Federal Register

Thursday
January 19, 1989

Part III

Department of Labor

Occupational Safety and Health Administration

29 CFR Part 1910

Air Contaminants; Final Rule

DEPARTMENT OF LABOR

Occupational Safety and Health Administration

29 CFR Part 1910

Air Contaminants

AGENCY: Occupational Safety and Health Administration, Labor.

ACTION: Final rule

SUMMARY: The Occupational Safety and Health Administration (OSHA) is amending its existing Air Contaminants standard, § 1910.1000 including Tables Z-1, Z-2 and Z-3. This amendment is limited to making more protective 212 Permissible Exposure Limits (PEL) listed in these three Tables; setting new PEL's for 164 substances not currently regulated by OSHA; and maintaining other PELs unchanged. Changes include revision of the PEL; inclusion of Short Term Exposure Limits (STEL) to complement 8 hour time weighted average (TWA) limits; establishment of skin designation; and addition of ceiling limits as appropriate.

All of the revised PELs are included in a single new Table Z-1-A which also includes the existing OSHA PELs under the Transitional Limits Columns. This regulation permits the use of any compliance methodology, until Dec. 31, 1992, to achieve the revised PEL. However, during this time period the established OSHA hierarchy of controls with preference for engineering controls will be applied to achieve the level of the transitional PELs. Tables Z-2 and Z-3 are temporarily maintained since they contain limits which cannot conveniently be included in the format used in Table Z-1-A.

OSHA has reviewed health, risk and feasibility evidence for all 428 substances for which changes to the PEL were considered. In each instance where a revised or new PEL is adopted, OSHA has determined that the new limits substantially reduce a significant risk of material health impairment among American workers, and that the new limits are technologically and economically feasible.

The revised standards will provide additional occupational health protection to 4.5 million workers at an annual cost of approximately $150 per employee protected. This cost is only a fraction of 1% of sales for all affected sectors.

DATES: This final rule shall become effective March 1, 1989. The start-up date for compliance with any combination of controls is September 1, 1989. The start-up date for compliance with preference for feasible engineering controls is December 31, 1992, or in certain circumstances December 31, 1993. See 29 CFR 1910.1000 (f) or Section X of the preamble.

ADDRESS: In compliance with 28 U.S.C. 2112(a), the Agency designates for receipt of petitions for review of the standard, the Associate Solicitor for Occupational Safety and Health, Office of the Solicitor, Room S-4004, U.S. Department of Labor, 200 Constitution Avenue NW., Washington, DC 20210.

FOR FURTHER INFORMATION CONTACT: Mr. James F. Foster, OSHA Office of Public Affairs, Room N-3647, Department of Labor, 200 Constitution Avenue NW., Washington, DC 20210 (202-523-8151). Copies of this document may be obtained two weeks after the publication date from the OSHA Publications Office, Rm. N-3101, at the above address (202-523-9667) or at any OSHA regional or area office.

SUPPLEMENTARY INFORMATION:

Organization of this Document

This **Federal Register** notice discusses health, feasibility, policy and legal issues, and includes amendments to 29 CFR 1910.1000, Tables Z-1, Z-2 and Z-3. All these amendments are included in a new Table Z-1-A which is part of Section X. Tables Z-2 and Z-3 are reprinted in Section X for reference purposes, and to assist during phased enforcement procedures. The preamble includes a discussion of the generic health effects for 18 individual groupings (e.g., neuropathic, ocular, cardiovascular, etc.) as well as a review of the health effects for all of the individual substances. It also includes the final feasibility and regulatory analysis with feasibility determinations organized by industry sector. All these discussions address the comments submitted to the public record for this rulemaking.

The Docket (H-020) includes considerable additional data, including many health studies, the complete preliminary and final feasibility and regulatory analysis with appendices, and additional feasibility information. This includes the final results of a large scale industry survey and many site visits. A four-volume printed version of this information, organized by substance, is also in the Docket. Also included in the record are extensive public comments which include additional health studies and feasibility analyses. The record includes 13 volumes of oral testimony and questioning of witnesses.

All this information is available for inspection and copying at the Docket Office. A list of exhibits is available in the Docket Office located in Room N-2634 at the above address, (202) 523-7894.

The preamble discussion is organized in the following manner:

I. Executive Summary

A. Background

Soon after adoption of the OSH Act in 1970, the Occupational Safety and Health Administration (OSHA) promulgated Permissible Exposure Limits (PELs) for many substances pursuant to the authority granted by section 6(a) which allowed the Agency to promulgate existing Federal Standards or national consensus standards as enforceable OSHA standards. Most of the PELs contained in the Z-Tables of 29 CFR 1910.1000 were adopted from the Walsh-Healey Public Contracts Act as existing Federal Standards. These in turn had been adopted from the 1968 Threshold Limit Values of the American Conference of Governmental Industrial Hygienists (ACGIH). Some consensus standards from the American Standards Association were also adopted at that time, following the 6(a) procedures.

Industrial experience, new developments in technology, and scientific data clearly indicate that in many instances these adopted limits are not sufficiently protective of worker health. In addition, there are no PELs for many toxic materials commonly used in the workplace. This has been demonstrated by the reduction in allowable exposure limits recommended by many technical, professional, industrial, and government organizations, both inside and outside the United States. In addition, these organizations have identified many other substances for which allowable exposure limits are needed to supplement the existing Z-Tables. Many large industrial organizations have felt obligated to supplement the existing OSHA PELs with their own internal corporate guidelines.

OSHA has focused its past priorities on the development of detailed and broad regulations for some high priority substances. This has resulted in major reductions in deleterious health effects for those 24 substances for which regulations have been adopted. However, OSHA has not been able to consider the need for regulating the thousands of substances commonly found in the workplace, or to review the scientific information to determine if different limits are required for the more than 400 substances now regulated under the provisions of the Z-Tables.

OSHA determined that it was necessary to modify this approach through the use of generic rulemaking, which would simultaneously cover many substances. The Hazard Communication Standard is an example of a regulation using such an approach. At this time, OSHA is also in the process of considering the need for development of generic standards to cover: Respiratory protection; medical surveillance; and exposure monitoring. Without a generic approach OSHA would not be able to provide the level of health protection required for many work situations.

OSHA concludes that it is of first priority to modify existing PELs, and to establish PELs for substances for which no exposure limits exist. The existing health literature and expert judgment indicates that such actions are required to protect against: Kidney and liver diseases; respiratory diseases; nerve disorders; carcinogenicity; irritation to various body organs; and many other material impairments to health. Millions of employees are potentially exposed to substances of concern, and adoption of such a regulation would represent one of the most significant steps to ensure the adequacy of health protection for workers. This regulation will achieve these objectives.

B. Proposal

On June 7, 1988, OSHA proposed to amend and expand the PELs for substances covered in the 29 CFR 1910.1000 Z-Tables and add new PELs to address this deficiency. To facilitate this major change for a large number of substances, OSHA initially considered available, generally accepted guidelines or recommendations as its starting point for establishing new PELs. Initially, this involved a review of 14 data bases which might serve this purpose. After analyses of the characteristics of each data base, compared to OSHA requirements, it was decided that OSHA would utilize the already published and widely accepted 1987-88 Threshold Limit Values (TLVs) published by the American Conference of Governmental Industrial Hygienists (ACGIH) and the Recommended Exposure Limits (RELs) developed by the National Institute for Occupational Safety and Health (NIOSH) as the starting point for its analysis. OSHA used both the TLVs and RELs as a starting point for making its own independent judgment regarding selection of the proper PEL. The TLV listing was used to define the bounds of substances included in this rulemaking.

The Proposal considered new PELs for 428 substances. OSHA reviewed the health evidence for each individual substance and preliminarily determined that available evidence would form a reasonable basis for proposing a new limit. It also preliminarily concluded that the new limits were technically and economically feasible. This proposed regulation was intended to reduce diseases (resulting from workplace exposure to chemicals) such as liver and kidney impairments; neuropathy; cardiovascular effects; respiratory effects; lung function deterioration; narcosis; biochemical and metabolic changes; and other material impairment of health. During the Public Hearing, extensive additional information was developed to permit OSHA to make a final determination of the health effects and risk associated with each substance under consideration for adoption of a new PEL.

OSHA also prepared a Preliminary Regulatory Impact Analysis (PRIA) which estimated average annual costs per establishment to achieve compliance, and total costs by industry sector. Preliminarily, OSHA determined that compliance with the proposed PELs would be technologically and economically feasible. As part of this analysis OSHA also identified health related benefits which would be achieved. These benefits included the reduction of occupational illness cases, lost workdays and fatalities.

C. Final Regulation

On the basis of all the information in the record, including the data upon which OSHA based its Proposal, public submissions, additional health and feasibility data (some of which became available during this rulemaking

process), additional analyses of all data, and consideration of the statutory requirements defined by the OSH Act, a revised set of PELs is issued in this regulation.

Through this regulation, the Occupational Safety and Health Administration (OSHA) is amending its existing Air Contaminants standards, § 1910.1000 including Tables Z-1, Z-2 and Z-3. This amendment is limited to changing many of the Permissible Exposure Limits (PEL) listed in these three Tables while maintaining other PELs unchanged. All PELs are listed in a new Table Z-1-A which replaces Table Z-1.

This amendment reduces the PEL for 212 substances now listed in the Z-Tables, and sets new PELs for 164 substances currently not regulated by OSHA. Changes include revision of the PEL; inclusion of Short Term Exposure Limits (STEL) to complement 8 hour time weighted average (TWA) limits; and, as appropriate, establishment of a skin designation and/or ceiling limits.

All of the revised PELs are included in a single new Table Z-1-A, which also includes the existing PELs enforced by OSHA. This side-by-side format is provided as a user convenience, and as a reference source since this regulation permits the use of any compliance procedures for the first 4 years following publication of the regulation. However, during this time period the established OSHA hierarchy of controls with preference for engineering controls will continue to be applied to achieve the level of the existing PELs.

Tables Z-2 and Z-3 are temporarily maintained since they cannot conveniently be included in the format for Table Z-1-A. The original Table Z-1 has been deleted from the regulation because all of the PELs in that Table have been included in the new Table Z-1-A. The design of this new Table Z-1-A makes identification of all changes to PELs possible by simply comparing Transitional Limits (left side of Table) with Revised Limits (right side of Table).

OSHA has reviewed health, risk and feasibility evidence for all 428 substances for which changes to the PEL were considered. In each instance where a revised or new PEL is adopted, OSHA has determined that the new limits substantially reduce a significant risk of material impairment of health or functional capacity among American workers, and that the new limits are technologically and economically feasible. This determination has been based on further review of the material discussed in the Proposal, public comments and a detailed review of the entire record for this rulemaking.

OSHA's analysis of all the data available following the issuance of the Proposal, receipt of comments and testimony during the public hearing resulted in changes to the proposed PELs. Details of these changes and determination of the PELs adopted in this regulation are included as part of the discussion of specific substances in Section VI. The changes noted above include:

(a) Reducing the PEL noted in the proposal;

(b) Increasing the PEL (not to exceed the existing Table Z-1 PEL) noted in the proposal; and

(c) Identifying the acceptability of respirators, due to feasibility considerations, to achieve compliance with the PEL for a small number of specific operations involving 4 substances.

The final Standard in 29 CFR 1910.1000 covers a total of 600 substances, this includes 428 substances for which OSHA opened the rulemaking process for consideration of revising or establishing new PELs.

(1) Addition of PELs for 164 new substances.

(2) Adoption of more protective PELs for 212 substances.

(3) No changes for 52 substances which were considered in this rulemaking.

In addition to these changes, the new final standard in 29 CFR 1910.1000 reprints existing exposure limits for the following substances which were either not covered or not considered for change in this rulemaking.

(a) No change to existing PELs for 9 substances which are currently undergoing 6(b) rulemaking.

(b) No change to existing PELs for 3 substances (benzene, cotton dust, and formaldehyde) where some segments are not covered by an individual 6(b) Regulation.

(c) PELs for 160 substances, which are unchanged, and were not evaluated during this rulemaking.

The final rule also includes minor changes to the introductory text, and definitions for the tabular listing of the new PELs in 29 CFR 1910.1000.

Specific changes between the Proposal and the final Regulation are noted below:

(A) *Reducing the PEL*
- (1) Camphor
- (2) Fluorine
- (3) Perchloroethylene

(B) *Increasing PEL (Less Than the Previous PEL in 29 CFR 1910.1000)*
- (1) Acetone
- (2) Acetonitrile
- (3) Ammonia
- (4) Borates
- (5) Carbon disulfide
- (6) Carbon tetrachloride
- (7) Chlorine
- (8) Chloroform
- (9) Grain dust
- (10) Mesityl Oxide
- (11) Methyl ethyl ketone peroxide
- (12) Trichloroethylene
- (13) Wood dust

(C) *Increasing PEL to Previously Existing Level in 29 CFR 1910.1000*
- (1) Acetic acid
- (2) Calcium oxide
- (3) Chromium metal
- (4) DDT
- (5) Iron oxide
- (6) Oil mist
- (7) o-Toluidine
- (8) Physical Irritants: 17 individual substances which might otherwise be classified as "Particulates Not Otherwise Regulated" (PNOR) and the generic PNOR classification. (See Section VI-C-10 for details).

(D) *No PEL*
- (1) Asphalt (delaying decision)
- (2) Chromyl chloride
- (3) Fibrous glass (delaying decision)
- (4) Mineral wool (delaying decision)

(E) *Increasing PEL*
- Carbon dioxide (adding STEL and also increasing TWA)

(F) *Special Respirator Provisions*
- (1) Carbon monoxide—Selected operations to meet the requirements of the STEL in the non-ferrous foundries and ferrous steel industry (SIC 33)
- (2) Carbon Disulfide—Selected Rayon Fiber Manufacturing Processes
- (3) Carbon Disulfide—Selected Sausage Casing Manufacturing Processes
- (4) Styrene—Selected Open Molding Boat Manufacturing Processes
- (5) Sulfur dioxide—Selected operations for meeting requirements of the STEL in the non-ferrous foundries and ferrous steel industry (SIC 33)

(G) *Deletion of Skin and STEL Limitations for Some Substances are Identified in Section VI*

Details of the rationale for changing these PELs is provided in the substance specific portions of Section VI. This includes general discussions of health effects in the introductory material to the individual sub-parts of Section VI, as well as detailed discussions for 428 substances.

The revised PELs will protect workers against a wide variety of health effects which could cause material impairment of health or functional capacity. This includes protection against catastrophic effects previously noted as well as more

subtle effects resulting in decrements to the central nervous system which produce significant sensory irritation. For each substance, the health evidence in the record provides an adequate basis for establishing a new or revised PEL.

Because of the nature of this rulemaking, OSHA relied heavily on the already published and widely accepted Threshold Limit Values (TLV) published by the American Conference of Governmental Industrial Hygienists (ACGIH) and the Recommended Exposure Limits (RELs) developed by the National Institute for Occupational Safety and Health (NIOSH). OSHA considered both the TLVs and RELs in making its own independent judgment regarding selection of the proper PEL.

Table Z-1-A is designed to include all substances covered by this regulation whether or not the PEL has been changed and whether or not a separate rulemaking is involved.

For four substances used in specific operations, the full record indicates that it is presently not technically feasible to achieve the PEL which is necessary (based on available health information) through engineering controls. For these few specific operations, the use of engineering controls to fully achieve the new PEL is required only where the Assistant Secretary demonstrates that such controls are feasible. In the absence of such a finding by the Assistant Secretary, the employer must use engineering controls to meet at least the level of the PEL existing prior to this revision as listed in Table Z-1-A (Transitional Limits columns), and Tables Z-2 and Z-3. However, any methods of control may be used in these identified situations to achieve the new PELs noted in Table Z-1-A. The specific operating situations falling in this category are identified in the individual substance discussions in Section VI, and the general concept is discussed in the Summary and Explanation of the Standard (Section VIII).

A phased enforcement schedule of 6 months (any control methods) following the March 1, 1989, effective date and approximately 4 years (December 31, 1992) following the regulation publication date (engineering controls preferred) is adopted. In certain circumstances, the December 31, 1992, deadline may be extended to December 31, 1993. See 29 CFR 1910.1000(f) in Section X of this preamble.

The final regulation is limited to consideration of revising the PELs. There is no consideration of the ancillary requirements which are typically developed as part of individual substance rulemaking but were not included in the original § 1910.1000 standard. OSHA has published ANPR's for Exposure Monitoring (53 FR 32591-32595), and Medical Surveillance (53 FR 32595-32598), and is developing a proposal covering revision to the respirator provisions of the OSHA Standards. OSHA has issued a final rule expanding the Hazard Communication Standard.

While medical surveillance, exposure monitoring and other industrial hygiene practices are important, OSHA is not in a position to develop these requirements while at the same time developing PELs for several hundred substances. OSHA has determined that lowering exposures through the development of reduced PELs is of higher priority because it is more effective in reducing occupational diseases and material impairment of health. These ancillary requirements will be addressed as priorities dictate.

OSHA has also determined that it is appropriate to limit this rulemaking to the General Industry sector. Application to the Construction, Maritime and Agriculture Segments may require some modifications to this proposed rule because of differences in exposures and work situations in the established PELs for these segments, and differences regarding feasibility for these sectors. OSHA will pursue this as part of second stage rulemaking and has informally notified the Construction Advisory Committee of its plans.

The average annual cost, per establishment affected by this rule, is estimated to range from $77,000 for petroleum refining (SIC 29) down to $400 per year for auto dealers (SIC 55). The annual cost is approximately $150 per worker protected, and is never more than a fraction of 1% of sales and less than 2% of profits (usually substantially less) except for a very few segments. Benefits will accrue to approximately 4.5 million workers who are currently exposed in excess of the PEL and are expected to include the reduction of over 55,000 occupational illness cases, including almost 24,000 lost workday illness cases and approximately 520,000 lost workdays annually. If not prevented, these illnesses would eventually result in approximately 700 fatalities each year.

OSHA will continue its practice of rulemaking for individual substances when substance specific regulations are necessary and appropriate. An expanded discussion is provided in Section IV-D.

OSHA has also considered the concerns identified regarding the need for extensively tested analytical methods (Ex. 3-960; Ex. 8-47) for enforcement purposes. OSHA believes that enforcement can be initiated without such detailed methods. The OSHA docket includes: (1) Reference to a fully developed and extensively tested OSHA or NIOSH sampling and analytical procedure or, (2) a description of an OSHA in-house sampling and analytical method for all but the seven substances listed in Table IV-E-1. OSHA therefore believes there will be no problems with enforcement of the PELs for all but seven substances. This is consistent with conclusions of NIOSH regarding implementation (Ex. 8-47). Since development of sampling and analytical procedures is a dynamic, rapidly progressing technology, OSHA also believes it is appropriate to adopt PELs for these seven substances, but stay enforcement of these PELs until adequate sampling and analytical methods are available. At such time, OSHA will publish in the **Federal Register** its determination that such methods exist (together with a copy of the method), and indicate the proposed effective date for enforcement of the PEL for the substance in question.

As resources permit, OSHA will attempt to initiate a program in conjunction with NIOSH to develop more extensively tested sampling and analytical methods for those substances where only in-house methods are noted in the Proposal. OSHA believes that this balanced approach is consistent with the statutory requirements of the OSH Act.

II. Index to Preamble Discussion of Individual Substances

The table below provides an index by preamble section and subsection to a discussion of the record and the health effects evidence for each of the 428 substances for which new or revised limits were considered. The substances in the index are arranged in alphabetical order and include H.S. and CAS numbers as well as the principal toxicological or other basis for the selection or revision of each limit. For some of these substances, OSHA determined that no change to the existing PEL was warranted. Section X presents the entire standard and includes Table Z-1-A, which shows the new and revised limits as well as those OSHA limits that were not changed by this rulemaking.

BILLING CODE 4510-26-M

II. Index to Preamble Discussion of Individual Substances

II. S Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1001	ACETALDEHYDE	75-07-0	SENSORY IRRITATION	VI.C.3
1002	ACETIC ACID	64-19-7	SENSORY IRRITATION	VI.C.3
1003	ACETIC ANHYDRIDE	108-24-7	ANALOGY	VI.C.12
1004	ACETONE	67-64-1	SENSORY IRRITATION	VI.C.3
1005	ACETONITRILE	75-05-8	SYSTEMIC TOXICITY	VI.C.8
1006	ACETYLSALICYLIC ACID (ASPIRIN)	50-78-2	SYSTEMIC TOXICITY	VI.C.8
1007	ACROLEIN	107-02-8	SENSORY IRRITATION	VI.C.3
1008	ACRYLAMIDE	79-06-1	CANCER	VI.C.15
1009	ACRYLIC ACID	79-10-7	ANALOGY	VI.C.12
1010	ALLYL ALCOHOL	107-18-6	SENSORY IRRITATION	VI.C.3
1011	ALLYL CHLORIDE	107-05-1	LIVER AND KIDNEY EFFECTS	VI.C.4
1012	ALLYL GLYCIDYL ETHER (AGE)	106-92-3	SENSORY IRRITATION	VI.C.3
1013	ALLYL PROPYL DISULFIDE	2179-59-1	SENSORY IRRITATION	VI.C.3
1014	ALPHA-ALUMINA	1344-28-1	PHYSICAL IRRITATION	VI.C.10
1015	ALUMINUM (ALKYLS)	7429-90-5	ANALOGY	VI.C.12
1016	ALUMINUM (METAL)	7429-90-5	PHYSICAL IRRITATION	VI.C.10
1017	ALUMINUM (PYRO POWDERS)	7429-90-5	RESPIRATORY EFFECTS	VI.C.6
1018	ALUMINUM (SOLUBLE SALTS)	7429-90-5	ANALOGY	VI.C.12
1019	ALUMINUM (WELDING FUMES)	7429-90-5	SYSTEMIC TOXICITY	VI.C.8

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1020	AMITROLE (3-AMINO-1,2,4-TRIAZOLE)	61-82-5	CANCER	VI.C.15
1021	AMMONIA	7664-41-7	SENSORY IRRITATION	VI.C.3
1022	AMMONIUM CHLORIDE (FUME)	12125-02-9	SENSORY IRRITATION	VI.C.3
1024	AMMONIUM SULFAMATE (AMMATE)	7773-06-0	PHYSICAL IRRITATION	VI.C.10
1025	ANILINE	62-53-3	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1028	ASPHALT FUMES	8052-42-4	CANCER	VI.C.15
1029	ATRAZINE	1912-24-9	NOAELs	VI.C.9
1031	BARIUM SULFATE	7727-43-7	PHYSICAL IRRITATION	VI.C.10
1032	BENOMYL	17804-35-2	PHYSICAL IRRITATION	VI.C.10
1033	BERYLLIUM & COMPOUNDS	7440-41-7	CANCER	VI.C.15
1034	BISMUTH TELLURIDE (SE-DOPED)	1304-82-1	RESPIRATORY EFFECTS	VI.C.6
1035	BISMUTH TELLURIDE (UNDOPED)	1304-82-1	PHYSICAL IRRITATION	VI.C.10
1036	BORATES, TETRA, SODIUM (ANHYDROUS)	1330-43-4	SENSORY IRRITATION	VI.C.3
1037	BORATES, TETRA, SODIUM (DECAHYDRATE)	1303-96-4	SENSORY IRRITATION	VI.C.3
1038	BORATES, TETRA, SODIUM (PENTAHYDRATE)	12179-04-3	SENSORY IRRITATION	VI.C.3

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1039	BORON OXIDE	1303-86-2	PHYSICAL IRRITATION	VI.C.10
1040	BORON TRIBROMIDE	10294-33-4	ANALOGY	VI.C.12
1041	BROMACIL	314-40-9	NOAELs	VI.C.9
1042	BROMINE	7726-95-6	SENSORY IRRITATION	VI.C.3
1043	BROMINE PENTAFLUORIDE	7789-30-2	ANALOGY	VI.C.12
1044	BUTANE	106-97-8	NARCOSIS	VI.C.2
1045	2-BUTANONE (MEK)	78-93-3	SENSORY IRRITATION	VI.C.3
1046	2-BUTOXY ETHANOL	111-76-2	SYSTEMIC TOXICITY	VI.C.8
1047	N-BUTYL ACETATE	123-86-4	SENSORY IRRITATION	VI.C.3
1048	BUTYL ACRYLATE	141-32-2	ANALOGY	VI.C.12
1049	SEC-BUTYL ALCOHOL	78-92-2	NARCOSIS	VI.C.2
1050	TERT-BUTYL ALCOHOL	75-65-0	NARCOSIS	VI.C.2
1051	N-BUTYL ALCOHOL	71-36-3	NEUROPATHY	VI.C.1
1052	N-BUTYL GLYCIDYL ETHER (BGE)	2426-08-6	SYSTEMIC TOXICITY	VI.C.8
1053	N-BUTYL LACTATE	138-22-7	SENSORY IRRITATION	VI.C.3
1054	BUTYL MERCAPTAN	109-79-5	SENSORY IRRITATION	VI.C.3
1055	O-SEC-BUTYL PHENOL	89-72-5	ANALOGY	VI.C.12
1056	P-TERT-BUTYLTOLUENE	98-51-1	NOAELs	VI.C.9

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1057	CALCIUM CARBONATE	1317-65-3	PHYSICAL IRRITATION	VI.C.10
1058	CALCIUM CYANAMIDE	156-62-7	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1059	CALCIUM HYDROXIDE	1305-62-0	ANALOGY	VI.C.12
1060	CALCIUM OXIDE	1305-78-8	ANALOGY	VI.C.12
1061	CALCIUM SILICATE, TOTAL DUST	1344-95-2	PHYSICAL IRRITATION	VI.C.10
1062	CALCIUM SULFATE	7778-18-9	PHYSICAL IRRITATION	VI.C.10
1063	CAMPHOR (SYNTHETIC)	76-22-2	INCREASING PEL	VI.C.16
1064	CAPROLACTAM (DUST)	105-60-2	SENSORY IRRITATION	VI.C.3
1065	CAPROLACTAM (VAPOR)	105-60-2	SENSORY IRRITATION	VI.C.3
1066	CAPTAFOL (DIFOLATAN)	2425-06-1	SENSITIZATION EFFECTS	VI.C.14
1067	CAPTAN	133-06-2	SYSTEMIC TOXICITY	VI.C.8
1068	CARBOFURAN (FURADAN)	1563-66-2	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1069	CARBON DIOXIDE	124-38-9	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1070	CARBON DISULFIDE	75-15-0	CARDIOVASCULAR EFFECTS	VI.C.7
1071	CARBON MONOXIDE	630-08-0	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1072	CARBON TETRABROMIDE	558-13-4	LIVER AND KIDNEY EFFECTS	VI.C.4
1073	CARBON TETRACHLORIDE	56-23-5	CANCER	VI.C.15
1074	CARBONYL FLUORIDE	353-50-4	ANALOGY	VI.C.12

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1075	CATECHOL (PYROCATECHOL)	120-80-9	ANALOGY	VI.C.12
1076	CELLULOSE	9004-34-6	PHYSICAL IRRITATION	VI.C.10
1077	CESIUM HYDROXIDE	21351-79-1	SENSORY IRRITATION	VI.C.3
1078	CHLORINATED CAMPHENE	8001-35-2	NEUROPATHY	VI.C.1
1079	CHLORINE	7782-50-5	SENSORY IRRITATION	VI.C.3
1080	CHLORINE DIOXIDE	10049-04-4	RESPIRATORY EFFECTS	VI.C.6
1081	1-CHLORO-1-NITROPROPANE	600-25-9	ANALOGY	VI.C.12
1082	2-CHLORO-6-TRICHLOROMETHYL PYRIDINE (NITRAPYRIN)	1929-82-4	PHYSICAL IRRITATION	VI.C.10
1083	CHLOROACETYL CHLORIDE	79-04-9	SENSORY IRRITATION	VI.C.3
1084	O-CHLOROBENZYLIDENE MALONONITRILE	2698-41-1	SENSORY IRRITATION	VI.C.3
1085	CHLORODIFLUOROMETHANE	75-45-6	NOAELs	VI.C.9
1086	CHLOROFORM	67-66-3	CANCER	VI.C.15
1087	CHLOROPENTAFLUOROETHANE	76-15-3	CARDIOVASCULAR EFFECTS	VI.C.7
1088	CHLOROPRENE	126-99-8	SYSTEMIC TOXICITY	VI.C.8
1089	O-CHLOROSTYRENE	2039-87-4	LIVER AND KIDNEY EFFECTS	VI.C.4
1090	O-CHLOROTOLUENE	95-49-8	NOAELs	VI.C.9
1091	CHLORPYRIFOS	2921-88-2	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1092	CHROMIC ACID & CHROMATES	Varies	CANCER	VI.C.15
1093	CHROMIUM, METAL	7440-47-3	RESPIRATORY EFFECTS	VI.C.6
1094	CHROMYL CHLORIDE	14977-61-8	CANCER	VI.C.15
1095	CLOPIDOL (COYDEN)	2971-90-6	PHYSICAL IRRITATION	VI.C.10
1096	COAL DUST, < 5% QUARTZ	None	RESPIRATORY EFFECTS	VI.C.6
1097	COAL DUST, > 5% QUARTZ	None	RESPIRATORY EFFECTS	VI.C.6
1098	COBALT CARBONYL	10210-68-1	ANALOGY	VI.C.12
1099	COBALT HYDROCARBONYL	16842-03-8	ANALOGY	VI.C.12
1100	COBALT METAL, FUME, DUST	7440-48-4	SENSITIZATION EFFECTS	VI.C.14
1101	COPPER (FUME)	7440-50-8	INCREASING PEL	VI.C.16
1102	CRAG HERBICIDE (SESONE)	136-78-7	PHYSICAL IRRITATION	VI.C.10
1103	CRUFOMATE	299-86-5	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1104	CYANAMIDE	420-04-2	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1105	CYANOGEN	460-19-5	SENSORY IRRITATION	VI.C.3
1106	CYANOGEN CHLORIDE	506-77-4	SENSORY IRRITATION	VI.C.3
1107	CYCLOHEXANOL	108-93-0	CHANGE IN SKIN DESIGNATION ONLY	VI.C.18
1108	CYCLOHEXANONE	108-94-1	LIVER AND KIDNEY EFFECTS	VI.C.4
1109	CYCLOHEXYLAMINE	108-91-8	SYSTEMIC TOXICITY	VI.C.8

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1110	CYCLONITE	121-82-4	NOAELs	VI.C.9
1111	CYCLOPENTANE	287-92-3	NARCOSIS	VI.C.2
1112	CYHEXATIN	13121-70-5	SYSTEMIC TOXICITY	VI.C.8
1113	DDT	50-29-3	SYSTEMIC TOXICITY	VI.C.8
1114	DECABORANE	17702-41-9	NEUROPATHY	VI.C.1
1116	DI-SEC-OCTYL-PHTHALATE	117-81-7	NEUROPATHY	VI.C.1
1117	2,6-DI-TERT-BUTYL-P-CRESOL	128-37-0	NOAELs	VI.C.9
1118	DIAZINON	333-41-5	ANALOGY	VI.C.12
1119	DIBUTYL PHOSPHATE	107-66-4	SENSORY IRRITATION	VI.C.3
1120	2-N-DIBUTYLAMINOETHANOL	102-81-8	SYSTEMIC TOXICITY	VI.C.8
1121	1,1-DICHLORO-1-NITROETHANE	594-72-9	ANALOGY	VI.C.12
1122	1,3-DICHLORO-5, 5-DIMETHYLHYDANTOIN	118-52-5	SENSORY IRRITATION	VI.C.3
1123	DICHLOROACETYLENE	7572-29-4	NEUROPATHY	VI.C.1
1125	P-DICHLOROBENZENE	106-46-7	ANALOGY	VI.C.12
1126	1,1-DICHLOROETHANE	75-34-3	INCREASING PEL	VI.C.16
1127	DICHLOROETHYL ETHER	111-44-4	SENSORY IRRITATION	VI.C.3
1128	DICHLOROMONOFLUOROMETHANE	75-43-4	ANALOGY	VI.C.12

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1129	1,3-DICHLOROPROPENE	542-75-6	LIVER AND KIDNEY EFFECTS	VI.C.4
1130	2,2-DICHLOROPROPIONIC ACID	75-99-0	SENSORY IRRITATION	VI.C.3
1131	DICROTOPHOS (BIDRIN)	141-66-2	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1132	DICYCLOPENTADIENE	77-73-6	LIVER AND KIDNEY EFFECTS	VI.C.4
1133	DICYCLOPENTADIENYL IRON	102-54-5	PHYSICAL IRRITATION	VI.C.10
1134	DIETHANOLAMINE	111-42-2	NOAELs	VI.C.9
1135	DIETHYL KETONE	96-22-0	ANALOGY	VI.C.12
1136	DIETHYLPHTHALATE	84-66-2	NOAELs	VI.C.9
1137	DIETHYLAMINE	109-89-7	SENSORY IRRITATION	VI.C.3
1138	DIETHYLENE TRIAMINE	111-40-0	ANALOGY	VI.C.12
1139	DIGLYCIDYL ETHER (DGE)	2238-07-5	SYSTEMIC TOXICITY	VI.C.8
1140	DIISOBUTYL KETONE	108-83-8	SENSORY IRRITATION	VI.C.3
1141	DIMETHYL 1,2-DIBROMO-2, 2-DICHLOROETHYL PHOSPHATE	300-76-5	CHANGE IN SKIN DESIGNATION ONLY	VI.C.18
1142	DIMETHYL SULFATE	77-78-1	CANCER	VI.C.15
1143	DIMETHYLANILINE	121-69-7	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1144	DINITOLMIDE (3,5-DINITRO-O-TOLUAMIDE)	148-01-6	NOAELs	VI.C.9

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1145	DIOXANE (DIETHYLENE DIOXIDE)	123-91-1	LIVER AND KIDNEY EFFECTS	VI.C.4
1146	DIOXATHION (DELNAV)	78-34-2	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1147	DIPHENYLAMINE	122-39-4	NOAELs	VI.C.9
1148	DIPROPYL KETONE	123-19-3	ANALOGY	VI.C.12
1149	DIPROPYLENE GLYCOL METHYL ETHER	34590-94-8	NEUROPATHY	VI.C.1
1150	DIQUAT	85-00-7	ANALOGY	VI.C.12
1151	DISULFIRAM	97-77-8	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1152	DISULFOTON	298-04-4	ANALOGY	VI.C.12
1153	DIURON	330-54-1	NOAELs	VI.C.9
1154	DIVINYL BENZENE	108-57-6	ANALOGY	VI.C.12
1155	EMERY	112-62-9	PHYSICAL IRRITATION	VI.C.10
1156	ENDOSULFAN	115-29-7	ANALOGY	VI.C.12
1158	EPICHLOROHYDRIN	106-89-8	SENSORY IRRITATION	VI.C.3
1159	ETHANOLAMINE	141-43-5	SYSTEMIC TOXICITY	VI.C.8
1160	ETHION (NIALATE)	563-12-2	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1161	ETHYL ACRYLATE	140-88-5	RESPIRATORY EFFECTS	VI.C.6
1162	ETHYL BENZENE	100-41-4	SENSORY IRRITATION	VI.C.3
1163	ETHYL BROMIDE	74-96-4	NARCOSIS	VI.C.2

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1164	ETHYL ETHER	60-29-7	SENSORY IRRITATION	VI.C.3
1165	ETHYL MERCAPTAN	75-08-1	SENSORY IRRITATION	VI.C.3
1166	ETHYL SILICATE	78-10-4	LIVER AND KIDNEY EFFECTS	VI.C.4
1167	ETHYLENE CHLOROHYDRIN	107-07-3	SYSTEMIC TOXICITY	VI.C.8
1168	ETHYLENE DICHLORIDE (1,2-DICHLOROETHANE)	107-06-2	LIVER AND KIDNEY EFFECTS	VI.C.4
1169	ETHYLENE GLYCOL	107-21-1	SENSORY IRRITATION	VI.C.3
1170	ETHYLENE GLYCOL DINITRATE	628-96-6	CARDIOVASCULAR EFFECTS	VI.C.7
1171	ETHYLIDENE NORBORNENE	16219-75-3	SENSORY IRRITATION	VI.C.3
1172	N-ETHYLMORPHOLINE	100-74-3	OCULAR EFFECTS	VI.C.5
1173	FENAMIPHOS	22224-92-6	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1174	FENSULFOTHION (DASANIT)	115-90-2	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1175	FENTHION	55-38-9	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1176	FERBAM	14484-64-1	PHYSICAL IRRITATION	VI.C.10
1177	FERROVANADIUM DUST	12604-58-9	RESPIRATORY EFFECTS	VI.C.6
1178	FIBROUS GLASS DUST	None	RESPIRATORY EFFECTS	VI.C.6
1179	FLUORINE	7782-41-4	INCREASING PEL	VI.C.16
1180	FLUOROTRICHLOROMETHANE	75-69-4	CARDIOVASCULAR EFFECTS	VI.C.7

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1181	FONOFOS	944-22-9	ANALOGY	VI.C.12
1182	FORMAMIDE	75-12-7	ANALOGY	VI.C.12
1183	FURFURAL	98-01-1	SENSORY IRRITATION	VI.C.3
1184	FURFURYL ALCOHOL	98-00-0	SENSORY IRRITATION	VI.C.3
1185	GASOLINE	8006-61-9	NARCOSIS	VI.C.2
1186	GERMANIUM TETRAHYDRIDE	7782-65-2	ANALOGY	VI.C.12
1187	GLUTARALDEHYDE	111-30-8	SENSORY IRRITATION	VI.C.3
1188	GLYCERIN (MIST)	56-81-5	PHYSICAL IRRITATION	VI.C.10
1189	GLYCIDOL (2,3-EPOXY-1-PROPANOL)	556-52-5	SYSTEMIC TOXICITY	VI.C.8
1190	GRAIN DUST	None	RESPIRATORY EFFECTS	VI.C.6
1191	GRAPHITE, NATURAL, RESPIRABLE	7782-42-5	RESPIRATORY EFFECTS	VI.C.6
1191A	GRAPHITE, SYNTHETIC	None	PHYSICAL IRRITATION	VI.C.10
1192	GYPSUM, TOTAL DUST	7778-18-9	PHYSICAL IRRITATION	VI.C.10
1194	N-HEPTANE	142-82-5	NARCOSIS	VI.C.2
1195	HEXACHLOROBUTADIENE	87-68-3	LIVER AND KIDNEY EFFECTS	VI.C.4
1196	HEXACHLOROCYCLOPENTADIENE	77-47-4	SENSORY IRRITATION	VI.C.3
1197	HEXACHLOROETHANE	67-72-1	INCREASING PEL	VI.C.16
1198	HEXAFLUOROACETONE	684-16-2	SYSTEMIC TOXICITY	VI.C.8

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1200	N-HEXANE	110-54-3	NEUROPATHY	VI.C.1
1201	HEXANE ISOMERS	Varies	NARCOSIS	VI.C.2
1202	2-HEXANONE	591-78-6	NEUROPATHY	VI.C.1
1203	HEXONE (METHYL ISOBUTYL KETONE)	108-10-1	LIVER AND KIDNEY EFFECTS	VI.C.4
1204	HEXYLENE GLYCOL	107-41-5	SENSORY IRRITATION	VI.C.3
1205	HYDRAZINE	302-01-2	LIVER AND KIDNEY EFFECTS	VI.C.4
1206	HYDROGEN BROMIDE	10035-10-6	SENSORY IRRITATION	VI.C.3
1207	HYDROGEN CYANIDE	74-90-8	SYSTEMIC TOXICITY	VI.C.8
1208	HYDROGEN FLUORIDE	7664-39-3	SENSORY IRRITATION	VI.C.3
1209	HYDROGEN SULFIDE	7783-06-4	OCULAR EFFECTS	VI.C.5
1210	HYDROGENATED TERPHENYLS	61788-32-7	SYSTEMIC TOXICITY	VI.C.8
1211	2-HYDROXYPROPYL ACRYLATE	999-61-1	SENSORY IRRITATION	VI.C.3
1212	INDENE	95-13-6	ANALOGY	VI.C.12
1213	INDIUM & COMPOUNDS	7440-74-6	RESPIRATORY EFFECTS	VI.C.6
1214	IODOFORM	75-47-8	ANALOGY	VI.C.12
1215	IRON OXIDE (DUST AND FUME)	1309-37-1	RESPIRATORY EFFECTS	VI.C.6
1216	IRON PENTACARBONYL	13463-40-6	NEUROPATHY	VI.C.1
1217	IRON SALTS (SOLUBLE)	Varies	SENSORY IRRITATION	VI.C.3

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1218	ISOAMYL ALCOHOL	123-51-3	NARCOSIS	VI.C.2
1219	ISOBUTYL ALCOHOL	78-83-1	ANALOGY	VI.C.12
1220	ISOOCTYL ALCOHOL	26952-21-6	ANALOGY	VI.C.12
1221	ISOPHORONE	78-59-1	NARCOSIS	VI.C.2
1222	ISOPHORONE DIISOCYANATE	4098-71-9	SENSITIZATION EFFECTS	VI.C.14
1223	2-ISOPROPOXYETHANOL	109-59-1	SYSTEMIC TOXICITY	VI.C.8
1224	ISOPROPYL ACETATE	108-21-4	SENSORY IRRITATION	VI.C.3
1225	ISOPROPYL ALCOHOL	67-63-0	SENSORY IRRITATION	VI.C.3
1226	ISOPROPYL ETHER	108-20-3	ODOR EFFECTS	VI.C.11
1227	ISOPROPYL GLYCIDYL ETHER	4016-14-2	SYSTEMIC TOXICITY	VI.C.8
1228	ISOPROPYLAMINE	75-31-0	SENSORY IRRITATION	VI.C.3
1229	N-ISOPROPYLANILINE	768-52-5	ANALOGY	VI.C.12
1230	KAOLIN, TOTAL DUST	None	PHYSICAL IRRITATION	VI.C.10
1231	KETENE	463-51-4	ANALOGY	VI.C.12
1232	LIMESTONE, TOTAL DUST	1317-67-3	PHYSICAL IRRITATION	VI.C.10
1233	MAGNESITE, TOTAL DUST	546-93-0	PHYSICAL IRRITATION	VI.C.10
1234	MAGNESIUM OXIDE FUME	1309-48-4	PHYSICAL IRRITATION	VI.C.10
1235	MALATHION	121-75-5	PHYSICAL IRRITATION	VI.C.10

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1236A	MANGANESE, FUME	7439-96-5	NEUROPATHY	VI C.1
1237	MANGANESE CYCLOPENTADIENYL TRICARBONYL	12079-65-1	NEUROPATHY	VI.C.1
1238	MANGANESE TETROXIDE	1317-35-7	NEUROPATHY	VI.C.1
1239	MARBLE, TOTAL DUST	1317-65-3	PHYSICAL IRRITATION	VI.C.10
1240	MERCURY (ARYL AND INORGANIC COMPOUNDS)	7439-97-6	NEUROPATHY	VI.C.1
1241	MERCURY (VAPOR)	7439-97-6	NEUROPATHY	VI.C.1
1242	MERCURY, (ORGANO) ALKYL COMPOUNDS	7439-97-6	NEUROPATHY	VI.C.1
1243	MESITYL OXIDE	141-79-7	SENSORY IRRITATION	VI.C.3
1244	METHACRYLIC ACID	79-41-4	ANALOGY	VI.C.12
1245	METHOMYL (LANNATE)	16752-77-5	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1246	METHOXYCHLOR	72-43-5	PHYSICAL IRRITATION	VI.C.10
1247	4-METHOXYPHENOL	150-76-5	ANALOGY	VI.C.12
1248	METHYL 2-CYANOACRYLATE	137-05-3	SENSORY IRRITATION	VI.C.3
1249	METHYL ACETATE	79-20-9	NOAELs	VI.C.9
1250	METHYL ACETYLENE/PROPADIENE MIXTURE	None	ANALOGY	VI.C.12

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H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1251	METHYL ACRYLONITRILE	126-98-7	NEUROPATHY	VI.C.1
1252	METHYL ALCOHOL	67-56-1	OCULAR EFFECTS	VI.C.5
1253	METHYL BROMIDE	74-83-9	NEUROPATHY	VI.C.1
1254	METHYL CHLORIDE	74-87-3	NARCOSIS	VI.C.2
1255	METHYL CHLOROFORM (1,1,1-TRICHLOROETHANE)	71-55-6	NARCOSIS	VI.C.2
1256	METHYL DEMETON	8022-00-2	ANALOGY	VI.C.12
1257	METHYL ETHYL KETONE PEROXIDE	1338-23-4	ANALOGY	VI.C.12
1258	METHYL FORMATE	107-31-3	ANALOGY	VI.C.12
1259	METHYL IODIDE	74-88-4	ANALOGY	VI.C.12
1260	METHYL ISOAMYL KETONE	110-12-3	ANALOGY	VI.C.12
1261	METHYL ISOBUTYL CARBINOL	108-11-2	SENSORY IRRITATION	VI.C.3
1262	METHYL ISOPROPYL KETONE	563-80-4	ANALOGY	VI.C.12
1263	METHYL MERCAPTAN	74-93-1	SENSORY IRRITATION	VI.C.3
1264	METHYL N-AMYL KETONE	110-43-0	SENSORY IRRITATION	VI.C.3
1265	METHYL PARATHION	298-00-0	ANALOGY	VI.C.12
1266	METHYL SILICATE	681-84-5	OCULAR EFFECTS	VI.C.5
1267	ALPHA-METHYL STYRENE	98-83-9	SENSORY IRRITATION	VI.C.3

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1268	METHYLCYCLOHEXANE	108-87-2	ANALOGY	VI.C.12
1269	METHYLCYCLOHEXANOL	25639-42-3	LIVER AND KIDNEY EFFECTS	VI.C.4
1270	O-METHYLCYCLOHEXANONE	583-60-8	SENSORY IRRITATION	VI.C.3
1271	METHYLCYCLOPENTADIENYL MN TRICARBONYL	12108-13-3	ANALOGY	VI.C.12
1272	METHYLENE BIS (4-CYCLOHEXYLISOCYANATE)	5124-30-1	RESPIRATORY EFFECTS	VI.C.6
1273	4,4'-METHYLENE BIS (2-CHLOROANILINE)	101-14-4	SYSTEMIC TOXICITY	VI.C.8
1275	METRIBUZIN	21087-64-9	NOAELs	VI.C.9
1276	MICA	12001-26-2	RESPIRATORY EFFECTS	VI.C.6
1277	MINERAL WOOL FIBER	None	RESPIRATORY EFFECTS	VI.C.6
1278	MOLYBDENUM (INSOLUBLE COMPOUNDS)	7439-98-7	PHYSICAL IRRITATION	VI.C.10
1279	MONOCROTOPHOS (AZODRIN)	6923-22-4	ANALOGY	VI.C.12
1280	MONOMETHYL ANILINE	100-61-8	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1281	MORPHOLINE	110-91-8	ANALOGY	VI.C.12
1282	NAPHTHALENE	91-20-3	OCULAR EFFECTS	VI.C.5
1283	NICKEL (SOLUBLE COMPOUNDS)	7440-02-0	RESPIRATORY EFFECTS	VI.C.6

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H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1284	NICKEL CARBONYL	13463-39-3	INCREASING PEL	VI.C.16
1286	NITRIC ACID	7697-37-2	ANALOGY	VI.C.12
1287	P-NITROANILINE	100-01-6	ANALOGY	VI.C.12
1288	P-NITROCHLOROBENZENE	100-00-5	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1289	NITROGEN DIOXIDE	10102-44-0	RESPIRATORY EFFECTS	VI.C.6
1290	NITROGLYCERIN	55-63-0	CARDIOVASCULAR EFFECTS	VI.C.7
1291	2-NITROPROPANE	79-46-9	CANCER	VI.C.15
1292	NITROTOLUENE (ALL ISOMERS)	99-08-1, 99-99-0, 88-72-2	ANALOGY	VI.C.12
1293	NONANE	111-84-2	ANALOGY	VI.C.12
1295	OCTACHLORONAPHTHALENE	2234-13-1	LIVER AND KIDNEY EFFECTS	VI.C.4
1296	OCTANE	111-65-9	NARCOSIS	VI.C.2
1297	OIL MIST (MINERAL)	8012-95-1	NOAELs	VI.C.9
1298	OSMIUM TETROXIDE	20816-12-0	SENSORY IRRITATION	VI.C.3
1299	OXALIC ACID	144-62-7	ANALOGY	VI.C.12
1300	OXYGEN DIFLUORIDE	7783-41-7	RESPIRATORY EFFECTS	VI.C.6
1301	OZONE	10028-15-6	RESPIRATORY EFFECTS	VI.C.6
1302	PARAFFIN WAX FUME	8002-74-2	SENSORY IRRITATION	VI.C.3

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1303	PARAQUAT, RESPIRABLE DUST	4685-14-7	RESPIRATORY EFFECTS	VI.C.6
1294	PARTICULATES NOT OTHERWISE REGULATED	None	PHYSICAL IRRITATION	VI.C.10
1304	PENTABORANE	19624-22-7	NEUROPATHY	VI.C.1
1305	PENTAERYTHRITOL, TOTAL DUST	115-77-5	PHYSICAL IRRITATION	VI.C.10
1306	PENTANE	109-66-0	NARCOSIS	VI.C.2
1307	2-PENTANONE (METHYL PROPYL KETONE)	107-87-9	NARCOSIS	VI.C.2
1308	PERCHLOROETHYLENE	127-18-4	CANCER	VI.C.15
1309	PERCHLORYL FLUORIDE	7616-94-6	ANALOGY	VI.C.12
1310	PERLITE	None	PHYSICAL IRRITATION	VI.C.10
1312	PETROLEUM DISTILLATES (NAPHTHA)	None	NOAELs	VI.C.9
1313	PHENOTHIAZINE	92-84-2	SENSITIZATION EFFECTS	VI.C.14
1314	PHENYL ETHER (VAPOR)	101-84-8	ODOR EFFECTS	VI.C.11
1315	PHENYL GLYCIDYL ETHER	122-60-1	SENSITIZATION EFFECTS	VI.C.14
1316	PHENYL MERCAPTAN	108-98-5	NEUROPATHY	VI.C.1
1317	PHENYLHYDRAZINE	100-63-0	SYSTEMIC TOXICITY	VI.C.8
1318	PHENYLPHOSPHINE	638-21-1	SYSTEMIC TOXICITY	VI.C.8
1319	PHORATE (THIMET)	298-02-2	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1320	PHOSDRIN (MEVINPHOS)	7786-34-7	ANALOGY	VI.C.12

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1321	PHOSPHINE	7803-51-2	SYSTEMIC TOXICITY	VI.C.8
1322	PHOSPHORIC ACID	7664-38-2	SENSORY IRRITATION	VI.C.3
1323	PHOSPHORUS OXYCHLORIDE	10025-87-3	ANALOGY	VI.C.12
1324	PHOSPHORUS PENTASULFIDE	1314-80-3	ANALOGY	VI.C.12
1325	PHOSPHORUS TRICHLORIDE	7719-12-2	SENSORY IRRITATION	VI.C.3
1326	PHTHALIC ANHYDRIDE	85-44-9	ANALOGY	VI.C.12
1327	M-PHTHALODINITRILE	626-17-5	NOAELs	VI.C.9
1328	PICLORAM (TORDOM)	1918-02-1	PHYSICAL IRRITATION	VI.C.10
1329	PICRIC ACID	88-89-1	SENSITIZATION EFFECTS	VI.C.14
1330	PIPERAZINE DIHYDROCHLORIDE	142-64-3	SYSTEMIC TOXICITY	VI.C.8
1331	PLASTER OF PARIS, TOTAL DUST	7778-18-9	PHYSICAL IRRITATION	VI.C.10
1332	PLATINUM, METAL	7440-06-4	NOAELs	VI.C.9
1333	PORTLAND CEMENT	None	PHYSICAL IRRITATION	VI.C.10
1334	POTASSIUM HYDROXIDE	1310-58-3	SENSORY IRRITATION	VI.C.3
1335	PROPARGYL ALCOHOL	107-19-7	ANALOGY	VI.C.12
1336	PROPIONIC ACID	79-09-4	ANALOGY	VI.C.12
1337	PROPOXUR (BAYGON)	114-26-1	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1338	N-PROPYL ACETATE	109-60-4	ANALOGY	VI.C.12

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1339	PROPYL ALCOHOL	71-23-8	ANALOGY	VI.C.12
1340	N-PROPYL NITRATE	627-13-4	SYSTEMIC TOXICITY	VI.C.8
1341	PROPYLENE DICHLORIDE	78-87-5	LIVER AND KIDNEY EFFECTS	VI.C.4
1342	1,2-PROPYLENE GLYCOL DINITRATE	6423-43-4	NEUROPATHY	VI.C.1
1343	PROPYLENE GLYCOL MONOMETHYL ETHER	107-98-2	SENSORY IRRITATION	VI.C.3
1344	PROPYLENE OXIDE	75-56-9	ANALOGY	VI.C.12
1346	RESORCINOL	108-46-3	NOAELs	VI.C.9
1347	RHODIUM (METAL, FUME & INSOLUBLE COMPOUNDS)	7440-16-6	INCREASING PEL	VI.C.16
1348	RHODIUM (SOLUBLE SALTS)	7440-16-6	INCREASING PEL	VI.C.16
1349	RONNEL	299-84-3	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1350	ROSIN CORE SOLDER PYROLYSIS PRODUCT (AS HCHO)	None	SENSORY IRRITATION	VI.C.3
1351	ROUGE, TOTAL DUST	None	PHYSICAL IRRITATION	VI.C.10
1352	SILICA, AMORPHOUS, DIATOMACEOUS EARTH	68855-54-9	INCREASING PEL	VI.C.16
1353	SILICA, AMORPHOUS, PRECIPITATED OR GEL	None	INCREASING PEL	VI.C.16

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1354	SILICA, CRYSTALLINE-CRISTOBALITE	14464-46-1	RESPIRATORY EFFECTS	VI.C.6
1355	SILICA, CRYSTALLINE QUARTZ, RESPIRABLE	14808-60-7	RESPIRATORY EFFECTS	VI.C.6
1356	SILICA, CRYSTALLINE TRIDYMITE	15468-32-3	RESPIRATORY EFFECTS	VI.C.6
1357	SILICA, CRYSTALLINE TRIPOLI (AS QUARTZ DUST)	1317-95-9	RESPIRATORY EFFECTS	VI.C.6
1358	SILICA, FUSED	60676-86-0	RESPIRATORY EFFECTS	VI.C.6
1359	SILICON	7440-21-3	PHYSICAL IRRITATION	VI.C.10
1360	SILICON CARBIDE	409-21-2	PHYSICAL IRRITATION	VI.C.10
1361	SILICON TETRAHYDRIDE	7803-62-5	ANALOGY	VI.C.12
1362	SILVER, METAL, DUST, AND FUME	7440-22-4	INCREASING PEL	VI.C.16
1363	SOAPSTONE, TOTAL DUST	None	RESPIRATORY EFFECTS	VI.C.6
1363A	SOAPSTONE, RESPIRABLE DUST	None	RESPIRATORY EFFECTS	VI.C.6
1364	SODIUM AZIDE	26628-22-8	CARDIOVASCULAR EFFECTS	VI.C.7
1365	SODIUM BISULFITE	7631-90-5	SENSORY IRRITATION	VI.C.3
1366	SODIUM FLUOROACETATE	62-74-8	SYSTEMIC TOXICITY	VI.C.8
1367	SODIUM HYDROXIDE	1310-73-2	SENSORY IRRITATION	VI.C.3
1368	SODIUM METABISULFITE	7681-57-4	SENSORY IRRITATION	VI.C.3

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1369	STARCH, TOTAL DUST	9005-25-8	PHYSICAL IRRITATION	VI.C.10
1371	STODDARD SOLVENT	8052-41-3	NARCOSIS	VI.C.2
1372	STYRENE (PHENYLETHYLENE)	100-42-5	NARCOSIS	VI.C.2
1373	SUBTILISINS (PROTEOLYTIC ENZYMES)	1395-21-7	SENSITIZATION EFFECTS	VI.C.14
1374	SUCROSE, TOTAL DUST	57-50-1	PHYSICAL IRRITATION	VI.C.10
1375	SULFUR DIOXIDE	7446-09-5	RESPIRATORY EFFECTS	VI.C.6
1376	SULFUR MONOCHLORIDE	10025-67-9	SENSORY IRRITATION	VI.C.3
1377	SULFUR PENTAFLUORIDE	5714-22-7	SENSORY IRRITATION	VI.C.3
1378	SULFUR TETRAFLUORIDE	7783-60-0	RESPIRATORY EFFECTS	VI.C.6
1379	SULFURYL FLUORIDE	2699-79-8	ANALOGY	VI.C.12
1380	SULPROFOS	35400-43-2	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1381	TALC (NON-ASBESTIFORM)	14807-96-6	RESPIRATORY EFFECTS	VI.C.6
1382	TANTALUM	7440-25-7	NOAELs	VI.C.9
1383	TEMEPHOS	3383-96-8	PHYSICAL IRRITATION	VI.C.10
1384	TERPHENYLS	26140-60-3	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1385	1,1,2,2-TETRACHLOROETHANE	79-34-5	LIVER AND KIDNEY EFFECTS	VI.C.4
1386	TETRAETHYL LEAD	78-00-2	INCREASING PEL	VI.C.16
1387	TETRAHYDROFURAN	109-99-9	SENSORY IRRITATION	VI.C.3

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1388	TETRAMETHYL LEAD	75-74-1	INCREASING PEL	VI.C.16
1389	TETRASODIUM PYROPHOSPHATE	7722-88-5	SENSORY IRRITATION	VI.C.3
1391	4,4'-THIOBIS (6-TERT-BUTYL-M-CRESOL)	96-69-5	PHYSICAL IRRITATION	VI.C.10
1392	THIOGLYCOLIC ACID	68-11-1	SENSORY IRRITATION	VI.C.3
1393	THIONYL CHLORIDE	7719-09-7	ANALOGY	VI.C.12
1394	TIN (ORGANIC COMPOUNDS)	7440-31-5	CHANGE IN SKIN DESIGNATION ONLY	VI.C.18
1395	TIN OXIDE	7440-31-5	RESPIRATORY EFFECTS	VI.C.6
1396	TITANIUM DIOXIDE	13463-67-7	PHYSICAL IRRITATION	VI.C.10
1397	TOLUENE	108-88-3	NARCOSIS	VI.C.2
1398	TOLUENE-2,4-DIISOCYANATE	584-84-9	SENSITIZATION EFFECTS	VI.C.14
1399	O-TOLUIDINE	95-53-4	CANCER	VI.C.15
1400	P-TOLUIDINE	106-49-0	CANCER	VI.C.15
1401	P-TOLUIDINE	108-44-1	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1402	TRIBUTYL PHOSPHATE	126-73-8	ANALOGY	VI.C.12
1403	1,1,2-TRICHLORO-1,2, 2-TRIFLUOROETHANE	76-13-1	CARDIOVASCULAR EFFECTS	VI.C.7
1404	TRICHLOROACETIC ACID	76-03-9	ANALOGY	VI.C.12

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1405	1,2,4-TRICHLOROBENZENE	120-82-1	SENSORY IRRITATION	VI.C.3
1406	TRICHLOROETHYLENE	79-01-6	NARCOSIS	VI.C.2
1407	1,2,3-TRICHLOROPROPANE	96-18-4	LIVER AND KIDNEY EFFECTS	VI.C.4
1408	TRIETHYLAMINE	121-44-8	SENSORY IRRITATION	VI.C.3
1409	TRIMELLITIC ANHYDRIDE	552-30-7	RESPIRATORY EFFECTS	VI.C.6
1410	TRIMETHYL PHOSPHITE	121-45-9	NOAELs	VI.C.9
1411	TRIMETHYLAMINE	75-50-3	ANALOGY	VI.C.12
1412	TRIMETHYLBENZENE	25551-13-7	SYSTEMIC TOXICITY	VI.C.8
1413	2,4,6-TRINITROTOLUENE (TNT)	118-96-7	BIOCHEMICAL/METABOLIC EFFECTS	VI.C.13
1414	TRIORTHOCRESYL PHOSPHATE	78-30-8	CHANGE IN SKIN DESIGNATION ONLY	VI.C.18
1415	TRIPHENYL AMINE	603-34-9	NOAELs	VI.C.9
1416	TUNGSTEN & COMPOUNDS (INSOLUBLE)	7440-33-7	SYSTEMIC TOXICITY	VI.C.8
1417	TUNGSTEN & COMPOUNDS (SOLUBLE)	7440-33-7	SYSTEMIC TOXICITY	VI.C.8
1418	URANIUM (INSOLUBLE COMPOUNDS)	7440-61-1	NOAELs	VI.C.9
1419	URANIUM (SOLUBLE COMPOUNDS)	7440-61-1	INCREASING PEL	VI.C.16
1420	N-VALERALDEHYDE	110-62-3	ANALOGY	VI.C.12
1421	VANADIUM (V_2O_5, DUST)	1314-62-1	SENSORY IRRITATION	VI.C.3
1422	VANADIUM (V_2O_5, FUME)	1314-62-1	SENSORY IRRITATION	VI.C.3
1423	VEGETABLE OIL MIST	None	PHYSICAL IRRITATION	VI.C.10

II. Index to Preamble Discussion of Individual Substances (continued)

H. S. Number	Substance Name	CAS Number	Primary Basis for Limits	Preamble Section
1424	VINYL ACETATE	108-05-4	SENSORY IRRITATION	VI.C.3
1425	VINYL BROMIDE	593-60-2	CANCER	VI.C.15
1426	VINYL CYCLOHEXENE DIOXIDE	106-87-6	CANCER	VI.C.15
1427	VINYL TOLUENE	25013-15-4	ODOR EFFECTS	VI.C.11
1428	VINYLIDENE CHLORIDE	75-35-4	SYSTEMIC TOXICITY	VI.C.8
1429	VM & P NAPHTHA	8032-32-4	SENSORY IRRITATION	VI.C.3
1430	WELDING FUMES (TOTAL PARTICULATE)	None	SYSTEMIC TOXICITY	VI.C.8
1430a	WOOD DUST, HARD WOOD	None	RESPIRATORY EFFECTS	VI.C.6
1430b	WOOD DUST, SOFT WOOD	None	RESPIRATORY EFFECTS	VI.C.6
1430c	WOOD DUST, WESTERN RED CEDAR	None	RESPIRATORY EFFECTS	VI.C.6
1431	XYLENE (O,M,P-ISOMERS)	1330-20-7	SENSORY IRRITATION	VI.C.3
1432	M-XYLENE-ALPHA,ALPHA'-DIAMINE	1477-55-0	ANALOGY	VI.C.12
1433	XYLIDINE	1300-73-8	ANALOGY	VI.C.12
1434	ZINC STEARATE	557-05-1	PHYSICAL IRRITATION	VI.C.10
1435	ZINC CHLORIDE FUME	7646-85-7	SENSORY IRRITATION	VI.C.3
1436	ZINC CHROMATES (CrVI)	Varies	CANCER	VI.C.15
1437	ZINC OXIDE (FUME)	1314-13-2	SYSTEMIC TOXICITY	VI.C.8
1438	ZINC OXIDE, TOTAL DUST	1314-13-2	PHYSICAL IRRITATION	VI.C.10
1439	ZIRCONIUM COMPOUNDS	7440-67-7	SYSTEMIC TOXICITY	VI.C.8

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III. Pertinent Legal Authority

The publication of a final standard is authorized by sections 6 and 8 of the Occupational Safety and Health Act of 1970 (the Act), 29 U.S.C. 655 and 657. Section 6(b)(5) governs the issuance of occupational safety and health standards dealing with toxic materials or harmful physical agents.

It states:

> The Secretary in promulgating standards dealing with toxic materials or harmful physical agents under this subsection shall set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to the hazard dealt with by such standard for the period of his working life. Development of standards under this subsection shall be based upon research, demonstrations, experiments, and such other information as may be appropriate. In addition to the attainment of the highest degree of health and safety protection for the employee, other considerations shall be the latest available scientific data in the field, the feasibility of standards, and experience gained under this and other health and safety laws. Whenever practicable, the standard promulgated shall be expressed in terms of objective criteria and of the performance desired.

Section 3 (8) defines an occupational safety and health standard as "a standard which requires conditions, or the adoption or use of one or more practices, means, methods, operations, or processes, reasonably necessary or appropriate to provide safe or healthful employment and places of employment."

The Supreme Court has held under the Act that the Secretary, before issuing any new standard, must determine that it is reasonably necessary and appropriate to remedy a significant risk of material health impairment. *Industrial union Department* v. *American Petroleum Institute, (IUD* v. *API).* 488 U.S. 607 (1980). The Court stated that "before he can promulgate any permanent health or safety standard, the Secretary is required to make a threshold finding that a place of employment is unsafe in the sense that significant risks are present and can be eliminated or lessened by a change in practices" (448 U.S. at 642). The Court also stated "that the Act does limit the Secretary's power to require the elimination of significant risk" (488 U.S. 644, n. 49).

The Court indicated, however, that the significant risk determination is "not a mathematical straitjacket," and that "OSHA is not required to support its finding that a significant risk exists with anything approaching scientific certainty." The Court ruled that "a reviewing court [is] to give OSHA some leeway where its findings must be made on the frontiers of scientific knowledge [and that] * * * the Agency is free to use conservative assumptions in interpreting the data with respect to carcinogens, risking error on the side of over protection rather than under protection" (448 U.S. at 655).

The Court also stated that "while the Agency must support its finding that a certain level of risk exists with substantial evidence, we recognize that its determination that a particular level of risk is 'significant' will be based largely on policy considerations." (488 U.S. at 655, n. 62).

After OSHA determines that a significant risk exists and that such risk can be reduced or eliminated by the proposed standard, it must set a standard which is technologically and economically feasible. In *American Textile Manufacturers Institute* v. *Donovan,* 452 U.S. 490, 531, n. 32 (1981) the Supreme Court held that "cost-benefit analysis is not required by statute because feasibility analysis is." The aim is to set the lowest feasible level necessary to eliminate significant risk.

As previously noted OSHA is required under its statutory authority to "set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to the hazard dealt with by such standard for the period of this working life" under the provisions of 6(b)(5) of the OSH Act. "Material impairment" is therefore a term which needs to be considered when issuing standards.

In this rulemaking, OSHA is considering establishing new or revised PELs for over 400 separate substances. The health effects for these substances cover a wide spectrum of severity including: Life threatening effects; disabling effects; various diseases; irritation to different organs or tissues; and changes in organ functions indicative of future health decrements.

The statutory requirements in section 3(8) and 6(b)(5) are quoted above. Other statutory criteria are set forth in section (2)(b) which states:

> The Congress declares it to be its purposes and policy, through the exercise of its powers to regulate commerce among the several States and with foreign nations and to provide for the general welfare, to assure so far as possible every working man and woman in the Nation safe and healthful working conditions and to preserve our human resources.

One of the earlier Senate drafts of the OSHA bill did not include the word "material" before the word "impairment." That word was added by an amendment of Senator Dominick. The Senator stated in respect to that amendment:

> What we were trying to do in the bill—unfortunately, we did not have the proper wording or the proper drafting—was to say that when we are dealing with toxic agents or physical agents, we ought to take such steps as are feasible and practical to provide an atmosphere within which a person's health or safety would not be affected. Unfortunately, we had language providing that anyone would be assured that no one would have a hazard, or at least, we would require the Secretary to set standards so stating, and that in the HEW standard there would be a requirement to proceed on that basis so that no one would have any problem for the rest of his working life. It was an unrealistic standard. As modified, we would be approaching the problem by looking at the problem and setting a standard or criterion which would not result in harm (Legislative History p. 502).

The D.C. Circuit Court considered the concept of material impairment and reviewed the Legislative History in the Lead Case.

It stated:

> The essential question under Section 6(b)(5) for this case is whether OSHA acted within the limits of this mandate to establish "material" impairment of health when it set a standard designed to protect workers from the subclinical effects of lead. As a statutory matter, after examining precedent and legislative history, we hold that Section 6(b)(5) empowers OSHA to set a PEL that prevents the subclinical effects of lead that lie on a continuum shared with overt lead disease. (United Steelworkers v. Marshall, 647 F. 2d at 1248–49. See also the more extended discussion there).

The legislative history and judicial analysis indicate that OSHA is to take a balanced but protective approach. Some impairments are so slight a discomfort that they are not material and do not provide a basis for regulation. A complaint of minor discomfort, in and of itself, is not material impairment. However, the OSH Act is designed to be protective of workers and is to protect against impairment with less impact than severe impairment.

These health effects are related to two different types of exposure: Acute and chronic. Because of the difference between the health effects associated with these two types of exposures, OSHA must consider different types of PELs (TWA, STEL, or ceiling) to protect against material impairment.

OSHA asked for comment on the subject of "material impairment" (Question 21), especially with regard to

whether sensory irritation should be considered to be material impairment. There was a relatively limited amount of comment. The most complete response was provided by NIOSH (Ex. 8-47) which stated:

> The recognition of sensory irritation as potentially being "material impairment of health" is consistent with the current scientific consensus related to health effects of environmental agents.
>
> Mucous membrane irritants can cause increased blink frequency and tearing; nasal discharge, congestion, and sneezing; and cough, sputum production, chest discomfort, wheezing, chest tightness, and dyspnea. Work environments often require levels of physical and mental performance considerably greater than encountered in daily living. Even in the absence of any permanent impairment, the symptoms listed can interfere with job performance and safety.
>
> Mucous membrane irritation can result in inflammation, which may lead to increased susceptibility to nonspecific irritants and infectious agents. For example, experimental ozone exposure in humans results in increased airway reactivity. Also, studies of exposure to environmental tobacco smoke have shown irritative symptoms and evidence of increased frequency of respiratory tract illnesses in young children and decreased pulmonary function in adults. * * *
>
> Mucous membrane irritation is associated with respiratory illnesses, depending on the composition of specific exposure and on the dose, duration, and frequency of exposure. No universally applicable conclusion can be drawn at this time regarding the association between irritative symptoms and permanent injury or dysfunction. Where certain individuals show no measurable impairment after an exposure, even when experiencing irritative symptoms, others may develop identifiable dysfunction.
>
> Aside from the effects of irritations, mucous membrane exposure may result in absorption of a substance, with resultant systemic toxicity. An inflamed mucous membrane may be an even more effective route of absorption, either for the irritant or for other substances. Furthermore, injury to bronchopulmonary membranes can impair removal of particulates from the respiratory system.

Thus, according to NIOSH, sensory irritants interfere with job performance and safety, cause inflammation, may increase the victim's susceptibility to other irritants and infectious agents, lead to permanent injury or dysfunction, or permit greater absorption of hazardous substances (Ex. 8-47). In sum, NIOSH and most other respondents agree that sensory irritation caused by occupational exposure to the irritant substances included in this final rule constitutes a material impairment of health (see Section VI.C.3).

Of course, irritation also covers a spectrum of effects, some serious and some trivial. Hence, complaints of minor irritation would not in and of itself constitute material impairment. In addition, OSHA would weigh irritation with physical manifestations more heavily than irritation with purely subjective responses. This does not mean that purely subjective responses would not constitute material impairment. That judgment would depend on the magnitude of the irritation.

OSHA also believes that clinical, tissue or organ changes, or properly documented pain, chest tightness, migraine headache or similar reactions reflected in responses by persons, may also represent material impairment of health. Each of these are considered on a case-by-case basis in this Rulemaking. OSHA believes that its approach is consistent with the Act and Congressional intent regarding material impairment of health.

All of the revised or new PELs in this Rulemaking are within reasonable interpretation of a general approach to identifying situations involving material impairment of health. In a few instances, PELs noted in the Proposal have been modified, or deleted, because it was determined that the proposed level was not needed to reduce a significant risk of material impairment of health. These substances are identified in the Preamble.

OSHA's analyses regarding material impairment of health, as applied in this Rulemaking, are provided in three separate ways. First, they are included in the introductory discussion for Sections VI-C-1 through VI-C-18 in the Preamble. Second, the discussion of each of the 428 substances contains health effect information for all new or revised PELs. Third, the following discussion provides examples and general guidance regarding the OSHA decision process, and accounts for the severity spectrum of health effects, and the separate cases involving chronic and acute exposures.

As previously indicated, health effects cover a wide range of severity levels. A precise delineation between material impairment and non-material impairment is not possible since a variety of factors must be considered, such as the composite health effect and frequency and duration of the effect, to determine if a substance represents a material impairment of health. For example, nerve damage would normally constitute material impairment of health. However, a small reduction in nerve conductivity may not constitute material impairment. Moreover, an occupational, transitory, non-progressive and/or non-intensive coughing reaction may represent non-material impairment. Major intensification of some of these factors could result in a health effect which represents material impairment of health.

Consequently, general considerations can be stated but they must be applied on a case-by-case basis taking into account the scientific evidence, public comments and agency expertise.

OSHA has concluded that updating the Z-Tables to reflect recent information is the highest priority for the Agency. This will reduce exposure limits for approximately 212 substances regulated currently by the Z-Tables and add exposure limits for approximately 164 substances which are currently unregulated. The health literature indicates this must be accomplished to improve worker health; it is one of Congress' concurrent goals and will greatly increase occupational health protection for a very large number of workers.

In order to accomplish this high priority task in a reasonable time in the light of limited administrative resources, it is necessary to narrow somewhat the issues to be faced by the Agency in this proceeding. Consequently, it is necessary to delay other worthwhile goals and concurrent Congressional purposes.

This approach is consistent with the general principles of administrative law. An Agency may set priorities within the framework of its statutory authority. Secondly, an Agency may take substantial steps towards its statutory goals, without having to achieve them completely, when Agency resources are not sufficient to complete all aspects initially.

Congress recognized that the Secretary could not address all occupational safety and health problems simultaneously. It therefore gave the Secretary discretion to set priorities in exercising his rulemaking authority. As section 6(g) of the Act, 29 U.S.C. 655(g), states:

> In determining the priority for establishing standards under this section, the Secretary shall give due regard to the urgency of the need for mandatory safety and health standards for particular industries, trades, crafts, occupations, businesses, workplaces or work environments.

In proposing this addition to the Act, Senator Javits explained that its purpose was "to relieve the Secretary of the necessity for waiting to promulgate whatever standards he wishes to promulgate across the board but, rather, allowing him to yield to more urgent demands before he tries to meet others." Leg. Hist 505. Thus, the Act has "built in

flexibilities" that the Secretary may use, such as establishing "the priorities between the various occupations that may require standards." *National Congress of Hispanic American Citizens* v. *Usery*, 554 F. 2d 1196, 1199 (D.C. Cir. 1977); see also *National Congress* v. *Marshall*, 626 F.2d 882 (D.C. Cir. 1979). The flexibility expressed in the statute and legislative history is consistent with the well-established principle that an administrator may adopt a "rational, 'one step at a time' approach" to rulemaking. *National Roofing Contractors Ass'n* v. *Brennan*, 495 F. 2d 1294, 1299 (7th Cir.), cert. denied, 419 U.S. 1105 (1974) (OSHA roofing standard); cf., *Industrial Union Dept.* v. *American Petroleum Institute*, 488 U.S. 607, 663 (1980) (Burger, concurring) (OSHA can act in its legislative capacity "to focus on only one aspect of a larger problem"); *United Steelworkers* v. *Auchter*, 763 F. 2d 728, 738 (3rd Cir. 1985) (Although OSHA's decision to exclude workers in some industries from a standard requires explanation. "[s]ection 6(g) clearly permits the Secretary to set priorities for the use of the agency's resources and to promulgate standards sequentially."); *IUD* v. *Hodgson*, 499 F. 2d 467, 480 n. 31 (D.C. Cir. 1974) ("The [OSHA] statutory scheme is generally calculated to give the Secretary broad responsibility for determining when standards are required and what those standards should be.").

OSHA has concluded that setting exposure limits for the large number of substances involved in this rulemaking has priority at this stage over exploring the need for accompanying medical surveillance, exposure monitoring and industrial hygiene provisions for a much smaller number of substances. Section 6(b)(7) of the Act, of course, indicates that "where appropriate" such provisions are to be included. That was a concurrent goal of Congress as was Congress's goal to lower exposure for the many unregulated or inadequately regulated substances when scientific data indicate lower exposures are needed. However, OSHA has inadequate resources to accomplish both goals at this time. Lower exposures is a higher priority because it is more effective in reducing diseases and material impairments of health.

OSHA has already addressed some of section 6(b)(7)'s goals, as they relate to labels and warnings, in the generic Hazard Communication Standard, 29 CFR 1910.1200. It is working on a standard to improve respirator use for all chemicals (47 FR 20803). It is considering generic regulation for exposure monitoring (53 FR 32591–32595) and medical surveillance (53 FR 32595–32598). OSHA does not have the resources to conclude this rulemaking in any reasonable time, and also consider these issues.

OSHA does have legal authority not to address ancillary provisions in this rulemaking and determinations about the appropriateness of ancillary provisions have not been made. That is a rational use of its priority setting authority in the light of OSHA's limited resources. The language of section 6(b)(7) is not an absolute requirement to include such provisions and this is an "appropriate" circumstance not to include them. The actions already initiated by OSHA indicate it is facing the issue of ancillary provisions in a responsible and reasonable manner.

OSHA is utilizing its priority setting authority for several other matters. There are several substances (both carcinogens and non-carcinogens) where more detailed analysis of the evidence might in the future lead to the conclusion that there is remaining significant risk. If that were the case in a single substance rulemaking, OSHA would explore that issue in great depth and do much more extended economic analysis of several different exposure levels to determine what the lowest feasible level might be.

Past experience has shown this to be a major undertaking both from a health and economic point of view. OSHA, for example, spends an average of $500,000 and takes one year of study to determine the lowest feasible level for a *single* substance. OSHA does not have the resources to engage in that kind of analysis for more than a few substances. The attempt to do so in this rulemaking would significantly reduce the chances of it ever being completed and would result in far more workers being exposed to significant risk in exchange for the incremental risk reduction attained by further lowering the PEL for a small group of substances in this Rulemaking.

When evidence has been available to determine that the proposed level resulted in remaining significant risk and a lower level was feasible, OSHA has issued that lower level. Three chemicals fit that category in this rulemaking. Two are based on OSHA's analysis of evidence presented by NIOSH and a third, perchloroethylene, is based on OSHA's analysis of evidence supplied by the Amalgamated Clothing and Textile Workers Union and the dry cleaning industry.

OSHA has indicated that further, more extensive analysis may lead to the conclusion that significant risk remains for other substances. However, the extensive investment of resources needed to arrive at such conclusions would be determined by the Agency's future priorities.

OSHA's first priority is to achieve substantial reductions in significant risk for this large number of substances. The accomplishment of this goal will create the greatest health benefits for the American worker. OSHA's decisions in this Rulemaking are consistent with the evidence on risk and feasibility which is available at this time. Further consideration of these issues for specific substances can be achieved in future rulemakings as new evidence becomes available and as priorities indicate.

OSHA concludes this is both rational priority setting and a reasonable integration of its priority setting authority and relevant case law. Clearly multi-issue and multi-substance rulemaking require a different balancing than single substance or single issue rulemaking.

OSHA has met legal requirements also in the regulation of carcinogens in the light of priorities and Agency resources. Potential carcinogens tend to require far more administrative resources per substance to regulate, in part because their regulation tends to create more controversy. There are a number of expert organizations which analyze and develop lists of suspect or probable carcinogens. These organizations include ACGIH, NIOSH, the National Toxicological Program (NTP) and the International Agency for Research on Cancer (IARC). These organizations utilize somewhat different criteria and categories for their lists.

The ACGIH TLVs include some substances which it categorizes as carcinogens, but does not set the exposure limit based on carcinogenicity. It includes other substances which it may or may not categorize as carcinogens but for which the exposure limit is set taking into account carcinogenicity. It is only this last category which OSHA considered as carcinogens in its proposal. (This is a separate issue from what exposure limit is set). If NIOSH recommended a different limit for a chemical in this grouping, OSHA considered which of the recommended limits was best supported. OSHA concludes this was a rational use of its priority setting authority. A categorization of carcinogenicity along with a proposed exposure limit is much more useful to OSHA as a starting point for its analysis than a categorization without a recommended numerical limit. IARC

never recommends numerical limits and in some instances neither has NIOSH.

Attemps to analyze for carcinogenicity a substantially larger number of substances would again require far more scientific and administrative resources than OSHA has available and would probably prevent the prompt completion of this rulemaking. In addition OSHA has spent much of its past efforts in regulating carcinogens. It concludes that it is important to concentrate some of its efforts through this project on noncarcinogenic health hazards. Substances which cause liver/kidney or cardiovascular disease can be just as deadly as carcinogens and can cause higher risks.

Accordingly, OSHA concludes it is a rational use of its priority authority not to consider for regulation as a carcinogen every sustance included, for which there is some evidence or recommendation that it may be carcinogenic. Such substances often require exposure limits to protect against other health hazards they cause, which results in OSHA setting limits in this rulemaking based on non-carcinogenic effects. In these circumstance 29 CFR Part 1990 need not apply when there is no determination to consider regulating a substance as a carcinogen.

There are several substances which OSHA proposed regulating as carcinogens, for which participants submitted evidence to the contrary. In some of those cases OSHA has concluded that it is inappropriate to regulate a substance as a carcinogen at this time because further analysis is necessary. In several cases participants submitted evidence that substances included in the proposal which OSHA did not propose to regulate as carcinogens should be regulating as carcinogens. When OSHA concluded there was sufficient evidence and analysis to meet legal requirements, OSHA has in this final rule regulated the substance as a carcinogen. However, as just stated, OSHA has not itself attempted to analyze further for carcinogenicity substances which it did not propose regulated as a carcinogen, unless participants supplied relevent evidence.

For the substances OSHA is regulating as carcinogens, it has met all requirements of *IUD* v. *API* and has acted consistently with 29 CFR Part 1990. Specifically, OSHA reviewed all data to determine whether there were studies of sufficient merit to determine that the substance was qualitatively a carcinogen.

Secondly, OSHA contracted with Dr. Nathan J. Karch, President of Karch & Associates, Inc., consultant experts in risk assessment, to analyze the available studies for two purposes (Ex. 85): first to determine whether there was sufficient evidence to perform a quantitative risk assessment or to state the reasons why there was not; second, to perform a quantitative risk assessment where the studies permitted, using techniques generally accepted by the scientific community. The risk assessments were presented in the preamble (52 FR 21190–209) and elaborated upon in Dr. Karch's statement. Dr. Karch responded to questions on the risk assessments. (OSHA did not ask Dr. Karch to independently review the strength of the qualitative date since OSHA had preliminarily made that review.)

Utilizing these data, public comments and OSHA analysis of the data, OSHA has issued exposure limits which will substantially reduce significant risk and are feasible. Where a public participant has supplied evidence that a lower level than that proposed would further reduce significant risk and was feasible, OSHA has promulgated that lower level.

In other words, OSHA's decisions in this Rulemaking are based on the entire evidence in the public record, and that the final PEL substantially reduces signficant risk and is feasible. OSHA may reconsider the issues for individual substances as further information becomes available.

For the reasons stated above and in line with its priority setting authority, OSHA concludes it has authority, in the circumstances of a rulemaking which considered changes to PELs for 428 substances, not to explore those issues at this time. This issue and the regulation of other substances as carcinogens may be considered in future 6(b) rulemakings where priorities indicate the issue is important and when administrative and scientific resources become available. OSHA concludes this is consistent with its legal authority and reasonable health policy.

A few of the substances which OSHA is regulating principally for non-carcinogenic effects, but for which there is some evidence of carcinogenicity, fit into an additional category. In these cases there was uncertainty concerning which of two levels would eliminate significant risk of the non-carcinogenic effect. In those cases OSHA used the evidence of carcinogenicity as an additional factor in determining whether the lower level was appropriate.

Most of the chemicals OSHA is regulating are not carcinogens. There are effects which occur immediately, or a relatively short time after exposure. Sometimes the effects are reversible after removal from exposure and treatment, and sometimes the effects are not.

Many of these substances are believed to have effective thresholds, that is there is a level of exposure above which some number of persons will suffer the effect. There is a level somewhat below that, where it appears that few persons would suffer the effect, and there is a lower level where scientists are more confident of that conclusion. At levels below a properly well defined threshold the risk would not be significant. However, there may be a smaller group of employees who might be susceptible at very low levels.

The studies which tend to be performed, examine relatively small groups of animals or persons exposed at one or several levels. A determination is made at which of these levels effects are seen and at which they are not.

The Supreme Court in *IUD* v. *API* was faced with the situation, as is often the case with carcinogens, that high risk is known at relatively high levels of exposure. But scientists do not have direct measurements of what the risk may be at lower levels. Indeed for statistical and methodological reasons they may never be in position to directly measure risk at lower levels or indeed to determine if it is ever eliminated. Consequently, modeling techniques must be used to estimate risk at various levels so as to determine the significance of risk at various levels.

In the case of this rulemaking the type of evidence available makes such modeling techniques generally unnecessary for significant risk determinations. The studies provide direct evidence of whether or not there is an effect and the probability of that effect occurring at the levels to which the Agency is regulating. Consequently, a judgment can be made whether the effect is a significant risk directly from the studies. Similarly, a judgment can be made directly from the studies whether that significant risk is reduced or eliminated.

An example may illustrate this method of analysis. The current OSHA exposure limit for hydrogen sulfide (H_2S) is a 20 ppm STEL and a 50 ppm peak. There are studies showing that at 20 ppm and perhaps below workers develop conjunctivitis, eye irritation and other ocular effects. Those effects present a significant risk of material impairment because they would prevent work, require medical treatment, and would make it difficult to work safely

with the condition. Another study shows that the deleterious health effects do not develop in workers exposed continually at 10 ppm. Consequently OSHA is issuing a final standard of a 10 ppm TWA with a 20 ppm ceiling which should eliminate that significant risk. Direct evidence of significant risk and its probable elimination exist and there would be no point in applying mathematical modeling to the data for purposes of making a significant risk determination.

Often the data are not as clear cut. There may be only an effect study. There may be uncertainty about exposures levels or conflicting results. There may be a no effect study with exposure data and an effect study without exposure data. Studies are often of small size with fairly wide ranges of uncertainty. Consequently, they do not take into account variability of response among humans. Of course, many studies are based on animal data and adjustments must be made to take into account the differences between human and animal susceptibility.

However, judgments as to levels which create, reduce or eliminate significant risk still have the greatest scientific validity in most cases when based directly on specific studies. The uncertainties which exist are likely to be magnified, rather than reduced, when combined with the uncertainties of modeling techniques.

Accordingly OSHA concludes that the type of significant risk analysis undertaken in this rulemaking is most consistent with the studies generally available and is a valid scientific approach. OSHA concludes it is fully consistent with the requirements of the Supreme Court in *IUD* v. *API.*

OSHA has made quantitative estimates of the benefits of this entire standard. These are discussed in Section VII D and are substantial. That analysis demonstrates that on a general basis there is a significant risk of occupationally related illness and death and that this new standard substantially reduces that risk.

Some of the exposure levels incorporate an uncertainty factor. This is sometimes referred to as a "safety factor." Studies are often of small size and, since there is a large variation in human susceptibility, a study because of its small size may not demonstrate an effect that actually exists. (In scientific terminology it lacks statistical power.) For this reason, it is not uncommon to set a limit below that level which the study may have indicated showed no effect.

This has been the standard approach for recommending exposure limits for non-carcinogens by scientists and health experts in the field for many years. (See testimony of Mastromatteo (Ex. 22) and Key (Ex. 17)). Generally, a greater safety or uncertainty factor is used for more severe health effects.

This use of uncertainty or safety factors in a reasonable manner generally does not lead to reducing exposures below the level of significance. Rather, it takes into account the likelihood that effects may exist at levels below the level of the study.

Of course, use of uncertainty factors or safety factors is even more clearly justified when the studies available determine only a level at which there is a significant risk of a health effect exists rather than the level at which there clearly is no significant risk, or when animal studies are used. Clearly, to substantially reduce significant risk, an exposure level must be set below the level where significant risk exists in humans. Similarly, to take into account the possibility of interspecies variability, a level set for humans usually is set below the observed no effect level in an animal. See the extended discussion in the Health Effects Section, specifically Section VI A.

Section 6(b)(5) of the OSH Act states that standards shall be based on many factors including "the best available evidence," the "latest available scientific data in the field," and "experience gained under this and other health and safety laws." OSHA concludes that its approach meets these provisions.

OSHA utilized the research and recommendations of two expert organizations, NIOSH and ACGIH, as the starting point for its analysis. Those organizations regularly review the literature and update their recommendations. (See testimony of Mastromatteo (Ex. 22) and NIOSH (Ex. 8–47)). When participants brought to OSHA's attention other studies, made other recommendations for exposures limits or the proposal was controversial, OSHA fully reviewed the available scientific information. (The final preamble does not always discuss studies which OSHA did not believe were important or relevant to the determination of an exposure limit.) Where there was little or no comment on particular parts of the OSHA proposal, and the evidence OSHA relied on appeared substantial, OSHA did not attempt additional searches of the literature.

OSHA concludes that this is a rational approach to make use of the best available and latest scientific information for making final decisions on exposure limits. An approach attempting to analyze and discuss every single study for substances where the proposed exposure limit is not seriously disputed would not add to the quality of OSHA's final decisions and would interfere with the statutory goal to protect employees from material health impairments. As discussed elsewhere OSHA also relied upon its experience in making final decisions as is encouraged by Section 6(b)(5).

It has been suggested to OSHA that two of its selected categories do not constitute material impairment of health. The first category includes particulates which cause physical irritation and other effects. In the Proposal, OSHA followed for clarity purposes the historical terminology of "nuisance" dust which is misleading. The term "nuisance" is used by ACGIH to cover dusts that, although they do not cause pneumoconiosis or permanent scarring of the lungs, can cause many material health impairments such as chronic bronchitis, chronic throat irritation, skin irritation or eye inflammation. Consequently there is a health need for an exposure limit for all particulates. See the discussion under particulates (Section VI, Health Effects).

Secondly, three chemicals are listed as odorants by ACGIH and also have other effects. In these cases OSHA has not changed the existing PEL. These levels were identified in the Proposal and there was not significant comment that the limits should be raised. No new chemicals are regulated as odorants. Accordingly, OSHA has not had to visit the issue of when an odorant has become so severe as to constitute a material impairment of health.

OSHA feasibility determinations are based on both the statute and on a consistent and extensive body of case law extending over its entire history. In addition to *ATMI* (supra) see for example: *AFL–CIO* v. *Hodgson*, 499 F. 2d 467 (D.C. Cir., 1974); *Society of Plastics Industries* v. *OSHA* (SOCMA), 509 F. 2d 1301 (2d Cir., 1975); *American Iron and Steel Inst.* v. *OSHA* (AISI), 577 F. 2d 825 (3rd. Cir., 1977); *United Steelworkers* v. *Marshall*, 647 F. 2d 1189 (D.C. Cir., 1980); *ASARCO* v. *OSHA*, 746 F. 2d 483 (9th Cir., 1984) and others.

Standards may be expensive and still be feasible if necessary to protect occupational health.

Standards may be economically feasible, though from the standpoint of employers, they are financially burdensome and affect profit margins adversely. Nor does the concept of economic feasibility necessarily guarantee the continued existence of

individual employers. It would appear to be consistent with the purpose of the Act to envision the economic demise of an employer who has lagged behind the rest of industry in protecting the health and safety of employees * * * (*Hodgson*, 499 F. 2d at p. 478).

An OSHA standard may be technology forcing. OSHA may demonstrate feasibility by showing that only a few plants are now in compliance. Moreover, a standard is still feasible even though some respirator use is needed to achieve compliance.

[T]he Secretary is not restricted by the status quo. He may set standards which require improvements in existing technologies or the development of new technology * * * (SOCMA, 509 F. 2d at p. 1309).

The experience at (2) batteries provides a sufficient basis for the Secretary's reasoned belief that the 0.15 mg/m³ limit could be met (for the entire industry) (AISI, 577 F. 2d at p. 834; See also SOCMA, p. 1309).

The limited respirator use that the standard requires does not in any way render the standard infeasible * * * (ASARCO, 746 F. 2d at p. 483. See also ATMI generally and SOCMA 509 F. 2d at p. 1310, etc.)

OSHA must show a general presumption of feasibility in most operations in an industry with engineering controls to place the burden of proof in enforcement action on the industry to show that compliance with engineering controls cannot be attained in a particular circumstance. But a showing by a particular industry that compliance requires respirators in certain operations or an admission by OSHA that that is the case does not make a standard infeasible. Rather it "will reduce the strength of the presumption a firm will have to overcome in justifying its use of respirators" in an enforcement or variance action. *United Steelworkers*, pp. 1272–73. See also *Building and Construction Trades* (Supra).

In addition, a gloss of experience has been added to this general legal guidance. In the two standards where OSHA thought at the time that it was regulating to the limits of its legal authority on feasibility, subsequent studies indicated the standard was achieved more easily than OSHA predicted.

OSHA predicted the cotton dust standard would cost $500 million in 1977 dollars whereas industry predicted twice the cost and anticipated substantial technical problems. As a matter of fact, a later detailed study indicated that the standard cost only $250 million in 1983 dollars, improved industry competitiveness and productivity as well, and improved health more than predicted. See 50 FR 51121, 51164–67 (Dec. 13, 1985).

OSHA's contractor predicted that the OSHA vinyl chloride standard could not generally be achieved with engineering controls and the attempt would cost $1.5 billion. As a matter of fact, compliance was achieved with engineering controls within three years at a cost of less than 10% of that predicted. See 49 FR 5001, 5253 (Jan. 22, 1980).

In this rulemaking, OSHA concludes that it has demonstrated feasibility without taking that concept to the full limits of its legal authority. Many of the substances regulated constitute acute hazards with apparent thresholds; the limit set to protect health does not approach the limits that could feasibly be achieved.

There are some substances covered by this regulation for which further analysis might indicate a lower limit is needed, possibly the lowest feasible limit. However, the limit set is clearly within the limits of feasibility.

In the case of a few substances, industry has argued that the limit proposed was not technically and/or economically "feasible." In some circumstances where OSHA believed it did not have enough evidence of feasibility in the record to support the level proposed, it has raised the limit to that level which the evidence available demonstrated is clearly feasible. In other circumstances where industry contended that engineering controls could not achieve the proposed level in a specific operation, and there was not sufficient evidence in the record indicating that it could be achieved with engineering and work practice controls, the preamble indicates that respirator use may be appropriate. In any event, the burden of proof would be on OSHA in an enforcement action to demonstrate the level in that operation could be achieved with engineering and work practice controls. Since OSHA's feasibility analysis was based on what industry is already achieving or what could be achieved with standard "off-the-shelf" technology, there are few if any cases where OSHA is attempting to force technology.

Several participants specifically argue that the proposed level was infeasible for a specific because in a specific substance operation it could not be achieved with engineering controls. As the case law clearly indicates, that does not make a standard infeasible. OSHA has in several cases concluded it did not have enough evidence to demonstrate that a level could be achieved with engineering controls in a specific operation. The case law clearly indicates that this does not make a standard infeasible. (See for example, *ASARCO* and *United Steelworkers*, supra). The industry does not have the burden of proving the technical infeasibility of engineering controls in an enforcement case involving these operations which are specifically identified in Section VII. The burden of proof would be on OSHA to prove that the level could be attained with engineering and work practice controls in an enforcement action if OSHA believed that was the case.

A few participants argued that, because engineering controls might not be appropriate in certain maintenance operations or in occasional or intermittent operations, the level set was therefore infeasible. First, these conditions relate to individual operations and do not indicate general difficulties of compliance with engineering controls. Secondly, as OSHA has stated, for some maintenance and intermittent operations, respirators may be the appropriate control methodology. See 52 FR 34549 for example.

Finally, 29 CFR 1910.1000(e) requires the use of engineering controls when feasible. If they are not feasible, the employer may use respirators. Consequently, this standard does not become infeasible simply because engineering controls may not achieve the PEP in a specific operation.

OSHA has a variety of data on technical feasibility. These include exposure data indicating that required levels are already being achieved by some employers in a sector. It also includes judgment by experts that standard controls have achieved or can achieve the required level in that or in analogous operations. Finally, the docket includes a significant amount of information on available existing control technology for each substance. (See for example, Ex. 6 and NIOSH Health Hazard Evaluation (HHE) reports). OSHA concludes that it was not its burden to demonstrate the technical feasibility for the substances it is setting new or more protective levels for. This is analyzed in Section VII.

OSHA also concludes, as discussed in the Economic Feasibility section, that the standard is economically feasible. Indeed, the costs do not approach the levels that would be the legal limits of economic feasibility in terms of affecting the economics of industry, either generally or by industry sector. The estimated ual cost is approximately $800 million. However, approximately, 4.5 million employees receive improved health protection making the cost per employee receiving additional protection approximately $150 each. The total cost is approximately $2 million

per substance being regulated. See the detailed feasibility discussion in Section VII.

Secondly, the cost per industry sector is never more than a small fraction of one percent of sales and with two limited exceptions not more than 2% of net profits, assuming no costs would be shifted to consumers. As the courts have recognized, it is likely that some costs will be shifted to consumers. In the few subsectors where costs reflect a slightly larger percent of profit, OSHA explains in the specific analysis of Section VII why the proposed standard is feasible and will not create disruption to an industry or to competition, although it may have some temporary effects requiring some adjustment.

OSHA's cost and economic feasibility conclusions have a high degree of validity on a sector basis. OSHA has provided much data at the subsector (4 digit SIC) level and has supplied more when requested by participants. That subsector data has probative merit, but there would not be quite as high a degree of confidence in the exact cost totals estimated. There was some questioning of OSHA and its economic panel on this matter. OSHA and the economics panel pointed out that OSHA's survey was designed to have a high degree of statistical certainty at the sector level, and provided useful evidence but not to the same high degree of statistical confidence at a subsector level.

OSHA's responsibility is to demonstrate economic feasibility for an industry. OSHA's feasibility analysis clearly shows feasibility for every sector. The costs are sufficiently low per sector to demonstrate feasibility not only for each sector but also for each subsector. Higher subsector costs would be reflected in higher sector costs. This is confirmed by the subsector data which OSHA initially provided or provided upon request. This also indicates costs are low in relation to sales and profits for subsectors. OSHA is not required to demonstrate feasibility for every plant in a subsector.

At the enforcement level, an employer's demonstration of economic infeasibility for a particular plant may lead to an extended period of time in which to come into compliance with engineering controls allowing the use of respirators during the extended interim period. The fact that this particular standard gives all employers a long period of time in which to comply will reduce feasibility problems for the few employers with possible economic difficulties.

As discussed in the approach and chronology sections, OSHA believes it has provided the public an extensive opportunity to comment and participate in this rulemaking and has complied fully with required administrative procedures. Nine months advance notice of the proposal was given. The proposal contained OSHA health reasoning for each substance proposed for revision or addition of a PEL and cited the studies OSHA relied upon. It discussed OSHA's feasibility reasoning for each sector. Underlying health studies were made available in the docket and are generally available in major libraries and/or computer data bases. The feasibility studies were also available in the docket.

The public was given more than the legal minimum time to comment. The time from proposal to final post-hearing briefs was 5 months. An oral hearing was held in which extensive presentations were made by participants and questioning was permitted of OSHA, its economic panel and witnesses, NIOSH and other participants. OSHA concludes that all of its decisions are based on substantial evidence in the docket which is analyzed in this final preamble.

Several procedural objections were made during the course of the proceeding. The first was that OSHA did not permit sufficient time for comments. OSHA believes the public has been given not only more than the legal minimum period in which to comment but has been given a fair amount of time to comment on the proposal.

The rulemaking permitted 47 days after proposal for prehearing comments, oral testimony up to 79 days after proposal, post hearing evidence up to 4 months after proposal and post hearing briefs up to almost 5 months after proposal. The OSHA Act only requires 30 days for comment. See section 6(b)(2). Three Courts of Appeals have held comment periods of 30–45 days legally sufficient: *Phillips Petroleum* v. *U.S. EPA* 802 F. 2d 549, 558–559 (10th Cir., 1986); *North American Van Lines* v. *I.C.C.*, 660 F. 2d 1087, 1092 (7th Cir., 1981) and *Conn. Light* v. *N.P.C.* 672 F/2d 529, 534 D.C. Cir., 1982.

The prehearing comment period alone met these requirements. In reality, and as OSHA agreed (Ex. 14 B p. 7), evidence not available by prescribed dates could be submitted as late as Oct. 7, 1988, the post hearing evidence deadline, which was four months after proposal. Final views did not have to be submitted until five months after the proposal. This far more than meets the legal minimums.

All data OSHA relied upon were cited in the preamble and available in the docket. The date were also usually available in libraries and on computer data bases as well. Few participants were interested in more than a few chemical and none indicated an interest in more than 20. There was time to analyze the studies and submit comments in the time periods specified. Indeed, the trade associations and individual participants who objected the most about the time for comments were interested in only one chemical (carbon disulfied, sulfur dioxide, grain dust and styrene). These trade associations had been in existence for more than a decade and had immediate access to available studies, analyses and position papers to support their views. The unions divided up the 20 or so chemicals they expressed an interest in among the various unions and ultimatley did not object to the final post hearing submission dates. Interested participants submitted extensive studies, comments and testimony averaging 3000 pages per controversial chemical. The reality was that all views were effectively presented.

The second procedural issue involves post hearing comments. The OSHA procedural rules initially grant the presiding officer authority to set the date for post hearing comments. (29 CFR 1911.16 (g)). The presiding officer set Nov. 14, 1988, for post-hearing submissions and Dec. 13, 1988, for post-hearing briefs (Ex. 81).

For the reasons just stated, OSHA concluded that these time periods were far more than required by legal minimums or for considerations of fairness. As stated in the approach section, such an extended time frame for post-hearing submissions would greatly delay the completion of this action. This would also interfere with OSHA's priority to complete in a timely fashion this project which is of such significant benefit in protecting the health of employees.

The Chief Administrative Law Judge held that he did not have jurisdiction to change the dates set by the presiding officer. (Ex. 81 B.) Accordingly, the Secretary of Labor and OSHA exercised their authority to set priorities for OSHA and by **Federal Register** Notice of Sept. 7, 1988, (53 FR 34708) set Oct. 7, 1988, for post hearing evidence and Oct. 31, 1988, for post hearing briefs.

The OSHA rule at 29 CFR 1911.4, gives authority to prescribe alternative requirements (such as the October date set) in order to expedite the conduct of the proceeding upon reasonable notice. Reasonable notice was given of the changed dates. There was a period of 30 days from the **Federal Register** notice to

the date that post hearing evidence was due and OSHA gave notice prior to the **Federal Register** notice orally and by letter that it desired to have shorter dates for post hearing submissions than initially set. Clearly, the purpose of the new date was to expedite the rulemaking process. Accordingly, all procedural requirements have been met. See also OSHA's discussions in Exs. 60 and 81 A on these two issues and at 53 FR 34708.

Third, for several substances covered by this proceeding, OSHA had proposed new standards in the middle 1970's, held hearings and closed the record. However, no new standard was ever issued, nor was a statement made of why a new standard was not issued.

There were comments on two of those substances in this rulemaking, sulfur dioxide and beryllium. In light of this circumstance and at the request of participants, OSHA submitted the complete earlier record of those proceedings into the record of this rulemaking.

For sulfur dioxide OSHA is issuing a new limit. OSHA has reviewed both the old record and new submissions, and has concluded that the new level is needed to reduce significant risk and is feasible. The discussion in this final preamble meets the procedural requirements of section 6(b)(4) for both the prior and current rulemaking.

For beryllium, OSHA is retaining the existing limit. That limit is already very low. Extensive additional evaluation would be needed to determine if that limit should be changed. Accordingly, OSHA has concluded it is not of sufficient priority to determine if the limit should be changed at this time.

Several participants stated that ACGIH was not a national consensus organization and should not be used as a starting point for OSHA's evaluation. ACGIH is not a national consensus organization as defined by the OSH Act. However, that is not relevant to this rulemaking. Section 6(a) of the Act permitted OSHA to issue as OSHA standards, without rulemaking, national consensus standards. However, that authority expired in May of 1973. This standard is not issued under section 6(a) but under the authority of section 6(b) of the OSH Act. OSHA intends this to be a section 6(b) standard and is following all of the procedures and meeting all of the requirements of section 6(b).

This is a 1900-page typed document covering 600 substances. As a result of the editing process, sometimes slightly different conclusory language is used when an identical conclusion is intended. OSHA wishes to make it clear that wherever a new or more protective exposure limit has been issued, OSHA has concluded based on evidence in the record, and its experience, that such limit is needed to substantially reduce a significant risk of material impairment of health or functional capacity. OSHA has also concluded that such limit is technically and economically feasible.

IV. Overview of Rulemaking

A. History of Health Standards and Need To Revise PELs

One of the principal reasons, if not the single most important basis, for Congress passing the Occupational Safety and Health Act of 1970 was Congress' recognition of the need to protect workers from occupational health hazards. In the preamble to the Act, Congress stated that one of the purposes was to protect employees by "exploring ways to discover latent diseases, establishing causal connections between diseases and work in environmental conditions, and conduct other research relating to health problems, in recognition of the fact that *occupational health standards present problems often different from those involved in occupational safety.*" (emphasis added).

The legislative history indicates Congressional concern for reduction in health risk from both the recognized hazards and from the many newly utilized chemicals. Congress stated in 1970:

> In the field of occupational health the view is particularly bleak, and due to the lack of information and records, may well be considerably worse than we currently know. Occupational diseases which first commanded attention at the beginning of the Industrial Revolution are still undermining the health of workers. Substantial numbers, even today, fall victim to ancient industrial poisons such as lead and mercury. Workers in the dustry trades still contact various respiratory diseases. Other materials in industrial use are only now being discovered to have toxic effects. In addition, technological advances and new processes in American industry have brought numerous new hazards to the workplace. Carcinogenic chemicals, lasers, ultrasonic energy, beryllium metal, epoxy resins, pesticides, among others, all present incipient threats to the health of workers. Indeed, new materials and processes are being introduced into industry at a much faster rate than the present meager resources of occupational health can keep up with. It is estimated that every 20 minutes a new and potentially toxic chemical is introduced into industry. New processes and new resources of energy present occupational health problems of unprecedented complexity. (Senate Report 91-1282, p.2).

To accomplish the goal of protecting workers from occupationally related disease Congress created a three-pronged approach in the OSH Act.

First, Congress desired that OSHA, as soon as possible after it was established, have in existence a set of basic, minimum health and safety standards. To accomplish this it provided in section 6(a) of the OSH Act that OSHA should adopt within its first two years, without hearing or public comment, established federal standards and national consensus standards.

At that time, under the Walsh-Healy Act, the Department of Labor had adopted for government contractors approximately 400 health standards based on the Threshold Limit Value (TLV) recommendations of the American Conference of Governmental Industrial Hygienists (ACGIH). Those were adopted as established federal standards. In addition about 25 exposure limits had been recommended by the American Standards Association (presently called the American National Standards Institute). Those were adopted as national consensus standards. OSHA adopted these initial exposure limits in May 1971. They are for the most part the maximum air contaminant levels set forth in Tables Z-1, Z-2, and Z-3 of 29 CFR 1910.1000.

Congress recognized the need to update and add new standards. It created two mechanisms for this purpose: Regular or "6(b)" standards and emergency or "6(c)" standards.

Congress specified the procedures for developing and promulgating regular standards in sections 6(b)(1)-(4) and 6(f). These sections provide that: The public may petition for new standards; OSHA may set up an advisory committee to assist in developing a standard; and, before issuing a standard, OSHA must publish a proposal with an explanatory preamble, request public comments and then publish an explanatory preamble with a final standard. In addition to these general requirements of informal rulemaking, Congress specified that OSHA must hold an oral hearing if requested and support its determination with substantial evidence in the rulemaking record.

Congress also provided in section 6(c) for the issuance of Emergency Temporary Standards (ETS) to take immediate effect without rulemaking. However, OSHA must then complete a section 6(b) rulemaking within 6 months. The criteria for issuing an ETS is that "employees are exposed to grave danger from exposure to substances or agents determined to be toxic or physically harmful or from new hazards, and that such emergency standard is necessary to protect employees from that danger."

OSHA has found that section 6(c) procedures have not generally accelerated the regulatory process. Most ETS's have been litigated and judicial stays have been issued either on procedural or substantive grounds.

Since the passage of the Act in 1970, OSHA has made substantial progress in improving the occupational health of workers for some priority health hazards. Asbestos and arsenic exposures have been dramatically reduced, substantially reducing cancer risk to employees. Lead exposures have been reduced and we are now seeing a major reduction in employee blood lead levels, and lead related diseases. Cotton dust exposures have been reduced and byssinosis has been nearly eliminated from the textile work force. OSHA has also substantially reduced significant health risk from some of the newer chemicals such as ethylene oxide and vinyl chloride.

Through the hazard communication and access to employee exposure and medical records standards, OSHA has greatly expanded the ability of employees to learn about and protect themselves from health hazards.

OSHA's standards have proven to be feasible, often costing less than estimated. The vinyl chloride standard cost one-tenth OSHA's contractor's estimate. The cotton dust standard has been credited with improving the industry's competitiveness and productivity by stimulating major technology improvements while costing one-half OSHA's estimate.

The preambles to OSHA standards have been lengthy, detailed and sophisticated. They have thoroughly analyzed health studies and controversial scientific issues about carcinogenicity and risk assessment. Extensive analyses of feasibility have been made.

OSHA has issued only 24 substance-specific health regulations since its creation. It has not been able to review the many thousands of currently unregulated chemicals in the workplace or to keep up with reviewing the several thousand new chemicals introduced since its creation.

Using past approaches and practices, OSHA could continue to regulate a small number of the high priority substances and those of greatest public interest. However, it would take decades to review currently used chemicals and OSHA would never be able to keep up with the many chemicals which will be introduced in the future.

OSHA believes it is a major priority to update its existing PELs and to make a substantial effort to control exposure to chemicals newly used in the workplace for which no exposure limits exist. The existing health literature and expert judgment indicate that such new or lower limits are needed to protect against many types of deleterious health effects. These include kidney and liver diseases, respiratory diseases, reductions in lung function, nerve disorders and reduction in nerve function, carcinogenicity, irritation to the eyes, throat, skin and other organs which prevent working safely, and many other disorders and dysfunctions.

As the final regulatory analysis indicates, millions of employees are exposed to levels of these chemicals which, the literature or expert opinion indicates, do or may create deleterious health effects. Clearly, it is a most important occupational health priority to reduce or eliminate such disease and material impairments of health.

Congress clearly indicated that it was a major Congressional priority to consider and control, when needed, the many thousands of unregulated chemicals, and update the existing Z-Table chemicals. For example, the previous quotation indicated Congress' concern with the thousands of newly introduced chemicals. Congress also stated:

> Accordingly, it is essential that such standards (Table Z chemicals) be constantly improved and replaced as new knowledge and techniques are developed. In addition there are occupational harzards, particularly those affecting health—which are not covered by any standards at all. (Senate Report 91-1282, p. 6.)

Government agencies and professional organizations have also recommended that OSHA lower exposures for many Z-Table substances and add limits for currently unregulated substances. The National Institute for Occupational Safety and Health has recommended new or lower exposure limits for approximately 190 chemicals (RELs) in its Recommendations for Occupational Safety and Health Standards, Sept. 1986.

The American Conference of Governmental Industrial Hygienists (ACGIH) 1987-88 Threshold Limit Values (TLVs) adopted new exposure limits for approximately 164 substances not regulated by OSHA, and lower limits, short-term exposure limits, ceiling limits or skin designations for 212 substances now regulated by OSHA.

In light of its priority to address the many unregulated health hazards and improve the existing Z-Table limits, OSHA commenced a review process to determine the best way to achieve this goal. It reviewed its past history and set up an internal task force to consider the matter. In addition, OSHA requested the Administrative Conference of the United States to study the issue and make recommendations.

OSHA's analysis indicated a number of reasons why the standards development process takes so long. These are discussed below. As can be seen some are within OSHA's control and some are not.

OSHA, in the past, has determined which substances it would commence standards development activity upon either through response to petitions or internal reviews. The time and resources spent analyzing what should be done next has been considerable.

An exhaustive review of the literature for each substance has been completed prior to initiating rulemaking. Detailed presentations for each study and lengthy discussions of every conceivable issue have been completed.

The lengthy preamble which has become a regular part of each standard is largely the result of the need for OSHA to defend its standards in suits brought inevitably by both industry and labor. See for example *Synthetic Organic Chemical Mfgs.* v. *Brennan*; *Oil Chemical and Atomic Workers* v. *Brennan*, 503 F.2d 1155, 506, F.2d 385 (3rd. Cir. 1974); Public Citizen, *Health Research Group et al.* v. *Tyson*; *Association of Ethylene Oxide Users* v. *Tyson*, 796 F.2d. 1479 (D.C. Cir. 1986). OSHA feels more confident in the successful defense of a standard if all possible issues have been exhaustively explored.

Individual standards have included a full range of ancillary provisions such as monitoring, medical surveillance, action levels and work practices. This increases the issues that must be studied and discussed, adding to the time taken to complete a standard.

OSHA has performed technical and economic feasibility analyses as required by statute. These have been made lengthier and more time consuming because a range of possible alternative exposure limits have been explored.

The regulatory process is also longer and more resource intensive because of analyses required either by statute or executive order. OSHA develops Environmental Impact Statements as required by the National Environmental Policy Act, conducts Regulatory Flexibility Analyses as required by the Regulatory Flexibility Act and conducts detailed analyses required by the Paperwork Reduction Act. All of the Presidents who have been in office during OSHA's existence have stressed the need to reduce inflation and improve the cost effectiveness of regulations.

Under various Executive Orders (E.O. 12044, 12291, etc.) OSHA has been required to perform extensive economic analyses.

OSHA has also followed more extensive and elaborate administrative procedures than other health regulatory agencies. In addition to extensive preambles to the proposed and final regulations, there is usually advance notice of a proposal in the **Federal Register**. There is a complete rulemaking docket into which the Agency places all the studies it relied upon. In addition to public comment and an oral hearing as required by law, opportunity is given for post hearing evidence and briefs. During the hearing, questioning of witnesses, OSHA and its contractors is permitted.

Consequently OSHA has permitted the public more extensive procedural opportunities than its statute, the Administrative Procedures Act, or legal doctrine require. See the procedural rules in 29 CFR Part 1911 and *International Harvester* v. *Rucklehaus*, 478 F.2d 619 (D.C. Cir. 1973). These procedures can increase agency knowledge and have been commended by the courts. (See *Industrial Union Dept.* v. *Hodgson* 499 F.2d 467 (D.C. Cir. 1974). However, they do mean that if delays and continuances are granted at each stage, the length of the rulemaking process is substantially extended.

OSHA in its first 17 years has also had to address difficult scientific feasibility and policy issues. These include extrapolation from animal data to humans (ETO supra), epidemiology, risk assessment and significant risk analysis (Arsenic, 48 FR 1864, January 14, 1983; Asbestos, 51 FR 22612, June 20 1986), feasibility for industries with aging facilities (lead, arsenic, supra), lowest feasible level (Benzene, 52 FR 34460, September 11, 1987, for example and others). Naturally when considering such issues for the first time, an Agency desires to go through extensive reviews before reaching final decisions.

OSHA consulted with the Administrative Conference of the United States (ACUS) to determine what would be appropriate procedures to respond to the issue of the large number of chemicals which need new exposure limits. The Conference issued two lengthy reports of a study conducted by two professors of administrative law. After extensive consideration, the ACUS made two sets of recommendations to OSHA. Recommendation 87–1, 52 FR 23629 (1987) and 87–10, 52 FR 40147 (December 30, 1987).

The Administrative Conference specifically recommended.

1. Updating the 1971 Consensus Standards. The Occupational Safety and Health Administration, as an interim step, should continue to update the Table Z national consensus standards adopted in 1971 if updating can be accomplished by expedited rulemaking procedure (e.g., including more concise preambles) appropriate to the nature of the revised table. OSHA should update the 1971 standards on a generic basis (i.e., include multiple standards in one proceeding) when consensus recommendations are available which are generally accepted by employers and workers in the affected industries, and when the new standards can be evaluated on the basis of risk and feasibility information reasonably available to the Agency. This interim step should not interfere with OSHA's continuing responsibility to promulgate and modify safety and health standards.

As this discussion indicates, there is a clear and generally recognized need to improve occupational health protection of workers from a substantial number of chemicals which are present in the workplace. Clearly an improved approach to regulation is needed to solve this problem in a reasonable time period. OSHA's traditional approach, which has permitted on the average less than two major health regulations per year, is not adequate to address the backlog of at least 400 chemicals generally recognized as needing new or lower exposure limits. OSHA has reviewed the law, Congressional intent, its history, and the recommendations of experts. Based on this review, OSHA adopted the approach described in Section IV–C which it has followed to accomplish the crucial goal of improving occupational health protection of workers. OSHA concluded that this approach has a greater health benefit and will prevent more deaths and various deleterious health effects, than could be achieved by allocating the same resources to comprehensive rulemaking for a small group of substances.

B. Chronology of Regulation

The public process followed by OSHA to implement this rulemaking was started on October 26, 1987, when the Department of Labor published its Semiannual Agenda of Regulations at 52 FR 40494–40534. The entry titled "Permissible Exposure Limit Update" states OSHA would propose a wide scale updating of its exposure limits. That entry scheduled March 1988 for the proposal and October 1988 for the final regulation. It also indicated that the American Conference of Governmental Industrial Hygienists had updated many of its recommended exposure limits (TLVs) since 1968. The 1968 TLVs were used as the basis of most of OSHA's existing 6(a) exposure limits. A similar notice was published in the next Semiannual Agenda on April 29, 1988, at 53 FR 14024.

OSHA published its proposal, "Air Contaminants, Proposed Rule" on June 7, 1988, at 53 20960 (Ex. 2). That document filled 433 **Federal Register** pages. It considered whether exposure limits should be changed for 428 substances. New or lower limits were proposed for 402 substances, one was proposed to be raised and 25 were proposed to be unchanged.

That document included a 21 page discussion of the history, approach and general issues. There followed 250 pages of health discussions which included general discussions by type of effect (cardiovascular, kidney/liver, etc.) and a concise individual discussion of the health effects associated with exposure to each substance. The more important or controversial substances had somewhat longer discussions than other substances. Each individual substance discussion stated the health effects, summarized the major relevant studies, and stated the reason OSHA preliminarily concluded significant risk did or did not exist. Finally, the complete Preliminary Regulatory Impact, Regulatory Flexibility and Feasibility Analysis was printed running 71 pages along with one of the supplements on methodology. (In this instance one **Federal Register** page equaled approximately five double-spaced typed pages.)

OSHA placed in the public docket either before June 7th, or shortly thereafter, all of the studies or documents upon which it relied. This included Exs. 1–1 to 1–1208, which constitute virtually all of the health studies upon which OSHA relied and that were discussed in the preamble (a few minor foreign studies referenced by ACGIH were not available). Also included were many data bases on occupational health such as the ACGIH documentation, NIOSH publications (for example, NIOSH–TIC, Ex. 7, is a 2500 page summary of health effects organized by chemical) and exposure limits of other countries.

Also placed in the Docket were 6 supplements to the Regulatory Impact and Feasibility Analysis (Exs. 4A–4F, approximately 1500 pages). Four volumes of exposure, control technology and feasibility data organized by chemical were placed in the record as Ex. 6 and a computer data tape for these volumes was made available.

The June 7 Proposal scheduled July 1 for notices of intent to appear at the hearing, July 8 for the submission of

comments and testimony, and July 20 for the hearing to begin. It suggested August 12 for post-hearing evidence and August 26 for post-hearing briefs.

OSHA received several requests for extensions of time, some for quite extended periods such as 9 months. On July 1 (53 FR 24956) (Ex. 5) OSHA granted a brief extension. Comments and testimony were due July 25 and the hearing was scheduled to commence July 28. August 19 and September 2 were recommended respectively for post-hearing evidence and briefs.

In response to the proposal, OSHA received 1248 timely comments (Ex. 3–1 to 3–1248) and 204 late comments (Ex. L–3–1249 to L–3–1452). Approximately 800 of the comments and most of the late comments were very similar letters generated by trade associations interested in the regulation of grain dust, wood dust or styrene. The balance of comments ranged from short to 3000 page submissions expressing views on the Proposal and including various studies.

OSHA also received 92 Notices of Intention to Appear (Exs. 8–1 to 8–92). Approximately half of those included copies of testimony and substantive evidence to be presented at the hearing. Established and published procedures require that persons may testify for no longer than 10 minutes at the hearing without submitting a notice.

The public comments, evidence and testimony totaled approximately 25,000 pages. This included actual comments and views, and attached health and feasibility studies.

OSHA submitted to the docket in a timely fashion on July 25, 1988, statements by the Director of Health Standards, the panel of economic witnesses, and 7 other witnesses requested to testify by OSHA. NIOSH also submitted on that date its comments, testimony and views.

In addition, for the convenience of the public, NIOSH placed in the record the paper copy of all health studies on each substance in the rulemaking organized by substance. This included the minor studies as well as the major ones that OSHA had relied on and had already submitted to the record. This submission was lengthy; however, all this information was indexed in the NIOSH Registry of Toxic Effects of Chemical Substances (RTECS) which is a 5 volume bibliography of occupational health studies which NIOSH is required by statute to create and maintain. (Section 20(a)(6) of the OSH Act). The RTECS have been available for many years. The vast majority of studies NIOSH placed in the docket were publicly available either in major libraries or from several computer data bases. Consequently they had all been readily available to the public for many years. Participants testified that they could be easily researched (Tr. August 4, Test. of Factor).

As OSHA had expected, a review of the comments and testimony indicates that of 428 chemicals in the rulemaking, approximately 280 received no comments or testimony and OSHA preliminary conclusions on risk and feasibility were reasonable and correct. Approximately 100 substances received only limited mention by one or a few participants. Approximately 40 substances received substantive comments but in some of these instances OSHA's proposed PEL was not necessarily controversial. For example, the comments may have been directed at technical improvements. Finally, approximately ten substances received substantial comments and were deemed to be controversial. Because of this filtering process, the rulemaking, as OSHA had anticipated, developed a narrower and more manageable form.

Also, as had been OSHA's prior experience, major industry trade associations were already formed, represented by major law firms, to express views on the more controversial substances. For example, the Styrene Institute, which has been in existence for 12 years, submitted about 2500 pages of comments, studies and attachments (Ex. 3–742) and was represented by Keller & Heckman. The Carbon Disulfide Committee, which has been in existence 11 years, submitted approximately 1000 pages of comments and attachments (Ex. 3–747) and was represented by Gibson, Dunn and Crutcher.

The Trade Unions also made major submissions on the chemicals they were interested in. The Food and Allied Trades Department of the AFL–CIO submitted extensive information on grain dust (Ex. 3–751). The Amalgamated Clothing and Textile Workers Union submitted extensive information on perchloroethylene (Ex. 43). The AFL–CIO (Ex. 39), United Autoworkers (Ex. 42), Workers' Institute of Safety and Health, United Paperworkers, Carpenters and Woodworkers, and other unions also made major submissions.

The oral hearing commenced on July 28. It extended for 13 hearing days with an average of approximately 7 hours of actual hearing time each day. The total transcript was approximately 4000 pages. Approximately 200 witnesses testified and responded to questions. Although there were some limits on questioning by an individual participant, the questioning in total was extensive since there often were more than 10 participants who questioned a particular witness. It was evident that all participants had been able to ask all the questions they wished by the end of the hearing.

The OSHA Staff panel responded to questions for three-quarters of one hearing day. The OSHA economics panel responded to questions on three separate occasions so that the public could complete questioning and have an opportunity to ask questions after having reviewed all OSHA submissions. The other OSHA witnesses fully responded to questions as did NIOSH (Tr. August 1). NIOSH agreed to return for further questions, but participants who initially requested the right to ask further questions withdrew their requests.

OSHA submitted to the Docket on July 25, 1988, approximately 40 site visit reports and on August 8, 1988, approximately 40 more site visit reports were submitted. The site visits were supplementary to the survey data and were not the basis of OSHA's initial feasibility conclusions. The OSHA economics panel returned on August 15, 1988, to answer questions specifically on those visits.

OSHA's initial goal was to complete 100 site visits. However, the visits needed employer approval both for the visit and the subsequent report before the visit could be undertaken and/or the report submitted to the Docket. Consequently OSHA could not completely control the completion of the site visits or submission of the report to the Docket. OSHA completed several additional site visits, but could not complete the related reports prior to the end of the hearing. OSHA submitted those reports to the employer in order to give the employer the option of putting them in the docket. Employers in the steel industry and styrene users did submit several such reports to the docket. Employers with grain exposures initially refused to permit site visits. Although some later agreed to site visits, it was by then too late to complete the visits and submit the reports to the record, so the site visits were not made.

At the end of the hearing, the presiding officer indicated that, because of the broad scope of the hearing, post-hearing evidence should be due 90 days after the close of the hearing and post-hearing briefs 120 days after the close of the hearing (Ex. 81). OSHA indicated both at the hearing and by letters and telephone calls to participants that a shorter period was required to maintain the schedule necessitated by the high

priority of the Proposal; OSHA stated that it did not think this would be unfair to participants (Exs. 60–9, 81 A). By Federal Register notice of September 7, 1988, (53 FR 34708; Ex. 100), the Secretary of Labor set October 7, 1988, for post-hearing evidence and October 31, 1988, for post-hearing briefs. (This matter is discussed more fully in the Section III, Legal Authority.)

OSHA received 57 post-hearing submissions from public participants totaling approximately 9000 pages. OSHA also received 41 post-hearing briefs. The total record includes substantially more than 4000 separate documents (individual studies, statements, comments, etc.).

The record was closed and certified by the presiding officer on November 10, 1988.

C. Details of Approach Used to Develop Regulation

The first step OSHA took to increase the pace of the regulatory process was to make a determination not to analyze individual substances in order to decide if they were of sufficient priority to be included in the project. Rather OSHA reviewed existing data bases and lists of recommended exposure limits, and determined which of these should be the starting point for the Proposal.

OSHA concluded that the National Institute for Occupational Safety and Health's (NIOSH) Recommended Exposure Limits (RELs) and the American Conference of Governmental Industrial Hygienist's (ACGIH) 1987–88 Threshold Limit Values (TLVs—a copyrighted term) provided the best two lists of substances to be considered for regulation and to provide a starting point for individual substance permissible exposure limits (PELs). See the discussion at 53 FR 20966–7. These lists of substances are developed by organizations of experts very knowledgeable both about the American work place and the health literature. See the testimony of Mastromatteo (Ex. 22) and NIOSH statement (Ex. 8–47). Both NIOSH and ACGIH publish documentation to support their recommendations and permit outside participation in the development of exposure limits.

Based on further analysis, the ACGIH TLVs were picked as the single best list to define the substances to be included in this rulemaking. The details of this OSHA analysis are provided in the Proposal (53 FR 20966–20967) and will not be repeated here. OSHA's major reason was that the TLVs are more extensive than the RELs and more generally used. There are over 600 TLVs and approximately 160 RELs.

By using the ACGIH list of TLVs as the basis for the selection of substances to be considered for this regulation, OSHA has greatly reduced the time it would take to proceed with this rulemaking. The ACGIH's list is broad in scope, attuned to the American workplace and developed by experts with substantial health expertise. Therefore, OSHA concludes that this approach to determining which substances will be considered for regulation is rational and allows OSHA to expedite the process of improving the health of American workers.

No changes to existing limits were considered in this rulemaking for substances covered by limits established in substance-specific section 6(b) rulemaking (24 substances) or substances for which the process of section 6(b) rulemaking has already been initiated (9 substances). Since OSHA had already begun the process of detailed analysis of these substances, additional review was determined to be unduly repetitive and confusing at this point.

OSHA then compared the permissible exposure limits in Tables Z–1, Z–2, and Z–3 to the TLV list. If the TLV and OSHA permissible exposure limits in the Z-Tables were identical, the substance was not considered for change of PEL in this proposal. The basis for this approach is that there is less likelihood to be a need for a change in an exposure limit if an organization which regularly reviews the literature has not changed its recommendation.

If the TLV and the PEL differed, the substance was considered for change of PEL in this rulemaking. Also substances for which there was a TLV, but no PEL, were included in this rulemaking. There is additional elaboration on this methodology in Section IV. D., Boundaries to Regulation.

The second approach OSHA used to shorten the rulemaking process was to rely to a greater extent than in the past on research and recommendations already made by NIOSH and ACGIH as a starting point for OSHA's analysis.

Both organizations have experts undertake a complete review of the literature for individual substances. Then they propose recommendations and permit outside comments on their proposed recommendations. At the next stage, each has a committee of experts again review the literature, as well as the comments on the initially published recommendations before determining the recommended exposure limit.

The approach OSHA followed was to first determine if the ACGIH–TLVs and NIOSH RELs were similar. If they were, or if there was no NIOSH REL, then OSHA reviewed the ACGIH documentation and recommendation. The ACGIH documentation includes summaries and analyses of the major studies. If the REL and TLV differed significantly, OSHA reviewed the studies and reasoning upon which both NIOSH and ACGIH recommendations were based, and then chose the recommendation which in OSHA's view was more appropriate.

In its review OSHA determined first whether the studies and analyses were valid and of reasonable scientific quality. Second, it determined, based on the studies, if the published documentation of the REL or TLV would meet OSHA's legal requirements for setting a PEL. Thus, OSHA reviewed the studies to see if there was substantial evidence of significant risk at the existing PEL or, if there was no PEL, at exposures which might exist in the workplace in the absence of any limit. Third, OSHA reviewed the studies to determine if the new PEL would lead to substantial reduction in significant risk. If this was so, and if the new PEL was feasible (see discussion below), OSHA proposed the new PEL.

OSHA then divided the chemicals into 18 categories, generally by health effect but, in a few instances, by other criteria. These categories included cardiovascular, liver-kidney, respiratory and other types of diseases or material health impairments. Each of these categories received an individual literature review and discussion in the preamble analyzing the etiology of substances which cause that health effect.

Some substances have several effects; the category chosen for each substance was based on the health effect which most influenced the exposure level proposed. However, OSHA individual discussions and conclusions referenced and were based on all health effects associated with the specific substance.

Following each general discussion in the preamble, OSHA summarized the documentation and provided references for each individual substance and stated the reasons for the proposed new exposure limit. OSHA also stated the reasons why it preliminarily concluded that the proposed new limit would substantially reduce significant risk for that substance. In the case of 25 substances, OSHA explained why after review it did not propose a new exposure limit. In one instance OSHA indicated the reasons for proposing to raise a limit.

This method fully informed the public of the basis for OSHA's decisions. The public was then in position to support or

challenge OSHA's proposal, to criticize the studies upon which OSHA relied, and to supply any additional studies, evidence or views during the comment period, during the hearing or as post hearing submissions.

OSHA is gratified by the degree of support for the proposal expressed by rulemaking commenters. For example:

We believe this proposal is one of the most significant steps taken by OSHA since its inception. The time and resources required for substance specific rulemaking have greatly limited the number of PELs that OSHA has been able to revise since they were adopted in 1971. Continuing individual rulemaking would result in adding to the backlog of outdated PELs, while the method chosen by OSHA for this revision assures that comprehensive update will be completed within a reasonable time (Tr. August 2, Testimony of Tamarelli; SOCMA).

CMA supports the concept of revising the Z Table PELs in order to conform with the threshold limit values, TLVs, that have been adopted or updated by the American Conference of Governmental Industrial Hygienists * * * It is hard for us to understand how anyone could say that the proceeding is too limited in scope. To the contrary, a much more valid criticism might be that OSHA has bitten off more than it can chew. The Agency quite reasonably has concluded that adjusting the permissible exposure limits for chemicals on the TLV list should be its first order of business (Tr. August 10, Testimony of Lynch/CMA).

OSHA has taken a truly significant step in updating and enhancing the regulatory provisions applicable to the workplace * * * OSHA was prudent in our view to rely on the ACGIH TLVs to establish the bounds of the rulemaking (Tr. August 9, Testimony of Holthouser/RMA).

GE strongly supports and endorses the Occupational Safety and Health Administration's 6(b) rulemaking efforts to revise and upgrade the Z Tables in 29 CFR 1910.1000 and encourages everyone concerned about employee health, along with those involved in the rulemaking effort, to pursue a timely conclusion to the process (Tr. August 9, Jones/GE).

The American Industrial Hygiene Association (AIHA), on the other hand, was entirely in favor of OSHA's use of either an ACGIH or NIOSH limit, as the case requires:

AIHA supports the adoption by OSHA of NIOSH REL values as PELs on a case-by-case basis where such values are supported by the scientific evidence and are feasible from the standpoint of implementation (Ex. 8–16).

NIOSH expressed strong support for this rulemaking in general but submitted specific comments on a number of substances that it believes should have different limits from those proposed (Ex. 8–47). NIOSH's substance-specific comments are addressed in connection with the preamble discussion of these substances in Section VI.

Union representatives concurred with the need to update the Z-Tables. For example, M. Seminario/AFL–CIO stated: "We are pleased that OSHA and many industry representatives have acknowledged finally that the current permissible exposure limits do not protect workers, and we do indeed support regulatory action to update the standards for toxic substances through the use of board-based rulemaking such as the Agency has proposed here. (Tr. August 4, Testimony of Seminario/AFL–CIO).

However, Ms. Seminario did not agree with the approach that OSHA followed stating that "it does not provide the workers with the kind of protection that the OSHA Act requires." Union representatives stated that the proposed standard was not adequate since it did not cover some substances of concern and did not include the ancillary provisions which they felt were important.

OSHA does not agree with this judgment. It is impossible to cover all substances, and OSHA has made a rational and reasonable judgment regarding the bounds of this standard which is supported by most industry and professional associations. OSHA is approaching the subject of ancillary provisions through separate generic rulemakings which have already been initiated. The basis of these judgments are discussed in detail in other parts of this preamble.

OSHA stated in the proposal that it would consider all the additional views and studies presented by participants. Based on what was best supported by the entire record, OSHA would issue as the final standard either the PEL it had proposed, make no change to the existing PEL, or issue a different PEL. OSHA has followed this method of analysis in issuing the final rule.

OSHA's approach has indeed made it possible to increase the efficiency of the regulatory process and issue new and revised PELs to protect the health of workers from a large number of substances which were unregulated, or for which existing exposure limits are out of date. It has also permitted OSHA to rely on the best available scientific information and its past experience, while giving the public both excellent notice and a full and fair opportunity to comment, submit additional studies and make recommendations.

The improved efficiency of this rulemaking effort has not come from sacrificing scientific validity. It has resulted from combining discussions by health effect, concentrating on major issues and studies, using as a starting point the research of expert organizations, and using public comments to bring to attention additional relevant studies and issues. OSHA has addressed in more detail those substances, issues and studies which have been identified in the comments as the most controversial.

In addition, OSHA has increased the efficiency of its feasibility analysis. Its prior substance-by-substance, industry sector by industry sector, process-by-process approach would have resulted in a vast body of duplicative information in a multi-substance rulemaking. In addition, it would have made it impossible to update very many substances in a reasonable period because of the time and resources required.

OSHA followed several approaches to increase the efficiency of its feasibility analysis process. First, it made maximum use of existing information. OSHA's Integrated Management Information System (IMIS) is probably the largest source of accurate exposure date in the world. It has 77,000 exposure measurements, is organized by industry and process, includes judgments by compliance officers who are experts in industrial hygiene, includes the number of workers represented by each measurement, and is computer readable.

OSHA also used the two National Occupational Hazard Surveys (NOHS) by NIOSH. These are good sources of the number of workers potentially exposed to substances in each industry segment. OSHA also analyzed the large volume of data in various publicly available data bases on control technology for various substances, processes and industries. For the convenience of the public, much of these data were combined into four volumes which were made available to the public in the docket. OSHA also stated it would supply these data on computer tape if requested.

OSHA had these data reviewed by approximately twenty experts in industrial hygiene and industrial engineering. They made estimates of substances likely to be used and processes likely to be present in each industry sector covered by this regulation. These estimates were used as starting points. Much more extensive information was gathered in a nationwide survey of 5700 firms.

These experts also made estimates of the cost to reduce exposure based on scale of operation, type of process, and degree of exposure reduction needed. Standard source materials such as industrial manuals were used. Many processes are relatively standardized throughout industry and are used for a

variety of substances. For example, vat mixing takes place for many substances in many industries. It can be uncontrolled without a cover and involve manual loading of dry chemicals. It can be partially controlled with covers and pump-loading of liquids. It can be fully controlled with enclosure, ventilation and automated loading and unloading. Likely exposures can be estimated by determining the amount of chemicals used and degree of existing controls. Costs can be generalized throughout much of industry from the size of the operation, estimated exposures, and the cost to go from one degree of control to the improved level of control needed to achieve the proposed reduction in exposures.

With this method of analysis, it is possible to make estimates of exposures, controls necessary, exposure levels which can be achieved and costs from data on substances and processes present and numbers of operations for each industry segment. This information is sufficient for determining technical feasibility and costs by industry segment. These data combined with publicly available sales and profit ratio data make it possible to estimate economic feasibility by industry segment.

To gather data on the substances present, types of processes, number of processes, and controls in place by industry segment, OSHA commissioned the largest survey it has ever conducted. Over 5700 questionnaires were administered throughout the covered industry segment based on statistically valid sampling techniques. A vast amount of information was received on substances present, processes used and controls in place.

OSHA concludes that this approach is accurate on an industry sector by industry sector basis for individual processes.

Overall, OSHA has a high degree of confidence that its estimates of technical feasibility, costs and economic feasibility are accurate. OSHA has had far more data available to it than it normally does in a single substance rulemaking. The data were gathered systematically and were combined using a methodology that was statistically valid and devised by persons with great expertise.

In addition, to increase the efficiency of the process, OSHA analyzed the feasibility of the specific proposed exposure level for each substance rather than considering a variety of different exposure levels.

To permit public comment on this approach and related data in an efficient manner, OSHA published the entire Preliminary Regulatory Impact Analysis with the Proposal in the **Federal Register**. In addition, it was made available to the public in the docket at the time of publication six supplements which described in great detail the methodology and results of the survey by sector.

OSHA concludes that it provides the public with all the information participants would need to comment on, criticize or support OSHA's feasibility conclusions. However, some participants requested more detailed analysis of their sectors. Although OSHA indicated that it believed the date it had made available were sufficient for these purposes, it did where possible supply additional data and make special computer runs when requested by the participants.

OSHA also stated it would consider all additional feasibility data submitted by the public. Many participants did supply additional data. OSHA has reviewed all data in the record in reaching its final feasibility conclusions.

OSHA concludes that the approach it took developed good feasibility data, permitted participants a reasonable opportunity to review OSHA's data and supply their own, and was necessary to make the feasibility analysis process more efficient.

The fourth difference in approach from single substance rulemaking was OSHA's decision to limit this rulemaking to the issue of exposure limits. OSHA has not considered medical surveillance, exposure monitoring, industrial hygiene requirements and other ancillary provisions which were not included in the existing 6(a) standard.

As stated in the Preamble, OSHA has concluded that the highest priority for protecting occupational health is to lower exposure limits for many substances where current knowledge indicates they are too high, or where currently there are no limits but recent scientific knowledge indicates limits are needed. This priority could not be achieved if ancillary provisions were considered at the same time. As discussed in Section III, Legal Authority, OSHA believes it is a rational use of its priority setting power to consider ancillary provisions subsequently either in other generic rulemakings or in substance specific section 6(b) rulemakings. It has already begun that process as discussed there. In any event, OSHA's approach significantly improves occupational health protection.

A final method OSHA has followed to make this rulemaking more manageable is to rely on its experience. OSHA has now made feasibility determinations for several dozen substances and significant risk determinations approximately one dozen times. Various issues regarding the analysis of data have been reviewed many times. OSHA's approaches have been reviewed by the courts and upheld or modified to meet judicial guidance. OSHA has not revisited all of the issues in quite the depth it has given them in the past in light of its experience. Of course, determinations and conclusions required by law have been fully analyzed and supported. OSHA concludes it is both rational to rely on its past experience and specifically permitted by section 6(b)(5) of the Act.

In the most important areas OSHA has not made any attempt to make the regulatory process shorter. First, as discussed, it included individual substance-by-substance health analyses and significant risk determinations. Second, it has made feasibility determinations on the impact of the regulation of all the substances for each industrial sector.

Third, OSHA has followed its traditional elaborate rulemaking process. Nine months, advance notice of the intent to issue a proposal was given. The Proposal explained OSHA's reasoning at great length by citing and discussing the evidence upon which OSHA relied. All the studies and analyses upon which OSHA relied were made available in the docket. More than the minimum period was allowed for comments.

Thirteen days were allowed for oral hearings. Testimony and evidence was required to be submitted in advance and the testimony of OSHA, its economic contractors and witnesses was made available in advance. Participants in the hearings were permitted to question the OSHA panel, contractors, witnesses and each other. Though the questioning permitted for each participant was not unlimited, time was provided at the end to ensure that each participant had completed all questioning he or she desired. As there were frequently more than 10 participants who questioned a single witness, the total amount of questioning was often extensive. The OSHA panel was questioned for approximately four hours and the economics panel for more than six hours. Various arrangements were made to bring back witnesses.

After the hearing, participants were allowed to file post-hearing evidence to respond to comments and testimony, and to supply materials which they could not submit by the deadline for

comments. An additional period was allowed for post-hearing briefs.

These are far more than the minimum procedural requirements of information rulemaking or hybrid rulemaking. Few if any agencies, for example, permit questioning of the agency and its contractors. The process also effectively permits participants a double round of comments.

The various time frames were shorter than some participants desired. However, there was approximately one year between initial notice and final opportunity for submissions and approximately five months from proposal to post-hearing briefs. Not only is this far more than the legal minimum, but it should have been ample to give sufficient time for participants to effectively present their views and supporting evidence.

Adhering to a schedule is crucial for an agency to accomplish a high priority, large scale project in a reasonable period of time. Among other reasons for this is the fact that extra staff must be borrowed and contractor assistance arranged. Both groups have other schedule commitments. If a rulemaking is delayed too long these resources become lost to the project. Moreover, medium length delays during the public participation period become very lengthy delays of a final rule. As OSHA has pointed out, the benefits of this standard to worker health are so significant that lengthy delays of the final rule would result in a major loss in health protection.

Finally, OSHA has fully met the requirements pursuant to statute and executive order to perform required analyses. OSHA has completed the Regulatory Impact, Regulatory Flexibility and other analyses as required.

D. Boundaries to Regulation

The Proposal defined the substances covered by this rulemaking as a sub-set of the substances listed in the 1987–88 Threshold Limit Values (TLV) published by the American Conference of Governmental Industrial Hygienists (ACGIH) (53 FR 20964–20966). OSHA pointed out in the Proposal that the TLV listing had several advantages over other possible lists that might be used for this purpose. Details of the OSHA analysis leading to this decision are noted in the Proposal (53 FR 20966–20967). The primary considerations leading to that OSHA decision were (1) number of substances covered by the TLV listing; (2) available written documentation for the TLVs; (3) potential employee exposures covered by TLVs; and (4) general acceptance of the TLVs by health professionals.

OSHA realized that there are different valid approaches to the question of identifying the boundaries for this type of rulemaking, and any decision must balance completeness with practicality. Several commenters recommended that the number of substances considered in this rulemaking be expanded to include: (1) Other lists; (2) the 160 substances in the existing Z-Tables which were not discussed in the Proposal since their current TLVs were identical with the existing OSHA PEL; and (3) substances which are in the process of active (6b) rulemaking.

Relative to the first point, the additional lists suggested included the following data bases: (a) Recommended Exposure Limits (REL) developed by the National Institute for Occupational Safety and Health; (b) Workplace Environmental Exposure Limits (WEEL's) developed by the American Industrial Hygiene Association; (c) EPA's Integrated Risk Information System (IRIS); (d) standards used by government agencies in the U.S.S.R. and other parts of eastern and western Europe; and (e) internal company limits. The following comments focus on this aspect: Ex. 8–47 (NIOSH), Ex. 43 (Frumin), Ex. 194 (AFL–CIO), Ex. 3–9 and Ex. 46 (Ziem), Ex. 42 C and 197 (UAW). Dr. Phillip J. Landrigan suggested (TR August 1, Test. of Landrigan): (a) Using a single alternative listing instead of the TLVs; (b) combining several lists to define the bounds of this rulemaking; or (c) adopting a smaller sub-set of the TLVs.

During the public hearing it was suggested that benefits would result from developing PELs for additional substances. Specific substances suggested for inclusion in this rulemaking included dimethylformamide (Ex. 47); polychlorinated biphenyls (Tr 7–123); and glycol ethers (Ex. 3–639).

OSHA considered these constructive suggestions intended to expand the scope of this rulemaking in an effort to improve the level of health protection afforded workers. OSHA realizes that there are various approaches to this type of rulemaking and believes that, while some of these suggestions have merit, they introduce untenable problems at this stage of the rulemaking process. OSHA has determined that it is preferable to consider some of these suggestions as part of possible follow-on rulemaking based on the following facts and analyses.

For many of the additional substances provided by these data bases there are no quantitative exposure limits (e.g. IRIS and some NIOSH RELs). For other substances it is not clear that the limits are actually applied to workplace compliance situations (e.g. U.S.S.R. and eastern Europe limit). For others, (internal corporate limits) an extended independent review procedure is not defined. Use of a multiplicity of data bases to define the bounds for this already large rulemaking would overwhelm the resources of OSHA and those concerned parties who wish to comment on any proposed changes. This would greatly delay prompt implementation of a regulation which is urgently needed to protect the health of approximately 17 million workers who are potentially exposed to the 428 substances for which revised PELs were considered in the Proposal. Additional delay would be necessary since OSHA would be required to public a new Proposal to include any substances not identified and discussed in the Proposal.

The record clearly shows that OSHA's decision to use the ACGIH TLVs as the bounds for this effort was generally supported by most commenters for a variety of reasons: Ex. 3–866 (ORC); Ex. 3–740 (ARCO); Ex. 3–741 and 196 (Dow); Ex. 170 (GE); Ex. 3–891 and 176 (SOCMA); Ex. 178 (API); Ex. 3–877 and 47 (RMA); Ex. 52 (HIMA) Ex. 3–678 and 58 (Abbott); Ex. 163 (Ergon Refining), Ex. 186 (Halogenated Solvents Industry Alliance) and Ex. 165 (CMA). These reasons include the general acceptance and probable feasibility of the TLVs, and the need to have clearly defined limits for this rulemaking so it can be concluded in a reasonable time period.

For example, Jeremiah Lynch speaking on behalf of CMA stated:

> It is hard for us to understand how anyone could say the proceeding is too limited in scope * * *. The Agency quite reasonably has concluded that adjusting the permissible exposure limits for chemicals on the TLV list should be its first order of business. Further refinements in the regulation of these chemicals can be dealt with at a later date, to the extent additional requirements are found to be necessary. (Ex. 64).

It is necessary to limit the number of substances included in this rulemaking so that it can be completed in a reasonable time frame. The total number of chemicals in existence is well over 100,000. The 1985–86 edition of the NIOSH Registry of Toxic Effects of Chemical Substances (RTECS; DHHS (NIOSH) Publication No. 87–114) contains 88,693 prime chemical substances. It is impossible to promulgate an OSHA regulation without limiting the number of substances under consideration to manageable proportions. In this regulation such limitations are based on several

considerations including the: (1) Extent of use in commerce; (2) potential for exposure; and (3) lack of any existing protective limits. The first two criteria are best satisfied by using the well established TLVs as the data base for defining inclusion in this rulemaking. Since the number of substances in the TLV listing would still overwhelm the available resources, OSHA determined that it was reasonable to defer for consideration at a later time those 160 substances for which an OSHA PEL already exists and for which no change in TLV has occurred.

Such exclusion from consideration of change of PEL in this rulemaking does not preclude OSHA from initiating 6(b) rulemaking in the future for any of these substances or for any of the other substances covered by this regulation. Because of this fact, OSHA believes that its initial decision not to consider changing the PEL for those substances where the 1987–88 TLV is identical with the existing OSHA PEL is appropriate.

OSHA also believes that it would unnecessarily complicate this rulemaking as well as the individual rulemakings if changes to existing PELs were considered at this time for the nine substances for which the 6(b) rulemaking process has already been started. The process of developing a 6(b) standard for a single substance differs from the process used in this proceeding since it involves consideration of various ancillary requirements (exposure monitoring, medical surveillance, use of personal protective equipment, labeling, etc.) which are not part of this rulemaking. Extensive dockets have already been developed for the nine substances in this category (Table IV–D–1). Since these 6(b) rulemakings should be completed in the near future as tentatively scheduled by the Regulatory Agenda (52 FR 40494–40542), it would unnecessarily complicate the rulemaking process without any significant benefit if these nine substances were included in this rulemaking.

Until the new regulations for these nine substances are adopted, the existing OSHA PELs will remain in effect as reflected in Table Z–1–A. This same procedure is also used for the 160 substances where the existing PEL is identical with the 1987–88 TLV, and for which new PELs are not proposed in this rulemaking. These 160 substances were listed in Table VII–D of the Proposal (53 FR 21254–21261).

It should be noted that no changes in PEL have been proposed for the 24 substances listed in Table VI–D–2 which are covered by individual 6(b) regulations. The existing PELs for some of these substances are incorporated into the Z–1–A Table both for reference purposes and because the individual 6(b) regulations for some of these substances do not cover all operations, making maintenance of these PELs necessary to provide protection to workers involved in these exempted activities (e.g. benzene, cotton dust, and formaldehyde).

While the TLVs and RELs were used as a starting point for defining PELs, it should be noted that OSHA made its own determination regarding each individual limit. This was based on further evaluation of: (a) The TLV Documentation and the Criteria Document supporting development of the REL; (b) submissions to the public hearing record; and (c) information used in developing some of the other data bases initially considered by OSHA in developing the Proposal.

For a few substances, commenters suggested that it would be preferable to delete a particular substance from this rulemaking and consider it as part of a separate single substance rulemaking. Such comments were specifically directed at wood dust (Ex. 3–748), grain dust (Ex. 3–752 and 3–755), sulfur dioxide (Ex. 8–65), and styrene (Ex. 3–742).

In the case of wood dust and grain dust it is imperative that OSHA act promptly since there is no existing accepted PEL for organic dusts. The Occupational Safety and Health Review Commission has held that the standard for nuisance dusts is *not* to be applicable to wood dust and grain dust. To initiate and complete a 6(b) standard to control these substances would take considerable time. Since there is a clear need for a PEL to protect against the significant risk associated with exposures to wood dust and grain dust, and there is now sufficient health and feasibility data to justify setting a PEL, it is imperative that OSHA act promptly to protect workers exposed to these hazardous substances. The analyses of these data are provided in the discussion of these substances in Section VI. In the case of wood dust, the Inter-Industry Wood Coordinating Committee indicated their concurrence regarding adoption of a 5 mg/m^3 standard, which represents part of the standard OSHA is proposing for wood dust. (Ex. 3–748 and 80).

In the case of sulfur dioxide, the commenter indicated that deletion was appropriate due to the existence of a past record (Ex. 8–65 and Docket No. H–039). OSHA agrees that the past public record must be considered, and has incorporated the previous SO_2 record into the record for this rulemaking (Ex. 10–45). OSHA has carefully considered all relevant information from the previous SO_2 record in making its decision regarding a PEL for sulfur dioxide, and the OSHA analysis of that record is included in the discussion establishing the PEL for sulfur dioxide.

In the case of styrene, acrylamide, and a few other substances, questions were raised regarding the adequacy of available information to develop a PEL in this rulemaking. Questions were raised regarding definition of carcinogenicity, feasibility (economic and technological), proper classifications of health effects, and the proper PEL (Ex. 3–742 and 70). In some instances, OSHA believes that sufficient information was not available to reach a final determination regarding carcinogenicity. However, information submitted by the commenters, together with material considered in the development of the Proposal, was adequate to permit OSHA to reach a conclusion regarding the PEL. The details of these analyses are included in Section VI.

In some instances OSHA has specifically indicated that a specific revised PEL may not fully eliminate significant risk of material impairment. In many instances this is due to information and data limitations noted in the discussion for that specific substance. However, the PEL is based on the best current interpretation of data available at the time of promulgation of the regulation. A PEL may change as more information becomes available, or more accurate analytical procedures are developed. As an example, the PEL for asbestos initially adopted in 1971 was revised in 1972. This level was modified in 1976 and revised again in 1986.

After due consideration of all suggestions to delete substances from this rulemaking, OSHA has determined that the only substance to be deleted from this rulemaking is chromyl chloride for which a PEL was considered but not adopted because OSHA had not given adequate notice in the Proposal. In the case of three other substances (asphalt, fibrous glass and mineral wool), a decision regarding a specific PEL is being delayed. OSHA has discussed the reasons for adopting each PEL in Section VI of the preamble to this standard.

As part of the public hearing submissions and presentations, several individuals suggested expansion of the rulemaking to include provisions for exposure monitoring and medical surveillance; Ex. 8–3 (Landrigan); Ex. 194 (AFL–CIO); Ex. 3–751 (Food & Allied Service Trades Dept.); (Ex. 42 and 197

(UAW); Ex. 43 (Frumin); Ex. 8–61 (Workers Institute); and 8–85 (Melius). OSHA has adopted ancillary provisions for each substance regulated through 6(b) rulemaking. After 17 years, these provisions are included in only the 24 existing individual substance OSHA standards. OSHA finds that this rulemaking is not the appropriate mechanism for extending the ancillary provisions to all substances covered by the Z–Tables and agrees with the following comments of PPG and Dow:

Further expansion of this rule to specifically impose additional regulatory requirements such as medical surveillance, recordkeeping, personal protective equipment, and training would unnecessarily complicate and confuse the main objective of this proposal rule. There is also a greater likelihood of challenge that has been an impediment to previous attempts to revise air contaminant levels such as the Standards Completion Progress Project. Ex. 3–1158 (PPG)

While we believe OSHA should not adopt medical surveillance and exposure monitoring provisions in this rulemaking, we do believe OSHA should promulgate generic medical surveillance and exposure monitoring standards in a timely fashion. Ex. 169 (Dow)

On September 27, 1988, OSHA published Advance Notices of Proposal Rulemaking (ANPRM) covering "Generic Standards for Exposure Monitoring" (53 FR 32591–32595) and "Medical Surveillance Programs for Employees" (53 FR 32595–32598).

It was pointed out by Dr. I. Rosenthal, Rohm and Haas Co., that "if in the future OSHA supplements up-to-date exposure standards with generic how-to standards addressing medical surveillance, monitoring, personal protective equipment and other similar items the Agency will have established over 400 defacto complete standards." (Ex. Tr. 3–17). OSHA therefore believes that consideration of exposure monitoring and medical surveillance is best achieved through the generic approach which has been initiated with the two September 27, 1988, ANPRM's.

The legal and policy justification for limiting this rulemaking to development of PELs is detailed in the Legal Authority Section of this Preamble. The appropriateness of addressing PELs prior to considering ancillary provisions was also endorsed on technical grounds by Dr. Marcus Key, former Director of NIOSH (Ex.– TR 1–233, TR 1–265; TR 1–266).

Several commenters were concerned with the computational formula presently noted in § 1910.1000(d)(2), for example, Ex. 3–742 (SIRC); Ex. 3–877 (RMA) and Ex. 165 (CMA). These concerns relate to the lack of a requirement that this formula should apply only to those situations in which an additive effect is present. In contrast, the ACGIH discussion of the Threshold Limit Value for Mixtures (TLV and BEI for 1988–89, p. 42) states that this equation is applicable, "when two or more hazardous substances, which act upon the same organ system, are present, their combined effect, rather than that of either individually, should be given primary consideration." This reference goes on to state that "exceptions to the above rule may be made when there is a good reason to believe that the chief effects of the different harmful substances are not in fact additive, but independent as when purely local effects on different organs of the body are produced by the various components of the mixture."

The Proposal only redesignated paragraph § 1910.1000(d) as §1910.1000(f) (53 FR 21263), a change which is no longer necessary. There was no intent to reconsider or clarify this paragraph as part of this rulemaking. This subject was not discussed in the Proposal and was *not* a topic for consideration as part of this rulemaking. Therefore, it is not appropriate to consider any changes to the mixture equation at this time.

During the Public Hearing a few commenters made specific suggestions regarding other expansions to the subjects covered under this rulemaking. These included:

(1) Expansion of the PELs to non-traditional work shifts (TR 3–231, 3–234 (Arco)). OSHA is aware that work schedules in excess of 8 hrs/day are becoming more common. However, it is clear that this rulemaking did not provide an appropriate platform for full discussion of the technical problems associated with adjusting PELs for work shifts other than an 8 hr/day. It appears that such a question is highly substance specific depending on the toxicology and body clearance mechanisms, and the significance of short term exposure peaks. As such it may be more appropriate to provide guidance in the form of an interpretation of acceptable alternate approaches to extrapolating the 8 hr. PEL to other work shift periods. This might be developed through the OSHA Industrial Hygiene Technical Manual or the Field Operations Manual so it could be implemented on a case-by-case basis. OSHA believes that this type of expansion of a Proposal intended to address only PELs is not justified. The use of PELs developed using a 10 hr. definition for developing 8 hr TWA PELs is supported by NIOSH testimony (Ex. 8–47) and this approach has been used in this rulemaking.

(2) Representatives of the Workers Institute for Health and Safety suggested incorporating surface contaminated limits, such as those recommended by NIOSH and EPA for polychlorinated biphenyls (PCB) that are used in the clean-up of the New Mexico State Highway Department Building (TR 7–123 and 7–124). Clearly this represents an exposure index which is significantly different from "Air Contaminants" which is the subject of this Proposal. OSHA therefore concludes that extension of this rulemaking to include consideration of surface contamination limits is not appropriate.

(3) Mr. Richard Henderson representing the Chlorine Institute recommended that, rather than reducing the PEL for mercury, OSHA maintain the existing PEL and develop a comprehensive standard which includes a requirement for periodic urinary mercury determination. The question of the appropriate PEL for mercury is discussed in Section VI of this preamble. Regarding the suggestion of urinary measurements which could be considered for many substances other than mercury, OSHA finds that this represents an exposure index which is significantly different from "Air Contaminants", the subject of this rulemaking. OSHA concludes that extension of this rulemaking to cover this subject is not appropriate.

In summary, OSHA has reviewed all comments to the record which might result in changes to the boundaries of the rulemaking defined in the Proposal. OSHA finds that the suggested additions and deletions would not be appropriate in light of the objectives for this rulemaking established by OSHA and dictated by the statutory requirements of the OSH Act.

Therefore, OSHA concluded that the 428 substances listed in Table I–E of the Proposal (53 FR 20968–20976) should be considered for change in the PEL as part of this regulation. These substances are listed in the Index-Locator Section (II) of this preamble.

The Z–1–A Table in this regulation also incorporates the existing PELs for: (1) The 160 substances from the existing Z–Tables, which were not considered for changes in the PEL; (2) 9 substances for which 6(b) rulemaking is in progress; and (3) some of the 24 substances covered by individual OSHA standards where some sectors are not covered by the individual substance standard.

The Z–1–A Table lists all substances covered by this regulation, whether or not the PEL has been changed, whether or not a 6(b) standard has been undertaken on a specific substance, and whether or not a 6(b) standard covers the substance either fully or partially. In the case of substances regulated by individual substance OSHA standards, the Z–1–A Table cross references the individual standard.

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Table IV-D-1 Substances for which OSHA Has Initiated 6(b) Rulemaking.

CHEMICAL NAME
1,3-Butadiene
Cadmium Dust and Fume
2-Ethoxyethanol (Cellosolve)
2-Ethoxyethyl Acetate
Ethylene Dibromide
Methyl Cellosolve
Methyl Cellosolve Acetate
Methylene Chloride
4,4'-Methylenedianiline

TABLE IV-D-2. Substances Regulated by OSHA Under Section 6(b).

CHEMICAL NAME	STANDARD
2-Acetylaminofluorine	1910.1014
Acrylonitrile	1910.1045
4-Aminodiphenyl	1910.1011
Arsenic (Inorganic)	1910.1018
Asbestos	1910.1001
Benzene	1910.1028
Benzidine	1910.1010
Bis-Chloromethyl Ether	1910.1008
Coke Oven Emissions	1910.1029
Cotton Dust	1910.1043
1,2-Dibromo-3-Dichloropropane	1910.1049
3,3'-Dichlorobenzidine	1910.1007
4-Dimethylaminoazobenzene	1910.1015
Ethylene Oxide	1910.1047
Ethyleneimine	1910.1012
Formaldehyde	1910.1048
Lead	1910.1025
Methyl Chloromethyl Ether	1910.1006
2-Naphthylamine	1910.1004

TABLE IV-D-2. Substances Regulated by OSHA Under Section 6(b) (continued).

CHEMICAL NAME	STANDARD
3-Naphthylamine	1910.1009
4-Nitrobiphenyl	1910.1003
n-Nitrosodimethylamine	1910.1016
b-Propiolactone	1910.1013
Vinyl Chloride	1910.1017

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E. Special Considerations

In the Proposal several substances were identified (53 FR 20978, Tables I–F–A, I–F–B, I–F–C and I–F–D) as requiring special attention. This was due to the presence of "significant differences" between the exposure limits recommended by the various data bases initially considered in this rulemaking. This same Section of the Proposal also identified some basic assumptions used to initially simplify the definition of "significant difference." The objective of this effort was to encourage comments during the Public Hearing process for those substances where there was greater potential for uncertainly regarding the proposed PEL.

Comments were received on some of these substances. NIOSH (Ex. 8–47) commented on all those substances for which a REL existed, as well as for many other substances noted in these four Tables. Extensive comments were submitted regarding the proposed PELs for Acetone (Ex. 8–54, 3–69, 3–661, 3–741, Tr. VI pp. 89–247); Acrylamide (Ex. 3–961); Carbon disulfide (Ex. 8–19, 8–45, 3–659, 3–674, 3–897, 3–945, 3–1158, 3–753); and Sulfur dioxide (Ex. 3–1123, 8–22, 8–57, 8–65, 3–349, 8–9). A considerable number of comments were also directed at the selection of RELs (SOCMA, Exs. 3–891 and 176; Dow Chemical, Ex. 3–741; Rubber Manufacturers, Ex. 3–877). For example, GE (Ex. 170) stated:

> It is apparent from the testimony given during the hearings and comments submitted into the rulemaking record that the Recommended Exposure Limits (RELs) are a source of considerable controversy * * * a wide cross-section of occupational health professionals from industry, government and academia believe there are substantial flaws in the the contract process under which NIOSH RELs were developed, including limitations in peer reviews, feasibility considerations, and the methodologies employed in the extrapolating animal toxicological data and limited epidemiology findings to workplace exposure limits. (Ex. 170)

NIOSH pointed out that "Each Criteria Document is reviewed by experts representing affected industries, organized labor, and trade or professional organizations, and by scientists, physicians, and other health professionals with related experience in academia, government, or industry. The number of these external peer reviewers normally is greater than 10 and often exceeds twice that number. In addition to the invaluable contribution their comments make to the completed Criteria Document, OSHA receives, along with the completed Criteria Document, the full text of each reviewer's written comments accompanied by itemized annotations indicating how the draft was modified in response, or providing the rationale if the comment or recommendation was not adopted. Each Criteria Document contains an extensive summary in which the basis for the Recommended Exposure Limit (REL) is carefully developed with clear and explicit citation of the data relied upon at all steps of the logical development. No other source of exposure limits approximates the comprehensiveness of these documents" OSHA believes that its use of the RELs in this rulemaking is appropriate.

These submissions achieved the OSHA objectives of stimulating the input of new data, analyses, and information to assist OSHA in this rulemaking based on the full record for each individual substance. The discussion and application of this information to set specific PELs is included in the individual substances discussions in Section VI of this Preamble.

While OSHA did initially identify basic assumptions regarding significant differences (53 FR 20977), the final OSHA decision regarding selection of a specific PEL is based on a case-by-case assessment of the health effects, significant risk, material impairment of health, available sampling and analytical methods, and technological and economic feasibility considerations.

Another special consideration was the question of dealing with substances for which there are sampling and analytical limitations (Table I–F–E of the Proposal, (53 FR 20978)). This was also identified as a concern in Question 9 of the Proposal (53 FR 20961). The responses to this question are detailed in Section V of this Preamble. OSHA concurs that adequate sampling and analytical methods are required to permit enforcement of a PEL. However, OSHA believes that an adequate sampling and analytical method exists when such methods are fully described in the open literature, or when otherwise readily available.

The Public Hearing did not provide much additional information regarding sampling and analytical methods for those substances identified as having inadequate sampling and analytical methods. However, a method was identified for substilisins and was entered into the docket (Ex. 8–70). Included in Table IV–E–1 of this section are the substances noted in Table I–F–E of the Proposal (53 FR 20978) and two additional substances (oxygen difluoride and phenylphosphine) which were inadvertently omitted from the listing of substances with inadequate sampling or analytical methods along with one substance, cyanamide, which was erroneously listed as having no method.

OSHA has also considered the concerns identified regarding the need for a more extensively tested analytical method (Ex. 3–960; Ex. 8–47) for enforcement purposes. OSHA believes that enforcement can be initiated without such detailed methods. The OSHA docket includes: (1) Reference to a fully developed and extensively tested OSHA or NIOSH sampling and analytical procedure or, (2) a description of an OSHA in-house sampling and analytical method for all but the seven substances listed in Table IV–E–1. OSHA therefore believes there will be no problems with enforcement of the PELs for all but these seven substances. This is consistent with conclusions of NIOSH regarding implementation (Ex. 8–47). Since development of sampling and analytical procedures is a dynamic, rapidly progressing technology, OSHA also believes it is appropriate to adopt PELs for the seven substances (based on the Proposal and the Public Hearing record), but stay enforcement of these PELs until adequate sampling and analytical methods are available. At such time, OSHA will publish in the **Federal Register** its determination that such methods exist (together with a copy of the method), and indicate the proposed effective date for enforcement of the PEL for the substance in question.

As time, resources and priorities permit, OSHA will attempt to initiate a program, in conjunction with NIOSH, to develop more extensively tested sampling and analytical methods for those substances where only in-house methods are noted in the Proposal.

OSHA further considered the three alternative approaches described in the Proposal regarding interim procedures for handling those substances requiring special attention (53 FR 20978–20979). OSHA encouraged public comment on this subject by including a question (number 14) in the Proposal. The responses to this question are detailed in Section V of this preamble and indicate the desireability of implementing new PELs promptly, even if they represent only interim values.

Therefore, OSHA is proceeding to promulgate limits for all of the substances included in the Proposal where statutory requirements are met, while also indentifying some substances which appear appropriate for future consideration.

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Table IV-E-1

Substances with Inadequate Analytical or Sampling Methods

1. Aluminum alkyls

2. Ethylidene norbornene

3. Hexafluoracetone

4. Mercury (alkyl compounds)

5. Oxygen Difluoride

6. Phenylphosphine

7. Sulfur pentafluoride

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F. Construction, Maritime and Agriculture Segments

Currently the exposure limits which apply to construction are the ACGIH Threshold Limit Values of Air Contaminants for 1970 and certain substance specific section 6(b) standards. See 29 CFR 1926.55, 58 and 29 CFR 1910.19. OSHA is required to consult with the Advisory Committee on Construction Safety and Health prior to proposing new standards that have a major impact on construction. See 29 CFR 1911.10(a). OSHA is in the process of formally consulting with the Construction Advisory Committee. After receiving their recommendations and studying feasibility issues for construction, OSHA intends to propose amendments covering exposures to toxic substances in construction reflecting the facts in this final preamble and standard, and the views of the Construction Advisory Committee.

Parts 1916, 1917 and 1918 of 29 CFR cover, respectively, employment in shipyards, marine terminals and longshoring. Part 1916 for shipyards references the 1970 TLVs, of the ACGIH. See 29 CFR 1915.5 and 1915.12(b)(3). Part 1917 for marine terminals references the current Z-Tables. See 29 CFR 1917.2(p), and 1917.23. Part 1918 for longshoring refers to "dangerous gaseous contaminants not immediately dangerous to life" and "heavy concentrations of dusts." See 29 CFR 1918.93 (e) and (f). Certain substance specific section 6(b) standards also cover these industries. See 29 CFR 1910.19.

OSHA, as part of the rulemaking covering construction and after studying feasibility for the maritime sectors, intends to consider applying the final standard to the maritime sectors.

Subpart Z of 29 CFR Part 1910, and the included Z-Tables specifically do not apply to Agriculture. See 29 CFR 1928.21(b). In addition, many of the chemicals which affect agriculture are pesticides regulated by the EPA over which OSHA may not have jurisdiction, pursuant to section 4(b)(1) of the OSHA Act. In the future OSHA will consider, based on relevance, priorities and administrative resources, whether or not it is appropriate to consider coverage for agriculture.

G. Federalism

This final regulation has been reviewed in accordance with Executive Order 12612 (52 FR 41685; October 30, 1987) regarding Federalism. Executive Order 12612 requires that agencies, to the extent possible, refrain from limiting state policy options, consult with states prior to taking any actions that would restrict state policy options, and take such actions only when there is clear constitutional authority and the presence of a problem of national scope. The Executive Order provides for preemption of state law only if there is a clear Congressional intent for the Agency to do so. Any such preemption is to be limited to the extent possible.

During the development of this rule, OSHA has, to the extent possible, refrained from limiting state policy options by developing a rule that permits flexibility on the part of the States through the use of performance language. OSHA also consulted with the States during the public comment and hearing period announced in the notice of proposed rulemaking for this rule. OSHA specifically invited Stephen Cant of the State of Washington to testify about the state's experience in a similar rulemaking. OSH will continue to work with the States that have occupational safety and health plans approved under section 18 of the OSH Act to encourage those states to develop their own policies to achieve program objectives and will continue to work with appropriate state officials as they present their state standards for approval.

Section 18 of the Occupational Safety and Health Act (OSH Act), permits any state to develop its own independent state occupational safety and health program that provides, among other things, worker protection "at least as effective as" that protection provided under the Federal program.

With respect to section 4 of Executive Order 12612, section 18 of the OSH Act also expresses Congress' clear intent to preempt state laws relating to issues with respect to which Federal OSHA has promulgated occupational safety or health standards. Under the OSH Act, a state can avoid preemption only if it submits, and obtains Federal OSHA approval of, a plan for the development of such standards and their enforcement as mentioned above. Occupational safety and health standards developed by such Approved Plan States must, among other things, be as least as effective in providing safe and healthful employment and places of employment as the Federal standards.

Under the OSH Act, if a state develops its own OSHA approved state program, it could include additional requirements in its standards. Moreover, the performance nature of this final rule, of and by itself, allows for flexibility by states to provide at least as much health protection, consonant with the conditions in each state.

In summary, there is a clear national problem, identified by Congress, related to occupational safety and health. While the individual states, if all acted collectively, might be able to deal with the health problems involved, most have elected not to do so in the seventeen years since the enactment of the OSH Act. However, some states such as Washington have taken action. Those states which have elected to participate under section 18 of the OSH Act, would not be preempted by this final regulation and would be able to address special, local conditions within the framework provided by this standard while ensuring that their standards are at least as effective as the Federal standard. State comments were invited on the proposal and those that were submitted to the record were fully considered prior to promulgation of this Final Rule.

The agency certifies that this document has been assessed in light of the principles, criteria, and requirements stated in sections 2 through 5 of Executive Order 12621. There are no provisions of this rulemaking that are inconsistent with the principles, criteria and requirements stated in sections 2 through 5 of Executive Order 12621. States which have approved state occupational safety and health plans may incur additional costs associated with standards development and enforcement as a result of this rulemaking. Funding for these approved state plan programs is available from OSHA under section 18 of the OSH Act. This rulemaking would not change the State's ability to discharge traditional State governmental functions or other aspects of State sovereignty.

H. Glossary

The following terms and acronyms appear in the standard and the preamble supporting it. This glossary is provided as a convenience to the reader.

ACGIH—The American Conference Governmental Industrial Hygienists is a professional society devoted to the development of administrative and technical aspects of worker health protection. Membership is limited to professional personnel in governmental agencies or educational institutions engaged in occupational safety and health programs. The ACGIH issues guidelines and recommendations in the form of Threshold Limit Values (TLVs®) which are published annually.

CAS—The Chemical Abstracts Service (CAS) Registry Number is a numeric designation assigned by the American Chemical Society's Chemical Abstracts Service which uniquely identifies a specific chemical compound.

This entry allows one to conclusively identify a substance regardless of the name or naming system used.

CHRIS—The Chemical Hazards Response Information System was developed by the U.S. Coast Guard in cooperation with the National Academy of Sciences to provide information on the handling and disposal of toxic substances. CHRIS consists primarily of the Hazardous Chemical Data Manual which contains chemical, physical and health hazard data on approximately 600 hazardous chemicals and substances; and a Hazard Assessment Computer System in an extensive data base of the information contained in the Hazardous Chemical Data Manual.

HSDB—The Hazardous Substances Data Bank, a part of the National Library of Medicine System, will soon be available on OSHA's Computerized Information System (OCIS). This data bank, currently available through TOXNET, contains health and safety profiles for over 4100 chemicals. It includes 144 data elements in 10 categories including use information, substance identification, animal and human toxicity, environmental fate, standards, personal protective equipment, fire, physical and chemical properties.

IARC—The International Agency for Research on Cancer (IARC) is a research organization authorized by the World Health Organization in 1965. IARC's mission is to study the causes of cancer in the human environment. IARC has published (and continues to update) a series of monographs on a substantial number of toxic chemicals and substance in which the carcinogenic risk of these chemicals is evaluated.

ILO—The International Labour Organization (ILO) is a specialized agency associated with the United Nations. Established in 1919 as part of the Versailles Peace Treaty, the ILO serves to band together governments, employers, and workers of 145 nations in an international effort to improve overall working conditions and to protect the life and health of workers.

IMIS—The Integrated Management Information System (IMIS) is a data base developed by OSHA in 1979 with sampling information on more than 100,000 substances. The IMIS contains exposure measurements obtained by OSHA compliance officers during thousands of health inspections; it is the most extensive data base of its kind.

Material—The term "material" is used in the original standard whereas "substance" is used in the revision. The meaning is the same.

MSDS—The Material Safety Data Sheet (MSDS) is a compilation of data and information on individual hazardous chemicals produced by the manufacturers and importers of that chemical, as required by OSHA's Hazard Communication Standard, 29 CFR 1910.1200. An MSDS contains data on chemical identification, current exposure limits, chemical reactivity, fire and explosion limits, and information on health hazards and emergency procedures, spill, leak, and disposal procedures, and any needed special protection or precautions.

NIOSH—The National Institute for Occupational Safety and Health (NIOSH) was created by the Occupational Safety and Health Act of 1970. NIOSH is part of the Centers for Disease Control under the Department of Health and Human Services. Its mandate includes conducting research in developing criteria and/or recommendations to be used in setting occupational exposure standards, identifying and evaluating workplace hazards, measurement techniques, and control technologies, and providing professional education as well as health and safety information.

NOES—The National Occupational Exposure Survey (NOES) is a data base completed in 1982 by NIOSH. NOES is the successor to the first such data base, completed by NIOSH in 1974, and known as the National Occupational Hazard Survey (NOHS). The NOES data base contains a sample of the number of persons exposed by substance and industry from approximately 4500 businesses in 98 geographic areas in the U.S. These surveys provide national estimates of potential exposure to workplace hazards, by industry and occupational group.

OCIS—The OSHA Computerized Information System is a comprehensive data base that contains information and data on standards interpretation, chemical information, hazardous waste activity, 5(a)(1) citations, a health hazard evaluation index, training materials, and other information compiled by OSHA on subjects related to occupational safety and health.

OSHA HS Number—A Health Standard (HS) number is a 4-digit code assigned, for ease in reference, to each of the hazardous substances or chemicals considered for change of PEL in this rulemaking.

PEL—Permissible Exposure Limits (PELs) are limits developed by OSHA to indicate the maximum airborne concentration of a contaminant to which an employee may be exposed over the duration specified by the type of PEL assigned to that contaminant.

Proposal—Refers to the June 7, 1988, Notice of Proposed Rulemaking (NPRM), Air Contaminants.

REL—Recommended Exposure Limits (RELs) are issued by NIOSH to aid in controlling hazards in the workplace. These limits are generally expressed as 8—or 10—hour TWAs for a 40-hour workweek and/or ceiling levels with time limits ranging from instantaneous to 120 minutes. RELs are published in a variety of NIOSH documents.

RTECS—The Registry of Toxic Effects of Chemical Substances (RTECS) is a data base that lists an identification number, synonyms, Department of Transportation (DOT) hazard label information, EPA Toxic Substances Control Act (TSCA) information, OSHA and Mine Safety and Health Administration (MSHA) air exposure limits, and animal and human toxicologic data.

Substance—The term "substance" is used in the revised standard whereas "material" is used in the original. The meaning is the same.

TLV^R—The Threshold Limit Value (TLV^R) is a registered trademark for an exposure limit developed by the American Conference of Governmental Industrial Hygienists (ACGIH). A listing of TLVs may be found in the ACGIH's "Documentation of the Threshold Limit Values and Biological Exposure Indices for 1988–1989." TLVs may be stated as a time-weighted average (TLV^R–TWA), a Short-Term Exposure Limit (TLV^R–STEL), or a Threshold Limit Value Ceiling (TLV^R–C).

TSCA—The Toxic Substances Control Act (TSCA), administered by the Environmental Protection Agency (EPA), was passed by Congress to protect human health and the environment by requiring testing and necessary use restrictions to regulate the commerce of certain chemical substances.

WHO—The World Health Organization (WHO) is part of the United Nations. WHO's programs in occupational health include development of an occupational health information system, criteria for early detection of health impairment, and the development of internationally recommended health-based permissible exposure limits for occupational exposure to toxic substances.

I. Clearance of Information Collection Requirements

On March 31, 1983, the Office of Management and Budget (OMB) published a new 5 CFR Part 1320, implementing the information collection provisions of the Paperwork Reduction Act of 1980, 44 U.S.C. 3501 *et seq.* (48 FR

13666). Part 1320, which became effective on April 30, 1983, sets forth procedures for agencies to follow in obtaining OMB clearance not later than the date of publication of the proposal in the **Federal Register** for collection of information requirements contained in proposed rules. It also requires agencies to include a statement in the notice of proposed rulemaking indicating that such information requirements have been submitted to OMB for review under section 3504(h) of the Paperwork Reduction Act. The PELs update standard will create no additional recordkeeping requirements.

J. Maintaining Z-Tables Current

This Rulemaking was designed to overcome an 18 year gap between the 1971 adoption of the PELs in the 29 CFR 1910.1000 Z-Tables and the present. During this time period there were extensive changes in toxicology and health effects information, and the application of available control technologies. This resulted in the existing Z-Tables in 29 CFR 1910.1000 being out of date and incomplete. The new information demonstrated that some workers are not protected from exposure levels which represent a significant risk of material impairment of health.

Because of the magnitude of changes during this 18-year period, this Rulemaking has involved a large number of substances and interested parties. Consequently, OSHA designed this Rulemaking to use as a starting point the well established TLV's and REL's. The record during the public hearing was then used to determine the appropriate final PEL, consistent with OSHA's statutory obligations.

As a follow-up to this Rulemaking OSHA plans to develop and implement a methodology which will permit OSHA to keep the PELs current as time goes on. This point was raised by several commentors during the public hearing (ORC, TR 3–266; NAM, TR 3–338; G.E., TR 9–172), without specific detail regarding the best procedures OSHA should follow. OSHA agrees with these suggestions, and from the start of this Rulemaking has been considering how this objective can be attained. The program would be designed to be applicable to all sectors (general industry, maritime, construction, and agriculture). The methodology adopted will be designed to avoid duplicative efforts or gaps in coverage, to be protective of workers, and to be cost effective.

Updating the PELs in the Z-Tables on a regular basis insures that the latest evidence is considered as it becomes available, and that appropriate action to either reduce or increase permissible levels is initiated promptly. Commenters to this rulemaking cited several substances for which they believed significant new information would become available in the near future. OSHA intends to evaluate such evidence and, if appropriate, to initiate prompt action to revise the Z-Tables. OSHA may take such action on a single substance or on several substances. This continual, dynamic process of evaluation and revision, consistent with the priorities of the Agency will insure that the Z-Tables reflect the latest scientific evidence on the risks posed by the listed substances.

OSHA will use a variety of sources to determine which substances will be considered for updating. Information developed as a result of NIOSH studies and the annaul updating of the TLVs will, of course, be used by OSHA.

At this time OSHA has not developed the details of the up-date process. It is anticipated that it will be planned with regularly scheduled update intervals, somewhere between 2 and 5 years. The process is expected to follow full 6(b) rulemaking procedures.

V. Summary of Commenters' Responses to NPRM Questions

In the preamble to the proposed rule (53 FR 20961), OSHA asked interested parties and the public for information related to 27 questions. These questions addressed a large number of issues, such as the scope of the rulemaking, the appropriateness of the proposed exposure limits, the availability of feasibility information for particular substances, the definition of material impairment of health, and the availability of engineering and cost data relevant to this rulemaking.

Many commenters (see, for example, Exs. 3–593, 3–660, 3–741, 3–744, 3–891, 3–896, 3–1095, 3–1161, 8–16, 8–19, and 8–47) submitted responses to the questions raised by OSHA. Of these commenters, most chose only to answer selected questions, while NIOSH provided answers to all of the issues raised. The responses of commenters to each of the questions specifically asked in the preamble to the proposal are summarized below. In addition, many participants addressed some of the issues raised by these questions in their comments on the health effects or feasibility of individual substances. These comments are addressed in connection with the preamble discussion for each substance. More detailed responses to some of these comments are discussed in other sections of the preamble, in connection with the health and feasibility discussions for specific substances.

1. Are substances included which should be excluded from this rulemaking?

Several commenters (See, for example, Exs. 3–593, 3–891, 3–896, 3–1095, and 8–47) responded to this question. There was widespread support for OSHA's selection of substances for regulation. For example, the Dow Chemical Company (Ex. 3–741) "supports the PEL project in changing outdated PELs." Several commenters, however, requested that certain aspects of the proposal be modified.

For example, Susan Kernus, Manager of Government Affairs for the Synthetic Organic Chemical Manufacturers Association (SOCMA), stated:

> We do not believe any substances included in the proposal should be excluded, but we strongly object to the adoption of [NIOSH] REL's. We recommend that ACGIH TLV's be adopted for these substances * * *. (Ex. 3–891, p. 6).

George Talley and Michael Garcia, industrial hygienists with the Los Alamos National Laboratory, argued that recommended exposure limits (RELs) with "insufficient or old * * * data" should be deleted, as also should substances for which sampling and analytical methods are unavailable (Ex. 3–1095).

Commenting for the Chevron Corporation, Stanley Dryden stated:

> We support the adoption of the ACGIH Short-Term Exposure Limits (STELs) that were established on the basis of careful review of documented short-term health effects. However, for several substances the proposed STELs are *not* adequately justified * * *. We recommend that the proposed STELs be removed from this rulemaking *except* where there is clear evidence that the STEL is required to protect against a material impairment of health (Ex. 3–896, p. 3).

U.V. Henderson, Jr., Director of Environmental Affairs for the Texaco Company, endorsed OSHA's choice of regulatory candidates by stating: "No substances are included in the listings which should be excluded from rulemaking" (Ex. 3–593). In response to this question, NIOSH expressed support for the inclusion of the proposed substances but urged OSHA to take further action "immediately upon completion of this rulemaking * * * to establish PELs for all substances that are excluded from this rulemaking" and for which NIOSH has made a recommendation to OSHA (Ex. 8–47, p. 17). NIOSH stated that OSHA should initiate "consolidated rulemaking * * * to adopt all NIOSH RELs pending [the

initiation of] chemical-specific Section 6(b) rulemaking * * *." (Ex. 8–47, p. 17).

The support voiced by these commenters is gratifying to OSHA and increases the Agency's confidence that the substances selected for this generic rulemaking are both necessary and appropriate. The ancillary issues raised by these commenters, such as the appropriate basis for short-term exposure limits, the use of ACGIH TLVs in lieu of NIOSH RELs, and the initiation of other rulemakings in the future, are addressed in other sections of this preamble, e.g., Section VI.C.17 (STELs), Section VI on the Agency's methods of selecting exposure limits, etc. Readers are referred to these sections for a detailed discussion of the record evidence on these topics.

2. Is additional health and feasibility documentation available relative to the proposed PELs beyond that described in the preamble?

Several participants (see, for example, Exs. 3–741, 3–891, 3–1043, and 8–47) specifically responded to this preamble question. (There were of course many responses directed to health effects or feasibility issues on specific substances; these are presented in the discussions for individual substances (see Section VI).) The Synthetic and Organic Chemical Manufacturers Association (SOCMA) reported that it does not possess additional data but has requested its members to submit to the docket any information available to them (Ex. 3–891). Richard Olsen, project manager for the Dow Chemical Company, noted his company's support for the PEL project but stated that Dow was limiting its submission of additional data to certain substances, such as styrene and those chemicals for which OSHA proposed the adoption of NIOSH RELs. According to Mr. Olsen, "Feasibility documentation is not readily available in the time allowed to prepare this submission because it resides mainly with our customers." However, Dow did ask several of its customers to submit feasibility information to the record (Ex. 3–741).

The American Federation of State, County, and Municipal Employees (AFSCME) stated that OSHA had not evaluated 14 other lists developed by professional organizations and foreign bodies" (Ex. 3–1043). AFSCME is of the opinion that OSHA should have started its analysis with the most protective standard.

NIOSH commented that additional health and feasibility data pertaining to these substances and affected sectors are available and urged OSHA to consider criteria documents, health hazard evaluations, current intelligence bulletins and other NIOSH publications when developing the final rule. NIOSH also noted that several foreign governments (e.g., Germany, Sweden, West Germany), organizations (e.g., the American Industrial Hygiene Association, the International Labour Organisation), and research groups (e.g., the National Toxicology Program, the International Agency for Research on Cancer) have provided extensive toxicity information (Ex. 8–47, pp. 17–18).

In the proposal, OSHA relied extensively on the health effects information made available by these and other organizations and individuals; the reference list for the health effects section of the proposal alone included more than 1,000 citations to the toxicological literature. In the development of the final rule, OSHA has gone beyond this initial list to include hundreds of additional citations and has additionally performed a thorough analysis of all data submitted to the rulemaking docket.

OSHA appreciates NIOSH's submission of data to the record and the efforts of SOCMA and the Dow Chemical Company to obtain feasibility data from their members and customers, repectively. Information submitted by NIOSH and these individuals is discussed in other portions of this preamble in connection with the specific feasibility concerns and health effects issues raised by these commenters.

3. Are substances included in this rulemaking used in industries other than those described in the preamble? and

4. Are substances included in this rulemaking used for purposes other than those described in the preamble?

NIOSH (Ex. 8–47) responded to these two questions together by noting that it has submitted to the record a printout of the complete NIOSHTIC data base file. This information often contains industry-specific data on exposures, operations, and controls, and OSHA has analyzed this information as part of this rulemaking. No other commenters provided responses specifically to these questions, and OSHA therefore believes that the proposal and its appendices accurately identified both the major chemical-using industries by Standard Industrial Classification and the major uses applicable to substances included in this rulemaking.

5. Do alternative unpublished exposure guidelines exist, such as those used in private workplaces, which may be suitable for general usage?

Several respondents (see, for example, Exs. 3–741, 3–1095, and 8–47) submitted information about internal corporate guidelines. George M. Talley and Michael Garcia, with the Los Alamos National Laboratory, reported that several industries have such limits (Ex. 3–1095), and the Dow Chemical Company (Ex. 3–741) acknowledged that it has developed internal limits for 250 chemicals used in its plants. However, Dow does not believe that these unpublished exposure guidelines are appropriate "for general usage" because they were developed specifically for Dow's operations and facilities. Dow reports that these limits have not been "appropriately peer reviewed for operations outside our company" (Ex. 3–741, p. 21).

NIOSH commented (Ex. 8–47) that many of the private workplaces it has surveyed have internal exposure guidelines and that, in many cases, these limits are considerably lower than OSHA's existing limits. NIOSH noted that exposure guidelines for two of the substances included in this rulemaking, soluble and insoluble uranium, have been established by the International Commission on Radiation Protection and the National Commission on Radiation Protection.

Several corporations, for example, Rohm and Haas and the Dow Chemical Company, submitted some or all of their internal exposure guidelines to the docket, and OSHA has reviewed these submittals carefully. For the reasons discussed in Section IV.D of this preamble, however, OSHA determined that the ACGIH and NIOSH exposure limits were the most appropriate data bases for OSHA to use as starting points for the rulemaking. In the overwhelming majority of cases, the record has supported this decision, and the limits included in this final rule are consistent with those proposed. In a few instances, OSHA has determined, based on evidence submitted to the record, that another limit is more appropriate; the record evidence in these cases is discussed in detail in the chemical-specific discussions in Section VI.

6. Is there information regarding laboratory analytical procedures which may be used in lieu of those suggested by OSHA to determine exposure to air contaminants?

Several commenters responded to this question (Exs. 3–1095, 8–19, and 8–47). Representatives of the Los Alamos National Laboratory gave OSHA specific information on an improved method for the analysis of methylene dianiline, a substance that is not included in this rulemaking because a section 6(b) rule is being developed for it at the present time (Ex. 3–1095). NIOSH noted several corrections to the NIOSH Analytical Methods published in

Appendix A of the proposal; these corrections have been incorporated into Appendix A of this final rule. In addition, NIOSH stated that, in Appendix A of the proposal, several existing NIOSH analytical methods "have been extended to compounds for which the suggested method has not been verified" (Ex. 8–47, p. 22). In several such instances, according to NIOSH, the compound to which the method has been extended differs from the compound for which the method was originally developed.

According to NIOSH, the analytical methods for the following substances would benefit from additional analysis:

1-3-dichloropropene
2-hydroxypropyl acrylate
propargyl alcohol
isooctyl alcohol
trichloroacetic acid
dichloroacetylene
chlorodifluoromethane
chloropentafluoroethane
o-chlorostyrene
o-chlorotoluene
cyclopentane
hexane isomers
hydrogenated terphenyls
N-isopropylaniline
methyl silicate
nonane
p-toluidine
m-toluidine.

Based on its experience, OSHA concludes that there are adequate methods for the sampling and analysis of the substances. As noted in this preamble, additional work is planned regarding further evaluation of these methods. In addition, OSHA's experience shows that the promulgation of new permissible exposure limits has often encouraged the development of appropriate analytical and sampling methods. In 1971, at the time of the adoption of the start-up standards, few sampling and analytical methods had been developed, and NIOSH was charged with the responsibility of developing and validating such methods. The success of this approach is evidenced by the fact that, at the time of the June proposal, only seven substances of the 428 included in the rulemaking were identified as lacking any sampling and analytical methods (53 FR 20978). (In the course of this rulemaking methods for two of these seven, the subtilisins and cyanamide, were submitted to OSHA.) Another example of the incentive to develop methods provided by the setting of new limits can be seen in the case of OSHA's recent ethylene oxide (EtO) standard. At the time of the promulgation of the final rule, in June of 1984, no accurate and easy-to-use method was available to measure short-term EtO exposures; however, by 1986, OSHA's Salt Lake City Laboratory had developed a simple and efficient method using hydrogen-bromine-impregnated charcoal tubes. By 1987, OSHA's research and development effort had led to the development of a commercial product that is now widely available: small, easy-to-use, and inexpensive charcoal tubes for taking employee-breathing-zone measurements of EtO STEL exposures. In addition, several manufacturers have developed passive dosimeters for EtO STEL monitoring. OSHA believes that this same course of research and development, which illustrates the successful working of the market, will occur for the very few substances currently without analytical methods in this rulemaking.

7. Are the proposed exposure limits for each substance appropriate?

OSHA received responses to this question from many rulemaking participants (see, for example, Exs. 3–593, 3–741, 3–891, 3–896, 8–16, 8–19, and 8–47). (In addition, many commenters addressed the appropriateness of the PELs for specific substances; these commenters are addressed in Section VI.C of the preamble.) There was substantial support among these commenters for adoption of the proposed limits that were based on the ACGIH TLVs (Exs. 3–593, 3–891, 3–741, and 3–1095). For example, the Texaco Company stated, "The TLVs are current, well documented, and widely accepted by the industrial hygiene community" (Ex. 3–593). Several respondents felt that the 17 proposed limits that were based on NIOSH Recommended Exposure Limits (RELs) were not appropriate (Ex. 3–593, 3–891, 3–741, and 3–1095). Typical of the reasoning of these commenters was the statement of U.V. Henderson, Jr., Director of Environmental Affairs for the Texaco Corporation:

> Only the ACGIH TLVs should be used as the best available source for OSHA to update * * * [its] exposure standards. The TLVs are current, well documented, and widely accepted by the industrial hygiene community. Many State-approved OSHA programs incorporate the TLVs as their basis for regulations. The NIOSH recommended limits are oftentimes outdated and conservative. Furthermore, feasibility and cost-effectiveness are not always addressed by NIOSH (Ex. 3–593, Attachment, p. 1).

The American Industrial Hygiene Association (AIHA), on the other hand, was entirely in favor of OSHA's used of either an ACGIH or a NIOSH limit, as the case required:

> AIHA supports the adoption by OSHA of NIOSH REL values as PELs on a case-by-case basis where such values are supported by the scientific evidence and are feasible from the standpoint of implementation.

NIOSH expressed strong support for this rulemaking in general but submitted specific comments on a number of substances that it believes should have different limits from those proposed (Ex. 8–47). However, NIOSH stated at the hearing that, for substances lacking NIOSH RELs, the use of the ACGIH's TLVs as a starting point is appropriate (Tr. pp. 3–130—3–131). NIOSH's substance-specific comments are addressed in connection with the preamble discussion of these substances (see Section VI).

OSHA is gratified by the degree of support for the proposal expressed by these and other rulemaking commenters. The Agency agrees with the AIHA that the appropriate way to establish exposure limits is on a case-by-case basis, considering health effects and feasibility concurrently. This is the methodology used by OSHA in the proposal, and the final rule applies these same principles to the setting of limits for individual substances.

8. Is additional information available for those substances for which ACGIH proposed a higher TLV which might affect OSHA's decision that such a change was not justified?

Only NIOSH (Ex. 8–47) responded specifically to this preamble question. NIOSH expessed the opinion that a comprehensive section 6(b) rulemaking is required if OSHA is considering raising, rather than lowering, a particular exposure limit. OSHA believes that the issue is not so much the *type* of rulemaking, i.e., generic vs. substance-specific, as the significance of the risk involved. For example, when raising a limit, the Agency must be able to show that "exposed workers will not be placed at increased risk for the health effects at issue even after the limit in question has been raised or revoked * * *" (53 FR 21213). The guiding principles were first enunciated by OSHA when the Agency revoked the cotton dust limit for facilities in specific nontextile industries (50 FR 51120 *et seq.*), and this issue was subsequently discussed in the present rulemaking in the proposal section pertaining to substances for which the ACGIH TLVs are higher than OSHA's existing limits (53 FR 21213). OSHA continues to believe that those principles, rather than the type of rulemaking, constitute the test the Agency must meet when a limit is proposed for raising or revocation.

9. Should implementation dates for some substances be delayed because of sampling/analytical limitations or short term feasibility impact considerations?

Several commenters (Exs. 3–823, 3–891, 3–905, 3–960, 3–1095, 8–16, 3–741, and 3–891) questioned OSHA's promulgation of PELS for substances for which available sampling/analytical methods are not adequate. The Los Alamos National Laboratory (Ex. 3–1095) commented that OSHA "should delay implementation dates for substances that do not have adequate sampling and analytical procedures until such methods are available and validated. It is unreasonable to expect compliance when the chemicals cannot be quantified." NIOSH (Ex. 8–47) commented that some substances have no sampling and analytical methods and that methods for others have not been validated by OSHA or NIOSH.

Appendix A to the proposed rule provided data on the status of sampling and analytical methods for all of the substances included in this rulemaking. For a number of substances, in-house sampling and analysis methods are available; copies of these methods have been supplied by OSHA to any party requesting them, and they are also available in the docket for this rulemaking. No commenter has suggested that any of these in-house methods is inadequate; however, commenters have made general comments on interlaboratory testing and exchange programs and their benefits in terms of method standardization. NIOSH has recommended additional evaluation of the sampling and analytical procedures for several substances.

OSHA has considerable expertise and experience in developing sampling and analytical methods. The Agency has determined that these in-house methods are adequate for enforcement purposes. (Any employer or laboratory wishing a copy of the entire set of methods can purchase them from the ACGIH. Copies of any individual method may be obtained by calling the OSHA Salt Lake City Laboratory, (801) 524–5287.) There have been no objections to any sampling and analytical method for any specific substance. Consequently, OSHA will enforce all of the exposure limits in the final rule except in the seven cases where no sampling and analytical method is known to OSHA.

OSHA identifed seven substances in Table 1–F–E of the proposal (53 FR 20978) as not having adequate sampling/analysis methods (aluminum alkyls, cyanamide, ethylidene norbornene, hexafluoroacetone, mercury [alkyl compounds], subtilisins, and sulfur pentafluoride). (In the course of this rulemaking, commenters submitted methods for two of the substances listed in the proposal as having no method: Cyanamide and the subtilisins. However, commenters also identifed two other substances, phenylphosphine and oxygen difluoride, as lacking methods.) In the final rule, OSHA is promulgating permissible exposure limits for aluminum alkyls, ethylidene norbornene, hexafluoroacetone, mercury [alkyl compounds], oxygen difluoride, phenylphosphine, and sulfur pentafluoride. However, the Agency is staying the enforcement of these limits until an acceptable sampling and analytical technology is devised. When such techniques are developed, OSHA will publish a **Federal Register** notice indicating that fact and setting forth the date on which enforcement will commence.

10. Is there additional information relative to the OSHA plans to adopt some recommended 10-hour TWA RELs as an 8-hour TWA PEL?

OSHA received few comments in response to this question (see, for example, Exs. 3–1095, 3–623, and 8–47).

Representatives of the Los Alamos National Laboratory supported OSHA's use of NIOSH 10-hour limits as 8-hour TWAs:

> [W]e support these plans * * * [because this] is a conservative approach and appropriate (Ex. 3–1095).

George Lathrop of Kerr-McGee Corporation (Ex. 3–623) observed:

> [T]he NIOSH Recommended Exposure Levels (REL's) are based upon 10-hour work shifts in a 40-hour work week. The OSHA PEL's, as well as the ACGH recommended TLV's, are based upon 8-hour work shifts in a 40-hour work week. OSHA preliminarily concludes that the NIOSH REL is equivalent to the OSHA PEL's definition. These two values are equivalent only if their interpretation is based on the length of the work (i.e., 40 hours). If the interpretation of the OSHA PEL's and the NIOSH REL is based on the length of the work shift (8 or 10 hours, respectively), then these values are not equivalent. If OSHA adopts a NIOSH REL for a particular substance, a notation should exist which identifies the exposure level as based on a 10-hour work shift or the level should be adjusted to represent an 8-hour work shift (Ex. 3–623, p. 3).

NIOSH (Ex. 8–47) provided a detailed response and explained that NIOSH 10-hour RELs are intended to apply to either 8-hour or 10-hour days in a 40-hour workweek. NIOSH explained that the 10-hour REL originated during the energy crisis of the 1970s. When many employers began to use 10-hour/4-day work schedules to conserve energy (Ex. 8–47, p. 25). Thus, the 40-hour workweek rather than the length of a workday is, in NIOSH's view, the important time element in the (concentration) X (time) equation: Any given REL can be applied to either four 10-hour days or five 8-hour days without being exceeded. NIOSH supports OSHA's proposal to apply 10-hour NIOSH RELs to 8-hour days by stating:

> The action proposed by OSHA in this rulemaking relative to these RELs is consistent with that original intent (Ex. 8–47, p. 26).

In this final rule. OSHA is therefore applying values derived from NIOSH RELs as 8-hour TWA PELs.

11. Does the most current scientific information generally support acceptance of the hypothesis that all C_{5-8} alkanes are not equally toxic because a metabolite of n-hexane exhibits unique neurotoxic properties?

The C_{5-8} alkanes include pentane, n-hexane, hexane isomers, n-heptane, octane, and the refined petroleum solvents, namely rubber solvent (naphtha), Stoddard solvent, and VM & P naptha. There is some disagreement regarding the question of equal toxicity for all C_{5-8} alkanes, which impacts on the determination of appropriate PELs on the basis of neuropathic effects resulting from exposure to these substances.

n-Hexane has been shown to produce distal axonopathy in both experimental animals and humans; it is metabolized to 2,5-hexanedione (2,5-HD), which is thought to be the agent that produces peripheral neuropathy after exposure to n-hexane (Schaumburg, and Spencer, Thomas 1983/Ex. 1–228). The ACGIH arrived at a TLV of 50 ppm for n-hexane, based primarily on studies by Miyagaki (1967/Ex. 1–198) and Inoue, Takeuchi. Takeuchi et al. (1970/Ex. 1–75) showing peripheral neuropathies at exposure levels as low 210 ppm. A number of studies have shown a consistent relationship between exposure levels of 500 to 2000 ppm n-hexane and the development of characteristic peripheral neuropathies (Yamamura 1969/Ex. 1–42; Yamada 1967/Ex. 1–192). Neuropathic effects have also been shown to occur at level between 210 and 500 ppm n-hexane (Takeuchi, Maluchi, and Takagi 1975/Ex. 1–217).

The NIOSH (1977a/Ex. 1–223) RELs for the C_{5-8} alkanes are based on the belief that polyneuropathy may be caused by other alkanes (or mixtures of alkanes) and their isomers. NIOSH (1977a/Ex. 1–223) relied heavily on two studies by Gaultier, Rancurel, Piva, and Efthymioc (1973/Ex. 1–123) and Truhaut et al. (1973, as cited in ACGIH 1986, p. 305, "n-Hexane"). The report by Gaultier et al. (1973/Ex. 1–123) reported that five workers in a belt-manufacturing shop

developed polyneuropathy as a result of exposure to a solvent that contained 80 percent pentane, 14 percent heptane, and 5 percent hexane. The authors concluded that pentane and heptane, as well as hexane, might also have caused this polyneuritis.

Truhaut et al. (1973, as cited in ACGIH 1986, p. 305, "n-Hexane") exposed Wistar rats to airborne hexane (technical grade) at a concentration of 2000 ppm and to heptane (technical grade) at a concentration of 1500 ppm for five hours/day, five days/week, for one to six months. The analysis of technical grade hexane was: 0.3 percent n-pentane, 25.1 percent 2-methylpentane + cyclopentane, 18.4 percent 3-methylpentane, 45 percent n-hexane, 8 percent methyl cyclopentane, 1.2 percent methyl hexane, and 1.2 percent benzene.

The analysis of technical grade heptane was; 9.8 percent 2-methylhexane, 2,3-dimethyl pentane, and cyclohexane; 16.2 percent 3-methylhexane; 52.4 percent n-heptane; 18.2 percent 2,4-dimethylene, methylcyclohexane, and toluene; 3.3 percent methylheptane; 0.1 percent benzene; and 2.8 percent toluene. The exposed rats developed polyneuropathy, and NIOSH considers this study as evidence indicating that different alkanes cause polyneuropathy.

Since 1977, when NIOSH published its criteria document on alkanes (C_{5-8}), considerable evidence has accumulated that demonstrates that peripheral neuropathies are caused only by n-hexane and gamma-diketone metabolites (O'Donoghue 1985).

The following summaries of publications show that n-hexane, and not the hexane isomers, n-pentane, n-hexane, of octane, is the primary cause of peripheral neuropathy.

1. Peripheral neuropathy comparable to that seen in human cases has been reproduced using rats, cats, monkeys, hens, and pigeons exposed to n-hexane, practical grade hexanes (which contain n-hexane and benzene), or gasoline containing n-hexane (O'Donoghue 1985).

2. Egan et al. (1980) exposed rats for 22 hours per day, for periods up to six months, at 500-ppm concentrations of an n-hexane "free" isomer mixture: no evidence of neurotoxic effects was observed. A second group of rats exposed to 1 ppm of methyl n-butyl ketone, a positive control, developed histological evidence of peripheral neuropathy after four months of continuous exposure.

3. Takeuchi et al. (1980) performed a comparative study on the neurotoxicity of n-pentane, n-hexane, and n-heptane in the rat. Rats were exposed to 3000 ppm of n-pentane, n-hexane, or n-heptane for 12 hours/day for 16 weeks. The experiment showed that n-hexane distributed the conduction velocity of the motor nerve and the mixed nerve and prolonged the distal latency in the rats' tails, but that n-pentane and n-heptane did not. Light- and electron-microscopic examinations showed that the peripheral nerve, the neuromuscular junction, and the muscle fibers of the rats exposed to n-hexane or n-heptane showed no particular changes after 16 weeks of exposure. These results show that n-hexane is far more toxic to the peripheral nerve of the rat than is n-pentane or n-heptane.

4. Frontali et al. (1981) exposed rats to n-hexane or n-heptane for 9 to 10 hours/day, 5 to 6 days/week, for a period of 30 weeks. Animals treated with n-hexane at 5000 ppm for 14 weeks or at 2500 ppm for 30 weeks developed the typical giant axonal degeneration already described by Spencer and Schaumburg (1976) in rats treated continuously with 400 to 600 ppm of n-hexane for seven weeks or more. No such alterations were found in the rats subjected to intermittent respiratory treatments with n-pentane at 3000 ppm for 30 weeks or to n-heptane at 1500 ppm for 30 weeks. Again, this demonstrates the greater neurotoxicity of n-hexane compared with that of its isomers.

5. Bahima et al. (1984) conducted a study on female Wistar rats exposed by inhalation to 2000 ppm n-heptane for 12 weeks. No clinical evidence of neurotoxicity was observed after n-heptane exposure. Urinary metabolites were identified by gas chromatography/mass spectrometry. The n-heptane metabolites were 1-, 2-, 3-, and 4-heptanols, 2- and 3-heptanones, 2,5- and 2,6-heptanediols, 5-hydroxy-2-heptanone, 6-hydroxy-2-heptanone, 6-hydroxy-3-heptanone, 2,5- and 2,6-heptanediones, and gamma-valerolactone. 2,5-Heptanedione, a known neurotoxic agent, was the metabolite found in least amounts in the urine. The authors concluded that the lack of neurotoxicity was due to the small amount of 2,5-heptanedione produced after n-heptane exposure.

6. Olson et al (1986) studied the metabolism of n-octane in Fischer 344 rats. The urinary metabolites of n-octane in rats given n-octane by gavage included 2-octanol, 3-octanol, 5-oxohexanoic acid, and 6-oxoheptanoic acid. n-Octane was not metabolized to a ketone, diketone, or diol derivative. None of the metabolites excreted are known to cause peripheral neuropathy in rats.

7. Spencer and Schaumburg (1985) point out that alkanes normally undergo subterminal carbon oxidation. The likelihood of producing neurotoxic levels of gamma-diketone metabolites from alkanes higher in the series than n-hexane is unlikely. Shorter-chain alkanes (pentane) and hexane isomers free of n-hexane also fail to produce the appropriate metabolite and do not induce neuropathy in experimental animals. The authors further conclude that n-hexane is unique among the alkanes in producing peripheral neuropathy in humans.

8. Recent studies have suggested a mechanism for the structural basis of the neurotoxicity of gamm-diketones. Studies reported by Sayre et al. (1986) and Genter et al. (1987) demonstrate that only those hydrocarbons capable of gamm-diketone and pyrrole formation are potentially neurotoxic. Chronic exposure to gamma-diketones results in the formation of giant neurofilament-containing axonal enlargements.

9. Several commenters (Exs. 3–896, 3–740, and 3–593) were in agreement with the points made in the discussion of this issue, above.

10. NIOSH (1988/Ex. 8–47) continues to support its conclusions as to the neurotoxicity of all of the C_{5-8} alkanes, as discussed in the 1977 criteria document (1977a/Ex. 1–223). NIOSH believes that n-hexane and other C_{5-8} alkanes or related chemicals are ultimately metabolized to a gamma-diketone and thus may have similar neurotoxic properties. Accordingly, in the 1977 criteria document on alkanes (C_{5-8}), NIOSH proposed a REL of 350 mg/m ^{3} as a TWA concentration for up to a 10-hour work shift for the straight and branched-chain aliphatic isomers of pentane, hexane, heptane, and octane (NIOSH 1977a/Ex. 1–223).

OSHA finds NIOSH's argument on this issue unconvincing in light of the consistent results obtained by a number of investigators using a variety of experimental procedures (see item 1 through 9, above). Therefore, the Agency concludes that only n-hexane has been proved to cause peripheral neuropathy at this time and that other alkanes, such as n-pentane, n-heptane, octane, and the hexane isomers, do not appear to cause peripheral neuropathy. Consequently, OSHA's initial assessment of the relative toxicity of the C_{5-8} alkanes (53 *FR* 20998) remains unchanged.

12. OSHA has Proposed to use Exposure Limits From two Well-Established sets of Guidelines as a Source of Values to Update the PELs. Is Information available about alternative sources which OSHA might consider for this purpose?

Several commenters (see, for example, Exs. 3–1095, 8–16, and 8–47) responsed

to this preamble question. Representatives of the Los Alamos National Laboratory (Ex. 3–1095) noted that many industries have voluntary guidelines that might be considered by OSHA if individual companies or trade associations submit them to the docket.

NIOSH (Ex. 8–47) mentioned as excellent sources the 9 data sets referred to by OSHA in the preamble to the proposal (53 FR 20967) and additionally recommended as a potential source the Nordic Expert Group for Documentation of Occupational Exposure Limits. NIOSH stated:

> No single source should be expected to stand alone as a comprehensive list of candidates for regulation. OSHA should construct its own comprehensive list by drawing information from all available sources (Ex. 8–47, p. 28).

The Agency used the ACGIH TLVs and NIOSH RELs as starting points and then carefully reviewed the testimony and comments submitted in the course of this rulemaking. If additional information was needed, the Agency examined additional toxicological sources. After careful review and evaluation of this body of information on any given substance and in conformance with Agency policy and statutory requirements, OSHA then determined the appropriate PEL or PELs for each substance.

The American Industrial Hygiene Association (AIHA) also responded to this question (Ex. 8–16). The AIHA submitted a complete set of that organization's Workplace Environmental Exposure Level (WEEL) Guides, with supporting documentation, to OSHA to consider as PEL replacement values. The AIHA described the process by which the WEEL committee establishes these levels and reported that such factors as production rate, acute toxicity, and extent of the interest expressed by the entire AIHA membership are taken into account when deciding what substances to consider for WEELs (Ex. 8–16).

As noted in the preamble to the proposal (53 FR 20967), OSHA cnsidered nine sets of exposure limits, including the WEELs, when the proposal was being developed. OSHA agrees in general that the WEEL values "constitute a well-established set of guidelines for more than 40 substances" (Ex. 8–16); however, the Agency was not able to use the WEELs as replacement PELs in the present rulemaking because, to date, fewer than 40 WEELs have been developed. OSHA concludes that the reasons identified by OSHA in the proposal (53 FR 20967) for using the ACGIH TLVs and the NIOSH RELs as starting points were appropriate.

13. OSHA has outlined its criteria for identifying special situations. Are alternative criteria available which might be used in lieu of these, or in addition to them?

Several rulemaking participants responded to this question; these comments were similar to those provided in response to questions 6 and 9 on analytic methods. See the responses to these questions for a discussion of this issue.

14. OSHA has outlined three alternative procedures for dealing with substances requiring special attention. Are additional approaches available which might be used in lieu of these, or in addition to them?

Four commenters (Exs. 3–1095, 3–593, 3–891, and 8–47) responded to this question, which referred to three approaches suggested by OSHA as possible ways of treating the substances in this rulemaking that require special attention (53 FR 20978–79). These three alternatives were:

(1) In-depth review of all available data for each substance and the establishment of a PEL at the level indicated by this review;

(2) Adoption of a limit in this rulemaking, to be followed later by separate rulemaking if the data warrant further analysis; or

(3) Retain the existing OSHA limits for special-attention substances and proceed later with follow-up review and possible 6(b) rulemaking.

A large majority of commenters endorsed the second approach for substances identified in the course of the rulemaking as warranting special attention (see, for example, Exs. 3–1095, 3–593, 3–891, and 8–47).

The Synthetic Organic Chemicals Manufacturing Association (SOCMA) had no suggestions for alternatives to the three approaches suggested by OSHA (Ex. 3–891). SOCMA found the second approach to dealing with special situations most appropriate; however, the association urged OSHA not to use NIOSH recommended exposure limits (RELs) as interim values but instead to rely on the ACGIH limits for this purpose (Ex. 3–891). The Texaco Company agreed with SOCMA that NIOSH RELs should not be used as interim PELs (Ex. 3–593), while representatives of the Los Alamos National Laboratory (Ex. 3–1095) believe that limits should not be promulgated for those substances lacking sampling and analytical methods. NIOSH (Ex. 8–47) supported OSHA's suggestion that it might be appropriate to mandate limits for all substances immediately and then follow this generic rulemaking with separate rulemaking, as the evidence dictates. NIOSH believes that in some instances, a full 6(b) rulemaking is required. According to NIOSH:

> NIOSH concurs with OSHA that it is in the best interest of the worker to promptly provide such increased health protection as is indicated by the evidence in the record (Ex. 8–47, p. 32).

OSHA has concluded that this second approach constitutes the best method of protecting the health and well-being of the largest possible number of workers in the shortest possible time frame. Accordingly, the Agency is today promulgating limits for all but a few of the substances for which limits were proposed. Depending on resources, OSHA may consider for additional rulemaking those substances identified in this preamble as warranting further consideration.

15. OSHA has performed feasibility analyses for the following substances, based on limited available information: acetonitrite, carbon disulfide, carbon monoxide, carbon tetrachloride, chloroform, ethylene dichloride, ethylene glycol dinitrate, fibrous glass dust, hydrogen cyanide, isophorone diisocyanate, nitrogen dioxide, nitroglycerin, and trichloro-ethylene. Is further information available which might be used to supplement the present findings regarding the feasibility of achieving these levels in the workplace?

The Synthetic Organic Chemicals Manufacturing Association (SOCMA) (Ex. 3–891), the Dow Chemical Company (Ex. 3–741), the Teepak Corporation (Ex. 8–19), and NIOSH (Ex. 8–47) each responded to this question. The Dow Chemical Company (Ex. 3–741) commented on the difficulty of obtaining feasibility information, especially for "the small business entities which will be most heavily impacted," while SOCMA (Ex. 3–891) opposed the promulgation of NIOSH limits if the feasibility information available was not adequate. NIOSH (Ex. 8–47) submitted feasibility information to OSHA for the substances listed and for acetone, chlorine, styrene, and sulfur dioxide as well. OSHA appreciates NIOSH's submission and is using this information in its feasibility analyses for individual substances (see Section VI).

For the final rule, OSHA went beyond the feasibility analyses presented in the proposal. The Agency incorporated the substantial amount of feasibility data submitted by NIOSH and other submitters. OSHA also reviewed the site visit reports submitted into the record. Based on the entire record, OSHA has

concluded that the Agency has sufficient feasibility data to support the final rule's PELs. These data are analyzed in depth, by industry sector, in Section VII.

16. OSHA has made a preliminary assessment of the proposed rulemaking's impact on large and small establishments. The Act requires OSHA to determine whether a regulation will have a significant impact on a substantial number of small entities, pursuant to the Regulatory Flexibility Act of 1980, 5 U.S.C. 601 et seq. Is there additional information regarding implementation of this rule for small businesses and entities which OSHA should consider?

The U.S. Borax and Chemical Corporation (Ex. 3–744), SOCMA (Ex. 3–891), and the Dow Chemical Company (Ex. 3–741) each responded to this preamble question. SOCMA expects "some adverse impact" on its smaller member companies because these companies are likely to have greater difficulty than others in absorbing the costs of controls (Ex. 3–891). SOCMA believes that such companies are particularly likely to be severely impacted if REL, rather than TLV, values are promulgated as OSHA PELs; in SOCMA's views, selection of TLV values as PELs "will substantially reduce the feasibility problems of the proposal" (Ex. 3–891). The Dow Chemical Company (Ex. 3–741) agrees with SOCMA that the largest impact of the rulemaking will be on smaller establishments, and also that promulgation of values consistent with those of the ACGIH, rahter than with those of NIOSH, will mitigate any such impact.

Eugene Smith, Vice President for Government and Public Affairs of the U.S. Borax and Chemical Corporation, believes that the uses of "sodium tetraborate as well as boron oxide are so ubiquitous in their applications that a complete documentation of their uses in industrial and household applications is virtually impossible" (Ex. 3–744, p. 5). Mr. Smith reports that he is aware of "very small companies who would find installation of the type of engineering controls" described in the proposal financially difficult to implement (Ex. 3–744, p. 5). OSHA is sympathetic to the concerns of U.S. Borax and has focused much of the economic and feasibility analysis for this rule on small entities that will be affected by this revision or expansion of permissible exposure limits. The Regulatory Flexibility Analysis (RFA) accompanying this final rule fully considers the impacts of this regulation on these entities and describes the magnitude of any differential small-business impacts. In the RFA, OSHA concludes that the final rule is feasible for small businesses.

17. OSHA has proposed PELs for some substances, where the basis for this proposal also includes a carcinogenicity designation (e.g., TLV with an A1 or A2 designation; REL with a Ca designation). Should OSHA include a similar carcinogen designation in the Z–4 table in this rulemaking?

Several commenters (Exs. 3–741, 3–1008, 3–1095, 3–593, 3–660, 3–891, 8–16, and 8–47) responded to this question. Some commenters (Exs. 3–741 and 3–891) indicated that OSHA's Hazard Communication Standard already requires employers to inform employees about the carcinogenic hazards of any substances listed as carcinogens by IARC or NTP. According to these respondents, identification of substances as carcinogens in the Z tables would therefore be duplicative and could cause confusion (Ex. 3–891). In addition, adding such information to a table could be confusing because there is no method of adding extensive explanatory material to a table. Other commenters (Exs. 3–593, 3–1095, 8–16 and 8–47) favored the addition of a cancer designation to carcinogenic substances included in the Z tables. For example, the American Industrial Hygiene Association (AIHA) stated:

> AIHA would support the inclusion of a designation on carcinogenicity * * * provided that such designation reflects the weight of evidence for carcinogenic effects * * *. (Ex. 8–16, p. 14).

NIOSH (Ex. 8–47) concurred in recommending the inclusion of such a designation in the final rule's Z tables.

OSHA has carefully reviewed the record evidence on this issue and has investigated the various evaluative critieria used by scientific and regulatory bodies to determine the classification of a substance as a carcinogen. The Agency notes that each organization has a different system and that the criteria used rarely coincide. Thus, the ACGIH uses two designations, A1 and A2, to refect the strength of the evidence for a substance's carcinogenicity, while the EPA has five classifications that represent different kinds of evidence. OSHA believes that the inclusion of a cancer designation on the Z tables would further complicate this already complex situation by adding yet another classification system to those already in use. OSHA is also concerned that adding a cancer designation to the Z-table limits would require frequent updating and revision as additional substances are identified as carcinogens in the future. Therefore, OSHA has determined that the present system (in which the Z tables present the exposure limits for a substance, while the Hazard Communication Standard (29 CFR 1910.1200) determines whether the evidence for a particular substance is such as to require employers to describe its carcinogenicity in their hazard communication programs) is the clearest and simplest approach to alerting workers to the hazards present in their workplaces.

18. OSHA has preliminarily decided that for substances where the ACGIH, TLV is a TWA and the NIOSH, REL is a Ceiling Value which is the same or one-half of the TWA, OSHA will propose that the TWA be adopted as the PEL. Should this approach be modified in the final rulemaking? What approach should be used when the converse of this situation (TLV, Ceiling-REL, TWA) exists.

Several commenters were cautious concerning this approach. Los Alamos (Ex. 3–1095) and NIOSH (Ex. 8–47) concurred, recognizing that an analysis of the data supporting a proposed limit must be developed on a case-by-case basis to discern which limit is appropriate. NIOSH (Ex. 8–47) also stated that the simple numerical relationship that OSHA has proposed is not a scientifically sound basis for choosing between a TWA and a ceiling value.

The Synthetic Organic Chemical Manufacturers Association (SOCMA) (Ex. 3–891) recommended that the TLV be adopted, whether the TLV is a ceiling value or a TWA, since TLVs are the most appropriate levels for adoption. The American Federation of State and County Municipal Employees (AFSCME) (Ex. 3–1043) recommended:

> OSHA should adopt most protective limit unless source dictates otherwise. Ceiling and STELS provide greater protection than TWA of the same numerical value (Ex. 3–1043, p. 5).

Other commenters (Exs. 3–1043, 3–42, and 3–1095) also wanted to ensure that OSHA understood the difference between TWA and ceiling values. OSHA understands that TWAs are not equal to ceiling limits and concurs with the definition of these two limits discussed in NIOSH's submission:

> A TWA is appropriate as a limit when the toxic effect of the substance is directly related to the total dose received in a daily exposure. Ceiling values are intended to minimize toxic effects related to the peak exposure. Ceiling values are necessary when there are immediate acute responses to an air contaminant independent of the total daily dose or when chronic effects are dose-rate

responses. Ceiling values are also used to minimize the total daily dose when there is intermittent occupational exposure, e.g., ethylene oxide (Ex. 8-47).

OSHA has always recognized the differences between TWA and ceiling limits; in the proposal, OSHA adopted the TWAs only as a starting point. Since that time, OSHA has analyzed the various docket submissions regarding individual substances. Based on these individual analyses, OSHA has developed updated PELs on a case-by-case basis. (For information regarding a specific substance, refer to the discussion for that individual substance.)

19. OSHA preliminarily plans to adopt a phased start-up schedule. This would include an initial start-up requirement permitting the use of alternate control methods for revised PELs, followed at a later date by the required use of control methods fully consistent with the methods of compliance priorities in effect at that time. OSHA will shortly be requesting comments on the heirarchy of controls. An alternate approach is to set compliance dates for engineering controls based on final determinations of that rulemaking. OSHA solicits comments on those approaches and suggestions regarding the appropriate times for the two proposed start-up dates.

The proposed rule (53 FR 20960 *et seq.*) suggested six months from the publication date of the final regulation as a reasonable time for employers to evaluate the exposures of their employees and to come into compliance using any combination of respirators, work practices, and engineering controls. Many commenters, such as the Texaco Company (Ex. 3-593) and the Synthetic Organic Chemical Manufacturers Association (SOCMA) (Ex. 3-891), indicated that the 6-month compliance date phase-in was appropriate. The Kerr-McGee Corporation (Ex. 3-623) was more specific in its comments and contended that the initial six-month period should be extended to a 24-month period to allow industry sufficient time to monitor and develop the necessary control measures. The American Paper Institute (Ex. 3-685) was also of the opinion that an initial six-month compliance period would be too short.

OSHA has extended the period to come into compliance using any method from six months from the date of publication to approximately six months from the effective date; this action adds two months to this period. OSHA concludes, based on the Agency's experience and many comments, that a six-month period after the effective date is sufficient to evaluate exposures and commence a respirator program. Most employers will only have employee overexposures to a relatively few substances. (See also the discussion under the Scope and Application of the standard.)

Several companies stated that OSHA should grant a specific extension for their particular industries as a consequence of feasibility concerns. OSHA has, however, considered the issues of feasibilty raised by rulemaking participants for specific industries and has determined that it is feasible, with few exceptions, for employers in affected industries to achieve compliance with the limits promulgated in the final rule. These exceptions are discussed in Section VII of this preamble.

In the proposal, OSHA also estimated that all employers, including those who would have to control exposures to several different chemicals, could achieve compliance within four years using the hierarchy of controls (i.e., preference for engineering controls and work practices, and, if not feasible, personal protective equipment) specified in 29 CFR 1910.1000(e). Regarding the four-year engineering implementation date schedule, OSHA received a number of comments. Most trade associations and employers supported the four-year period, and most unions suggested that one to two years would be sufficient. The State of Washington used 60 days for a similar regulation and reports that there were few difficulties with compliance. NIOSH (Ex. 8-47) suggested that two years was a reasonable time for compliance. The longest period suggested was the period recommended by the Fibre Box Association (Ex. 3-823), which stated that 10 years for compliance by industry was a reasonable time frame. OSHA has evaluated the data from various industries regarding the feasibility of compliance and has determined that it is feasible for employers in nearly all operations to achieve compliance, using engineering and work practice controls, by December 31, 1992. In light of the scope of this regulation, OSHA concludes that a shorter period would not be appropriate.

Since OSHA is in the process of reviewing the hierarchy of controls, OSHA requested comment on whether the compliance period should be tied into the completion of that rulemaking. There were few responses to this query. A few companies (see, for example, Exs. 3-669 and 3-527) suggested that the Agency delay the four-year coming-into-compliance period until after publication of any new regulations on this subject; these commenters cited costs of compliance as a major concern. The Dow Chemical Co. (Ex. 3-741) urged the Agency not to wait to set a start-up date for this rule. OSHA concludes that the December 31, 1992 deadline specified is appropriate and is supported by most of industry. Section 1910.1000(e) has been in effect for 18 years and reflects the view of most industrial hygienists. (See the Summary and Explanation section of the preamble for further details.)

20. OSHA requests comment on whether the establishment of margins of safety below lowest observed or no-effect levels is consistent with the concept of "significant risk," and on whether the specific margins of safety proposed for specific chemicals are appropriate.

Several commenters (see, for example, Exs. 3-744, 3-1095, 3-660, 8-16, and 8-47) submitted information to OSHA in response to this question. Representatives of the Los Alamos National Laboratory (Ex. 3-1095) noted that safety factors must be established on a case-by-case basis, while the U.S. Borax and Chemical Corporation (Ex. 3-744) commented that dose-response information is needed before safety factors can be applied to set an exposure limit. The American Industrial Hygiene Association (AIHA) (Ex. 8-16) is of the opinion that OSHA should adopt a "uniform toxicologic basis for assigning such factors" and should change the term "safety factor" to "uncertainty factor" in the final rule.

NIOSH (Ex. 8-47) stated that safety factors cannot be used to estimate human risk and are therefore not related to the magnitude or significance of a risk; instead, safety factors are intended to reflect uncertainty in knowledge or available data. NIOSH endorsed the use of safety factors as a "pragmatic method" to develop standards except when a nonthreshold process, such as the induction of cancer, is the outcome of concern (Ex. 8-47). NIOSH believes that "standards based on a margin of safety * * * as well as standards derived from a case-by-case evaluation, [should] be periodically reviewed to determine what new information is available" (Ex. 8-47).

OSHA is pleased that these commenters believe that the use of safety factors or margins of safety is an appropriate method of adjusting for the absence of complete information in the standards-setting process. OSHA agrees with NIOSH that this approach to limit-setting is appropriate when threshold effects are the endpoints of concern. (For a full discussion of safety factors,

see Section VI.A and the Legal Analysis section of this preamble.)

21. OSHA has identified sensory irritation, which causes rhinitis, cough, sputum production, chest pain, wheezing and dyspnea, as material impairment of health. OSHA invites comments on this understanding.

Many commenters (see, for example, Exs. 3–744, 3–1095, 3–896, 8–47, 3–660, 3–593 and 3–665) responded to this preamble question. A few were of the opinion that transitory or acute effects should not be considered material impairment of health; the U.S. Borax and Chemical Corporation (Ex. 3–744) believes that transitory "rhinitis, cough, sputum production, chest pain, wheezing, and dyspnea" do not constitute material health impairment. Stanley Dryden of Chevron Corporation believes that "mild irritants and odorants" should not be considered to pose a risk of material health impairment (Ex. 3–896).

Most commenters, however, agreed with OSHA that the signs and symptoms listed in this question should be regarded as material health impairments (Exs. 8–47, 3–1095, 3–660 and 3–593). NIOSH stated:

> The recognition of sensory irritation as potentially being "material impairment of health" is consistent with the current scientific consensus related to health effects of environmental agents.
>
> Mucous membrane irritants can cause increased blink frequency and tearing; nasal discharge, congestion, and sneezing; and cough, sputum production, chest discomfort, wheezing, chest tightness, and dyspnea. Work environments often require levels of physical and mental performance considerably greater than encountered in daily living. Even in the absence of any permanent impairment, the symptoms listed can interfere with job performance and safety.
>
> Mucous membrane irritation can result in inflammation, which may lead to increased susceptibility to nonspecific irritants and infectious agents. For example, experimental ozone exposure in humans results in increased airway reactivity. Also, studies of exposure to environmental tobacco smoke have shown irritative symptoms and evidence of increased frequency of respiratory tract illnesses in young children and decreased pulmonary function in adults * * *.
>
> Mucous membrane irritation is associated with respiratory illnesses, depending on the composition of specific exposure and on the dose, duration, and frequency of exposure. No universally applicable conclusion can be drawn at this time regarding the association between irritative symptoms and permanent injury or dysfunction. Where certain individuals show no measurable impairment after an exposure, even when experiencing irritative symptoms, others may develop identifiable dysfunction.
>
> Aside from the effects of irritation, mucous membrane exposure may result in absorption of a substance, with resultant systemic toxicity. An inflamed mucous membrane may be an even more effective route of absorption, either for the irritant or for other substances. Furthermore, injury to bronchopulmonary membranes can impair removal of particulates from the respiratory system (Ex. 8–47).

Thus, according to NIOSH, sensory irritants interfere with job performance and safety, cause inflammation, may increase the victim's susceptibility to other irritants and infectious agents, lead to permanent injury or dysfunction, or permit greater absorption of hazardous substances (Ex. 8–47). In sum, NIOSH and most other respondents agree with OSHA that sensory irritation caused by occupational exposure to the irritant substances included in this final rule constitutes a material impairment of health (see Section VI.C.3).

22. The question also arises of whether odorants present material impairment of health. That issue also might arise in the context of other substances. Based on the evidence in the final record concerning this issue, OSHA will determine if the criteria detailed in Section IV–C–16 have been met, and take appropriate action. OSHA requests comment on this issue.

Section IV.C.11 of the preamble to the proposed rule (53 FR 21135–21136) described the adverse effects associated with exposure to four substances included in the category of odorants. These substances are: Isopropyl ether, phenyl ether, propylene glycol monomethyl ether, and vinyl toluene. NIOSH (Ex. 8–47), the National Renderers Association (Ex. 3–11), the E.I. du Pont Company (Ex. 3–660), the Pharmaceutical Manufacturers Association (Ex. 3–1161), and the Los Alamos National Laboratory (Ex. 3–1095), among others, commented on this issue. According to representatives of the Los Alamos National Laboratory, odorants should not be considered as causing a material health impairment (Ex. 3–1095); John Beary, III, a physician speaking for the Pharmaceutical Manufacturers Association, is of the same opinion (Ex. 3–1161).

NIOSH described several important reasons for minimizing objectionable odors in the workplace.

> Odors emitted by industrial chemicals often play an important role in occupational safety and health. When odors can be detected before health effects occur, they may provide early warning of exposure. A number of chemicals have strong odors at concentrations which are otherwise minimally toxic. These odors may cause undue health concerns among exposed workers or may create safety hazards by distracting workers from their tasks. Strong odors in the workplace may also mask the presence of other, more toxic substances. Strong odors can produce irritation and/or nausea at high concentrations, although these effects may be reversible following cessation of exposure. Olfactory fatigue often occurs and should be considered a functional impairment that can result in increased worker exposure. Olfactory fatigue can reduce the wearer's ability to sense inadequate respirator performance of air-purifying respirators (Ex. 8–47, p. 41).

NIOSH thus concurs with OSHA that intolerable odors may have serious adverse effects in the workplace. And, although it is true that there is wide variation in individual responses to odor (i.e., in the ability to detect an odor), it is also true that one individual may respond to an odor with only mild discomfort, while another becomes overtly nauseated.

OSHA has carefully weighed all of the evidence in the record on the toxicological significance of exposures to odorant chemicals. The Agency finds that odor effects alone do not constitute material health impairment; however, OSHA notes that it is exceedingly rare for a substance only to cause odor effects. It is generally the case that odorant chemicals also cause toxic effects, such as sensory irritation or incipient central nervous system effects that manifest as headaches, nausea, vertigo, or diplopia. However, for the purposes of this rulemaking, OSHA concludes that odor alone does not constitute material health impairment.

23. Is there exposure information available which can be supplied which will refine OSHA's estimates of employee exposures and overexposures to the substances being regulated?

Although two other commenters (Exs. 3–744, 3–742) mentioned this proposal question, the only substantive comment received by OSHA in response specifically to this question came from NIOSH (Ex. 8–47), which noted that it was submitting to the docket all relevant Health Hazard Evaluations (HHEs). OSHA has reviewed these HHEs as they apply to the substances and sectors of interest and has used data from these documents in the Regulatory Impact Analysis that accompanies this final rule.

24. Is there information available which can be supplied to improve or supplement the engineering controls identified as necessary in order to reduce exposure levels? Is there additional cost data which can be supplied to refine the annual costs associated with these controls?

In response to this preamble question, the U.S. Borax and Chemical

Corporation (Ex. 3-744) submitted data to show that, for the years 1979 through 1987, the average per-year cost for environmental control units at its Borax Operation was $37,609. According to this commenter, since a large plant would have many such units, the cost per plant for SIC 28 facilities presented by OSHA in the proposal (53 FR 21376) is "orders of magnitude" lower than the costs plants would actually experience. In response to U.S. Borax, OSHA points out that the costs presented in the proposal were average annual compliance costs for *all* large plants across *all* of SIC 28. Thus, it is likely that OSHA's costs may not exactly approximate those of any particular plant but will, in the aggregate, reflect those of the average plant in this sector. OSHA has received no cost data or information that calls these average compliance cost estimates into question.

In general, feasibility and cost data were directed toward individual substances rather than being submitted in response to this question. These data are discussed in detail in Section VII.

25. Under what conditions, involving which industrial processes, will respirators be needed during the start-up period, for maintenance operations, or where other controls are infeasible in order to protect employees at the proposed exposure levels? Are respirators currently being used under the conditions identified, or would they need to be purchased? Please describe the type of respirator currently in use or needed.

This question elicited responses from several commenters (Exs. 3-593, 3-741, 3-891, 3-1095 and 8-47). The Texaco Corporation (Ex. 3-593) identified several operations where respirators are required; these included field maintenance of process equipment in refinery and petrochemical plants, confined space operations, asbestos stripping, and equipment repair. The Synthetic Organic Chemical Manufacturing Association (SOCMA) (Ex. 3-891) noted that respirator use varies from job to job and that engineering controls are not always feasible. The Dow Chemical Company (Ex. 3-741) reported that respirators are currently being used in industry where infrequent tasks make the costs of engineering controls infeasible; examples of such operations are maintenance operations, emergency operations, and certain infrequently performed process operations. Dow had no information on the type or extent of current respirator use. NIOSH (Ex. 8-47) submitted data from a 1982 NIOSH-sponsored contractor report that shows that, in 1980, 19.1 percent of mining, manufacturing, and construction workers wore or had access to certified respirators. The same report showed that the market shares of self-contained breathing apparatus, single-use, and chemical cartridge respirators were approximately equal and ranged from 25 to 30 percent each (Ex. 8-47).

OSHA did not raise the issue of methods of compliance in this rulemaking. This question was asked to gather factual information. Section VII discusses those few areas identified where respirator use may be needed. (See also the Legal Analysis sector.)

26. As a result of simultaneously regulating many substances, what cost savings will be realized in purchasing and installing engineering controls? Are alternate engineering controls available to achieve the lower permissible exposure limits being proposed?

OSHA received no substantive responses to this question. Several participants, including NIOSH and the unions presented evidence on situations in which lower levels than those proposed could be achieved by means of engineering and work practice controls. These cases are discussed in Section VII of the preamble.

27. What is the current state of technology control and financing in firms which would need to comply with reduced exposure limits to wood dust?

No commenters provided substantive responses to this question. Much information was submitted on the issues alluded to in this question; however, this information was not submitted in response to this question but rather in relation to the technological and economic feasibility of achieving the proposed limits for wood dust. This information is discussed in Section VII.

In addition to these 27 specific questions, OSHA solicited comments on the appropriateness of considering 10-, 15-, and 20-minute NIOSH RELs as 15-minute STELs and on the appropriateness of adopting PELs having other durations (i.e., 30-, 60-, or 120-minute "ceilings"), such as those recommended by NIOSH (53 FR 21242).

Only the Kerr-McGee Corporation specifically addressed this question. Kerr-McGee (Ex. 3-623) was concerned that OSHA might, in the final rule, establish the NIOSH 30, 60-, or 120-minute ceilings as 15-minute STELs. OSHA agrees with Kerr-McGee that this approach would not be appropriate at this time without additional analysis. Where the NIOSH limit for a substance was for a duration of 30, 60, or 120 minutes, OSHA has generally maintained these intervals in the final rule. The final rule adopts 15-minute STELs in cases where NIOSH has recommended a 10-, 15-, or 20-minute limit.

VI. Health Effects Discussion and Determination of Final PEL

A. General Principles of Toxicology and Dose Response

Introduction

As long ago as the 16th century, people recognized that there is no such thing as an absolutely safe chemical. The Swiss physician Paracelsus, who lived from 1493 to 1541, said:

> All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy.

On the other hand, methods have been devised to permit any chemical, no matter how poisonous, to be handled safely; this is done either by limiting the dose or controlling the exposure. However, before the necessary degree of control can be determined for a particular exposure or situation, the toxicity of the substance in question must be known. The paragraphs that follow describe the methods used by scientists to measure the relative toxicity of substances and to select exposure limits that will prevent exposed individuals from suffering adverse effects from such exposures. As this discussion demonstrates, methods of choosing exposure limits must, because of the lack or inadequacy of dose-response information for many chemicals, rely on experience in the use of these substances and on scientific and professional judgment.*

Chemicals range in inherent toxicity from those that are relatively harmless even after large doses have been administered to others that cause death if encountered even in small quantities. Toxicologists rank chemicals by categories that range from practically nontoxic (an adult human would have to consume a quart) to supertoxic (fewer than 7 drops would be lethal for most people).

In the occupational setting, it is the risk associated with a particular use of a chemical rather than its inherent toxicity that is important. *Risk* can be defined as the probability that a substance will produce harm under certain conditions of use. The converse of risk is *safety*, which is the probability that no harm will occur under specific circumstances.

* The material in this section derives principally from the following sources: Klaasen, Amdur, and Doull 1986; National Research Council 1986; Cohen 1986a, b; and Tardiff and Rodricks 1987.

The degree of hazard associated with exposure to a specific substance depends on the manner in which it is handled in a particular situation: A supertoxic chemical that is processed in a closed, isolated system may be less hazardous in actual use than a low-toxicity compound handled in an open batch process. Another factor affecting the ability of a chemical to elicit a toxic response is the susceptibility of the biological system or individual. For the relative degree of hazard to be known in a particular instance, this requires knowledge about the chemical agent, the exposure situation, and the exposed subject. In addition, the route of administration and the duration and frequency of exposure must be known.

Route of Exposure

There are four principal routes of exposure by which toxic substances can invade humans or animals. These are inhalation, ingestion, dermal absorption, and parenteral administation (i.e., administration through routes other than the intestinal canal). The route of administration of a toxin also affects the relative toxicity of the agent. For example, a chemical that can be detoxified in the liver will be less toxic if it is administered orally than if it is given systematically (i.e., inhaled). Studies that provide information about the relative toxicity of an agent via different routes of exposure can provide a considerable amount of information about the absorbability of the agent. For example, if exposure to a certain dose of a chemical via all routes of administration causes death within the same time period, it can be assumed that the substance in question is easily and rapidly absorbed. On the other hand, if the dermal dose of a chemical that is required to kill a subject is much higher than the dose required to produce the same effect when the chemical is ingested, one can deduce that the skin provides, to some degree, a barrier against that agent's toxicity. Other, less important, elements affecting the response to a toxic substance include the relative concentration of the substance, the volume of the vehicle used to administer the chemical, the chemical and physical properties of the vehicle, and the dose rate (i.e. the period of time over which the dose is administered).

Duration and Frequency of Exposure

Scientists conduct animal experiments that involve four different types of exposure: Acute, subacute, chronic, and subchronic. Acute exposures are limited to periods of less than 24 hours and can involve either single or repeated exposures within that period. Subacute exposures are repeated exposures that last for one month or less, while subchronic exposures have a duration of one to three months. When a research project having a chronic regimen is conducted, the test animals are dosed repeatedly for a period lasting more than three months. Animals exposed acutely can have both immediate and delayed-onset responses. Similarly, chronic exposures can cause immediate reactions as well as long-term effects.

The frequency of dosing also has an important influence on the magnitude of the toxic effect: A large single dose of an acute toxin will usually have more than three times the effect of one-third the dose given at three different times, and the same dose administered in 10 or 15 applications might have no effect whatsoever. The pattern of dosing is important because it is possible for some of the substance to be excreted between successive administrations or because the lesion caused by the toxin has a chance to be partially or completely repaired between applications. Thus a chronic effect is said to occur: (1) If a toxic sustance accumulates in the system of an exposed person or animal because the dose absorbed is greater than the body's ability to transform or eliminate the substance; (2) if it produces adverse effects that are not reversible; or (3) if it is administered in a manner that permits inadequate time for repair or recovery.

Variation in Response

Responses to toxic insults vary in a number of ways. For example, some toxicants have immediate effects, while others are associated with delayed symptom onset. The latency period for carcinogenic agents may be as long as 40 years for some types of cancer, and even some acute agents, such as some chemicals that have adverse ocular effects, may not cause overt symptoms until hours after exposure.

Another difference in type of response concerns the reversibility or irreversibility of the effect. Reversibility depends on the site of action as well as the magnitude of the insult. That is, some tissues of the body, such as the liver, have considerable ability to regenerate; others, like the kidney or central nervous system, do not.

The site of action associated with toxic substances also varies widely. Local effects are those lesions caused at the site of first contact between the agent and the organisms. Examples of localized effects are skin burns caused by contact with a caustic substance and site-of-contact tumors that develop at the locus of the injection of the carcinogen.

In contrast to localized effects, systemic effects involve the absorption and distribution of the toxic agent from the point of entry to a distant site; the toxic response is manifested at this distant point. An example of a systemic poison is mercury, which produces its toxic effect on the central nervous system. Often, the site of deposition for a chemical is not the organ system most affected by the toxin. For example, although lead is deposited and concentrated in the bone, it affects the central nervous system. Any sites that are adversely affected by the toxic effects, of exposure to a substance, whether they are sites of contact or distal sites, are called the target organs of toxicity.

In cases of systemic poisoning, the system most often affected is the central nervous system (CNS); it is common for the CNS to be involved even when another target, such as the liver, is the primary target organ of toxicity. In descending order of frequency, the systems or organs most often involved in cases of systemic poisoning are the central nervous system, the circulatory system, the blood and hematopoietic system, the visceral organs (liver, kidneys, lungs), and the skin.

Dose-Response

The relationship that associates the dose of a chemical with the effects it causes is called the dose-response relationship. A single data point relating a dose to a response is sufficient to establish a dose-response relationship. As additional data become available, it is possible to expand our understanding of the dose-response relationship to cover a range of doses or exposures. Dose-response is an important principle in toxicology, and an understanding of dose-response is important in establishing occupational or other exposure limits. Knowing how toxic substances act makes it easier to predict the potential effects of exposure. (It is, of course, generally true that lowering dose reduces response, and data are often available to demonstrate that lower doses reduce responses, at least on the grossly observable level. However, data showing that more subtle responses (e.g., those at the subcellular level) have been reduced are rarely available.)

To apply dose-response relationships, it is helpful if several types of data are available. First, it must be possible to relate a response to a particular chemical. Although basic data pointing toward causality may be available, it is

often difficult to refine the dose-response relationship further. For example, epidemiological studies often identify an association between a disease and one or more causative agents. However, since information on the precise identity of the etiologic agent, the actual dose received, and the true site of the response is usually not available, it is often impossible to use data from epidemiological studies to establish a precise dose-response relation between a specific dose of a toxin and an effect.

The second condition to be met before dose-response can be established is that it must be possible to relate the response to the dose. It is relatively easy to determine that a large dose causes an obvious response. Refining the relationship, however, involves three other requirements: (1) That there be a receptor site; (2) that the response and the intensity of the response be related to the concentration of the toxin at the receptor site; and (3) that the concentration of the toxin at the site be related to the dose given.

The third principle underlying the concept of dose-response is that there must be a quantifiable means of measuring the toxicity of a substance and a method of expressing this measured toxicity. Although lethality in test animals is often used to measure toxicity, the best form of measurement would involve quantification of the sequence of molecular events occurring during the toxic response. In the absence of such endpoints, other good methods are available. For example, it is common to measure an effect believed to be related to the substance in question. The level of activity of an enzyme in the blood is often used as a measure of effect, e.g., serum glutamic-oxaloacetic transaminase (SGOT) levels are used to measure liver damage. Many different endpoints can be used to measure toxic effects, such as changes in muscle tone, heart rate, blood pressure, electrical activity of the brain, motor functioning, and behavior.

The most widely used endpoint, especially when a new substance is involved, is lethality in an animal test system. Lethality studies allow scientists to make comparative assessments of a chemical's toxicity as it relates to that of many other substances. Research of this type also permits the gathering of essential information on dose, duration, route of administration, site of action, and the target organ of toxicity.

Form of the Response

The classic form of dose-response is sigmoidal (Figure 1). This form characterizes the relationship between the amount of a toxin administered and the degree of response to that dose. The response is measured on the ordinate, and the dose is represented on the abscissa.

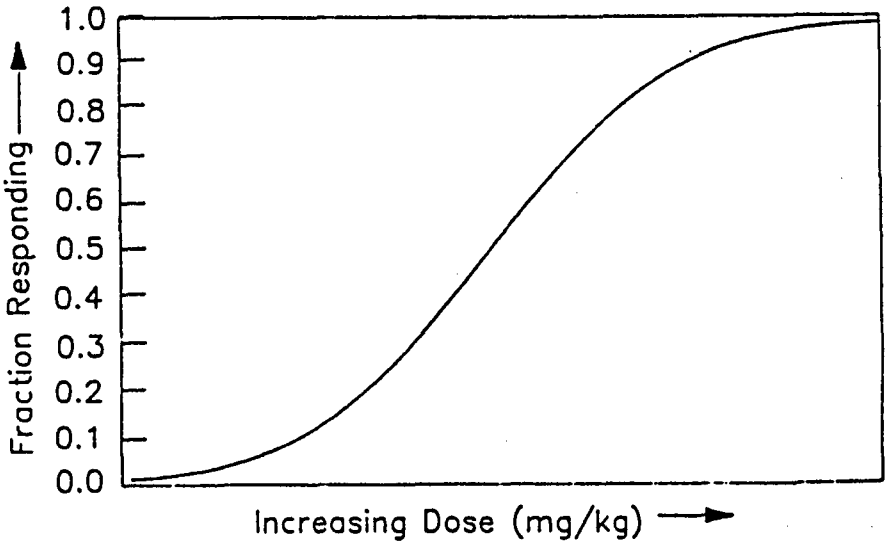
Dose-response can be thought of in two ways:

- As exposure increases, the proportion of the population that manifests the response increases (quantal response); and
- As exposure increases, the intensity of an individual's response increases (graded response).

A relatively flat dose-response curve means that a large change in dose is required before there is a significant change in response. A steep curve, on the other hand, means that a small change in dose will elicit a large increase in response. Although it is sometimes possible to generate a curve of the type shown in Figure 1, it is not necessary to do so to demonstrate that exposure at a given level is associated with a particular response. That is, it is not necessary to have sufficient data to define, in mathematical terms, the dose-response relationship to know that exposure at a given level is associated with adverse consequences.

Figure 1

Diagram of Dose-Response Relationship



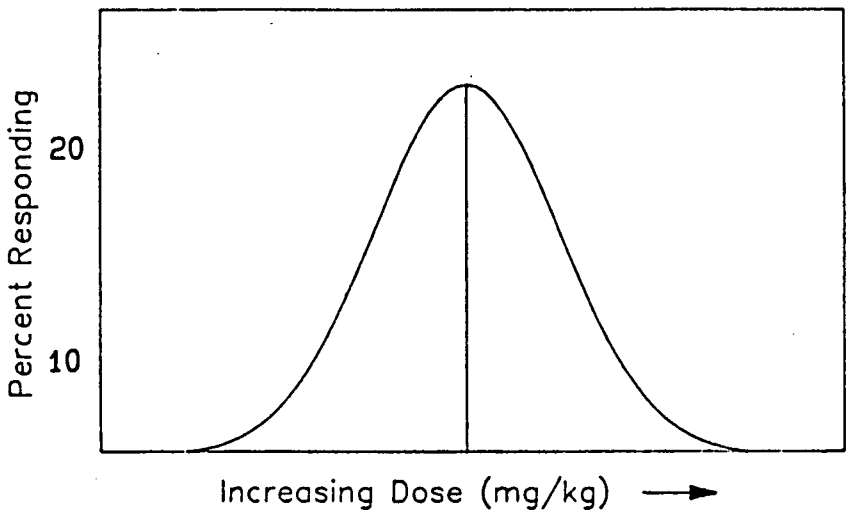
In the regulatory context, it is most common to express dose-response relations in terms of the percentage of the population responding. However, before this information can be evaluated, the endpoint being considered must be known. For every substance, there are several dose-response relationships, depending on endpoint: A substance that produces irritation at low doses may cause more severe symptoms or even death at high doses and in other conditions. For example, many substances that are mucosal irritants at low doses will produce pulmonary edema and nervous system effects at high doses.

Plotting the cumulative percentage of individuals responding against dose produces the typical sigmoid curve. Such a curve reflects the fact that at the lowest dose, zero percent of the population responds, while 100 percent of the population will respond at the highest dose. However, if the percentage responding is plotted against incremental rather than total dose, the curve produced is a normal distribution (Figure 2). This curve says that a relatively small percentage of the population will manifest the response at the lowest dose and that a similarly small percentage of the population will exhibit the effect only at the highest dose. What this normal distribution of response reflects is individual and species variation in exposed populations. A wide degree of variation

occurs even in inbred, homogeneous laboratory animals, and such variability increases dramatically when a heterogeneous population, such as workers, is involved. Individuals responding at the left end of the curve shown in Figure 2 are hypersusceptible, while those at the right end could be termed resistant.

FIGURE 2

Diagram of Quantal Dose-Response Relationship



Because the relationship between dose and response is sigmoidal, response approaches zero as dose approaches zero. However, because of the mathematical form used to express this relationship, a true zero response can never be achieved. In the strictest sense, therefore, a true threshold dose level (i.e., the dose with which a zero response is associated) can never be established on the basis of experimental research. Instead, scientists attempt to define the minimum dose associated with a specific endpoint, which is customarily termed the "threshold" dose for that particular endpoint. However, unless a specific endpoint (such as respiratory irritation, cholinesterase inhibition, the development of a tumor, or death) is specified, the concept of a threshold is essentially meaningless. In fact, a separate threshold could be said to exist for each of these endpoints.

The extent to which an experimentally derived "threshold" actually reflects the true threshold for a substance (i.e., the level above which a response will occur and below which no response will occur) depends on several factors, such as the number of animals used to determine the experimental threshold, the number of dose levels tested, and the degree of variation represented in the test subjects. For example, to determine an LD_{50} (the lethal dose that will kill 50 percent of the animals tested) with a high degree of precision requires the use of a minimum of 50 test animals and five dose groups (10 animals in each group). Other factors that can influence the magnitude of the median lethal dose include the sources involved, the sex and age of the animals, the environmental condition prevailing during the test conditions, diet, the health status of the subjects being tested, and the subjects' past exposure to other toxic substances.

In toxicological research, the experimentally observed threshold dose is called the low-observed-effect level (LOEL) or the low-observed-adverse-effect level (LOAEL). Alternatively, the threshold may be expressed as the highest no-observed-effect level (NOEL), i.e., the highest dose administered and found not to produce a given response. Determination of an accurate NOEL requires both a careful interpretation of the toxicological data and the use of an adequate number of test animals. The National Academy of Sciences (1985) has concluded that the chance of finding a no-adverse-effect level (that is, of missing an adverse effect) at a given dose is statistically greater in experiments having a small number of animals than in studies involving a large number of animals. Thus, the degree of confidence one has that a NOEL actually represents a "safe" dose, rather than a research design artifact, increases with the number of animals tested. The greatest degree of confidence is associated with studies involving a large number of animals that were tested at several doses that were administered at close intervals.

In a recent publication (Tardiff and Rodricks 1987), David W. Gaylor of the National Center for Toxicological Research explained that experimentally derived thresholds represent statistical limitations in study design rather than biological characteristics:

> The existence of dose-response relationships might lead one to assume incorrectly the existence of threshold doses below which no toxic effects could occur. As dosage is decreased, the prevalence of an observable toxic effect * * * diminishes to zero. Eventually, a dosage is reached below which the experiment has essentially no resolving power to distinguish between the spontaneous background rate and small induced toxic effects * * *.
>
> If no toxic effects are detected at a specified dosage, this dosage is called the no-effect, or more correctly the no-observed-effect dosage. Because of the limitations of any given experiment, the no-observed-effect dosage is not a precise estimate of a true no-effect level. Lack of statistical significance is not equivalent to no toxic effect. It may or may not be, and further experimentation would be required to resolve this equivocal issue * * *. The no-observed-effect level is not a biological property, but, rather, a statistical property or operational threshold that is highly dependent on sample size.

The scientific issues surrounding the concept of no-observed-effect levels or experimentally derived thresholds have important implications for their use in establishing protective occupational exposure limits. Because the no-observed-effect level cannot represent the "true" threshold for an adverse effect, given the design of most toxicologic studies, regulators and others have used the concept of safety factors (also known as uncertainty factors) to aid them in setting permissible exposure limits; that is, the exposure limit is established at some interval below the no-observed-effect level to provide additional assurance that exposed populations are not likely to suffer harm.

The size of the interval between the permissible exposure limit and the no-

observed-effect level depends on a professional judgment as to whether the no-observed-effect level is likely to represent a level that is not harmful to humans. Thus, if the available data include a NOEL derived from a well-conducted human study, a smaller safety factor might be used to establish an exposure limit than would be used if the data to be used to establish the limit consisted of a NOEL from an animal study; in the latter case, there is greater uncertainty regarding the relationship between the animal NOEL and human NOEL. Safety factors have also been used to recognize the fact that the human population is heterogeneous and that there may be a wide variation in individual responses to toxic substances (the wide range in the odor thresholds reported for some substances is a good illustration of individual variability in response).

The use of NOELs, LOAELs, and safety factors to develop permissible exposure limits is not a recent development:

> For more than half a century, evaluation of the safe use of chemicals has been focused mainly on the development of toxicity data and on the application of professional judgment to the ad hoc interpretation of such data to derive acceptable levels of exposure for humans. Generally, this practice has taken the form of identifying from studies in laboratory animals the no-observed-effect level and dividing it by a safety factor (usually 100 for NOELs derived from chronic studies) reflecting the uncertainties of relating data to humans under their conditions of exposure and the quality and appropriateness of the data base * * *.
>
> Safety factors are usually chosen prospectively to address the uncertainties of interspecies extrapolation. Although safety factors as small as 2 and as large as 2000 have been used * * * the safety factor of 100 is used most commonly, at least for NOELs derived from chronic toxicity studies, and incorporates adjustments for interspecies variability (usually 10) and intrahuman variability (usually 10) * * *. The resulting value is equivalent to a NOEL in humans (Tardiff and Rodricks 1987, pp. 391, 421.)

Tardiff and Rodricks caution, however, that the use of safety factors has been questioned because these factors "often create the impression that human population thresholds have been identified and that there is virtually no risk below that level of exposure" (Tardiff and Rodricks 1987, p. 421).

Although safety factors have traditionally been used to establish exposure limits for chronic or lifetime exposure situations, they have also been applied to establish limits for acute effects resulting from short-term exposure. The National Academy of Sciences' Committee on Toxicology has been using a safety-factor approach to establish emergency exposure guidance levels (EEGLs), which are exposure levels judged to be acceptable for military personnel performing tasks during emergency situations. An EEGL is not considered to be a safe exposure level for routine or normal operations, but these levels are considered acceptable when tasks must be performed to prevent greater risks (e.g., death or injury caused by fires or explosions). In developing EEGLs, safety factors are generally applied to account for uncertainties in the use of animal data and when extrapolating between different dose routes. The NAS also develops short-term public emergency exposure guidance levels (SPEGLs) to apply to the exposures of the general public to contaminants during airborne chemical releases; SPEGLs are generally set at a level of 0.1 to 0.5 times the EEGL (i.e., an additional safety factor of from 2 to 10) (*Criteria and Methods for Preparing Emergency Exposure Guidance Level (EEGL), Short-Term Public Emergency Guidance Level (SPEGL), and Continuous Exposure Guidance level (CEGL) Documents.* Washington, DC: National Academy Press, National Academy of Sciences 1986).

The use of the safety factor approach in establishing occupational exposure limits was addressed by many rulemaking participants (Exs. 3-744, 3-1095, 8-16, 8-47, 116, and 144; Tr. 1-221, Tr. 2-163 to 2-164). NIOSH (Ex. 8-47) stated that safety factors cannot be used to estimate human risk and are therefore not related to the magnitude or significance of a risk; instead, NIOSH believes that safety factors are intended to reflect uncertainty in the available data. This comment echoes the observation made by Tardiff and Rodricks, i.e., that safety factors do not necessarily identify a human population thereshold. NIOSH (Ex. 8-47) also endorsed the use of safety factors as a "pragmatic method" of developing standards (except when a nonthreshold process, such as the induction of cancer, is the outcome of concern). NIOSH also believes that "standards based on a margin of safety * * * as well as standards derived from a case-by-case evaluation, [should] be periodically reviewed to determine what new information is available" (Ex. 8-47).

Dr. Marcus Key, Professor of Occupational Medicine at the University of Texas School of Public Health, also testified on the appropriateness of using safety factors to establish occupational exposure limits:

> We seldom, if ever, know with any precision where a significant risk level begins or ends; hence, the need for safety factors. Safety factors depend on several considerations. * * * mainly on toxicity and the nature of the health effects, but also on the availability of scientific evidence of effects at lower levels.
>
> Professional judgment must be relied on in selecting safety factors, with one to three orders of magnitude being commonly used for serious effects, and 50 percent, or [a] safety factor of 2, [being used] for acute, less harmful effects (Tr. 1-221).

Both Dr. Key (Tr. 1-221) and Dr. Ernest Mastromatteo, Chairman of the ACGIH TLV Committee (Tr. 2-163 to 2-164) testified that safety factors are frequently used by the ACGIH to develop recommended exposure limits.

Some commenters (Exs. 8-16, 116, and 144; Tr. 7-121) were of the opinion that OSHA should adopt a uniform system of assigning safety factors to establish permissible exposure limits. For example, the Workers' Institute for Safety and Health (WISH) (Ex. 116, p. 13) commented that OSHA should review the toxicology profiles prepared by the Agency for Toxic Substances and Disease Registry (ATSDR), in which Reference Doses (RfD) are computed. The RfD, as described by WISH, is "an estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure of the human population to a potential hazard that is likely to be without risk of deleterious effects during a lifetime" (Ex. 116, p. 13). The RfD is derived by applying uncertainty factors to experimentally derived NOAELs in a consistent manner. The uncertainty factors used by ATSDR include factors of 10 to account for each of the following:

- Human variation in response;
- Extrapolation from animals to humans;
- Extrapolation of effects associated with lifetime exposure from less-than-lifetime studies; and
- Additional uncertainty in relying on a LOAEL rather than a NOAEL.

In addition, ATSDR applies a factor of from 1 to 10 to account for the overall quality of the scientific evidence.

EPA uses the same approach to develop RfDs for noncarcinogens; EPA's application of this approach is described in a concept paper presented by the EPA Reference Dose Work Group (Ex. 144, Appendix A). As explained by the Work Group:

> The RfD is useful as a reference point for gauging the potential effects of other doses. Usually, doses that are less than the RfD are not likely to be associated with any health risks, and are therefore less likely to be of regulatory concern * * * Nonetheless, a clear conclusion cannot be categorically drawn that all doses below the RfD are

"acceptable" and that all doses in excess of the RfD are "unacceptable" (Ex. 144, Appendix A, p. A-10).

The EPA has been compiling dose-response data and information on RfDs for almost 2,000 chemicals in a database called the Integrated Risk Information System (IRIS). The system is described by Dr. Rebecca T. Zagraniski, Assistant Commissioner of the Division of Occupational and Environmental Health, New Jersey Department of Health (Exs. 144 and 144A). In her posthearing submission, Dr. Zagraniski presents an analysis in which EPA RfDs from the IRIS system are converted to Workday Ambient Air Concentrations (WACs) for 43 of the substances included in this rulemaking. These WACs were then compared to OSHA's proposed PELs for the same substances. After converting the RfDs to equivalent lifetime occupational exposure levels, Dr. Zagraniski found that all but three of the resulting WACs were lower than 1 mg/m^3 and that the WACs for noncarcinogens were generally 100 to 1,000 times lower than the PELs being proposed by OSHA in this rulemaking. Dr. Zagraniski commented on these findings as they relate to OSHA's proposal:

> The WACs are not recommended exposure limits because they do not take into account numerous significant considerations including feasibility, anecdotal reports of effects following human exposure, routes of exposure other than inhalation, and other critical information. Also, the WACs for non-carcinogens are based primarily on oral exposure studies. In some cases, there may be inhalation studies which are more appropriate for use in setting an occupational exposure guideline, but which were not discussed in IRIS due to their focus on the oral exposure route. In spite of these constraints, the WACs may be considered preliminary health-based guidelines which are useful as indicators that current PELs and TLVs may need reevaluation (Ex. 144A, p. 4).

In response to Dr. Zagraniski's comments, OSHA notes that the approach suggested by this commenter is new and was not supported by other participants. It is also inconsistent with the recommendation of most expert organizations in this field and would require extensive analysis by OSHA before its merit could be ascertained. Accordingly, OSHA finds this approach inappropriate for use in the present rulemaking.

In this rulemaking, OSHA has evaluated the efficacy of the final rule's limits on a case-by-case basis, although the initial evaluation presented in the NPRM relied heavily on analyses conducted by the ACGIH and NIOSH, the limits promulgated in the final rule are based on an expanded toxicologic assessment using information contained in the rulemaking record. OSHA believes that, at this time, this case-by-case assessment is the best way to establish new and revised limits for the numerous substances addressed in this rulemaking.

Types of Toxicological Evidence

The evidence available to scientists wishing to evaluate the toxicity of a substance can be derived from studies in laboratory animals, *in vitro* studies in cell or tissue systems, reports of clinical observations, studies of exposed human populations, or from intervention studies conducted with human volunteers. The preceding paragraphs have described animal studies (or "bioassays"). The following section discusses the two most common types of human evidence: Data derived from clinical observations and information from epidemiological studies.

Clinical observations. Much of the data on the toxic effects associated with human exposures have come from industrial accidents, fatal poisonings, or other such tragedies. This information is generally more useful in delineating broad categories of pathological effects than in refining a specific dose-response relationship, because the exposure levels causing the accident are known to be high but cannot be quantified with precision.

Epidemiological studies. Studies conducted by epidemiologists are designed to reveal the patterns of disease or mortality prevailing in certain groups of people (usually workers) exposed to a single toxin or to a group of substances. One of the advantages of epidemiological studies is that they involve humans and their responses to actual situations. The interpretation of the results of epidemiological studies is complicated by the inevitable presence of confounding variables that occur whenever human populations are involved. Ideally, the populations being studied (i.e., the study population and the control population) should be fully comparable with regard to every variable except the single characteristic under study. Because it is rarely possible to achieve this degree of comparability, statistical techniques are often used to attempt to adjust for this lack of comparability. In addition, if the measured effect is relatively large, it is unlikely that confounding factors will obscure the true picture.

Broadly speaking, epidemiological studies can have two possible outcomes: They can report an effect or they can report no effect; in the former case, the study is termed a positive study, and in the latter, a negative one. Within each of these categories, it is possible for the study to be correct (that is, to give a true-positive or true-negative result) or to be incorrect (that is, to give a false-positive or a false-negative result). A false-positive result reports that there is an increased risk when in fact there is not, and a false-negative study reports that there is no increased risk when in fact there is.

The probability that a study will detect a statistically significant effect if that effect is actually present is called the power of the study. As the power of a study increases, the likelihood of producing a false-negative error decreases. Power is dependent on two factors: The level of relative risk being evaluated and the number of cases of the effect (i.e., disease) that are expected in the population being studied. The number of expected cases depends both on the sample size and the expected disease frequency in the comparison population. For example, a study involving a small population and a common disease can have the same power as a study of a rare disease in a large population. Consequently, studies of larger samples have sufficient power to detect smaller increases in risk, and studies of smaller samples will be able only to detect large increases in relative risk.

Because epidemiological studies have limitations, it is essential that the power of such studies, particularly of negative studies, be examined to ensure that their sample sizes are adequate to detect the absence of increased risk with validity. When the power of a study is not adequate, negative studies cannot be said either to contradict or to support the conclusion that increased risk exists. Essentially, a negative epidemiologic study identifies a NOAEL, which, as discussed above, reflects the statistical limitations of a study more than the "true" population threshold for an effect. However, a study with a positive result may indicate a relationship if the excess risk is high, even if the study's sample size is small and the effects of some factors are not controlled for.

Quality of Evidence

Dose-response models have often been used in the quantitative assessment of the risks associated with exposures to carcinogenic substances. However, less scientific effort has been devoted to models to be used with non-carcinogenic substances. Mathematically precise methods to establish the true no-effect level or to define the dose-response curves have not been developed for most of the more

than 400 substances involved in this rulemaking.

Most of the scientific work that has been done was designed to identify lowest observed effect or no-effect levels for a variety of acute effects. As described above, experts in industrial hygiene and occupational health have developed factors to be used to offset, at least to some extent, the insensitivity of NOELs and LOELs to such factors as subcellular effects, sensitive individuals, and chronic effects. It is possible to use these data, combined with professional judgment and OSHA's expertise and experience, to determine that significant risk exists at current levels of exposure and that a reduction in these levels will substantially reduce this risk of material impairment of health. OSHA is also confident that it is not attempting in this rulemaking to reduce exposures to insignificant levels. However, additional analysis may well reveal that the levels being established in the final rule can be refined further in the future.

B. Historical Development of Occupational Exposure Limits Early Limits

Until the development of occupational health standards, the occurrence of adverse health effects resulting from exposures to hazardous substances or conditions in the workplace could only be determined *ex post facto*—after impairment had already occurred to the health and welfare of exposed employees. In her 1910 studies of lead poisoning, Dr. Alice Hamilton was forced to rely on "personal observations of working conditions and the illness and deaths of workers to demonstrate the existence of harmful exposures" (Paull 1984/Ex. 1–255). The concept of occupational exposure limits thus represents a dramatic breakthrough in the battle against occupational disease and remains "one of the most useful and indispensable tools yet devised for safeguarding the health and well-being of industrial workers" (Thomas 1979/Ex. 1–96).

Occupational exposure limits are air quality values that apply in workplaces, and they are derived by studying the correlation between the amount of a toxic substance absorbed by the body and its effects on health. Within the context of occupational exposure, knowledge of this relationship permits quantification of the etiology "of a large number of occupational health impairments, [evaluation of] the risk of such impairments and, if necessary, [consideration of] the effectiveness of preventive measures" (Parmeggiani 1973/Ex. 1–229). More specifically, an understanding of the levels at which disease or other health effects occur can be used to establish limits of occupational exposure below which health hazards are unlikely to occur in most workers.

The historical development of occupational exposure limits began with the published reports of a German scientist whose investigations in 1883 into the effects on experimental animals (and on himself) of carbon monoxide in known air concentrations caused him to conclude that "the boundary of injurious action of carbon monoxide lies at a concentration in all probability of 500 parts per million, but certainly [not less than] 200 parts per million" (Cook 1987/Ex. 1–187). Shortly after the appearance of this first documented dose-response value, another German researcher, K. N. Lehmann, published a series of reports on a number of chemical substances under the title "Experimental Studies on the Effect of Technically and Hygienically Important Gases and Vapors on the Organism." This series culminated in 1936 with a comprehensive paper on chlorinated hydrocarbons, published as Volume 116 of *Archiv für Hygiene*.

In 1912, Rudolf Kobert published a table of exposure limits, based on animal studies, for 20 compounds. One of the first tables of hazardous air concentrations to originate in the United States was a technical paper published in 1921 by the U.S. Bureau of Mines. The 33 substances included in this table were those frequently encountered in the workplace. In addition to limits based on acute toxic effects, this table provided some information on the least detectable odor concentration and the lowest airborne concentration required to cause irritation (Paull 1984/Ex. 1–255; Cook 1987/Ex. 1–187).

Throughout the 1920s and 1930s, data became available that correlated concentrations of harmful substances with observed effects on worker health for such materials as lead and mercury compounds, benzene, and granite dusts. These early occupational health studies, which were based on animal experiments and on findings in exposed workers, provided the kind of data needed to link human exposures "to concentrations that were capable of producing not only acute, but chronic health effects" (Paull 1984/Ex. 1–255).

After 1935, the emphasis of researchers had shifted, for the most part, from the reporting of a series of values for a range of acute effects to results that yielded a single limit based on studies of repeated exposures. Over the years, a sizable amount of data about the levels of exposure that would not produce injurious effects had been amassed for a considerable number of substances. "By the early 1940s, control of the occupational environment to prevent the harmful absorption of toxic materials was becoming an accepted principle, and the practical problem of defining what was 'harmful' was beginning to be met by employing maximum allowable concentrations" (Paull 1984/Ex. 1–255). In 1943, Sterner (Ex. 1–806) explained the meaning of the term maximum allowable concentrations as "the upper limit of concentration of an atmospheric contaminant which will not cause injury to an individual exposed continuously during his working day and for indefinite periods of time" (Paull 1984/Ex. 1–255).

The first lists of maximum allowable concentrations of airborne toxic substances were issued between 1933 and 1938. The Union of Soviet Socialist Republics (U.S.S.R.) was the first country to make occupational exposure limits a statutory obligation; in 1933 it published a list that included 14 substances (although health standards for some air pollutants apparently were used in the Soviet Union during the 1920s). The first American list was published four years later by the State of Massachusetts, and in 1938 Germany issued occupational health standards for a number of organic solvents (Holmberg and Winell 1977/Ex. 1–141). Additionally, the United States "imposed limited occupational safety and health requirements on certain contractors with the Federal government" when the Walsh-Healey Act was passed in 1936 (Mintz 1984/Ex. 1–840).

Standards Developed by Professional Organizations

During the 1940s, American organizations led in the development of occupational health standards, beginning with the American Standards Association (now the American National Standards Institute, or ANSI) list of "maximum acceptable concentrations" (MACs), which appeared in 1941. This list represented a consensus of opinion by the ASA and a number of industrial hygienists who had formed the American Conference of Governmental Industrial Hygienists (ACGIH) in 1938 (Baetjer 1980/Ex. 1–223). Originally conceived of as a time-weighted concentration to be maintained as an average over the working shift, the MAC was redefined in 1957 to mean an upper level (ceiling level) that should never be exceeded (Turner 1976/Ex. 1–79).

An important contribution to occupational health standard-setting was made in 1945 by Warren Cook (Ex. 1–726), who published a list of maximum allowable concentrations for 132 industrial atmospheric contaminants. These limits had been developed by six states, the U.S. Public Health Service, and the American Standards Association, and included Cook's own list of "accepted or tentative values" based on industrial experience, animal experimentation, human sensory response, or a combination of these factors. This table was followed by

> Documentation supported by 187 specific references, indicating the basis and reliability of each value. Cook was the first investigator to codify all of the available data on MAC's and present it in one publication. His list of recommended values was incorporated, practically without changes, by the ACGIH in establishing the TLVs. In support of Cook's inferences, it should be noted that 50 of the * * * values that he recommended in 1945 were subsequently adopted as federal standards, and are still in use today (Paull 1984/Ex. 1–255).

The American Conference of Governmental Industrial Hygienists Subcommittee on Threshold Limits presented its second report at the Eighth Annual Meeting of the ACGIH in 1946. The report included values for 131 gases, vapors, dusts, fumes, mists, and 13 mineral dusts "compiled from the list reported by this subcommittee * * * in 1942, from the list published by Warren Cook in * * * 1945, and from published values of the Z–37 Committee of the American Standards Association" (Cook 1987/Ex. 1–87). The Committee's report noted that:

> Considerable difficulty attends the fixing of satisfactory values for maximal allowable concentrations of chemicals in respirable atmospheres because of the lack of a uniform definition of the maximum allowable concentration concept. One concept is that the M.A.C. value should represent as accurately as possible that concentration at which a worker exposed for a sufficient period of time will just escape physiological or organic injury and occupational disease.
>
> A second concept is that the M.A.C. should represent some fraction of that concentration which will injure the worker in order to allow a margin of safety in the design of protective equipment and guard against possible synergistic effects in the case of multiple exposures. A third concept is that the M.A.C. should perform the functions of the former concepts and in addition provide a work environment free of objectionable but non-injurious concentrations of smokes, dusts, irritants and odors. Obviously all of these concepts cannot be fulfilled with the establishment of a single value. M.A.C. values in use at the present time represent examples of all of these concepts. The committee feels that the establishment of dual lists or a single definition is not possible at the present time (ACGIH 1946).

The report concluded by stressing that the 1946 list of M.A.C. values was presented "with the definite understanding that it be subject to annual revision" (ACGIH 1946).

Papers presented at both the Ninth International Congress on Industrial Medicine in London (1948) and at the Fifteenth International Congress of Occupational Health in Vienna (1966) also dealt with maximum acceptable concentrations. The first of these proposed that zones of toxicity be set up to facilitate an understanding of the relative hazards of substances, "since the boundaries of MAC values were not sharp lines of demarcation" (Cook 1987/Ex. 1–87). At the 1966 meeting, discussion took place on the advantages of the concept of a "peak level" of exposure—an extension of the "ceiling level" notion inherent in the definition of a MAC since 1957. A "peak level" was defined as one "that can be applied to certain substances for brief designated periods and for a strictly limited number of times during the work shift, with a designated time interval between peaks. The 'peak' concept places a limit on the intermittent higher exposures that occur in many industrial operations. The time-weighted average exposure limit is of course to be observed [even when a peak has also been assigned to a substance]" (Cook 1987/Ex. 1–87).

Terminology and definitions throughout this early period were ambiguous and imprecise, reflecting uncertainty as to exactly what needed to be and could be done in the realm of occupational health standard setting. Initially, the ACGIH designated its recommended limits as "maximum allowable concentrations." although this term was often used interchangeably with "threshold limit values." Confusion about the meaning, interpretation, and relative significance of the terms being employed during this embryonic period was common. After 1953, the ACGIH defined the concept of threshold limit values in the preface to its annual published list of occupational health standards as "maximum average atmospheric concentrations * * * for an eight-hour day." This definition of the TLVs as average concentrations differed from the general understanding of the original term "maximum allowable concentrations," which were essentially ceiling values (Stokinger 1962/Ex. 1–998).

Documentation for the 238 substances included in the TLV list for 1956 was provided by Smyth (Ex. 1–759) in a separate paper in which the author:

> Recommended that the TLV's include references to the underlying data, and that the concepts represented by the values be restated in more realistic toxicological terms. In his analysis of the TLVs, he [Smyth] concluded that nine categories of objectionable action were guarded against: Chronic toxicity, acute toxicity, narcosis, irritation, asphyxiation, fume fever, eye pigmentation, allergic response, and cancer (Paull 1984/Ex. 1–255).

At about the same time, Stokinger stated that, in his opinion, the Threshold Limits Committee had avoided grappling with the issue of developing a method for establishing limits for industrial carcinogens and noted that, with the exception of nickel carbonyl, limits had not been assigned for potential carcinogens (Paull 1984/Ex. 1–255). In 1962, however, the TLV Committee included three carcinogens as additions to the TLV list, although these were listed separately in an appendix and did not have assigned TLVs.

Despite the fact that the ACGIH had stressed early on that TLVs were intended as guides and not as rigidly enforceable limits, the American Standards Association's MAC values (or, where none was available, the TLV) were included as mandatory limits in the Safety and Health Standards for Federal Supply Contracts, which were published in 1960 under the Walsh-Healey Act. Following this action the ACGIH issued a statement on the definitions and interpretations of TLVs and MACs (Stokinger 1962/Ex. 1–998). At the same time, the ACGIH announced the production of the first edition of the *Documentation for Threshold Limit Values* (ACGIH 1962); this was followed by another paper in which the work and intentions of the Threshold Limits Committee were reviewed. Turner states that:

> [a]t this time the concept of ceiling values and excursion factors around the time-weighted average values was introduced in order to reduce conflict or confusion with the "maximal" values in the American [ANSI] Standerds. A "C" (ceiling value) listing was to be given to those fast-acting substances thought likely to be injurious if the concentration exceeded the limit value by more than a designated factor for a relatively short period (about 15 min.). The factor varied between 3 and 1.25, depending inversely upon the magnitude of the TLV. A corollary was that the factor would also indicate the limit of permissive excursion of the concentration above the TLV for a substance not given a "C" listing, always provided that the time-weighted average concentration did not exceed the TLV. This rule of thumb approach to limiting exposure is no doubt appropriate to certain substances when they are used routinely throughout the working day. It seems to have little relevance

in other instances where exposure is irregular or where the basis for fixing the TLV is on grounds other than toxicity (Turner 1976/Ex. 1–79).

Several commenters (Tr. pp. 6–30 to 6–31, 7–119, 8–139 to 8–141, and 8–167) were of the opinion that the ACGIH's procedures for establishing TLVs were not open to comment and that its reasons for selecting certain TLVs were not clear. Dr. Ernest Mastromatteo, Chairman of the ACGIH's TLV Committee, explained the organization's limit-setting process at the hearing (Tr. pp. 2–113 to 2–128). He stated that the Committee's minutes have recently been made public and explained that the committee often invited industry or union consultants to help the committee in its work on the TLVs (however, these consultants do not vote on the recommended limits). In addition, Dr. Mastromatteo described the ACGIH's process of placing new or revised limits on an "Intended" list for a period of two years, during which time comments on the proposed limits are invited, and considered.

Permissible Exposure Limits in the Era of OSHA

The enactment of the Occupational Safety and Health Act of 1970 marked the first "comprehensive and serious attempt * * * to protect the health and safety of American workers" (Mintz 1984/Ex. 1–840); it also greatly extended the use of MACs and TLVs by authorizing the newly established Occupational Safety and Health Administration (OSHA) to adopt as its own standards "national consensus standards" and established federal standards (29 U.S.C. 655 (a)). Mintz notes that "in addition to the safety standards adopted under section 6(a), OSHA also adopted permissible exposure limits for approximately 400 toxic substances. These [start-up] health standards, now appearing in 29 CFR 1910.1000 * * * were derived from both national consensus and established federal standards. The national consensus standards had been issued by ANSI, while the established federal standards had been adopted under the Walsh-Healey Act from the TLVs * * * recommended by the * * * ACGIH" (Mintz 1984/Ex. 1–840).

Since OSHA's large-scale adoption of the ANSI consensus standards and the 1968 ACGIH TLVs, the Agency has promulgated standards under section 6(b) of the OSH Act to regulate the industrial use of 24 substances, most of which have been identified as occupational carcinogens, but the ANSI and ACGIH start-up standards continue to comprise the major part of the Agency's occupational health and safety program.

In the interval since the establishment of OSHA and the adoption of the ACGIH and ANSI limits by the Agency, the ACGIH has continued to revise, update, and document the recommended limits that appear in its annual list of TLVs. Since 1968, annual revisions have been made to these limits by the ACGIH. During this time, the TLVs have been "accepted on an international basis as the best available guides for providing healthful occupational environments, and at least 18 countries, including the United States, have either adopted them as legal standards or as guides to legal action, thus verifying their efficacy in accomplishing this purpose" (Paull 1984/Ex. 1–255).

The action OSHA takes today initiates the process of updating the Agency's Z-table permissible exposure limits. That these limits were seriously out of date is attested to by the fact that the ACGIH has found it necessary to revise or add nearly 400 limits to its list in the 20 years since the limits that were later adopted by OSHA were initially published. Recognition that OSHA's Z-table limits need updating to reflect recent developments in toxicology and new data on the health effects associated with exposure to these substances is widespread throughout industry: For example, OSHA's Hazard Communication standard (29 CFR 1910.1200) requires organizations that develop Material Safety Data Sheets (MSDSs) to include on these MSDSs the ACGIH's current TLV values as well as OSHA's limits.

The following section describes the methodology used by OSHA in selecting the limits it is promulgating today. The Agency believes that promulgation of these limits will address a broad range of significant risks now prevalent in industry. As many industrial hygienists and occupational safety and health professionals have noted, the use of permissible exposure limits continues to be the single most efficacious way of protecting the health, functional capacity, and well-being of the American worker.

C. Description of the Substances For Which Limits Are Being Established

In this rulemaking, OSHA considered revising 428 substances, and the final rule is revising existing or adding new limits for several hundred toxic substances currently being manufactured, used, or handled in workplaces throughout general industry. This section of the preamble identifies the PELs being established, describes the available toxicological data, and explains the Agency's rationale for selecting the final permissible exposure limits for these substances.

The universe of substances included in this rulemaking is bounded by the substances for which the American Conference of Governmental Industrial Hygienists (ACGIH) has established a Threshold Limit Value (TLV) for exposures in the work environment. That is, OSHA is not at this time establishing exposure limits for any hazardous substance that is not included in the ACGIH's 1987–88 List of TLVs. In addition, where the limit included in the current ACGIH list was identical to OSHA's existing Z-table limit for the same substance, OSHA did not consider revising its existing limit.

Although new limits are not being established for chemicals excluded from the ACGIH's 1987–88 list, OSHA has not limited its initial consideration of appropriate limits to those levels established by the ACGIH. The Agency has also carefully evaluated the exposure limits recommended by the National Institute for Occupational Safety and Health (NIOSH), OSHA's sister agency. In instances where both NIOSH and the ACGIH have recommended substantially different limits for the same substance, OSHA has thoroughly analyzed the evidence presented by each organization and has made its own judgment of the appropriate level at which to establish the PEL. For all substances addressed in this rulemaking, OSHA has also evaluated the extensive record evidence. The limits being established today thus represent, in the Agency's professional judgment, those levels found to be most consistent with the best available toxicological data, OSHA's mandate, and the case law that has subsequently developed to interpret that mandate. (For a discussion of the relevant legislative and judicial principles, see the sections of this preamble entitled Pertinent Legal Authority, History and Need for Revision of the PELs, and Approach).

For ease of analysis and presentation, the substances included in the scope of this rulemaking have been grouped into 18 separate sub-sections. In general, these groupings reflect the primary basis underlying the ACGIH or NIOSH recommended limits for these substances. In addition, three additional sections cover substances for which the ACGIH has increased its limits, substances for which OSHA is adding short-term limits, and those for which the Agency is adding skin notations.

The following sections are included:

1. Substances for which Limits Are Based on Avoidance of Neuropathic Effects.
2. Substances for which Limits Are Based on Avoidance of Narcotic Effects.
3. Substances for which Limits Are Based on Avoidance of Sensory Irritation.
4. Substances for Which Limits Are Based on Avoidance of Liver or Kidney Effects.
5. Substances for Which Limits Are Based on Avoidance of Ocular Effects.
6. Substances for Which Limits Are Based on Avoidance of Respiratory Effects.
7. Substances for Which Limits Are Based on Avoidance of Cardiovascular Effects.
8. Substances for Which Limits Are Based on Avoidance of Systemic Toxicity.
9. Substances for Which Limits Are Based on Observed-No-Adverse-Effect Levels.
10. Substances for Which Limits Are Based on Avoidance of Physical Irritation and Other Effects.
11. Substances for Which Limits Are Based on Avoidance of Odor Effects.
12. Substances for Which Limits Are Based on Analogy to Related Substances.
13. Substances for Which Limits Are Based on Avoidance of Biochemical/Metabolic Effects.
14. Substances for Which Limits Are Based on Avoidance of Sensitization Effects.
15. Substances for Which Limits Are Based on Avoidance of Cancer.
16. Substances for Which Current ACGIH TLVs Are Less Stringent than Former OSHA PELs.
17. Substances for Which OSHA is Establishing Short-Term Exposure Limits.
18. Substances for Which OSHA is Adding Skin Notations.

A list of the references that OSHA relied on in evaluating the toxicological evidence pertaining to these chemicals appears in Section VI–D.

1. Substances for Which Limits Are Based on Avoidance of Neuropathic Effects

Introduction

Many industrial chemicals have been shown to cause severe neurological effects in exposed workers, and in many cases these effects are irreversible. Limits have been set on the basis of avoidance of neuropathic effects for 20 substances. Table C1–1 lists the former, proposed, and final rule limits, CAS number, and OSHA HS number for each of these substances. The table shows time-weighted averages (TWAs), ceiling limits, and short-term exposure limits (STELs). For this group of 20 substances, OSHA is lowering its former TWA–PEL for three substances; adding a STEL to a former or a revised TWA for four substances; changing a ceiling to a TWA or a TWA to a ceiling for four substances; establishing permissible exposure limits for seven substances not formerly regulated by OSHA; retaining an existing TWA but changing its accompanying ceiling to a STEL for one substance; and lowering the former TWA and changing its accompanying ceiling to a STEL for one substance.

Description of the Health Effects

The human nervous system comprises the central nervous system (CNS) and peripheral nervous system (PNS). The CNS is made up of the brain and spinal cord, while the PNS consists of a network throughout the body of nerves that communicate with the CNS via connections to the spinal cord. The brain and spinal cord are bathed in cerebrospinal fluid, which supplies nutrients to the CNS and also acts as a barrier against some foreign substances. This barrier protects the central nervous system. In general, fat-soluble substances readily diffuse across this barrier and water soluble substances do not.

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TABLE C1-1. Substances for Which Limits Are Based on Avoidance of Neuropathic Effects

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1051 n-Butyl alcohol	71-36-3	100 ppm TWA	50 ppm Ceiling, Skin	50 ppm Ceiling, Skin
1078 Chlorinated camphene	8001-35-2	0.5 mg/m^3 TWA, Skin	0.5 mg/m^3 TWA 1 mg/m^3 STEL, Skin	0.5 mg/m^3 TWA 1 mg/m^3 STEL, Skin
1114 Decaborane	17702-41-9	0.05 ppm TWA, Skin	0.05 ppm TWA, Skin 0.15 ppm STEL	0.05 ppm TWA, Skin 0.15 ppm STEL
1116 Di-sec-octyl-phthalate	117-81-7	5 mg/m^3 TWA	5 mg/m^3 TWA 10 mg/m^3 STEL	5 mg/m^3 TWA 10 mg/m^3 STEL
1123 Dichloroacetylene	7572-29-4	—	0.1 ppm Ceiling	0.1 ppm Ceiling
1149 Dipropylene glycol methyl ether	34590-94-8	100 ppm TWA, Skin	100 ppm TWA, Skin 150 ppm STEL	100 ppm TWA, Skin 150 ppm STEL
1200 n-Hexane	110-54-3	500 ppm TWA	50 ppm TWA	50 ppm TWA
1202 2-Hexanone	591-78-6	100 ppm TWA	5 ppm TWA	5 ppm TWA
1216 Iron pentacarbonyl (as Fe)	13463-40-6	--	0.1 ppm TWA 0.2 ppm STEL	0.1 ppm TWA 0.2 ppm STEL
1236A Manganese, fume (as Mn)	7439-96-5	5 mg/m^3 Ceiling	1 mg/m^3 TWA 3 mg/m^3 STEL	1 mg/m^3 TWA 3 mg/m^3 STEL

TABLE C1-1. Substances for Which Limits Are Based on Avoidance of Neuropathic Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1237 Manganese cyclopentadienyl tricarbonyl (as Mn)	12079-65-1	—	0.1 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA, Skin
1238 Manganese tetroxide (as Mn)	1317-35-7	—	1 mg/m^3 TWA	1 mg/m^3 TWA
1240 Mercury (aryl and inorganic compounds) (as Hg)	7439-97-6	0.1 mg/m^3 TWA	0.1 mg/m^3 Ceiling	0.1 mg/m^3 Ceiling, Skin
1241 Mercury, vapor (as Hg)	7439-97-6	0.1 mg/m^3 TWA	0.05 mg/m^3 TWA, Skin	0.05 mg/m^3 TWA, Skin
1242 Mercury, (organo) alkyl compounds (as Hg)	7439-97-6	0.01 mg/m^3 TWA 0.04 mg/m^3 Ceiling	0.01 mg/m^3 TWA 0.03 mg/m^3 STEL, Skin	0.01 mg/m^3 TWA 0.03 mg/m^3 STEL, Skin
1251 Methylacrylonitrile	126-98-7	—	1 ppm TWA, Skin	1 ppm TWA, Skin
1253 Methyl bromide	74-83-9	20 ppm Ceiling, Skin	5 ppm TWA, Skin	5 ppm TWA, Skin
1304 Pentaborane	19624-22-7	0.005 ppm TWA	0.005 ppm TWA 0.015 ppm STEL	0.005 ppm TWA 0.015 ppm STEL

TABLE C1-1. Substances for Which Limits Are Based on Avoidance of Neuropathic Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1316 Phenyl mercaptan	108-98-5	--	0.5 ppm TWA	0.5 ppm TWA
1342 1,2-Propylene glycol dinitrate	6423-43-4	--	0.05 ppm TWA, Skin	0.05 ppm TWA

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

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Chemicals that affect the central nervous system may manifest their toxic effects peripherally. An example of this is the tremor associated with elemental and organic mercury poisoning. Exposure to some chemicals (for example, n-hexane) is associated with axonal degeneration of the nerves in both the central and peripheral nervous systems. Baker (1983/ Ex. 1–230) refers to this dual-system effect as central-peripheral distal axonopathy.

Nervous system toxicants can affect motor function, sensory function, or integrative processes, and they can also cause changes in the behavior of exposed persons. Substances that cause demyelination or neuronal damage can produce motor dysfunction that is expressed as muscular weakness or unsteadiness of gait, while exposures to chemicals that are associated with loss of sensory function may result in alterations in touch, pain, or temperature sensation or damage to sight or hearing. Other neuropathic chemicals affect the way in which information is processed in the brain and can interfere with learning and memory. All of the health effects described above constitute material impairments of health within the meaning of the Act.

Although mature neurons cannot divide and be replaced, the nervous system has considerable ability to restore function lost as a result of exposure to toxic chemicals. This capability to restore function even after neurons have been killed is achieved by two mechanisms: Plasticity of organization and redundancy of function. That is, when some neurons die, other cells that perform the same function may be able to maintain an adequate level of functioning, or other neurons may be able to "learn" how to perform the lost function. However, even when one of these mechanisms comes into play to compensate for neuronal damage, the overall reserve capacity of the nervous system will have been diminished. The loss of this reserve could be critical in a situation in which additional demands are placed on the nervous system. Thus, even so-called reversible neuropathic effects should be seen as toxic effects causing alterations in and material impairment of the normal functioning of the nervous system.

The neurological effects potentially associated with chemical exposures are numerous, and it is not always easy to identify the precise target site. However, recent medical advances have made tests available that can detect neurological damage that was not detectable several years ago. For example, electrophysiological methods have been developed to measure damage to the visual pathway caused by such exposures. Because of the variation in individual responses to chemical exposures, exposure limits should be set with a view toward this range of susceptibility and the avoidance of any neuropathic effects.

Peripheral Nervous System Effects

The pathological mechanisms associated with peripheral neuropathies result from segmental demyelination or axonal degeneration. Segmental demyelination destroys the myelin sheath but leaves the axon intact; this causes a slowing in nerve conduction velocity. Muscle weakness is often the first sign of such segmental demyelination, and this effect can progress to a decline in motor function or paralysis. Although remyelination may occur within weeks after injury, even a temporary loss in motor or sensory function places the affected worker or others at risk of injury.

Axonal degeneration is a more serious effect in that recovery is often slow or incomplete. It causes demyelination secondary to the degeneration of the distal portion of the nerve. This effect occurs when a chemical interferes with the physiologic dynamics of the nerve, e.g., when it decreases the transport of nutrients to the nerve. The axon will degenerate (die back) sufficiently to accommodate the cell's capacity to supply it with nutrients. Axonal degeneration can also occur as a result of biochemical or metabolic derangement of the central nervous system. Alkyl mercury and elemental mercury are examples of chemicals causing this type of effect (Cavanaugh 1977/Ex. 1–202).

Central Nevous System Effects

The mechanism of action of central nervous system toxins is not well understood but is believed to be associated with neurochemical alteration in the brain. Seizures, Parkinsonism, intellectual impairment, narcosis, dementia, cranial neuropathy, and visual disturbances are all examples of effects that can occur after overexposures to neuropathic chemicals. The more serious CNS effects, such as Parkinsonism, dementia, intellectural impairment, and cranial neuropathy, are generally irreversible (Baker 1983/Ex. 1–230). Before these effects are manifested, subtle changes in behavior may occur; if these subtle signs are interpreted correctly, exposure can be stopped before irreversible damage occurs.

Dose-Response Relationships and Neuropathic Effects

The development of chemically induced neurological effects is believed to follow a dose-response pattern. At an exposure intensity or duration below the no-effect level, detectable effects are unlikely to be evident. As exposure intensity/duration increases to and beyond this level, the toxin begins to interfere with the normal cellular processes of the neurological system. At this early stage, transient signs and symptoms may appear. Overt effects become more severe as exposure continues and finally progress to serious loss of neurological function and possible permanent damage to neural tissue. Increases in our ability to detect neurological changes at lower levels of exposure have shown that neurobehavioral changes or impairment may occur at levels previously thought to be innocuous. These early effects can be important indicators of potential functional impairment at exposure levels below those that produce either transient or permanent damage. Heavy metals, solvent, and pesticides are examples of chemicals that can cause symptoms that include nausea, sensory and motor function impairments, depression, sleep disturbances, cognitive impairment, and sexual dysfunction. Limits for substances in this group are generally designed to maintain worker exposures below the level associated with such symptoms. This approach ensures that employees will not be likely to suffer these material impairments of health and provides a margin of safety against the risk of more severe or permanent neurological impairment.

The following discussions describe the record evidence and OSHA's findings for all of the substances in this group and illustrate the material impairments of health faced by workers exposed to these toxicants.

n-BUTYL ALCOHOL

CAS: 71–36–3; Chemical Formula: $CH_3CH_2CH_2CH_2OH$

H.S. No. 1051

OSHA's former PEL for n-butyl-alcohol was a 100-ppm 8-hour TWA: the ACGIH limit is a 50-ppm ceiling, with a skin notation. The proposed and final rule PEL is a 50-ppm ceiling, with a skin notation. NIOSH (Ex. 8–47, Table N1) concurs that these limits are appropriate. n-Butyl alcohol is a colorless, highly refractive liquid with a mild vinous odor that has long been known to cause irritation of the eyes and headaches in occupational settings.

Systemic effects in the form of vestibular and auditory nerve injuries have been reported in workers in France and Mexico (Seitz 1972 and Velasquez 1964, both as cited in ACGIH 1986/Ex. 1-3, p. 76; Velasquez, Escobar, and Almaraz 1969/Ex. 1-1174). Contact dermatitis of the hands may occur due to the defatting action of liquid n-butyl alcohol, and toxic amounts can be absorbed through the skin. Based on data describing the rate of n-butyl alcohol uptake through the skin of dogs, DiVincenzo and Hamilton (1979, as cited in *Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., Vol. 2C, pp. 4571-78, Clayton and Clayton 1982) suggested that direct contact of human hands with n-butyl alcohol for one hour results in an absorbed dose that is four times that resulting from inhalation of 50 ppm for one hour.

The former OSHA limit of 100 ppm (TWA) was based on the studies of Tabershaw, Fahy, and Skinner (1944, as cited in ACGIH 1986/Ex. 1-3, p. 76) and of Smyth (1956/Ex. 1-759). These studies indicated that workers experienced no narcotic or systemic effects at levels lower than 100 ppm. However, irritation has been reported in humans exposed to 24 ppm; this irritation became uncomfortable and was followed by headaches at 50 ppm (Nelson, Enge, Ross et al. 1943/Ex. 1-66).

More recent data reported by Seitz (1972, as cited in ACGIH 1986/Ex. 1-3, p. 76), Velasquez (1964, as cited in ACGIH 1986/Ex. 1-3, p. 76), and Velasquez, Escobar, and Almaraz (1969/Ex. 1-1174) indicate serious exposure-related long-term systemic effects on the auditory nerve and hearing loss (hypoacusia); the magnitude of the hearing loss was related to length of exposure. Nine of 11 workers exposed without hearing protection to 80 ppm for periods of from 3 to 11 years displayed impaired hearing. This phenomenon was particularly evident in younger workers (Velasquez 1964, as cited in ACGIH 1986/Ex. 1-3, p. 76; Velasquez, Escobar, and Almaraz 1969/Ex. 1-1174).

Three commenters, ConAgra (Ex. 3-635), the Motor Vehicle Manufacturers Association (MVMA) (Ex. 3-902), and ARCO (Tr. p. 3-237) submitted comments on n-butyl alcohol. Con Agra (Ex. 3-635) misinterpreted OSHA's discussion of a 1964 study (Velasquez, as cited in ACGIH 1986/Ex. 1-3, p. 76) to mean that OSHA attributed all hearing loss found in the workers in this study to n-butyl alcohol exposure. ARCO (Tr. p. 3-237) also questioned n-butyl alcohol's effect on hearing. In response to these commenters, OSHA notes that n-butyl alcohol has been shown in many studies to damage the auditory nerve and further, that workplace noise may also have contributed to the hearing loss observed in these studies. The MVMA comment (Ex. 3-902) lists n-butyl alcohol as a substance for which rulemaking should be delayed, but provides no other details.

OSHA finds that the former PEL of 100 ppm is not sufficiently protective against the acute effects associated with exposure to n-butyl alcohol; in addition, the possibility of auditory nerve damage from exposures below the 100-ppm level makes the former PEL inadequate. A skin notation is necessary because data in beagle dogs suggest that dermal contact with n-butyl alcohol can result in a systemic dose greater than that obtained by inhalation (DiVincenzo and Hamilton 1979). The Agency is establishing a permissible exposure limit of 50 ppm as a ceiling, with a skin notation, for n-butyl alcohol. OSHA concludes that this limit will protect workers against the significant risks of possible vestibular and auditory nerve injury as well as of headaches and irritation, which constitute material impairments of health and are associated with exposure to this substance at levels above the new limit.

CHLORINATED CAMPHENE (60 Percent)

CAS: 8001-35-2; Chemical Formula: $C_{10}H_{10}Cl_8$
H.S. No. 1078

Previously, OSHA had a limit of 0.5 mg/m³, with a skin notation, for chlorinated camphene. The ACGIH has a TLV-TWA limit of 0.5 mg/m³ and a TLV-STEL of 1 mg/m³ for chlorinated camphene (60 percent), with a skin notation, and these were the limits proposed. The final rule retains the 0.5-mg/m³ 8-hour TWA and the skin notation, and adds a 1-mg/m³ STEL for chlorinated camphene, an amber waxy solid with a pleasant, pine-like odor.

Chlorinated camphene has demonstrated a moderately high acute toxicity in animal studies (ACGIH 1986/Ex. 1-3, p. 115). Toxic doses cause varied central nervous system effects, including nausea, muscle spasms, confusion, and convulsions (Hayes 1963/Ex. 1-982). Data indicate that rats and guinea pigs show no significant effects at dietary levels of 800 ppm daily for a six-month period (Alderson Reporting Co., as cited in ACGIH 1986/Ex. 1-3, p. 115). Monkeys tolerate daily feeding at 10 ppm but show toxic symptoms after two weeks' feeding at the 60-ppm level (Sosnierz, Szczurek, Knapek, and Kolodziejczyk 1972/Ex. 1-760). Although chlorinated camphene may accumulate in fatty tissues, it clears quickly when ingestion is terminated (Sosnierz, Szczurek, Knapek, and Kolodziejczyk 1972/Ex. 1-760).

In humans, the acute lethal dose of chlorinated camphene is between 2 and 7 grams, and a dose of 10 mg/kg causes nonfatal convulsions in some exposed individuals. The ACGIH (1986/Ex. 1-3, p.115) concludes that the acute toxicity of chlorinated camphene is equivalent to that of chlordane, for which the fatal human dose is estimated to be around 6 grams; the ACGIH TLV-TWA for chlordane is 0.5 mg/m³. One study of 25 human volunteers failed to reveal toxic responses to daily 30-minute exposures to 500 mg/m³ for 10 consecutive days, followed by similar exposures for three consecutive days three weeks later (Shelansky 1947, as cited in ACGIH 1986/Ex. 1-3, p. 115). There are no reports of occupational poisonings, and a review of the medical records of employees engaged in the manufacture and handling of chlorinated camphene showed no ill effects in workers exposed for an average of 3.7 years (Frawley 1972, as cited in ACGIH 1986/Ex. 1-3, p. 115).

NIOSH does not concur with OSHA's PELs for this substance; NIOSH believes that chlorinated camphene is a potential occupational carcinogen and should have lower exposure limits (Ex. 8-47, Table N6B; Tr. pp. 3-97, 3-98). No other comments on the health effects of this substance were submitted to the record.

OSHA is retaining the 8-hour TWA PEL of 0.5 mg/m³ TWA and adding a 15-minute STEL of 1.0 mg/m³ for this insecticide. The Agency's skin notation is retained. OSHA concludes that both a TWA and a STEL are required to protect exposed workers against the significant risks of bioaccumulation and neuropathic and systemic effects; the Agency finds that these effects constitute material impairments of health. The STEL ensures that TWA exposures will be maintained under good industrial hygiene control.

DECABORANE

CAS: 17702-41-9; Chemical Formula: $B_{10}H_{14}$
H.S. No. 1114

OSHA's former limit for decaborane was 0.05 ppm TWA, with a skin notation. The ACGIH has a TLV-TWA of 0.05 ppm and a TLV-STEL of 0.15 ppm, also with a skin notation. The proposal retained the 8-hour TWA of 0.05 ppm and added a 0.15-ppm STEL, with a skin notation, and the final rule establishes these limits. NIOSH (Ex. 8-47, Table N1) concurs that these limits are appropriate. Decaborane forms colorless crystals that are stable at ordinary temperatures and have a pungent odor.

The acute toxicity of decaborane is extremely high for small laboratory animals. The 40-hour LC_{50}s for rats and mice are 46 and 12 ppm, respectively (Schechter 1958/Ex. 1–363). Dermal LD_{50}s for rabbits and rats are 71 and 740 mg/kg, respectively (Svirbely 1954a/Ex. 1–385). Acute exposures to decaborane cause loss of coordination, convulsions, weakness, tremors, and hyperexcitability. Decaborane's primary effects are on the kidneys and liver. Studies of repeated exposures to this substance suggest that the toxicity of decaborane is intermediate between that of pentaborane and diborane. The ability of decaborane to penetrate the skin is particularly notable, as is its toxicity to the central nervous system in some species, e.g., rats and rabbits (Svirbely 1954a/Ex. 1–385, 1954b/Ex. 1–530, and 1955/Ex. 1–386). Monkeys showed decreased ability for certain operant behaviors when injected with doses of 3 to 6 mg/kg decaborane (Reynolds et al. 1964, as cited in ACGIH 1986/Ex. 1–3, p. 169). Central nervous system toxicity has been observed in humans exposed occupationally (Krackow 1953/Ex. 1–344). No comments other than NIOSH's were received on the health effects of decaborane.

OSHA is retaining its 8-hour TWA PEL of 0.05 ppm TWA and skin notation, and adding a 15-minute STEL of 0.15 ppm for decaborane. The Agency concludes that these limits will provide protection against the significant risks of material health impairment in the form of neuropathy and kidney and liver damage possible in the absence of a short-term limit for decaborane.

Di-sec-OCTYL PHTHALATE

CAS: 117–81–7; Chemical Formula: $C_{24}H_{38}O_4$
H.S. No. 1116

OSHA formerly had a limit of 5 mg/m³ TWA for di-sec-octyl phthalate. The ACGIH has a TLV–TWA of 5 mg/m³ and a TLV–STEL of 10 mg/m³, and these are the limits that were proposed. In the final rule, OSHA is retaining the 8-hour TWA limit of 5 mg/m³ and adding a 15-minute STEL of 10 mg/m³ for this light-colored, viscous, odorless, combustible liquid.

Di-sec-octyl phthalate (DEHP) is not acutely toxic in small laboratory animals via the oral route. The oral LD_{50} reported for mice is 26.3 g/kg; for rats, it is 33.8 g/kg (Krauskopf et al. 1973/Ex. 1–495). No skin irritation or sensitization potential has been demonstrated in either animals or humans, and the lethal dermal dose in rabbits is about 25 ml/kg (Singh, Lawrence, and Autian 1972/Ex. 1–436). Shaffer, Carpenter, and Smyth (1945/Ex. 1–369) and Lawrence (unpublished data, as cited in ACGIH 1986/Ex. 1–3, p. 223) have reported deaths in rats and chronic inflammation of the lung in mice exposed to DEHP at unspecified levels.

Long-term dietary toxicity studies in rats, guinea pigs, and dogs have established a no-effect dose level of about 60 mg/kg/day, and no carcinogenic or histologic abnormalities were observed at this level (Gesler 1973/Ex. 1–481). Higher doses were associated with growth retardation and increased liver and kidney weights but not histologic abnormalities. Metabolic studies have demonstrated that laboratory animals do not appreciably metabolize DEHP (Dillingham and Autian 1973/Ex. 1–477). Teratogenicity studies in pregnant rats indicated that fertility is unaffected at doses of 0.1, 0.2, or 0.33 percent of the acute intraperitoneal LD_{50} dose for rats, although slight effects on embryonic and fetal development were observed in these animals; skeletal deformities were the most common teratogenic effects observed (Dillingham and Autian 1973/Ex. 1–477). Mutagenic effects were observed at intravenous doses of one-third, one-half, and two-thirds of the acute LD_{50}; these effects are consistent with DEPHP's ability to produce dominant lethal mutations (Dillingham and Autian 1973/Ex. 1–477).

A study of workers exposed to a mixture of the vapors of diethyl phthalate, dibutyl phthalate, and di-2-ethylhexyl phthalate reported that exposures to 1 to 6 ppm caused no peripheral polyneuritis (Raleigh, as cited in ACGIH 1986/Ex. 1–3, p. 223). However, Russian investigators examined male and female workers exposed to between 1.7 and 66 mg/m³ of various combinations of airborne phthalates (including butyl phthalate, higher aryl phthalates, dioctyl phthalate and others) and noted complaints of pain, numbness, and spasms in the upper and lower extremities after six to seven years of exposure. Polyneuritis was observed in 32 percent of the workers studied, and 78 percent of these workers showed depression of vestibular receptors (Milkov, Aldyreva, Popova et al. 1973/Ex. 1–646).

OSHA received a comment from the Chemical Manufacturers Association Phthalate Esters Program Panel (Ex. 3–900). Although the Panel did not oppose the proposed PEL for di-sec-octyl phthalate, it objected to this substance's categorization as a neuropathic agent on the grounds that (1) confounding exposures to tricresyl phosphate and vinyl chloride, which are known neurotoxicants occured in the study referenced in the NPRM; and (2) other studies (in humans or animals) have not substantiated that this substance is neuropathic:

> Including [di-sec-octyl phthalate] in this category of compounds [i.e., neuropathic agents] is not justified and could lead to improper labeling of the material or unwarranted regulations, and restrictions on the use of the material based on unfounded conclusions (Ex. 3–900, p. 1).

In response to this comment, OSHA notes that the classfication scheme used in the preamble to the proposed and final rules is not intended to have regulatory implications. As explained earlier in the preamble, OSHA is using this scheme simply to facilitate generic rulemaking; the various categories reflect the health endpoint used by the ACGIH or NIOSH as the point of reference in setting a limit. Most of the substances included in this rulemaking produce multiple health effects and could be classified in more than a single health effects category. Di-sec-octyl phthalate is no exception, and exposure to this substance has been associated with liver damage, testicular injury, and teratogenic and carcinogenic effects in experimental animals, as well as with possible neuropathic effects.

Another commenter, Lawrence H. Hecker of Abbott Laboratories feels that the STEL for di-sec-octyl phthalate is unwarranted (Ex. 3–678, p. 8). OSHA disagrees with Dr. Hecker and finds that, for substances posing serious health hazards, such as those associated with di-sec-octyl phthalate exposure, the STEL further protects workers from the signficant adverise effects that could occur in the short-term excursions above the TWA limit permitted in the absence of a STEL.

NIOSH concurs in OSHA's selection of limits for di-sec-octyl phthalate but believes it should be designated as a potential occupational carcinogen (Ex. 8–47, Table N6A). On the other hand, the Chemical Manufacturers Association's (Ex. 140) analysis of the evidence for DEHP's carcinogenicity led the CMA to conclude that this substance is not a carcinogen. OSHA is aware of di-sec-octyl phthalate's carcinogenic effects in experimental animals and notes that IARC has determined that sufficient evidence exists to designate it as an animal-positive carcinogen. However, adequate data are not available to evaluate the risk of cancer to humans. The Agency will continue to monitor the scientific evidence for di-sec-octyl phthalate and will re-evaluate this substance in the future if such evidence suggests that this is appropriate.

In the final rule, OSHA is retaining the 8-hour PEL of 5 mg/m³ and adding a 15-

minute STEL of 10 mg/m³ for di-sec-octyl phthalate. The Agency concludes that these limits together will protect workers from the significant risks of neuropathic, hepatic, and other systemic injuries, which constitute material health impairments and are associated with exposure to this substance.

DICHLOROACETYLENE

CAS: 7572-29-4; Chemical Formula: ClC ≡ CCl
H.S. No. 1123

OSHA previously had no limit for dichloroacetylene. The ACGIH has a TLV-ceiling of 0.1 ppm for this liquid, which explodes upon boiling. OSHA proposed a ceiling limit of 0.1 ppm, and this is the limit established by the final rule.

In preliminary inhalation exposure studies, guinea pigs demonstrated a 4-hour LC_{50} of 20 ppm; death occured two or three days after exposure and was caused by pulmonary edema. In rats, similar exposures to dichloroacetylene in the presence of 330 ppm of trichloroethylene indicated an LC_{50} of 55 ppm (Siegal 1967, as cited in ACGIH 1986/Ex. 1-3, p. 177). When dichloroacetylene was mixed with 9 parts of ether, the 4-hour LC_{50} in rats was 219 ppm; in combination with 7 parts of trichloroethylene, the 4-hour LC_{50} in rats was 55 ppm; and exposure to dichloroacetylene with 10 parts of trichloroethylene caused a 4-hour LC_{50} in guinea pigs of 15 ppm (Siegal, Jones, Coon, and Lyon 1971/Ex. 1-371).

In humans, dichloroacetylene exposure causes headache, loss of appetite, extreme nausea, and vomiting; it affects the trigeminal nerve and facial muscles and exacerbates facial herpes. Disabling nausea was experienced by approximately 85 percent of individuals exposed for prolonged periods of time (not further specified) at concentrations from 0.5 to 1 ppm (Saunders 1967/Ex. 1-361). A number of occupational fatalities have been attributed to exposure to dichloroacetylene (Humphrey and McClelland 1944/Ex. 1-491; Firth and Stuckey 1945, as cited in ACGIH 1986/Ex. 1-3, p. 177). Humphrey and McClelland (1944/Ex. 1-491) reported 13 cases of cranial nerve palsy, nine of which had labial herpes, following exposure to dichloroacetylene. These patients also had symptions of nausea, headache, jaw pain, and vomiting. Autopsies of two of these fatalities revealed edema at the base of the brain (Humphrey and McClelland 1944/Ex. 1-491).

NIOSH concurs with OSHA's limit for dichloroacetylene but believes that this substance should be designated as a potential occupational carcinogen (Ex. 8-47, Table N6A). However, as explained elsewhere in the preamble, OSHA has decided not to designate substances specifically as carcinogens since so many other organizations already do so. OSHA received no other comments regarding the health effects of dichloroacetylene.

In the final rule, OSHA is establishing a ceiling limit of 0.1 ppm for dichloroacetylene. The Agency concludes that this limit will substantially reduce the significant risks of disabling nausea and serious systemic effects posed to workers exposed to dichloroacetylene at the levels formerly permitted by the absence of any OSHA limit. OSHA finds that these health effects constitute material impairments of health.

DIPROPYLENE GLYCOL METHYL ETHER

CAS: 34590-94-8; Chemical Formula: $CH_3OC_3H_6OC_3H_6OH$
H.S. No. 1149

OSHA formerly had an 8-hour TWA limit of 100 ppm for dipropylene glycol methyl ether (DPGME), with a skin notation. The ACGIH recommends a TLV-TWA of 100 ppm and a TLV-STEL of 150 ppm, with a skin notation, for this colorless liquid with a mild, pleasant, ethereal odor and a bitter taste. OSHA proposed to retain the 8-hour permissible exposure limit of 100 ppm TWA, to add a 150-ppm STEL, and to retain the skin notation for dipropylene glycol methyl ether. NIOSH (Ex. 8-47, Table N1) concurs that these limits are appropriate, and the final rule establishes these limits.

Intact dogs receiving intravenous injections of DPGME exhibited central nervous system depression and died as a result of respiratory failure (Shideman and Procita 1951/Ex. 1-667). Rowe and associates (1954/Ex. 1-435) reported a single acute oral LD_{50} for rats of 5.4 ml/kg. Even at the highest levels tested (not further specified), no single application of DPGME to the skin of rabbits was lethal, although some narcosis and transient weight loss did occur. However, a significant number of deaths occurred in a group of rabbits treated with 65 repeated dermal applications containing DPGME concentrations of 3 ml/kg or higher during a 90-day period. Four animal species, including the monkey, were exposed repeatedly to seven-hour daily inhalation exposures of between 300 and 400 ppm DPGME; the animals exhibited narcosis and changes in the lung and liver (Rowe, McCollister, Spencer et al. 1954/Ex. 1-435).

Humans inhaling DPGME concentrations of 300 to 400 ppm judged this level to be very disagreeable, but 100 ppm was tolerable and, in the opinion of the authors, was unlikely to produce organic injury (Rowe, McCollister, Spencer et al. 1954/Ex. 1-435). Patch tests on the skin of 250 human subjects produced neither irritation nor sensitization (ACG1H 1986/Ex. 1-3, p. 221). Humans exposed to DPGME vapor concentrations at levels between 50 to 2000 ppm experienced eye, nose, and throat irritation before the onset of CNS impairment, which occurred at 1000 ppm in one of two subjects (Stewart, Baretta, Dodd, and Torkelson 1970/Ex. 1-379).

NIOSH (Ex. 150, Comments on Dipropylene Glycol Monomethyl Ether) reported that it is developing a criteria document on the glycol ethers; NIOSH submitted recent toxicity data on DPGME, including the following: rats and mice inhaling concentrations of 50, 140, or 330 ppm DPGME six hours/day for nine days showed increased liver weights (at 50 and 140 ppm for the rat and at 330 ppm for the mouse), but no effects were observed when rats inhaled 15, 50, or 220 ppm DPGME six hours/day, five days/week for 13 weeks (Landry and Yano 1984, as cited in Ex. 150). NIOSH also reported results of a 1985 study by Miller et al. indicating that DPGME is metabolized via the same routes to the same types of metabolites—propylene glycol, and sulfate and glucuronide conjugates of DPGME—as previously identified for PGME (1-methoxy-2-propanol) (Miller, Hermann, Calhoun et al. 1985, as cited in Ex. 150). The Landry and Yano study (1984, as cited in Ex. 150) further indicated that at the concentrations tested, DPGME exerted no teratogenic or reproductive effects (NIOSH/Ex. 150, Comments on Dipropylene Glycol Monomethyl Ether).

The ARCO Chemical Company (Ex. 3-740) questioned the appropriateness of a skin notation for this substance. In response to ARCO, the Agency notes that DPGME, applied essentially according to the Draize method, is absorbed in sufficient quantities through rabbit skin to cause transient narcosis, although the absorption rate was not considered acutely dangerous (*Patty's Industrial Hygiene and Toxicology,* 3rd rev. ed., Vol. 2C, p. 3990, Clayton and Clayton 1982). Topical administration of 10 mg/kg DPGME five times per week for 13 weeks to shaved rabbit skin caused six deaths among seven animals (*Chemical Hazards of the Workplace,* 2nd ed., p. 221, Proctor, Hughes, and Fischman, 1988). To date, there are no human data demonstrating that dermal contact with DPGME is without a significant adverse health risk; therefore, in accordance with the policy

described in Section VI.C.18, OSHA finds that the available evidence does not meet the criterion for deleting an existing skin notation.

In the final rule, OSHA is retaining a PEL of 100 ppm TWA and adding a STEL of 150 ppm for dipropylene glycol methyl ether; the skin notation is retained. The Agency concludes that this combined limit will substantially reduce the significant risks of central nervous system effects and irritation, which constitute material health impairments, that exist when workers are exposed to DPGME for short periods above the 100-ppm PEL.

n-HEXANE

CAS: 110-54-3; Chemical Formula: $CH_3(CH_2)_4CH_3$
H.S. No. 1200

OSHA's former PEL for n-hexane was 500 ppm. The ACGIH has a 50-ppm TWA limit for this substance, and the NIOSH REL is 100 ppm as a 10-hour TWA. OSHA proposed a limit of 50 ppm TWA for n-hexane, and the final rule establishes this limit. NIOSH (Ex. 8-47, Table N1) concurs that a PEL of 50 ppm is appropriate for n-hexane. Normal hexane is a clear, volatile liquid.

n-Hexane has been shown to produce distal axonopathy in both experimental animals and humans; it is metabolized to 2,5-hexanedione (2,5-HD), which is thought to be the causative agent of most of the adverse neurological effects observed after exposure to hexane (Schaumburg, Spencer, and Thomas 1983/Ex. 1-228).

In the preamble to the proposed rule, OSHA asked:

> Does the most current scientific information generally support acceptance of the hypothesis that the C_{5-8} alkanes are not equally toxic because a metabolite of n-hexane exhibits unique neurotoxic properties?

Several commenters (Exs. 3-593, 3-1246, and 124; Tr. III, pp. 109-110) responded to this question, and their detailed responses are discussed in Section V of this preamble, Summary of Commenters' Responses to NPRM Questions.

The C_{5-8} alkanes include pentane, n-hexane, the hexane isomers, n-heptane, octane, and the refined petroleum solvents. Whether all of these alkanes exhibit the same degree of toxicity or whether one (or more) is uniquely toxic has a direct bearing on the appropriate exposure limits for these substances. Based on a thorough review of the chemical and toxicological literature and the responses of these commenters, OSHA has determined that n-hexane is uniquely toxic to the peripheral nervous system. The Agency finds that 2,5-hexanedione (2,5-HD), a metabolite of n-hexane, is likely to be responsible for this unique toxicity, and the American Petroleum Institute (Ex. 124) agrees with this finding. NIOSH (Tr. III, pp. 109-110), on the other hand, is of the opinion that any ketone or related chemical that can be metabolized to a gamma diketone has the potential to cause peripheral neuropathy. However, representatives of the Texaco Company (Ex. 3-1246) agree with OSHA that n-hexane is uniquely toxic because its toxicity is mediated by 2,5-HD.

The ACGIH established a TLV of 50 ppm for this substance, based primarily on studies (Miyagaki 1967/Ex. 1-198; Inoue, Takeuchi, Takeuchi et al. 1970/Ex. 1-75) showing peripheral neuropathies at exposure levels as low as 210 ppm. NIOSH based its 100-ppm REL on the same studies as those cited by the ACGIH (Miyagaki 1967/Ex. 1-198; Inoue, Takeuchi, Takeuchi et al. 1970/Ex. 1-75). NIOSH reasoned as follows:

> The absence of definitive epidemiologic or toxicologic evidence makes it difficult to determine how much lower the environmental limit should be. Professional judgment suggests [that] a TWA concentration of 350 mg/m³ (100 ppm) offers a sufficient margin of safety to protect against the development of chronic nerve disorders in workers (NIOSH 1977a/Ex. 1-233, p. 74).

The adverse neurological effects of hexane exposure are manifested as both sensory and motor dysfunctions. Initially, there is a symmetric sensory numbness of the hands and feet, with loss of pain, touch, and heat sensation. Motor weakness of the toes and fingers is often present; as the neuropathy becomes more severe, weakness of the muscles of the arms and legs may also be observed (Schaumburg, Spencer, and Thomas 1983/Ex. 1-228). There are no known conditions that predispose an individual to hexane neurotoxicity (Schaumburg, Spencer, and Thomas 1983/Ex. 1-228). The onset of neurological symptoms may not be evident for several months to a year after the beginning of exposure. Recovery may be complete, but severely exposed individuals often retain some degree of sensorimotor deficit.

OSHA received comments on n-hexane from several participants, including NIOSH, the National Cotton Council, the American Petroleum Institute, the Corn Refiners Association, the AFL-CIO, and the United Auto Workers. Two commenters, the National Cotton Council (Tr. pp. 9-45 to 9-47) and the Corn Refiners Association (Ex. 177), stated that the revised PEL for n-hexane would impact their members, but did not provide further detail.

Some commenters (Exs. 194 and 197: Tr. pp. 3-290 to 3-293) urged OSHA to regulate all of the refined petroleum solvents on the basis of neurotoxicity. For example, the AFL-CIO recommended a 10-ppm PEL for all such solvents, and Dr. Franklin Mirer of the United Auto Workers described feasible controls that could be used, in his opinion, to achieve this level. Dr. Philip Landrigan (Tr. pp. 3-290 to 3-293) described the neurotoxic effects of exposure to any of the refined petroleum solvents. In response to these commenters, OSHA notes that it is reducing the limits for a number of these solvents in this rulemaking; however, the scale of this undertaking is such that OSHA was unable to perform the detailed analysis necessary to evaluate the health effects, risks, and feasibility for all of the solvents in this large group of substances.

The dose-response relationship for n-hexane exposure in humans is not well defined, although it is clear that the severity of the resulting neuropathy increases as the exposure level of n-hexane increases. A number of studies have shown a consistent relationship between exposure levels of 500 ppm (OSHA's former exposure limit) to 2000 ppm and the development of characteristic peripheral neuropathies (Yamamura 1969, as cited in ACGIH 1986/Ex. 1-3, p. 305; Yamada 1967/Ex. 1-192). Neuropathic effects have also been shown to occur at levels between 210 and 500 ppm (Takeuchi, Maluchi, and Takagi 1975/Ex. 1-217).

Reports of effects occurring at levels of 210 to 500 ppm indicate that the former OSHA PEL of 500 ppm was not adequate to protect exposed workers from adverse sensorimotor neuropathic effects, and exposure at this level thus represents a significant risk to workers. The decreased sensitivity to pain, touch, and temperature associated with n-hexane exposure can also make a worker more susceptible to injuries and accidents. Further, the delayed onset of a clinical response, which is typical of hexane exposure, increases the probability that exposure will continue until irreversible effects occur.

Both the presence of peripheral neuropathies at 210 ppm and the delay in onset of neurological symptoms indicate that workers exposed at levels above the new limit are at significant risk of developing these symptoms. OSHA therefore establishes a PEL of 50 ppm TWA for n-hexane. The Agency concludes that this PEL will substantially reduce the significant risk

of peripheral neurophathies and other adverse neuropathic effects, which constitute material impairments of health and are associated with the exposures permitted at levels above the new limit.

2-HEXANONE (METHYL n-BUTYL KETONE)

CAS: 591-78-6; Chemical Formula: $CH_3CO-CH_2CH_2CH_2CH_3$
H.S. NO. 1202

OSHA's former PEL for 2-hexanone was 100 ppm TWA; the NIOSH REL is a 1 ppm (10-hour) TWA; and the ACGIH recommends a TLV-TWA of 5 ppm. The Agency proposed, and the final rule establishes, a permissible exposure limit of 5 ppm as an 8-hour TWA for 2-hexanone. 2-Hexanone is a colorless, volatile liquid with a characteristic acetone-like odor that is more pungent than that of acetone.

Industrial exposure to 2-hexanone causes distal neuropathy manifesting as interference with motor and sensory function; even in cases characterized by minimal intensity, electrodiagnostic abnormalities were seen (ACGIH 1987/Ex. 1-16). In animals, exposure to 2-hexanone causes axonal swelling and thinning of the myelin sheath. A metabolite of 2-hexanone, 2,5-hexanedione, appears to be responsible for the neural damage; this same metabolite is formed when n-hexane (discussed above) is metabolized. Exposures of rats, cats, dogs, monkeys, hens, and guinea pigs to 2-hexanone have resulted in peripheral neuropathies (O'Donoghue 1985). Krasavage, O'Donoghue, and Terhaar (1978) reported that 2,5-hexanedione is 3.3 times more neurotoxic than 2-hexanone and 38 times more neurotoxic than n-hexane in rats. Thus, 2-hexanone would be approximately eleven times more neurotoxic than n-hexane in rats.

The limit of 5 ppm TWA for 2-hexanone recommended by the ACGIH is based on the results of several studies. These include studies showing evidence of peripheral neuropathy at levels of 50 ppm and above after exposures lasting six months or more (Johnson, Anger, Setzer et al. 1979/Ex. 1-984; Streletz, Duckett, and Chambers 1976/Ex. 1-1067). Another study identified 2,5-hexanedione (the metabolite believed responsible for neurotoxic effects) in the serum of humans after a one-day exposure to 50 ppm (DiVincenzo, Kaplan, and Dedinas 1976/Ex. 1-1049).

The NIOSH REL for 2-hexanone of 1 ppm (10-hour TWA) is based on an epidemiologic study describing an outbreak of neurologic disease among workers in a plant that manufactures printed fabrics (Allen, Mendall, Billmaier et al. 1975/Ex. 1-80). This study reported that a screening of 1,157 exposed workers revealed 86 verified cases of distal neuropathy. 2-Hexanone was suspected of being the neurotoxicant because it had only recently been introduced into the process (Allen, Mendall, Billmaier et al. 1975/Ex. 1-80). When recommending its limit, NOISH relied on an industrial hygiene survey of the plant conducted by Billmaier, Yee, Allen et al. (Ex. 1-76) in 1974, which showed that 2-hexanone concentrations near the textile printing machines ranged from 1 to 156 ppm (10-minute area samples). After reviewing this evidence, NIOSH concluded that 1 ppm could not be considered a no-effect level for 2-hexanone-induced neuropathy, and NIOSH (Ex. 8-47, Table N2; Tr. p. 3-86) continues to recommend a limit of 1 ppm TWA for 2-hexanone. The AFL-CIO (Ex. 194) also supports the adoption of the lower NIOSH REL. Dr. Franklin Mirer of the AFL-CIO (Ex. 197) described controls for use in workplaces where solvents present exposure problems.

The ACGIH (1987/Ex. 1-16) stated that interpretation of the results of the Billmaier, Yee, Allen et al. (1974/Ex. 1-76) study was complicated because the exposure measurements reported in the study had been taken after the outbreak of neuropathic effects had occurred. In addition, the ACGIH pointed out that Billmaier and colleagues (1974/Ex. 1-76) found poor work practices at the plant (gloves were rarely used, employees washed their hands with solvent, etc.); thus, dermal exposures may have contributed substantially to the outbreak.

Both human and animal studies show the development of disease at exposure levels well below the former 100-ppm PEL, clearly indicating the need to reduce this significant risk. In the final rule, OSHA is establishing a 5-ppm (8-hour TWA) PEL for 2-hexanone. The Agency concludes that this limit will substantially reduce the significant risk of distal neuropathy, which constitutes a material impairment of health and has been demonstrated to occur at concentrations above the new limit.

IRON PENTACARBONYL

CAS: 13463-40-6; Chemical Formula: $Fe(CO)_5$
H.S. No. 1216

OSHA previously had no exposure limit for iron pentacarbonyl. The ACGIH has a TLV-TWA of 0.1 ppm with a TLV-STEL of 0.2 ppm, measured as iron, for this highly flammable, oily, colorless to yellow liquid. The Agency proposed, and the final rule establishes, permissible exposure limits of 0.1 ppm TWA and 0.2 ppm STEL for iron pentacarbonyl, measured as Fe. NIOSH (Ex. 8-47, Table N1) concurs that these limits are appropriate.

In studies of rats, iron pentacarbonyl has been reported to have approximately one-third the acute toxicity of nickel carbonyl (for which the ACGIH has recommended a TLV of 0.05 ppm TWA) (Sunderman, West, and Kincaid 1959/Ex. 1-384). In 1970, Gage found that a 5.5-hour exposure at 33 ppm caused fatalities in three of eight rats; four of eight animals died after two 5.5-hour exposures at 18 ppm. At 7 ppm, no ill effects were observed in rats exposed 18 times in 5.5 hours (Gage 1970/Ex. 1-318). There are no reports of long-term dose-response exposure studies in laboratory animals, and no evidence exists that iron pentacarbonyl is carcinogenic in either humans or animals (ACGIH 1986/Ex. 1-3, p. 327).

Immediate symptoms of acute exposure to high concentrations of iron pentacarbonyl include headache and dizziness, followed in 12 to 36 hours by fever, cyanosis, cough, and shortness of breath. Another clinical effect of overexposure to this substance is lung injury, and degenerative changes in the central nervous system have also been reported (ACGIH 1986/Ex. 1-3, p. 327). No comments (other than NIOSH's) on the health effects of iron pentacarbonyl were submitted to the rulemaking record.

In the final rule, OSHA establishes a permissible exposure limit of 0.1 ppm TWA and a STEL of 0.2 ppm for iron pentacarbonyl. The Agency concludes that these limits will protect workers from the significant risks of material health impairment in the form of headache, dizziness, fever, dyspnea, cyanosis, pulmonary injury, and central nervous system effects, which are potentially associated with exposures at levels above the new limits.

MANGANESE FUME

CAS: 7439-96-5; Chemical Formula: MnO
H.S. No. 1236a

OSHA previously had a ceiling limit of 5 mg/m^3 for manganese fume, measured as manganese. Because of this substance's potential for damage to the lungs and central nervous system, the ACGIH recommends an 8-hour TWA of 1 mg/m^3 and a 3-mg/m^3 STEL for manganese fume. These limits were proposed and are now established by the final rule. NIOSH (Ex. 8-47, Table N1) concurs that these limits are appropriate.

Symptoms of manganese poisoning range from sleepiness and weakness in the legs (Fairhall 1957a, as cited in ACGIH 1986/Ex. 1–3, p. 354) to difficulty in walking and uncontrolled laughter (Fairhall and Neal 1943, as cited in ACGIH 1986/Ex. 1–3, p. 354). Health surveys of employees exposed to manganese fume have demonstrated a high incidence of pneumonia in these workers (Davies 1946, as cited in ACGIH 1986/Ex. 1–3, p. 354). Tanaka and Lieben (1969/Ex. 1–388) found seven cases of pneumonia and 15 borderline cases of pneumonia among 144 workers exposed to manganese dust or fume concentrations greater than 5 mg/m^3; three of these cases were associated with fume rather than dust exposure. Those workers exposed to fume levels below 5 mg/m^3 exhibited no signs of pneumonia. In a separate study by Smyth, Ruhf, Whitman, and Dugan (1973/Ex. 1–990), three cases of manganese poisoning were detected among 71 employees exposed to levels of 13.3 mg/m^3 manganese fume.

OSHA received several comments on manganese fume and dust (Exs. 3–189, 3–673, 3–675, 3–829, 8–22, and 129). Some commenters stated that reducing the PEL for manganese fume would have a large impact on their industries but did not provide any details (Exs. 3–673, 3–.675, and 8–22). Chemetals, Inc., a manufacturer of manganese products, supports the reduction in the PEL for manganese *fume* from a ceiling of 5 mg/m^3 to an 8-hour TWA of 1 mg/m^3 and a STEL of 3 mg/m^3. According to Chemetals:

[We] agree that the fumes of metals and their compounds have higher toxicities than the dusts * * * and that a time-weighted average is more appropriate for manganese (Ex. 3–189, p. 2).

However, Chemetals urged OSHA to also revise the Agency's limit for manganese dust from a ceiling to an 8-hour TWA (Ex. 3–189). OSHA did not propose a change to its existing 5-mg/m^3 ceiling limit for manganese dust. In response to this comment, OSHA notes that manganese dust is not a substance that is included in this rulemaking; the Agency did not propose to regulate manganese dust and is not revising its limits for this substance in the final rule (see the preamble section entitled "Boundaries to the Regulation").

One other commenter, the Specialty Steel Industry of the United States (Ex. 3–829), stated that, in its opinion, there was no basis for reducing OSHA's former PEL for manganese fumes or for supplementing this limit with an STEL. OSHA does not agree with the views of this commenter, because exposures to these fumes have been demonstrated to cause toxic effects in both humans and animals. Workers exposed to manganese fumes developed pneumonia (Tanaka and Lieben 1969/Ex. 1–388), and Stokinger (1981f, in *Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., Vol. 2A, p. 1767) reports that the 1-mg/m^3 limit "is supported by the finding in animals that the higher oxides are more toxic, and the report of an occasional case of Mn poisoning in susceptible workers exposed to ferro Mn fumes around the 1-mg/m^3 limit."

Based on a review of all of the record evidence, the final rule establishes a 1-mg/m^3 TWA and a 3-mg/m^3 STEL for manganese fume. The Agency concludes that both a TWA limit and a STEL are required to protect employees from the significant risks of manganese poisoning, lung damage, and pneumonia, all of which constitute material health impairments, associated with exposure to these fumes.

MANGANESE CYCLOPENTADIENYL TRICARBONYL

CAS: 12079–65–1; Chemical Formula: $C_5H_5—Mn(CO)_3$
H.S. No. 1237

OSHA formerly had no limit for exposure to manganese cyclopentadienyl tricarbonyl (MCT). The ACGIH has a TLV–TWA of 0.1 mg/m^3 (measured as manganese), with a skin notation. The Agency proposed, and the final rule establishes, a permissible exposure limit of 0.1 mg/m^3 TWA (measured as manganese), with a skin notation, for this substance. NIOSH (Ex. 8–47, Table N1) concurs that these limits are appropriate.

A Russian study reported that a single two-hour exposure to MCT at 120 mg/m^3 was fatal to 80 percent of albino rats, although rabbits, guinea pigs, and rats survived a single two-hour exposure at 20 to 40 mg/m^3. Chronic exposure of rats for 11 months at levels averaging 1 mg/m^3 for four hours daily showed delayed effects (seven months from onset of exposure) of neuromuscular excitability, evidence of kidney damage, and decreased resistance to infection (Arkhipova, Tolgskaya, and Kochetkova 1965/Ex. 1–1046). The tails of 10 white mice were dipped in a gasoline mixture containing 1 gram MCT per 100 ml; a second group of mice had their tails immersed in gasoline without MCT. An equal number of fatalities were observed in the gasoline plus MCT and gasoline-only groups after four or five two-hour applications, and all tails exhibited necrosis. The authors concluded that these effects were caused by the gasoline and not by the MCT (Arkhipova, Tolgskaya, and Kochetkova 1965/Ex. 1–1046). Further studies in rabbits showed that MCT applied dermally as an oil emulsion caused irritation of the skin. These authors also investigated the dermal toxicity of MCT solutions in tetrahydrofuran versus solutions of tetrahydrofuran in oil. All animals whose tails had been dipped in the hydrofuran solution of MCT died within an hour, while animals whose tails had been dipped in pure tetrahydrofuran did not (Arkhipova, Tolgskaya, and Kochetkova 1965/Ex. 1–1046). The same authors concluded that MCT is toxic at low concentrations, has cumulative properties, affects the nervous system, is irritating to the skin, and causes early histological changes in the respiratory tract.

More recent reports describe MCT-induced pulmonary edema and convulsions in the rat (Penney, Hogberg, Traiger, and Hanzlik 1985/Ex. 1–431). The ED_{50_s} for convulsions were 32 mg/kg orally and 20 mg/kg intraperitoneally; LD_{50_s} were 24 mg/kg orally and 14 mg/kg intraperitoneally. Necrosis of the bronchiolar tissue and pulmonary parenchymal damage were seen in mice and rats given intraperitoneal doses (Haschek, Hakkinen, Witschi et al. 1982/Ex. 1–1083). No comments other than NIOSH's werre received on MCT.

OSHA has concluded that occupational exposure to MCT poses a risk of neuropathic effects, kidney damage, skin irritation, pulmonary edema, and tissue damage, which together constitute material health impairments. The Agency is therefore establishing an 8-hour TWA PEL of 0.1 mg/m^3 for manganese cyclopentadienyl tricarbonyl, with a skin notation, to protect workers against the significant risk of these effects, which have been shown to occur at levels above the new standard.

MANGANESE TETROXIDE

CAS: 1317–35–7; Chemical Formula: Mn_3O_4
H.S. No. 1238

OSHA previously had no exposure limit for manganese tetroxide (compound and fume). The ACGIH recommends a TLV–TWA of 1 mg/m^3, measured as manganese, for this brownish-black powder and its dust and fume. The Agency proposed a PEL of 1 mg/m^3 TWA for manganese tetroxide, measured as Mn, and the final rule establishes this limit. Ferromanganese fume has been determined by X-ray diffraction analysis to consist primarily of manganese tetroxide.

Findings from a Russian study indicated that intratracheal suspensions of manganese oxide, manganese dioxide, and manganese tetroxide particles (particle size less than 3 μm) produced pneumonitis and other similar

pulmonary effects in rats (Levina and Robachevskiau 1955/Ex. 1–1041). These investigators also determined that manganese tetroxide has a greater toxicity than do the lower oxides of manganese and that freshly prepared oxides were more potent than those stored for six months to one year.

Two cases of manganese fume poisoning were reported in a plant where concentrations were between 2.7 and 4.7 mg/m³ (Whitlock, Amuso, and Bittenbender 1966/Ex. 1–455), but other investigators have questioned these air sampling results and believe that exposures to manganese tetroxide concentrations of 5 mg/m³ or less cause no harmful effects (Whitman and Brandt 1966/Ex. 1–1103). In a seven-year study, Smyth and co-workers (1973/Ex. 1–990) investigated chronic manganese poisoning in workers exposed to both ferromanganese fumes and dust. Five of 71 employees suffered from chronic manganism; of these five cases, three resulted from fume exposure and two from dust exposure. Two of the three fume-exposure victims were exposed over a five-year period to an estimated average ferromanganese concentration of 13.3 mg/m³; however, the third victim worked in an operation where air concentrations of manganese were less than 1 mg/m³, which suggests that certain individuals may be hypersusceptible to manganese poisoning. The dust-exposed victims worked in areas where air concentrations were in the range of 30 to 50 mg/m³ throughout the study period (Smyth, Ruhf, Whitman, and Dugan 1973/Ex. 1–990).

Martonik (1976, as cited in ACGIH 1986/Ex. 1–13, p. 357) reported that the fume of manganese has greater toxicity than does the dust. During a two-year period, at least one case of acute manganese poisoning was documented at a fume concentration level of 7.5 mg/m³, and another case at the same welding operation may also have involved manganism.

OSHA received two comments on this substance, one from NIOSH (Ex. 8–47; Tr. p. 3–86), and one from Chemetals, a manganese manufacturer (Ex. 3–189). NIOSH (Ex. 8–47, Table N2) does not concur with the limits being established by OSHA. NIOSH (Ex. 8–47, Table N2) notes that, based on the results of the Smyth and co-workers study (1973/Ex. 1–990), the 1-mg/m³ PEL being established by OSHA "may not be protective, especially to the potentially sensitive individual." In response to this NIOSH comment, OSHA states that the Agency intends to monitor the literature on manganese tetroxide closely in the future to determine whether the new limit for this substance is adequately protective.

Chemetals (Ex. 3–189) asked OSHA to promulgate separate limits for the dust and fume of manganese tetroxide based on the relative toxicities of these two particulate forms. OSHA recognizes that some information in the literature (including some discussed above) points to the greater toxicity of the fume and that fumes are generally the more toxic form of particulate. However, the Agency notes that intratracheal suspensions of manganese tetroxide dust caused pneumonitis and other pulmonary effects in Russian workers (Levina and Robachevskiau 1955/Ex. 1–1041) and that several cases of manganism have been caused by dust exposure (Smyth, Ruhf, Whitman, and Anger 1973/Ex. 1–990). The Agency believes it prudent not to distinguish at this time between the dust and the fume but to set the TWA PEL at a level that will protect against the effects of exposure to both forms of particulate.

OSHA is establishing a 1-mg/m³ 8-hour TWA for manganese tetroxide (compound and fume). The Agency concludes that this limit will provide protection against the significant risks of material health impairment in the form of chronic manganese poisoning, pneumonitis, and other respiratory effects that are associated with exposure to manganese tetroxide at levels above 1 mg/m³.

MERCURY (ARYL AND INORGANIC COMPOUNDS)

CAS: 7439–97–6; Chemical Formula: Hg
H.S. No. 1240

The former OSHA limit for all inorganic forms of mercury (Hg) was 0.1 mg/m³ as a ceiling limit, as indicated on Table Z–2; this limit was adopted from ANSI standard Z37.8 (1943). In a compliance directive issued in 1978 (OSHA Instruction CPL 2–2.6), however, the Agency stated that the PEL for inorganic mercury should be expressed as an 8-hour TWA of 1 mg/10 m³ (0.1 mg/m³) rather than as a ceiling. The ACGIH has a 0.1-mg/m³ TLV–TWA for aryl and inorganic mercury compounds. NIOSH (1973b, as cited in ACGIH 1986/Ex. 1–3, p. 358) has recommended a 0.05-mg/m³ limit as an 8-hour TWA. OSHA proposed to return to its 0.1-mg/m³ ceiling limit (measured as mercury) and this limit is being established, together with a skin notation, in the final rule. This action cancels the 1978 compliance directive.

In 1971, shortly after OSHA had adopted the 0.1-mg/m³ ceiling, the ACGIH reduced the TLV–TWA for all forms of mercury, including the inorganic compounds, to 0.05 mg/m³. ANSI also reduced its standard to 0.05 mg/m³ in 1972, and NIOSH recommended the same limit in 1973. The 0.05-mg/m³ limit was based largely on the study of Smith, Vorwald, Patil, and Mooney (1970/ Ex. 1–373) of workers exposed to mercury levels ranging from less than 0.1 to 0.27 mg/m³ in chlor-alkali plants. The authors reported a significant dose-related increase in the incidence of weight loss, tremors, abnormal reflexes, nervousness, and insomnia among workers exposed to concentrations of 0.1 mg/m³ or more. There were slight increases in incidences of insomnia and loss of appetite among workers exposed to 0.1 mg/m³ or less. Smith, Vorwald, Patil, and Mooney (1970/Ex. 1–373) concluded that a limit of 0.1 mg/m³ contained little or no margin of safety. Other studies (Bidstrup, Bonnell, Harvey, and Locket 1951/Ex. 1–1014; Turrian, Grandjean, and Turrian 1956, as cited in ACGIH 1986/ Ex.1–3, p. 358) have also reported symptoms of mercury poisoning among workers exposed below 0.1 mg/m³. The 0.05-mg/m³ limit established by the ACGIH, ANSI, and NIOSH also follows the 1968 recommendation of an international committee (Permanent Commission & International Association on Occupational Health 1968, as cited in ACGIH 1986/Ex. 1–3, p. 358).

In 1980, the ACGIH revised its recommended TLV for aryl and inorganic mercury compounds to 0.1 mg/m³. In revising this limit, the ACGIH cited discrepancies in the literature regarding the ratio of blood and urinary mercury levels to airborne concentrations of mercury (Bell, Lovejoy, and Vizena 1973/Ex. 1–1078; Stopford et al. 1978/Ex. 1–1100). These studies reported lower ratios of mercury body burden to airborne concentration when personal sampling is used rather than area sampling. According to Bell, Lovejoy, and Vizena (1973/Ex. 1–1078), the lower ratio results because mercury exposure measurements are generally found to be higher when personal sampling is conducted, presumably as a consequence of contamination of clothing. The ACGIH argued that the 0.05-mg/m³ limit may be too stringent to apply when personal sampling is conducted. The ACGIH also stated that, in contrast to the effects of elemental or alkyl mercury, little mercury is deposited in the brain following exposure to aryl or inorganic mercury compounds. Based on this reasoning, the ACGIH adopted the higher 0.1-mg/m³ TLV–TWA for aryl and inorganic compounds of mercury. However, the

ACGIH (1986/Ex. 1-3, p. 358) also noted that, although central nervous system effects are less likely to occur from exposure to mercury salts than from other forms of mercury, the risk of renal and oral effects would "presumably be just as great." Therefore, they cautioned that the higher limit for mercury salts "may be subject to debate" (ACGIH 1986/Ex. 1-3, p. 358).

Robert G. Smerko, President of the Chlorine Institute (Ex. 3-828; Tr. pp. 10-171 to 10-177), reviewed the pharmacologic evidence on the various forms of mercury. He concluded that, contrary to the statement by the ACGIH, there is little difference in brain deposition between elemental mercury and mercury compounds:

> The ACGIH differentiated between aryl mercury and inorganic salts of mercury in comparison with elemental mercury vapor * * *. While this is true for large doses of mercury, it overlooks for the fact that absorbed elemental mercury is rapidly oxidized in the blood as reported by Clarkson et al. (1967) * * *.
>
> Only when the rate of absorption exceeds the rate at which the body can oxidize mercury between the point of absorption and the brain does elemental mercury behave differently than aryl mercury and inorganic salts of mercury at the blood-brain barrier (Ex. 3-828, p. 7).

Mr. Smerko requested that OSHA retain its 0.1-mg/m^3 limit as an 8-hour TWA, but supplement the limit with requirements for monitoring of urinary mercury levels; Dr. James Melius of the New York State Department of Health (Tr. pp. 11-105, 11-106, 11-109 to 11-111) also stressed the importance of biological monitoring for mercury. This issue is discussed below, for mercury vapor.

In light of this information, which counters the basis for the 0.1-mg/m^3 ACGIH TLV, and given the caution expressed by the ACGIH (1986/Ex. 1-3, p. 358) that the 0.1-mg/m^3 TWA limit "may be subject to debate," OSHA concludes that the PEL for aryl and inorganic mercury should be 0.1 mg/m^3 as a ceiling limit, as indicated in Table Z-2. The health studies cited above indicate that reducing the limit for these forms of mercury will ensure that employees are not at significant risk of adverse neuropathic effects from exposure to these forms of mercury and their compounds. Accordingly, OSHA is establishing a 0.1-mg/m^3 ceiling limit (measured as mercury) for aryl and inorganic mercury and compounds. Dr. Grace Ziem (Ex. 46) supported lowering the mercury limit in the final rule, and the Workers Institute for Safety and Health urged OSHA to restore the ceiling (Ex. 116). OSHA is also adding a skin notation to alert employers to the fact that mercury readily penetrates the skin, causing systemic poisoning; several cases of poisoning from this route have been reported (NIOSH 1973b, as cited in ACGIH 1986/Ex. 1-3, p. 358; Ex. 3-828).

One commenter, Stuart B. Cooper, Manager of Regulatory Affairs for Cosan Chemical Corporation (Ex. 3-1162), expressed concern that establishing different PELs for inorganic mercury and elemental mercury vapor would confuse the interpretation of monitoring results in cases in which more than one form of mercury is present. He suggested that, where one form of mercury is present to a greater extent than another form, only the PEL for the predominant form should apply. OSHA agrees that, for some workplaces, such an approach may be reasonable; however, since the limits for inorganic mercury and mercury vapor differ, both in numerical value and required sampling duration, OSHA believes that employers may wish to conduct both ceiling and full-shift air sampling in cases where both forms of mercury are present.

In the final rule, OSHA is establishing a PEL of 0.1 mg/m^3 as a ceiling for aryl mercury and the inorganic compounds of mercury, along with a skin notation. The Agency concludes that these limits are necessary to protect exposed workers from the significant risks of neuropathy and systemic toxicity (both of which constitute material impairments of health) that are associated with exposure to these substances at higher levels.

MERCURY (VAPOR)

CAS: 7439-97-6; Chemical Formula: Hg
H.S. No. 1241

OSHA formerly had a TWA limit of 0.1 mg/m^3 for mercury (including vapor). The ACGIH recommends a TLV-TWA of 0.05 mg/m^3 for mercury vapor, measured as mercury, and a skin notation. NIOSH has a REL of 0.05 mg/m^3 as an 8-hour TWA. The Agency proposed a PEL of 0.05.mg/m^3 TWA mercury and its vapor, measured as Hg, and the final rule establishes this limit, also with a skin notation. NIOSH (Ex. 8-47, Table N1) concurs that this limit is appropriate. Elemental mercury is a silvery, oderless, heavy liquid.

Inhalation of high concentrations of mercury vapor for relatively brief periods can cause pneumonitis, bronchitis, chest pain, dyspnea, coughing, stomatitis, gingivitis, salivation, and diarrhea (NIOSH 1973b, as cited in ACGIH 1986/Ex. 1-3, p. 359; Ashe, Largent, Dutra et al. 1953/Ex. 1-502). Chronic mercurialism is manifested by central nervous system effects, including tremor, a variety of neuropsychiatric disturbances, and loss of appetite (Kazantzis 1968, as cited in ACGIH 1986/Ex. 1-3, p. 359; Smith, Vorwald, Patil, and Mooney 1970/Ex. 1-373).

Severe organ damage occurred in rabbits exposed for four hours to an average vapor concentration of 28.8 mg/m^3. Damage was observed in the kidneys, liver, brain, heart, lungs, and colon (Ashe, Largent, Dutra et al. 1953/Ex. 1-502). A study by Smith, Vorwald, Patil, and Mooney (1970/Ex. 1-373) of 567 male workers exposed to a mean exposure level of 0.065 mg/m^3 (S.D. $\pm$ 0.085) showed a significant dose-related increase in the incidence of weight loss, tremors, abnormal reflexes, nervousness, and insomnia among workers exposed to 0.1 mg/m^3 or higher. There were slight increases in the incidence of insomnia and loss of appetite among workers exposed to 0.1 mg/m^3 or less. Smith, Vorwald, Patil, and Mooney (1970/Ex. 1-373) concluded that a limit of 0.1 mg/m^3 contained little or no margin of safety. Six of 75 workers regularly exposed to 0.05 to 0.1 mg/m^3 of mercury vapor in a glassware manufacturing plant reported insomnia, and one was found to have tremors (Danziger and Possick 1973/Ex. 1-504). One of 11 workers, employed in a mercury mine or refining plant and exposed at vapor concentrations below 0.1 mg/m^3 had sore gums, loose teeth, or excess salivation (Rentos and Seligman 1968/Ex. 1-523).

NIOSH (1973b, as cited in ACGIH 1986/Ex. 1-3, p. 358) has recommended a 10-hour TWA limit of 0.05 mg/m^3 for inorganic mercury and concluded that hyperactivity, rather than tremor, may be the most typical symptom of chronic mercurialism. Two studies report no evidence of mercury vapor poisoning in industrial settings where characteristic exposures ranged between 0.05 and 0.1 mg/m^3 (Danziger and Possick 1973/Ex. 1-504; McGill, Ladd, Jacobs, and Goldwater 1964/Ex. 1-520).

In workers exposed at levels above 0.1 mg/m^3, toxic symptoms were seen (Rentos and Seligman 1968/Ex.1-523). Turrian, Grandjean, and Turrian (1956, as cited in ACGIH 1986/Ex. 1-3, p. 358) found that 33 percent of workers exposed to the vapor at levels above 0.05 mg/m^3 exhibited hyperexcitability, while only 8 percent of those exposed below that level manifested this symptom. About 20 percent of workers in both groups exhibited tremor. The ACGIH notes that, after exposure to the vapor, "a relatively high percentage of the absorbed mercury remains in the brain," compared with the amount

deposited in the brain after exposure to the aryl and inorganic compounds (ACGIH 1986/Ex. 1-3, p. 359). The ACGIH accordingly recommends a higher TLV-TWA for aryl and inorganic mercury then for mercury vapor (see, however, the discussion of aryl and inorganic mercury above).

Robert G. Smerko, President of the Chlorine Institute (Ex. 3-828), and the Laboratory Products Association (Ex. 135) urged OSHA to retain its 0.1-mg/m^3 PEL and to require urinary mercury analysis in lieu of a reduced PEL because dermal contact with mercury may contribute substantially to its toxicity (Ex. 3-828; Tr. pp. 10-171 to 10-177). Mr. Smerko cited several reports of such effects in his testimony and submission, including reports of poisoning resulting from contact with contaminated clothing. Because dermal contact is a significant route of exposure for mercury, Mr. Smerko commented:

> There is a large probability that air measurements of mercury concentrations (aryl mercury, inorganic salts, or elemental mercury vapor) either overestimate or underestimate the extent of exposure to mercury. The extreme accuracy and precision of the urinary mercury analysis and the amount of work that has been done in correlating urinary mercury concentrations with the presence or absence of effects from exposure to mercury warrant the propsal that a biological standard, or a comprehensive standard that includes an air concentration and urinary mercury concentration, be established for aryl mercury, inorganic salts of mercury, and elemental mercury vapor (Ex. 3-828, p. 9).

Mr. Robert F. Adams, Senior Industrial Hygienist for Occidental Chemical Corporation (Ex. 3-1174), supported the position of the Chlorine Institute on this issue, as did Dr. James Melius of the New York State Department of Health (Tr. pp. 11-105, 11-106, 11-109 to 11-111).

OSHA agrees that prevention of mercury contamination of skin and clothing, as well as the proper handling of contaminated clothing, are essential elements of a program to protect employees from the health hazards of mercury. OSHA also believes that mercury presents one of the rare instances in which a biological-monitoring-based standard may represent an effective and reasonable approach for ensuring worker protection. Margaret Seminario, Associate Director of Occupational Safety, Health, and Social Security for the AFL-CIO, also supported provisions for biological monitoring of mercury (Ex. 194, Appendix 1, p. 3). However, developing such a standard is beyond the scope of this rulemaking, which is being conducted solely to revise OSHA's air contaminant limits.

Despite some of the uncertainties in the studies described above regarding the relationship between airborne exposure levels and health effects, OSHA concludes that the data suggest that the former PEL of 0.1 mg/m^3 is not sufficiently protective. Given the severity of the neuropathic effects caused by mercury poisoning, OSHA finds that a reduction in the airborne limit is necessary to ensure that workers are not at significant risk of mercury-related neuropathic effects. Therefore, OSHA is revising its PEL for elemental mercury vapor to 0.05 mg/m^3 as an 8-hour TWA. In addition, because skin absorption is a significant route of exposure and leads to systemic poisoning, as evidenced by Mr. Smerko's written testimony, OSHA is including a skin notation in the final rule.

OSHA is establishing an 8-hour TWA PEL of 0.05 mg/m^3 TWA for mercury vapor, with a skin notation. The Agency concludes that this limit will substantially reduce the significant risks of acute and chronic mercury poisoning (which constitute material health impairments) that have been demonstrated to occur at exposure levels above 0.05 mg/m^3. The skin notation is added because the vapors of elemental mercury can be readily absorbed through the skin.

MERCURY, (ORGANO) ALKYL COMPOUNDS

CAS: 7439-97-6
H.S. No. 1242

OSHA had a former 8-hour PEL of 0.01 mg/m^3 TWA and a ceiling limit of 0.04 mg/m^3 for the alkyl compounds of mercury. The ACGIH has a TLV-TWA of 0.01 mg/m^3 and a TLV-STEL of 0.03 mg/m,^{3} with a skin notation, for these compounds, measured as mercury. The Agency proposed, and the final rule is establishing, permissible exposure limits of 0.01 mg/m^3 as an 8-hour TWA and 0.03 mg/m^3 as a STEL, with a skin notation, for the alkyl compounds of mercury (measured as Hg). NIOSH (Ex. 8-47, table N1) concurs with these limits. Alkyl mercury compounds include volatile liquids, such as dimethyl and diethyl mercury, as well as many complex salts, which are usually solids.

Alkyl mercury compounds pose greater health hazards than do the inorganic compounds of mercury because they can penetrate the blood-brain barrier and the placenta very quickly. The primary toxic effects associated with exposure to the organic compounds of mercury are injuries to the central and peripheral nervous systems and to the kidneys (Casarett and Doull 1975/Ex. 1-1144). In addition, data concerning mouse and rat exposures to alkyl mercury compounds have demonstrated toxicity to the gastrointestinal system, pancreas, liver, gonads, and cardiovascular system. Suppression of the immune system and impairment of the endocrine system have also been observed (Shakbazyan, Shevchenko, Borisenko et al. 1977/Ex. 1-933). Fatalities in mice have been reported following exposures of 10 to 30 mg/m^3 for 3 to 5 hours (Trakhtenberg 1950/Ex. 1-447).

Methyl mercury is among the most damaging of the alkyl compounds to humans because it accumulates in the body and causes developmental effects (Wilson 1977/Ex. 1-457). A three-month exposure to approximately 1 mg/m^3 diethyl mercury caused death in two individuals (Hill 1943/Ex. 1-786). Another fatal case of alkyl mercury poisoning has also been described (Hook, Lundgren, and Swensson 1954/Ex. 1-333). On the basis of his work with laboratory animals, Trakhtenberg (1950/Ex. 1-447) stated that even a concentration as low as 0.00001 mg/m^3 could not be tolerated by humans on a continuing basis. However, a later study reported no consistent, acute effects of mercury poisoning at air concentrations between 0.01 and 0.1 mg/m^3, despite the fact that brief excursions considerably above this range occurred (Dinman, Evans, and Linch 1958/Ex. 1-311). Organic mercury compounds can be absorbed through the skin (*Dangerous Properties of Industrial Materials,* 7th ed., Sax and Lewis 1989).

Lawrence H. Hecker, representing Abbott Laboratories (Ex. 3-678), objected to the inclusion of a STEL for the alkyl mercury compounds, stating that there is no health basis for such a limit. OSHA believes that both the seriousness of the neurological effects caused by exposure to low levels of alkyl mercury and the ability of alkyl mercury to accumulate in the body necessitate the establishment of a STEL to ensure that the PEL is not exceeded. As discussed in Section VI.C.17 of this preamble, OSHA has determined that a STEL is warranted in instances where extremely hazardous substances are involved.

OSHA is retaining its 8-hour TWA PEL of 0.01 mg/m^3 and adding a 15-minute STEL of 0.03 mg/m^3 for the alkyl compounds of mercury (measured as Hg), with a skin notation. The Agency concludes that exposure to the alkyl mercury compounds poses significant risks of severe neuropathic and other systemic injuries, which constitute material health impairments, and that both the short-term and 8-hour limits are necessary to reduce these risks. OSHA

has added the skin notation to protect against the dermal absorption possible in the absence of a skin notation.

METHYLACRYLONITRILE

CAS: 126-98-7; Chemical Formula: $CH_2=C(CH_3)C=N$
H.S. No. 1251

OSHA previously had no standard for methylacrylonitrile. The ACGIH recommends a 1-ppm TLV-TWA with a skin notation to protect workers who are occupationally exposed to methylacrylonitrile. The Agency proposed, and the final rule establishes, a permissible 8-hour TWA exposure limit of 1 ppm, with a skin notation, for methylacrylonitrile, which is a colorless liquid. NIOSH (Ex. 8-47, Table N1) concurs that these limits are appropriate.

Methylacrylonitrile has been shown to be extremely toxic in animals, both by inhalation and dermal absorption. The dermal LD_{50} in rabbits is 0.35 ml/kg (280 mg/kg) (ACGIH 1986/Ex. 1-3, p. 370). Beagles exposed for 90 days to 13.5 ppm convulsed and lost motor control in their hind limbs. Microscopic brain lesions were detected in one of the dogs. The level at which no effects were detected was determined to be between 3.2 and 8.8 ppm (ACGIH 1986/Ex. 1-3, p. 370). No comments (other than NIOSH's) on the health effects of methylacrylonitrile were submitted to the rulemaking record.

OSHA is establishing a 1-ppm 8-hour TWA PEL and a skin notation for this substance. The Agency concludes this limit will substantially reduce the significant risk of neurological damage (which constitutes a material health impairment) that formerly existed in the absence of an OSHA exposure limit for this substance.

METHLY BROMIDE

CAS: 74-83-9; Chemical Formula: CH_3Br
H.S. No. 1253

OSHA's former PEL for methyl bromide was a 20-ppm ceiling with a skin notation, while the ACGIH limit is 5 ppm as an 8-hour TWA, with a skin notation. NIOSH recommends that the REL for this substance be set at the lowest feasible level. The Agency proposed, and the final rule establishes, a permissible exposure limit of 5 ppm (8-hour TWA), with a skin notation, for methyl bromide. Methyl bromide is a colorless, nonflammable gas with no taste and no odor at low temperatures. At levels above 5 ppm, it has a sweetish odor.

Acute poisoning from methyl bromide is characterized by lung irritation, pulmonary edema, convulsions, and coma. Chronic exposure to low concentrations of methyl bromide generally produces central nervous system effects, including muscle weakness and pain, incoordination, inability to focus one's eyes, and behavioral changes (ACGIH 1986/Ex. 1-3, p. 376; Craft 1983/Ex. 1-196). The onset of neurological signs and symptoms may be delayed for from several hours to a few days after exposure.

Methyl bromide is a gas and is predominantly an inhalation hazard, although there are suggestions that it can also be absorbed through the skin (*Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., Vol. 2B, p. 3443, Clayton and Clayton 1981). A report by Hine (1969/Ex. 1-70) notes that methyl bromide has been responsible for more deaths among occupationally exposed workers in California than have the organophosphates. It is hypothesized that methyl bromide has a greater potential for toxicity than do other organic bromides because its greater lipophilicity provides increased access to the brain.

Various studies demonstrate methyl bromide's toxicity in humans. Ingram (1951/Ex. 1-175) reported ill effects (symptoms not specified) after exposure to methyl bromide at concentrations of 100 ppm. Similar exposure concentrations were also reported by Hine (1969/Ex. 1-70) in a case study of two date packers in California. Johnson, Setzer, Lewis, and Anger (1977/Ex. 1-87) indicated that 34 packers became sick when exposed to an average methyl bromide concentration of 50 ppm, although concentrations in the packing room may have been as high as 100 to 150 ppm during the purging of a fumigation chamber.

Watrous (1942/Ex. 1-275) described nausea, vomiting, and headache in 90 workers who were exposed for two weeks to concentations "generally below" 35 ppm. These symptoms emphasized the need to create a TLV to protect workers from the nausea, vomiting, and headaches (which together constitute material impairments of health) associated with lower levels of exposure. This need is strengthened by the fact that, since these symptoms are usually delayed in onset, workers may not have sufficient warning of this substance's potential neurotoxicity.

The AFL-CIO (Ex. 194, p. A-12) supports the inclusion of methyl bromide in this rulemaking, but notes that it is a potential occupational carcinogen. NIOSH takes the same position and believes that methyl bromide should be addressed in a full section 6(b) rulemaking (Ex. 8-47, Table N6B; Tr. pp. 3-97, 3-98). OSHA shares the concerns of these commenters and intends to monitor the scientific evidence on methyl bromide's toxicity in the future. The Workers Institute for Safety and Health (WISH) (Ex. 116) is of the opinion that a ceiling limit is more appropriate than an 8-hour TWA for methyl bromide. OSHA finds, however, that the 5-ppm TWA will provide protection against the levels shown to produce poisoning in humans (generally in the 50- to 150-ppm range).

The presence of neurologic symptoms at levels below 35 ppm indicates that the former ceiling limit of 20 ppm is not adequate to protect workers from the effects of methyl bromide poisoning. OSHA is establishing a PEL of 5 ppm TWA, with a skin notation, to protect workers more adequately against these incapacitating symptoms. The Agency concludes that these limits will reduce this significant risk substantially.

PENTABORANE

CAS: 19624-22-7; Chemical Formula: B_5H_9
H.S. No. 1304

OSHA's former limit for pentaborane was 0.005 ppm as an 8-hour TWA. The ACGIH has the same 8-hour TWA but additionally recommends a 15-minute STEL of 0.015 ppm. The Agency proposed, and the final rule establishes, permissible exposure limits of 0.005 ppm as an 8-hour TWA and 0.015 ppm as a 15-minute STEL for pentaborane. NIOSH (Ex. 8-47, Table N1) concurs that these limits are appropriate. Pentaborane is a colorless liquid with a strong and penetrating odor.

In both humans and animals, inhalation of pentaborane vapor causes central nervous system effects (Svirbely 1954a/Ex. 1-385; Rozendaal 1951/Ex. 1-525; Lowe and Freeman 1957/Ex. 1-518; Cordasco, Cooper, Murphy, and Anderson 1962/Ex. 1-545).

The 5-minute LC_{50} for rats and mice is 67 and 40 ppm, respectively; for 60 minutes, these values are 10 and 6 ppm for rats and mice, respectively (Weir, Bath, and Weeks 1961, as cited in ACGIH 1986/Ex. 1-3, p. 459). Rats exposed repeatedly to 3 ppm pentaborane by inhalation exhibited tremors, hyperexcitability, belligerence, and weight loss (Svirbely 1954a/Ex. 1-385). Rats, rabbits, monkeys, and dogs exposed repeatedly to pentaborane vapor at concentrations of 1 ppm for four weeks or 0.2 ppm for six months lost weight (Levinskas, Paslian, and Bleckman 1958/Ex. 1-517). In the same experiments, rats and rabbits exposed at 1 ppm showed reduced activity and impaired locomotor ability, respectively, and monkeys and dogs exhibited apathy, loss of appetite, insensitivity to

pain, loss of mobility, tremor, and impaired coordination. The ACGIH (1986/Ex. 1-3, p. 459) notes that the 0.2-ppm concentration reported in the Levinskas, Paslian, and Bleckman (1958/Ex. 1-517) study was a calculated rather than measured value and that the actual exposure level was probably closer to 0.01 ppm.

Humans accidentally overexposed to pentaborane experienced tremors, convulsions, behavioral changes, loss of memory, impaired judgment, and other symptoms of central nervous system intoxication (Svirbely 1954a/Ex. 1-385; Rozendaal 1951/Ex. 1-525; Lowe and Freeman 1957/Ex. 1-518; Cordasco, Cooper, Murphy, and Anderson 1962/Ex. 1-545). No comments other than those from NIOSH were received on the health effects associated with pentaborane exposure.

OSHA is establishing an 8-hour TWA PEL of 0.005 ppm and a 15-minute STEL of 0.015 ppm for pentaborane. The Agency concludes that these limits will protect workers against the significant risk of central nervous system effects, such as tremors and convulsions, behavioral changes, and loss of judgment, potentially associated with exposure to pentaborane at levels only slightly above those formerly permitted by the 8-hour TWA alone. OSHA finds that these neuropathic effects constitute material health impairments within the meaning of the Act.

PHENYL MERCAPTAN

CAS: 108-98-5; Chemical Formula: C_6H_5SH
H.S. No. 1316

OSHA previously had no exposure limit for phenyl mercaptan. The ACGIH has a TLV-TWA of 0.5 ppm. NIOSH recommends a 15-minute ceiling limit of 0.1 ppm for phenyl mercaptan (benzenethiol). The Agency proposed a permissible exposure limit of 0.5 ppm as an 8-hour TWA, and the final rule establishes this limit. Phenyl mercaptan is a colorless liquid with an offensive, garlic-like odor.

The primary acute hazards of exposure to phenyl mercaptan are central nervous system stimulation followed by post-convulsive CNS depression, severe eye and skin irritation, systemic toxicity to spleen, kidney, lung, and liver tissues, and narcotic effects (ACGIH 1986/Ex. 1-3, p. 478).

Phenyl mercaptan has been reported to have 4-hour inhalation LC_{50} values of 33 and 28 ppm for rats and mice, respectively (Doull and Plzak 1962, as cited in ACGIH 1986/Ex. 1-3, p. 478; Fairchild and Stokinger 1958/Ex. 1-415). The oral LD_{50} for the rat is reported to be 46 mg/kg (McCord and Witheridge 1949/Ex. 1-882; Robles 1975, as cited in ACGIH 1986/Ex. 1-3, p. 478). For the rabbit and rat, the dermal LD_{50} values are 134 mg/kg and 300 mg/kg, respectively (Doull and Plzak 1962, as cited in ACGIH 1986/Ex. 1-3, p. 478; Fairchild and Stokinger 1958/Ex. 1-415); Schafer 1972/Ex. 1-362). The responses of animals to phenyl mercaptan exposure were uniform regardless of species, and progressed from CNS stimulation to incoordination, skeletal and muscular paralysis, and respiratory depression, followed at high concentrations by coma and death. High doses (not further specified) administered via inhalation produced lung, liver, and kidney changes in mice (Doull and Plzak 1962, as cited in ACGIH 1986/Ex. 1-3, p. 478; Fairchild and Stokinger 1958/Ex. 1-415); Schafer 1972/Ex. 1-362). In rabbits, phenyl mercaptan is a severe eye and skin irritant (McCord and Witheridge 1949/Ex. 1-882; Robles 1975, as cited in ACGIH 1986/Ex. 1-3, p. 478; Schafer 1972/Ex. 1-362).

In humans, phenyl mercaptan is a moderately toxic skin irritant and causes severe dermatitis, headaches, and dizziness at unspecified levels (Fairchild and Stokinger 1958/Ex. 1-415; McCord and Witheridge 1949/Ex. 1-882). NIOSH (Ex. 8-47, Table N7; Tr. p. 3-99) believes that the limit for phenyl mercaptan is better expressed as a ceiling than as a time-weighted average; however, OSHA believes that a TWA limit set at 0.5 ppm will protect against phenyl mercaptan's toxic effects. No other comments on the health effects of phenyl mercaptan were submitted to the rulemaking record.

OSHA is establishing an 8-hour TWA limit of 0.5 ppm for phenyl mercaptan. The Agency concludes that this limit will protect workers from the significant risks of CNS effects, skin irritation, and systemic injury, all material impairments of health that are potentially associated with exposure to phenyl mercaptan at the uncontrolled levels formerly permitted by the absence of any OSHA limit.

PROPYLENE GLYCOL DINITRATE

CAS: 6423-43-4; Chemical Formula: $C_3H_6N_2O_6$
H.S. No. 1342

OSHA previously had no exposure limit for propylene glycol dinitrate. The ACGIH recommends a TLV-TWA of 0.05 ppm, with a skin notation. The Agency proposed a permissible exposure limit of 0.05 ppm TWA, with a skin notation, for this substance, and NIOSH (Ex. 8-47, Table N1) concurred with the proposed limit. The final rule establishes a PEL of 0.05 ppm but does not include the proposed skin notation. When freshly prepared, propylene glycol dinitrate is a colorless liquid with a disagreeable odor.

Exposure to this substance affects blood pressure, causes methemoglobinuria and respiratory toxicity, injures liver and kidney tissues, and distorts vision. Propylene glycol dinitrate can also cause headache and incoordination.

The oral LD_{50} value for the rat is between 480 and 250 mg/kg (Clark and Litchfield 1969/Ex. 1-543; Andersen and Mehl 1973/Ex. 1-536), and the subcutaneous LD_{50} is 530 mg/kg (Andersen and Mehl 1973/Ex. 1-536). Mice are reported to be somewhat more resistant, with a subcutaneous LD_{50} of slightly more than 1200 mg/kg; however, cats appear to be even more susceptible to propylene glycol dinitrate and exhibit a subcutaneous LD_{50} of between 200 and 300 mg/kg (Clark and Litchfield 1969/Ex. 1-543). In all species studied, death occurs by anoxia, which is caused by almost complete conversion of hemoglobin to methemoglobin (Clark and Litchfield 1969/Ex. 1-543). Skin tests in albino rabbits did not produce irritation, but ocular instillation caused transient conjunctival redness (Jones, Strickland, and Siegel 1972/Ex. 1-742). Twenty-day skin exposures in rabbits at 1 g/kg caused minor irritation, and at 2 g/kg, rabbits became weak and cyanotic; one of five rabbits died, and this animal's hemoglobin and hematocrit values had decreased. When the dose was increased to 4 g/kg, the rabbits' methemoglobin values rose to 34.5 percent at death (Jones, Strickland, and Siegel 1972/Ex. 1-742). Continuous 90-day inhalation exposures at 10 ppm caused kidney and liver changes in dogs; exposures at 35 ppm caused heavy iron deposits in the liver, spleen, and kidneys. Female (but not male) rats showed a drop in blood pressure within 30 minutes after injection of doses above 5 mg/kg. Rhesus monkeys displayed mydriasis in 90-day exposures at 35 ppm but no change in avoidance behavior during a visual discrimination and acuity threshold test (Jones, Strickland, and Siegel 1972/Ex. 1-742).

In humans, eight-hour exposures to 0.2 ppm or higher concentrations of propylene glycol dinitrate resulted in visual distortion and headache (Stewart, Peterson, Newton et al. 1974, as cited in ACGIH 1986/Ex. 1-3, p. 502). Although subjects developed a tolerance for the headache response, the visual effects were cumulative. Impaired balance occurred after 6.5 hours of exposure to 0.5 ppm, and a 40-minute exposure to 1.5 ppm caused eye irritation. Subjects

exposed at 0.5 ppm for 8 hours experienced a consistent elevation in diastolic pressure but no pulmonary irritation. At concentrations of 0.03 to 1.5 ppm, no hematologic effects were observed (Stewart, Peterson, Newton et al. 1974, as cited in ACGIH 1986/Ex. 1–3, p. 502). Studies of human exposures to levels below 0.1 ppm do not report chronic neurotoxicity (Horvath, Ilka, Boyd, and Markhan 1981/Ex. 1–557).

The skin notation included in the proposal for this substance is not included in the final rule because evidence demonstates that the dermal LD_{50} in rabbits is even greater than 2 g/kg (see the discussion in Section VI.C.18 for OSHA's policy on skin notations). No comments except those from NIOSH were received on the health effects of propylene glycol dinitrate.

OSHA is establishing an 8-hour TWA limit of 0.05 ppm for propylene glycol dinitrate. The Agency concludes that this limit will protect workers against the significant risks of hepatotoxic, hematologic, and central nervous system effects (all of which constitute material health impairments) that exist from workplace exposure at the levels permitted in the absence of any OSHA PEL.

Conclusions

OSHA concludes that significant risks are associated with occupational exposure to the group of neuropathic toxicants shown in Table C1–1. The effects caused by such exposures include brain lesions, nausea, vomiting, general depression of the central nervous system, interference with sensory and motor functions, and alterations in the ability of the brain to process information. Affected workers may experience drowsiness, dizziness, loss of ability to concentrate, mood changes, reduced awareness, learning difficulty, unsteadiness, and auditory and visual disturbances. In addition, employees experiencing these effects are imperiled and are likely to hurt themselves or others in accidents caused by their reduced functional capacities. The final rule's promulgation of new or revised exposure limits for these neurotoxins substantially reduces such risks and affords protection to workers against these material health impairments.

2. Substances for Which Limits Are Based on Avoidance of Narcotic Effects

Introduction

OSHA is establishing new or revised limits for 19 substances based primarily on evidence showing that occupational exposure to these substances causes narcosis. The narcotic effects of exposure to such substances as the alcohols, aliphatic hydrocarbons, and chlorinated hydrocarbons have been recognized as serious for many years. Table C2–1 lists these chemicals, their CAS and HS numbers, and their former, proposed, and final rule limits. For seven of these substances, the Agency is lowering the 8-hour TWA permissible exposure limit and revising or adding a STEL. In five additional cases OSHA is retaining its former 8-hour TWA permissible exposure limit and adding a STEL. Eight-hour TWAs and/or STELs are being established for four previously unregulated substances, and in three other cases, OSHA is lowering its 8-hour TWA permissible exposure limit.

Description of the Health Effects

Narcosis is caused by a general depression of central nervous system (CNS) function. When the CNS becomes sufficiently depressed, the awareness or consciousness of affected persons is diminished. Initial symptoms of narcosis include drowsiness, difficulty in concentration, and mood changes; these effects may progress to slurred speech, dizziness, loss of coordination, and, in more severe cases, loss of consciousness, coma, and death. Except in more serious cases, CNS depression is reversible if the exposure ceases. However, because narcosis adversely affects the concentration and coordination of affected workers, these workers and their co-workers are at increased risk of injuries and accidents caused by slowed reaction times, incoordination, and mistakes and errors in judgment. Moreover, these effects constitute material impairments of health or functional capacity within the meaning of the Act, even if they are not permanent.

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TABLE C2-1. Substances for Which Limits Are Based on Avoidance of Narcosis

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1044 Butane	106-97-8	—	800 ppm TWA	800 ppm TWA
1049 sec-Butyl alcohol	78-92-2	150 ppm TWA	100 ppm TWA 150 ppm STEL	100 ppm TWA
1050 tert-Butyl alcohol	75-65-0	100 ppm TWA	100 ppm TWA 150 ppm STEL	100 ppm TWA 150 ppm STEL
1111 Cyclopentane	287-92-3	—	600 ppm TWA	600 ppm TWA
1163 Ethyl bromide	74-96-4	200 ppm TWA	200 ppm TWA 250 ppm STEL	200 ppm TWA 250 ppm STEL
1185 Gasoline	8006-61-9	--	300 ppm TWA 500 ppm STEL	300 ppm TWA 500 ppm STEL
1194 Heptane	142-82-5	500 ppm TWA	400 ppm TWA 500 ppm STEL	400 ppm TWA 500 ppm STEL
1201 Hexane isomers	Varies with compound	--	500 ppm TWA 1000 ppm STEL	500 ppm TWA 1000 ppm STEL

TABLE C2-1. Substances for Which Limits Are Based on Avoidance of Narcosis (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1218 Isoamyl alcohol (primary and secondary)	123-51-3	100 ppm TWA	100 ppm TWA 125 ppm STEL	100 ppm TWA 125 ppm STEL
1221 Isophorone	78-59-1	25 ppm TWA	4 ppm TWA	4 ppm TWA
1254 Methyl chloride	74-87-3	100 ppm TWA 200 ppm Ceiling (5 min/3 hrs) 300 ppm Peak	50 ppm TWA 100 ppm STEL	50 ppm TWA 100 ppm STEL
1255 Methyl chloroform (1,1,1-Trichloroethane)	71-55-6	350 ppm TWA	350 ppm TWA 450 ppm STEL	350 ppm TWA 450 ppm STEL
1296 Octane	111-65-9	500 ppm TWA	300 ppm TWA 375 ppm STEL	300 ppm TWA 375 ppm STEL
1306 Pentane	109-66-0	1000 ppm TWA	600 ppm TWA 750 ppm STEL	600 ppm TWA 750 ppm STEL
1307 2-Pentanone (Methyl propyl ketone)	107-87-9	200 ppm TWA	200 ppm TWA 250 ppm STEL	200 ppm TWA 250 ppm STEL

TABLE C2-1. Substances for Which Limits Are Based on Avoidance of Narcosis (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1371 Stoddard solvent	8052-41-3	500 ppm TWA	100 ppm TWA	100 ppm TWA
1372 Styrene	100-42-5	100 ppm TWA 200 ppm Ceiling (5 min/3 hrs) 600 ppm Peak	50 ppm TWA 100 ppm STEL	50 ppm TWA 100 ppm STEL
1397 Toluene	108-88-3	200 ppm TWA 300 ppm Ceiling (10 min/8 hrs) 500 ppm Peak	100 ppm TWA 150 ppm STEL	100 ppm TWA 150 ppm STEL
1406 Trichloroethylene	79-01-6	100 ppm TWA 200 ppm Ceiling (5 min/2 hrs) 300 ppm Peak	25 ppm TWA	50 ppm TWA 200 ppm STEL

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

BILLING CODE 4510-26-C

The mechanism by which exposure to substances induces narcosis is poorly understood. It is believed that CNS depressants may have the same mechanism of action as general anesthetics, which appear to produce a reversible effect on electrically excitable neuronal membranes.

Dose-Response Relationship and Narcotic Effects

The induction of narcosis following exposure to narcotic agents is expected to follow the classical S-shaped (sigmoidal) dose-response relationship. As exposure level increases, both the percent of exposed persons affected and the severity of the effect increase. Although it is not known whether a true threshold exists for the occurrence of the molecular events leading to narcosis (i.e., disruption of electrical impulses in neurons), there is usually a level at which most exposed individuals will manifest the onset of symptoms associated with narcosis. The no-effect level for a particular substance is determined largely by individual susceptibility, the extent to which the material is absorbed, and the rate at which it is metabolized and eliminated.

The following discussion describes the record evidence and OSHA's findings for the substances in this group and illustrates the material health impairments associated with workplace exposure to these substances.

BUTANE

CAS: 106-97-8; Chemical Formula: C_4H_{10}
H.S. No. 1044.

Previously, OSHA had no limit for butane. The ACGIH has a TLV-TWA of 800 ppm for this colorless, flammable gas. The proposed PEL was 800 ppm as an 8-hour TWA, and NIOSH (Ex. 8-47, Table N1,) concurs with this limit. The final rule promulgates an 8-hour TWA of 800 ppm.

The primary risk of exposure to butane is narcosis, which occurs at high exposure levels. Exposure to 10,000 ppm butane for 10 minutes causes drowsiness, but there are no reports of systemic toxicity or irritation at this level (Gerarde 1963a, as cited in ACGIH 1986/Ex. 1-3, p. 10).

In rats, the 4-hour LC_{50} for butane is 658 g/m^3, or about 280,000 ppm (NIOSH 1977i/Ex. 1-1182). Humans exposed to 1000 ppm for a single eight-hour day, or to 500 ppm for two-week periods of eight-hour workdays, showed no harmful subjective or abnormal physiological responses but did show a reduced visual evoked response (VER) wave amplitude during the second week (Stewart, Herrman, Baretta et al. 1977/ Ex. 1-575). OSHA received no comments, other than NIOSH's on butane.

In the final rule, OSHA is establishing a permissible exposure limit of 800 ppm TWA for butane. The Agency concludes that this limit will protect workers against the significant risks of drowsiness and other narcotic effects, which together consitute material health impairments and are associated with exposures at the uncontrolled levels permitted in the past by the absence of an OSHA limit.

sec-BUTYL ALCOHOL

CAS: 78-92-2; Chimical Formula: CH_3 CH_2 $CHOHCH_3$
H.S. No. 1049

OSHA's former limit for sec-butyl alcohol was 150 ppm as an 8-hour TWA. The proposed PELs were 100 ppm as an 8-hour TWA and 150 ppm as a 15-minute STEL, and NIOSH (Ex. 8-47) concurred with these limits. In the final rule, OSHA is establishing an 8-hour TWA of 100 ppm but is not adding a STEL (see the discussion of the Agency's policy on STELs for this rulemaking in Section VI.C.17). sec-Butyl alcohol is a colorless liquid with a strong, wine-like odor.

The acute toxicity of sec-butyl alcohol is reported to be lower than that of n-butanol, for which OSHA is establishing a ceiling of 50 ppm. The oral LD_{50}s in rats for these two substances are 6.5 g/kg for sec-butyl alcohol and 4.4 g/kg for n-butanol, respectively (Smyth, Carpenter, and Weil 1951/Ex. 1-439). Liquid sec-butyl alcohol is less injurious to the eyes than liquid n-butanol (ACGIH 1986/Ex. 1-3, p. 77). Occupational exposures to sec-butyl alcohol at levels of about 100 ppm were reported not to be associated with difficulties (Banks 1966, as cited in ACGIH 1986/Ex. 1-3, p. 77).

OSHA received a comment on this substance from the American Industrial Hygiene Association (AIHA) (Ex. 8-16). The AIHA noted that there was no evidence to support a STEL for this substance and reported that the ACGIH intends to delete this STEL. OSHA has arrived at the same conclusion, and the final rule thus has no STEL for sec-butyl alcohol.

OSHA is reducing the permissible exposure limit for sec-butyl alcohol to 100 ppm TWA to afford protection against the significant risks of narcosis and irritation, which are material health impairments that are caused by exposures to sec-butyl alcohol at concentrations above the revised PEL. The Agency concludes that this limit will substantially reduce this risk.

tert-BUTYL ALCOHOL

CASL 75-65-0; Chemical Formula: $(CH_3)_3COH$
H.S. No. 1050

OSHA formerly had a limit of 100 ppm for tert-butyl alcohol. The ACGIH has a TLV-TWA of 100 ppm, with a TLV-STEL of 150 ppm. OSHA proposed to retain the 8-hour TWA limit of 100 ppm and to add a STEL of 150 ppm for tert-butyl alcohol, and NIOSH (Ex. 8-47, Table N1) concurs. These limits are established by the final rule. At ordinary temperatures and pressures, tert-butyl alcohol exists in the form of colorless, hygroscopic crystals (ACGIH 1986/Ex. 1-3).

Although similar to the other butyl alcohols in many respects, tert-butyl alcohol is more volatile and has a greater potential for narcotic effects than other butyl alcohols (Weese 1928/ Ex. 1-1073). Mice exposed to tert-butyl alcohol exhibit a stronger narcotic response than they show when exposed to normal or isobutyl alcohol (Weese 1928/Ex. 1-1073). Repeated daily doses of tert-butyl alcohol that produced narcosis were not fatal in animals (Schaffarzick and Brown 1952/Ex. 1-868). In humans, contact with t-butyl alcohol produces erythema and hyperemia (Oettel 1936/Ex. 1-921). Except for NIOSH's submittal, OSHA received no comments on tert-butyl alcohol.

In the final rule, OSHA is retaining the 8-hour TWA PEL of 100 ppm and adding a 15-minute STEL of 150 ppm for tert-butyl alcohol. The Agency concludes that this combination of limits will protect against the significant risk of narcosis, which constitutes a material health impairment that potentially occurs at levels above the 8-hour TWA PEL.

CYCLOPENTANE

CAS: 287-92-3; Chemical Formula: $CH_2CH_2CH_2CH_2CH_2$
H.S. No. 1111

Previously, OSHA had no limit for cyclopentane. The proposed PEL was 600 ppm as an 8-hour TWA, and NIOSH concurred with this limit (Ex. 8-47, Table N1). The final rule promulgates this limit, which is consistent with that of the ACGIH. Cyclopentane is a mobile, colorless, and flammable liquid.

The existing animal data indicate that cyclopentane is a narcotic agent. As with other alicyclic hydrocarbons, exposure to high concentrations causes excitement, loss of equilibrium, stupor, coma, and, rarely, respiratory failure (Gerarde 1963a, as cited in ACGIH 1986/ Ex. 1-3, p. 164). No major animal studies on the effects of cyclopentane exposure have been reported, and evaluations of the toxic properties of this substance have therefore relied on the animal data

for n-pentane. n-Pentane has been shown to cause narcosis in animals at exposures of 90,000 to 120,000 ppm for 5 to 60 minutes (Abbritti, Siracusa, Cianchetti et al. 1976/Ex. 1–406). Swann, Kwon, and Hogan (1974/Ex. 1–124) reported that a concentration of 130,000 ppm is fatal. Almost no data are available concerning the chronic effects of cyclopentane exposure.

Abbritti, Siracusa, Cianchetti et al. (1976/Ex. 1–406) reported that petroleum solvents used in the Italian shoe industry contain up to 18 percent cyclopentane. Workers exposed to these solvents have developed polyneuropathy, and Oettel (1936/Ex. 1–921) reported that skin exposure to such solvents caused burning and skin blistering after 15 minutes of confined contact. It has not been determined whether the irritation was caused by cyclopentane or by cyclopentane and other substances, such as n-hexane, in the solvent. OSHA received no comments other than those from NIOSH.

In the final rule, OSHA is establishing a PEL of 600 ppm as an 8-hour TWA for cyclopentane. OSHA concludes that occupational exposure to cyclopentane poses a significant risk of irritation and narcosis, which constitute material impairments of health that occur at levels somewhat above the PEL established in the final rule.

ETHYL BROMIDE

CAS: 74–96–4; Chemical Formula: C_2H_5Br
H.S. No. 1163

OSHA formerly had an 8-hour TWA limit of 200 ppm for ethyl bromide. The ACGIH also has a limit of 200 ppm as an 8-hour TWA and 250 ppm as a 15-minute STEL. The proposal retained the PEL of 200 ppm and added a STEL of 250 ppm; these limits are established by the final rule. Ethyl bromide is a colorless, highly volatile, flammable liquid with an ether-like odor; it becomes yellow when exposed to light and air.

The concentrations of ethyl bromide reported as lethal to guinea pigs are 3200 ppm for 9 hours and 1700 ppm for 12.5 hours (Sayers, Yant, Thomas, and Berger 1929/Ex. 1–803). von Oettingen (1955/Ex. 1–876) reported the minimal lethal concentration of this substance for mice as 3500 ppm.

Ethyl bromide acts as a central nervous system depressant (narcotic); additionally, exposure causes irritation of the lungs and congestion and fatty degeneration of the liver, intestinal hemorrhage, and kidney swelling. Several deaths have been reported from the use of ethyl bromide as a general anesthetic (von Oettingen 1955/Ex. 1–876). The record contains no submissions on the health effects of ethyl bromide exposure other than a submission from NIOSH (Ex. 8–47, Table N2; Tr. 3–86) indicating its nonconcurrence. NIOSH noted that one study (Karimullna and Gizatullina 1969) demonstrated liver injury and disrupted liver function in rats exposed 4 hours daily for 6 months to 540 ppm ethyl bromide. NIOSH also reported that an NTP inhalation bioassay to assess the carcinogenicity of ethyl bromide in rats and mice exposed at 100, 200, or 400 ppm was scheduled for peer review in October 1988. OSHA will review this study and any others that become available on this substance to determine whether further action is warranted.

In the final rule, OSHA is retaining its PEL of 200 ppm as an 8-hour TWA and adding a 15-minute STEL of 250 ppm for ethyl bromide. The Agency concludes that these limits will work together to reduce the significant risks of narcosis, kidney and liver damage, and respiratory irritation, all material impairments of health that are associated with occupational exposure to elevated levels of ethyl bromide.

GASOLINE

CAS: 8006–61–9; Chemical Formula: None
H.S. No. 1185

Previously, OSHA had no PEL for gasoline. The ACGIH has a 300-ppm 8-hour TWA and a 500-ppm 15-minute STEL for this substance. OSHA proposed a TWA PEL of 300 ppm and a STEL of 500, and these limits are established in the final rule.

Studies have shown that exposure to 2000 ppm of gasoline for 30 minutes produces mild anesthesia, while exposure to concentrations between 500 and 900 ppm for one hour produces dizziness (Gerarde 1963a and Runion 1975, both as cited in ACGIH 1986/Ex. 1–3, p. 283). However, these authors also found that people exposed to gasoline at concentrations of 160 to 270 ppm for several hours do not experience any symptoms of narcosis but may, as Dr. Liem (Ex. 46) points out, experience eye and throat irritation.

Several commenters noted that gasoline, or specific types of gasoline (i.e., unleaded), may cause kidney and liver damage and cancer, in addition to CNS effects (Exs. 3–746, 8–47, 194, 197; Tr. VII, pp. 70–76). Dr. Franklin Mirer, Director of the Health and Safety Department of the United Auto Workers, made the following statement, which is typical of the views of this group of commenters:

> The crucial study in redefining the toxicity of aliphatic hydrocarbons is an inhalation bioassay of unleaded gasoline conducted by the American Petroleum Institute in 1984. The study found increased kidney tumors in male rats and liver tumors in female mice.
>
> The rat portion of the study gave definitely clear evidence of carcinogenic activity. Kidney tumors appeared in a group of rats exposed at 292 parts per million, although a statistical analysis was not documented in the published report. Of greater concern to me than the carcinogenic effect was that male rats also suffered a characteristic toxic kidney effect[s]. Indications of this toxicity appeared as early as three to six months in rats exposed at 47 parts per million (Tr. VII, pp. 70–71).

NIOSH shares these concerns and commented (Ex. 8–47, Table N6B) that gasoline would be an appropriate candidate for a full Section 6(b) rulemaking.

OSHA is aware that there is a recent and rapidly developing body of evidence about other health effects associated with exposure to gasoline and other petroleum materials and that this is an active area of toxicological research. However, the Agency agrees with the American Petroleum Institute (Ex. 124, p. 4) that complex and difficult scientific questions remain to be answered before conclusions can be drawn about these other potentially toxic effects of gasoline exposure. OSHA believes that it would be inappropriate to delay action on this substance at the present time. NIOSH representatives at the hearing (Tr. pp. 3–130, 3–131) agreed that, in the absence of a NIOSH REL for gasoline, promulgation of the proposed limits would constitute an appropriate first step in affording exposed workers protection against these health effects.

OSHA is establishing an 8-hour TWA of 300 ppm, supplemented with a STEL of 500 ppm, to ensure that workplace exposure levels to gasoline do not exceed the TWA level for any length of time; these limits are intended to protect against narcosis. OSHA concludes that the 8-hour TWA and STEL being promulgated in the final rule will substantially reduce the significant risks posed to workers exposed to gasoline in their places of work. These exposure-related health effects, which include narcosis and liver and kidney damage, clearly constitute material impairments of health within the meaning of the Act.

HEPTANE

CAS: 142–82–5; Chemical Formula: $CH_3(CH_2)_5CH_3$
H.S. No. 1194

The former OSHA limit for heptane was 500 ppm as an 8-hour TWA. The ACGIH TLVs for heptane are 400 ppm as a TWA and 500 ppm as a STEL. NIOSH (1977a/Ex. 1–233) has recommended that workplace exposures to heptane not exceed 85 ppm as a full-

shift TWA or 440 ppm as a 15–minute ceiling limit. The proposed PEL and STEL were 400 and 500 ppm, respectively, and these limits are established by the final rule. Heptane is a clear, flammable liquid which is highly volatile.

Patty and Yant (1929, as cited in ACGIH 1986/Ex. 1–3, p. 297) reported that exposure to 1000 ppm of heptane for 6 minutes caused slight dizziness in humans; exposures to higher levels caused vertigo, incoordination, and inappropriate behavior. These authors also reported that a four-minute exposure to 5000 ppm produced complaints of loss of appetite and nausea. Based on this information, as well as on animal data showing 10,000 to 15,000 ppm to be an effect level for heptane-induced narcosis (Fuhner 1921, as cited in ACGIH 1986/Ex. 1–3, p. 297), the ACGIH concluded that heptane was more acutely toxic than hexane. The ACGIH therefore recommended limits for heptane that are somewhat lower than the limits for the hexane isomers.

As discussed in connection with pentane and the hexane isomers, NIOSH (1977a/Ex. 1–233) has recommended the same occupational exposure limits for all of the C_5-C_8 alkanes (i.e., 350 mg/m^3 TWA and 1800 mg/m^3 as a 15-minute ceiling). This recommendation is based on NIOSH's belief that all C_5-C_8 alkanes possess a potential neurotoxic capability similar to that of n-hexane. OSHA disagrees with this concept (see the discussion of this issue in Section V of the preamble); the Agency finds that the neurotoxicity caused by exposure to n-hexane is the result of the action of a unique metabolite, 2,5-hexanedione; the majority of record commenters agreed with OSHA that n-hexane is uniquely toxic (Exs. 3–593, 3–896, and 3–1246).

NIOSH does not concur with the limits being established for heptane (Ex. 8–47, Table N2) because NIOSH believes that "it would be incorrect to conclude that the neurotoxic properties ascribed to n-hexane are unique to this compound [n-hexane]. Other alkanes or related chemicals [such as heptane] that are ultimately metabolized to gamma diketone may have similar toxicity" (Tr. III, p. 110). However, OSHA does not agree with NIOSH that all of the C_5-C_8 alkanes have equal toxicity (see the discussion in Section V of the preamble); OSHA believes that n-hexane is uniquely toxic.

The AFL–CIO (Ex. 194, p. A–7) reiterated its position that OSHA should promulgate a 10-ppm limit for all of the petroleum solvents, including heptane. However, OSHA has determined (see Section IV.D) that it would be inappropriate at this time to enlarge the scope of this already extensive regulation. The United Auto Workers (Ex. 197) described engineering controls that could be used to achieve the lower levels the unions advocate for all petroleum solvents; these are discussed in Section VII.

Because heptane is considered to be more acutely toxic than hexane, OSHA concludes that it is appropriate to revise its limit for heptane to a level below that established for the hexane isomers to reduce the significant risk of narcosis, which is a material health impairment. Therefore, OSHA is revising its limit for heptane to 400 ppm as an 8-hour TWA and 500 ppm as a 15-minute STEL. The Agency concludes that the TWA and STEL together will substantially reduce this significant occupational risk.

HEXANE ISOMERS

CAS: None; Chemical Formula: $(CH_3)_3C_3H_5$; $n(CH_3)_4C_2H_2$
H.S. No. 1201

Previously, OSHA had no limit for the hexane isomers. The ACGIH TLVs for the hexane isomers are 500 ppm as an 8-hour TWA and 1000 ppm as a 15-minute STEL. NIOSH has a recommended TWA limit for these isomers of 100 ppm, supplemented with a 510-ppm 15-minute ceiling. The proposed and final rule PELs are an 8-hour TWA of 500 ppm and a 15-minute STEL of 1000 ppm. The hexanes are clear, highly volatile liquids with a mild gasoline-like odor.

A study by Drinker, Yaglou, and Warren (1943/Ex. 1–730) shows that humans exposed to 1400 to 1500 ppm of hexane experienced nausea and headache. Patty and Yant (1929, as cited in ACGIH 1986/Ex. 1–3, p. 307) found that a 10-minute exposure to 5000 ppm caused giddiness and dizziness in exposed subjects. A study by Nelson, Enge, Ross et al. (1943/Ex. 1–66) showed no effects in unacclimated subjects exposed to hexane isomers in concentrations of 500 ppm, but narcotic effects have often been seen in subjects exposed at levels above 1000 ppm (Elkins 1959d, as cited in ACGIH 1986, Ex. 1–3, p. 307). The ACGIH based its limit primarily on the Nelson, Enge, Ross et al. (1943/Ex. 1–66) study.

NIOSH recommends limits for the hexane isomers of 100 ppm as a 10-hour TWA and 510 ppm as a 15-minute short-term limit. These recommendations are based on human and animal evidence showing that exposure to n-hexane below concentrations of 500 ppm is associated with the development of polyneuropathy (Inoue, Takeuchi, Takeuchi et al. 1970/Ex.1–75; Miyagaki 1967/Ex. 1–198); NIOSH (1977a/Ex. 1–233) did not distinguish between n-hexane and other hexane isomers when making its recommendation for an exposure limit. NIOSH concluded that all of the C_5–C_8 alkanes are potential neuropathic agents and should have the same PELs as those established for n-hexane.

OSHA disagrees with NIOSH that all C_5–C_8 alkanes are potential neuropathic agents. As discussed in Section V of the preamble, OSHA believes that a metabolite of n-hexane (2,5-hexanedione) is responsible for the unique neurotoxic properties of n-hexane (see also the discussion of n-hexane in Section VI.C.1 of the Preamble). Thus OSHA agrees with the ACGIH that "it seems unlikely that all the hexanes would follow the same metabolic route in the body [as n-hexane], in view of the marked variations in structure of the molecule" (ACGIH 1986/Ex. 1–3, p. 307). The majority of commenters supported OSHA's conclusion that n-hexane is uniquely toxic because of the presence of 2,5-hexanedione and that the other alkanes are not toxic in this way (Exs. 3–593, 3–896, and 3–1246). However, the AFL–CIO (Ex. 194, p. A–7) argued for a lower limit for the hexane isomers and all petroleum solvents (see the discussion for heptane, above), and the UAW (Ex. 197) noted that controls are available to reduce exposures (see Section VII for a discussion of feasibility).

After reviewing the evidence cited by the ACGIH (1986/Ex. 1–3), NIOSH (1977a/Ex.1–233), and commenters to the record, OSHA finds that workers exposed to hexane isomers are at significant risk of experiencing narcosis and of developing neuropathy at exposure levels above the new PELs. The Agency concludes that establishing an 8-hour TWA of 500 ppm and a 15-minute STEL of 1000 ppm will substantially reduce these risks. OSHA finds that both narcosis and neuropathy constitute material health impairments.

ISOAMYL ALCOHOL (PRIMARY AND SECONDARY)

CAS: 123–51–3; Chemical Formula: $(CH_3)_2CHCH_2CH_2OH$—Primary; $(C_2H_5)_2CHOH$—Secondary
H.S. No. 1218

OSHA's former limit for the isoamyl alcohols was 100 ppm as an 8-hour TWA. The ACGIH has established an 8-hour TLV–TWA of 100 ppm and a 15-minute STEL of 125 ppm for these substances, which are colorless liquids that have pungent tastes and an alcoholic odor that causes coughing. OSHA proposed to retain the 8-hour TWA limit of 100 ppm and to add a 125-ppm 15-minute STEL; NIOSH (Ex. 8–47,

Table N1) concurs with these limits. The final rule retains the 100-ppm 8-hour TWA and adds a 125-ppm STEL for isoamyl alcohol.

In rats, the oral LD_{50} for the primary isoamyl alcohol is 7.07 mg/kg (Smyth, Carpenter, Weil et al. 1969/Ex. 1–442). Haggard, Miller, and Greenberg (1945/Ex. 1–956) determined that isoamyl alcohol's anesthetic toxicity was approximately 12 times higher than that of ethyl alcohol, which has a TLV-TWA of 1000 ppm. Exposure to isoamyl alcohol is not associated with chronic effects.

Smyth (1956/Ex. 1–759) reported that the principal effect of inhalation exposure to this substance is narcosis, and that a 100-ppm level would protect exposed workers against significant narcosis but not against some irritation. Nelson, Enge, Ross, and co-workers (1943/Ex. 1–66) stated that unacclimatized human volunteers reported upper respiratory tract irritation after brief exposures to an isoamyl alcohol concentration of 100 ppm, and objectionable eye and mucous membrane irritation at short-term exposures to 150 ppm. With the exception of NIOSH's submittal, OSHA received no comments on isoamyl alcohol.

In the final rule, OSHA is retaining the 8-hour TWA of 100 ppm and adding a 15-minute STEL of 125 ppm for the isoamyl alcohols (primary and secondary). OSHA concludes that a short-term limit is necessary because the chemically induced eye and throat irritation associated with exposure to the isoamyl alcohols is an acute effect that occurs at concentrations only slightly higher than the 100-ppm 8-hour TWA; in addition, significant narcosis occurs at the levels permitted by the absence of a STEL. The Agency concludes that both the TWA and STEL limits are necessary to ensure that workers are protected against the material impairments represented by significant narcosis, as well as the eye, nose, and upper respiratory tract irritation known to be associated with brief exposures to isoamyl alcohol at levels above 100 ppm.

ISOPHORONE

CAS: 78–59–1: Chemical Formula: $C_9H_{14}O$
H.S. No. 1221

The former OSHA limit for isophorone was 25 ppm as an 8-hour TWA. The ACGIH has established a 5-ppm TLV as a ceiling limit, and NIOSH recommends a workplace standard of 4 ppm as an 8-hour TWA for isophorone. Isophorone is a colorless liquid at room temperature, and it has a camphor-like odor. The proposed limit was 4 ppm as an 8-hour TWA; NIOSH (Ex. 8–47, Table N1) concurs. This is the limit promulgated by the final rule.

Studies in animals and with human volunteers indicate that exposures to high concentrations of isophorone cause nephrotoxic and other adverse effects. A paper by Smyth, Seaton, and Fischer (1942/Ex. 1–378) reported that guinea pigs and rats exposed to 550 ppm isophorone for six weeks demonstrated degenerative changes in the kidneys and liver. At an exposure level of 25 ppm, no adverse effects were noted, but at 50 ppm, the liver of one animal and the kidneys of four others were damaged. The entire group of 20 animals exposed at 50 ppm survived, but 2 of 16 animals died after this level was raised to 100 ppm (Smyth, Seaton, and Fischer 1942/Ex. 1–378). Volunteers exposed for a few minutes to isophorone vapor at concentrations between 40 and 400 ppm experienced eye, nose, and throat irritation; several subjects exposed at the 200-ppm level developed headache, nausea, faintness, dizziness, and a feeling of suffocation (Smyth and Seaton 1940a/Ex. 1–377). Silverman, Schulte, and First (1946/Ex. 1–142) reported that volunteers exposed to 25 ppm isophorone, the former OSHA PEL, complained of irritation of the eyes, nose, and throat. Another study conducted by the Western Electric Company (Ware 1973, as cited in ACGIH 1986/Ex. 1–3, p. 333) reported that workers exposed for a one-month period to levels of 5 to 8 ppm isophorone demonstrated fatigue and malaise. When the workplace level was reduced to between 1 and 4 ppm, there were no complaints of adverse effects. The NIOSH criteria document for the ketones (1978f, as cited in ACGIH 1986/Ex. 1–3, p. 333) notes that all of the ketones are central nervous system depressants and that workplace exposures to more than one ketone may produce additive effects.

A comment from the New Jersey Department of Public Health (Ex. 144) urged OSHA to use EPA's IRIS data to set a limit for isophorone. The use of IRIS data is discussed in Section VI.A.

In the final rule, OSHA is reducing its 8-hour TWA PEL of 25 ppm to an 8-hour TWA of 4 ppm to protect workers against the significant risk of fatigue, nausea, and headaches, which together constitute material health impairments that have been demonstrated to occur at isophorone levels between 5 and 8 ppm. The Agency concludes that this limit will substantially reduce these occupational risks.

METHYL CHLORIDE

CAS: 74–87–3; CHEMICAL FORMULA: CH_3Cl
H.S. No. 1254

OSHA's former limits for methyl chloride were 100 ppm as an 8-hour TWA, 200 ppm as a ceiling (not to be exceeded for more than five minutes in any three-hour period), and 300 ppm as a peak. The ACGIH has a 50-ppm 8-hour TLV-TWA limit and a 100-ppm 15-minute STEL for this substance, and NIOSH recommends the lowest feasible limit because it considers methyl chloride a potential occupational carcinogen. The proposed PELs were 50 ppm as an 8-hour TWA and 100 ppm as a 15-minute STEL; the final rule establishes these limits. Methyl chloride is a colorless, sweet-smelling gas.

There is considerable evidence in humans and some in animals demonstrating that exposure to methyl chloride by inhalation or dermal absorption produces narcosis and other central nervous system effects, including respiratory failure and death (ACGIH 1986/Ex. 1–3, p. 380). In animals, repeated exposures to 500 ppm or to higher concentrations can be life-threatening, but exposures to 300 ppm for 64 weeks caused no apparent effects (Smith and von Oettingen 1947/Ex. 1–527).

Reports in earlier literature described by Fairhall (1969a/Ex. 1–848) indicate that moderate (not further specified) exposure causes ocular symptoms that may persist for weeks, while high (not further specified) exposure has severe effects on the central nervous system. Patty (1963a/Ex. 1–855) states that serious exposure causes central nervous system, liver and kidney, and bone marrow effects, with symptoms of ataxia, staggering gait, weakness, tremors, vertigo, speaking difficulty, and blurred vision. Symptoms may be of several weeks' duration or may even be permanent (Patty 1963a/Ex. 1–855).

The Dow Chemical Company (as cited in ACGIH 1986/Ex. 1–3, p. 380) studied the methyl chloride exposures of employees in 54 job classifications over a four-month period. Exposures ranged from 5 to 78 ppm methyl chloride (8-hour TWAs), averaged 30 ppm over the work shift, and occasionally included peaks as high as 440 ppm. Medical examination of these workers revealed no detectable effects of methyl chloride exposure. However, average eight-hour exposures in the range of 195 to 475 ppm caused symptoms of weakness, drowsiness, staggering gait, thickness of the tongue, and memory lapses in some of the exposed employees 'Dow

Chemical Company, as cited in ACGIH 1986/Ex. 1–3, p. 380).

In a study of six cases of industrial methyl chloride poisoning, workers chronically exposed to levels between 200 and 400 ppm developed neurotoxic symptoms after two or more weeks of exposure (Scharnweber, Spears, and Cowles 1974/Ex. 1–664). Symptoms included drowsiness, dizziness, mental confusion, clouded vision, staggering gait, and slurred speech, and symptoms sometimes recurred after apparent recovery and in the absence of renewed exposure.

Repko and co-workers (1976/Ex. 1–1165) found that workers exposed to concentrations of methyl chloride ranging from 7.4 to 70 ppm but averaging 33.6 ppm displayed a significant performance decrement, and that exposures below 100 ppm produced significant but transitory changes in functional capacity. OSHA will continue to monitor the toxicological evidence for methyl chloride and will re-evaluate the substance if this evidence suggests that this is appropriate.

OSHA received comments on methyl chloride from NIOSH and the Methyl Chloride Industry Association. NIOSH believes that methyl chloride is an appropriate substance for a section 6(b) rulemaking because, in NIOSH's view, methyl chloride is a potential occupational carcinogen (Ex. 8–47; Tr. 3, pp. 97–98). The AFL–CIO (Ex. 194) agrees with NIOSH on this point. The Methyl Chloride Industry Association (MCIA) indicated its support of OSHA's proposed PELs for this substance and submitted material suggesting that methyl chloride may not be a potential occupational carcinogen (Ex. 148, pp. 2–4). MCIA submitted to the record a copy of the IARC monograph and recent supplement on methyl chloride, which conclude that the evidence for the carcinogenicity of methyl chloride is inadequate in both animals and humans.

In the final rule, OSHA is establishing an 8-hour TWA of 50 ppm and a 15-minute STEL of 100 ppm for methyl chloride. The Agency concludes that these two limits together will substantially reduce the significant risk of neurotoxic effects, including functional impairment, performance decrements, headaches, dizziness, slurred speech, and staggering gait, which together constitute material impairments of health. These effects have been associated with exposure to this substance at the levels permitted by OSHA's former PEL. OSHA will continue to monitor the literature on the toxicity of methyl chloride to determine whether other action is appropriate.

METHYL CHLOROFORM (1,1,1-TRICHLOROETHANE)

CAS: 71–55–6; Chemical Formula: CH_3CCl_3
H.S. No. 1255

Previously, OSHA had an 8-hour TWA limit of 350 ppm for methyl chloroform. The ACGIH has established the same TWA limit in addition to a TLV–STEL of 450 ppm; NIOSH recommends a 15-minute ceiling limit of 350 ppm. The Agency proposed to retain its 8-hour TWA limit and to add a STEL of 450 ppm; NIOSH concurs that these limits are appropriate but would express them as ceilings rather than as TWAs (Ex. 8–47, Table N7). The final rule retains an 8-hour TWA of 350 ppm and adds a STEL of 450 ppm for methyl chloroform, which is a clear, nonflammable liquid.

The primary health effects associated with exposure to methyl chloroform are anesthesia and cardiac sensitization. The oral toxicity of methyl chloroform is low, with LD_{50} values ranging from 5.7 to 12.3 g/kg for rats, mice, rabbits, and guinea pigs. This substance does, however, defat the skin on contact, causing redness and scaling (Torkelson, Oyen, McCollister, and Rowe 1958/Ex. 1–768). Skin absorption is relatively insignificant: The acute precutaneous LD_{50} in rabbits is greater than 16 g/kg, and slight, reversible irritation was observed from applications of 0.5 g/kg to rabbit skin for 90 days (Torkelson, Oyen, McCollister, and Rowe 1958/Ex. 1–768). Repeated exposures of animals to concentrations between 1000 and 10,000 ppm for three months produced anesthesia and lung and liver damage in some species, but exposure to 500 ppm of methyl chloroform vapor for seven hours daily, five days/week for six months caused no toxic changes in guinea pigs, rabbits, or monkeys (Torkelson, Oyen, McCollister, and Rowe 1958/Ex. 1–768). Other animal studies (Gehring 1968/Ex. 1–637; Plaa, Evans, and Hine 1958/Ex. 1–754; Rowe, Wujkowski, Wolf et al. 1963/Ex. 1–687) have reported that methyl chloroform has low hepatotoxicity, but cardiac sensitization has occurred at high doses (5000 to 10,000 ppm) (Rennick, Malton, Moe, and Seevers 1949/Ex. 1–864; Trochimowicz, Reinhardt, Mullin et al. 1976/Ex. 1–992). Tests in rats and mice for teratogenicity and carcinogenicity have demonstrated negative results (Schwetz, Leong, and Gehring 1975/Ex. 1–757; NIOSH 1976m, as cited in ACGIH 1986/Ex. 1–3, p. 382; Weisberger 1977/Ex. 1–694).

In humans, it has been reported that anesthetic effects may begin to occur at methyl chloroform concentrations approaching 500 ppm (Stewart, Gay, Schaffer et al. 1969/Ex. 1–529). Deaths from anesthesia and/or cardiac sensitization have been noted in employees working in confined areas (Patty 1963d/Ex. 1–856). Kramer and co-workers (1978/Ex. 1–515) conducted an epidemiological study of men and women exposed for periods ranging from several months to six years to methyl chloroform at levels that occasionally exceeded 200 ppm; when compared to matched-pair controls, no adverse exposure-related effects were found (Kramer, Ott, Fulkerson et al. 1978/Ex. 1–515).

Commenters supplied conflicting evidence to the record on the toxicity of methyl chloroform. The Workers Institute for Safety and Health (WISH) (Ex. 116, Tr. pp. 7–134, 135) noted that there is an extensive amount of recent information on this substance. In particular, WISH mentioned three recent studies (McLeod et al. 1987, Karlsson et al. 1987, and Mackay et al. 1987) that demonstrate that methyl chloroform causes chronic cardiac toxicity on long-term exposure, may have toxic effects on brain cells, and may cause behavioral changes after 3.5-hour exposures to 175 to 350 ppm. WISH believes that these studies and others warrant a further reduction in the PELs for methyl chloroform. However, the Halogenated Solvents Industry Alliance (Ex. 186) criticized these studies and believes that the PELs for methyl chloroform are appropriate.

In the final rule, OSHA is retaining its PEL of 350 ppm as an 8-hour TWA and adding a STEL of 450 ppm for methyl chloroform. The Agency concludes that this combined PEL-STEL limit will protect workers against the significant risk of narcotic and cardiac-sensitizing effects, which constitute material health impairments that are potentially associated with exposure to methyl chloride at the elevated short-term levels permitted by an 8-hour TWA limit alone.

OCTANE

CAS: 111–65–9; Chemical Formula: $CH_3(CH_2)_6CH_3$
H.S. No. 1296

OSHA's former limit for octane was 500 ppm as an 8-hour TWA. The ACGIH has a 300-ppm TWA and a 375-ppm STEL; NIOSH (1977a/Ex. 1–233) recommends a 75-ppm 10-hour TWA and a 385-ppm 15-minute ceiling limit. The proposed PELs were an 8-hour TWA of 300 ppm and a 15-minute STEL of 375 ppm, and these are the limits promulgated in the final rule. n-Octane is a colorless, flammable liquid with an odor like that of gasoline.

Mice exposed to octane concentrations of 6600 to 13,700 ppm developed narcosis within 30 to 90 minutes (Fuhner 1921, as cited in ACGIH 1986/Ex. 1–3, p. 448). Flury and Zernik (1931h, as cited in ACGIH 1986/Ex.1–3, p. 448) believed the narcotic concentration in humans to be 5000 ppm; Patty and Yant (1929, as cited in ACGIH 1986/Ex. 1–3, p. 448) placed the narcotic concentration at 8000 ppm. Based on this information, the ACGIH concluded that octane was 1.2 to 2 times more toxic than heptane, and recommended TLVs of 300 ppm TWA and 375 ppm STEL.

As discussed in more detail in Section V of the preamble and in the discussions above for the other C_5–C_8 alkanes, the NIOSH (1977a/Ex. 1–233) recommended limits for octane are based on NIOSH's belief that all C_5–C_8 alkanes present a neurotoxic hazard similar to that of n-hexane. OSHA disagrees with this conclusion and has found instead that the neurotoxic properties of n-hexane are unique among the substances in the alkane series. NIOSH continues to recommend these lower limits for all of the C_5–C_8 alkanes, including octane (Ex. 8–47, Table N2; Tr. 3–86 to 122). The AFL–CIO (Ex. 194) and the UAW (Ex. 197) made the same comments for octane as for heptane (see the discussion, above).

The Chevron Corporation (Ex. 3–896) objected to the proposed short-term exposure limit for octane on the grounds that studies showing narcosis at concentrations of 5000 and 8000 ppm do not provide a justification for a STEL. In addition, Chevron stated that, "as a practical matter, a STEL that is only 25 percent greater than the TWA value suggests a level of precision that simply does not exist in exposure assessment techniques. Variations in sampling and analytical methodologies combined with normal statistical variability in exposure patterns make it impossible to reliably distinguish between exposures that differ by only 20 to 25 percent. Intuitively, it is not reasonable to conclude that a concentration that is slightly above an acceptable 8-hour exposure level would be unsafe for a 15-minute exposure" (Ex. 3–896. p. 3).

In response to Chevron, OSHA notes that octane is considered more toxic than heptane, for which OSHA is establishing limits of 400 ppm as an 8-hour TWA and 500 ppm as a 15-minute STEL. Short-term effects have been observed in humans and animals exposed to the hexane isomers at levels below 500 ppm (Nelson, Enge, Ross et al. 1943/Ex. 1–66), and OSHA finds it appropriate to establish a STEL for octane and several other alkanes to protect against these narcotic effects. OSHA disagrees with Chevron that it is not possible to distinguish between octane exposures of 300 ppm and those of 375 ppm; although a ± 25-percent level of precision may be difficult to achieve at very low contaminant concentrations, there should be no sampling and analytical difficulty at the levels being considered here. Finally, OSHA notes that a theoretically possible, although unlikely, exposure scenario that could occur with an 8-hour TWA limit of 300 ppm alone would be an excursion of up to 9600 ppm; such an exposure could produce serious CNS effects in exposed workers. Thus, the purpose of the 375-ppm STEL is to ensure that the TWA limit is not exceeded for any substantial period of time and that exposures are effectively controlled.

In the final rule, OSHA is revising its limits for octane to 300 ppm as an 8-hour TWA and 375 ppm as a 15-minute STEL. The Agency concludes that these limits will protect workers from the significant risks of narcosis, a material health impairment that is associated with octane exposures. OSHA believes that these limits will substantially reduce these significant risks.

PENTANE

CAS: 109–66–0; Chemical Formula: C_5H_{12}
H.S. No. 1306

Previously, OSHA's limit for pentane was 1000 ppm TWA. In 1976, the ACGIH adopted a 600–ppm TLV–TWA and a 750–ppm TLV–STEL. NIOSH (1977a/Ex. 1–233; Ex. 8–47, Table N2) has recommended that workplace exposures to pentane not exceed 120 ppm as a 10-hour TWA and 610 ppm as a 15-minute short-term limit. The proposed and final rule PELs are 600 ppm as an 8-hour TWA and 750 ppm as a 15-minute STEL. Pentane, a colorless, flammable liquid with a gasoline-like odor, is usually encountered in volatile petroleum fractions, some of which are used as solvents. Pure pentrane is used as a blowing agent for plastics, in solvent extraction, and in ice manufacture.

Fairhall (1957c/Ex. 1–184) stated that narcosis and mucous membrane irritation were the only reported toxic effects resulting from exposure to pentane. The reported lethal concentration in humans is 130,000 ppm (Flury and Zernik 1931j/Ex. 1–994; Swann, Kwon, and Hogan 1974/Ex. 1–124). According to Patty and Yant (1929, as cited in ACGIH 1986/Ex. 1–3, p. 463), humans exposed for 10 minutes to 5000 ppm did not complain of any adverse symptoms.

In a report by Gaultier, Rancurel, Piva, and Efthymioc (1973/Ex. 1–123), five cases of polyneuropathy occurred among employees exposed to a solvent containing 80 percent pentane, 14 percent heptane, and 5 percent hexane. Based largely on this report, NIOSH (1977a/Ex. 1–233) recommended the same occupational limit for all C_5–C_8 alkanes as for the neurophatic agent n-hexane (350–mg/m^3 TWA and 1800-mg/m^3 15-minute short-term limits; these limits are equal to about 120-ppm TWA and 610-ppm 15-minute short-term limits for pentane).

OSHA points out that the rationale used by NIOSH in setting a limit for pentane ignores the theory that n-hexane is uniquely neuropathic via metabolism to 2,5-hexanedione, which is the same metabolite that is formed during exposure to another neuropathic agent, methyl butyl ketone (see the discussion in Section V of this preamble). OSHA finds that all C_5–C_8 alkanes are not equally toxic; the Agency concludes that a metabolite of n-hexane exhibits unique neurotoxic properties. In OSHA's view, the Gaultier, Rancurel, Piva, and Efthymioc (1973/Ex. 1–123) study does not provide specific isomer exposure data supporting the NIOSH RELs of 120 ppm (TWA) and 610 ppm (STEL).

The Chevron Corporation (Ex. 3–896) objected to the proposed STEL for pentane because, in Chevron's opinion, the health evidence did not justify this addition. However, OSHA finds that the STEL is needed to protect workers from the significant neurotoxic effects of pentane exposure by ensuring that the high short-term excursions possible in the absence of a STEL do not occur. The Workers Institute for Safety and Health (Ex. 116) and the UAW (Ex. 197) submitted the same comments on pentane as on heptane (which see).

In the final rule, the Agency is establishing an 8-hour TWA of 600 ppm and a 15-minute STEL of 750 ppm as the permissible exposure limits for pentane. OSHA concludes that these limits will protect exposed workers from the narcosis long known to be associated with pentane exposure; the Agency finds that narcosis constitutes a material health impairment within the meaning of the Act.

2-PENTANONE (METHYL PROPYL KETONE)

CAS: 107–87–9; Chemical Formula: $CH_3COC_3H_7$
H.S. No. 1307

The former OSHA limit for 2-pentanone was 200 ppm as an 8-hour TWA. The ACGIH has a 200-ppm TLV–TWA and a 250-ppm TLV–STEL; NIOSH (1978k, as cited in ACGIH 1986/Ex. 1–3,

p. 408) has recommended a 150-ppm limit as a 10-hour TWA. The proposed PELs were 200 ppm as an 8-hour TWA and 250 ppm as a 15-minute STEL, and these limits are established in the final rule. 2-Pentanone is a clear, flammable liquid with a strong odor resembling acetone and ether.

Both the ACGIH- and NIOSH-recommended limits are based on a study by Specht, Miller, Valaer, and Sayers (1940/Ex. 1–1179), which found that guinea pigs exhibited irritation and weakness on exposure to 2500 ppm, and that exposure to 5000 ppm produced narcosis and coma. The authors concluded that 2-pentanone is considerably less toxic than methyl butyl ketone but is more toxic than methyl ethyl ketone, and, in addition, is likely to be more irritating than either methyl ethyl ketone or acetone. The ACGIH-recommended limits are based on a judgment that the 200-ppm TLV–TWA and 250-ppm TLV–STEL are low enough to prevent narcosis and irritation.

NIOSH (1978k, as cited in ACGIH 1986/Ex. 1–3, p. 408) applied the findings of the Specht, Miller, Valaer, and Sayers (1940/Ex. 1–1179) study to the results of the Nelson, Enge, Ross et al. study (1943/Ex. 1–66); these latter authors reported that volunteers complained of slight irritation on exposure to 100 ppm methyl ethyl ketone. Because 2-pentanone was found by Specht, Miller, Valaer, and Sayers (1940/Ex. 1–1179) to be at least as irritating as methyl ethyl ketone, NIOSH (1978k, as cited in ACGIH 1986/Ex. 1–3, p. 408) stated that a "slight reduction" in the standard was warranted for 2-pentanone. Therefore, NIOSH recommended a 150-ppm limit for 2-pentanone, and NIOSH reiterates this recommendation in the present rulemaking (Ex. 8–47, Table N2; Tr. 3–86). No other comments were submitted regarding the health effects of 2-pentanone.

OSHA has concluded that the combination of a 200-ppm TWA and a 250-ppm STEL will work together to ensure that workplace levels are maintained at levels that will prevent the occurrence of the adverse health effects associated with exposures to this chemical. In the final rule, OSHA is establishing these limits to reduce the significant risks of narcosis, a material impairment of health, which is associated with exposures to 2-pentanone at elevated short-term levels.

STODDARD SOLVENT

CAS: 8052–41–3; Chemical Formula: C_9H_{20}
H.S. No. 1371

OSHA's former limit for Stoddard solvent was 500 ppm as an 8-hour TWA. The ACGIH has established a TLV–TWA of 100 ppm, and NIOSH (1977g, as cited in ACGIH 1986/Ex. 1–3, p. 537) recommends limits of 350 mg/m^3 as a 10-hour TWA and 1800 mg/m^3 as a 15-minute ceiling for all refined petroleum solvents; these limits correspond approximately to a 60-ppm TWA and a 310-ppm STEL, respectively. Stoddard solvent is a refined petroleum solvent having a flash point in the range of 102 to 110 °F, a boiling point in the range of 154 to 202 °C, and containing 65 percent or more C_{10} and higher-molecular-weight hydrocarbons. OSHA proposed to reduce its 8-hour TWA to 100 ppm, and the final rule promulgates this limit. NIOSH (Ex. 8–47, Table N1) agreed with the Agency's selection of this PEL.

The former OSHA limit of 500 ppm (equivalent to the limit in the 1968 ACGIH TLV list) was based largely on analogy to the irritant and narcotic effects of gasoline vapor in humans (ACGIH 1966/Ex. 1–13, pp. 176–177). The revised ACGIH limit of 100 ppm was based on a report by Carpenter, Geary, Myers et al. (1978/Ex. 1–301), which found slight kidney damage among rats exposed to 330 ppm Stoddard solvent for 65 days. The ACGIH TLV for Stoddard solvent was calculated from the TLVs for nonane and trimethyl benzene, the major components of Stoddard solvent (ACGIH 1986/Ex. 1–3); the TLV for nonane is 200 ppm, based on the Carpenter, Geary, Myers et al. (1978/Ex. 1–301) study's findings of a non-effect level for nonane in rats of 590 ppm, while the TLV for trimethyl benzene is 25 ppm, because there is evidence that humans exposed to the isomers of trimethyl benzene exhibited central nervous system effects (ACGIH 1986/Ex. 1–3).

THE ACGIH (1986/Ex. 10–3, p. 537) notes that guinea pigs exposed for 30 eight-hour days to 290 ppm Stoddard sovlent developed congestion and emphysema of the lungs. The eye irritation threshold in humans is approximately 150 ppm for 15 minutes (ACGIH 1986/Ex. 1–3, p. 537).

The NIOSH limits of a 350-mg/m^3 (60-ppm) TWA and an 1800-mg/m^3 (310-ppm) 15-minute short-term limit are derived from NIOSH's recommended limits for all of the C_5–C_8 alkanes; NIOSH recommended the same limit for Stoddard solvent as for all C_5–C_8 alkanes both because of the lack of scientific data on Stoddard solvent's chronic effects and because of a report of polyneuropathy occurring among workers exposed to jet fuels containing mixtures of kerosene and gasoline. NIOSH reasoned that, although the C_5–C_8 alkanes present in jet fuel may have been implicated, it was possible that the heavier hydrocarbon components may also have been responsible. Thus, the NIOSH recommended limits for Stoddard solvent reflect a concern that higher-molecular-weight hydrocarbons may be neuropathic. However, no evidence exists that the C_{10} and higher molecular weight hydrocarbons cause neuropathies. NIOSH has re-examined the health evidence for Stoddard solvent in this rulemaking and concurs with OSHA that the 100-ppm 8-hour TWA limit is appropriate for this substance (Ex. 8–47, Table N1). Several commenters (TR. 7–70 to 7–95; Exs. 46, 116, 194, 197) urged OSHA to reevaluate the final rule's limits for this substance because recent evidence points to hepatic and hematopoietic effects. OSHA is aware of the emerging literature and will monitor developments in the future.

In the final rule, OSHA is establishing an 8-hour TWA of 100 ppm to reduce the significant risk, of eye irritation, narcosis, polyneuropathy, and kidney damage, all of which constitute material health impairments that have been demonstrated to occur in either humans or animals at levels well below the former PEL. OSHA finds that the study of Carpenter and co-workers (1978/Ex. 1–301) in animals and the study reported by the ACGIH showing that exposed workers develop eye irritation at levels of 150 ppm and above clearly indicate that a reduced PEL is needed for Stoddard solvent to diminish these significant occupational risks.

STYRENE

CAS: 100–42–5; Chemical Formula: C_6H_5CH-CH_2
H.S. No. 1372

OSHA's former exposure limits for styrene (listed in 29 CFR 1910.1000, Table Z–2) were 100 ppm as an 8-hour TWA, 200 ppm as a STEL, not to be exceeded for more than 5 minutes in any 3-hour period, and 600 ppm as a ceiling limit. OSHA proposed revising these limits to 50 ppm as an 8-hour TWA and 100 ppm as a 15-minute STEL, based on both the ACGIH TLVs and the NIOSH RELs, which are identical. NIOSH (Ex. 150, Comments on Styrene) concurs that these limits are appropriate for styrene, and they are established in the final rule. Styrene monomer is a colorless, oily liquid with an aromatic odor.

In the proposal, styrene was located in the cancer category; in the final rule, it has been moved into the narcotics section, for the reasons discussed below. According to the generic methodology used by OSHA to group the 428 substances included in this rulemaking, substances were grouped

according to the guidelines given by the ACGIH for assigning an appropriate exposure limit for a particular substance. In other words, if the ACGIH noted that a particular TLV was designed to protect against irritant effects, that substance was classified by OSHA in the sensory irritant category. This classification scheme was chosen by OSHA because it facilitated the rulemaking process (made unusually complex by the broad scope of the issues addressed) and made the discussion of hundreds of substances easier. However, as is often the case with classification schemes, this methodology oversimplifies the issues, particularly in those situations where a substance has more than one serious health effect.

Styrene is a case in point. This widely used substance is an irritant, a narcotic, and a neuropathic agent; some studies also show that animals exposed to styrene vapor develop tumors. the ACGIH *Documentation* (1986/Ex.1-3) for styrene states:

> [A] time-weighted average TLV of 50 ppm, one-tenth the lowest concentration possibly causing *lymphoid or hematopoietic tumors* in female rats, and a STEL of 100 ppm are suggested as reasonable limits [for styrene] (emphasis added) (ACGIH 1986/Ex. 1-3, p. 539).

Because the ACGIH limit had been set with reference to tumorigenicity (notwithstanding the lack of an A1 or A2 cancer designation), styrene fell into the category of carcinogens for the purposes of the proposal (53 FR 21202).

Many commenters objected to the proposal's classification of styrene as a carcinogen (Exs. 3-741, 3-742, 3-1059, L3-1312B, 8-12, 8-32, 8-48, 8-54, 34, 36, 103, 155, and 187; Tr. 8/3/88, pp 5-9 to 5-127; Tr. pp. 11-265, 11-266). For example, the Styrene Information and Research Council (SIRC) stated:

> Regarding the long-term animal studies on styrene * * * there have been nine * * * seven of which were via the oral route and two via inhalation * * * . All of these studies showed either no evidence of cancer or gave inconclusive results due to study limitations, e.g., faulty study design, high background tumor incidence and/or high morbidity in test and control groups of animals (Ex. 3-742, p. 10).

Other commenters echoed the view of the SIRC. For example, a paper prepared by the Epidemiology Department of the Dow Chemical Company and reported on in Dow's prehearing submission (Ex. 3-741, p. 55) concludes: "[O]verall these data do not support a causal link between lymphatic and hematopoietic cancer and styrene." Dr. Gregory Bond (Ex. 103 and testimony) also criticized the epidemiology studies relied on by OSHA in the proposal, as did the Chemical Manufacturers Association (Ex. 8-54). J. Roger Crawford, Director of Environmental Control for the Outboard Marine Corporation, a manufacturer of outboard and inboard engines, lawn care equipment, and marine products, commented that OSHA's conclusion in the proposal about the carcinogenicity of styrene "is clearly outside the mainstream of most scientific opinion" (Ex. 8-12, p. 3).

In posthearing testimony on behalf of the SIRC, Dr. Robert G. Tardiff, Director of Versar, Incorporated's Risk Focus Division, described the comments of EPA's Science Advisory Board (SAB) on a draft EPA *Water Criteria Document on Styrene*. Dr. Tardiff reported that the SAB had advised EPA to consider styrene a "possible human carcinogen (Category C) at best" (Ex. 34, p. 4). Dr. Tardiff further commented that the Category C classification "would generally lead EPA to regulate the compound based on protection against non-cancer pathology" (Ex. 34, pp. 4-5).

However, EPA's *Guidelines for Carcinogen Risk Assessment* (51 FR 33992) interpret the meaning of a Category C designation somewhat differently than does Dr. Tardiff. In a letter dated March 9, 1988 from the SAB to EPA's Administrator, Lee M. Thomas (Attachment to Ex. 124), the SAB makes clear that factors other than category are important to consider for regulatory purposes:

> From a scientific point of view, it seems inappropriate for EPA and other agencies to regulate substances that are classified as B2 [probable human carcinogens] and not to consider regulation of compounds classified as C * * *. A substance classified as C (limited evidence in animals) for which human exposure is high may represent a much greater potential threat to human health [than substances with classifications of B2, B1, or A where exposures are lower].
>
> EPA and other agencies * * * may, therefore, wish to take steps to reduce high exposures to substances in the C category whenever there appears to be a potentially significant threat to human health (in the sense [where risk estimates are] * * * above the threshold where regulation may be judged appropriate) (Attachment to Ex. 124).

Several animal and human studies have suggested that styrene may be a carcinogen. A nested case-control study conducted by McMichael, Spirta, Gamble, and Tousey (1976/Ex. 1-206) found significantly increased risks of lymphatic and hematopoietic cancer, lymphatic leukemia, and stomach cancer among workers exposed to both styrene and butadiene. A retrospective cohort mortality study by Meinhardt, Lemen, Crandall, and Young (1982/Ex. 1-199), also among workers exposed concurrently to styrene and butadiene, reported an excess risk of leukemia and aleukemia. In a study sponsored by the Chemical Manufacturers Association (Dow 1978, as cited in EPA 1987/Ex. 1-836), male and female Sprague-Dawley rats were exposed to styrene vapor at concentrations of 600 to 1200 ppm, six hours per day, five days per week, for 18 or 20 months. The higher exposure level was reduced to 1000 ppm after the first two months of exposure because of excessively reduced weight in the male rats. A statistically significant increased incidence of mammary tumors was reported in low-dose female rats (7 of 87) compared with controls (1 of 85); no increase in mammary tumors was reported among high-dose female rats. The authors questioned the significance of this response, since historical control animals from the same laboratory showed a higher background incidence of mammary tumors than did the controls used in this study.

In a 1979 NCI study (NCI 1979b/Ex. 1-948), male and female B6C3F1 mice and Fischer 344 rats were treated by gavage five days per week for 78 weeks (low-dose rat groups were treated for 103 weeks). The study was terminated at 91 weeks for mice and at 104 to 105 weeks for rats. Dose-related increases in alveolar/bronchiolar adenomas and carcinomas were observed only in the low-dose (150 mg/kg) and high dose (300 mg/kg) male mice; the incidence of tumors for vehicle controls, low-dose, and high-dose male mice was 0/20, 6/44, and 9/43, respectively. Although the historical incidence of tumors among untreated controls was 12 percent (32/271), the historical incidence of vehicle controls was 0/40.

However, the human studies cannot be used to demonstrate styrene's carcinogenicity because there were confounding exposures in these cohorts to butadiene, a substance identified by the NTP as carcinogenic. The animal studies also have limitations, such as high background rates of cancer in the controls and non-treatment-related mortality in some of the test animals.

Thus, at this time, OSHA believes that the current evidence on styrene's carcinogenicity does not support its classification in the final rule as a carcinogen. OSHA has reviewed additional evidence and has determined that the most appropriate basis for classifying styrene in this rulemaking is the substance's demonstrated narcotic effects. In its criteria document (1983a), NIOSH agrees that styrene is primarily a narcotic and central-nervous-system toxin.

The principal health effects due to styrene exposure involve the central nervous system. These effects include subjective complaints of headache, fatigue, dizziness, confusion, drowsiness, malaise, difficulty in concentrating, and a feeling of intoxication * * *. There have also been reports of liver injury, peripheral nervous system dysfunction, abnormal pulmonary function, chromosomal changes, reproductive effects, and carcinogenicity related to styrene exposures. Although data concerning these latter adverse effects are not well defined at this time, they do provide cause for concern (NIOSH 1983a, p. 150).

Accordingly, OSHA has placed the health-effects discussion for styrene in the preamble section labeled "Narcotic Effects" in this final rule.

OSHA proposed to reduce its former exposure limits for styrene to 50 ppm as an 8-hour TWA and 100 ppm as a 15-minute STEL. The Agency finds clear evidence, based on styrene's narcotic effects, to support these limits. Richard Olsen, representing the Dow Chemical Company, agrees, and stated at the hearing that 50 ppm is likely to be the most "appropriate" limit for styrene (Tr. 3, pp. 250, 251). There is a considerable body of health-effects information in humans for styrene in the toxicological literature. Subjects exposed at 800 ppm for four hours experienced eye and throat irritation and also reported listlessness, drowsiness, and impaired balance (NIOSH 1983a, p. 150). At a concentration of 376 ppm, five human volunteers experienced eye and respiratory tract irritation within 20 minutes and demonstrated decrements in motor function (NIOSH 1983a, p. 150). Three subjects exposed to 100 ppm of styrene for 90 minutes had slower reaction times; on repeated exposure, sleepiness, fatigue, headache, difficulty in concentration, malaise, nasal irritation, and nausea occurred in another group of subjects (NIOSH 1983a, p. 150).

Effects attributable to central nervous system depression were seen in a six-week study involving human subjects exposed to 20, 100, or 125 ppm styrene; the authors of the study reported visual-evoked-response and electroencephalogram changes in these subjects (NIOSH 1983a, p. 150). Other studies report irritation of the eyes and throat at concentrations ranging from 1 to 100 ppm (NIOSH 1983a, p. 151).

Workers in reinforced plastics (RP) facilities in many countries have also evidenced narcotic effects as a consequence of styrene exposure. Swedish, Dutch, and Czechoslovakian workers in RP plants complained of headache, fatigue, drowsiness, giddiness, and dizziness at exposure levels in the range of 4 to 195 ppm (NIOSH 1983a, p. 151).

Respiratory effects were observed in U.S. RP workers exposed to from 9 to 111 ppm styrene; symptoms included wheezing, shortness of breath, and chest tightness. Another study showed a significantly greater number of RP workers with abnormal pulmonary function when compared with workers from a nonstyrene facility (NIOSH 1983a, p. 154).

NIOSH concluded, based on its extensive review of the health-effects literature for styrene, that an 8-hour TWA exposure limit of 50 ppm was appropriate to protect against the health effects observed in workers exposed to styrene at levels of 100 ppm and below. NIOSH also recommends a STEL of 100 ppm for styrene to prevent acute eye and upper-respiratory-tract irritation (NIOSH 1983a, p. 156). The State of New Jersey's Department of Public Health (Ex. 144) urged OSHA to derive a PEL for styrene on the basis of EPA's IRIS data, but this approach was criticized by other commenters (Ex. 187). The use of IRIS data for limit-setting purposes is addressed in Section VI.A of the preamble. At the hearing, representatives of the International Chemical Workers Union urged OSHA to adopt a lower PEL because considerable risk remains at the 50-ppm level (Tr. 9, p. 216). However, the AFL-CIO (Ex. 194) agrees with NIOSH that the 50-ppm and 100-ppm TWA and STEL limits are appropriate.

OSHA finds that workplace exposures to styrene are associated with health effects ranging from narcosis to neuropathies and irritation, which together constitute material impairments of health. The Agency finds that an 8-hour TWA of 50 ppm and a STEL of 100 ppm are necessary to protect against these significant risks of material health impairment. The Agency also notes that large chemical companies (for example, Rohm and Haas and the Dow Chemical Company) have already established internal corporate limits of 25 to 50 ppm (8-hour TWAs) for styrene to protect their workers from the range of serious health effects associated with exposure to this substance (Ex. 25, Appendix II, pp. 1–3).

Some commenters (Ex. 155; Tr. p. 10–111) pointed to the fact that the State of Washington has not yet adopted a 50-ppm limit for styrene as evidence of this limit's infeasibility; however, OSHA notes that Stephen Cant, for the State of Washington's Department of Public Health, stated that his department was monitoring the health evidence for styrene and considered the State's 100-ppm limit an "incremental improvement" (Tr. 2, pp. 105, 106).

OSHA notes that, with the exception of two operations in a single industry (i.e., the boat-building industry), these limits have been found to be achievable with engineering and work-practice controls in all styrene-using operations, including styrene manufacture and other reinforced-plastics operations. OSHA finds that general dilution ventilation, local exhaust ventilation, and process enclosure can be used effectively in tub, shower, and diving board manufacturing because the size and configuration of these items lend themselves to effective control. However, in two operations, manual layup and sprayup, in the boat-building industry, there is insufficient data in this record to indicate that compliance can generally be achieved with engineering and work-practice controls. For these boat-building operations, employers may use any combination of engineering controls, work practices, and respiratory protection to achieve these limits (see the discussion in Section VII of this preamble). For these operations, engineering controls and work practices will only be required to achieve full compliance with the final rule's PELs in cases where the Assistant Secretary can demonstrate that engineering controls and work practices can generally achieve these limits. In the absence of such a finding, the employer must nonetheless use engineering controls and work practices to achieve compliance with the Agency's former PELs for styrene.

TOLUENE

CAS: 108-88-3; Chemical Formula: $C_6H_5CH_3$
H.S. No. 1397

The former OSHA standard for toluene was 200 ppm as an 8-hour TWA limit, with a 300-ppm ceiling (not to be exceeded for more than 10 minutes in any eight-hour period), and a 500-ppm peak. The ACGIH has an exposure limit for toluene of 100 ppm as an 8-hour TWA and 150 ppm as a 15-minute STEL; NIOSH recommends a 100-ppm 8-hour TWA and a 10-minute ceiling of 200 ppm. The proposed PELs were 100 ppm as an 8-hour TWA and 150 ppm as a STEL; NIOSH (Ex. 8-47, Table N1) concurs with these limits, which are established in the final rule. Toluene is a flammable, colorless liquid with an aromatic hydrocarbon odor.

The acute toxicity of toluene in animals is greater than that of benzene. Patty (1963b, as cited in ACGIH 1986/ Ex. 1-3, p. 578) reports that the lethal doses of toluene and benzene in mice are 10,000 and 14,000 ppm, respectively.

The oral LD_{50} for toluene in rats is 7.53 ml/kg (Smyth, Carpenter, Weil et al. 1969/Ex. 1–442). Exposure of rats to 2500 or 5000 ppm of toluene caused a temporary decrease in white cell count but no evidence of damage to the blood-forming organs or the liver. Fairhall (1957d, as cited in ACGIH 1986/Ex. 1–3, p. 578) stated that severe toluene exposure can cause a marked drop in the red blood cell count and partial destruction of the blood-forming elements of the bone marrow, but other researchers report that numerous animal studies indicate that toluene is not a bone marrow toxin (Gerarde 1960c, as cited in ACGIH 1986/Ex. 1–3, p. 578).

A study by Greenberg, Mayers, Heinmann, and Moskowitz (1942/Ex. 1–325) reported that painters exposed to toluene levels of 100 to 1100 ppm exhibited enlarged livers, a moderate decrease in red blood cell counts, enlarged red blood cells, and absolute lymphocytosis, but no leukopenia. Wilson (1943/Ex. 1–403) observed 1,000 workers exposed to toluene at levels ranging from 50 ppm to 1500 ppm for periods of one to three weeks. One hundred of these workers developed symptoms severe enough to require hospitalization. At levels less than 200 ppm, 60 of these employees experienced headache, fatigue, and lack of appetite. Those workers exposed to 200 to 500 ppm toluene experienced headache, nausea, bad taste in the mouth, lassitude, temporary amnesia, impaired coordination, and anorexia. Levels of exposure from 500 to 1500 ppm resulted in nausea, headache, dizziness, anorexia, marked loss of coordination, diminished reaction time, pronounced weakness, and heart palpitations. Red cell counts were also decreased, and two cases of aplastic anemia required lengthy hospital treatment; however, the author noted that he could not rule out the possibility that benzene contamination of the toluene was the cause of these effects. Aplastic anemia (including one fatal case) has been noted in six glue sniffers; toluene was the base solvent in the glue (Powars 1965/Ex. 1–433). A man who had inhaled toluene regularly at unspecified levels for 14 years developed permanent encephalopathy (Knox and Nelson 1966/Ex. 1–421).

von Oettingen, Neal, Donahue et al. (1942/Ex. 1–875) exposed human volunteers to toluene levels ranging from 50 ppm to 800 ppm for 8 hours/day. These authors report that exposures to 50 ppm cause drowsiness and headaches and that exposures at 100 ppm result in sleepiness, moderate fatigue, and headaches. At 200 ppm, effects included impairment of coordination and reaction times. Later studies by Ogata, Tomokuni, and Takatsuka (1970/Ex. 1–352) showed an increase in reaction time, a decrease in pulse rate, and a decrease in systolic blood pressure in humans exposed to 200 ppm toluene for seven hours.

The Chevron Corporation (Ex. 3–896) objected to the short-term exposure limit for toluene as being unjustified by either the discussion in the preamble or that in the ACGIH *Documentation* (1986/Ex. 1–3). Chevron also urged OSHA to clarify the proposal's discussion of blood dyscrasias occurring as a result of toluene exposure because, according to Chevron:

> [T]he majority of later studies show no such evidence [of blood dyscrasias]. Due to the tighter specifications for benzene contamination of toluene, we question whether blood dyscrasias will occur (Ex. 3–896, p. 14).

As discussed above in connection with octane and pentane, OSHA finds that a short-term exposure limit is necessary to ensure that workers are not exposed at the elevated levels possible with a TWA limit alone. Levels only slightly above the 8-hour TWA may cause incoordination and amnesia. For example, workers could be exposed to toluene at levels as high as several hundred ppm if the 8-hour TWA limit was promulgated alone. In addition, OSHA notes that the Agency has always had a short-term and ceiling limit for toluene, to protect against this substance's narcotic and neuropathic effects; OSHA continues to find a short-term limit necessary to ensure that workers do not experience the effects seen at levels only slightly above 100 ppm. On the question of blood dyscrasias, OSHA noted in the preamble to the proposal that the author of the study in question (Wilson 1943/Ex. 1–403) himself noted that benzene contamination may have been the cause of these blood effects; OSHA agrees that this may have been the case.

NIOSH (Ex. 150, Comments on Toluene) reports that "[s]everal recent studies indicate measurable biological changes in liver function" as a consequence of exposures to 100 ppm (Seiji et al. 1987) but not at 46 ppm (Yin et al. 1987). NIOSH also states that volunteers' performance on psychological test scores was reduced during 100-ppm exposures to toluene and that these volunteers expressed exposure-related complaints. NIOSH also notes that there is some evidence that toluene causes reproductive effects at levels currently being experienced in the workplace (NIOSH, Ex. 150, Comments on Toluene). NIOSH concluded that "there are significant health effects at the * * * [former] PEL of 200 ppm which will be reduced by the * * * [final rule] PEL of 100 ppm." The New Jersey Department of Health, represented by Dr. Rebecca Zagriniski, also notes that there are more recent studies on toluene (Tr. 11–266).

In the final rule, OSHA is establishing an 8-hour TWA PEL of 100 ppm and a STEL of 150 ppm for toluene. The Agency concludes that studies clearly indicate that a significant risk of hepatotoxic, behavioral, and nervous system effects exists at toluene levels substantially at or only slightly above the Agency's former PEL. OSHA finds that the new limits will protect workers against the significant risk of serious health effects that have been demonstrated to occur even during less than full-shift exposures to toluene.

TRICHLOROETHYLENE

CAS: 79–01–6; Chemical Formula: $CCl_2=CHCl$

H.S. No. 1406

OSHA's former limit for trichloroethylene, adopted from the American National Standards Institute, was 100 ppm TWA, 200 ppm as a ceiling limit not to be exceeded for more than five minutes every two hours, and 300 ppm as a peak limit. The proposed PEL for trichloroethylene was 25 ppm as an 8-hour TWA, and NIOSH (Ex. 8–47, Table N1) supported the proposed limit, which is consistent with the NIOSH REL. The ACGIH has a 50-ppm TLV–TWA and a 200-ppm TLV–STEL for trichloroethylene. Based on its review of the record evidence, OSHA has determined that a 50-ppm TWA PEL and 200-ppm STEL are appropriate limits for trichloroethylene; the final rule establishes these limits. Trichloroethylene is a colorless, nonflammable, noncorrosive liquid with the sweet odor characteristic of some chlorinated hydrocarbons.

The ACGIH (1986/Ex. 1–3) cited several studies establishing that trichloroethylene primarily affects the central nervous system and liver; some of these studies have indicated that chronic exposure to less than 100 ppm trichloroethylene is associated with a variety of nervous disturbances. Haas (1960, as cited in ACGIH 1986/Ex. 1–3, p. 595) and Grandjean, Muchinger, Turrian et al. (1955/Ex. 1–324) reported nervous symptoms among workers exposed for five years or more to trichloroethylene concentrations ranging from 1 to 335 ppm; the frequency of complaints increased when average exposures exceeded 40 ppm. Bardodej and

Vyskocil (1956/Ex. 1–461) also reported symptoms of trichloroethylene poisoning, including tremors, giddiness, anxiety, and alcohol intolerance, among workers exposed above 40 ppm. In contrast, controlled laboratory experiments with human subjects exposed for up to several days to 100 or 200 ppm have generally reported no behavioral or subjective responses. The ACGIH concluded that, although the symptoms reported by workers are subjective and commonly found among individuals having no chemical exposure, the consistency of the reports "suggests the possibility of some subjective complaints as concentrations exceed about 50 ppm" (ACGIH 1986/Ex. 1–3, p. 596). Therefore, the ACGIH recommended a TLV–TWA of 50 ppm and a TLV–STEL of 200 ppm for trichloroethylene to minimize symptoms of headache, fatigue, and irritability.

The ACGIH (1986/Ex. 1–3) also reviewed some of the carcinogenicity data on trichloroethylene. In an NCI bioassay (1976b/Ex. 1–168), mice given trichloroethylene by gavage developed hepatocellular carcinomas, but rats did not. The species difference in response was attributed to a difference in the way trichloroethylene is metabolized between the mouse and rat (Stott, Quast, and Watanabe 1982/Ex. 1–833). An inhalation study in mice, rats, and Syrian hamsters (Henschler, Romen, Reichert et al. 1980/Ex. 1–330) found only an increase in the occurrence of malignant lymphomas in mice, which the authors attributed to the strain of mouse used (NMRI). The ACGIH also cited a number of epidemiologic investigations having cohorts as large as 7,688 workers, in which no correlation between cancer mortality and exposure to trichloroethylene was found (Novotna, David, and Malek 1971, as cited in ACGIH 1986/Ex. 1–3, p. 595; Axelson, Andersson, Hogstedt et al. 1978/Ex. 1–713; Tola, Vilhunen, Jarvinen, and Korkala 1980/Ex. 1–391).

After reviewing all of the available health data, NIOSH (1978m/Ex. 1–1121) concluded that the results of the NCI (1976b/Ex. 1–168) gavage study indicate trichloroethylene (TCE) to be a potential human carcinogen, although NIOSH noted that TCE was "not considered to be a potent carcinogen." NIOSH also stated that a 100-ppm limit would not protect against the neuropathic symptoms, such as headache and fatigue, caused by exposure to trichloroethylene. In support of this conclusion, NIOSH (1978m/Ex. 1–1121) cited three health hazard evaluations conducted in facilities using trichloroethylene as a degreasing agent. In all three facilities, employees consistently experienced symptoms of dizziness, fatigue, nausea, headache, sensory irritation, and difficulty in breathing. Personal TWA exposures to trichloroethylene ranged from 37 to 112 ppm in one plant, 10 to 100 ppm in the second plant, and 10 to 95 ppm in the third plant. NIOSH (1978m/Ex. 1–1121) concluded that these reports documented the presence of adverse effects caused by acute exposure to trichloroethylene at levels of one-fourth to one-half the 100-ppm OSHA limit, at 25 to 50 ppm.

NIOSH recommended a 25-ppm TWA limit for trichloroethylene based on the health hazard reports described above as well as on a NIOSH evaluation of several NIOSH industrial hygiene reports showing that degreasing operations, including those using open-top tanks, are able to achieve 25 ppm uniformly by the use of engineering controls. NIOSH reasoned that these open-tank operations would be among the most difficult of all TCE-using operations to control.

Since publication of the NIOSH (1978m/Ex. 1–1121) report, several recent bioassays on trichloroethylene have been published and are currently being reviewed by EPA. Fukuda, Takemoto, and Tsuruta (1983/Ex. 1–1109) exposed female rats and mice to 50, 150, or 450 ppm trichloroethylene for 103 weeks and reported an increased incidence of lung tumors among mice only. Maltoni, Lefemine, and Cotti (1986/Ex. 1–1160) exposed rats and mice to 100, 300, or 600 ppm trichloroethylene and reported a significant increase of renal adenocarcinomas and Leydig cell tumors in rats, as well as a significant increase in hepatomas and lung tumors in mice. In 1986, the NTP reported an increase in the incidence of kidney tumors in rats given trichloroethylene by gavage; however, the NTP considered the tumor response to be weak (3 of 49 animals) and reported that the results were only statistically significant after corrections for high mortality were made.

Based on the information discussed above, OSHA proposed to revise the PEL for trichloroethylene to 25 ppm as an 8-hour TWA. The proposed limit was supported by NIOSH (Ex. 8–47) and by the AFL–CIO (Ex. 194), which consider trichloroethylene a potential carcinogen. However, the Dow Chemical Company objected to this proposed limit on the grounds that:

> OSHA does not provide justification for reduction of the PEL to 25 ppm based on CNS effects. Although NIOSH (1978m/Ex. 1–1121) mentions [the] CNS effects of trichloroethylene, the 25-ppm REL was not based on concern for these effects * * *. After reviewing the data on the reported [CNS and subjective response] effects of TCE, ACGIH concluded [that] a 50-ppm TWA protects workers from potential adverse effects (Ex. 3–741, pp. 61–62).

Dow also pointed out that neither the ACGIH nor IARC has classified trichloroethylene as a potential carcinogen and that EPA's Science Advisory Board concluded that the weight of evidence for TCE's carcinogenicity "lies on a continuum between their categories B2 [probable human carcinogen] and C [possible human carcinogen]" (Ex. 3–741, p. 62). Dow concluded:

> Since justification for reduction of the PEL below that recommended by ACGIH has not been provided, based on either CNS effects or carcinogenicity, we recommend adoption of the ACGIH TWA of 50 ppm with a 200-ppm STEL * * * (Ex. 3–741, p. 63).

The Halogenated Solvents Industry Alliance (Ex. 8–89, pp. 3–18) expressed an opinion similar to that of Dow Chemical.

In its posthearing submission, Dow submitted the written findings of the EPA's Science Advisory Board (SAB) on trichloroethylene (letter dated March 9, 1988 to Lee M. Thomas, Administrator of EPA, Ex. 106D). In this letter, the SAB concluded that "[t]richloroethylene has the potential to cause cancer in humans, but its potency is low." The Science Advisory Board also stated:

> The endpoints with the most biological plausibility, based upon what is known about the effects of structurally related compounds, are liver and lung tumors in mice and renal tumors in rats * * *. While [the incidence of these tumors] is clearly in excess, [it does] * * * not approach the incidence of 100 percent that occurred for chloroform, for example. This suggests a lower or more moderate potency for trichloroethylene (Ex. 106D).

OSHA believes that the evidence described above supports OSHA's preliminary conclusion in the NPRM (53 FR 21013) that the former 100-ppm TWA PEL for trichloroethylene is insufficiently protective against CNS effects and, further, that exposure to trichloroethylene may present a possible carcinogenic hazard. However, OSHA concludes that the evidence for adverse CNS effects below concentrations of 50 ppm is equivocal; exposures exceeding 50 ppm were found in each of the facilities studied by NIOSH in which symptoms of CNS disturbances were reported. Furthermore, OSHA finds that it is premature to establish a PEL for trichloroethylene based on evidence of its carcinogenicity, given the uncertainties in the evidence. Therefore,

OSHA concludes that it is appropriate at this time to establish a TWA PEL of 50 ppm and a STEL of 200 ppm to reduce the significant risk of adverse CNS effects that are associated with exposure to trichloroethylene at the former OSHA limits. The Agency considers the adverse effects resulting from exposure to trichloroethylene to be material impairments of health. Accordingly, the Agency is establishing a 50 ppm TWA PEL and 200 ppm STEL for trichloroethylene in the final rule.

Conclusions for This Group of Narcotic Agents

OSHA concludes that workers exposed to these narcosis-causing substances in the workplace are at significant risk of experiencing a broad range of narcotic effects, including loss of consciousness, uncoordinated movements, inability to concentrate, drowsiness, irritability, poor judgement, and inappropriate behavior. These highly undesirable and potentially serious health effects, which are viewed by OSHA as material impairments of health, additionally have the potential to cause serious workplace accidents and injuries because they interfere with reaction times, muscle coordination, and the ability to make good decisions and exercise good judgment. The new or revised exposure limits being established by OSHA in the final rule will protect employees from experiencing these significant risks in their places of work and will contribute to a substantial reduction in these risks.

3. Substances for Which Proposed Limits Are Based on Avoidance of Sensory Irritation

Introduction

Exposure to many chemical agents is associated with the development of sensory irritation, which is initiated when these substances come into contact with mucous membrances or skin. Limits have been set for a large group of chemicals on the basis of their sensory irritant effects. These substances, which number 79, are shown in Table C3–1, along with their former OSHA limits, the limits proposed by OSHA in the June 7, 1988 NPRM, and the final exposure limits being promulgated today. For five of these chemicals, OSHA is reducing the 8-hour TWA and for an additional eight, the Agency is both reducing the 8-hour limit and adding a STEL. In 21 cases, the 8-hour limit remains unchanged but a STEL is being added. In eight instances, a ceiling is being deleted, and this limit is being replaced by an 8-hour TWA and/or STEL value; in five instances, a TWA limit is being deleted and a ceiling value added in its place. For one chemical, methyl n-amyl ketone, OSHA is retaining its existing PEL. Thirty-one of these substances were previously unregulated by OSHA, and for these, the Agency is establishing 8-hour limits, 8-hour limits supplemented by a STEL, or ceiling limits.

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TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1001 Acetaldehyde	75-07-0	200 ppm TWA	100 ppm TWA 150 ppm STEL	100 ppm TWA 150 ppm STEL
1002 Acetic acid	64-19-7	10 ppm TWA	10 ppm TWA 15 ppm STEL	10 ppm TWA
1004 Acetone	67-64-1	1000 ppm TWA	250 ppm TWA	750 ppm TWA 1000 ppm STEL
1007 Acrolein	107-02-8	0.1 ppm TWA	0.1 ppm TWA 0.3 ppm STEL	0.1 ppm TWA 0.3 ppm STEL
1010 Allyl alcohol	107-18-6	2 ppm TWA, Skin	2 ppm TWA, Skin 4 ppm STEL	2 ppm TWA, Skin 4 ppm STEL
1012 Allyl glycidyl ether	106-92-3	10 ppm Ceiling	5 ppm TWA 10 ppm STEL, Skin	5 ppm TWA 10 ppm STEL
1013 Allyl propyl disulfide	2179-59-1	2 ppm TWA	2 ppm TWA 3 ppm STEL	2 ppm TWA 3 ppm STEL
1021 Ammonia	7664-41-7	50 ppm TWA	25 ppm TWA 35 ppm STEL	35 ppm STEL

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1022 Ammonium chloride fume	12125-02-9	--	10 mg/m^3 TWA 20 mg/m^3 STEL	10 mg/m^3 TWA 20 mg/m^3 STEL
1036 Borates, tetra, Na (anhydrous)	1330-43-4	--	1 mg/m^3 TWA	10 mg/m^3 TWA
1037 Borates, tetra, Na (decahydrate)	1303-96-4	--	5 mg/m^3 TWA	10 mg/m^3 TWA
1038 Borates, tetra, Na (pentahydrate)	12179-04-3	--	1 mg/m^3 TWA	10 mg/m^3 TWA
1042 Bromine	7726-95-6	0.1 ppm TWA	0.1 ppm TWA 0.3 ppm STEL	0.1 ppm TWA 0.3 ppm STEL
1045 2-Butanone (MEK)	78-93-3	200 ppm TWA	200 ppm TWA 300 ppm STEL	200 ppm TWA 300 ppm STEL
1047 n-Butyl acetate	[illegible]	150 ppm TWA	150 ppm TWA 200 ppm STEL	150 ppm TWA 200 ppm STEL
1053 n-Butyl lactate	138-22-7	--	5 ppm TWA	5 ppm TWA
1054 n-Butyl mercaptan	109-79-5	10 ppm TWA	0.5 ppm TWA	0.5 ppm TWA

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1064 Caprolactam (dust)	105-60-2	--	1 mg/m^3 TWA 3 mg/m^3 STEL	1 mg/m^3 TWA 3 mg/m^3 STEL
1065 Caprolactam (vapor)	105-60-2	--	5 ppm TWA 10 ppm STEL	5 ppm TWA 10 ppm STEL
1077 Cesium hydroxide	21351-79-1	--	2 mg/m^3 TWA	2 mg/m^3 TWA
1079 Chlorine	7782-50-5	1 ppm Ceiling	0.5 ppm STEL	0.5 ppm TWA 1.0 ppm STEL
1083 Chloroacetyl chloride	79-04-9	--	0.05 ppm TWA	0.05 ppm TWA
1084 o-Chlorobenzylidene malononitrile	2698-41-1	0.05 ppm TWA	0.05 ppm Ceiling, Skin	0.05 ppm Ceiling, Skin
1105 Cyanogen	460-19-5	--	10 ppm TWA	10 ppm TWA
1106 Cyanogen chloride	506-77-4	--	0.3 ppm Ceiling	0.3 ppm Ceiling
1119 Dibutyl phosphate	107-66-4	1 ppm TWA	1 ppm TWA 2 ppm STEL	1 ppm TWA 2 ppm STEL
1122 1,3-Dichloro-5,5-dimethylhydantoin	118-52-5	0.2 mg/m^3 TWA	0.2 mg/m^3 TWA 0.4 mg/m^3 STEL	0.2 mg/m^3 TWA 0.4 mg/m^3 STEL

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1127 Dichloroethyl ether	111-44-4	15 ppm Ceiling, Skin	5 ppm TWA 10 ppm STEL, Skin	5 ppm TWA 10 ppm STEL, Skin
1130 2,2-Dichloropropionic acid	75-99-0	--	1 ppm TWA	1 ppm TWA
1137 Diethylamine	109-89-7	25 ppm TWA	10 ppm TWA 25 ppm STEL	10 ppm TWA 25 ppm STEL
1140 Diisobutyl ketone	108-83-8	50 ppm TWA	25 ppm TWA	25 ppm TWA
1158 Epichlorohydrin	106-89-8	5 ppm TWA, Skin	2 ppm TWA, Skin	2 ppm TWA, Skin
1162 Ethyl benzene	100-41-4	100 ppm TWA	100 ppm TWA 125 ppm STEL	100 ppm TWA 125 ppm STEL
1164 Ethyl ether	60-29-7	400 ppm TWA	400 ppm TWA 500 ppm STEL	400 ppm TWA 500 ppm STEL
1165 Ethyl mercaptan	75-08-1	10 ppm Ceiling	0.5 ppm TWA	0.5 ppm TWA
1169 Ethylene glycol	107-21-1	--	50 ppm Ceiling	50 ppm Ceiling

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1171 Ethylidene norbornene	16219-75-3	--	5 ppm Ceiling	5 ppm Ceiling
1183 Furfural	98-01-1	5 ppm TWA, Skin	2 ppm TWA, Skin	2 ppm TWA, Skin
1184 Furfuryl alcohol	98-00-0	50 ppm TWA	10 ppm TWA 15 ppm STEL, Skin	10 ppm TWA 15 ppm STEL, Skin
1187 Glutaraldehyde	111-30-8	--	0.2 ppm Ceiling	0.2 ppm Ceiling
1196 Hexachlorocyclo-pentadiene	77-47-4	--	0.01 ppm TWA	0.01 ppm TWA
1204 Hexylene glycol	107-41-5	--	25 ppm Ceiling	25 ppm Ceiling
1206 Hydrogen bromide	10035-10-6	3 ppm TWA	3 ppm Ceiling	3 ppm Ceiling
1208 Hydrogen fluoride	7664-39-3	3 ppm TWA	3 ppm TWA 6 ppm STEL	3 ppm TWA 6 ppm STEL
1211 2-Hydroxypropyl acrylate	999-61-1	--	0.5 ppm TWA, Skin	0.5 ppm TWA, Skin
1217 Iron salts (soluble)	Varies with compound	--	1 mg/m^3 TWA	1 mg/m^3 TWA

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1224 Isopropyl acetate	108-21-4	250 ppm TWA	250 ppm TWA 310 ppm STEL	250 ppm TWA 310 ppm STEL
1225 Isopropyl alcohol	67-63-0	400 ppm TWA	400 ppm TWA 500 ppm STEL	400 ppm TWA 500 ppm STEL
1228 n-Isopropylamine	75-31-0	5 ppm TWA	5 ppm TWA 10 ppm STEL	5 ppm TWA 10 ppm STEL
1243 Mesityl oxide	141-79-7	25 ppm TWA	15 ppm TWA 25 ppm STEL	15 ppm TWA 25 ppm STEL
1248 Methyl 2-cyano-acrylate	137-05-3	--	2 ppm TWA 4 ppm STEL	2 ppm TWA 4 ppm STEL
1261 Methyl isobutyl carbinol	108-11-2	25 ppm TWA, Skin	25 ppm TWA 40 ppm STEL, Skin	25 ppm TWA 40 ppm STEL, Skin
1263 Methyl mercaptan	74-93-1	10 ppm Ceiling	0.5 ppm TWA	0.5 ppm TWA
1264 Methyl n-amyl ketone	110-43-0	100 ppm TWA	100 ppm TWA	100 ppm TWA
1267 alpha-Methyl styrene	98-83-9	100 ppm Ceiling	50 ppm TWA 100 ppm STEL	50 ppm TWA 100 ppm STEL

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1270 o-Methylcyclo-hexanone	583-60-8	100 ppm TWA, Skin	50 ppm TWA 75 ppm STEL, Skin	50 ppm TWA 75 ppm STEL, Skin
1298 Osmium tetroxide	20816-12-0	0.002 mg/m^3 TWA	0.002 mg/m^3 TWA 0.006 mg/m^3 STEL	0.002 mg/m^3 TWA 0.006 mg/m^3 STEL
1302 Paraffin wax fume	8002-74-2	--	2 mg/m^3 TWA	2 mg/m^3 TWA
1322 Phosphoric acid	7664-38-2	1 mg/m^3 TWA	1 mg/m^3 TWA 3 mg/m^3 STEL	1 mg/m^3 TWA 3 mg/m^3 STEL
1325 Phosphorus trichloride	7719-12-2	0.5 ppm TWA	0.2 ppm TWA 0.5 ppm STEL	0.2 ppm TWA 0.5 ppm STEL
1334 Potassium hydroxide	1310-58-3	--	2 mg/m^3 Ceiling	2 mg/m^3 Ceiling
1343 Propylene glycol monomethyl ether	107-98-2	--	100 ppm TWA 150 ppm STEL	100 ppm TWA 150 ppm STEL
1350 Rosin core solder pyrolysis products, as formaldehyde	--	--	0.1 mg/m^3 TWA	0.1 mg/m^3 TWA
1365 Sodium bisulfite	7631-90-5	--	5 mg/m^3 TWA	5 mg/m^3 TWA

TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1367 Sodium hydroxide	1310-73-2	2 mg/m^3 TWA	2 mg/m^3 Ceiling	2 mg/m^3 Ceiling
1368 Sodium metabisulfite	7681-57-4	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1376 Sulfur monochloride	10025-67-9	1 ppm TWA	1 ppm Ceiling	1 ppm Ceiling
1377 Sulfur pentafluoride	5714-22-7	0.025 ppm TWA	0.01 ppm Ceiling	0.01 ppm Ceiling
1387 Tetrahydrofuran	109-99-9	200 ppm TWA	200 ppm TWA 250 ppm STEL	200 ppm TWA 250 ppm STEL
1389 Tetrasodium pyrophosphate	7722-88-5	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1392 Thioglycolic acid	68-11-1	--	1 ppm TWA, Skin	1 ppm TWA, Skin
1405 1,2,4-Trichloro-benzene	120-82-1	--	5 ppm Ceiling	5 ppm Ceiling
1408 Triethylamine	121-44-8	25 ppm TWA	10 ppm TWA 15 ppm STEL	10 ppm TWA 15 ppm STEL
1421 Vanadium (V_2O_5, respirable dust)	1314-62-1	0.5 mg/m^3 Ceiling	0.05 mg/m^3 TWA	0.05 mg/m^3 TWA

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TABLE C3-1. Substances for Which Limits Are Based on Avoidance of Irritant Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1422 Vanadium (V_2O_5, fume)	1314-62-1	0.1 mg/m^3 Ceiling	0.05 mg/m^3 TWA	0.05 mg/m^3 TWA
1424 Vinyl acetate	108-05-4	--	10 ppm TWA 20 ppm STEL	10 ppm TWA 20 ppm STEL
1429 VM & P Naphtha	8032-32-4	--	300 ppm TWA 400 ppm STEL	300 ppm TWA 400 ppm STEL
1431 Xylenes (o-, m-, and p-isomers)	1330-20-7	100 ppm TWA	100 ppm TWA 150 ppm STEL	100 ppm TWA 150 ppm STEL
1435 Zinc chloride fume	7646-85-7	1 mg/m^3 TWA	1 mg/m^3 TWA 2 mg/m^3 STEL	1 mg/m^3 TWA 2 mg/m^3 STEL

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

Description of the Health Effects

Irritant effects are readily perceived by affected individuals. The symptoms of sensory irritation include stinging, itching, and burning of the eyes, tearing (or lacrimation), a burning sensation in the nasal passages, rhinitis (nasal inflammation), cough, sputum production, chest pain, wheezing, and dyspnea (breathing difficulty). In the majority of cases, the onset of symptoms occurs rapidly upon exposure to the irritant; it is therefore easy to associate the causative agent with the irritant effect.

These effects may cause severe discomfort and be seriously disabling, as is the case with dyspnea or wheezing. The tearing and eye irritation associated with exposure to sensory irritants are often severe and can be as disabling as the weeping caused by exposure to tear gas. In addition to these primary effects, workers distracted by material irritant effects are more likely than nonexposed workers to have accidents and thus to endanger both themselves and others. (These adverse health effects also clearly have substantial productivity impacts.)

The eye irritation caused by exposure to irritants is believed to result from stimulation of the sensory nerve endings in the cornea. There is little information available on the relationship between the severity of the effect and the physical or chemical properties of the irritating substance. In addition, the mechanism of action underlying this irritant effect is not well understood. Mechanisms that have been suggested include physical action of the irritant on nerve endings, binding of the irritant to sulfhydryl groups of protein, inhibition of cellular respiration, and cholinesterase inhibition (Grant 1986/ Ex. 1-975). The symptoms of eye irritation are usually transient and do not generally persist after cessation of exposure; however, exposure to concentrations of lacrimators that exceed the levels associated with transient eye irritation may produce corneal or conjunctival injury that requires medical treatment (Grant 1986/ Ex 1-975).

Sensory irritation of the pulmonary system primarily affects the upper respiratory tract and causes an increase in sputum production; inflammation of the nasal passages, trachea, and upper bronchial tree; and decreased cilial clearance. These effects produce a burning sensation in the nasal passages and throat; coughing; sneezing; and acute bronchitis. The development of bronchitis indicates that the cilial clearance mechanism has been compromised, and the resulting mucous retention increases the risk of secondary bacterial infection. Wheezing may also be apparent, particularly if the affected individual has a history of hyperreactive airway disease. If exposure is sufficiently intense, the irritant may reach the lower portion of the bronchial tree, causing a chemical burn of the parenchyma and the sudden collection of fluid in interstitial spaces and alveoli (pulmonary edema). Irritation-induced edema may have a delayed onset (12 hours or more) and can cause hypoxia and difficulty in breathing. All of the effects described above are considered to constitute material impairment of health or functional capacity within the meaning of the Act.

For the great majority of substances in this group, current limits are derived from human evidence that exposure to the chemical agent at a particular airborne concentration will be associated with sensory irritation. For a few substances in this group, animal evidence provided the basis for limit setting. Several general types of evidence may be used to revise existing limits:

- Consideration of new human evidence;
- Reinterpretation of human data that formed the basis for setting the 1968 TLV;
- Consideration of evidence from industrial experience showing that employees are not experiencing irritation; and
- Evaluation of new animal evidence.

The studies that provide the basis for the sensory irritant levels being proposed by OSHA are generally controlled-exposure experiments using human volunteers or reports of employee complaints arising in industrial settings.

Dose-Response Relationships and Sensory Irritation

The onset of sensory irritation is considered a "threshold" or NOE level; that is, for any sensory irritant, there is an exposure level below which very few, if any, individuals will experience sensory irritation. As exposure increases above this level, a larger proportion of exposed individuals will notice the effect and the effect will become increasingly severe. At some level above this NOEL, all exposed persons will experience sensory irritation, although the intensity of the response may vary.

The risk of experiencing irritation that is associated with exposures below the NOEL will be minimal (except in the hypersensitive individual), while the risk of experiencing the irritant effect will increase directly as exposure increases. At some point above the NOE level (i.e., at some dose of the substance) the response will be 100 percent, and all exposed persons will experience irritation. According to general toxicologic principles, the shape of the curve that describes responses above the NOEL is sigmoidal, and the steepness of the curve is a function of the variability in individual responses to the particular irritant. For example, if nearly all persons exposed to the substance will experience a response at approximately the same concentration (dose), the curve will be steep; if, on the other hand, the percentage of people responding increases only slowly as concentration rises, the curve will be considerably flatter.

In addition to the relationship between increasing dose and increasing proportion of exposed persons being affected, the intensity of the response also increases with increasing exposure level. Slightly above the NOE level, affected individuals will experience itching and burning of the eyes, nose, and throat; this is a transient effect and disappears upon removal from exposure. For some substances, workers may become inured to the sensations and higher exposure levels are necessary to elicit a subjective response. As exposure levels increase, the irritant effects become more severe to the point where objective signs of mucous membrane irritation are apparent (i.e., redness of the eyes, rhinitis, coughing, and lacrimation).

During the rulemaking, the question arose as to the level of irritation that constitutes a significant risk of material health impairment; OSHA posed this question in the NPRM and a discussion of the responses received appears earlier in this preamble (see Section V, Question 21). Some commenters (Exs. 3-744 and 3-896) were of the opinion that transient irritant effects should not be considered material impairment of health. For example, the U.S. Borax and Chemical Corporation (Ex. 3-744) stated that transient "rhinitis, cough, sputum production, chest pain, wheezing, and dyspnea" do not constitute material impairments of health.

Most commenters, however, recommended that these signs and symptoms be regarded as material health impairments (see, for example, Exs. 8-47, 3-1095, 3-660, and 3-593). For example, NIOSH stated:

> The recognition of sensory irritation as potentially being "material impairment of health" is consistent with the current

scientific consensus related to health effects of environmental agents.

Mucous membrane irritants can cause increased blink frequency and tearing; nasal discharge, congestion, and sneezing; and cough, sputum production, chest discomfort, sneezing, chest tightness, and dyspnea. Work environments often require levels of physical and mental performance considerably greater than encountered in daily living. Even in the absence of any permanent impairment, the symptoms listed can interfere with job performance and safety.

Mucous membrane irritation can result in inflammation, which may lead to increased susceptibility to nonspecific irritants and infectious agents. For example, experimental ozone exposure in humans results in increased airway reactivity. Also, studies of exposure to environmental tobacco smoke have shown irritative symptoms and evidence of increased frequency of respiratory tract illnesses in young children and decreased pulmonary function in adults * * *.

Mucous membrane irritation is associated with respiratory illnesses, depending on the composition of specific exposure and on the dose, duration, and frequency of exposure. No universally applicable conclusion can be drawn at this time regarding the association between irritative symptoms and permanent injury or dysfunction. Where certain individuals show no measurable impairment after an exposure, even when experiencing irritative symptoms, others may develop identifiable dysfunction.

Aside from the effects of irritation, mucous membrane exposure may result in absorption of a substance, with resultant systemic toxicity. An inflamed mucous membrane may be an even more effective route of absorption, either for the irritant or for other substances. Furthermore, injury to bronchopulmonary membrances can impair removal of particulates from the respiratory system (Ex. 8-47, pp. 38-40).

Thus, according to NIOSH, sensory irritants interfere with job performance and safety, cause inflammation, may increase the victim's susceptibility to other irritants and infectious agents, lead to permanent injury or dysfunction, or permit greater absorption of hazardous substances (Ex. 8-47).

Another commenter, E.L. DeWitt, an occupational health consultant for the du Pont Company, remarked:

Irritation takes many forms * * * with the effect being perhaps no more than transient, slight to mild discomfort. Again, this type of irritation needs to be prevented but the 'safety factor' [applied] might be somewhat less in this case. There are also situations where 'irritation' is perceived but is without any accompanying manifestations. In these cases, there may be no real need to modify the exposure limit. The exposure conditions required to produce these findings need to be considered also (Ex. 3-660, p. 4).

OSHA concludes that exposure limits are needed for those substances for which PELs are being established in this rulemaking to protect against sensory irritant effects that result in objective signs of irritation, such as coughing, wheezing, conjunctivitis, and tearing. Such levels of mucous membrane irritation may require medical treatment, adversely affect the well-being of employees, and place the affected individual at risk from increased absorption of the substance and decreased resistance to infection. Exposing workers repeatedly to irritants at levels that cause subjective irritant effects may cause workers to become inured to the irritant warning properties of these substances and thus increase the risk of overexposure. In addition, the long-term effects of repeated low-level sensory irritation have not been well studied.

Therefore, OSHA finds that the sensory irritation caused by exposure to those substances for which PELs are being established in this rulemaking constitutes a material impairment of health and functional well-being and has established exposure limits for these substances at levels that will protect workers from the significant risk of experiencing this material impairment of health.

Analyses of the toxicologic data for the substances in this group of chemicals and OSHA's findings in each case are presented below.

ACETALDEHYDE

CAS: 75-07-0; Chemical Formula: CH_3CHO
H.S. No. 1001

OSHA's previous PEL for acetaldehyde was 200 ppm as an 8-hour TWA. In its NPRM, OSHA proposed revising its limit for acetaldehyde to 100 ppm as an 8-hour TWA and supplementing this with a STEL of 150 ppm; these are the limits currently recommended by the ACGIH. OSHA is establishing permissible exposure limits of 100 ppm as an 8-hour TWA and 150 ppm as a 15-minute STEL in the final rule. Acetaldehye is a colorless liquid with a pungent, fruity odor.

The 200-ppm 1968 TLV established by the ACGIH for acetaldehyde was based on a sensory irritation study conducted by Silverman, Schulte, and First (1946/Ex. 1-142) that showed that unacclimatized individuals experienced eye irritation at 50 ppm, but that a level of 200 ppm was tolerable for an 8-hour day. Reexamination of the data reported by Silverman, Schulte, and First (1946/Ex. 1-142) reveals that, at 200 ppm of acetaldehyde, all exposed persons experienced inflammation of the conjuctivae of the eyes, which manifested as redness. OSHA therefore concluded that its previous PEL of 200 ppm placed exposed employees at risk of conjunctivitis and other irritation and that a reduction to 100 ppm was necessary to reduce this risk. OSHA also proposed a STEL of 150 ppm to supplement the 8-hour limit because, without a STEL, workers could be exposed to levels many times those that have been shown to cause corneal injury, sensitization, and respiratory tract irritation. NIOSH (Ex. 8-47, Table N6B; Tr. pp. 3-97 to 3-98) indicated that acetaldehyde might be a candidate for an individual 6(b) rulemaking. As pointed out by the Workers Institute for Safety and Health (WISH) (Tr. 7-117, Ex. 116, p. 8), IARC has classified acetaldehyde as a possible human carcinogen based on animal data. There is also evidence that acetaldehyde is teratogenic and fetotoxic in animals (Ex. 116). The Agency will continue to monitor the scientific evidence for this substance to examine whether a further reduction in the PEL is warranted.

OSHA concludes that employees are placed at significant risk of conjunctivitis and irritation at the current 8-hour TWA limit of 200 ppm. The Agency has determined that conjunctivitis and sensory irritation represent material impairments of health or functional capacity. Therefore, OSHA is revising the limit for acetaldehyde to 100 ppm as an 8-hour TWA and 150 ppm as a 15-minute STEL to substantially reduce this risk.

ACETIC ACID

CAS: 64-19-7; Chemical Formula: CH_3COOH
H.S. No. 1002

The former OSHA PEL for acetic acid was a 10-ppm 8-hour TWA. OSHA proposed to retain the TWA limit and to supplement it with a 15-ppm STEL, Based on the acute irritant properties of acetic acid. These limits are consistent with the ACGIH recommended TLVs (1986/Ex. 1-3). NIOSH (Ex. 8-47, Table N1) concurred with these proposed limits. However, OSHA's review of the evidence for acetic acid has demonstrated that there is no basis at this time for a STEL, and the final rule thus retains the 8-hour TWA PEL. Acetic acid is a clear, colorless, flammable liquid with a pungent odor.

Sterner (1949/Ex. 1-1207) reported that exposures to concentrations of acetic acid ranging from 800 to 1200 ppm cannot be tolerated by humans for longer than three minutes. The AIHA (Ex. 8-16) stated that unacclimatized workers experience eye and nasal irritation at acetic acid levels in excess of 26 ppm, and that exposure to 50 ppm is intolerably irritating. The ACGIH also reported that acclimatized workers are sometimes able to tolerate exposure to concentrations as high as 30 ppm.

Guinea pigs exhibited minor changes in respiration after exposure to 5 ppm; exposure to 100 ppm produced a significant increase in pulmonary flow resistance and a decrease in breathing rate and minute volume, which suggests that bronchial constriction is the primary irritant action of acetic acid (Amdur 1961/Ex. 1–601).

The 10-ppm TWA was established on the basis of studies indicating that industrial exposure to acetic acid at 10 ppm was nonirritating (Sterner 1943/Ex. 1–806). However, conjunctival irritation has been reported in humans exposed below 10 ppm (duration not specified) (Baldi 1953/Ex. 1–602), and workers exposed to concentrations of 60 ppm during the workshift, plus one hour daily at 100 to 260 ppm, for 7 to 12 years developed respiratory irritation, conjunctivitis, bronchitis (which was asthma-like in some workers), pharyngitis, erosion of exposed teeth, and gastritis (Parmeggiani and Sassi (1954/Ex. 1–753). Vigliani and Zurlo (1955/Ex. 1–164) observed respiratory, gastrointestinal, and skin irritation in the same group of workers.

In a prehearing comment, Eastman Kodak (Ex. 3–661) argued that there was no toxicologic basis for a 15-ppm STEL, citing Vigliani and Zurlo (1955/Ex. 1–164), who reported that exposure to 20 to 30 ppm is without danger. In addition, Kodak stated that irritation has only been observed "with prolonged and repeated exposures" above the 10-ppm TWA PEL. Eastman Kodak concluded that "[no] significant irritation or other ill effects have been reported by employees that periodically are exposed to levels of acetic acid in excess of the proposed 15-ppm STEL" (Ex. 3–661, p. 4).

OSHA has carefully reviewed the toxicologic evidence in the record and has determined that the evidence supporting a STEL for acetic acid is equivocal. Because information on exposure durations is lacking in the studies cited above (Baldi 1953/Ex. 1–602; Parmeggiani and Sassi 1954/Ex. 1–753), it is not known whether the conjunctival irritation found among exposed workers was due to short-term or prolonged exposure to acetic acid. Eastman Kodak (Ex. 3–661) has maintained that prolonged exposure to acetic acid at levels above the 10 ppm TWA PEL is necessary to cause irritant responses among exposed workers. Therefore, in the final rule, OSHA is retaining its 10 ppm TWA PEL for acetic acid, but is not supplementing this limit with a STEL.

ACETONE

CAS: 67–64–1: Chemical Formula: CH_3COCH_3
H.S. No. 1004

OSHA's previous Z-table limit for acetone was 1000 ppm as an 8-hour TWA. In the NPRM, the Agency proposed to lower this limit to 250 ppm as an 8-hour TWA. This proposed limit was derived from the NIOSH-recommended limit, which was based on a number of industrial and human volunteer studies reporting irritant and central nervous system effects resulting from exposure to acetone concentrations at levels below 1000 ppm; NIOSH (Ex. 8–47, Table N1) and the AFL–CIO (Ex. 194) concurred with the proposed limit. The ACGIH TLVs for acetone are 750 ppm as an 8-hour TWA and 1000 ppm as a 15-minute STEL. OSHA has carefully reviewed the scientific evidence and comments in the record and has determined that it is appropriate to revise the acetone PEL in the final rule to 750 ppm as an 8-hour TWA and to add a short-term limit of 1000 ppm. Acetone is a colorless, highly volatile, flammable liquid with an aromatic odor.

OSHA's proposed 250-ppm TWA limit for acetone was largely based on controlled human studies conducted by Nelson, Enge, Ross et al. (1943/Ex. 1–66) and Matsushita, Yoshimune, Inoue et al. (1969/Ex. 1–191), as well as studies in workers conducted by Vigiliani and Zurlo (1955/Ex. 1–164) and Parmeggiani and Sassi (1954/Ex. 1–753). OSHA's reliance on these studies to establish a revised limit for acetone was criticized by Dr. William C. Thomas, Manager of Toxicology for the Hoechst Celanese Corporation, who testified on behalf of the Ketones Program Panel of the Chemical Manufacturers Association (CMA) (Ex. 8–54; Tr. 8/4/88, pp. 6–114 to 6–127; Exs. 149A, 149C). The National Marine Manufacturers Association (Ex. 181) agreed with Dr. Thomas' remarks. Summaries of each of these studies and of OSHA's response to Dr. Thomas' remarks follow.

In a controlled-exposure experiment, Nelson, Enge, Ross et al. (1943/Ex. 1–66) exposed an average of 10 human subjects (both male and female) to a variety of solvents, including acetone, for three to five minutes. Subjects were asked to judge the level of sensory irritation as absent, slightly irritating, or very irritating. Tests were conducted in a 1200-cubic-foot gas cabinet equipped with an anemostat to distribute the air uniformly. Acetone was reported to produce slight irritation on exposure to 300 ppm, but a concentration of 500 ppm produced a degree of eye, nose, and throat irrigation that was still described by a majority of the subjects as "tolerable."

Dr. Thomas expressed five criticisms of the Nelson, Enge, Ross et al. (1943/Ex. 1–66) study. These were: (1) The short duration of exposure used; (2) the study's failure to account for adaptation because "naive" subjects who had not had previous acetone exposure were used; (3) the authors' reliance on subjective responses rather than on objective medical examination; (4) the use of nominal (calculated) exposures rather than measured exposures; and (5) the introduction of potential bias because students who were involved in the experiment were used as test subjects (Tr. 8/4/88, pp. 6–114 to 6–117; Exs. 149A, 149C).

NIOSH addressed some of these issues in its criteria document for ketones (NIOSH 1978f, as cited in ACGIH 1986/Ex. 1–3, p. 6). In its analysis of the Nelson, Enge, Ross et al. (1943/Ex. 1–66) study, NIOSH (1978f) concluded:

> The concentrations of ketones in the exposure chamber were calculated (nominal) rather than measured analytically, so the true concentration may have been lower than reported * * *.
>
> [T]he use of experimenters as subjects was a possible source of bias, and the exposure periods of 3–5 minutes were not long enough to show if adaptation would occur * * *. The fact that exposure duration did not approach that of a normal workshift is a major limitation of * * * [this study]. However, the data are useful as a guide to the relative irritating properties of ketones and the concentrations at which these [properties] appear (NIOSH 1978f, p. 31).

Thus, despite these experimental limitations, NIOSH concluded that the Nelson, Enge, Ross et al. (1943/Ex. 1–66) study was useful in identifying ketone concentrations that are irritating, and it relied on this study, at least in part, when recommending a 250–ppm TWA limit for acetone (NIOSH 1978f, as cited in ACGIH 1986/Ex. 1–3, p. 6).

The second paper discussed by Dr. Thomas is the report by Matsushita, Yoshimune, Inoue et al. (1969/Ex. 1–191). In this study, the authors exposed 25 healthy male subjects to 0, 100, 250, 500, or 1000 ppm acetone. Subjects were exposed for three hours in the morning and three hours in the afternoon, with a 45-minute period between exposures. Irritant responses were scored on a scale from 0 to 12, with a score of 12 representing severe irritation.

Most of the subjects exposed to 500 or 1000 ppm acetone reported irritation (scored between 4 and 5 in severity) during the first 90 minutes of exposure in the morning and the first 60 minutes of exposure in the afternoon. Subjects ceased to report irritation at the 90-minute mark during the afternoon exposure. A lesser degree of irritation was reported to occur among subjects

exposed to 100 or 250 ppm acetone; however, this irritation subsided after the first 90 minutes of exposure in each of the two exposure periods. Subjects exposed to 250 ppm or higher reported feeling general weakness and a sense of tension even as long as 24 hours after exposure. Blood and urine samples taken during and after exposure showed increasing blood and urinary acetone levels among subjects exposed to 250 ppm or higher. Following the exposure period, these levels fell to normal values within about 25 to 35 hours after exposure was terminated. The authors also reported an increased leukocyte count in subjects exposed to 500 or 1000 ppm acetone; the increased white cell count persisted for about 24 hours after the cessation of exposure. The authors attributed this increased leukocyte count to acetone's irritant properties (Matsushita, Yoshimune, Inoue et al. 1969/Ex. 1–191).

Dr. Thomas criticized this study because it did not describe the methods used by its authors for measuring acetone exposures, and the blood acetone levels reported by Matsushita and colleagues (1969/Ex. 1–191) were about 2.5 times higher than those reported after similar exposures conducted by DiVincenzo, Yanno, and Astill (1973, as cited in ACGIH 1986/Ex. 1–3, p. 6). After a two-hour exposure to 500 ppm acetone, Matsushita, Yoshimune, Inoue et al. (1969/Ex. 1–191) found a blood acetone level of 25 mg/L, compared to a level of 10 mg/L reported by DiVincenzo, Yanno, and Astill (1973, as cited in ACGIH 1986/Ex. 1–3, p. 6). Dr. Thomas suggested that the actual exposure levels employed by Matsushita and associates (1969/Ex. 1–191) may actually have been substantially higher than reported by these authors (Tr. 8/4/88, pp. 6–118 to 6–119; Exs. 149A, C).

OSHA has reviewed the report by DiVincenzo, Yanno, and Astill (1973, as cited in ACGIH 1986/Ex. 1–3, p. 6) and finds that the blood acetone results reported in this paper cannot be directly compared, as Dr. Thomas has done, with those reported by Matsushita, Yoshimune, Inoue et al. (1969/Ex. 1–191), for a number of reasons. First, the subjects studied by DiVincenzo, Yanno, and Astill fasted for eight hours prior to exposure; it is not clear that the subjects studied by Matsushita, Yoshimune, Inoue et al. fasted before they were exposed. Second, the blood acetone values reported by DiVincenzo, Yanno, and Astill were corrected for endogenous acetone (i.e., acetone levels that existed prior to exposure). The authors reported that endogenous acetone levels ranged from 0 to 10 mg/L of blood, or about as high as would occur after a two-hour exposure to 500 ppm of acetone. Whether Matsushita, Yoshimune, Inoue et al. corrected for endogenous blood acetone levels is uncertain; if they did not, their reported blood acetone levels may be as much as two times overstated. The third consideration is that the studies used different methods to measure blood acetone levels. Matsushita, Yoshimune, Inoue, et al. used a colorimetric method, while DiVincenzo, Yanno, and Astill used a gas chromatographic approach. The use of different analytical methods by the two investigative groups complicates any comparison of their blood acetone results. Thus, OSHA does not agree that the results by DiVincenzo, Yanno, and Astill (1973, as cited by ACGIH 1986/Ex. 1–3, p. 6) demonstrate that the exposure levels used by Matsushita, Yoshimune, Inoue et al. (1969/Ex. 1–191) are necessarily understated.

In addition to the two controlled-exposure studies discussed above, two industry studies were relied on by OSHA to support the reduction in the acetone PEL. One report by Parmeggiani and Sassi (1954/Ex. 1–759) indicated that six employees exposed to 307 to 918 ppm acetone in a rayon acetate plant experienced eye and throat irritation, dizziness, and inebriation. Five of the employees showed objective signs of pharyngeal irritation, four had lung irritation, and three had conjunctivitis. Although the authors attributed the observed CNS effects to excessive concomitant exposure to carbon disulfide, the irritant effects are more likely to have been the result of exposure to acetone, because carbon disulfide is not a primary irritant by vapor inhalation (*Chemical Hazards of the Workplace,* 2nd ed., Proctor, Hughes, and Fischman 1988, pp. 120–121). The other report, by Vigliani and Zurlo (1955/Ex. 1–164), found that acetone production workers exposed to 700 ppm acetone for three hours daily for 7 to 15 years experienced inflammation of the respiratory tract, stomach, and duodenum; giddiness; and loss of strength.

Dr. Thomas (Exs. 8–54, 149A, 149C; Tr. 8/4/88, pp. 6–114 to 6–127) criticized these two studies on the basis that the urinary acetone levels reported by Parmeggiani and Sassi (1954/Ex. 1–759) and by Vigliani and Zurlo (1955/Ex. 1–164) indicated that airborne exposures were much higher than the reported values. He stated that, based on these values, the employees observed in both of these studies were likely to have been exposed to acetone levels approximating 5000 ppm. OSHA is not convinced that the exposure levels reported in these two studies are understated. The studies by Matsushita, Yoshimune, Inoue et al. (1969/Ex. 1–191) and DiVincenzo, Yanno, and Astill (1973, as cited in ACGIH 1986/Ex. 1–3, p. 6) clearly demonstrate that blood and urinary acetone levels can increase with continued, daily exposure. Furthermore, in its criteria document, NIOSH (1978f, as cited in ACGIH 1986/Ex. 1–3, p. 6) cites a number of studies that demonstrate that skin absorption of acetone can result in elevated blood and urinary acetone levels. OSHA believes that the high urinary acetone levels reported in the workers studied by Parmeggiani and Sassi (1954/Ex. 1–759) and by Virgiliani and Zurlo (1955/Ex. 1–164) were most likely the result of an accumulated body burden of acetone brought about by long-term exposure and dermal absorption. Given these considerations, it does not appear appropriate to approximate airborne exposure levels on the basis of the urinary acetone levels reported in these two studies.

To summarize, OSHA finds that the studies discussed above show that acetone is capable of producing sensory irritation at concentrations below 1000 ppm and that long-term exposure to acetone at levels below 1000 ppm can cause CNS disturbances. In addition, the ACGIH (1986/Ex. 1–3, p.6) reports that chronic exposure to acetone causes respiratory irritation and headaches. Despite the methodological shortcomings of all of these studies, OSHA is impressed with the consistency of their findings. Both the Nelson, Enge, Ross et al. (1943/Ex. 1–66) and the Matsushita, Yoshimune, Inoue et al. (1969/Ex. 1–91) studies demonstrate that exposure to concentrations of acetone below 1000 ppm are associated with eye, nose, and throat irritation. Both industry studies (Parmeggiani and Sassi 1954/Ex. 1–759; Vigliani and Zurlo 1955/Ex. 1–164) report similar signs and symptoms of irritation and CNS disturbances in workers exposed to concentrations of acetone between 700 and 1000 ppm. OSHA is not persuaded by Dr. Thomas' arguments that exposure levels are understated in these reports; OSHA believes that the quantitative relationship between long-term exposure to acetone and urinary acetone levels is not sufficiently established to draw this conclusion. Therefore, OSHA concludes that the findings of these four studies are consistent in demonstrating the acute and long-term effects of acetone exposure at levels below 1000 ppm.

The Ketones Panel of the CMA (Tr. 8/4/88, pp. 6–100 to 6–113; Exs. 149A, 149B, and 179) also presented testimony by Dr. Robert Raleigh, Adjunct Professor of Medicine at the University of Rochester School of Medicine. Dr. Raleigh testified on a study he conducted among filter press operators who were exposed exclusively to acetone (Raleigh and McGee 1972, as cited in Ex. 8–54). In this study, 13 workers were asked about symptoms and were medically examined over a one-week period. Using grab bags, acetone samples were taken at random periods during each workshift. Subjective symptoms were recorded with each grab sample. Samples were analyzed by gas chromatography.

Over the period studied, TWA exposures to acetone varied from 950 to 1060 ppm. Of the 13 workers studied, nine (69 percent) reported eye irritation, five (38 percent) reported nasal irritation, and five (38 percent) reported throat irritation. Three (23 percent) employees reported experiencing lightheadedness. Some employees reported these symptoms more than once during the study period. There were four cases of eye irritation following short-term exposures to acetone concentrations below 1000 ppm. Eye irritation that was reported to be "strong" occurred following short-term exposures to 1200 ppm. Physical examination revealed a few instances of redness of the nasal mucosa and slight infection of the mucosa of the nose and throat.

In his written testimony regarding this study, Dr. Raleigh concluded:

> Considering the number of samples taken, the variability of human response, the slight to mild nature of the response, and the lack of *objective* evidence of eye irritation as noted by the examining physician, I do not believe * * * [instances of irritation occurring below 1000 ppm] indicate the need for a safe level being set below 1000 parts per million (Ex. 8–54, p. 9).

Dr. Raleigh also testified that the occurrence of transient dizziness was no cause for concern:

> [T]his symptom is usually very transient and in my experience I have never noted any adverse consequences from an occasional person * * * who complains of dizziness (Tr. 8/4/88, p. 6–103).

OSHA does not agree with Dr. Raleigh's interpretation of his study or with his view that dizziness, irritation and mild infections of the mucous membranes of the respiratory tract do not constitute material impairments of health. After reviewing the Raleigh and McGee report (1972, as cited in Ex. 8–54), OSHA notes that more than half the workers studied experienced sensory reactions from exposure to acetone at TWA levels equal to the former 1000-ppm OSHA limit. Furthermore, some of these reactions were characterized as "strong." OSHA believes that this study further demonstrates that the Agency's former 1000-ppm 8-hour TWA limit is insufficiently protective and does not prevent workers from experiencing these sensory effects. In addition, in contrast to Dr. Raleigh, OSHA characterizes transient dizziness in and of itself as an "adverse consequence." Dizziness connotes an effect on the central nervous system; in addition, dizziness is a serious safety hazard in the workplace. For the reasons stated earlier in this section, OSHA finds that such effects constitute material impairments of health. Thus, OSHA finds that the Raleigh and McGee study (1972, as cited in Ex. 8–54) is a recent, well-conducted study that provides additional support for the need to lower the former 1000-ppm TWA limit for acetone. Furthermore, OSHA finds the evidence that adverse effects can result from short-term exposures to levels of acetone at or near 750 ppm convincing; two controlled human studies (Nelson, Enge, Ross et al. 1943/Ex. 1–66; Matsushita, Yoshimune, Inoue et al. 1969/Ex. 1–191) reported sensory irritant effects upon short-term exposure to such levels of acetone, and two industry studies (Parmeggiani and Sassi 1954/Ex. 1–759; Vigliani and Zurlo 1955/Ex. 1–164) reported irritation and CNS effects among employees exposed to acetone levels ranging from 307 to 918 ppm in one instance and about 700 ppm in the other. In addition, two studies (Matsushita, Yoshimune, Inoue et al. 1969/Ex. 1–191; DiVincenzo, Yanno, and Astill 1973, as cited in ACGIH 1986/Ex. 1–3, p. 6) suggest that chronic exposure to acetone on a daily basis leads to the bioaccumulation of acetone.

In light of the studies discussed above, OSHA concludes that it is necessary to reduce the limit for acetone to 750 ppm as an 8-hour TWA and 1000 ppm as a STEL to protect workers from the acute and chronic effects of acetone exposure. OSHA finds that the chemically induced sensory irritation associated with acute exposures to acetone can occur at levels only slightly above the 750-ppm level being established as an 8-hour TWA. In the absence of a STEL, the 750-ppm limit would permit excursions to levels as high as 12,000 ppm for brief periods. Such levels "depress the central nervous system, causing dizziness, weakness, and loss of consciousness" (Proctor, Hughes, and Fischman 1988, p. 49). An 8-hour TWA of 750 ppm is necessary to protect workers against the bioaccumulation of acetone, chronic irritation of the respiratory tract, and headaches associated with long-term acetone exposures. OSHA considers both the short-term sensory irritation associated with brief exposures to acetone and the increased blood and urinary accumulation and chronic respiratory irritation characteristic of long-term acetone exposures to be material impairments of health. Accordingly, OSHA is establishing in the final rule an 8-hour TWA PEL of 750 ppm and a STEL of 1000 ppm for acetone.

ACROLEIN

CAS: 107–02–8; Chemical Formula: $CH_2=CHCHO$
H.S. No. 1007

OSHA formerly had an 8-hour TWA PEL of 0.1 ppm (0.25 mg/m^3) for acrolein. OSHA proposed the addition of a 0.3-ppm STEL to this TWA limit, and the final rule adopts this short-term limit. NIOSH (Ex. 8–47, Table N1) concurred with these proposed limits. These limits for acrolein are the same as those recommended by the ACGIH (1986/Ex. 1–3). Acrolein is a colorless or yellowish flammable liquid with a disagreeable, choking odor.

In early inhalation studies of cats (Iwanoff 1911, as cited in ACGIH 1986/Ex. 1–3, p. 11), exposure to 10 ppm acrolein for 3.5 hours was found to have only transient effects, including salivation, lacrimation, respiratory irritation, and mild narcosis. However, later studies reported that an exposure to 1 ppm of acrolein produced marked nose and eye irritation in five minutes or less (Cook 1945/Ex. 1–726). Over longer periods, studies have demonstrated fatalities in one of six rats exposed for four hours to airborne concentrations of acrolein at 8 ppm; at 16 ppm, the mortality was 100 percent (Smyth 1956/Ex. 1–759). Irritation of the upper respiratory tract is the primary symptom of acrolein inhalation, but lung edema can occur after exposure to high concentrations (Henderson and Haggard 1943a/Ex. 1–881). In addition, skin contact with acrolein causes skin burns and severe injury to the cornea.

No comments (other than NIOSH's) were received on OSHA's proposed 8-hour time-weighted-average limit or its 15-minute short-term limit of 0.3 ppm. OSHA concludes that, in the absence of a STEL, the current 0.1-ppm TWA limit would not protect employees from short-term exposures to airborne concentrations in excess of 1 ppm, the level found by Cook (1945/Ex. 1–726) to cause severe eye and nose irritation. OSHA considers these adverse effects

to represent material impairments of health or functional capacity. Therefore, OSHA finds that the 0.3-ppm STEL is necessary to protect employees from the significant risk associated with mucous membrane irritation, and the Agency is revising the exposure limit for acrolein to 0.1 ppm as an 8-hour TWA and 0.3 ppm as a 15-minute STEL.

ALLYL ALCOHOL

CAS: 107-18-6; Chemical Formula: $CH_2=CHCH_2OH$
H.S. No. 1010

OSHA previously had a PEL of 2 ppm TWA for allyl alcohol, with a skin notation. OSHA proposed to supplement this TWA limit with a STEL of 4 ppm and to retain the existing skin notation. NIOSH (Ex. 8-47, Table N1) concurred with this proposal. The final rule establishes a 2-ppm TWA limit, a 4-ppm STEL, and a skin notation for allyl alcohol, which is consistent with the ACGIH (1986/Ex. 1-3) recommendation. Allyl alcohol is a colorless liquid with a pungent, mustard-like odor.

The most important adverse effects of occupational exposures to allyl alcohol are upper-respiratory-tract irritation and burns of the eyes. In a controlled human sensory response study (Dunlap, Kodama, Wellington et al. 1958/Ex. 1-630), a five-minute exposure to 25 ppm resulted in severe eye irritation. Milder irritation has been reported to occur at 5 ppm (McCord 1932, as cited in ACGIH 1986/Ex. 1-3, p. 18). Necrosis of the cornea and temporary blindness occurred in one individual exposed to allyl alchol at a level irritating to the eyes and nose (Smyth 1956/Ex. 1-759). Skin absorption may lead to serious systemic injury (visceral congestion, periportal congestion of the liver, hematuria, and nephritis); in addition, when evaporation from the skin is prevented or reduced, skin contact causes burns (ACGIH 1986/Ex. 1-3, p. 18).

Exposure to airborne concentrations of allyl alcohol causes a series of characteristic effects, including lacrimation, photophobia, blurred vision, and retrobulbar pain (Dunlap, Kodama, Wellington et al. 1958/Ex. 1-630). Exposed individuals do not develop a tolerance for this substance, and they also do not become sensitized (Kodama and Hine 1958/Ex. 1-1088).

The New Jersey Department of Public Health (Ex. 144, 144A) urged OSHA to set its limits for allyl alcohol on the basis of EPA's IRIS data. The use of such an approach is discussed in Section VI.A of the preamble.

In a prehearing comment, Dr. Lawrence Hecker of Abbott Laboratories (Ex. 3-678) stated that a STEL did not appear to be warranted for allyl alcohol, based on his review of the literature. However, Dr. Hecker did not specifically discuss the evidence or rationale underlying this contention. In reviewing the evidence for allyl alcohol, OSHA notes that severe eye irritation has been reported to occur in human subjects exposed to 25 ppm for as short an interval as five minutes (Dunlap, Kodama, Wellington et al. 1958/Ex. 1-630); such an exposure would be permitted under the current limit of 2 ppm as an 8-hour TWA. OSHA also notes that short-term exposure to allyl alcohol produces characteristic effects more severe than those caused by other sensory irritants; these effects include photophobia and blurred vision. OSHA considers the effects of sensory irritation and disturbed vision to constitute material impairments of health or functional capacity. Sax and Lewis (1989) report that the dermal LD_{50} in rabbits is 53 mg/kg, indicating that allyl alcohol readily permeates the skin and causes systemic toxicity.

OSHA concludes that the scientific evidence clearly shows a significant health risk associated with short-term exposure to the levels of allyl alcohol that would be permitted under the former standard; accordingly, the Agency is establishing a 4-ppm 15-minute STEL to supplement its 8-hour TWA limit of 2 ppm. The final rule retains the skin notation for this substance to protect workers from dermal absorption.

ALLYL GLYCIDYL ETHER

CAS: 106-92-3; Chemical Formula: $C_6H_{10}O_2$
H.S. No. 1012

OSHA's former PEL for allyl glycidyl ether (AGE) was 10 ppm (45 mg/m^3) as a ceiling. OSHA proposed to revise this limit to a TWA of 5 ppm, and to add a 15-minute STEL of 10 ppm and a skin notation, consistent with the recommended limits of the ACGIH (1986/Ex. 1-3). NIOSH (Ex. 8-47, Table N1) concurred with this proposal. In the final rule, OSHA is establishing the air contaminant limits as proposed, but is not establishing a skin notation for this substance (see Section VI.C.18 for a discussion of the Agency's policy on skin notations). Allyl glycidyl ether is a colorless liquid of characteristic, but not unpleasant, odor.

In limited human exposure studies, AGE has been demonstrated to cause dermatitis and eye irritation; the substance produces moderate primary skin irritation and severe eye irritation in animals (Hine, Kodama, Wellington et al. 1956/Ex. 1-331). At 260 ppm, animals experienced irritation of the eyes and respiratory distress; at higher levels (e.g., 400, 600, and 900 ppm), corneal opacities and severe respiratory difficulties occurred (Hine, Kodama, Wellington et al. 1956/Ex. 1-331). The percutaneous LD_{50} for rabbits is 2.55 g/kg. Intragastric administration of AGE in mice, rats, and rabbits has also been demonstrated to cause depression of the central nervous system (Hine, Kodama, Wellington et al. 1956/Ex. 1-331).

In humans, skin sensitization occurs readily (Hine and Rowe 1963a, as cited in ACGIH 1986/Ex. 1-3, p. 20). In addition to primary irritation and sensitization, the potential exists for cross-sensitization with other epoxy agents (ACGIH 1986/Ex. 1-3, p. 20).

Sax and Lewis (*Dangerous Properties of Industrial Materials*, 7th ed., 1989) report the dermal LD_{50} in rabbits to be 2.25 g/kg; there is no other evidence of systemic poisoning occurring from skin absorption in humans or other animal species. Therefore, in accordance with the general policy described in Section VI.C.18 of this preamble, OSHA is not establishing a skin notation for AGE. Other than those submitted by NIOSH, OSHA received no comments on its proposed revision of the exposure limit for AGE.

In the final rule, OSHA is establishing PELs of 5 ppm (8-hour TWA) and 10 ppm (15-minute STEL) for allyl glycidyl ether. OSHA concludes that these combined limits will reduce the significant risks of sensitization and primary irritation to which employees could otherwise be exposed. OSHA considers these adverse effects material impairments of health and functional capacity.

ALLYL PROPYL DISULFIDE

CAS: 2179-59-1; Chemical Formula: $CH_2=CHCH_2S_2C_3H_7$
H.S. No. 1013

The previous OSHA PEL for allyl propyl disulfide was 2 ppm (12 mg/m^3) as an 8-hour TWA. OSHA proposed to supplement this limit with a 3-ppm (18-mg/m^3) 15-minute STEL, and NIOSH (Ex. 8-47, Table N1) concurred with this proposal. The final rule establishes a 2-ppm TWA limit and 3-ppm STEL for this substance; these limits are the same as those recommended by the ACGIH (1986/Ex. 1-3). Allyl propyl disulfide is a liquid with a pungent, irritating odor.

Nearly all occupational exposures to allyl propyl disulfide, the primary volatile constituent of onion oil, occur in the processing of onions and onion products. Allyl propyl disulfide's irritative effects on the human eye, nose, and upper respiratory tract are well recognized. The most severe irritation effects have occurred when workers were exposed to allyl propyl disulfide in

the vicinity of onion slicing machines, where average concentrations of 3.4 ppm have been measured (Feiner, Burke, and Baliff 1946/Ex. 1-604).

No rulemaking participants other than NIOSH commented on the addition of a STEL to the current TWA limit for allyl propyl disulfide. OSHA concludes that, in the absence of a STEL, the 2-ppm TWA limit would not prevent employees from being exposed to short-term concentrations of sufficient magnitude to cause acute irritant effects. The Agency considers this effect to constitute material impairment of health and functional capacity. Accordingly, OSHA finds that a limit on short-term exposure is necessary to protect workers from significant acute irritation and is supplementing its current 2-ppm TWA limit with a 3-ppm 15-minute STEL in the final rule.

AMMONIA

CAS: 7664-41-7; Chemical Formula: NH_3
H.S. No. 1021

OSHA's former exposure limit for ammonia was 50 ppm as an 8-hour TWA. OSHA proposed to revise this limit to 25 ppm TWA and to add a 35-ppm 15-minute STEL, based on the limits established by the ACGIH. NIOSH indicated its agreement with these proposed limits (Ex. 8-47, Table N1). However, in the final rule, the Agency has determined that it is not appropriate to establish a 25-ppm TWA limit for ammonia; the final rule does revise OSHA's exposure limit to 35 ppm as a 15-minute STEL. Ammonia is principally used as a feedstock in the manufacture of fertilizers and other chemical substances and is also used as a refrigerant.

Ammonia is a primary eye and upper respiratory tract irritant. An unpublished study conducted by the Detroit Department of Health and cited by the ACGIH (1986/Ex. 1-3, p. 27) reports that ammonia concentrations in the range of 20 to 25 ppm elicited complaints of discomfort from workers engaged in blueprinting and copying operations. In addition, a study of pigs conducted by Stombaugh et al. (1969) appeared to demonstrate that exposure to ammonia causes systemic effects. Thus the ACGIH established both a full-shift TWA of 25 ppm to protect against chronic effects and a 35-ppm STEL to protect against ammonia's irritant effects.

OSHA also considered NIOSH's recommended 5-minute ceiling limit for ammonia of 50 ppm. When making this recommendation, NIOSH relied on several reports that ammonia concentrations as low as 50 ppm are moderately irritating (Vigliani and Zurlo 1955/Ex. 1-164; Mangold 1971; Industrial Bio-test Laboratories 1873, all as cited in NIOSH 1974a/Ex. 1-238; MacEwen, Theodore, and Vernot 1970/Ex.1-827; Pagnotto 1973, as cited in ACGIH 1986/Ex. 1-3, p. 27). NIOSH concluded that the "irritating or annoying effects * * * [of exposure to ammonia are] more dependent upon concentration than length of exposure," and that "a standard expressed as a time-weighted average is inappropriate since it would permit fluctuations to concentrations considerably higher than 50 ppm" (NIOSH 1974a/Ex. 1-238, p. 69). In the proposal, OSHA preliminarily concluded that NIOSH's recommended 50-ppm ceiling limit was above the effect level reported in the Detroit Department of Health studies (1965-1970, as cited in ACGIH 1986/Ex. 1-3, p. 27) for sensory irritation.

Several rulemaking participants objected to a reduction in the current 50-ppm TWA limit (Exs. 3-375, 3-582, 3-756, 3-869, 3-888, 3-902, 3-939, 3-1012, 3-1118, 8-25, 8-29, 8-62, 8-68, 8-123, 8-136, 113, and 122). At the rulemaking hearing, Lucas Seeman, Technical Advisor for the Association of Reproduction Materials Manufacturers (ARMM), testified that there was no basis for the proposed revision since the effects associated with exposure to 50 ppm of ammonia did not, in his opinion, constitute impairment of health:

> The Detroit Health Department studies, which make reference to "worker complaints" of ammonia exposures, appear to be based on subjective reactions of workers and not any manifestation of health impairment or physical evidence of severe irritation.
>
> None of the reference data added [by the ACGIH] in 1980 * * * made reference to any health impairment at the 25-ppm TWA or 35-ppm STEL levels of exposure. References added in 1980 did indicate that at 50 ppm workers reported no irritation, or minor to moderate irritation, and that they quickly became accustomed to the ammonia exposure up to that level (Tr. VII, pp. 222-224).

In reviewing the record evidence, OSHA finds that the 50-ppm 5-minute ceiling limit recommended by NIOSH is not sufficiently protective against ammonia's irritant effects. The evidence discussed by NIOSH (Ex. 150) and the testimony presented by Mr. Seeman (Tr. VII, pp. 222-224) show that, at levels below 50 ppm, some workers experience eye and upper respiratory tract irritation. This view is supported by Proctor, Hughes, and Fischman (*Chemical Hazards of the Workplace*, 2nd ed., 1988, p. 71), who report that even 5-minute exposures to 32 ppm caused nasal dryness in 10 percent of exposed volunteers, and that 5-minute exposures to 50 ppm ammonia caused nasal irritation and dryness in 20 percent of exposed volunteers. Deborah Berkowitz of the AFL-CIO testified that two companies in the meat packing industry evacuate the work place if airborne concentrations of ammonia reach 25 ppm (Tr. pp. 6-310 to 6-311).

OSHA finds that sensory irritation, such as that experienced by volunteers exposed to ammonia (Proctor, Hughes, and Fischman, 1988) constitutes material impairment of health. OSHA also finds that the fact that some workers may become acclimatized to ammonia exposures at concentrations as high as 50 ppm may account for the belief expressed by Mr. Seeman and others that 50 ppm is an acceptable exposure level. However, OSHA does not agree with this view of acclimatization because the long-term consequences of a continual assault on the sensory nerves are not known. In addition, acclimatization lessens the ability of workers to discern airborne concentrations of other hazardous materials.

The ACGIH (1986/Ex. 1-3) believes that an 8-hour TWA limit is necessary for ammonia because a study by Stombaugh, Teague, and Roller (1960/Ex. 1-29) reports that pigs exposed continuously to 103 to 145 ppm ammonia reduced their consumption of food and lost weight. The ACGIH interprets this study to mean that systemic toxicity occurs as a result of chronic exposure to ammonia. However, OSHA interprets this study differently, believing instead that it shows a secondary effect of the irritation traditionally associated with ammonia exposure. That is, in OSHA's view, these pigs stopped eating because they were experiencing too much respiratory and eye irritation to be interested in their food.

Thus, OSHA does not find it necessary in the final rule to establish an 8-hour TWA limit for ammonia to protect against chronic effects. Instead, the Agency concludes that a 15-minute STEL of 35 ppm will protect against this substance's irritant effects, which have been demonstrated to occur in workers exposed to ammonia at and below 50 ppm. OSHA concludes that the eye and upper respiratory tract irritation associated with ammonia exposure constitute material impairments of health and pose a significant risk to exposed workers.

AMMONIUM CHLORIDE (FUME)

CAS: 12125-02-9 Chemical Formula: NH_4Cl
H.S. No. 1022

No previous OSHA PEL had been established for ammonium chloride fumes. Based on the ACGIH recommendation, OSHA proposed a TWA limit of 10 mg/m^3 and a 20-mg/m^3 STEL, and NIOSH (Ex. 8–47, Table N1) concurred with these proposed limits, and they are established in the final rule. Ammonium chloride is a white crystalline solid, somewhat hygroscopic, with a cool, saline taste.

Ammonium chloride fume is an irritant to the skin and respiratory passages when inhaled and produces mild systemic toxicity when ingested (Sax 1968a/Ex. 1–867). Although exposure-response data are lacking for this substance, the ACGIH (1986/Ex. 1–3) judged that these workplace limits would be sufficient to prevent workers from experiencing respiratory irritation.

OSHA received no comment on the proposed addition of exposure limits for ammonium chloride fume to the Z tables, other than those submitted by NIOSH. OSHA finds that, in the absence of any limit on airborne exposure, employees are at significant risk of respiratory irritation caused by exposure to high concentrations of ammonium chloride fume. OSHA concludes that the respiratory irritation caused by exposure to ammonium chloride fume constitutes a material impairment of health. To substantially reduce this risk, OSHA is establishing an 8-hour TWA limit of 10 mg/m^3 and a 15-minute STEL of 20 mg/m^3 in the final rule.

BORATES, TETRA, SODIUM SALTS (ANHYDROUS, PENTAHYDRATE, AND DECAHYDRATE)

CAS: 1303–96–4 (Decahydrate); Chemical formula: $Na_2B_4O_7\ 10H_2O$
1330–43–4 (Anhydrous); Chemical Formula: $Na_2B_4O_7$
12179–04–3 (Pentahydrate); Chemical Formula: $Na_2B_4O_7\ 5H_2O$
H.S. Nos. 1036, 1038, and 1037

OSHA formerly had no exposure limits for the anhydrous or hydrated forms of sodium tetraborate. Based on the ACGIH-recommended TLVs for these substances, OSHA proposed a 1-mg/m^3 8-hour TWA PEL for the anhydrous and pentahydrate forms of sodium tetraborate and a 5-mg/m^3 TWA PEL for the decahydrate form. NIOSH (Ex. 8–47, Table N1) concurred with these proposed limits. However, during the rulemaking proceeding, OSHA received several comments on the proposed limits and obtained information on a large health survey currently being conducted by the U.S. Borax and Chemical Corporation. Based on this evidence, the Agency has determined that it is appropriate at this time to establish a 10-mg/m^3 8-hour TWA limit for all forms of the sodium tetraborates. Anhydrous sodium tetraborate is a light gray, orderless solid; the pentahydrate and decahydrate forms are white, orderless, and crystalline.

OSHA's proposed limits were based on some early studies cited by the ACGIH (1986/Ex. 1–3) and on observation that the anhydrous and pentahydrate forms of sodium tetraborate present a greater irritant hazard than does the decahydrate form. These early studies reported that exposure to the tetraborates produces irritation of the skin, eyes, and upper respiratory tract and can cause shortness of breath and nosebleeds. These studies were criticized at the hearings by John Middleton, Manager of Product Safety for the U.S. Borax Research Corporation, because they did not have sufficient exposure data to define a dose-response relationship (Tr. p. 9–113).

During the rulemaking, commenters discussed two NIOSH health hazard evaluations (HHEs) relevant to the borates. The first study (HHE 75–059–496, NIOSH 1978o) was conducted at the Kerr-McGee Chemical Corporation plant in Trona, California. NIOSH performed clinical examinations of nine employees exposed to tetraborates and collected total dust samples for each employee. Clinical examination revealed symptoms of eye irritation in five employees, nose irritation with bleeding in three workers, throat irritation in three employees, and chapping of the hands in four workers. Four of the nine dust samples exceeded 10 mg/m^3, with the highest being 29.9 mg/m^3. In testimony before the Occupational Safety and Health Standards Board in California in 1985, Dr. Charles Hine of Kerr-McGee stated that dust exposures at the California plant were probably well above the 10-mg/m^3 level because employees commented that dust from "frequent windstorms" was the main problem at the plant. Dr. Hine also noted that the NIOSH HHE reported that dust levels at the plant were excessive and that the visibility of employees was impaired (Ex. 3–744, Attachment I).

The second NIOSH HHE (conducted in 1980) reported on a walk-through survey of the U.S. Borax and Chemical Corporation's Boron, CA Operation. This HHE identified health complaints among employees, and its findings led to a larger, more comprehensive health survey in 1981 (HETA 80–109), a report of which was subsequently published in a peer-reviewed journal (Garabrant, Bernstein, Peters et al. 1985). Data on employees' respiratory symptoms were obtained by questionnaire, and total dust measurements were collected from historical data obtained between 1977 and 1981. The authors found no evidence of X-ray abnormalities or declines in pulmonary function among the 629 active employees examined. There was a dose-related and statistically significant increase in the frequency of reported symptoms, which included eye irritation, dry cough, nosebleeds, sore throat, shortness of breath, and chest tightness. Over 10 percent of employees having mean TWA exposures of 8.6 mg/m^3, measured as total tetraborate dust, reported experiencing nosebleeds, dry cough, eye irritation, and dryness of the mouth, nose, or throat. At a mean exposure level of 14.6 mg/m^3, between 15 and 30 percent of the employees examined reported these symptoms. The authors concluded that borax dust appears to act as a simple respiratory irritant and may cause small changes in pulmonary function among smokers who are also heavily exposed to borate dust.

U.S. Borax submitted to the record the written testimony of Dr. David Heilbron, a biostatistician (Ex. 3–744, Attachment 2), and of Dr. Ralph C. Smith, Professor of Occupational and Environmental Health, School of Public Health, University of Michigan (Ex. 3–744, Attachment 3), both of whom were of the opinion that the Garabrant et al. (1985) study's treatment of exposure data was biased. For example, Dr. Heilbron objected to the grouping of employees into three exposure categories, commenting that such aggregation "can seriously distort a dose-response relationship and particularly, the estimation of an effect threshold * * *" (Ex. 34–744, Attachment 2, p. 4). Dr. Heilbron also took issue with these authors' use of geometric means to describe the tetraborate exposure data; in the opinion of Dr. Heilbron, there was no statistical justification for the use of geometric means because of the heterogeneity of jobs within each exposure group.

OSHA believes that it is not possible to determine whether arithmetic or geometric means are appropriate without having access to the raw data. OSHA notes further that Garabrant and his co-authors (1985) both gathered and analyzed the data and that neither Dr. Heilbron nor Dr. Smith has access to these data.

Dr. Smith (Ex. 3–347, Attachment 3) believes that the exposure data in the Garabrant et al. (1985) study substantially underreported the actual exposures of the workers comprising the

study group. According to Dr. Smith, when the data are reanalyzed using arithmetic means, the observed health effects would be associated with exposures to much higher dust levels than those presented in the report (Ex. 3–744, Attachment 3, p. 13).

Because the raw exposure data from the study were not available to Dr. Smith, he based his reanalysis of the exposure data on an assumption that all individuals in a job category had exposures equal to the mean exposure level for the job category as a whole. For example, according to Dr. Smith, if "four laborers in the fusing building had average exposures of 49.2 mg/m^3 * * * [it was assumed] that all four had the same exposure" (Ex. 3–744, Attachment 3, p. 8). OSHA believes that Dr. Smith's approach provides less information about the actual exposures of the members of the cohort than does Dr. Garabrant's because Dr. Garabrant took two factors (representative data by job category as well as subjective self-reporting of exposure levels by employees) into account, while Dr. Smith only considered a single factor (job category). That is, Dr. Smith assumes that all workers in a job category have the same exposure, while Dr. Garabrant's approach recognizes the impact of such factors as individual differences in work practices, differences in control effectiveness at different workstations, etc., on the exposures of individuals in the same job category. OSHA is therefore unpersuaded by Dr. Smith's reanalysis; the Agency finds Dr. Garabrant's analysis convincing and believes that it more accurately reflects the true exposures of members of this cohort.

Largely because of questions raised concerning the dose-response relationship for tetraborates, U.S. Borax has been conducting a large epidemiologic study at its facility. This study, described at the informal hearing by Mr. Middleton (Tr. pp. 9–114 to 9–115, Ex. 120), will span a three- to four-year period and will obtain about 400 measurements of workplace tetraborate dust. The test protocols have been reviewed by representatives of OSHA, the U.S. Bureau of Mines, NIOSH, the ACGIH TLV Committee, and the Mine Safety and Health Administration. The final report is expected to be released in mid-1989. At the hearing, Mr. Middleton stated the position of U.S. Borax:

> Based on the fact that the present data does not support the establish[ment] of PEL's for these compounds and that U.S. Borax is presently collecting data that could be meaningful in establishing PEL's, we request that OSHA delay action on these compounds until these data are available and can be analyzed by OSHA and MSHA (Tr. pp. 9–115).

OSHA commends U.S. Borax for undertaking this effort to study the relationship between exposure to tetraborates and respiratory effects. OSHA believes that such data are essential to inform employees properly about hazards present in their workplaces and to guide employers in the development of effective occupational health programs. However, OSHA does not agree that the evidence currently available is inadequate to serve as a basis for establishing a PEL for the tetraborates at the present time. The study by Garabrant et al. (1985) does demonstrate a dose-response relationship for respiratory symptoms and exposure to sodium borates. OSHA finds that employees should be protected from experiencing the symptoms that have been reported. These symptoms, which have been reported in the more recent Garabrant et al. (1985) and NIOSH (1978o; 1980b) studies, as well as in the older literature, include nosebleeds, upper respiratory tract irritation, dermatitis, and dyspnea. OSHA believes that this evidence clearly indicates that the tetraborates act as primary respiratory and skin irritants, and that a 10-mg/m^3 PEL is clearly warranted. In light of the research currently being conducted by U.S. Borax, however, OSHA notes that the Agency will consider new evidence as it becomes available and will revise its limits if such action appears to be appropriate.

OSHA agrees with U.S. Borax that, at this time, there are insufficient data upon which to establish different PELs for the different hydrated forms of tetraborate. OSHA believes that current sampling and analytical procedures cannot distinguish among the various hydrated forms of tetraborate (Ex. 3–744, Attachment 3, pp. 4–5) and therefore that separate standards of 5 mg/m^3 and 1 mg/m^3 are not feasible at this time.

OSHA concludes that an 8-hour TWA of 10 mg/m^3 is appropriate for the tetraborates, and the final rule establishes this limit. OSHA finds that, in the absence of any limit on exposure, employees are at significant risk of experiencing acute eye, skin, and respiratory irritation effects, and that a 10-mg/m^3 PEL will substantially reduce these risks. The Agency considers the eye, skin, and upper respiratory tract irritation caused by exposure to all forms of sodium tetraborates to be material impairments of health.

BROMINE

CAS: 7726–95–6; Chemical Formula: Br_2
H.S. No. 1042

OSHA's previous exposure limit for bromine was 0.1 ppm as an 8-hour TWA. OSHA proposed to supplement this TWA limit with a STEL of 0.3 ppm, the same limit recommended by the ACGIH, and NIOSH (Ex. 8–47, Table N1) concurred with this proposal. In the final rule, the Agency is establishing a 0.1-ppm TWA limit and a 0.3-ppm STEL for bromine. Bromine is a dark, reddish-brown, noncombustible, diatomic liquid that has irritating vapors.

Early studies of bromine exposure indicated that workers exposed to 0.75 ppm for 6 hours exhibited no symptoms (Flury and Zernik 1931a, as cited in ACGIH 1986/Ex. 1–3, p. 65). Later studies reported physiological responses to different concentrations of bromine and used these findings to make the following recommendations: The maximum allowable concentration for prolonged exposures should be 0.1 to 0.15 ppm, and the maximum allowable concentration for short-term exposures (i.e., 30 minutes to one hour) should be 4 ppm (Henderson and Haggard 1943b, as cited in ACGIH 1986/Ex. 1–3, p. 65). These investigators found levels of 40 to 60 ppm dangerous for short-term exposures, and a level of 1000 ppm proved rapidly fatal even during short exposures. These authors reported that the effects of exposure to bromine include respiratory irritation and lung edema. Elkins (1959a/Ex. 1106) reported that workers exposed to 1 ppm in a plant handling liquid bromine found this level excessively irritating.

OSHA received no comments on its proposed STEL for bromine, other than the NIOSH concurrence statement. The Agency finds that both the TWA and the short-term limits are necessary to substantially reduce the risk of respiratory irritation and lung damage that could occur following short-term exposures to concentrations of bromine that would be permitted by the 8-hour TWA limit alone. OSHA considers the effects related to bromine exposure material impairments of health. Therefore, OSHA is revising the limit for bromine to 0.1 ppm as an 8-hour TWA and 0.3 ppm as a 15-minute STEL.

2-BUTANONE (METHYL ETHYL KETONE)

CAS: 78–93–3; Chemical Formula: $CH_3COCH_2CH_3$
H.S. No. 1045

OSHA's former exposure limit for 2-butanone was 200 ppm TWA. OSHA proposed to supplement this limit with a STEL of 300 ppm, based on the ACGIH (1986/Ex. 1–3) recommendation. NIOSH (Ex. 8–47, Table N1) concurred with this proposal. The final rule establishes a 200-ppm TWA limit and a 300-ppm STEL

for 2-butanone. 2-Butanone is a colorless, flammable liquid with an objectionable odor.

2-Butanone is an ocular and upper respiratory tract irritant. One study (Nelson, Enge, Ross et al. 1943/Ex. 1–66) reported that exposures to 200 ppm for 3 to 5 minutes caused mild eye irritation in some subjects and that others experienced slight nose and throat irritation at concentrations of 100 ppm. Exposure to 350 ppm caused eye and nasal irritation in a majority of subjects tested. Studies conducted in the 1940s noted low-grade intoxication resulting from exposure to 300 to 600 ppm (Smith and Mayers 1944, as cited in ACGIH 1986/Ex. 1–3, p. 395). Later studies have shown that approximately 50 percent of trained panelists experienced eye and nose irritation at 200 ppm (as reported in ACGIH 1986/Ex. 1–3, p. 395).

In the preamble discussion on 2-butanone, OSHA noted that a number of studies indicate that the proposed limits may not be sufficient to fully protect workers from the irritant effects of this substance (ACGIH 1986/Ex. 1–3; Nelson, Enge, Ross et al. 1943/Ex. 1–66). The ACGIH also cited a manufacturer's publication that stated that 200 ppm was the highest concentration judged by human subjects to be "satisfactory" for eight hours. In addition, another study cited by the ACGIH (1986/Ex. 1–3) reported that exposure to 200 ppm was associated with a 50-percent response rate for eye and nasal irritation (the degree of irritation was not specified).

OSHA specifically requested comment on whether its proposed limits for 2-butanone were sufficiently protective. The New Jersey Department of Public Health (Exs. 144, 144A) urged OSHA to set its limits for 2-butanone based on EPA's IRIS data. The use of such an approach is discussed in Section VI.A of the preamble. The AFL–CIO (Ex. 194) supported the establishment of a STEL for butanone.

OSHA has determined that its previous 8-hour TWA limit of 200 ppm was not sufficient to protect workers from experiencing the significant irritation and narcotic effects that are associated with short-term exposures to high concentrations of 2-butanone. After reviewing the available reports describing human sensory responses to short-term exposures to 2-butanone, the Agency concludes that a 300-ppm STEL is also necessary to reduce the significant risk of sensory irritation; exposure to 350 ppm for three to five minutes was reported to cause eye, nose, and throat irritation in a majority of subjects (Nelson, Enge, Ross et al. 1943/Ex. 1–66). Accordingly, OSHA is establishing a 200-ppm TWA limit and a 300-ppm 15-minute STEL for 2-butanone to protect employees from the significant risk of sensory irritation; OSHA considers the irritation caused by 2-butanone to be a material impairment of health or functional capacity.

n-BUTYL ACETATE

CAS No. 123-86-4; Chemical Formula: $CH_3COO(CH_2)_3CH_3$
H.S. No. 1047

The previous OSHA exposure limit for n-butyl acetate was 150 ppm, measured as an 8-hour TWA. OSHA proposed the adoption of a 15-minute STEL of 200 ppm to supplement the TWA limit. NIOSH (Ex. 8–47, Table N1) concurred with this proposal. The final rule establishes limits of 150 ppm as an 8-hour TWA and 200 ppm as a 15-minute STEL for this substance; these are the same limits as those recommended by the ACGIH (1986/Ex. 1–3). n-Butyl acetate is a colorless liquid with a fruity odor.

n-Butyl acetate is an irritant to the eyes, skin, and respiratory system. In a study involving cats exposed for six hours to 6100 ppm, slight narcotic effects were noted (Flury and Wirth 1933, as cited in ACGIH 1986/Ex. 1–3, p. 72). When exposed to 4200 ppm n-butyl acetate for six days at six hours per day, cats experienced slight irritation of the respiratory passage; at 3100 ppm, changes in blood cell morphology were recorded. At exposures of 1600 ppm, these cats exhibited slight irritation of the eyes and increased salivation (Flury and Wirth 1933, as cited in ACGIH 1986/Ex. 1–3, p. 72). Air concentrations of 10,000 ppm n-butyl acetate proved fatal to rats after eight hours; four hours of exposure at the same level produced no deaths (Smyth 1956/Ex. 1–759). A paper by Sayers, Schrenk, and Patty (1936/Ex. 1–802) reported that guinea pigs demonstrated eye irritation effects at 3300 ppm, became unconscious after nine hours of exposure to 7000 ppm, and died after four hours of exposure to 14,000 ppm.

Human volunteers complained that throat irritation, which began at an exposure level of 200 ppm n-butyl acetate, worsened and became quite severe at 300 ppm (Nelson, Enge, Ross et al. 1943/Ex. 1–66). NIOSH was the only commenter to the record in response to OSHA's proposed STEL for n-butyl acetate.

OSHA finds that workers are at significant risk of experiencing severe eye, skin, and respiratory irritation, in addition to narcotic effects, that are associated with short-term exposures to this substance at levels above the 8-hour limit. The Agency considers the irritant and narcotic effects resulting from exposure to n-butyl acetate to be material impairments of health and functional capacity. OSHA concludes that a STEL is necessary to reduce this risk, and the Agency is therefore revising its limit for n-butyl acetate to 150 ppm as an 8-hour TWA and 200 ppm as a 15-minute STEL.

n-BUTYL LACTATE

CAS: 138–22–7; Chemical Formula: $C_7H_{14}O_3$
H.S. No. 1053

OSHA previously had no limit for n-butyl lactate but proposed a 5-ppm 8-hour TWA limit, based on the ACGIH recommendation. NIOSH (Ex. 8–47, Table N1) concurred with the proposed 5-ppm TWA limit, and this limit is established in the final rule. Butyl lactate is a colorless liquid ester of lactic acid.

In humans, prolonged exposures to n-butyl lactate at approximately 7 ppm, with brief peak excursions to 11 ppm, caused headache, irritation of the pharyngeal and laryngeal mucosa, and coughing in all workers, and occasional nausea, vomiting, and sleepinessh in some (Zuidema and Pel 1969, as cited in ACGIH 1986/Ex. 1–3, p. 82). Headache, coughing, and irritation of the pharynx were sometimes related to n-butyl lactate concentrations of 4 ppm; however, no adverse effects were observed at a concentration of 1.4 ppm. Studies employing improved sampling and analytic methods have subsequently concluded that, although the odor of n-butyl lactate is discernible at the 7-ppm level, this concentration does not produce objectionable or injurious effects (Turner 1972/as cited in ACGIH 1986/Ex. 1–3, p. 82).

In the preamble discussion of the proposed limit for this substance, OSHA noted that some studies reported acute adverse effects associated with exposure levels below the proposed 5-ppm TWA limit. This was also pointed out by Dr. Grace Ziem, an independent physician (Ex. 46). Based on the study by Turner (1972, as cited in ACGIH 1986/Ex. 1–3, p. 82), which employed improved sampling and analytical techniques as compared to earlier studies, OSHA judges that promulgation of a 5-ppm 8-hour TWA limit will effectively protect workers from the significant risks of irritation, headache, and nausea caused by exposure to higher conentrations of n-butyl lactate. OSHA considers these adverse effects to represent material impairments of health. Therefore, OSHA is establishing a 5-ppm 8-hour TWA limit for n-butyl lactate.

n-BUTYL MERCAPTAN

CAS: 109-79-5; Chemical Formula: $CH_3CH_2CH_2CH_2SH$
H.S. No. 1054

n-Butyl mercaptan is a colorless, flammable liquid and has a strong, obnoxious, garlic-like odor. It is used as a solvent, a chemical intermediate, and an odorant for natural gas. OSHA's previous limit for n-butyl mercaptan was 10 ppm as an 8-hour TWA. OSHA proposed a lower limit of 0.5 ppm TWA, based on the ACGIH recommendation, and the final rule establishes this limit.

Humans exposed to concentrations of n-butyl mercaptan report that the "readily noticeable" odor level for this substance is between 0.1 and 1 ppm, although the odor threshold is significantly below this level (ranging from 0.001 to 0.0001 ppm) Gobbato and Terribile (1968/Ex. 1-178) have reported that symptoms of CNS toxicity occurred in humans exposed for one hour to concentrations of n-butyl mercaptan believed to lie in the range of 50 to 500 ppm. These same authors reported that mucosal irritation occurred in human volunteers exposed to 4 ppm of ethyl mercaptan, a closely related substance. Irritation did not occur at exposures to 0.4 ppm. The ACGIH established the TLV for n-butyl mercaptan at 0.5 ppm, to protect against the intolerable odor effects, mucosal irritation, and CNS toxicity that occur on exposure to higher concentrations of this substance.

The current PEL of 10 ppm is between 10 and 100 times higher than the concentration of n-butyl mercaptan that is readily detected by smell and is more than twice the concentration reported as causing mucosal irritation for a closely related substance. OSHA finds that workers are at risk of significant acute effects in the absence of a more stringent limit.

In its prehearing comments, NIOSH (Ex. 8-47, Table N7) pointed out that it has recommended a 0.5-ppm ceiling limit for n-butyl mercaptan, rather than a TWA limit, for this substance. No other comments were submitted to the record. In accordance with the criteria in its June 7, 1988 NPRM (53 *FR* 20977), OSHA is establishing the 0.5-ppm TWA limit for n-butyl mercaptan. The Agency concludes that this PEL will substantially reduce the risks of irritation, CNS toxicity, and intolerable odor effects, which together constitute material health impairments.

CAPROLACTAM (DUST)

CAS: 105-60-2; Chemical Formula: $C_6H_{11}NO$
H.S. No. 1064

OSHA had no previous permissible exposure limit for caprolactam dust; however, a 1-mg/m³ 8-hour TWA and a 3-mg/m³ STEL were proposed, based on the recommended limits adopted by the ACGIH, and NIOSH (Ex. 8-47, Table N1) concurred with these limits. The final rule establishes these limits. Caprolactam is a white crystalline solid with an unpleasant odor.

In humans, caprolactam has been shown to be a convulsant, a dermal and respiratory irritant, and a dermal sensitizer; however, dosage levels in humans are ill-defined (Ferguson and Wheeler 1973/Ex. 1-1108; Tuma, Orson, Fossella, and Waidhofer 1981/Ex. 1-1071). In animals, exposure to caprolactam by several routes can cause convulsions, tremors, mydriasis, opisthotonus (Elison, Lien, Zinger et al. 1971/Ex. 1-1050; Lien, Lien, and Tong 1971/Ex. 1-1089) and salivation (Goldblatt, Farquharson, Bennett, and Askew 1954/Ex. 1-1044). Cardiovascular and respiratory effects have been reported in rabbits and cats, with an initial increase in blood pressure followed by a decrease in blood pressure and an increased respiratory rate (Goldblatt, Farquharson, Bennett, and Askew 1954/Ex. 1-1044). Weight loss and initial growth depression occurred in rats and mice (Morrison, Ross, and Ruth 1980/Ex. 1-1062).

One animal study observed that caprolactam's convulsant effects on rats, rabbits, and cats occur at injection doses above 100 mg/kg (Goldblatt, Farquharson, Bennett, and Askew 1954/Ex. 1-1044). Results of studies in guinea pigs were consistent with these findings (Hohensee 1951, as cited in ACGIH 1986/Ex. 1-3, p. 95). In a 90-day feeding study of dogs, Burdock, Kolwick, Alsakor, and Marshall (1984, as cited in ACGIH 1986/Ex. 1-3, p. 95) reported that dogs given dietary dose levels of 0.1, 0.5, or 1.0 percent caprolactam showed weight losses at both the 1.0-percent and 0.5-percent levels. Hematologic and opthalmologic changes did not occur. In a two-year bioassay of rats and mice, caprolactam was not observed to be carcinogenic (NCI/NTP 1982, as cited in ACGIH 1986/Ex. 1-3, p. 95). A Polish study observed hematologic and systemic changes, increased mortality, kidney and liver damage, and growth inhibition in animals given daily doses of 50 or 100 mg/kg (Zwierzchowski, Kowalski, Szendzikowski, and Slusarchzyk-Zalobna 1967, as cited in ACGIH 1986/Ex. 13, p. 96.1). The results of early studies of caprolactam's teratogenicity in rats and rabbits indicate that it is not teratogenic even at doses as high as 1000 mg/kg/day (Gad, Powers, Robinson et al. 1984, as cited in ACGIH 1986/Ex. 1-3, p. 96.1).

Studies of industrial exposures to caprolactam dust in Germany report severe irritation on inhalation of 10 percent caprolactam in dust (Hohensee 1951, as cited in ACGIH 1986/Ex. 1-3, p. 95). Workers experienced a bitter taste, nervousness, epistaxis, upper respiratory tract irritation, and dry and splitting skin on the lips and nose (Hohensee 1951, as cited in ACGIH 1986/Ex. 1-3, p. 95). Direct contact with the solid form of caprolactam produces primary skin irritation (Ferguson 1972, as cited in ACGIH 1986/Ex. 1-3, p. 96.1). Brief (1972, as cited in ACGIH 1986/Ex 1-3, p. 96.1) also reports that the dust produces skin irritation.

OSHA received no comments, other than NIOSH's, on its proposed limits for caprolactam dust. Based on its review of the health evidence, OSHA concludes that, in the absence of any limit on employee exposure to caprolactam dust, workers are at significant risk of respiratory irritation, adverse nervous system effects, and possible cardiovascular effects; the Agency considers these effects to be material impairments of health. OSHA finds that promulgation of the 1-mg/m³ TWA and 3-mg/m³ STEL limits for caprolactam dust will substantially reduce this risk.

CAPROLACTAM (VAPOR)

CAS: 105-60-2; Chemical Formula: $C_6H_{11}NO$
H.S. No. 1065

OSHA had no previous permissible exposure limit for caprolactam as vapor. The Agency proposed a TWA of 5 ppm (20 mg/m³) for the vapor, supplemented by a STEL of 10 ppm (40 mg/m³), based on the limits adopted by the ACGIH. These limits are established in the final rule. Caprolactam is a white crystalline solid at room temperature; thus, high vapor levels occur only at elevated temperatures.

The health effects of exposure to caprolactam vapor are identical to those described for caprolactam dust, except that contact with the vapor is reported to be even more irritating (Hohensee 1951, as cited in ACGIH 1986/Ex. 1-3, p. 95). Workers exposed to the vapor at approximately 12 ppm complained of a bitter taste in the mouth, nervousness, epistaxis, upper respiratory tract congestion, and dry and splitting skin; other workers reported experiencing heartburn, flatulence, and a heavy feeling in the stomach (Hohensee 1951, as cited in ACGIH 1986/Ex. 1-3, p. 95).

In another report of industrial exposure to the vapor, Ferguson and Wheeler (1973/Ex. 1-1108) reported that workers routinely exposed to unspecified levels and occasionally to concentrations as high as 100 ppm for 18 years reported severe discomfort from burning nose, throat, and eyes. This

irritation response was dose-related, with no workers reporting effects at 7 ppm or below, some experiencing transient upper respiratory tract irritation at levels above that, and others reporting eye irritation at concentrations of 25 ppm and above (Ferguson and Wheeler 1973/Ex. 1–1108). Ferguson (1972, as cited in ACGIH 1986/Ex. 1–3, p. 96.1) noted that a group of 143 workers, some of whom were exposed for as long as 17 years to vapor concentrations of 5 to 10 ppm, showed no evidence of adverse effects. At higher vapor exposures (13 to 130 ppm), all subjects experienced eye irritation (Ferguson 1972, as cited in ACGIH 1986/Ex. 1–3, p. 96.1). Human volunteers exposed at low relative humidities to concentrations of the vapor in the range of 10 to 100 ppm showed a dose-related response, but at higher relative humidities, no irritation was observed below a concentration of 14 ppm (Ferguson and Wheeler 1973/Ex. 1–1108).

NIOSH (Ex. 8–47, Table N2; Tr. p. 3–86) did not concur with the Agency's proposal to establish an 8-hour TWA exposure limit of 5 ppm (20 mg/m^3) and a 10-ppm (40-mg/m^3) STEL for caprolactam vapor. NIOSH (Ex. 8–47) noted that the 1986 ACGIH *Documentation* (Ex. 1–3) lists a TLV–TWA of 1 mg/m^3 for the combined vapor and aerosol of caprolactam and 0.22 ppm if the substance is present only as a vapor. The proposed change in the 1986 TLV was recommended to prevent early signs of irritation in some workers. NIOSH observed that "the proposed PEL does not appear to provide a sufficient margin of safety to caprolactam vapor," based on available human exposure responses. No other comments on this substance were submitted to the rulemaking record.

As explained in Section IV.D. of this preamble, which discusses the boundaries of today's rulemaking, the Agency confined its attention to the substances and exposure limits listed in the 1987–1988 edition of ACGIH's *Threshold Limit Values and Biological Exposure Indices* (ACGIH 1987/Ex. 1–16). Caprolactam vapor is listed on ACGIH's Notice of Intended Changes but new limits have neither been reviewed nor adopted by that organization to date. Under these circumstances, OSHA believes it prudent to promulgate the limits as proposed for caprolactam vapor. The Agency is establishing an 8-hour TWA limit of 5 ppm (20 mg/m^3) and a 15-minute STEL of 10 ppm (40 mg/m^3) for this previously unregulated substance. OSHA concludes that these PELs will substantially reduce the significant risk of eye, upper respiratory tract, and skin irritation that are permitted in the absence of any exposure limit for caprolactam vapor. OSHA considers the irritant effects resulting from exposure to caprolactam vapor to be material impairments of health. The Agency will continue to monitor the health evidence for this substance to determine whether further action is warranted.

CESIUM HYDROXIDE

CAS: 21351–79–1; Chemical Formula: CsOH
H.S. No. 1077

OSHA formerly had no limit for cesium hydroxide; however, based on the ACGIH recommendation, OSHA proposed the establishment of a 2-mg/m^3 limit as an 8-hour TWA. NIOSH (Ex. 8–47, Table N1) concurred with OSHA's proposed limit for this substance, and the Agency is establishing this limit for cesium hydroxide in the final rule. Cesium hydroxide is a colorless or yellowish fused crystalline mass; it is the strongest base known and is highly soluble in both water and alcohol.

Animal studies indicate that cesium hydroxide has an acute oral toxicity of about one-third that of potassium hydroxide, which causes lesions of the nasal septum and irritation of the eyes and respiratory tract (Karpov 1971/Ex. 1–1115). The oral LD_{50} for cesium hydroxide in rats is 1016 mg/kg. Although a concentration of 5 percent cesium hydroxide did not produce skin irritation, contact with this concentration did result in severe eye irritation. Cesium hydroxide does not cause skin sensitization (Johnson, Lewis, and Perone 1972, as cited in ACGIH 1986/Ex. 1–3, p. 113). No rulemaking participants other than NIOSH commented on the proposed 2-mg/m^3 TWA limit for cesium hydroxide.

In the final rule, the Agency is promulgating an 8-hour TWA PEL of 2 mg/m^3 for cesium hydroxide and concludes that this limit will protect workers from the significant risk of severe eye irritation associated with exposure to this substance at levels above the new PEL. The Agency considers the severe eye irritation caused by exposure to cesium hydroxide a material impairment of health and functional capacity.

CHLORINE

CAS: 7782–50–5; Chemical Formula: Cl_2
H.S. No. 1079

The previous OSHA limit for chlorine was 1 ppm as a ceiling limit. OSHA proposed to revise this limit to 0.5 ppm measured over 15 minutes, which was the limit recommended by NIOSH (1976b/Ex. 1–276) in its criteria document; NIOSH (Ex. 8–47, Table N1) concurred with the proposed limit. However, the final rule establishes a PEL of 0.5 ppm TWA with a 15-minute short-term exposure limit of 1 ppm for chlorine. Chlorine is a greenish-yellow, noncombustible gas at atmospheric pressure; it has a suffocating odor. At −35 C, it condenses to an amber liquid.

Exposure to chlorine at concentrations around 5 ppm has been associated with respiratory symptoms, erosion of the teeth, and inflamation of the mucous membranes (Flury and Zernik 1931c/Ex. 1–1199; Patty 1963c/Ex. 1–854). Ferris, Burgess, and Worcester (1967/Ex. 1–316) reported slight effects on the respiratory system in workers exposed to chlorine concentrations ranging from negligible to 7 ppm. Rupp and Henschler (1967/Ex. 1–1122) reported burning of the eyes among human subjects exposed to 0.5 ppm; an unspecified number of these subjects reported painful eyes after 15 minutes' exposure to this level. In a separate test, subjects reported respiratory irritation on exposure to 0.5 ppm, and a concentration of 1 ppm was described as being uncomfortable.

At the time of OSHA's proposal, the limits adopted by the ACGIH were a 1-ppm TLV–TWA and a 3-ppm TLV–STEL; these limits were based on the reports described above and were established to "minimize chronic changes in the lungs, accelerated aging, and erosion of the teeth" (ACGIH 1986/Ex. 1–3, p. 117). NIOSH (1976b/Ex. 1–276) reviewed these studies, as did others (Matt 1889, as cited in Flury and Zernick 1931c/Ex. 1–1199; Beck 1959, as cited in NIOSH 1976b/Ex. 1–276) that reported ocular and respiratory irritation associated with exposure to chlorine levels of around 1 ppm for 30 minutes or less. NIOSH (1976b/Ex. 1–276) recommended a 15-minute 0.5-ppm limit to prevent possible eye and respiratory tract irritation.

The United Paperworkers International Union (UPIU) (Ex. 8–37) cited the NIOSH Criteria Document (Ex. 1–276) and ACGIH Documentation (Ex. 1–3) as evidence that exposure to 0.5 ppm chlorine causes respiratory irritation. The UPIU also submitted several studies indicating that decrements in pulmonary function may persist for several days or weeks following acute exposure to concentrations of chlorine requiring medical treatment. In addition, the UPIU cited a number of studies indicating that pulp mill workers and chlorine production plant workers experience declines in pulmonary function as a result of chronic exposure to low levels of chlorine (Ex. 8–37); however,

interpretation of many of these studies is complicated by a lack of exposure data or the presence of confounding exposure to other respiratory toxins, such as sulfur dioxide. The UPIU (Ex. 8–37) supported the promulgation of a 0.2 ppm limit for chlorine.

In 1986, the ACGIH proposed revising the TLVs for chlorine to 0.5 ppm as an 8-hour TWA and 1 ppm as a 15-minute STEL. This proposal was based on a review of two recent studies. One study, a 1981 doctoral dissertation by Anglen (Ex. 108A), was sponsored by the Chlorine Institute and was conducted on 29 human subjects. This study reported statistically significant changes in pulmonary function and subjective irritation resulting from exposure to 1 ppm chlorine for eight hours. No significant ocular effects were noted at this exposure level and duration. Exposure to 0.5 ppm for eight hours was not associated with significant declines in pulmonary function, and subjective irritation was also less severe at this level than at 1 ppm (Anglen 1981, Ex. 108A). During the eight-hour exposure to 1 ppm, sensory responses of itching or burning of the throat were reported to be "just perceptible" or "distinctly perceptible." A short-term (30)-minute) exposure to 2 ppm produced no increase in subjective irritation compared with controls.

These findings were confirmed in a study of eight healthy volunteers exposed to 0.5 or 1 ppm chlorine concentrations (Rotman, Fliegelman, Moore et al. 1983/Ex. 1–108B). Significant declines in pulmonary function were associated with exposure to 1 ppm but not to 0.5 ppm.

The Chlorine Institute (Ex. 3–828) described a recent animal study conducted by the Chemical Industry Institute of Toxicology (CIIT). In this study, groups of 20 rats were exposed to 1, 3, or 9 ppm chlorine for six hours/day, five days/week, for six weeks. Exposure to the two highest levels resulted in significant decreases in body weight. Inflammation of the upper and/or lower respiratory tract was observed in the 9-ppm group and, to a lesser extent, in the 3- and 1-ppm groups. Pathological and clinical changes were not observed in the 1-ppm group, but were seen in the 3- and 9-ppm groups.

Several rulemaking participants urged OSHA to adopt the more recent ACGIH limits of 0.5 ppm TWA and 1 ppm STEL (Exs. 3–677, 3–741, 3–828, and 3–1150; Tr. pp. 10–165 to 10–170; Tr. pp. 10–178 to 10–180). For example, the Chlorine Institute commented as follows:

> The imposition of an instantaneous ceiling PEL is inappropriate. The Chlorine Institute's University of Michigan and CIIT studies demonstrate conclusively that sensory effects and adverse pulmonary function effects are directly related to prolonged chlorine exposures and are correctly controlled by a PEL expressed as a Time Weighted Average (TWA) * * *. The Chlorine Institute supports * * * [the ACGIH limits] as the correct PEL for adoption by OSHA, and we submit that the evidence is conclusive that such a PEL is totally protective of worker health in chlorine-producing and chlorine-using industries (Ex. 3–828, p. 3).

In its posthearing comment, NIOSH (Ex. 150) reaffirmed its recommended TWA of 0.5 ppm as a 15-minute limit, based on the findings of Rupp and Henschler (1967/Ex. 1–1122):

> The studies of Anglen (1981) and Rotman (1983), as summarized by the ACGIH, if considered alone, would support the ACGIH TWA TLV of 0.5 ppm with a STEL of 1 ppm. However, in the studies of Rupp and Henschler (1967), exposure to chlorine at concentrations of approximately 0.5 ppm resulted in conjunctival pain in several subjects after 15 minutes; in their second study, subjects reported respiratory irritation after exposure to 0.5 ppm for 25 minutes * * *.
>
> The Rupp and Henschler study (1967), although it has been criticized for lack of a control group (Ex. 3–685) confirms the Anglen (1981), Rotman et al. (1983), and CIIT studies (Ex. 3–828) that there is a significant risk of irritation and a risk of respiratory inflammation at the present PEL of 1 ppm ceiling. Reduction of the current PEL to 0.5 ppm ceiling will reduce the risk of respiratory irritation and pulmonary function changes, and minimize the subjective complaints of irritation (Ex. 150, Comments on Chlorine).

The Dow Chemical Company submitted a critical review of the NIOSH (1976b/Ex. 1–276) criteria document on chlorine and the Rupp and Henschler (1967/Ex. 1–1122) study that was prepared in 1979 by Dr. Ralph G. Smith, who directed the University of Michigan (Anglen 1981) study (Ex. 3–741, Appendix B; Tr. pp. 10–165 to 10–170). In his review, Dr. Smith criticized the Rupp and Henschler (1967/Ex. 1–1122) study because the design of the exposure facility led to uncertainties in determining actual exposure levels present in the test room. He also remarked that the chlorine was passed through "liquid paraffin," which may have produced chlorinated hydrocarbons. In addition, Dr. Smith felt that the air compressor used may have caused contamination of the air in the test room by carbon monoxide and other impurities. Dr. Smith believed these observations were important "because one of the effects allegedly resulting from short exposures to low levels of chlorine was headaches, a symptom which we have never had reported to us by a subject in the University of Michigan (Anglen 1981) exposures" (Ex. 3–741, Appendix B, pp. 9–10).

After reviewing the evidence and testimony presented in the record on the effects of exposure to chlorine gas, OSHA concludes that there is clearly a significant risk of pulmonary function impairment and sensory irritation at the current 1-ppm ceiling PEL; such effects have been demonstrated by the Anglen (1981/Ex. 108A) and Rotman, Fliegelman, Moore et al. (1983/Ex. 108B) studies in human subjects exposed to 1 ppm for 8 hours, an exposure level and duration that would be permitted by the former PEL. In addition, pulmonary inflammation has been observed in rats exposed daily for six weeks to 1 ppm chlorine. Therefore, OSHA finds that it is necessary to revise its current limit for chlorine.

The human studies by Anglen (1981/Ex. 108A) and by Rotman, Fliegelman, Moore et al. (1983/Ex. 108B) also indicate that exposure to 0.5 ppm chlorine for as long as 8 hours is not associated with impairment of pulmonary function or significant sensory irritation; these findings are in contrast to the earlier German reports upon which the NIOSH REL of 0.5 ppm (15 minutes) is based. However, the German studies, in particular those of Rupp and Henschler (1967/Ex. 1–1122), appear to have had methodological shortcomings that call into question the finding that exposure to 0.5 ppm chlorine is associated with significant acute effects. Therefore, OSHA judges, based on the more recent University of Michigan study, that an exposure limit of 0.5 ppm TWA with a 1-ppm 15 minute STEL will reduce the risk of irritation and pulmonary function decline in workers, and is today revising its limit for chlorine to these values. OSHA considers the effects of respiratory irritation and the declines in pulmonary function associated with chlorine exposure to be material impairments of health.

CHLOROACETYL CHLORIDE

CAS: 79–04–9; Chemical Formula: $ClCH_2COCl$
H.S. No. 1083

No previous exposure limit existed for chloroacetyl chloride. OSHA proposed a 0.05-ppm 8-hour TWA limit for this substance, based on the ACGIH recommendation, and NIOSH (Ex. 8–47, Table N1) concurred with this proposal. This limit is established in the final rule. Chloroacetyl chloride is a colorless liquid with a pungent odor.

The oral LD_{50} in rats fed this substance is between 0.12 and 0.25 g/kg. Chloroacetyl chloride is corrosive to the

skin and eyes, and skin absorption of this substance can be lethal. Inhalation of 4 ppm for five to ten minutes caused respiratory problems in rats; however, no effect was observed in these animals when they inhaled 2.5 ppm for a period of seven hours (Dow Chemical Company 1977a, as cited in ACGIH 1986/Ex. 1–3, p. 122). Thirty-day inhalation studies with rats, mice, and hamsters showed eye and respiratory irritation at 2.5 ppm and no effect at 0.5 ppm (Dow Chemical Company 1977a, as cited in ACGIH 1986/Ex. 1–3, p. 122).

Reports of the acute effects associated with exposure to chloroacetyl chloride in humans include mild to moderate skin burns and erythema, eye burns and tearing, cough, dyspnea, and cyanosis, as well as mild gastrointestinal effects. Eye and respiratory irritation occurred in an industrial setting characterized by an exposure level of 0.009 to 0.017 ppm, with excursions as high as 0.140 ppm (Dow Chemical Company 1977a, as cited in ACGIH 1986/Ex. 1–3, p. 122). An accidental drenching with a mixture containing chloroacetyl chloride resulted in extensive first- and second-degree burns, pulmonary edema, and three episodes of cardiac arrest, followed by coma and anoxia-induced brain damage (Pagnotto 1978, as cited in ACGIH 1986/Ex. 1–3, p. 122). Other ingredients of the mixture involved in the accident included xylidine, benzene, and sodium carbonate. Rescuers of this victim experienced hand blisters, chest tightness, and nausea for two days. OSHA received no comments other than NIOSH's on the proposed 0.05-ppm TWA limit for chloroacetyl chloride.

The Agency concludes that an 8-hour TWA limit of 0.05 ppm for chloroacetyl chloride is necessary to protect employees from the significant risk of eye, skin, and respiratory irritation; gastrointestinal effects; and severe systemic effects, including life-threatening coma, cardiac arrest, and pulmonary edema, to which they could otherwise be exposed in the absence of any OSHA limit; the Agency considers each of these exposure-related adverse effects to be material impairments of health and functional capacity. Accordingly, OSHA is establishing an occupational limit of 0.05 ppm as an 8-hour TWA for chloroacetyl chloride.

o-CHLOROBENZYLIDENE MALONONITRILE

CAS: 2698–41–1; Chemical Formula: $ClC_6H_4CH = C(CN)_2$
H.S. No. 1084

OSHA's previous PEL for o-chlorobenzylidene malononitrile (OCBM) was 0.05 ppm as an 8-hour TWA. The Agency has proposed revising this limit to 0.05 ppm as a ceiling, with a skin notation, based on the ACGIH (1986/Ex. 1–3) recommendation. This revision is incorporated in the final rule. NIOSH (Ex. 8–47, Table N1) concurred with OSHA's proposed limit for this substance. o-Chlorobenzylidene malononitrile is a white crystalline solid with a pepper-like odor.

OCBM has extremely irritating properties. It couses intense eye and skin irritation, coughing, difficulty in breathing, chest tightness, running nose, dizziness, nausea, and vomiting. These effects are evident on exposure to concentrations between 12 and 20 mg/m^3 (1.5 to 2.5 ppm), and they become incapacitating within 20 seconds of exposure; the effects persist for approximately 5 to 10 minutes after the victim has been removed to fresh air (*Military Chemistry and Chemical Agents* 1963, as cited in ACGIH 1986/Ex. 1–3, p. 124).

OCBM is only slightly toxic to laboratory animals when they are exposed intravenously, subcutaneously, or through inhalation (Punte, Weimer, Ballard, and Wilding 1962/Ex. 1–354). In animals, it has been demonstrated that OCBM is metabolized by the body into cyanide (Frankenberg and Sorbo 1973/Ex. 1–480). Short-term exposures to high levels of OCBM did not cause carcinogenic, teratogenic, or embryolethal effects in animals (McNamara et al. 1973, as cited in ACGIH 1986/Ex. 1–3, p. 124).

Three of four human volunteers exposed to a 1.5-mg/m^3 (0.19-ppm) concentration of OCBM aerosol dispersed from a 10-percent solution of methylene chloride for 90 minutes developed headaches, and one showed mild eye and nose irritation. Headache persisted for 24 hours in two subjects. At 4 to 5 mg/m^3 (0.5 to 0.6 ppm), subjects' problem-solving abilities were affected and they showed eye irritation, conjunctivitis, lacrimation, and skin burning (Punte, Owens, and Gutentag 1963/Ex. 1–353). Other researchers observed no persistent clinical abnormalities in seven subjects exposed to OCBM at concentrations ranging from 1 to 13 mg/m^3 (0.13 to 1.6 ppm) over a 15-day period; however, none of these subjects developed a tolerance for the compound. Severe skin sensitization has also been reported in workers handling OCBM (Shmunes and Taylor 1973/Ex. 1–370). No comments, except those submitted by NIOSH, were received on OSHA's proposed revision of the limit for OCBM.

In the final rule, OSHA is establishing a PEL 0.05 ppm as a ceiling, with a skin notation, to reduce the risks associated with elevated short-term exposures to OCBM. The Agency concludes that workers are at significant risk of experiencing the severe eye and upper respiratory tract irritation, skin sensitization, dyspnea, nausea, lacrimation, vomiting, and performance decrements that are associated with brief exposures to this substance at the former 8-hour TWA PEL. Furthermore, OSHA considers the effects related to exposure to OCBM to represent material impairments of health.

CYANOGEN

CAS: 460–19–5; Chemical Formula: $(CN)_2$
H.S. No. 1105

OSHA previously had no limit for cyanogen. The Agency proposed a limit of 10 ppm as an 8-hour TWA for this colorless gas, which has a pungent, almond-like odor. NIOSH (Ex. 8–47, Table N1) concurred with this proposal, and the final rule establishes the 10 ppm TWA limit, which is the same as that recommended by the ACGIH (1986/Ex. 1–3).

The acute toxicity for cyanogen in various animal species is high (Flury and Zernick 1931d, as cited in ACGIH 1986/Ex. 1–3, p. 154). One hundred ppm was fatal to cats in two to three hours, and 400 ppm was fatal to rabbits in less than two hours. However, rabbits exposed to 100 ppm for four hours showed practically no effects. Cats exposed to 50 ppm were severely affected but recovered (Flury and Zernik 1931d, as cited in ACGIH 1986/Ex. 1–3, p. 154). Investigations in the rat suggest that cyanogen is approximately 10 times less acutely toxic than is hydrogen cyanide (McNerney and Schrenk 1960/Ex. 1–426).

Human tests showed that subjects experienced almost immediate eye and nasal irritation at exposures of 16 ppm (McNerney and Schrenk 1960/Ex. 1–426).

The New Jersey Department of Public Health (Exs. 144, 144A) urged OSHA to set a limit for cyanogen on the basis of EPA's IRIS data. The use of such an approach is discussed in Section VI.A of the preamble.

In the final rule, OSHA is establishing an 8-hour TWA limit for cyanogen. The Agency concludes that this limit is necessary to protect against the significant risk of irritation and systemic effects associated with exposure at the levels permitted in the absence of any OSHA limit. OSHA considers the irritant and systemic effects caused by exposure to cyanogen to be material impairments of health.

CYANOGEN CHLORIDE

CAS: 506-77-4; Chemical Formula: ClCN
H.S. No. 1106

OSHA previously had no limit for cyanogen chloride; however, a ceiling limit of 0.3 ppm was proposed for this colorless liquid or gas, which has a pungent odor. NIOSH (Ex. 8-47, Table N1) concurred with this proposal. In the final rule, OSHA is establishing a 0.3-ppm ceiling limit, which is the same as that recommended by the ACGIH.

The chronic effects of exposure to cyanogen chloride, which include hoarseness, conjunctivitis, and edema of the eyelid, have long been recognized (Reed 1920/Ex. 1-355). Flury and Zernik (1931d, as cited in ACGIH 1986/Ex. 1-3, p. 155) observed the effects of exposure to cyanogen chloride in five animal species. In mice, a concentration of approximately 500 ppm was fatal within three minutes; in cats, 120 ppm was fatal in 3.5 minutes; 48 ppm was fatal to dogs in six hours; in goats, a 1000-ppm exposure for three minutes caused death after 70 hours; and 1200 ppm was fatal to the rabbit. Several other studies have demonstrated that animals exposed to cyanogen chloride exhibit pulmonary edema and interference with cellular metabolism (Jandorf and Bodansky 1946/Ex. 1-334; Aldridge and Evans 1946/Ex. 1-708).

Human data indicate that 1 ppm is the lowest irritant concentration that can be tolerated for a 10-minute exposure; 2 ppm was intolerable for this time period, and 48 ppm was fatal in 30 minutes (Prentiss 1937/Ex. 1-1164). The Michigan Department of Health (1977, as cited by ACGIH 1986/Ex. 1-3, p. 155) reported that a concentration of about 0.7 ppm caused severe eye and nasal irritation, forcing workers to evacuate the area. NIOSH submitted the only comment received by OSHA on its proposed ceiling limit of 0.3 ppm for cyanogen chloride.

OSHA is establishing this 0.3-ppm ceiling limit for cyanogen chloride in the final rule. The Agency concludes that a ceiling limit is necessary to protect workers from the significant risks of severe irritation, metabolic effects, and pulmonary edema associated with short-term exposures to this substance at levels above the former PEL. The Agency considers the irritant, metabolic, and respiratory effects associated with exposure to cyanogen chloride to be material impairments of health and functional capacity.

DIBUTYL PHOSPHATE

CAS: 107-66-4; Chemical Formula: $(n\text{-}C_4H_9O)_2(OH)PO$
H.S. No. 1119

OSHA previously had an 8-hour TWA PEL of 1 ppm for dibutyl phosphate. The Agency proposed to supplement this limit with a 2-ppm STEL, based on the ACGIH recommendation. NIOSH (Ex. 8-47, Table N1) concurred with this proposal, and the final rule establishes a 1-ppm TWA limit with a 2-ppm STEL for this substance.

There are no published reports of toxic reactions caused by exposure to dibutyl phosphate. However, in a personal communication to the ACGIH, Mastromatteo reported that workers exposed to relatively low levels of dibutyl phosphate developed respiratory tract irritation and headache (Mastromatteo 1964a, as cited in ACGIH 1986/Ex. 1-3, p. 236). No additional data or health effects comment was introduced into the record during the rulemaking proceeding.

OSHA concludes that both a TWA and a STEL are necessary to protect workers from the risk of respiratory tract irritation and headaches reported to occur at low levels of exposure. OSHA judges it likely that, in the absence of a STEL, short-term exposure permitted by the 1-ppm TWA limit alone may be sufficiently high to present a significant risk of respiratory tract irritation and headache to workers; the Agency considers these exposure-related effects to be material impairments of health. Therefore, the Agency is supplementing its 1-ppm 8-hour TWA limit with a 2-ppm 15-minute STEL in the final rule.

1,3-DICHLORO-5,5-DIMETHYL HYDANTOIN

CAS: 118-52-5; Chemical Formula: $C_5H_6Cl_2N_2O_2$
H.S. No. 1122

OSHA previously had a limit of 0.2mg/m^3 TWA for 1,3-dichloro-5,5-dimethyl hydantoin (DCDMH). Based on the ACGIH (1986/Ex. 1-3) recommendation, the Agency proposed a TWA limit of 0.2 mg/m^3 and a STEL of 0.4 mg/m^3 for this white powder, which has a mild odor similar to that of chlorine. NIOSH (Ex. 8-47, Table N1) concurred with OSHA's proposed limits for this substance, and they are established in the final rule.

1,3-Dichloro-5,5-dimethyl hydantoin produces systemic toxicity in laboratory animals. The acute oral LD_{50} in rats of both sexes is 542±84 mg/kg when DCDMH is administered as a 10-percent aqueous suspension. Rats dying within 48 hours of administration showed gastrointestinal hemorrhage at necropsy. The animals tolerated aqueous solutions of DCDMH maintained at 20 ppm available chlorine (Industrial Bio-Test Laboratories 1961 and 1962, as cited in ACGIH 1986/Ex. 1-3, p. 183).

Limited human exposure data have been provided by Baier, who reported that individuals experienced extreme respiratory irritation at an average level of 1.97 mg/m^3, but that some experienced this degree of irritation even at 0.7 mg/m^3 (Baier 1964, as cited in ACGIH 1986/Ex. 1-3, p. 183). Other than the NIOSH submission, OSHA received no comments on its proposal to revise the limit for DCDMH.

The 0.2-mg/m^3 TWA and 0.4-mg/m^3 STEL limits that were proposed are based on evidence of systemic toxicity in laboratory animals and respiratory irritation at low exposure levels in human subjects. The Agency concludes that both a TWA and a STEL are required to protect exposed workers from the risk of respiratory irritation that has been shown to occur at levels only slightly above the level specified by the 8-hour TWA limit. OSHA considers the respiratory irritant effects associated with exposure to DCDMH to represent material impairment of health and functional capacity. OSHA also concludes that the combined TWA-STEL limits will reduce this risk substantially and is therefore establishing a 0.2-mg/m^3 TWA and a 0.4-mg/m^3 STEL for DCDMH.

DICHLOROETHYL ETHER

CAS: 111-44-4; Chemical Formula: $(CH_2ClCH_2)_2O$
H.S. No. 1127

OSHA previously had a 15-ppm ceiling limit, with a skin notation, for dichloroethyl ether. The Agency proposed to revise its limit for dichloroethyl ether to 5 ppm as an 8-hour TWA, with a 10-ppm STEL, and to retain the skin notation. NIOSH (Ex. 8-47, Table N6A; Tr. pp. 3-96 to 3-97) concurred with the proposed limits but indicated that a carcinogen designation should be added to the PEL. The final rule establishes the proposed limits, which are consistent with the ACGIH recommendation. Dichloroethyl ether is a colorless, flammable liquid with a nauseating odor.

The primary health hazards associated with exposure to this substance are irritation of the eyes and respiratory system and pulmonary damage. Schrenk, Patty, and Yant (1933/Ex. 1-665) reported that guinea pigs exposed to the vapor of dichloroethyl ether at 500 ppm experienced immediate and severe eye and nose irritation, respiratory disturbances after 1.5 to 3 hours, and death after five to eight hours. Lung, kidney, liver, and brain damage were also observed in these

animals; exposure to a reduced level of 105 ppm caused eventual death after 10 hours of continuous exposure. A one-hour exposure to 105 ppm caused irritation only (Carpenter, Smyth, and Pozzani 1949/Ex. 1-772). At 35 ppm, for an upspecified duration, irritation but no other adverse effects were observed (Schrenk, Patty, and Yant 1933/Ex. 1-665). Rats responded similarly, with four-hour exposures to 250 ppm proving lethal (Carpenter, Smyth, and Pozzani 1949/Ex. 1-722).

Repeated exposures to 69 ppm (seven hours/day, five days/week for 130 days) caused no serious injury in rats or guinea pigs; only mild stress-related effects were noted (Kosyan 1967/Ex. 1-914). However, other studies of guinea pigs have shown mild primary irritative effects on the skin, and fatalities occurred when 300 mg/kg was applied dermally as a pure liquid for 24 hours (Smyth and Carpenter 1948/Ex. 1-375). Direct contact of dichloroethyl ether with the eye causes moderate pain, conjunctival irritation, and transient corneal injury (Carpenter and Smyth 1946/Ex. 1-859). A sufficient amount of dichloroethyl ether can be absored through the skin to be lethal: Sax and Lewis (*Dangerous Properties of Industrial Materials,* 7th ed., 1989) report the dermal LD_{50} in rabbits as 720 mg/kg. Mice have been reported to develop hepatomas after prolonged oral administration (80 weeks) of dichloroethyl ether at 300 mg/kg (Innes, Ulland, Valerio et al. 1969/Ex. 1-270).

Humans exposed briefly to dichloroethyl ether at concentrations above 550 ppm experienced intolerable eye and nasal irritation, with coughing, nausea, and retching. Concentrations between 100 and 260 ppm were irritating but tolerable; however, the odor of dichloroethyl ether was still nauseating at 35 ppm (Schrenk, Patty, and Yant 1933/Ex. 1-665). Eye irritation has been reported from industrial exposure to a concentration of dichloroethyl ether at 2.5 ppm (Bell and Jones 1958/Ex. 1-714). A single fatality, presumably from inhalation of the vapor, has been reported but not documented (Elkins 1959c, as cited in ACGIH 1986/Ex. 1-3, p. 186). NIOSH submitted the only comments on OSHA's proposed revision of the PEL for dichloroethyl ether.

In the final rule, OSHA is establishing a 5 ppm TWA and 10 ppm STEL for this substance. The Agency concludes that a 5-ppm TWA and a 10-ppm STEL will protect workers against the significant risk of irritation, lung injury, and nausea associated with occupational exposure to elevated levels of dichloroethyl ether, and these limits are established in the final rule. OSHA considers the eye and nasal irritation, lung injury, and other symptoms associated with exposure to dichloroethyl ether to be material impairments of health and functional capacity. The skin notation is retained because dichloroethyl ether can cause systemic toxicity if percutaneously absorbed.

2,2-DICHLOROPROPIONIC ACID

CAS: 75-99-0; Chemical Formula: CH_3CCl_2COOH

H.S. No. 1130

OSHA previously had no limit for 2,2-dichloropropionic acid; however, the Agency proposed a 1-ppm 8-hour TWA limit for this liquid, based on the ACGIH (1986/Ex. 1-3) recommendation. NIOSH (Ex. 8-47, Table N1) concurred with the proposed 1-ppm TWA limit, and the final rule establishes it.

In a communication to the ACGIH, the Dow Chemical Company (1977b, as cited in ACGIH 1986/Ex. 1-3, p. 190) reported that 2,2-dichloropropionic acid is corrosive to the skin and can cause permanent injury to the eye. The oral LD_{50} in rats is between 0.7 and 1 g/kg. Seven-hour exposures to a saturated atmosphere of the acid vapor caused no ill effects in rats, and a 120-day study of dietary exposure in rats showed a no-effect level of 15 mg/kg/day (Dow Chemical Company 1977b, as cited in ACGIH 1986/Ex. 1-3, p. 190). Dr. Grace Ziem, an independent occupational physician (Ex. 46), commented that Dow's material safety data sheet on 2,2-dichloropropionic acid reports that the liver and kidneys are target organs in rats fed higher dietary levels.

Acute human exposures have been reported to cause mild to moderate skin, eye, respiratory, and gastrointestinal irritation. Minimal respiratory irritation was observed in workers exposed at concentrations of between 2 and 7 ppm (ACGIH 1986/Ex. 1-3, p. 190).

The Agency concludes that a 1 ppm TWA limit for 2,2-dichloropropionic acid will protect workers from the significant risk of eye, respiratory, and gastrointestinal irritation, and possible liver or kidney injury, at exposure levels permitted in the absence of any OSHA limit. The Agency considers the irritant and adverse organ effects associated with exposure to this substance to be material impairments of health and functional capacity. Therefore, OSHA is establishing a 1-ppm 8-hour TWA limit for 2,2-dichloropropionic acid.

DIETHYLAMINE

CAS: 109-89-7; Chemical Formula: $(C_2H_5)_2NH$

H.S. No. 1137

OSHA's previous limit for diethylamine was 25 ppm as an 8-hour TWA. The Agency proposed to lower this limit to an 8-hour TWA of 10 ppm and to add a 15-minute STEL of 25 ppm, based on the ACGIH (1986/Ex. 1-3) recommendation. NIOSH (Ex. 8-47, Table N1) concurred with these proposed limits, which are established in the final rule. Diethylamine is a colorless liquid with an ammonia-like odor.

Diethylamine is a strong irritant of the eyes, skin, and mucous membranes, and chronic sublethal exposures cause tracheitis, bronchitis, pneumonitis, and pulmonary edema (ACGIH 1986/Ex. 1-3, p. 197). In rabbits, the dermal LD_{50} is 0.82 ml/kg, and instillation of solutions of 1 percent or greater into the eyes of rabbits caused corneal opacity (Sutton 1963/Ex. 1-1101). Direct contact of the skin with diethylamine causes necrosis (ACGIH 1986/Ex. 1-3, p. 197). Rabbits exposed seven hours/day, five days/week for six weeks to 50 or 100 ppm diethylamine survived; those exposed to 50 ppm showed marked lung and corneal irritation, and, occasionally, degeneration of the heart muscle (Brieger and Hodes 1951/Ex. 1-408). In the animals exposed to 100 ppm, these changes were more severe, and the parenchymatous degeneration of the heart muscle was marked (Brieger and Hodes 1951/Ex. 1-408).

OSHA finds that its previous limit of 25 ppm as an 8-hour TWA is only one-half the level found to cause marked lung and corneal irritation in animals exposed for six weeks. The Agency concludes that the 25-ppm limit is not sufficient to protect workers from the significant risk of skin burns, corneal injury, pulmonary irritation, and skin, eye, and upper respiratory tract irritation potentially associated with more prolonged exposures to this substance. OSHA considers the exposure-related effects of diethylamine on the eyes, skin, and respiratory tract to be material impairments of health. To afford workers greater protection from these adverse effects, OSHA is revising its limit for diethylamine to 10 ppm as an 8-hour TWA and 25 ppm as a 15-minute STEL; these limits are established in the final rule.

DIISOBUTYL KETONE

CAS: 108-83-8; Chemical Formula: $[(CH_3)_2CHCH_2]_2CO$

H.S. No. 1140

OSHA previously had an 8-hour limit of 50 ppm TWA for diisobutyl ketone. The Agency proposed to reduce this limit to 25 ppm TWA, based on both the ACGIH and NIOSH recommendations. NIOSH (Ex. 8-47, Table N1) concurred with this proposal, and the final rule

revises OSHA's limit for diisobutyl ketone to 25 ppm as an 8-hour time-weighted average.

The primary health effects associated with exposure to diisobutyl ketone are eye, nose, and throat irritation, although experimental animals have shown some systemic effects. Diisobutyl ketone has a uniformly low acute toxicity by all routes of exposure. Rats and guinea pigs survived single exposures of from 7.5 to 16 hours to essentially saturated vapor (McOmie and Anderson 1949/Ex. 1-918). Smyth, Carpenter, and Weil (1949/Ex. 1-528) reported that five of six rats died after exposure to 2000 ppm for eight hours; these investigators also reported a percutaneous LD_{50} for rabbits of greater than 20 ml/kg. Direct application of diisobutyl ketone to rabbit skin was only mildly irritating, and no eye irritation was reported after instillation of this substance into the rabbit eye. The oral toxicity for the rat was reported as 5.8 g/kg (Smyth, Carpenter, and Weil 1949/Ex. 1-528). Carpenter and Smyth (1946/Ex. 1-859) reported a no-effect level for diisobutyl ketone of 125 ppm in rats and guinea pigs given 30 seven-hour exposures. At 250 ppm, the liver and kidney weights of female rats increased, and the liver weights of male guinea pigs decreased; at levels of 530 and 920 ppm, rats showed increased liver and kidney weights; and at 1650 ppm, increased mortality was noted (Carpenter and Smyth 1946/Ex. 1-859).

Silverman, Schulte, and First (1946/Ex. 1-142) reported eye irritation and complaints of objectionable odor in volunteer human exposures to concentrations above 25 ppm. No worker illnesses have been linked to diisobutyl ketone exposure (ACGIH 1986/Ex. 1-3, p. 203).

NIOSH (Ex. 150, Comments on Diisobutyl Ketone) concurred with OSHA's proposal to reduce the limit for diisobutyl ketone and reported that there are no new toxicological data beyond those described above; no other comments on this substance were received. The Agency concludes that the previous 50-ppm TWA limit is inadequate to protect workers against the significant risk of irritation associated with workplace exposures to diisobutyl ketone levels greater than 25 ppm. The Agency has determined that the irritation associated with exposure to diisobutyl ketone constitutes a material impairment of health and functional capacity. Therefore, OSHA is revising its limit for diisobutyl ketone to 25 ppm as an 8-hour TWA.

EPICHLOROHYDRIN

CAS: 106-89-8; Chemical Formula: C_3H_5ClO
H.S. No. 1158

OSHA previously had a limit of 5 ppm TWA, with a skin notation, for epichlorohydrin. OSHA proposed to reduce this limit to 2 ppm TWA, also with a skin notation, based on the ACGIH (1986/Ex. 1-3) recommendation, and the final rule establishes an 8-hour TWA limit of 2 ppm and retains the skin designation. Epichlorohydrin is an unstable liquid with an odor like that of chloroform.

In animals, epichlorohydrin is irritating and systemically toxic by all routes of exposure (Shell Chemical Corporation 1958, as cited in ACGIH 1986/Ex. 1-3, p. 233). Fatalities are caused by central nervous system and respiratory tract effects resulting from exposure to high concentrations.

In mice, single 30-minute exposures to 8300 ppm of epichlorohydrin vapor caused muscular paralysis and death from respiratory failure; similar results have been reported for dermal application of the liquid at 0.5 ml/kg in rats, and repeated oral administration at 0.1 mg/kg in mice (Shell Chemical Corporation 1958, as cited in ACGIH 1986/Ex. 1-3, p. 233). At 32 ppm (seven hours/day, five days/week) for 91 days, rats failed to show normal weight gain, and at 16 ppm they showed increased kidney size (ACGIH 1986/Ex. 1-3, p. 233). Gage (1959/Ex. 1-1052) confirmed these findings and demonstrated lung, liver, and kidney injury in rats from repeated six-hour exposures at concentrations ranging from 17 to 120 ppm. No effects were observed by this author at 9 ppm. The oral LD_{50} in rats is reported as 260 mg/kg, and the dermal LD_{50} in rabbits is reported as 755 mg/kg (Lawrence, Malik, Turner, and Autian 1972/Ex. 1-1058). A four-hour exposure at a level of 250 ppm was fatal to rats (Carpenter, Smyth, and Pozzani 1949/Ex. 1-722).

NIOSH (Ex. 8-47, Table N6B) did not concur with OSHA's proposed limit for epichlorohydrin, and considers this substance a potential human carcinogen and a likely candidate for a 6(b) rulemaking. There have been reports of carcinogenicity in mice resulting from both dermal application and subcutaneous injection of epichlorohydrin (Van Duuren, Goldschmidt, Katz et al. 1974/Ex. 1-969), as well as indications of reproductive effects resulting from ingestion; in addition, mutagenic effects have been observed in microbial systems and in the fruit fly (NIOSH 1976c/Ex. 1-972).

In humans exposed to concentrations above 100 ppm for brief periods, lung edema and kidney lesions have been reported (NIOSH 1976c/Ex. 1-972). Exposure at 20 ppm caused burning of eyes and nasal mucosa (Wexler 1971, as cited in NIOSH 1976c/Ex. 1-972). Another exposure to an unknown concentration caused eye and throat irritation, nausea, dyspnea, bronchitis, and an enlarged liver (Schultz 1964/Ex. 1-1064). Painful irritation of subcutaneous tissues follows skin contact in humans (ACGIH 1986/Ex. 1-3. p. 233). The New Jersey Department of Public Health (Exs. 144, 144A) urged OSHA to establish a PEL for epichlorohydrin on the basis of EPA's IRIS data. The use of such an approach is discussed in Section VI.A of the preamble.

OSHA is establishing an 8-hour TWA limit of 2 ppm, with a skin notation, for epichlorohydrin. The Agency concludes that this limit will protect workers from the significant risk of dermal, respiratory, liver, and kidney effects that are potentially associated with exposure to epichlorohydrin at elevated concentrations. OSHA has determined that the respiratory, liver, kidney, and dermal effects associated with exposure to epichlorohydrin represent material impairments of health. The skin notation is retained because of this substance's capacity to penetrate the skin and cause toxicity; according to Lawrence, Malik, Turner, and Autian 1972/Ex. 1-1058, the dermal LD_{50} of epichlorohydrin in rabbits is 755 mg/kg.

ETHYL BENZENE

CAS: 100-41-4; Chemical Formula C_8H_{10}
H.S. No. 1162

OSHA's former limit for ethyl benzene was 100 ppm as an 8-hour TWA. Based on the skin and mucous membrane irritant properties associated with exposure to ethyl benzene, OSHA proposed permissible exposure limits for this substance of 100 ppm as an 8-hour TWA and 125 ppm as a 15-minute STEL. NIOSH (Ex. 8-47, Table N1) concurred with this proposal. The final rule establishes limits of 100 ppm TWA and 125 ppm STEL for ethyl benzene; these limits are consistent with the ACGIH recommendation. Ethyl benzene is a colorless, flammable liquid with an aromatic odor.

The Agency's decision to add a STEL to the existing time-weighted average limit reflects evidence that transient eye irritation occurs in humans at vapor concentrations of 200 ppm; the short-term limit is necessary to protect exposed workers from the risk of such irritation as a result of even brief excursions above the 100-ppm level.

Written comments submitted by ARCO Chemical Company (ACC) (Ex. 3-638) include a detailed discussion of ethyl benzene's toxicity in animals, as reported in several recent studies (ECETOC 1986; Dynamac Corporation 1986) and in a personal communication from the National Toxicology Program's Chemical Manager for Ethyl Benzene. The findings of these investigators

include: Moderate dermal irritation on intact and abraded rabbit skin after a 24-hour application; mild conjunctival irritation (without corneal effects) from direct instillation of undiluted ethyl benzene in rabbit eyes; erythema and edema with superficial necrosis, resulting in exfoliation of large patches of skin, following repeated and prolonged application of the undiluted material to rabbit skin; "a slight, cloudy swelling of hepatocytes" in animals subchronically exposed to the vapor as a "result of an increase in the endoplasmic reticulum (SER), which is an adaptive process responsible for increased microsomal enzyme activity and, presumably, increased metabolism of ethyl benzene"; congestion of the lungs, nasal mucosa, liver, and kidneys in mice and rats exposed six hours/day for four consecutive days to ethyl benzene concentrations of 2360 ppm and in mice exposed to 1190 ppm; and lacrimation and salivation in rats exposed at 400 and 800 ppm for six hours/day, five days/week (ECETOC 1986 and Dynamac Corporation 1986, both as cited in Ex. 3-638). ACC stressed the fact that, except at very high concentrations, significant systemic toxicity does not appear to be a manifestation of ethyl benzene exposure.

In addition to providing the results of these up-to-date studies on the health effects in animals of ethyl benzene exposure, the ACC indicated its support for both the retention of the current 100-ppm TWA limit and the adoption of a 125-ppm 15-minute STEL for ethyl benzene. Both concentrations, according to the ACC, "provide a wide safety margin for eye irritation compared to the concentration which can be tolerated in the workplace (1000 ppm)."

The New Jersey Department of Health (Exs. 144, 144A) urged OSHA to set a PEL for ethyl benzene on the basis of EPA's IRIS data. The use of such an approach is discussed in Section VI.A of the preamble.

OSHA concludes that workers exposed to concentrations of ethyl benzene above the 100-ppm level, even briefly, are at significant risk of experiencing irritation; the Agency considers this to be a material impairment of health. Accordingly, the Agency is establishing a short-term limit of 125 ppm for a 15-minute period to supplement the existing 100-ppm time-weighted-average limit for ethyl benzene.

ETHYL ETHER

CAS: 60-29-7; Chemical Formula: $C_2H_5OC_2H_5$
H.S. No. 1164

OSHA's previous limit for ethyl ether was a 400-ppm TWA. The Agency proposed the same time-weighted-average TWA limit, with the addition of a 15-minute STEL of 500 ppm. These limits are established in the final rule and are consistent with those recommended by the ACGIH. Ethyl ether is a colorless, volatile, mobile liquid with a distinct odor and a burning, sweet taste. It is extremely flammable and is a severe fire and explosion hazard when exposed to heat or flame.

Ethyl ether causes narcosis and general anesthesia. Concentrations of 3.6 to 6.5 volumes percent in air are anesthetic to humans; 7- to 10-percent concentrations cause respiratory arrest, and concentrations greater than 10 percent are fatal (ACGIH 1986/Ex. 1-3, p. 259). Repeated workplace exposures deliberately induced to produce the so-called "ether jag" have caused narcosis, exhaustion, headache, dizziness, sleepiness, excitation, and other psychic disturbances (Hake and Rowe 1963a/Ex. 1-1152). In women, albuminuria and polycythemia may result (Browning 1965a/Ex. 1-1017). Repeated exposure may cause skin desiccation; irritation of the mucous membranes and eyes occurs on contact with the liquid or after exposure to high concentrations of the vapor (Hake and Rowe 1963a/Ex. 1-1152). Nelson and co-workers (1943/Ex. 1-66) reported that workers began to experience nasal irritation at 200 ppm (Nelson, Enge, Ross et al. 1943/Ex. 1-66). Henderson and Haggard (1943c, as cited in ACGIH 1986/Ex. 1-3, p. 259) calculated that the amount of ether absorbed by a man of average height at a concentration of 400 ppm would not cause intoxication. Armor (1950, as cited in ACGIH 1986/Ex. 1-3, p. 259) observed that exposure effects occur only at levels of 500 ppm and above.

NIOSH (Ex. 8-47, Table N2; Tr. pp. 3-86 and 3-89) did not concur with OSHA's proposed limits and noted that some individuals may experience sensory irritation upon exposure to these levels, as evidenced by the Nelson, Enge, Ross et al. (1943/Ex. 1-66) study. However, this finding was not supported by Armor (1950, as cited in ACGIH 1986/Ex. 1-3, p. 259). OSHA received no other comments on its proposed limits. The Agency concludes that both of these limits are necessary to protect exposed workers against the significant risk of narcosis and irritation potentially associated with excursions above the 8-hour TWA level, and OSHA is establishing PELs of 400 ppm as an 8-hour TWA and 500 ppm as a 15-minute STEL for ethyl ether in today's rule. The Agency has determined that irritation and narcosis caused by excessive exposure to ethyl ether constitute material impairments of health and functional capacity.

ETHYL MERCAPTAN

CAS: 75-08-1; Chemical Formula: C_2H_5SH
H.S. No. 1165

OSHA previously had a ceiling limit of 10 ppm for ethyl mercaptan. An 8-hour TWA limit of 0.5 ppm was proposed for this substance, based on the ACGIH (1986/Ex. 1-3) recommendation; NIOSH (Ex. 8-47, Table N1) concurred with OSHA's proposal. The final rule establishes a PEL of 0.5 ppm as an 8-hour TWA for ethyl mercaptan. Ethyl mercaptan is a colorless liquid with a persistent and penetrating leek-like odor.

Acute animal toxicity data concerning ethyl mercaptan are taken from a single study that reports the following findings. The 4-hour inhalation LC_{50} values in rats and mice are 2770 ppm and 4420 ppm, respectively. In the rat, the intraperitoneal LD_{50} is reported to be approximately 450 mg/kg. One drop applied to rabbit eyes caused only slight irritation, but high concentrations of vapor caused considerable irritation within 15 minutes. Maximal sublethal intraperitoneal doses have been reported to induce deep sedation, with higher exposures causing restlessness, muscular incoordination, skeletal muscular paralysis, cyanosis, respiratory depression, coma, and death. Although inhalation tests showed no noteworthy pathology in rats, intraperitoneal injection caused lymphatic infiltration of liver with occasional necrosis (Fairchild and Stokinger 1958/Ex. 1-415). In chronic inhalation studies of rabbits, rats, and mice, a five-month exposure to 40 ppm caused minimal cardiovascular and other systemic effects (Blinova 1965/Ex. 1-603).

Studies of human volunteers, exposed at 4 ppm for three hours daily for 5 to 10 days, have reported adverse effects. At this level, all subjects experienced altered taste and olfactory reactions, periodic nausea, mucous membrane irritation, and fatigue. Exposure to 0.4 ppm produced no unpleasant symptoms (ACGIH 1986/Ex. 1-3, p. 262).

The Workers Institute for Safety and Health (WISH) (Ex. 116) was critical of OSHA's proposal to establish an 8-hour TWA limit rather than a STEL or ceiling for ethyl mercaptan. OSHA believes that the health evidence on ethyl mercaptan shows that a 0.5 ppm TWA limit will be sufficient to reduce the adverse acute effects associated with exposure to this

substance; for example, a 3-hour exposure to 4 ppm, which caused adverse acute effects in human volunteers (ACGIH 1986/Ex. 1-3, p. 262), would exceed 0.5 ppm as an 8-hour TWA. The health evidence discussed above demonstrates that, at the previous PEL of 10 ppm (ceiling), employees were at risk of nausea, fatigue, and irritation; these effects have been demonstrated to occur on exposure to 4-ppm concentrations of this substance for just a few days. OSHA considers these exposure-related effects of nausea, fatigue, and irritation to be material impairments of health. The Agency concludes that the revised limit of 0.5 ppm will substantially reduce this significant risk. Therefore, OSHA is lowering its limit for ethyl mercaptan to 0.5 ppm as an 8-hour TWA.

ETHYLENE GLYCOL

CAS: 107-21-1; Chemical Forumla: CH_2OHCH_2OH
H.S. No. 1169

OSHA previously had no limit for ethylene glycol and proposed a ceiling limit of 50 ppm (approximately 125 mg/m^3) for this clear, colorless, odorless, hygroscopic liquid. The final rule establishes a limit of 50 ppm as a ceiling, which is consistent with the limit recommended by the ACGIH for ethylene glycol. Ethylene glycol poses virtually no exposure risk at room temperature because of its low vapor pressure; at elevated temperatures, however, exposures are possible and adverse effects have been reported as a result of exposure to mists.

In studies of rats, guinea pigs, rabbits, dogs, and monkeys, Coon and colleagues (1970/Ex. 1-84) reported that animals exposed over a 30-day period to concentrations of 10 or 57 mg/m^3 for eight hours daily, five days per week, showed no adverse effects. Moderate to severe eye irritation did occur in rats and rabbits exposed at 12 mg/m^3 for 24 hours per day for 90 days (Coon, Jones, Jenkins, and Siegel 1970/Ex. 1-84). Wiley and co-workers (1936/Ex. 1-600) reported no ill effects in animals exposed to approximately 350 to 400 mg/m^3, eight hours per day, for 16 weeks (Wiley, Hueper, and von Oettingen 1936/Ex. 1-600).

Rowe (1963/Ex. 1-865) concluded that daily exposure to 100 ppm of the vapor did not cause systemic or eye injuries, although Troisi (1949/Ex. 1-598) described nystagmus in overexposed workers (concentrations not reported). In a human inhalation study, Wills and colleagues (1974/Ex. 1-582) reported that volunteers exposed to the aerosol from 20 to 22 hours per day for four weeks, at an average concentration of 12 ppm, complained of throat irritation, mild headache, and lower back pain. Complaints were more pronounced when the concentration was raised to 140 mg/m^3 (50 ppm) for part of a day. Average concentrations of 80 ppm were found intolerable by the subjects, who reported a burning sensation in the throat and respiratory passages; irritation was also common at 60 ppm (Wills, Coulston, Harris et al. 1974/Ex. 1-582). Based primarily on this study, NIOSH (Ex. 8-47, p. 6; Tr. p. 3-86) suggested that OSHA reconsider its proposed 50-ppm ceiling limit; however, NIOSH acknowledged that the exposure concentrations used by Wills et al. (1974/Ex. 1-582) were "significantly erratic." NIOSH also described recent evidence that ethylene glycol may be a potential teratogen. OSHA will continue to monitor the toxicologic literature on this substance to evaluate ethylene glycol's potential teratogenicity.

Gary L. Melampy, counsel for the Independent Lubricant Manufacturers Association (ILMA) (Ex. 3-830), commented that OSHA should apply the 50-ppm ceiling limit only to those workplaces where ethylene glycol is used at elevated temperatures. In the final rule, OSHA has not restricted the application of any new or revised PEL to a particular industry segment or industrial process. OSHA recognizes that industrial processes vary in characteristics that affect the degree of risk to which workers are exposed; these characteristics include the amount of material processed or handled, the frequency with which a substance is present, the extent to which a process is open or closed, and the temperatures and pressures at which materials are used. OSHA's policy, which is reflected in all of its previous health standards, has been to base its permissible exposure limits on scientific evidence that exposure to a substance at a given concentration or dose is associated with a health risk and that promulgating a PEL will reduce that risk. Thus, a relationship between exposure level and degree of risk is established and is deemed applicable in all situations where a substance is present. If the characteristics of a process are such that employee exposure to a substance is nonexistent or is well below the levels associated with a health risk, the promulgation of a limit on employee exposure will have little or no effect on the operation or process and imposes no additional burden on the employer. Therefore, in the specific case of ethylene glycol, OSHA sees no reason to limit application of the 50-ppm ceiling limit to those processes where exposure to airborne ethylene glycol is most likely.

Based on evidence of an occupational risk of severe throat and respiratory irritation associated with exposure to the vapor and mist, OSHA is promulgating a ceiling limit of 50 ppm for ethylene glycol; this level is just below the level at which clinical symptoms have been noted in humans. OSHA considers these symptoms, which include throat and respiratory irritation and headache, to be material impairments of health. The Agency concludes that this limit will substantially reduce the significant risk associated with exposures to higher levels that would be permitted in the absence of a PEL.

ETHYLIDENE NORBORNENE

CAS: 16219-75-3; Chemical Formula: C_9H_{12}
H.S. No. 1171

OSHA had no previous limit for ethylidene norbornene. The Agency proposed a ceiling limit of 5 ppm, based on the ACGIH recommendation, and is establishing this limit in the final rule. NIOSH (Ex. 8-47, Table N1) agreed with the selection of this limit. Ethylidene norbornene is a colorless liquid which reacts with oxygen.

In a range-finding study, five of six rats died following a 4-hour exposure to 4000 ppm 5-ethylidene-2-norbornene (Smyth, Carpenter, Weil et al. 1969/Ex. 1-442). Other studies of longer duration have reported that exposures to 237 ppm for seven hours per day, five days per week, for 88 days resulted in death for 21 of 24 rats. No deaths resulted from repeated exposures at 90 ppm, but renal lesions and enlarged livers were observed; liver lesions, testicular atrophy, and hydrothorax occurred only at the 237-ppm level (Kinkead, Pozzani, Geary, and Carpenter 1971/Ex. 1-606). Beagle dogs similarly exposed to 93 ppm for 89 days survived but exhibited such effects as testicular atrophy, hepatic lesions, and slight blood changes. Less pronounced effects were seen after exposures to 61 ppm, and no effects were seen at 22 ppm (Kinkead, Pozzani, Geary, and Carpenter 1971/Ex. 1-606).

Human volunteers exposed for 30 minutes to ethylidene norbornene concentrations of 11 ppm experienced eye and nose irritation; at 6 ppm, transient eye irritation occurred (ACGIH 1986/Ex. 1-3, p. 261). Other than the comment submitted by NIOSH, OSHA receive no comments on its proposal to establish a ceiling limit of 5 ppm for ethylidene norbornene.

In the final rule, OSHA is establishing a 5-ppm ceiling for this substance. The Agency finds that this limit is necessary

to minimize the risk of irritation that has been documented to occur in occupational exposures to concentrations as low as 6 ppm for 30-minute periods. OSHA has determined that the eye and nasal irritation associated with exposure to ethylidene norbornene constitute material impairments of health. The Agency concludes that this limit will reduce this risk substantially.

FURFURAL

CAS: 98-01-0; Chemical Formula: $C_5H_4O_2$
H.S. No. 1183

OSHA previous exposure limit for furfural was an 8-hour TWA limit of 5 ppm, with a skin notation. The Agency proposed reducing this limit to 2 ppm TWA and retaining the skin notation, based on the ACGIH recommendation; these limits are established in the final rule. Furfural is a colorless, oily liquid that turns rust-colored when exposed to air and light.

An inhalation exposure to 260 ppm of furfural was fatal to rats but not to mice or rabbits. A four-week exposure of dogs to 130 ppm for six hours a day caused liver damage, but no adverse effects were observed at 63 ppm (AIHA 1965, as cited in ACGIH 1986/Ex. 1-3, p. 280).

Bugyi and Lepoid (1949/Ex. 1-1077) described numbness of the tongue and oral mucosa, absence of a sence of taste, and labored breathing in workers exposed to furfural (at unspecified levels) in a poorly ventilated facility. Korenman and Resnik (1930, as cited in ACGIH 1986/Ex. 1-3, p. 280) stated that inhalations of from 1.9 to 14 ppm furfural caused headaches, itching throat, and eye irritation; Kuhn (1944/Ex. 1-883) reported that exposure to furfural damages the eyesight in some individuals. NIOSH (1975e/Ex. 1-1183) described widespread eye and respiratory tract irritation in workers at a grinding wheel plant exposed to furfural vapor at levels ranging from 5 to 16 ppm. NIOSH (Ex. 8-47, Table N2; Tr. p. 3-86) did not concur with the proposed limit on the basis of these findings and, in addition, urged the Agency to follow up on a recent NTP assay with regard to a possible carcinogenic response in animals exposed to furfural. OSHA notes that Dunlop and Peters (1953/Ex. 1-1189) report that a 15-year study of furfural use in the synthetic resin industry revealed that this substance is not hazardous to employee health in facilities that are adequately ventilated, and that only occasional individual sensitivity was found. The Agency will carefully monitor the results of the NTP Study, currently in peer review, as well as any other scientific evidence pertaining to the health effects of furfural. NIOSH was the only commenter on this substance in the rulemaking record.

After reviewing the evidence above, OSHA concludes that its former 5-ppm limit is not sufficient to protect workers from eye and respiratory tract irritation; this is evidenced by the NIOSH study (1975e/Ex. 1-1183), in which widespread irritation was reported to occur among workers esposed to 5 to 16 ppm. OSHA considers the eye and respiratory tract irritation caused by exposure to furfural to be material impairment of health. Therefore, to protect workers from eye and respiratory tract irritation, OSHA is revising its limit for furfural to 2 ppm as an 8-hour TWA; this limit is established in today's rule. OSHA is also retaining its skin notation; Sax and Lewis *Dangerous Properties of Industrial Materials*, 7th ed., 1989) reported the dermal LD_{50} in rabbits to be 620 mg/kg, indicating that furfural penetrates the skin and can cause systemic effects.

FURFURAL ALCOHOL

CAS: 98-00-0; Chemical Formula: $C_6H_6O_2$
H.S. No. 1184

OSHA's previous limit for furfuryl alcohol was 50 ppm as an 8-hour TWA. In the NPRM, OSHA proposed revising its limit to 10 ppm as an 8-hour TWA and 15 ppm as a 15-minute STEL, and adding a skin notation, based on the ACGIH recommendation. NIOSH (Ex. 8-47, Table N1) concurred with this proposal, and these limits are established in the final rule. Furfuryl alcohol is a colorless liquid which turns red or brown on exposure to light and air.

The bases for the proposed OSHA limits, which were derived from ACGIH-recommended limits, are two foundry studies in which furfuryl alcohol was released during core preparation. Apol (1973/Ex. 1-1180) reported no discomfort among workers exposed to 10.8 ppm furfuryl alcohol, but severe lacrimation occurred at 15.8 ppm. Formaldehyde was also present at a concentration of 0.33 ppm. Burton and Rivera (1972/Ex. 1-944) found no irritation, headache, or dizziness among workers exposed to 8-hour TWA concentrations of 5 and 6 ppm, with excursions up to 16 ppm.

In its criteria document, NIOSH (1979a/Ex. 1-236) also reviewed these studies but concluded that it was unknown whether the lacrimation reported by Apol (1973/Ex. 1-1180) was caused by furfuryl alcohol, formaldehyde, or both combined. NIOSH also noted that the current OSHA limit (50 ppm) is five times lower than the concentration reported to cause no adverse effects in monkeys (Woods and Seevers 1954-1956, as cited in NIOSH 1979a/Ex. 1-236). At the time, NIOSH (1979a/Ex. 1-236) recommended that the 50-ppm limit should remain, since no information existed that showed that this limit offered inadequate protection.

Mr. H.K. Thompson, Corporate Industrial Hygiene Manager of Caterpillar, Inc. (Ex. 3-349), commented that formaldehyde probably contributed more than furfuryl alcohol to the lacrimation observed by Apol (1973/Ex. 1-1180). He also agreed that the 50-ppm PEL was too high, since his personal experience has indicated that eye irritation occurs between 25 and 30 ppm furfuryl alcohol. Mr. Thompson recommended that OSHA revise its limit to 25 ppm TWA and add a 50 ppm STEL.

In its final rule for formaldehyde, OSHA analyzed extensively the dose-response data on formaldehyde's irritant effects. In that analysis, OSHA concluded that severe irritation and lacrimation occur in most individuals when the formaldehyde levels reach 3 ppm or above; at levels between 0.1 and 0.5 ppm, slight eye irritation may occur in some individuals (52 FR 46235). In the foundry study by Apol (1973/Ex. 1-1180), formaldehyde was present at a concentration of 0.33 ppm, about 10 times below the level associated with severe eye irritation. Therefore, OSHA believes that exposure to furfuryl alcohol levels of about 16 ppm was most likely the cause of the lacrimation reported by Apol (1973/Ex. 1-1180).

NIOSH (Ex. 150, Comments on Furfuryl Alcohol) concurred with OSHA's proposal to revise the limits for this substance to 10-ppm TWA and 15-ppm STEL. In its posthearing submission, NIOSH cited a study by Cockcroft et al. (1980, as cited in Ex. 150), who reported that a 50-year-old moldmaker developed asthma after working with a mixture containing furfuryl alcohol, paraformaldehyde, xylene, and a catalyst containing sulfuric acid, phosphoric acid, and butyl alcohol. The patient's bronchial response to inhaled histamines was two to three times more severe following exposure to furfuryl alcohol mixed with butyl alcohol.

OSHA finds that the additional evidence submitted by NIOSH further justifies the proposed limits. This evidence indicates that exposure to furfuryl alcohol may potentiate asthmatic responses that are suggestive of an allergic or hypersensitive condition. Individuals that are so affected frequently respond adversely to exposure levels below those that affect

most other persons, and the asthmatic response is much more severe than that of respiratory tract irritation.

Therefore, OSHA concludes that the Apol (1973/Ex. 1–1180) study shows that severe eye irritation is associated with exposure to about 16 ppm furfuryl alcohol, and that furfuryl alcohol is capable of inducing more serious asthmatic responses in at least some workers. OSHA has determined that the severe eye irritation and asthma caused by exposure to furfuryl alcohol represent material impairments of health and functional capacity. The Agency is establishing PELs for this substance of 10 ppm as an 8-hour TWA and 15 ppm as a 15-minute STEL, with a skin notation, to reduce these significant risks among exposed employees. The skin notation is added to alert employers that excessive exposure may result from dermal contact; according to Proctor, Hughes, and Fischman (1988, p. 263), furfuryl alcohol is readily absorbed through the skin of animals in sufficient quantity to be lethal.

GLUTARALDEHYDE

CAS: 111–30–8; Chemical Formula: OCH $(CH_2)_3CHO$
H.S. No. 1187

OSHA previously had no limit for glutaraldehyde and proposed establishing a ceiling limit of 0.2 ppm, based on the ACGIH (1986/Ex. 1–3) recommendation. NIOSH (Ex. 8–47, Table N1) concurred with this proposal, and the final rule establishes this limit. Glutaraldehyde is an aliphatic dialdehyde that forms colorless crystals.

Glutaraldehyde is strongly irritating to the nose, eyes, and skin (*Human Sensory Irritation Threshold of Glutaraldehyde Vapor* 1976, as cited in ACGIH 1986/Ex. 1–3, p. 285) and can cause allergic contact dermatitis from occasional or incidental occupational exposure (Jordan, Dahl, and Albert 1972/Ex. 1–1056). The rat oral LD_{50} has been variously reported as 250, 820, and 2380 mg/kg (Stonehill, Krop, and Borick 1963/Ex. 1–1066; Smyth 1963 and NIOSH 1975f, both as cited in ACGIH 1986/Ex. 1–3, p. 285). The dermal LD_{50} in the rabbit is 2560 mg/kg, and the 4-hour inhalation LD_{50} in the rat is 5000 ppm (NIOSH 1975f, as cited in ACGIH 1986/Ex. 1–3, p. 285).

Mice exposed to alkalinized glutaraldehyde at 8 and 33 ppm for 24 hours have shown marked nervous behavior with panting and compulsive washing of the face and limbs; those exposed to 33 ppm exhibited signs of toxic hepatitis at autopsy (Varpela, Otterstrom, and Hackman 1971/Ex. 1–1072).

In a study of a cold-sterilizing operation in which the operator was exposed for 12 minutes to an activated 2-percent aqueous solution, a measurement of 0.38 ppm glutaraldehyde was taken in the operator's breathing zone; the operator and the investigators experienced severe eye, nose, and throat irritation as well as sudden headache at the end of this procedure (Schneider and Blejer 1973, as cited in ACGIH 1986/Ex. 1–3, p. 285). Another study employing very precise methods of airborne concentration measurement reported the irritation response level for glutaraldehyde to be 0.3 ppm and the odor recognition threshold to be 0.04 ppm (Colwell 1976, as cited in ACGIH 1986/Ex. 1–3, p. 285).

Other than the NIOSH submission, OSHA received no comments on its proposal to establish a ceiling level of 0.2 ppm for glutaraldehyde. The Agency finds that the human evidence cited above clearly demonstrates a significant risk of irritation to the eyes nose, and throat associated with short-term exposures to glutaraldehyde at concentrations of 0.3 ppm or above. OSHA considers the irritation effects associated with exposure to glutaraldehyde to be material impairments of health. Therefore, OSHA is establishing a 0.2 ppm ceiling limit for this substance in the final rule.

HEXACHLOROCYCLOPENTADIENE

CAS: 77–47–4; Chemical Formula: C_5Cl_6
H.S. No. 1196

No previous OSHA limit existed for hexachlorocyclopentadiene. The Agency proposed to establish a 0.01–ppm 8-hour TWA limit for this substances, based on the ACGIH (1986/Ex. 1–3) recommendation; NIOSH (Ex. 8–47, Table N1) concurred with this proposal, and the final rule adds this limit to the Z table. Hexachlorocyclopentadiene is a yellow to amber-colored, nonflammable liquid with a pungent odor.

Hexachlorocyclopentadiene has a high order of acute toxicity in laboratory animals. Rabbits, mice, rats, and guinea pigs died from inhaling 89.5 percent of the vapor in air (Treon, Cleveland, and Cappel 1955/Ex. 1–497). In 150 daily exposures of seven hours each, rabbits, rats, and guinea pigs survived concentrations of 0.15 ppm, but a similar exposure was fatal to four of five mice. At approximately twice this concentration, mice, rats, and most rabbits died by or before the 25th exposure, but guinea pigs survived 30 exposures. The hexachlorocyclopentadiene vapors caused tearing, labored respiration, and, at high concentrations, tremors. Treon and associates (1955/Ex. 1–497) observed degenerative changes in the brain, heart, liver, adrenal glands, and kidneys, and pulmonary irritation occurred in all species, even at the lowest concentration of 0.15 ppm. At higher concentrations, pulmonary edema, hyperemia, necrotizing bronchitis, and bronchiolitis were observed (Treon, Cleveland, and Cappel 1955/Ex. 1–497).

In humans, there are few data concerning hexachlorocyclopentadiene's toxicity. Irritation is known to occur, but the intolerable odor and eye irritation associated with exposure to this substance have discouraged prolonged exposures (McGilvray 1971, as cited in ACGIH 1986/Ex. 1–3, p. 300).

The New Jersey Department of Public Health (Exs. 144, 144A) urged OSHA to establish a PEL for hexachlorocyclopentadiene on the basis of EPA's IRIS data. The use of this approach is discussed in section VI.A of the preamble.

The proposed TWA PEL of 0.01 ppm for this severly toxic substance is about 10 times below the level associated with systemic damage and pulmonary irritation in experimental animals. In the absence of any limit on exposure, OSHA finds that employees are at significant risk of intense eye and pulmonary irritation and multiple organ damage; the Agency considers these effects to be material impairments of health and functional capacity. To substantially reduce these risks, OSHA is establishing an 8-hour TWA limit of 0.01 ppm for hexachlorocyclopentadiene.

HEXYLENE GLYCOL

CAS: 107–41–5; Chemical Formula: $(CH_3)_2$-$COHCH_2$-$CHOH$-CH_3
H.S. No. 1204

OSHA previously had no limit for hexylene glycol. Based on the ACGIH recommendation, OSHA proposed a ceiling limit of 25 ppm for this liquid, which has a mild, sweetish odor. NIOSH (Ex. 8–47, Table N1) concurred with this proposed limit, and the final rule establishes it.

In mice, the LD_{50} for hexylene glycol is reported to be 3.8 ml/kg, and it is reported to be 4.79 g/kg in rats. A single dose of 2.0 ml/kg induced hypnosis in mice. Undiluted hexylene glycol instilled into the rabbit eye caused irritation and corneal injury (Smyth and Carpenter 1948/Ex. 1–375).

The Shell Chemical Corporation has reported that oral administration of hexylene glycol can cause nervous system depression that is manifested by an initial state of excitation, followed by deep depression (Shell Chemical

Corporation, as cited in ACGIH 1986/Ex. 1-3, p. 309). When the liquid is applied to the skin, mild to moderate irritation occurs, although skin absorption does not. At high concentrations, hexylene glycol vapors evoke a strong sensory response: a five-minute exposure at 1000 ppm produced eye irritation and throat and respiratory discomfort. At concentrations of 50 ppm for 15 minutes, slight eye irritation was reported (ACGIH 1986/Ex. 1-3, p. 309).

Mr. Melampy, Counsel to the ILMA, commented that the proposed 25-ppm ceiling limit "is far below the hazard levels found to exist . . . ," given that exposures to hexylene glycol concentrations of 50 ppm for brief periods of time cause only slight eye irritation. OSHA does not agree with the assessment that a 25-ppm ceiling limit is too low. As discussed earlier in this section, OSHA has determined that no employee should be subjected to mucous membrane or respiratory irritation caused by exposure to toxic agents and that this effect represents material impairment of health and adversely affects the well-being and functional capacity of employees. For hexylene glycol, 50 ppm represents an adverse-effect level, and establishing the limit at this level would not be sufficiently protective. OSHA also concludes that 25 ppm is a reasonable level at which to establish the PEL; this level provides some margin against this substance's irritant effects. Therefore, OSHA finds that establishing a 25-ppm ceiling limit for hexylene glycol is necessary to reduce the risks of eye and respiratory irritation, which occur at exposure levels above the new PEL.

HYDROGEN BROMIDE

CAS: 10035-10-6; Chemical Formula: HBr
H.S. No. 1206

The previous OSHA PEL for hydrogen bromide was 3 ppm as an 8-hour TWA. The Agency proposed revising this limit to 3 ppm as a ceiling limit not to be exceeded at any time during the working day; NIOSH (Ex. 8-47, Table N1) concurred with this proposal. In the final rule, OSHA is establishing this ceiling limit, which conforms to the recommendation made by the ACGIH. Hydrogen bromide (HBr) is a colorless, corrosive, nonflammable gas with an acrid odor.

Animal studies have demonstrated that hydrogen bromide has a considerably higher acute toxicity than hydrogen chloride (HCl) in mice and a somewhat higher acute toxicity than this chemical in rats (NIOSH 1977i/Ex. 1-1182). In mice, the LC_{50} is 800 ppm HBr in air for 60 minutes (and 2500 ppm HCl in air for 30 minutes); in rats, the LC_{50} is 2800 ppm HBr in air for 60 minutes (and 5000 ppm HCl in air for 30 minutes).

The chief toxic effect of hydrogen bromide in humans is primary irritation of the nose and throat. Irritation begins within several minutes at levels of between 3 and 6 ppm. At 2 ppm, the odor of HBr is detectable, but no irritation is experienced (Connecticut State Department of Health 1955, as cited in ACGIH 1986/Ex. 1-3, p. 312). No chronic effects have been associated with exposure to hydrogen bromide. No comments, other than NIOSH's, were received on this substance.

OSHA finds that, under its previous 3-ppm TWA limit, workers were at significant risk of experiencing irritant effects due to short-term exposures to levels of hydrogen bromide exceeding 3 ppm. The Agency considers the irritant effects of exposure to hydrogen bromide to be material impairments of health. Therefore, OSHA is establishing a 3-ppm ceiling limit for this substance in the final rule to limit short-term exposures to hydrogen bromide and reduce this risk.

HYDROGEN FLUORIDE

CAS: 7664-39-3; Chemical Formula: HF
H.S. No. 1208

The previous OSHA standard for hydrogen fluoride was 3 ppm as an 8-hour TWA. OSHA proposed supplementing its 3-ppm TWA with a 15-minute STEL of 6 ppm. These limits are established in the final rule and are the same as those recommended by NIOSH (1976f, as cited in ACGIH 1986/Ex.1-3, p. 315). In its posthearing comments, NIOSH (Ex. 150, Comments on Hydrogen Fluoride) concurred with OSHA's proposed limits for hydrogen fluoride. The ACGIH (1986/Ex. 1-3) recommends a 3 ppm TLV-ceiling for hydrogen fluoride. Hydrogen fluoride is a fuming, colorless liquid; at temperatures above 19 °C (66 °F), it becomes a colorless gas.

Guinea pigs and rabbits survived 40-ppm hydrogen fluoride concentrations for 41 hours, but exposure to 300 ppm for two hours or more was fatal (Machle, Thamann, Kitzmiller, and Cholak 1934/Ex. 1-519). Animals exposed to 3 ppm hydrogen fluoride for 30 days showed no adverse effects (Ronzani 1909, as cited in ACGIH 1986/Ex. 1-3, p. 315). Stokinger (1949a, as cited in ACGIH 1986/Ex. 1-3, p. 315) reported that animals repeatedly exposed to 7 ppm on a daily basis exhibited mild respiratory tract irritation. One study by Largent (1961/Ex. 1-1158) demonstrated kidney, liver, and lung damage in laboratory animals repeatedly exposed to 17 ppm hydrogen fluoride. At 8.6 ppm, the pathologic changes seen in exposed animals were minor, except for lung damage in one dog (Largent 1961/Ex. 1-1158).

In studies with humans, Largent (1960/Ex. 1-516; 1961/Ex. 1-1158) reported that volunteers exposed repeatedly to concentrations of hydrogen fluoride as high as 4.7 ppm for six hours/day for 10 to 50 days experienced irritation and burning of the eyes and nose, in addition to reddening of the skin, at concentrations above 3 ppm. Industrial experience has shown that direct contact of the skin with hydrogen fluoride results in severe burns that may have a delayed onset but later develop into ulcers that eventually scar (Stokinger 1981b/Ex. 1-1127). A report by Eagers (1969, as cited in Stokinger 1981b, above) described several industrial accidents in which workers died in a matter of hours after accidental splashing from ruptured containers of hydrogen fluoride (the cause of death was respiratory failure and cardiac arrest). Kleinfeld (1965/Ex. 1-514) reported a fatal case of hydrogen fluoride poisoning that caused death from pulmonary edema.

NIOSH (1976f, as cited in ACGIH 1986/Ex. 1-3, p. 315), in its criteria document, cites numerous studies that consistently show that long-term occupational exposures to hydrogen fluoride lead to fluorosis in workers. The NIOSH limit is based in part on a study by Derryberry, Bartholomew, and Fleming (1963/Ex. 1-506) showing that the threshold limit for minimal increases in bone density caused by fluoride (fluorosis) is below 4.3 ppm of hydrogen fluoride. The limits proposed by OSHA are the current NIOSH-recommended limits for this substance, and NIOSH's concurrence statement was the only comment received in the record.

Because of hydrogen fluoride's potential to cause respiratory irritation, OSHA finds that a STEL is necessary to reduce the risk associated with elevated, short-term exposures, which would be permitted under the 3 ppm TWA limit alone. The Agency has determined that the irritation caused by exposure to hydrogen fluoride constitutes a material impairment of health. Therefore, OSHA is revising the limits for hydrogen fluoride to 3 ppm as an 8-hour TWA and 6 ppm as a 15-minute STEL; these limits are established in the final rule.

2-HYDROXYPROPYL ACRYLATE

CAS: 999-61-1; Chemical Formula: $CH_2CHCOOCH_2CHOHCH_3$
H.S. No. 1211

OSHA previously had no limit for 2-hydroxypropyl acrylate. A limit of 0.5

ppm as an 8-hour TWA, with a skin notation, was proposed, based on the ACGIH recommendation. NIOSH (Ex. 8–47, Table N1) concurred with the proposal, and this limit is established in the final rule. 2-Hydroxypropyl acrylate (HPA) is a colorless liquid at room temperature.

In experimental animals, 2-hydroxypropyl acrylate has a high acute toxicity. The Dow Chemical Company (1977c, as cited in ACGIH 1986/Ex. 1–3, p. 320) has reported an oral LD_{50} for the rat of 0.25 to 0.5 g/kg, and a dermal LD_{50} for the rabbit approximately 0.25 mg/kg. In guinea pigs, direct contact with HPA caused severe eye burns and skin corrosion and sensitized some of the experimental animals. Rats exposed to a concentration of 650 ppm HPA in air for seven hours survived. Longer-term inhalation studies (30 days for two hours/day, six days/week) in rats, dogs, rabbits, and mice resulted in some irritation at 5 ppm (Dow Chemical Company 1977c, as cited in ACGIH 1986/Ex. 1–3, p. 320).

OSHA received no comment (other than NIOSH's) on its proposed 0.5-ppm TWA limit and skin notation for this substance. The Agency finds that this limit is necessary to protect workers from the risks of irritant effects, skin and eye burns, and sensitization effects associated with exposure to 2-hydroxypropyl acrylate; OSHA considers these effects material impairments of health. Therefore, OSHA is promulgating a TWA limit of 0.5 ppm, which is below the effect level for irritation found in experimental animals. OSHA is also adding a skin notation to the limit because 2-hydroxypropyl acetate readily penetrates the skin to cause systemic effects; the dermal LD_{50} in rabbits has been reported to be 0.25 mg/kg (ACGIH 1986/Ex. 1–3).

IRON SALTS (SOLUBLE)

CAS: Varies with compound; Chemical Formula: Varies with compound
H.S. No. 1217

OSHA previously had no limit for the soluble iron salts and proposed establishing the ACGIH-recommended limit of 1 mg/m^3, measured as iron, for these substances. NIOSH (Ex. 8–47, Table N1) concurred with OSHA's proposed limit for the soluble salts of iron, and the final rule establishes an 8-hour TWA PEL of 1 mg/m^3.

When injected into the bloodstream of experimental animals, iron salts (especially the ferric salts) are highly toxic (ACGIH 1986/Ex. 1–3, p. 328). The acute intravenous dose of ferric chloride that is lethal to rabbits is about 7.2 mg/kg (Drinker, Warren, and Page 1935/Ex. 1–315). The ACGIH (1986/Ex. 1–3, p. 328) considers the salts to be irritants to the respiratory tract when inhaled as dusts and mists. Stewart and Faulds (1934/Ex. 1–764) described the ferric salts as skin irritants. The oral toxicities of iron salts are considered to be moderate to low, although marked gastrointestinal irritation results from ingestion (U.S. Department of Labor 1941, as cited in ACGIH 1986/Ex. 1–3, p. 328); 30 grams is the estimated fatal dose for humans (Smyth 1956/Ex. 1–759).

NIOSH was the only commenter on OSHA's proposed 8-hour TWA PEL of 1 mg/m^3, measured as iron, for the soluble salts of iron. The Agency concludes that, in the absence of any limit, employees are at risk of skin and mucous membrane irritation associated with exposure to high concentrations of these salts. OSHA considers these effects to be material impairments of health and deems this risk to be significant. Therefore, OSHA is establishing a 1 mg/m^3 8-hour TWA PEL for the soluble iron salts.

ISOPROPYL ACETATE

CAS: 108–21–4; Chemical Formula: $CH_3COOCH(CH_3)_2$
H.S. No. 1224

OSHA previously had a 250-ppm 8-hour TWA limit for isopropyl acetate. The Agency proposed supplementing this limit with a 15-minute STEL of 310 ppm, based on the ACGIH (1986/Ex. 1–3) recommendation. OSHA is establishing these limits for this substance in the final rule. Isopropyl acetate is a colorless liquid and has a fruity odor.

The oral LD_{50} for rats is reported to be 6.75 g/kg; five of six rats died after a four-hour exposure to 32,000 ppm, and one of six rats died after a four-hour exposure to 16,000 ppm (Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1–400).

The primary problems in occupational exposures to isopropyl acetate are eye and mucous membrane irritation. In humans, exposure to 200 ppm isopropyl acetate for 15 minutes caused eye irritation, with nose and throat irritation occurring at higher concentrations (Silverman, Schulte, and First 1946/Ex. 1–142). NIOSH (Ex. 8–47, Table N2) notes that the majority of subjects exposed to 200 ppm in the Silverman, Schulte, and First (1946/Ex. 1–142) study experienced eye irritation and that the authors of this study recommended an 8-hour TWA of 100 ppm to prevent sensory irritation. OSHA agrees with NIOSH that this substance presents a hazard at elevated short-term levels and has accordingly added a STEL to ensure that worker exposures are maintained under good industrial hygiene control.

OSHA concludes that, in the absence of a short-term limit on exposure, the 250-ppm TWA limit alone will not protect employees from experiencing the irritant effects associated with elevated short-term exposures to isporopyl acetate. OSHA has determined that the irritant effects related to exposure to isopropyl acetate are material impairments of health. Therefore, to reduce the risk of irritation among exposed employees, the Agency is establishing a 250-ppm 8-hour TWA limit and a 310 ppm STEL for this substance.

ISOPROPYL ALCOHOL

CAS: 67–63–0; Chemical Formula: $CH_3CHOHCH_3$
H.S. No. 1225

The previous PEL for isopropyl alcohol was 400 as an 8-hour TWA. OSHA proposed adding a 15-minute STEL of 500 ppm to this TWA, based on the ACGIH recommendation, and the final rule establishes these limits. In its posthearing comment, NIOSH (Ex. 150, Comments on Isopropyl Alcohol) endorsed OSHA's proposal, stating that a STEL is necessary to reduce the risks of irritation and narcosis that can occur on short-term exposure to elevated concentrations of isopropyl alcohol. Isopropyl alcohol is a colorless, flammable liquid with a slight odor resembling that of rubbing alcohol.

Rats exposed at isopropyl alcohol concentrations of 12,000 ppm for four hours survived, but extending the duration of exposure to eight hours killed the animals (Smyth 1937–1955, as cited in ACGIH 1986/Ex. 1–3, p. 337).

Isopropyl alcohol has been demonstrated to be irritating to the eyes, nose, and throat in humans exposed for brief periods to 400 ppm (Nelson, Enge, Ross et al. 1943/Ex. 1–66); at 800 ppm, these symptoms were more intense. In addition, isopropyl alcohol has narcotic and irritative acute effects at higher concentrations. Weil and associates have reported that an excess of paranasal sinus cancers has been observed among workers manufacturing isopropyl alcohol (Weil, Smith, and Nale 1952/Ex. 1–453). However, it has been established that the cancers associated with isopropyl alcohol manufacture were caused by isopropyl oil and not by the isopropyl alcohol itself (NIOSH 1976g, as cited in ACGIH 1986/Ex. 1–3, p. 337).

No comments, other than NIOSH's, were received on this substance. The irritant effects associated with exposure to isopropyl alcohol occur at concentrations only twice as high as the 8-hour TWA limit, even when the

exposure lasts only for a brief period; exposures at this level clearly cause irritation, as documented by the study by Nelson et al. (1943/Ex. 1–66).

OSHA concludes that, in the absence of a STEL, workers are at significant risk of experiencing the narcotic and irritative effects associated with short-term exposures to isopropyl alcohol above the 8-hour TWA PEL of 400 ppm. Therefore, the Agency is retaining its 400 ppm 8-hour TWA limit for isopropyl alcohol and adding a 500 ppm 15-minute STEL to substantially reduce this significant risk. OSHA has determined that the narcosis and eye and mucous membrane irritation associated with chronic and acute exposures to isopropyl alcohol constitute material impairments of health and that a STEL is needed to protect workers from experiencing these harmful effects.

n-ISOPROPYLAMINE

CAS: 75–31–0; Chemical Formula: $(CHG53)_2CHNH_2$
H.S. No. 1228

OSHA's previous limit for n-isopropylamine was 5 ppm as an 8-hour TWA. The Agency proposed retaining this TWA limit and adding a 10-ppm 15-minute STEL, based on the ACGIH recommendation. NIOSH (Ex. 8–47, Table N1; Tr. p. 3–86) concurred with this proposal, and these limits are established in the final rule. This substance is a flammable, volatile, colorless liquid that has an odor similar to that of ammonia.

The most serious effect of n-isopropylamine in laboratory animals is respiratory tract irritation, which can be severe enough to cause lung edema (Smyth 1956/Ex. 1–759). Rats survived a four-hour inhalation at 4000 ppm, but an 8000-ppm exposure resulted in fatalities (Smyth, Carpenter, and Weil 1951/Ex. 1–439). Proctor and Hughes (1978/Ex. 1–1136) have reported that the odor of n-isopropylamine becomes strong and unpleasant at the 10- to 20-ppm level; nose and throat irritation is experienced even as a result of brief exposures.

Except for NIOSH, no rulemaking participants commented on OSHA's proposal to issue a 5-ppm TWA and 15-minute STEL of 10 ppm for this substance. The Agency concludes that both a TWA and STEL are required to protect exposed workers from the significant risk of upper respiratory tract irritation that is known to occur even at brief excursions above the 8-hour PEL. The Agency considers upper respiratory tract irritation resulting from exposure to this substance to be a material impairment of health. Therefore, OSHA is revising the PEL for n-isopropylamine to 5 ppm as an 8-hour TWA and 10 ppm as a 15-minute STEL; these limits are established in the final rule.

MESITYL OXIDE

CAS: 141–79–7; Chemical Formula: $(CH_3)_2C{=}CHCOCH_3$
H.S. No. 1243

OSHA's previous limit for mesityl oxide was 25 ppm as an 8-hour TWA. The Agency proposed revising this limit to 15 ppm as an 8-hour TWA and 25 ppm as a 15-minute STEL, based on the ACGIH (1986/Ex. 1–3) recommendation. NIOSH has a 10-ppm REL for mesityl oxide. The final rule establishes a 15-ppm 8-hour TWA and a 25-ppm 15-minute STEL for mesityl oxide, which is an oily, colorless liquid with a peppermint odor.

Silverman, Schulte, and First (1946/Ex. 1–142) found that a majority of test subjects experienced eye irritation on exposure to 25 ppm mesityl oxide and nasal irritation at 50 ppm. A toxicity data sheet published by the Shell Chemical Corporation (1957, as cited in ACGIH 1986/Ex. 1–3, p. 361) confirms 25 ppm as the maximum comfort level. Smyth, Seaton, and Fischer (1942/Ex. 1–378) reported liver and kidney damage among rats and guinea pigs exposed to 100 ppm mesityl oxide for six weeks; no adverse effects were reported for animals exposed to 50 ppm. After reviewing these data, the ACGIH (1986/Ex. 1–3, p. 361) concluded that the former TLV of 25 ppm should be reduced to 15 ppm TWA and 25 ppm as a 15-minute STEL because of the greater systemic toxicity of mesityl oxide compared with that of other saturated ketones. NIOSH (1978f, as cited in ACGIH 1986/Ex. 1–3, p. 361), relying on the same data, recommended a limit of 10 ppm as a 10-hour TWA.

Studies indicate that eye irritation occurs following brief exposure to 25 ppm of mesityl oxide, and nasal irritation is experienced at the 50-ppm level. Animal studies show liver and kidney damage in experimental animals exposed to 100 ppm. NIOSH's comment (Ex. 8–47, Table N2; Tr. p. 3–86) was the only one received by the Agency on its proposal to revise the limits for mesityl oxide. NIOSH based its lower recommended limit on a belief that the eye irritation caused by exposure to mesityl oxide might be more severe than the irritation caused by exposure to the other ketones because mesityl oxide has a higher molecular weight than the lower ketones. OSHA is not persuaded by this argument because the evidence that brief exposure to 25 ppm mesityl oxide causes eye irritation is based on actual human exposures to mesityl oxide at that level; that is, NIOSH's argument would be reasonable if the 25 ppm short-term limit were being established by analogy to the effects of another (lower-molecular-weight) ketone.

After reviewing the health evidence for this substance, OSHA finds that the proposed 15-ppm TWA and 25-ppm STEL limits are protective against both the acute and chronic effects demonstrated to be caused by exposure to this substance. In the final rule, OSHA concludes that a TWA PEL of 15 ppm and a STEL of 25 ppm are necessary to protect employees both from the possible liver and kidney damage associated with chronic exposures and the eye irritation resulting from elevated short-term exposures to mesityl oxide. The Agency considers both the systemic and the irritant effects of exposure to mesityl oxide material impairments of health and functional capacity. To reduce these risks, OSHA is establishing limits for mesityl oxide of 15 ppm as an 8-hour TWA and 25 ppm as a 15-minute STEL.

METHYL 2-CYANOACRYLATE

CAS: 137–05–3; Chemical Formula: $CH_2{=}C(C{=}N)COOCH_3$
H.S. No. 1248

No previous limit existed for methyl 2-cyanoacrylate. OSHA proposed establishing a limit of 2 ppm as an 8-hour TWA and 4 ppm as a STEL, based on the ACGIH recommendation, and the final rule establishes these limits. NIOSH (Ex. 8–47, Table N1) concurred with the selection of these limits. Methyl 2-cyanoacrylate is a colorless, viscous liquid.

In a personal communication to the ACGIH TLV Committee in 1985, Eastman Kodak reported on the toxicity of methyl 2-cyanoacrylate in experimental animals. The oral LD_{50} in rats is reported to be 1.6 to 3.2 g/kg, and the dermal LD_{50} in guinea pigs is 10 ml/kg. The adverse effects reported in laboratory animals are slight irritation of the skin and corneal damage. An inhalation LC_{50} of 101 ppm has been reported in rats exposed for six hours to methyl 2-cyanoacrylate. Repeated exposures (six hours/day for five days/week) to 31.3 ppm for a total of 12 exposures caused only a slight decrease in the rate of weight gain in rats and no nasal or tracheal lesions or systemic toxicity. No changes were observed in rats similarly exposed to 3.1 ppm (Eastman Kodak 1985, as cited in ACGIH 1986/Ex. 1–3, p. 383).

In a simulated workbench exposure, McGee and co-workers reported nasal irritation in humans at 3 ppm and eye irritation at 5 ppm (McGee, Oglesby, Raleigh, and Fassett 1968/Ex. 1–424).

There are no reports of occupational poisonings. No comments, other than NIOSH's, were received on OSHA's proposed PELs for this substance.

The report by McGee et al. (1968/Ex. 1–424) clearly establishes that employees are at risk of nasal irritation on exposure to 3 ppm or above and of eye irritation at 5 ppm or above. The Agency has determined that these adverse effects constitute material impairment of health and should be avoided in the workplace. Therefore, to substantially reduce these significant risks, OSHA is establishing a 2-ppm 8-hour TWA limit and a 4-ppm STEL for methyl 2-cyanoacrylate in the final rule.

METHYL ISOBUTYL CARBINOL

CAS: 108–11–2; Chemical Formula: $CH_3CHOHCH_2CH(CH_3)_2$
H.S. No. 1261

OSHA previously had an 8-hour TWA limit of 25 ppm, with a skin notation, for methyl isobutyl carbinol. OSHA proposed supplementing these limits with a STEL of 40 ppm, based on the ACGIH (1986/Ex. 1–3) recommended limits, and NIOSH (Ex. 8–47, Table N1) concurred with this proposal. The final rule establishes a TWA limit of 25 ppm and a STEL of 40 ppm for this substance, with a skin notation. Methyl isobutyl carbinol is a colorless, stable liquid.

In rabbits, a 24-hour skin applicatioin of 3.56 ml/kg (2.9 g/kg) was lethal to half the animals (Smyth, Carpenter, and Weil 1951/Ex. 1–439). Rats exposed by inhalation to 2000 ppm of methyl isobutyl carbinol vapor died, and the same authors report that the oral LD_{50} for rats is 2.6 g/kg (Smyth, Carpenter, and Weil 1951/Ex. 1–439).

Humans volunteers exposed to methyl isobutyl carbinol reported eye irritation upon 15-minutes' exposure to 50 ppm (Silverman, Schulte, and First 1946/Ex. 1–142). Other than NIOSH's, OSHA received no comments regarding the basis for its proposed limits for methyl isobutyl carbinol.

In view of the finding that exposure to 50 ppm can result in eye irritation in as little as 15 minutes, OSHA has determined that a risk of eye irritation exists in the absence of a limit on short-term exposure. The Agency considers the eye irritation caused by exposure to this substance to be a material impairment of health. Therefore, to reduce this risk, OSHA is establishing a 15-minute STEL of 40 ppm, while retaining the 25-ppm 8-hour TWA PEL and skin notation for this substance.

METHYL MERCAPTAN

CAS: 74–93–1; Chemical Formula: CH_3SH
H.S. No. 1263

OSHA previously had a ceiling limit of 10 ppm for methyl mercaptan. Based on the ACGIH recommendation, the Agency proposed revising this limit to an 8-hour TWA of 0.5 ppm, and OSHA is establishing this limit in the final rule. Methyl mercaptan is a flammable, water-soluble gas with a disagreeable odor like that of rotten cabbage.

Methyl mercaptan acts on the respiratory center, producing death by respiratory paralysis. DeRekowski (1893, as cited in ACGIH 1986/Ex. 1–3, p. 405) and Frankel (1927/Ex. 1–1033) have reported that the acute toxicity of methyl mercaptan is similar to but somewhat lower than that of hydrogen sulfide; however, Ljunggren and Norberg (1943/Ex. 1–916) have concluded that the two substances exhibit toxicities of the same magnitude. Pulmonary edema results from exposures to lower, less acute concentrations of methyl mercaptan (Fairchild, personal communication, as cited in ACGIH 1986/Ex. 1–3, p. 405).

Inhalation of (an unspecified concentration of) methyl mercaptan produced coma and death in one worker; acute hemolytic anemia and methemoglobinemia developed after this exposure (Schultz, Fountain, and Lynch 1970, as cited in ACGIH 1986/Ex. 1–3, p. 405). A 1918 report by Pickler (as cited by E.E. Sandmeyer in Clayton and Clayton 1981) describes the accidental exposure (for several hours) of 28 students to a concentration of methyl mercaptan estimated at 4 ppm. The individuals had headache and nausea, and one student showed some liver involvement, demonstrated by the appearance of epithelial cells, protein, and erythrocytes, in the excretion fluid. This condition subsided in six weeks (Sandmeyer 1981).

John L. Festa, Director of Chemical Control and Health Programs at the American Paper Institute, Inc. (Ex. 3–685) objected to OSHA's proposal for methyl mercaptan, stating that the basis for the ACGIH TLV, from which the OSHA proposal was derived, was not to reduce irritant effects but to limit odor intensity. He commented further that regulating substances on the basis of "unpleasant sensory stimuli . . . embarks upon a new precedent" (Ex. 3–685, p. 3). Mr. Festa reported that the odor of methyl mercaptan is relatively faint at 0.05 ppm, although the substance may be mildly irritating over long periods of exposure to concentrations of 4 to 5 ppm.

OSHA does not agree with the suggestion made by Mr. Festa that the effects associated with exposure to methyl mercaptan below 10 ppm (the previous OSHA limit) do not warrant attention. First, Mr. Festa acknowledges that prolonged exposure to 4 to 5 ppm causes irritation; as discussed earlier in this section, OSHA has determined that sensory irritation constitutes material impairment of health. Furthermore, a single inhalation exposure to 7.9 ppm has been reported to result in nauseating odor (NIOSH 1978b, as cited in ACGIH 1986/Ex. 1–3 p. 405); clearly, this effect adversely affects the performance and functional capacity of employees. OSHA is also concerned about the possible liver effects that were reported from a single exposure to approximately 4 ppm methyl mercaptan (Pickler 1918, as cited by E.E. Sandmeyer in Clayton and Clayton 1981). Although this report is dated, OSHA has found no evidence that comprehensive studies have been undertaken in humans to examine the potential for liver or other organ damage as a result of long-term exposure to low levels of methyl mercaptan. Liver and other organ defects have been reported to occur in animals exposed to 50 ppm for only 90 days. Because of these considerations, OSHA concludes that a significant risk of acute sensory effects, as well as possible organ damage, exists at the former 10-ppm ceiling, and that a 0.5-ppm limit is necessary to ensure that these significant risks are adequately reduced. NIOSH (Ex. 8–47, Table N7) recommends a ceiling limit at the same 0.5 ppm level. OSHA is revising its limit for methyl mercaptan to 0.5 ppm as an 18-hour TWA, and this limit is promulgated in today's rule.

METHYL n-AMYL KETONE

CAS: 110–43–0; Chemical Formula: $CH_3COC_5H_{11}$
H.S. No. 1264

The current OSHA limit for methyl n-amyl ketone is 100 ppm TWA. OSHA did not propose a revision to its current limit of 100 ppm, and this limit is being retained in the final rule. NIOSH (Ex. 150) agreed that the 100-ppm PEL was sufficiently protective.

Johnson et al. (1978/Ex. 1–335) found no neurologic impairment in rats and monkeys exposed to 131 ppm or 1025 ppm methyl n-amyl ketone for nine months. No gross or histopathologic changes were found (Johnson, Setzer, Lewis, and Hornung 1978/Ex. 1–335). Because of the absence of any human data indicating the concentration of methyl n-amyl ketone that produces sensory irritation, ACGIH (1986/Ex. 1–3, p. 374) believed it prudent to reduce the TLV–TWA from 100 ppm to 50 ppm. NIOSH (1978f, as cited in ACGIH 1986/Ex. 1–3, p. 374) concluded that there was no basis for revising the 100-ppm OSHA limit, since the evidence showed methyl

n-amyl ketone's irritant effects to be equivalent to those of 2-pentanone, which had a recommended limit of 150 ppm.

No neurological or histopathological effects were observed at 131 ppm. The ACGIH's 50-ppm TLV applies an additional factor of safety to this no-observed-effect level, while the NIOSH REL is based on a judgment that such a reduction is unnecessary. In the NPRM, OSHA requested additional information on the health effects of methyl n-amyl ketone; however, no information was received into the record.

OSHA notes that the current 100-ppm PEL is well below the highest level (1025 ppm) reported to be associated with any adverse effects. Because histopathological examination was conducted on the organs of the rats and monkeys tested, OSHA is confident that the existing 100-ppm limit is not likely to be associated with adverse affects and that further reducing this limit would not result in a substantial reduction in risk. Therefore, OSHA is not revising its 100-ppm TWA limit for methyl n-amyl ketone at this time.

alpha-METHYL STYRENE

CAS: 98-83-9; Chemical Formula: $C_6H_5C(CH_3)=CH_2$
H.S. No. 1267

OSHA previously had a ceiling limit of 100 ppm for alpha-methyl styrene. The Agency proposed revising this limit to 50 ppm TWA with a STEL of 100 ppm, and NIOSH (Ex. 8-47, Table N1) concurred with OSHA's proposed limits for this substance, which are established in the final rule. alpha-Methyl styrene is a polymerizable, colorless liquid.

OSHA's former ceiling limit of 100 ppm is based on data developed in 1955 by the Dow Chemical Company (as cited in ACGIH 1986/Ex. 1-3, p. 410) and by Wolf, Rowe, McCollister et al. (1956/Ex. 1-404). These data demonstrated that seven-hour-per-day, five-day-per-week exposures to 200 ppm alpha-methyl styrene for six months produced no ill effects in rats, guinea pigs, rabbits, or monkeys.

In humans, however, these authors reported that a two-minute exposure to 200 ppm caused eye irritation and complaints about this substance's unpleasant odor. OSHA received no comments, other than NIOSH's, on its proposal to revise the limit for alpha-methyl styrene.

Therefore, to ensure that workers are protected against the acute irritant effects of this substance, OSHA is establishing a 50-ppm 8-hour TWA limit and a 100-ppm 15-minute STEL in the final rule. The Agency concludes that these combined limits will substantially reduce the exposure-related risk of irritation and odor effects, which together constitute material impairments of health.

o-METHYLCYCLOHEXANONE

CAS: 583-60-8; Chemical Formula: $CH_3C_5H_9CO$
H.S. No. 1270

OSHA's former limit for o-methylcyclohexanone was 100 ppm as an 8-hour TWA, with a skin notation. The Agency proposed revising this limit to 50 ppm as a TWA and 75 ppm as a STEL, and to retain the skin notation; NIOSH (Ex. 8-47, Table N1) concurred with this proposal. These limits are established in the final rule and are consistent with the limits recommended by the ACGIH. ortho-Methylcyclohexanone is a somewhat viscous liquid with an acetone-like odor.

o-Methylcyclohexanone has both irritative and narcotic effects at relatively low concentrations. The commercial product contains a mixture of isomers; however, toxicity data describe the effects of the ortho isomer only. Gross (as cited in Lehman and Flury 1943a/Ex. 1-962) reported that 450 ppm had irritative effects on the eyes and respiratory systems of rabbits, and 2500 ppm produced narcotic effects (Gross, as cited in Lehman and Flury 1943a/Ex. 1-962). Treon et al. (1943a/Ex. 1-393) reported the oral LD_{50} to be between 1 and 1.25 g/kg for rabbits. Eye problems were observed at about 500 ppm, but exposure to 182 ppm produced no adverse effects (Treon, Crutchfield, and Kitzmiller 1943a/Ex. 1-393).

Rowe and Wolf (1963, as cited in ACGIH 1986/Ex. 1-3, p. 386) reported that concentrations of 100 ppm had no narcotic effects in humans but could cause irritation. No comments, other than NIOSH's, were received on OSHA's proposal to revise the limit for this substance.

Because a level of 100 ppm may present an effect level for irritation in humans (Rowe and Wolf 1963, as cited in ACGIH 1986/Ex. 1-3, p. 386), OSHA finds that a reduction in its 100-ppm PEL is warranted. The Agency considers the irritation caused by exposure to this substance to be a material impairment of health. Therefore, OSHA is revising its limit for o-methylcyclohexanone to 50 ppm as an 8-hour TWA and 75 ppm as a 15-minute STEL. OSHA is also retaining its skin notation for this substance.

OSMIUM TETROXIDE

CAS: 20816-12-0; Chemical Formula: OsO_4
H.S. No. 1298

OSHA previously had an 8-hour TWA limit of 0.002 mg/m³ for osmium tetroxide. Based on the ACGIH recommendation, OSHA proposed to revise this limit to 0.002 mg/m³ as a TWA and to add a STEL of 0.006 mg/m³; NIOSH (Ex. 8-47, Table N1) concurred with this proposal. The final rule establishes these limits for this substance. Osmium tetroxide is a noncombustible, colorless to pale yellow solid with a disagreeable, chlorine-like odor.

Exposure to osmium tetroxide is known to produce ocular effects and respiratory irritation. In 1933, Brunot (Ex. 1-776) reported that rabbits died from pulmonary edema four days after a 30-minute exposure to osmium tetroxide at 130 mg/m³ or higher. Visual problems (e.g., delayed lacrimation and "halo" effects) were reported by this investigator after a brief exposure to osmium tetroxide at a significantly lower concentration (Brunot 1933/Ex. 1-776). A four-hour LC_{50} value of 40 ppm has been reported in rats and mice (NIOSH 1977i/Ex. 1-1182). Toxic effects to bone marrow have been reported in guinea pigs (Hamilton and Hardy 1974a/Ex. 1-957).

Industrial experience indicates that concentrations in a precious metal refining plant ranged from 0.1 to 0.6 mg/m³; intermittent exposures produced symptoms (sometimes delayed) of lacrimation, vision disturbances, headache, conjunctivitis, and cough (McLaughlin, Milton, and Perry 1946/Ex. 1-749). Complaints of persistent and severe nose and throat irritation have been reported (Hamilton and Hardy 1974a/Ex. 1-957). Fairhall (1949d, as cited in ACGIH 1986/Ex. 1-3, p. 450) reported a human fatality resulting from inhalation exposure to OsO_4. Flury and Zernik (1931i, as cited in ACGIH 1986/Ex. 1-3, p. 450) reported that 0.001 mg/m³ is the highest concentration of osmium tetroxide that can be tolerated for six hours without harmful effects.

Except for NIOSH, no rulemaking participants commented on the proposed addition of a STEL for osmium tetroxide. The study by McLaughlin, Milton, and Perry (1946/Ex. 1-749) used a calibrated, calorimetric procedure, together with well-described case reports, to assess the dose-response relationship. OSHA finds this study superior to the report by Flury and Zernik (1931i, as cited in ACGIH 1986/Ex. 1-3, p. 450), which is more anecdotal. The McLaughlin et al. (1946/Ex. 1-749) study demonstrates serious, acute effects resulting from intermittent and short-term exposure. OSHA concludes that, in the absence of a limit on short-term exposures, the 0.002-mg/m³ 8-hour TWA PEL alone is not sufficient to protect employees from experiencing these effects, which are deemed to constitute material

impairments of health. Therefore, to reduce the risk from short-term elevated exposures to osmium tetroxide, OSHA is establishing a 15-minute STEL of 0.006 mg/m³ to supplement the 0.002-mg/m³ TWA limit.

PARAFFIN WAX FUME

CAS: 8002-74-2; Chemical Formula: C_nH_{2n+2}; H.S. No. 1302

OSHA previously had no limit for paraffin wax fume and proposed establishing an 8-hour TWA limit of 2 mg/m³; NIOSH (Ex. 8-47, Table N1) concurred with this proposal. The final rule establishes this limit, which is the same as the limit recommended by the ACGIH. Paraffin is a white or slightly yellow, odorless solid that is derived from petroleum.

Paraffin is considered nontoxic in its solid state, but fume generated when it is in the molten state may cause discomfort and nausea (Queries and Minor Notes, *JAMA* 1938/Ex. 1-308). In the most recent report of industrial exposure effects, paraffin fume is reported to cause no discomfort in most cases when the concentration is maintained at or below 2 mg/m³, although one instance of mild discomfort was reported at concentrations between 0.6 and 1 mg/m³ (Massachusetts Division of Occupational Hygiene 1970, as cited in ACGIH 1986/Ex. 1-3, p. 455).

Dr. William Zeiler, President of the College of American Pathologists (Ex. 3-976), objected to OSHA's inclusion of paraffin wax fume in the final rule, stating that the scientific basis for the limit was lacking. Specifically, Dr. Zeiler commented that the *JAMA* article (1938/Ex. 1-308) reported "vague and nonspecific" symptoms and that the reference from the Massachusetts Division of Occupational Hygiene was unpublished. He also reported that a literature search dating back to 1965 produced no references on the toxicology of paraffin wax fume. Dr. Zeiler expressed concern that, if a final limit is promulgated for paraffin wax fume, "nonspecific complaints about the workplace environment may implicate this substance as the cause" (Ex. 3-976).

OSHA is aware that the dose-response data for paraffin wax fume are dated; nevertheless, OSHA finds it notable that two different sources (cited by ACGIH 1986/Ex. 1-3, p. 455) report acute adverse effects associated with the use of molten paraffin. OSHA also believes that promulgation of a PEL for paraffin wax fume will present little problem for pathology laboratories, since Dr. Zeiler commented that most clinical laboratories already comply with the ACGIH TLVs and that the services of certified industrial hygienists are used when new laboratories are designed or old ones are remodeled. OSHA is more concerned about workplaces in which paraffin is used in large quantities, such as the food industry, and a greater degree of exposure control is needed. To protect employees in these industries from experiencing acute adverse effects, such as discomfort and nausea, OSHA believes that a PEL for paraffin was fume is necessary. The Agency has determined that the adverse effects associated with excessive exposure to paraffin wax fume constitute material impairments of health. The limit of 2 mg/m³ has been shown to be effective in reducing this risk (ACGIH 1986/Ex. 1-3, p. 455); therefore, OSHA is establishing this limit for paraffin wax fume.

PHOSPHORIC ACID

CAS: 7664-38-2; Chemical Formula: H_3PO_4; H.S. No. 1322

OSHA's former limit for phosphoric acid was 1 mg/m³ as an 8-hour TWA. The Agency proposed adding a 15-minute STEL of 3 mg/m³ based on the ACGIH recommendation, and NIOSH (Ex. 8-47, Table N1) concurred with this proposal. In the final rule, the Agency is establishing a 1-mg/m³ TWA and a 3-mg/m³ STEL for this substance. Phosphoric acid is a colorless, odorless solid at temperatures below 21°C but becomes a viscous, clear liquid at higher temperatures.

In humans, there have been reports of respiratory irritation from exposure to phosphorus pentoxide fume at concentrations of between 3.6 and 11.3 mg/m³; concentrations of 100 mg/m³ were unendurable except to workers who had developed a tolerance to the fume over time (Rushing 1957, as cited in ACGIH 1986/Ex. 1-3, p. 483). The AIHA Hygiene Guide for phosphoric acid reports that this substance is less hazardous than either nitric or sulfuric acid (AIHA 1957/Ex. 1-709).

To protect unacclimatized workers from the risk of respiratory irritation, OSHA proposed a TWA limit of 1 mg/m³, with a STEL of 3 mg/m³, for phosphoric acid. No comments, other than NIOSH's, were received on this proposal. The Agency concludes that the combined 8-hour TWA and STEL limits are necessary to reduce this significant risk of irritation, which is considered by OSHA to be material impairment of health and which has been shown to occur at levels only slightly above those permitted by the TWA alone. Therefore, OSHA is establishing a 15-minute STEL of 3 mg/m³ to supplement its 8-hour TWA PEL of 1-mg/m³ TWA PEL for phosphoric acid.

PHOSPHORUS TRICHLORIDE

CAS: 7719-12-2; Chemical Formula: PCl_3; H.S. No. 1325

OSHA's former limit for phosphorus trichloride was 0.5 ppm as an 8-hour TWA. The Agency proposed revising this limit to 0.2 ppm as an 8-hour TWA with a STEL of 0.5 ppm; NIOSH (Ex. 8-47, Table N1) concurred with this proposal. The final rule establishes these limits, which are consistent with the limits recommended by the ACGIH. Phosphorus trichloride is a fuming, colorless, noncombustible liquid.

The primary occupational hazards associated with exposure to phosphorus trichloride are respiratory irritation and intoxication involving cough, bronchitis, pneumonia, and conjunctivitis (Henderson and Haggard 1943c/Ex. 1-1086; International Labour Office 1934b, as cited in ACGIH 1986/Ex. 1-3, p. 486; Sassi 1954/Ex. 1-931).

Early studies indicate that severe symptoms did not occur in cats and guinea pigs until concentration levels reached 50 to 90 ppm for exposures lasting one hour, although slight illness was observed at 0.7 ppm after an exposure of six hours (Butjagin 1904, as cited in ACGIH 1986/Ex. 1-3, p. 486). However, by 1934, the effects of phosphorus trichloride were considered to be 5 to 10 times as intense as those of hydrolyzed hydrochloric acid (International Labour Office 1934b, as cited in ACGIH 1986/Ex. 1-3, p. 486). More recently, Weeks, Musselman, Yevich et al. (1964, as cited in ACGIH 1986/Ex. 1-3, p. 486) reported studies in which 4-hour LC_{50} values of 104 ppm for rats and 50 ppm for guinea pigs were obtained.

OSHA received comments only from NIOSH on its proposal to establish a PEL of 0.2 ppm TWA and a STEL of 0.5 ppm for phosphorus trichloride. Because of the acutely irritating effects of this substance, the Agency concludes that both a TWA and a STEL are required to reduce the risk of respiratory and eye irritation that exists for workers exposed to this substance. OSHA considers these effects to be material impairments of health. Therefore, OSHA is revising its limit for phosphorus trichloride to 0.2 ppm as an 8-hour TWA and 0.5 ppm as a 15-minute STEL; these PELs are promulgated in the final rule.

POTASSIUM HYDROXIDE

CAS: 1310-58-3; Chemical Formula: KOH H.S. No. 1334

OSHA had no former limit for potassium hydroxide. A ceiling limit of 2 mg/m³ was proposed by the Agency based on the ACGIH recommendation, and NIOSH (Ex. 8-47, Table N1)

concurred with this proposal. OSHA has concluded that this limit is necessary to afford workers protection from irritant effects and is establishing the 2-mg/m³ ceiling limit for potassium hydroxide in the final rule. Potassium hydroxide is a white, deliquescent material that occurs in the form of pellets, sticks, lumps, or flakes.

Potassium hydroxide is corrosive to tissues. The health hazards of potassium hydroxide are similar to those of the other strong alkalies, such as sodium hydroxide. These substances gelatinize tissue on contact, causing deep, painful lesions. Dust or mist exposures may cause eye or respiratory system irritation and nasal septum lesions (Karpov 1971/Ex. 1–1115).

Mr Gary Melampy of the Independent Lubricant Manufacturers Association (ILMA) (Ex. 3–830) commented that there was no basis for establishing an occupational limit for potassium hydroxide. OSHA disagrees and notes that the irritant effects of potassium hydroxide dusts, mists, and aerosols have been documented (ACGIH 1986/Ex. 1–3, p. 495; Karpov 1971/Ex. 1–1115). Although dose-response data are lacking for this substance, it is reasonable to expect potassium hydroxide to exhibit irritant properties similar to those of sodium hydroxide, a structurally related strong alkali. In its criteria document, NIOSH (1976k/Ex. 1–965) cites a personal communication (Lewis 1974), which reported that short-term exposures (2 to 15 minutes) to 2-mg/m³ sodium hydroxide caused "noticeable" but not excessive upper respiratory tract irritation. Therefore, OSHA finds that the 2-mg/m³ ceiling limit will provide workers with an environment that minimizes respiratory tract irritation, which the Agency considers to be material impairment of health. To reduce these risks, OSHA is establishing a ceiling limit of 2 mg/m³ for potassium hydoxide.

PROPYLENE GLYCOL MONOMETHYL ETHER

CAS: 107–98–2; Chemical Formula: $CH_3OCH_2CHOHCH_3$
H.S. No. 1343

OSHA had no former limit for propylene glycol monomethyl ether (PGME). The ACGIH recommends a TWA of 100 ppm and a STEL of 150 ppm, and these were the limits proposed. NIOSH has no REL for this substance but concurred (Ex. 8–47, Table N1; Ex. 150, Comments on PGME) with OSHA's proposed limits. The final rule promulgates an 8-hour TWA of 100 ppm and a STEL of 150 ppm for PGME, which is a colorless liquid.

Propylene glycol monomethyl ether is an irritant, neurotoxin, teratogen, and nasal tumorigen (Sax and Lewis 1989, p. 2904). Exposure causes anesthesia at a level of approximately 1000 ppm and eye tearing at levels above 100 ppm; at 100 ppm, PGME also has an objectionable odor (Stewart, Baretta, Dodd, and Torkelson 1970/Ex. 1–379). Ingestion of 3 g/kg in a 35-day period caused changes in the livers and kidneys of rats, and repeated dermal applications of 7 to 10 ml/kg/day caused death in rats treated over a 90-day period (Rowe, McCollister, Spencer et al. 1954/Ex. 1–435). Sax and Lewis (1989) report that exposure to this substance causes nausea, and that inhalation has induced nasal tumors.

Unlike many other members of the glycol ethers family, PGME has been shown not to cause testicular effects at levels below 3000 ppm (NIOSH 1988/Ex. 150). However, Sax and Lewis (1989) note that PGME is an experimental teratogen. Rats exposed by inhalation to 3000 ppm for six hours on days 6 through 15 of gestation produced offspring with delayed skeletal ossification (Sax and Lewis 1989; Proctor, Hughes, and Fischman 1988).

The final rule PELs for PGME of 100 ppm TWA and 150 ppm STEL are designed to protect workers from experiencing the acute effects of exposure to PGME, which include eye and skin irritation and nausea, and the chronic effects of exposure, which include possible liver and kidney changes. Because PGME was not formerly regulated by OSHA, previous workplace exposures could attain essentially uncontrolled levels, and OSHA has determined that an 8-hour TWA of 100 ppm and a STEL of 150 ppm are necessary to protect against these significant occupational risks, which constitute material impairments of health. The Agency finds that the new limits will substantially reduce these significant risks.

ROSIN CORE SOLDER PYROLSIS PRODUCTS, AS FORMALDEHYDE

CAS: None; Chemical Formula: None
H.S. No. 1350

OSHA previously had no limit for rosin core solder pyrolysis products. Based on the ACGIH TLV, the Agency proposed an 8-hour TWA of 0.1 mg/m³ for these compounds, measured as formaldehyde. OSHA had determined that a TWA limit of 0.1 mg/m³ is necessary to prevent workers from experiencing servere irritant reactions, and the Agency is including this limit in its final rule. This limit applies to the thermal decomposition products of gum rosin soldering flux (3 to 6 percent rosin and 30 to 70 percent tin-lead solder) (Lozano and Melvin, unpublished data, as cited in ACGIH 1986/Ex. 1–3, p. 514).

A two-week exposure of guinea pigs and rats to these products at average concentrations of 0.96 mg/m³ caused reduction in rate of weight gain in male guinea pigs, abnormal liver-to-body-weight ratios in guinea pigs of both sexes, and abnormal heart-to-body-weight ratios in male rats (industrial Bio-test Lab, Inc., as cited in ACGIH 1986/Ex. 1–3, p. 514). Lungs of the animals expose in this same study were hyperemic.

In humans, slight bronchial irritation has been reported at 1 mg/m³ (Industrial Bio-test Laboratories, Inc. 1967, as cited in ACGIH 1986, p. 514). Several workers who were chronically exposed to levels as high as 0.15 mg/m³ had to be removed from exposure because of intractable upper respiratory tract irritation; when concentrations were kept below 0.1 mg/m³, such irritation was not reported (Christy 1965, as cited in ACGIH 1986/Ex. 1–3, p. 514). In a study designed to quantify dose-response levels for irritation in human volunteers, subjects were exposed for 15 minutes to these products at aldehyde concentrations (measured as formaldehyde, which is the best indirect measure of rosin pyrolysis products) of 0.04 to 0.2 mg/m³ (U.S. Public Health Service 1965, as cited in ACGIH 1986/Ex. 1–3, p. 514). Subjects detected the odor at 0.07 mg/m³, and 80 percent of subjects reported moderate to severe irritation of the eyes, nose, and throat at cencentrations of 0.12 mg/m³ or above. At levels below 0.05 mg/m³, fewer than 10 percent of subjects experienced irritation. Mucous membrane irritation occurred in 30 percent of subjects exposed at 0.07 mg/m³ (U.S. Public Health Service 1965, as cited in ACGIH 1986/Ex. 1–3, p. 514).

NIOSH (Ex. 8–47, Table N6B; Tr. p. 3–97 to 3–98) did not concur with OSHA's selection of a TWA limit of 0.1 mg/m³ and recommended a ceiling limit of 0.1 ppm for a 15-minute period. In addition, NIOSH (the only commenter to the rulemaking record) considers these thermal decomposition products to be likely candidates for a separate 6(b) rulemaking.

OSHA is establishing an 8-hour TWA limit of 0.1 mg/m³, measured as formaldehyde, for rosin core solder pyrolysis products. OSHA concludes that this limit will protect employees from the significant risk of respiratory tract irritation, which is a material impairment of health, that exists at levels above the new PEL.

SODIUM BISULFITE

CAS: 7631-90-5; Chemical Formula: $NaHSO_3$
H.S. No. 1365

OSHA's Z tables previously included no exposure limit for sodium bisulfite. The Agency proposed to establish a limit of 5 mg/m³ as an 8-hour TWA, and it is establishing the PEL in the final rule. NIOSH (Ex. 8-47, Table N1) agrees with the selection of this limit, which is the same as that recommended by the ACGIH. Sodium bisulfite is a white crystalline powder and has an odor like that of sulfur dioxide.

The oral LD_{50} in rats fed this substance is 2 g/kg (Dow Chemical Company 1977d, as cited in ACGIH 1986/Ex. 1-3, p. 534), and the intraperitoneal LD_{50} for rats is 115 mg/kg (Hoppe and Goble 1951/Ex. 1-490). The ACGIH reports that sodium bisulfite is an eye, skin, and mucous membrane irritant; acute exposures have resulted in mild eye and respiratory effects (ACGIH 1986/Ex. 1-3, p. 534).

One rulemaking participant, Mr. Gary Melampy of the Independent Lubricant Manufacturers Association (ILMA), remarked that OSHA's discussion of the proposed limit for sodium bisulfite in the preamble failed to demonstrate an adequate basis for the limit. OSHA notes that dose-response data to demonstrate a no-effect level are lacking. The 5-mg/m³ limit was proposed because it represents a limit below that established for physical irritant particulates, and this limit reflects the irritant properties of sodium bisulfite. In the professional judgment of the ACGIH (1986/Ex. 1-3, p. 534), "inhalation of or contact with the dust would result in high local concentrations [of sodium bisulfite] in contact with high local concentrations of sensitive tissue." The ACGIH further states that an occupational limit below that for physical irritant particulates "seems definitely in order." OSHA concurs with this assessment.

Dr. Grace Ziem, an independent occupational physician (Ex. 46), expressed concern about the adverse effects of sodium bisulfite on sensitized individuals. Although cases of severe, and even lethal, allergic reactions to this material have been documented from the use of sodium bisulfite as a food additive, OSHA does not believe that there is sufficient information to use as a basis for an exposure limit to protect against inhalation-induced allergic reactions.

OSHA finds that exposure to this substance presents a significant risk of irritant effects at high concentrations, and that these effects constitute material impairments of health. Accordingly, to substantially reduce this risk, OSHA is establishing a 5-mg/m³ 8-hour TWA for sodium bisulfite.

SODIUM HYDROXIDE

CAS: 1310-73-2; Chemical Formula: NaOH
H.S. No. 1367

The former OSHA limit for sodium hydroxide (also known as caustic soda or lye) was 2 mg/m³ as an 8-hour TWA. OSHA proposed a 2-mg/m³ ceiling limit for sodium hydroxide, based on the ACGIH- and NIOSH-recommended limits. NIOSH (Ex. 8-47, Table N1) concurred with the proposed limit, and this limit is established in the final rule. Sodium hydroxide is a white, deliquescent solid.

Sodium hydroxide is a severe irritant of the eyes, mucous membranes, and skin. Exposure to sodium hydroxide in the form of a caustic dust irritates the uppper respiratory tract and may cause ulceration of the nasal passages (ACGIH 1986/Ex. 1-3, p. 535). Although inhalation of sodium hydroxide is usually of secondary importance in industrial exposures, the effects of inhaling the dust or mist vary from mild irritation of the nose, which occurs on brief exposure to 2 mg/m³, to severe pneumonitis, which occurs at very high exposures. The greatest industrial hazard is rapid tissue destruction of the eyes or skin upon contact either with the solid or with concentrated solutions (*Chemical Hazards of the Workplace*, 2nd ed., p. 444, Proctor, Hughes, and Fischman 1988).

Contact with the eyes causes disintegration and sloughing of conjunctival and corneal epithelium, corneal opacification, marked edema, and ulceration; after 7 to 13 days, either gradual recovery begins or there is a progression to ulceration and corneal opacification. Complications of severe eye burns are symblepharon with overgrowth of the cornea by a vascularized membrane, progressive or recurrent corneal ulceration, and permanent corneal opacification (Proctor, Hughes, and Fischman 1988, p. 444). Grant (1986/Ex. 1-975) states that sodium hydroxide causes "some of the most severe, blinding injuries of the eye. Because it may be considered public enemy number one for causing chemical burns of the eye, sodium hydroxide has been the chemical caustic most extensively studied in animal and clinical investigations." Clinically, the worst features of sodium hydroxide burns of the eye are the great rapidity with which extreme damage can be done to the anterior segment of the eye and the tendency for the cornea to ulcerate and perforate or to become densely vascularized and opaque.

On the skin, solutions of 25 to 50 percent sodium hydroxide cause the sensation of irritation within about three minutes; with solutions of 4 percent, the sensation of burning does not occur until several hours later. If not removed from the skin, sodium hydroxide causes severe burns with deep ulcerations. Exposure to the dust or mist of sodium hydroxide may cause multiple small burns with temporary loss of hair (Proctor, Hughes, and Fischman 1988, p. 445). Nagao and co-workers (1972) examined skin biopsies from volunteers who had had a 1 N solution (equal to a 4-percent solution) of sodium hydroxide applied to their arms for 15 to 180 minutes. Progressive changes, beginning with dissolution of the cells in the horny layer and progressing through edema to total destruction of the epidermis, occurred within 60 minutes (Nagao, Stroud, Hamada et al. 1972).

Rats were exposed to an aerosol of 40 percent aqueous sodium hydroxide whose particles were less than 1 um in diameter. Exposures lasted for 30 minutes and were administered twice a week. The experiment was terminated after three weeks because two of the 10 rats died. Histopathological examination showed mostly normal lung tissue with foci of enlarged alveolar septae, emphysema, bronchial ulceration, and enlarged lymph adenoidal tissues (Wands 1981b, in *Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., vol. 2B, p. 3062).

OSHA received only one comment on sodium hydroxide, from NIOSH (Ex. 150, Comments on Sodium Hydroxide); NIOSH supported OSHA's proposed limit and reported that no new information on the health effects of sodium hydroxide had become available since the publication of the NIOSH criteria document (NIOSH 1976k/Ex. 1-965).

The irritant effect of sodium hydroxide and its markedly corrosive action on all body tissue can result even from brief (one minute or more) exposures to airborne concentrations above the 2-mg/m³ level; the acute nature of these effects is evident in the studies described above. Therefore, OSHA concludes that establishing a ceiling of 2 mg/m³ is necessary to reduce the significant risks of eye and skin burns and respiratory irritation that occur as a result of very brief exposures to the higher levels of sodium hydroxide that would be permitted with an 8-hour TWA PEL alone. OSHA considers the irritant effects resulting from exposure to sodium hydroxide material impairments of health. In the final rule, OSHA is accordingly revising its former

8-hour TWA limit for sodium hydroxide to a ceiling limit of 2 mg/m³.

SODIUM METABISULFITE

CAS: 7681-57-4; Chemical Formula: $Na_2S_2O_5$ H.S. No. 1368

OSHA previously had no exposure limit for sodium metabisulfite. The Agency proposed a 5-mg/m³ limit as an 8-hour TWA, based on the ACGIH recommendation, and is establishing this limit in the final rule. NIOSH (Ex. 8-47, Table N1) concurred with the selection of this limit. Sodium metabisulfite can occur either in the form of a solid or as white crystals; this substance smells like sulfur dioxide.

A two-year study at the Dow Chemical Company (1977e, as cited in ACGIH 1986/Ex. 1-3, p. 535), in which rats ingested 0.215 percent sodium metabisulfite, demonstrated no adverse effects in the rats. Other animal studies show a median lethal dose of 192 mg/kg for rabbits and 115 mg/kg for rats when sodium metabisulfite is injected intravenously (NIOSH 1973c, as cited in ACGIH 1986/Ex. 1-3, p. 535). Inhalation of sodium metabisulfite dust is irritating to the lungs, nose, and throat (ACGIH 1986/Ex. 1-3, p. 535).

Dr. Grace Ziem, an independent physician (Ex. 46), expressed concern that sensitized individuals may experience severe allergic reactions on exposure to sodium metabisulfite dust. Cases of severe, and even fatal, reactions have been documented in individuals exposed by consuming food items containing metabisulfite additive. At this time, OSHA believes there is insufficient data on oral toxicity to use as a basis to extrapolate to the airborne concentration likely to cause sensitization.

OSHA proposed an 8-hour TWA of 5 mg/m³ for sodium metabisulfite. The agency concludes that establishing this limit is necessary to reduce the risk of skin and eye irritation associated with exposure to high concentrations of sodium metabisulfite dust. OSHA has determined that these effects constitute material impairments of health. Accordingly, OSHA is promulgating a 5-mg/m³ limit as an 8-hour TWA for this substance.

SULFUR MONOCHLORIDE

CAS: 10025-67-9; Chemical Formula: S_2Cl_2 H.S. No. 1376

OSHA's former PEL for sulfur monochloride was 1 ppm as an 8-hour TWA. Based on the ACGIH recommendation, the Agency proposed revising this limit to 1 ppm as a ceiling limit. NIOSH (Ex. 8-47, Table N1) concurred with OSHA's proposed limit for this substance, and the final rule establishes it. Sulfur monochloride is an amber, oily, nonflammable, fuming liquid, and has a penetrating odor.

Sulfur monochloride is a primary irritant that affects the upper respiratory tract by releasing hydrochloric acid (HCl) on contact with moisture (Henderson and Haggard 1943g, as cited in ACGIH 1986/Ex. 1-3, p. 545). This same study noted that "undecomposed vapor [of sulfur monochloride] might reach the lungs, in which case it would be more toxic than an equivalent quantity of HCl." The ACGIH (1986/Ex. 1-3, p. 545) considers these data indicative of a far greater acute toxicity for sulfur monochloride than for hydrochloric acid. Animal toxicity studies revealed that a dose of 150 ppm sulfur monochloride resulted in death to mice exposed for one minute (Flury and Zernik 1931k/Ex. 1-979). Cats exposed to 60 ppm sulfur monochloride for 15 minutes all died within a few days, but concentrations of 12 ppm for 15 minutes were tolerated (Henderson and Haggard 1943g, as cited in ACGIH 1986/Ex. 1-3, p. 545).

A study by Elkins (1959g, as cited in ACGIH 1986/Ex. 1-3, p. 545) of workers in the rubber industry found that concentrations of 2 to 9 ppm sodium monochloride were mildly irritating; however, the concentrations to which these workers were exposed may have included a high proportion of hydrochloric acid. NIOSH was the only commenter on sulfur monochloride.

The Agency concludes that the former TWA PEL of 1 ppm is inadequate to protect exposed workers against the risk of primary irritation that could occur upon short-term exposure to elevated concentrations of sulfur monochloride. Since 2 ppm was reported to be an effect level for mild irritation, OSHA finds that revising its limit to 1 ppm as a ceiling limit is a reasonable and necessary action to protect workers from the significant risk associated with lung irritation, which constitutes a material impairment of health. Therefore, OSHA is establishing a ceiling limit for sulfur monochloride of 1 ppm.

SULFUR PENTAFLUORIDE

CAS: 5714-22-7; Chemical Formula: S_2F_{10} H.S. No. 1377

The previous OSHA limit for sulfur pentafluoride was 0.025 ppm as an 8-hour TWA. OSHA proposed revising this limit to 0.01 ppm as a ceiling, and NIOSH (Ex. 8-47, Table N1) concurred with this proposal. The Agency is establishing this limit in the final rule. This limit is consistent with the ACGIH (1986/Ex. 1-3) recommended limit. Sulfur pentafluoride is a colorless gas or liquid with a sulfur-dioxide-like odor.

Sulfur pentafluoride's toxic effects include lung congestion and lesions, and pulmonary edema. In a study in which rats were exposed to sulfur pentafluoride for 16 to 18 hours, levels of 0.1 ppm caused lung irritation, 0.5 ppm resulted in severe pulmonary lesions, and 1 ppm proved fatal (Greenberg and Lester 1950/Ex. 1-590). One-hour exposures to 10 ppm sulfur pentafluoride resulted in diffuse hemorrhagic lesions in the lungs of rats, while rats exposed to 1 ppm for one hour had severe congestion of the lungs. Rats exposed for one hour to 0.1 ppm showed no effects. Subsequent examination of rats surviving the 10- and 1-ppm exposures revealed that the lungs had returned to normal after 24 hours (Greenberg and Lester 1950/Ex. 1-590). Saunders, Shoshkes, DeCarlo, and Brown (1953/Ex. 1-610) established that the LD_{50} for sulfur pentafluoride in rabbits is 5.8 mg/kg, and that death was due to fulminant pulmonary edema. According to this study, sulfur pentafluoride does not injure the columnar epithelium of the respiratory tract, and exposure is not followed by bronchopneumonia.

Other than NIOSH's submission, OSHA received no comments on its proposal to revise the sulfur pentafluoride limit to 0.01 ppm as a ceiling. The 0.01-ppm ceiling was selected on the basis of evidence showing that even brief exposures to 1 ppm caused pulmonary affects in animals and prolonged exposures to 0.1 ppm caused lung irritation in animals. OSHA concludes that this limit for sulfur pentafluoride will reduce the risks of irritation and pulmonary effects to which workers could be exposed in the absence of a ceiling limit. The Agency considers these effects material impairments of health. Therefore, OSHA is promulgating a ceiling limit for sulfur pentafluoride of 0.01 ppm.

TETRAHYDROFURAN

CAS: 109-99-9; Chemical Formula: $(C_2H_4)_2O$ H.S. No. 1387

OSHA's former PEL for tetrahydrofuran was 200 ppm as an 8-hour TWA. The Agency proposed revising this limit to 200 ppm TWA with a 15-minute STEL of 250 ppm and is establishing these limits, which are consistent with those recommended by the ACGIH, in the final rule. NIOSH (Ex. 8-47, Table N1) concurred with OSHA's proposal to add a STEL for this substance. Tetrahydrofuran is a colorless liquid with an odor like that of ether.

This proposed limit was selected on the basis of extensive data from experimental animal studies. Lehmann

and Flury (1943c/Ex. 1–879) reported irritation of the upper respiratory tract as well as kidney and liver injury in a number of animals exposed by inhalation to more than 3000 ppm tetrahydrofuran for 20 days, eight hours daily. Aqueous solutions exceeding a concentration of 20 percent tetrahydrofuran proved irritating to the skin of rabbits. One study (Stoughton and Robbins 1936/Ex. 1–597) found that tetrahydrofuran concentrations in excess of 25,000 ppm were needed to anesthetize dogs. The anesthesia process in these animals showed a delayed induction period and poor recovery. In other studies with dogs (Zapp 1971, as cited in ACGIH 1986/Ex. 1–3, p. 564, 200 ppm tetrahydrofuran in daily six-hour inhalation exposures produced an observable effect on the pulse pressure of these animals within three to four weeks; despite an exposure of nine weeks at this dosage level followed by three weeks at nearly twice this concentration, no histopathologic changes were observed in the critical organs. Studies (Jochmann 1961/Ex. 1–1021) in which tetrahydrofuran was given orally and peritoneally to a variety of laboratory animals resulted in both liver and kidney damage; however, some of the effects observed by this author may have been caused by peroxide contamination of the tetrahydrofuran. Oettel (as cited in ACGIH 1986/Ex. 1–3, p. 564) observed no kidney or liver damage in cats, rabbits, rats, or mice exposed repeatedly by inhalation to tetrahydrofuran at concentrations of 3400 to 17,000 ppm for as long as six hours. Technicians involved in the experiment of Stoughton and Robbins (1936/Ex. 1–597, described above) experienced severe headaches when conducting these experiments.

Dr. Larry Hecker, Director of Corporate Industrial Hygiene and Toxicology for Abbott Laboratories, commented that there was no toxicological basis to justify a STEL for tetrahydrofuran (Ex. 3–678). However, OSHA believes that the severe headaches experienced by researchers conducting animal experiments (Stoughton and Robbins 1936/Ex. 1–597) are indicative of an acute effect that constitutes material impairment of health and is best avoided by establishing a short-term limit. OSHA also notes that the ACGIH (*Threshold Limit Values and Biological Exposure Indicies for 1988–1989*, ACGIH 1988b) has not proposed to delete its recommended STEL for this substance. Therefore, OSHA finds that both a 200-ppm 8-hour TWA and a 250-ppm STEL are necessary to reduce the risk of long-term systemic and acute effects associated with exposure to tetrahydrofuran and is establishing these limits in the final rule.

TETRASODIUM PYROPHOSPHATE

CAS: 7722–88–5; Chemical Formula: $Na_4P_2O_7$
H.S. No. 1389

The OSHA Z tables previously included no limit for tetrasodium pyrophosphate. OSHA proposed a PEL of 5 mg/m^3 as an 8-hour TWA, and NIOSH (Ex. 8–47, Table N1) concurred with OSHA's proposed limit for this substance. This limit is established in the final rule and is consistent with the ACGIH recommendation. Tetrasodium pyrophosphate may occur as either a white powder or a crystalline substance.

Tetrasodium pyrophosphate is an alkaline dust and therefore causes irritation to the eyes and the respiratory tract (ACGIH 1986/Ex. 1–3, p. 567). For this reason, the ACGIH recommended a time-weighted average TLV of 5 mg/m^3, which is one-half the value recommended for irritant dusts. NIOSH's comments was the only one submitted on OSHA's proposal to issue a 5-mg/m^3 8-hour TWA for this substance.

The Agency concludes that this previously unregulated chemical poses a significant risk of eye and respiratory tract irritation to workers potentially exposed to high concentrations. OSHA has determined that these irritant effects represent material impairments of health. Accordingly, OSHA is promulgating a 5-mg/m^3 8-hour TWA limit for tetrasodium pyrophosphate in the final rule.

THIOGLYCOLIC ACID

CAS: 68–11–1; Chemical Formula: $C_2H_4O_2S$
H.S. No. 1392

OSHA had no former PEL for thioglycolic acid. The Agency proposed a 1-ppm 8-hour TWA, with a skin notation, for this colorless liquid, which has an unpleasant odor; NIOSH (Ex. 8–47, Table N1) concurred with this proposal. The 1-ppm TWA limit and the skin notation, which are the same limits as recommended by the ACGIH, are established in the final rule.

A study by the Dow Chemical Company (1973b, as cited in ACGIH 1986/Ex. 1–3, p. 571) in which thioglycolic acid was instilled into the eyes of rabbits resulted in severe conjunctival inflammation and pain, dense opacity of the cornea, and severe inflammation of the iris. These effects had not improved 14 days after exposure and washing immediately after exposure did not modify the severity of this ocular response. A single dermal application of thioglycolic acid to rabbit skin caused necrosis within five minutes and was accompanied by hyperemia and edema. The LD_{50} for a 10-percent solution applied percutaneously was 848 mg/kg for rabbits (ACGIH 1986/Ex. 1–3); further studies by Dow (1973b, as cited in ACGIH 1986/Ex. 1–3, p. 571), in which female rats were fed a single oral dose of a 10-percent solution of thioglycolic acid, showed that this dose resulted in death at the level of 125 mg/kg. Autopsy revealed damage to the liver and gastrointestinal tract. Fassett (1963b, as cited in ACGIH 1986/Ex. 1–3, p. 571) reported that the oral LD_{50} for undiluted thioglycolic acid in rats was 50 mg/kg, and that a 10-percent solution applied to the skin of guinea pigs caused fatalities at doses of less than 5 ml/kg. Symptoms prior to death included gasping, convulsions, and weakness.

No rulemaking participants, other than NIOSH, commented on OSHA's proposal to establish a 1-ppm 8-hour TWA limit for thioglycolic acid. The evidence described above clearly demonstrates that this substance is a potent irritant; accordingly, OSHA finds that a limit on airborne exposure is necessary to protect workers from the risk of eye and skin irritation and systemic effects, which constitute material impairments of health. Therefore, OSHA is establishing a 1-ppm 8-hour TWA limit for this substance. In addition, the animal evidence shows that thioglycolic acid solutions readily penetrate the skin in lethal quantities (the dermal LD_{50} in rabbits is 848 mg/kg). Thus, OSHA finds that a skin notation is necessary to limit dermal contract and is adding this notation to its limit for thioglycolic acid.

1,2,4-TRICHLOROBENZENE

CAS: 120–82–1; Chemical Formula: $C_6H_3Cl_3$
H.S. No. 1405

OSHA formerly had no limit for 1,2,4-trichlorobenzene and proposed to establish a limit of 5 ppm as a ceiling for this substance. NIOSH (Ex. 8–47, Table N1) concurred with this proposal. The final rule establishes this limit, which is consistent with the ACGIH recommendation. 1,2,4-Trichlorobenzene is a colorless, stable liquid at room temperature, with an odor similar to that of o-dichlorobenzene.

The inhalation toxicity of 1,2,4-Trichlorobenzene was studied by Treon (1950, as cited in ACGIH 1986/Ex. 1–3, p. 593), who determined that the target organs of exposure in cats, dogs, rats, rabbits, and guinea pigs included the liver, kidneys, ganglion cells at all brain levels, and mucous membranes. Irritation of the lungs and changes in respiration were seen in animals that

later died as a result of exposure. Brown, Muir, and Thorpe (1969/Ex. 1–537) reported that 1,2,4-trichlorobenzene's single-dose oral LD_{50} is 756 mg/kg for rats and 766 mg/kg for mice. The acute percutaneous LD_{50} for rats was 6139 mg/kg. Sublethal doses administered repeatedly to guinea pigs caused liver damage; acute and short-term (15 six-hour exposures to 70 to 200 ppm) inhalation studies failed to kill these animals (Gage 1970/Ex. 1–318). In a separate study reported on by Rowe (1975, as cited in ACGIH 1986/Ex. 1–3, p. 593), 20 male rats, 4 rabbits, and 2 dogs were exposed at levels of 30 or 100 ppm, 1,2,4-trichlorobenzene for seven hours/day, five days/week, for a total of 30 exposures in 44 days. No adverse effects were detected in exposed animals belonging to 30 species as a result of exposure to 30 ppm, with the exception of an elevation of urinary porphyrins in the rats at days 15 and 30 of exposure. A second inhalation study was performed with 1,2,4-trichlorobenzene administered seven hours/day, five days/week for 26 consecutive weeks (Coate, Schoenfisch, Busey, and Lewis 1977, as cited in ACGIH 1986/Ex. 1–3, p. 593). Thirty rats, 16 rabbits, and 9 monkeys, all males were exposed at 0, 25, 50, or 100 ppm. Microscopic changes were seen in the parenchymal cells of the livers and kidneys of all rats after weeks 4 and 13 of exposure to 1,2,4-trichlorobenzene, but no adverse effects were seen in any of the other species.

In workers exposure to 1,2,4-trichlorobenzene caused dermal irritation, which may have been attributable to the defatting action of this chemical (Powers, Coate, and Lewis 1975/Ex. 1–658), and in some cases, exposure levels of 3 to 5 ppm caused eye and throat irritation (Rowe 1975, as cited in ACGIH 1986/Ex. 1–3, p. 593). NIOSH was the only rulemaking participant to submit comments on 1,2,4-trichlorobenzene.

The Agency concludes that the PPL being established today will protect workers from the risk of eye, throat, and dermal irritation associated with exposure to this substance; these adverse effects represent material impairments of health. To afford workers this protection, OSHA is promulgating a ceiling limit of 5 ppm for 1,2,4-trichlorobenzene.

TRIETHYLAMINE

CAS: 121–44–8; Chemical Formula: $(C_2H_5)_3N$
H.S. No. 1408

OSHA previously had a limit of 25 ppm TWA for triethylamine. Based on the ACGIH recommendation, the Agency proposed revising this limit to 10 ppm as a TWA and 15 ppm as a 15-minute STEL for this colorless liquid with a strong, ammonia-like odor. NIOSH (Ex. 8–47, Table N1) concurred with this proposal, and OSHA establishing these limits for triethylamine.

Exposure to triethylamine is associated with pulmonary, skin, and eye irritation and central nervous system effects. Guinea pigs exposed for 30 minutes to a concentration of 2000 ppm triethylamine survived, but four of six animals died when exposed to this level for two hours; two of six guinea pigs died during a four-hour exposure to a concentration of 1000 ppm, but all survived similar exposures at the 250- and 500-ppm levels (Carpenter, Smyth, and Shaffer 1948/Ex. 1–892). The single-dose oral LD_{50} value in rats is 0.46 g/kg (range: 0.25 to 0.85) (Smyth, Carpenter, and Weil 1951/Ex. 1–439). These investigators also reported that triethylamine readily penetrated rabbit skin on contact, with an LD_{50} value of 0.57 ml/kg (range: 0.36 to 0.90); skin irritation and eye injury were also noted from contact with the liquid. One of six rats died from an acute four-hour inhalation exposure to 1000 ppm triethylamine (Smyth, Carpenter, and Weil 1951/Ex. 1–439). Rabbits exposed repeatedly to a level of 50 ppm exhibited marked irritation of the cornea and of pulmonary tissue (Brieger and Hodes 1951/Ex. 408; Carpenter and Smyth 1946/Ex. 859). The effects of repeated triethylamine exposure correspond to those of ethylamine and dietyhylamine (Brieger and Hodes 1951/Ex. 1–408). Triethylamine was also found to inhibit monoamine oxidase activity, resulting in central nervous system stimulation (De Bruin 1976/Ex. 1–895).

OSHA received a comment on its proposal to revise the limit for triethylamine from Mr. H.K. Thompson, Corporate Industrial Hygienist for Caterpiller, Inc. (Ex. 3–349), who agreed that the 25-ppm PEL is too high, but recommended that OSHA establish a 15-ppm TWA and a 25-ppm STEL. He stated that, in his experience, where triethylamine is used as a catalyst in the making of foundry cores, 16 ppm "produces no irritation or 'halo' effect."

OSHA appreciates the suggestion made by Mr. Thompson; however, the Agency is concerned that his suggestion STEL of 25 ppm is not sufficiently protective, given that rabbits exposed repeatedly to 50 ppm exhibited marked irritation of the cornea and pulmonary tissue. OSHA judges that a somewhat greater margin of safety is called for to protect employees who may regularly be exposed to short-term elevated concentrations of triethylamine. Therefore, OSHA is establishing the limits originally proposed for triethylamine, which are 10 ppm as an 8-hour TWA and 15 ppm as a 15-minute STEL. The Agency believes that these limits are necessary to reduce the significant risk of irritation, which constitutes a material impairment of health that is associated with exposure to this substance.

VANADIUM (V_2O_5) DUST, RESPIRABLE

CAS: 1314–62–1; Chemical Formula: V_2O_5
H.S. No. 1421

The former OSHA PEL for vanadium pentoxide dust was a ceiling of 0.5 mg/m^3. The Agency proposed a limit of 0.05 mg/m^3 as an 8-hour TWA for the respirable dust of vanadium, as vanadium pentoxide, and is establishing this limit today in its final rule. This limit is the same as that recommended by the ACGIH. Vanadium pentoxide is a yellow to rust brown crystalline compound.

Several studies indicate that OSHA's current exposure limit is insufficient to protect exposed workers against vanadium dust's respiratory effects, which include bronchitis, emphysema, tracheitis, pulmonary edema, and bronchial pneumonia. According to Hudson (1964/Ex. 1–880), vanadium is poisonous to all animals by all routes of administration. The LD_{50} in rabbits injected intravenously was 1.5 mg/kg, and rats fed 25 ppm demonstrated toxic responses within a short time (Hudson 1964/Ex. 1–880).

Seven cases of upper respiratory tract irritation were reported in boiler cleaners exposed to concentrations ranging from 2 to 85 mg/m^3 vanadium pentoxide dust (Sjöberg 1951/Ex. 1–437). Williams (1952/Ex. 1–456) reported eight cases of vanadium poisoning in workers cleaning boilers in an atmosphere ranging from 30 to 104 mg/m^3. Gul'ko (1956, as cited by Hudson 1964/Ex. 1–880) observed eye and bronchial irritation in workers exposed to 0.5 to 2.2 mg/m^3. A study by Lewis (1959/Ex. 1–345) indicated that workers exposed to levels of 0.2 to 0.5 mg/m^3 experienced a higher incidence of respiratory symptoms than did controls. Tebrock and Machle (1968/Ex. 1–446) reported that workers exposed to average concentrations of 1.5 mg/m^3 vanadium pentoxide in a mixed dust developed conjunctivitis, tracheobronchitis, and dermatitis. A single average eight-hour exposure to 0.2 mg/m^3 respirable vanadium dust caused severe upper respiratory tract irritation in five human volunteers, and two other subjects exposed to a 0.1-mg/m^3 concentration also developed delayed cough and an

increase in mucous production (Zenz and Berg 1967/Ex. 1–405).

NIOSH (Ex. 8–47, Table N7; Tr. p. 3–99) recommended a ceiling limit of 0.05 mg/m³ for a 15 minute period for this substance. The Workers Institute for Safety and Health (WISH) (Ex. 116, pp. 53) supported NIOSH's recommendation.

In the final rule, OSHA is establishing a limit of 0.05 mg/m ³ as an 8-hour TWA for respirable vanadium dust, measured as vanadium pentoxide. The Agency concludes that this limit will prevent or substantially reduce the risks of eye and bronchial irritation, respiratory symptoms, conjunctivitis, and coughing seen in workers exposed at levels ranging from 0.1 to 2.2 mg/m³. OSHA considers these exposure-related effects material impairments of health.

VANADIUM (V_2O_5) FUME

CAS: 1314–62–1; Chemical Formula: V_2O_5
H.S. No. 1422

OSHA'S former PEL for vanadium pentoxide fume was 0.1 mg/m³ as a ceiling limit. The Agency proposed to revise this limit to 0.05 mg/m³ as an 8-hour TWA, based on the ACGIH recommendation. OSHA is establishing this limit in the final rule.

Vanadium pentoxide fume's chief toxic effects are manifested in the respiratory passages: bronchitis, emphysema, tracheitis, pulmonary edema, and bronchial pneumonia can result from exposure. According to Hudson (1964/Ex. 1–880), vanadium is poisonous to all animals by all routes of administration. The LD_{50} in rabbits injected intravenously is 1.5 mg/kg, and rats fed 25 ppm demonstrated toxic responses within a short time (Hudson 1964/Ex. 1–880).

Seven cases of upper respiratory tract irritation were reported in boiler cleaners exposed to concentrations of from 2 to 85 mg/m³ vanadium pentoxide fume (Sjôberg 1951/Ex. 1–437). Williams (1952/Ex. 1–456) reported eight cases of vanadium poisoning in workers cleaning boilers in an atmosphere ranging from 30 to 104 mg/m³. Gul'ko (1956, as cited by Hudson 1964/Ex. 1–880) observed eye and bronchial irritation in workers exposed to 0.5 to 2.2 mg/m³. A study by Lewis (1954/Ex. 1–345) indicated that workers exposed to levels of 0.2 to 0.5 mg/m³ experienced a higher incidence of respiratory symptoms than did controls. Tebrock and Machle (1968/Ex. 1–446) reported that workers exposed to average concentrations of 1.5 mg/m³ vanadium pentoxide in a mixed dust developed conjunctivitis, tracheobronchitis, and dermatitis. A single average eight-hour exposure to 0.2 mg/m³ respirable vanadium dust caused severe upper respiratory tract irritation in five human volunteers, and two other subjects exposed to a 0.1-mg/m³ concentration also developed delayed cough and an increase in mucous production (Zenz and Berg 1967/Ex. 1–405).

NIOSH (Ex. 8–47, Table N7) recommended a 15-minute ceiling limit of 0.05 mg/m³ for vanadium fume as vanadium pentoxide. However, OSHA is concerned about cumulative exposures below the former 0.1 mg/m³ ceiling, and the Agency concludes that the TWA limit originally proposed will protect workers from the significant risks of eye, skin, and upper respiratory tract irritation; conjunctivitis; pulmonary damage; and systemic poisoning associated with exposure to vanadium pentoxide fume at even brief excursions to higher levels. The Agency considers these irritant and systemic effects to be material impairments of health. Accordingly, OSHA is establishing a PEL of 0.05 mg/m³ as an 8-hour TWA for this substance in today's rule.

VINYL ACETATE

CAS: 108–05–4; Chemical Formula: $CH_3COOCH{=}CH_2$
H.S. No. 1424

There was no previous OSHA limit for vinyl acetate. OSHA proposed establishing a 10-ppm TWA and a 20-ppm STEL for this substance, based on the ACGIH recommendation, and the final rule establishes these limits. Vinyl acetate is a volatile liquid that polymerizes in light to a colorless, transparent mass and usually contains an inhibitor, such as hydroquinone.

The basis for the proposed limits is an epidemiologic report by Deese and Joyner (1969/Ex. 1–412) describing 15 years of industrial experience with vinyl acetate production. These authors reported that vinyl acetate is not a significant irritant at exposure levels of 5 to 10 ppm but causes cough and hoarseness at around 22 ppm. They also found no evidence of adverse chronic effects resulting from exposure to 5 to 10 ppm, as determined from medical records and examinations. While conducting air sampling for the study, the primary author (Deese) experienced hoarseness at concentrations of 4.2 to 5.7 ppm, and eye irritation at 5.7 to 6.8 ppm. Three chemical operators and one technician did not report any subjective responses at these levels. The ACGIH (1986/Ex. 1–3, p. 621) also cited a personal communication from the Mellon Institute (1968) stating that vinyl acetate concentrations of less than 5 ppm are detectable by odor, although some individuals may detect the odor at concentrations of 0.5 ppm (Mellon Institute 1968, as cited by ACGIH 1986/Ex. 1–3, p. 621).

NIOSH (1978i, as cited in ACGIH 1986/Ex. 1–3, p. 621) reviewed these data and concluded that the recommended exposure limit be designed to protect even the most sensitive individuals from sensory irritant effects. Since the lowest level reported to cause upper respiratory tract irritation was 4.2 ppm (Deese and Joyner 1969/Ex. 1–412), NIOSH recommended that workplace exposure not exceed 4 ppm measured over a 15-minute period. In its prehearing submission (Ex. 8–47, Table N2), NIOSH continued to recommend its earlier limit.

The NIOSH REL of 4 ppm (ceiling) relies on a report concerning the experience of a single individual; in contrast, the limits being established today are based on a 15-year epidemiology study that suggests that a 10-ppm TWA and a 20-ppm STEL will provide protection against the risk of irritation associated with exposure to vinyl acetate at higher levels. OSHA considers the irritation caused by exposure to vinyl acetate a material impairment of health. Therefore, the Agency is promulgating this 8-hour TWA and STEL combination as the revised limits for vinyl acetate.

VM & P NAPHTHA

CAS No. 8032–32–4; Chemical Formula: none
H.S. No. 1429

OSHA formerly had no PEL for VM & P (Varnish Markers' and Printers') naphtha. The Agency proposed to establish an 8-hour TWA of 300 ppm and a STEL of 400 ppm for this substance. NIOSH (Ex. 8–47, Table N7) concurred with these limits, which are based on the ACGIH TLVs. These limits are established in the final rule. VM & P naphtha, also known as ligroin, is a colorless, flammable liquid.

A study in which rats and beagles received inhalation doses of 500 ppm VM & P naphtha for 30 hours per week for 13 weeks resulted in no chronic or latent effects (Carpenter, Kinkead, Geary et al. 1975a/Ex. 1–302). These authors also noted that the acute toxicity of VM & P naphtha for rats and other species was four times greater than that of rubber solvent naphtha, which has a limit of 400 ppm. Carpenter and associates (1975a/Ex. 1–302) also reported on an experiment in which rats lost coordination and went into convulsions within 15 minutes during exposures to saturation concentrations at ambient room temperature. The 4-hour inhalation LC_{50} was 3400 ppm, and the acclimated rats survived 5800 ppm for six hours.

Seven human volunteers exposed to 880 ppm VM & P naphtha for 15 minutes reported upper respiratory tract, eye, and nose irritation, in addition to olfactory fatigue (ACGIH 1986/Ex. 1-3, p. 631). Elkins (1959d, as cited in ACGIH 1986/Ex. 1-3, p. 631) noted one case of a worker, exposed to levels of VM & P naphtha averaging 800 ppm, who developed unspecified chronic effects. Elkins also reported that the VM & P naphtha level producing significant irritation in human volunteers was about half as great for this form of naphtha as for rubber solvent naphtha.

The Agency concludes that the 300-ppm TWA is necessary to protect workers against the risk of possible chronic effects associated with naphtha exposure. In addition, OSHA finds that a STEL is necessary to prevent upper respiratory tract and eye irritation, which are considered by OSHA to be material impairments of health that have been demonstrated to occur on short-term exposure to 880 ppm VM & P naphtha (ACGIH 1986/Ex. 1-3, p. 631); the proposed 300-ppm TWA limit alone would permit such excursions. Therefore, OSHA is establishing both a 300-ppm 8-hour TWA and a 400-ppm STEL for VM & P naphtha in the final rule.

XYLENES, (o-, m-, AND p-ISOMERS)

CAS: 1330-20-7; Chemical Formula: $C_6H_4(CH_3)_2$
H.S. No. 1431

The previous OSHA limit for the xylenes was 100 ppm as an 8-hour TWA. Based on the ACGIH recommendation, OSHA proposed to revise this limit to a TWA of 100 ppm and a 15-minute STEL of 150 ppm. NIOSH (Ex. 8-47, Table N1) as well as the AFL-CIO (Ex. 194) concurred with these limits, and they are established in the final rule. The xylene isomers are clear, flammable liquids with an aromatic hydrocarbon odor.

Rats and rabbits exposed to a mixture of xylene isomers at a concentration of 690 ppm for eight hours daily, six days per week showed no blood abnormalities, but rabbits exposed on the same regimen at 1150 ppm for 55 days showed a decrease in red and white blood cell counts and an increase in platelet count (Fabre and Truhaut 1954, as cited in ACGIH 1986/Ex. 1-3, p. 637).

Studies of workers exposed to xylene revealed headache, fatigue, lassitude, irritability, and gastrointestinal disturbances as the most common symptoms (Gerarde 1960d/Ex. 1-738a). At unspecified exposure levels, Browning (1965b/Ex. 1-1016) also noted gastrointestinal disturbances, in addition to kidney, heart, liver, and neurological damage; blood dyscrasias, some of which resulted in death, were also reported in these workers. A study by Nelson, Enge, Ross et al. (1943/Ex. 1-66), in which human volunteers were exposed to 200 ppm xylene, found eye, nose, and throat irritation in the subjects at this level of exposure.

NIOSH developed a criteria document for xylene in 1975 (NIOSH 1975; as cited in ACGIH 1986/Ex. 1-3, p. 637), in which the work of Morley, Eccleston, Douglas, and colleagues (1970/Ex. 1-794) was discussed). These authors observed liver dysfunction and renal impairment in three workers overexposed to xylene (estimated concentration of 10,000 ppm). One of these workers died, but the others recovered slowly. Furniture polishers were reported by Matthaus (1964/Ex. 1-830) to have suffered corneal damage as a result of exposure to xylene at unknown concentrations.

One other commenter, Stanley L. Dryen of Chevron Corporation (Ex. 3-896, p. 15), objected to OSHA's issuing of a STEL, stating that there was no basis for one. OSHA disagrees and points out that a 100-ppm TWA limit alone would permit short-term exposure to several hundred ppm xylene, well above the 200-ppm level reported to be irritating as a result of short-term exposures. OSHA notes that NIOSH also recommends a short-term limit to supplement the TWA.

After reviewing this evidence, OSHA concludes that both a TWA and a STEL are necessary to prevent the risks of narcosis, blood effects, and irritant effects at the elevated levels possible at the current exposure limit. The Agency considers the effects of narcosis, irritation, and blood effects to constitute material impairments of health and functional capacity. Therefore, to reduce the risk of irritation to workers exposed to the xylenes, OSHA is establishing a 150-ppm STEL and a 100-ppm TWA for xylene isomers in the final rule.

ZINC CHLORIDE (FUME)

CAS: 7646-85-7; Chemical Formula: $ZnCl_2$
H.S. No. 1435

OSHA's former PEL for zinc chloride was 1 mg/m ^{3} as an 8-hour TWA. The Agency proposed a TWA of 1 mg/m ^{3}, with a STEL of 2 mg/m ^{3}, for this substance, based on the ACGIH recommendation. NIOSH (Ex. 8-47, Table N1) concurred with this proposal, and these limits are established in the final rule. Zinc chloride fume is white and has an acrid odor.

Zinc chloride fume is highly caustic and damages the mucous membranes of the nasopharynx and respiratory tract. Exposure to the fumes of the zinc chloride may result in a severe pneumonitis that is caused by irritation of the respiratory tract (Gafafer 1964/Ex. 1-1149). One instance in which a worker inhaled zinc chloride fumes resulted in advanced pulmonary fibrosis that ended in death (Milliken, Waugh, and Kadish 1963/Ex. 1-751), and 10 deaths and 25 nonfatal cases of pneumonitis occurred in workers caught in a tunnel when 79 smoke generators caught fire and generated zinc chloride fumes (Hunter 1955/Ex. 1-853). Other studies have shown that zinc chloride exposures cause skin ulceration (Sax 1957/Ex. 1-1095). It has also been suggested that zinc chloride exposure may have chronic effects (Hamilton and Hardy 1974b/Ex. 1-958). In an investigation of the adverse effects of zinc chloride fume exposures, Ferry (1966, as cited in ACGIH 1986/Ex. 1-3, p. 643) reported that no sensory effects occurred when 30-minute exposures were limited to 0.07 and 0.4 mg/m^3; however, this researcher noted that these levels did corrode metal. Other than NIOSH's submission, no comments were received by OSHA on the proposed limits for zinc chloride fume.

OSHA concludes that the risk of damage to the eyes, skin, and respiratory tract associated with short-term exposure to zinc chloride fume, which are considered by OSHA to be material impairments of health, should be substantially reduced by establishing both a STEL and a TWA. Therefore, in the final rule, OSHA is promulgating a 1-mg/m^3 TWA limit and 2-mg/m^3 STEL for this substance.

Conclusions for the Group of Sensory Irritants

OSHA finds that sensory irritation poses an occupational health risk to workers exposed to these substances at the Agency's former exposure limits. Among the adverse health consequences of exposure to sensory irritants are acute breathing difficulty, eye tearing, conjunctivitis, sensitization, persistent coughing, and upper respiratory tract irritation. OSHA has determined that these effects constitute material impairments of health and functional capacity within the meaning of the Act. In addition to the pain and suffering associated with these signs and symptoms, workers experiencing irritant effects find it difficult if not impossible to concentrate on the job at hand; they therefore work less safely and less productively than nonexposed employees. Reducing exposures from levels that have been associated with these effects to levels where such consequences are substantially less

likely to occur will reduce the significant risk posed to workers at current levels. Furthermore, many of the substances in this group have been demonstrated to have adverse effects on other organ systems, including the cornea, lungs, kidney, liver, central nervous system, and gastrointestinal tract. OSHA finds that promulgation of the new or revised limits for the substances in this group will also further reduce the possibility of harm to these organ systems.

OSHA concludes that the health evidence for these substances forms a reasonable basis for establishing revised or new limits, and that establishing these limits is necessary to reduce the risk of sensory irritation effects to exposed workers. OSHA concludes that sensory irritation constitutes a material impairment of health and functional capacity.

4. Substances for Which Limits Are Based on Avoidance of Liver or Kidney Effects

Introduction

The liver or the kidneys are the primary target organs affected by toxic exposures to a number of industrial chemicals. In recognition of this target organ toxicity, OSHA is establishing new or revised limits for 17 hepato- or nephrotoxic compounds (12 hepatotoxins and five nephrotoxins). For these substances, the liver or kidney appears to be the organ most sensitive to the effects of exposure. Thus, establishing permissible exposure limits that are low enough to prevent toxicity to these target organs generally also protects other organ systems.

For seven of the 12 substances for which limits were based on liver toxicity, OSHA is lowering the PEL, and for three substances, OSHA is adding a short-term exposure limit. For two substances, OSHA is adding a PEL where none previously existed. For three kidney toxins, OSHA is establishing new PELs; in one case, it is reducing an existing TWA-PEL, and, in another case, it is reducing its current PEL and adding a STEL. The sections below discuss liver and kidney toxins separately. Table C4–1 shows these hepatotoxic substances and their former, proposed, and final rule limits, CAS, and HS numbers; Table C4–2 provides the same information for the nephrotoxins in this group.

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Table C4-1. List of Substances For Which Limits Are Based Primarily on Avoidance of Liver Toxicity

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1011 Allyl chloride	107-05-1	1 ppm TWA	1 ppm TWA 2 ppm STEL	1 ppm TWA 2 ppm STEL
1072 Carbon tetrabromide	558-13-4	--	0.1 ppm TWA 0.3 ppm STEL	0.1 ppm TWA 0.3 ppm STEL
1089 o-Chlorostyrene	2039-87-4	--	50 ppm TWA 75 ppm STEL	50 ppm TWA 75 ppm STEL
1108 Cyclohexanone	108-94-1	50 ppm TWA	25 ppm TWA, Skin	25 ppm TWA, Skin
1145 Dioxane	123-91-1	100 ppm TWA, Skin	25 ppm TWA, Skin	25 ppm TWA, Skin
1168 Ethylene dichloride	107-06-2	50 ppm TWA 100 ppm STEL (5 min/3 hrs) 200 ppm Ceiling	1 ppm TWA 2 ppm STEL	1 ppm TWA 2 ppm STEL
1205 Hydrazine	302-01-2	1 ppm TWA, Skin	0.1 ppm TWA, Skin	0.1 ppm TWA, Skin

Table C4-1. List of Substances For Which Limits Are Based Primarily on Avoidance of Liver Toxicity (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1269 Methylcyclohexanol	25639-42-3	100 ppm TWA	50 ppm TWA	50 ppm TWA
1295 Octachloro-naphthalene	2234-13-1	0.1 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA 0.3 mg/m^3 STEL, Skin	0.1 mg/m^3 TWA 0.3 mg/m^3 STEL, Skin
1341 Propylene dichloride	78-87-5	75 ppm TWA	75 ppm TWA 110 ppm STEL	75 ppm TWA 110 ppm STEL
1385 1,1,2,2-Tetrachloro-ethane	79-34-5	5 ppm TWA, Skin	1 ppm TWA, Skin	1 ppm TWA, Skin
1407 1,2,3-Trichloro-propane	96-18-4	50 ppm TWA	10 ppm TWA, Skin	10 ppm TWA

* OSHA's TWA limits are for 8-hour exposures and its STELs are for 15 minutes only unless otherwise specified.

Table C4-2. List of Substances For Which Limits are Based Primarily on Avoidance of Kidney Toxicity

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1129 1,3-Dichloropropene	542-75-6	--	1 ppm TWA, Skin	1 ppm TWA, Skin
1132 Dicyclopentadiene	77-73-6	--	5 ppm TWA	5 ppm TWA
1166 Ethyl silicate	78-10-4	100 ppm TWA	10 ppm TWA	10 ppm TWA
1195 Hexachlorobutadiene	87-68-3	--	0.02 ppm TWA, Skin	0.02 ppm TWA
1203 Hexone (Methyl isobutyl ketone)	108-10-1	100 ppm TWA	50 ppm TWA 75 ppm STEL	50 ppm TWA 75 ppm STEL

* OSHA's TWA limits are for 8-hour exposures and its STELs are for 15 minutes unless otherwise specified.

BILLING CODE 4510-26-C

Liver Toxicity

Description of the Health Effects

Although the precise mechanisms by which these compounds cause liver damage are only partly understood, the development and manifestation of liver toxicity are similar for all of them. In general, liver toxicity is a graded response (i.e., the severity of the lesion is directly proportional to the intensity/duration of exposure). Although many of the effects caused by exposure to these substances are reversible, some are not.

Liver damage is not a single entity; the manner in which it is manifested depends upon the dose, duration, and particular chemical agent involved. For example, acute exposures may cause lipid accumulation in the hepatocytes, cellular death, and hepatobiliary dysfunction. In contrast, chronic exposures may lead to cirrhotic changes and the development of neoplasms. Fatty accumulation and necrosis can be either localized or widespread, and chemically induced lesions resulting from chromic exposures can cause marked changes of the entire liver (Plaa 1986/Ex. 1–183).

Typically, the earliest and most sensitive indicators of liver toxicity are alterations in biochemical liver functions, such as changes in specific enzyme activities. These may be accompanied by changes in the morphology of specific organelles in hepatocytes. For example, relatively low doses of halogenated aliphatic hydrocarbons, such as allyl chloride, carbon tetrabromide, and ethylene dichloride, cause an increase in the activity of microsomal mixed-function oxidase enzymes. This increase is ordinarily accompanied by proliferation of the endoplasmic reticulum.

Many compounds that damage the liver, such as 1,1,2,2-tetrachloroethane, also cause an abnormal accumulation of fat, especially of triglycerides, in liver cells. In experimental animals, this effect is manifested as an accumulation of microscopic vacuoles in liver cells. In humans, however, the only grossly detectable manifestation of this effect is increased liver size, which is an indication of severe fat accumulation in the liver.

At sufficiently high doses, most substances that damage the liver cause cell death that leads to tissue necrosis or gangrene. This necrosis may initially be localized, but, at higher or more sustained exposure levels, the entire liver may be involved. Moderate to severe liver necrosis is usually accompanied by increased concentrations of marker enzymes such as glutamate-pyruvate transaminase or glutamate-oxaloacetate transaminase in the serum; the detection of these substances in the serum of exposed individuals can thus be a useful diagnostic tool.

Dose-Response Characteristics

The development of liver and other organ damage in humans and animals is progressive; it begins with subcellular changes, progresses to the cellular level, and is finally manifested as whole-organ damage. This progression is related to the intensity/duration of dose (i.e., as dose increases, cellular death becomes widespread and eventually causes liver dysfunction). The extent to which liver damage is reversible follows a similar continuum; since the liver can regenerate, minor cellular damage or transient disease states are usually reversible if exposure ceases. However, if exposure continues, the capacity of the liver to regenerate is exceeded and permanent damage results. As is the case for some chemically induced toxic effects, there appears to be a NOE level below which hepatotoxic effects do not occur.

The following paragraphs describe OSHA's findings for all of the substances in this group of hepatotoxins and discuss the record evidence and the nature of the material health impairments experienced by exposed workers.

ALLYL CHLORIDE

CAS: 107–05–1; Chemical Formula: $CH_2 = CHCH_2Cl$
H.S. No. 1011

The former OSHA PEL for allyl chloride was a 1-ppm (3-mg/m^3) 8-hour TWA; the proposed PEL was also 1 ppm, with a 15-minute STEL of 2 ppm. NIOSH (Ex. 8–47, Table N1) concurred with the proposed limits. In the final rule, OSHA is establishing an 8-hour TWA limit of 1 ppm and a STEL of 2 ppm for this substance; these limits are consistent with those of the ACGIH. Allyl chloride is a colorless liquid with an unpleasant, pungent odor.

Studies of animal exposures to allyl chloride indicate that this chemical is among the most toxic of the halogenated aliphatic hydrocarbons, producing mucous membrane irritation, mild narcosis, and, at higher concentrations, histologic lesions of the lungs and kidneys (Adams, Spencer, and Irish 1940/Ex. 1–584). Even single exposures lasting only a few minutes at concentrations between 1 and 100 mg/liter (332 to 32,000 ppm) caused mucous membrane irritation in various laboratory animals; at 8-ppm concentrations for five weeks, kidney and liver damage were observed (Adams, Spencer, and Irish 1940/Ex. 1–584). Further animal studies have confirmed liver and kidney pathology in many species (Torkelson, Wolf, Oyen, and Rowe 1959/Ex. 1–691), and female rats exhibited kidney pathology after exposure to 3 ppm for six months.

Exposures of 50 to 100 ppm for five minutes in humans caused eye and nose irritation, and five-minute exposures below 25 ppm have been associated with pulmonary irritation (Shell Chemical Corp. 1974, as cited in Ex. 150). Humans exposed to concentrations of 1 to 113 ppm showed abnormal liver test results (Hausler and Lenich 1968/Ex. 1–1035).

In a posthearing comment (Ex. 150, Comments on Allyl Chloride), NIOSH reported the results of a recent National Cancer Institute monograph (Santodonato et al. 1985, as cited in Ex. 150) showing that allyl chloride is a tumor initiator in mice and a mutagen in bacterial test systems. NIOSH (Ex. 150) and Drs. Grace Ziem and Barry Castleman (Ex. 114A) discussed recent epidemiological and clinical studies from the People's Republic of China (He et al. 1985, as cited in Exs. 114A and 150), which also found toxic polyneuropathy in workers exposed to between 2.6 and 6650 mg/m^3 allyl chloride for durations ranging from 2.5 months to 6 years; in contrast, workers at another facility with allyl chloride exposures below 25 mg/m^3 for 1 to 4.5 years had few neurological disorders, but 50 percent showed abnormal electroneuromyographic results. Animal studies confirm this substance's neuropathic potential (Ex. 114A and Ex. 150, Comments on Allyl Chloride).

The final rule establishes an 8-hour TWA limit of 1 ppm and a STEL of 2 ppm for allyl chloride. The Agency concludes that both TWA and STEL limits are necessary to protect workers from the significant risk of kidney and liver damage and neuropathic effects which constitute material health impairments and are potentially associated with the elevated short-term exposures to ally chloride currently permitted by the 8-hour TWA alone.

CARBON TETRABROMIDE

CAS: 558–13–4; Chemical Formula: CBr_4
H.S. No. 1072

OSHA formerly had no limit for exposure to carbon tetrabromide. The proposed limits were 0.1 ppm as an 8-hour TWA and 0.3 ppm as a 15-minute STEL; the final rule establishes these limits, which are consistent with those of the ACGIH. NIOSH (Ex. 8–47, Table N1) concurred with OSHA's proposed limits for carbon tetrabromide. At room

temperature, pure carbon tetrabromide is a colorless, nonflammable solid. However, samples are usually yellow-brown in color.

Carbon tetrabromide's hepatotoxic effects include both fatty infiltration and necrosis. The 0.1-ppm and 0.3-ppm TWA and STEL levels were selected based on an observed no-effect level of 0.1 ppm; this finding derives from a study in which rats were exposed to carbon tetrabromide by inhalation for seven hours per day, five days per week for six months (Torkelson and Rowe 1981a/Ex. 1–974). Dr. Grace Ziem (Ex. 46) submitted information to OSHA showing that exposure to 0.07 ppm has caused sensory irritation in rats.

The final rule establishes limits of 0.1 ppm as an 8-hour TWA and 0.3 ppm as a 15-minute STEL for carbon tetrabromide. OSHA concludes that establishing these limits for this previously unregulated chemical will protect workers against the significant risk of hepatotoxic effects, which constitute material health impairments.

o-CHLOROSTYRENE

CAS: 2039–87–4; Chemical Formula: C_8H_7Cl
H.S. No. 1089

OSHA formerly had no limit for o-chlorostyrene. The proposed limits were an 8-hour TWA of 50 ppm and a STEL of 75 ppm, and NIOSH (Ex. 8–47, Table N1) concurred with these limits. The final rule establishes a 50-ppm TWA PEL and a 75-ppm STEL, limits that are consistent with those of the ACGIH. o-Chlorostyrene is a colorless liquid at room temperature.

In an unpublished report, the Dow Chemical Company (1973a, as cited in ACGIH 1986/Ex. 1–3, p. 136) describes the results of an o-chlorostyrene inhalation study in rats, rabbits, guinea pigs, and dogs. Dow exposed the animals to an average concentration of 101 ppm for seven hours daily, five days per week, for a total of 130 exposures in 180 days. No adverse effects were observed in any species in terms of appearance, growth, behavior, mortality, hematology, BUN, alkaline phosphatase, SGPT, BSP, organ weights, or gross pathology (Dow Chemical Company 1973a, as cited in ACGIH 1986/Ex. 1–3, p. 136). Microscopic examination of animal tissue revealed a somewhat higher incidence of pathological changes in the liver and kidneys. There is evidence indicating that the warning properties of o-chlorostyrene do not permit workers to be aware of o-chlorostyrene concentrations of 100 ppm. Based on o-chlorostyrene's structural analogy to styrene, for which short-term exposure of 100 ppm have been demonstrated to produce neuropathic and narcotic effects (Stewart, Dodd, Baretta, and Schaffer 1968/Ex. 1–380), OSHA finds that a short-term limit is necessary. OSHA received no comments (other than NIOSH's) on this substance.

The final rule establishes a PEL of 50 ppm as an 8-hour TWA and a 15-minute STEL of 75 ppm for o-cholorostyrene. The Agency concludes that both of these limits will protect workers from the significant risks of liver and kidney damage, narcosis, and neuropathy to which they could potentially be exposed in the absence of any OSHA limit. OSHA finds that these health effects constitute material health impairments and that the TWA and STEL limits will substantially reduce these significant occupational risks.

CYCLOHEXANONE

CAS: 108–94–1; Chemical Formula: $C_6H_{10}O$
H.S. No. 1108

OSHA's former limit for cyclohexanone was 50 ppm as an 8-hour TWA. The Agency proposed to reduce this limit to 25 ppm and to add a skin notation for this substance. NIOSH (Ex. 8–47, Table N1) concurred with this proposed limit. The final rule establishes an 8-hour TWA PEL of 25 ppm and includes a skin notation. Both the ACGIH and NIOSH recommend a time-weighted average for cyclohexanone of 25 ppm, and the ACGIH also recommends a skin notation. Cyclohexanone is a white to pale yellow, oily liquid with an odor similar to that of acetone and peppermint.

Cyclohexanone has been studied in several experimental animal species. A concentration of 2000 ppm inhaled for four hours was lethal to one of six rats; at 4000 ppm, all of the exposed animals died. In rabbits, the dermal LD_{50} was 1000 mg/kg (Smyth, Carpenter, Weil et al. 1969/Ex. 1–442). Rabbits showed marked irritation and some corneal injury when undiluted cyclohexanone was instilled in the eye (Carpenter and Smyth 1946/Ex. 1–859). Guinea pigs exposed to 4000 ppm for six hours showed narcotic symptoms, lacrimation, salivation, depression of body temperature and heart rate, and corneal opacity (Specht, Miller, Valaer, and Sayers 1940/Ex. 1–1179). Rabbits exhibited degenerative changes of the liver and kidneys after 50 daily six-hour inhalation exposures to 190 ppm (Treon, Crutchfield, and Kitzmiller 1943b/Ex. 1–394). Exposures to 309 ppm cyclohexanone on the same regimen caused conjunctival congestion, while exposures to 3000 ppm were lethal to some of the exposed animals (Treon, Crutchfield, and Kitzmiller 1943b/Ex. 1–394).

In humans, Nelson and co-workers (1943/Ex. 1–66) reported that irritation caused by exposure to cyclohexanone was intolerable at 50 ppm; however, 25 ppm was not objectionable to most subjects in three- to five-minute exposures (Nelson, Enge, Ross et al. 1943/Ex. 1–66).

OSHA is adding a skin notation for cyclohexanone based on this substance's ability to cause systemic toxicity through dermal absorption. L.H. Hecker, Director of Corporate Industrial Hygiene and Toxicology at Abbott Laboratories, commented that, in his opinion, there was no evidence for cyclohexanone's dermal toxicity, and thus that no skin notation was necessary (Ex. 3–678). However, OSHA has determined, based on a review of the evidence for this substance, that cyclohexanone has a dermal LD_{50} of 100 mg/kg in rabbits (*Dangerous Properties of Industrial Materials*, 7th ed., p. 831, Sax and Lewis 1989). The Agency believes it appropriate to establish a skin notation for substances posing a percutaneous hazard, which OSHA is defining as any substance having a dermal LD_{50} in rabbits of 1000 mg/kg or less. Accordingly, the Agency is including a skin notation for cyclohexanone in the final rule (see section VI.C.18 for a full discussion of the Agency's policy on skin notations).

In the final rule, OSHA is establishing an 8-hour TWA for cyclohexanone of 25 ppm, with a skin notation. The Agency has determined that these limits will protect workers from the significant risks of liver and kidney damage, skin and respiratory-tract irritation, and percutaneous absorption associated with exposure to this substance. OSHA finds that skin and respiratory-tract irritation and liver and kidney damage all constitute material health impairments.

DIOXANE

CAS: 123–91–1; Chemical Formula: $O(CH_2CH_2)_2O$
H.S. No. 1145

OSHA's former PEL for dioxane was 100 ppm as an 8-hour TWA, with a skin notation. The Agency proposed a 25-ppm 8-hour TWA PEL for this substance, with retention of the skin notation; these limits, which are consistent with those of the ACGIH, are established in the final rule. NIOSH (Ex. 8–47, Table N6A) agreed with the selection of this PEL. Dioxane is a colorless liquid with an ethereal odor.

A two-year drinking water study conducted by the Dow Chemical Company (1972b, as cited in ACGIH 1986/Ex. 1–3, p. 217), in which male and female rats were given water containing

1.0, 0.1, or 0.01 percent dioxane, showed that animals given the highest dose developed liver and nasal tumors, in addition to pathological changes in the liver and kidney. Rats in the 0.1-percent group showed renal tubular sloughing and hepatocellular degeneration but no significant increase in neoplasms. Because this study demonstrated hepato- and nephrotoxic effects at doses 10 times lower than the dose causing cancer in animals, the permissible exposure limit has been set at a level that will prevent dioxane's liver and kidney effects. A study by Torkelson et al. (1974/Ex. 1-111) in four species of animals exposed to multiple daily airborne exposures of dioxane at 50 ppm showed no gross or histopathologic organ changes; this study demonstrates that the 25-ppm level should protect against the risk of liver and kidney effects in exposed workers. Dioxane has been shown in several studies to readily penetrate the skin of humans and animals and cause liver and kidney damage (NIOSH 1977n, p. 151, as cited in ACGIH 1986/Ex. 1-3, p. 218).

NIOSH (Ex. 8-47, Table N6A; Tr. 3-96 to 3-97) concurs with OSHA's exposure limit for dioxane, but notes its cancer potential. The AFL-CIO (Ex. 194, p. A12) also urged OSHA to designate dioxane as a carcinogen, as did the International Chemical Workers Union (Tr. 9-217 to 9-218). IARC (1987) has classified dioxane as a Group 2B (possible human) carcinogen based on a finding of sufficient evidence in animals. OSHA is aware of the emerging literature on dioxane's carcinogenic potential and intends to monitor this substance in the future.

Thomas Robinson, representing Vulcan Chemicals, stated that it was "most appropriate" for OSHA to adopt a TWA limit of 25 ppm for dioxane (Ex. 3-677), and the Halogenated Solvents Industry Alliance also supported OSHA's proposed PELs.

OSHA finds that the evidence for dioxane indicates that it is a liver and kidney toxin at levels substantially lower than those at which it produces a carcinogenic response. The Agency concludes that an 8-hour TWA of 25 ppm for dioxane, with a skin notation, is necessary to protect exposed workers against the significant risks of kidney and liver damage and cancer, all material health impairments that are associated with exposure at levels above the new PELs. OSHA has determined that the 25-ppm TWA limit will substantially reduce this risk.

ETHYLENE DICHLORIDE

CAS: 107-06-2; Chemical Formula: $ClCH_2CH_2Cl$

H.S. No. 1168

The former OSHA standard for ethylene dichloride (EDC) was 50 ppm as an 8-hour TWA, with a 100-ppm STEL (maximum duration of five minutes in any three hours) and a 200-ppm ceiling; these limits were derived from limits recommended by the American National Standards Institute in 1969. In 1980, the ACGIH reduced its TLV for ethylene dichloride to 10 ppm as an 8-hour TWA. NIOSH (1978q/Ex. 1-1120 and Ex. 8-47, Table N6A) has concluded that ethylene dichloride should be considered a potential human carcinogen and has recommended a 1-ppm TWA REL and a 2-ppm 15-minute short-term limit. OSHA proposed an 8-hour TWA of 1 ppm and a STEL of 2 ppm, and the final rule establishes these limits. Ethylene dichloride is a colorless liquid with an odor typical of the chlorinated hydrocarbons.

Several studies indicate that the former OSHA PELs are insufficient to protect workers against the hepatotoxic and carcinogenic effects of exposure to EDC. A paper by Kozik (1957/Ex. 1-182) reported that workers generally exposed to ethylene dichloride at levels of 10 to 15 ppm but occasionally exposed to levels of 40 ppm experienced increased morbidity, diseases of the liver and bile ducts, and nervous system effects. In addition, Brzozowski and associates (1954/Ex. 1-63) reported abnormal changes in the blood of 50 percent of workers (8 of 16) exposed to EDC levels of 10 ppm and above (Brzozowski, Czajka, Dutkiewicz et al. 1954/Ex. 1-63).

Many commenters submitted information to the docket on ethylene dichloride (Exs. 3-624, 3-677, 3-678, 3-741, 3-874, 3-1174, 8-47, and 150). Most commenters were of the opinion that a permissible exposure limit of 10 ppm, rather than the proposed 1-ppm limit, would provide adequate protection against EDC's hepatoxic, central nervous system, and hematopoietic effects (Exs. 3-624, 3-677, 3-678, 3-741, 3-874, and 3-1174). Several of these participants also expressed concern about the feasibility of the 1-ppm limit (Exs. 3-624, 3-741, and 3-874). The comments of Richard Olson, representing the Dow Chemical Company, were typical of those of this group of commenters. According to Mr. Olson, OSHA's proposed limit was based on two outdated studies (Kozik 1957/Ex. 1-182 and Brzozowski, Czajka, Dutkiewicz et al. 1954/Ex. 1-63) that are incomplete, reflect outdated work practices, and present results that are based on effects caused by dermal as well as airborne exposures (Ex. 3-741, p. 52). The Chemical Manufacturers Association (Ex. 3-874) pointed out that the jobs being performed by the workers monitored in the Brzozowski et al. (1954/Ex. 1-63) study are no longer permitted because EPA has prohibited the use of EDC as a fumigant (Ex. 3-874).

In response to these commenters, OSHA notes that there are many studies reporting serious EDC-related effects among workers exposed to airborne concentrations in the 10- to 15-ppm range. For example, the aircraft workers in the Kozik (1957/Ex. 1-182) study (average 8-hour TWA exposures of 10 to 15 ppm) experienced increased morbidity and lost more workdays than did non-EDC-exposed workers at the same factory. These workers experienced high rates of gastrointestinal disease and liver and gallbladder diseases; these symptoms and diseases are typical EDC exposure effects. Another study (Cetnarowicz 1959) examined refinery workers exposed to EDC at levels ranging from 10 to 200 ppm and found that these workers experienced many of the same symptoms as those seen in the aircraft workers. Clinical analyses confirmed that the liver and gastrointestinal tract were the principal target organs affected by EDC exposure. Rosenbaum (1947) also reported that EDC exposures below 25 ppm (not further specified) caused functional nervous system disorders, including headache, insomnia, and fatigue, and also slowed the heartbeat rate in affected workers.

OSHA finds the evidence presented in these studies consistent, biologically plausible, and convincing. Although specific exposure levels and precise industrial hygiene measurements are not available for some of these studies, the weight of the evidence presented demonstrates that occupational exposures to EDC at levels of 10 ppm or somewhat higher (i.e., in the 14- to 15-ppm range) cause severe health effects in specific target organ systems (i.e., the liver and gastrointestinal tract). The symptoms and signs of EDC's effects have been confirmed both clinically (palpitation of enlarged livers, X-ray evidence of pyloric spasms) and by laboratory analysis (elevated urobilinogen levels, positive Takata-Ara liver function tests, negative glucose tolerance tests). Thus, OSHA finds that EDC's hepatotoxic and gastrointestinal effects clearly warrant a reduction in the PEL to levels substantially below the level (10 ppm) shown to cause toxic liver and other effects. In response to the CMA, OSHA agrees that EPA's ban has eliminated the fumigant exposures described in the Brzozowski et al. (1954/Ex. 1-63) study, which involved concomitant dermal exposures.

However, OSHA notes that the dermal LD_{50} in rabbits is in the range of 2.8 to 4.9 g/kg, indicating that EDC is not readily absorbed t hrough the skin in toxic quantities. OSHA therefore finds that, although dermal exposure undoubtedly contributed somewhat to the toxic effects seen in the workers in the Brzozowski et al. (1954/Ex. 1–63) study, airborne exposure was the predominant contributor to these effects.

Some commenters also took issue with OSHA's reference in the proposal to EDC's carcinogenicity. According to these commenters (Exs. 3–677, 3–741, and 3–874), because the NCI bioassay (1978d/Ex. 1–947) in mice and rats involved the use of corn oil as a vehicle, carcinogenic responses may have been enhanced. In addition, because EDC gavage produced greater amounts of the potentially genotoxic glutathione conjugate than did equivalent inhalation doses of EDC, these commenters believe that route of administration may play a critical role in the carcinogenicity of EDC, and thus, that occupational exposures, which are predominantly via inhalation, may not be carcinogenic.

OSHA is aware that inhalation bioassays of EDC did not produce a statistically significant increase in tumors in rats or mice. However, the NCI gavage study (1978d/Ex. 1–947) was positive in rats and mice, and intraperitoneal administration of EDC produced an elevated increase in lung adenomas in strain A mice (*Health Assessment Document (HAD) for 1,2-Dichloroethane (Ethylene Dichloride)*, EPA/600/8–84/006F, p. 1–5, EPA 1985a). Dermal application caused a statistically significant increase in benign lung tumors in mice, although this route did not cause a significant increase in skin tumors. EPA (1985a) concludes that the direct and supporting evidence for the carcinogenicity of EDC includes:

(1) Multiple tumor types in oral bioassays in two species;

(2) Suggestive evidence in two other animal bioassays;

(3) Demonstrated evidence of reactive metabolites and formation of a DNA adduct; and

(4) Evidence that EDC is also a mutagen (EPA 1985a, p. 1–5).

In posthearing comments, NIOSH (Ex. 150, Comments on Ethylene Dichloride) emphasized that the NCI bioassay (NCI 1978d/Ex. 1–947) demonstrated EDC-induced lung neoplasms and lymph system cancers in mice of both sexes, liver cancer in males, and mammary and uterine cancers in females. The AFL–CIO also emphasized EDC's carcinogenicity (Ex. 194). In rats, it produced cancers of the forestomach in males, mammary neoplasms in females, and hemangiosarcomas in animals of both sexes. NIOSH (Ex. 150, Comments on Ethylene Dichloride) concluded its comments by quoting the summary of the IARC (1979b, as cited in Ex. 150) monograph on EDC:

> There is sufficient evidence that ethylene dichloride is carcinogenic in mice and rats. In the absence of adequate data in humans, it is reasonable for practical purposes to regard ethylene dichloride as if it presented a carcinogenic risk to humans.

In regard to the technological feasibility of achieving a 1-ppm limit for EDC, the Chemical Manufacturers Association (CMA) states that

> uniform compliance with the proposed PEL will not be achieved. Manufacturing operations appear to be able to meet a 10-ppm, 8-hour TWA PEL for many routine operations. However, maintenance tasks, sampling, and loading operations will have difficulty meeting a 10-ppm PEL (Ex. 3–874, p. 3).

Both the Vinyl Institute (Ex. 3–624) and the Dow Chemical Company (Ex. 3–741) share the CMA's view on the feasibility of achieving the 1-ppm limit. However, OSHA notes that ethylene dichloride is manufactured and used in closed systems (Ex. 3–874) and that 90 percent of all EDC produced in this country is used captively by the producers themselves (84 percent of all EDC produced in the United States is used to make vinyl chloride monomer) (EPA 1985a, p. 1–1). Emissions from closed systems, which include fugitive emissions from process equipment such as pumps, seals, and flanges; emissions during process sampling; emissions during loading operations; and emissions during maintenance operations, are all readily amenable to control through the use of engineering methods or improved work practices. For example, implementation of a rigorous schedule of manual leak detection and repair, the use of sampling bombs or ventilated sampling ports, the use of loading arms for closed-hatch loading of EDC into railcars and tank trucks, installation of vapor return lines or vapor recovery systems on loading docks, and installation of improved maintenance procedures are all inexpensive and effective methods of controlling fugitive emissions from process machinery. In addition, because of the intermittent, nonroutine, and varied nature of maintenance operations, OSHA typically permits the use of respirators during the performance of maintenance tasks. OSHA is also cognizant of the potential for feasibility problems in loading and sampling operations. The Agency will consider the use of respirators for these operations on a case-by-case basis or, as appropriate, on a sector-by-sector basis. However, OSHA finds that EDC producers will generally be able to achieve the 1-ppm 8-hour TWA and the 2-ppm short-term limit by using readily available control technologies and implementing additional work practices.

The Agency concludes that an 8-hour TWA of 1 ppm and a 15-minute STEL of 2 ppm are necessary to protect workers against the significant risks of liver damage, gastrointestinal toxicity, and cancer, all material health impairments that are associated with exposure to ethylene dichloride. OSHA further concludes that the revised limits will substantially reduce these significant occupational risks.

HYDRAZINE

CAS: 302–01–2; Chemical Formula: H_2N-NH_2
H.S. No. 1205

The former OSHA limit for hydrazine was 1 ppm as an 8-hour TWA, with a skin notation. OSHA proposed an 8-hour TWA PEL of 0.1 ppm, also with a skin notation, and the final rule establishes this limit. Hydrazine is an odorless, fuming, oily liquid with an ammonia-like odor. Because of hydrazine's potential carcinogenic hazard, NIOSH (1978e/Ex. 1–263; Ex. 8–47) has recommended that workplace exposures to hydrazine not exceed 0.03 ppm, as determined by a two-hour air sample; this level represents the lowest detectable concentration over this sampling period.

A hepatotoxic response in mice and anemia and weight loss in dogs were reported to occur following a six-month exposure to 1 ppm of hydrazine for six hours per day, five days per week, or to 0.2 ppm continuously (Haun and Kinkead 1973/Ex. 1–824). The ACGIH has assigned an A2 designation (suspect human carcinogen) to hydrazine, based on a study by MacEwen, Vernot, and Haun (1979/Ex. 1–193) showing significant increases in nasal tumors in rats exposed to 1 or 5 ppm hydrazine, in thyroid adenocarcinomas in rats exposed to 5 ppm, and in lung adenomas among mice exposed to 1 ppm. NIOSH (1978e/Ex. 1–263) cites studies that demonstrate the carcinogenicity of hydrazine in rodents by a variety of dose routes. NIOSH (Ex. 8–47, Table N6B) believes that hydrazine should be labelled a potential occupational carcinogen. Based on sufficient evidence of hydrazine's carcinogenicity in animals, IARC (1987) classified hydrazine as a Group 2B (possible human) carcinogen.

The animal studies conducted by Haun and Kinkead (1973/Ex. 1–824) and by MacEwen, Vernot, and Haun (1979/

Ex. 1–193) clearly demonstrate that exposure to hydrazine at the former 1-ppm PEL presents a significant risk of respiratory cancer, liver disease, and adverse blood effects; animals exposed to airborne concentrations at this level have exhibited all of these responses. Reported dermal LD_{50}s in rabbits and dogs were 91 and 90 mg/kg, respectively, showing that hydrazine can readily penetrate the skin and cause systemic effects.

Some commenters (Ex. 8–16, 194, Tr. 9–218; Tr. 3–309) misunderstood the classification scheme used by OSHA to group substances in the proposal and commented that, in their opinion, hydrazine should have been classified as a carcinogen rather than a hepatotoxin. However, as discussed in other sections of the preamble, OSHA did not intend this classification scheme to have regulatory implications but to facilitate generic rulemaking. OSHA's approach was to classify substances in accordance with the health effect on which the ACGIH has based its TLV. In response to the American Industrial Hygiene Association's question about a risk assessment for hydrazine, OSHA notes that, in this rulemaking, OSHA has performed risk assessments only for some of the substances classified in Section VI.C.15 of the preamble.

The Agency is establishing an 8-hour TWA PEL of 0.1 ppm, with a skin notation, for hydrazine. OSHA concludes that this limit will substantially reduce the significant risks of cancer, liver disease, and hematopoietic effects, all clearly material impairments of health, that have been demonstrated to occur in animals at exposures about the revised PEL.

METHYLCYCLOHEXANOL

CAS: 25639–42–3; Chemical Formula: $CH_3C_6H_{10}OH$
H.S. No. 1269

OSHA formerly had an 8-hour TWA limit of 100 ppm for methylcyclohexanol. The Agency proposed a limit of 50 ppm TWA for this substance, and is establishing this limit in the final rule. NIOSH (Ex. 8–47, Table N1) concurred with OSHA's proposed limits for methylcyclohexanol. Methylcyclohexanol is a colorless, viscous liquid with an aromatic odor, and usually exists as a mixture of isomers in which the meta and para forms predominate.

Exposure to methylcyclohexanol produces liver and kidney impairment, narcotic effects, and eye and respiratory irritation. Treon, Crutchfield, and Kitzmiller (1943a/Ex. 1–393) have reported the oral LD_{50} in rabbits to be between 1.25 and 2 g/kg; liver damage was observed in surviving animals. Repeated inhalation exposures to the vapor caused salivation, eye irritation, and lethargy in rabbits exposed at 500 ppm, but exposures to 230 ppm caused no observable effects. Fifty 6-hour exposures at a level of 120 ppm caused microscopic changes in the liver and kidney tissue of rabbits (Treon, Crutchfield, and Kitzmiller 1943b/Ex. 1–394).

In humans, headaches and eye and respiratory irritation have been reported to occur following prolonged exposures to high concentrations of methylcyclohexanol (Fillipi 1914, as cited in ACGIH 1986/Ex. 1–3, p. 385). Smyth (1956/Ex. 1–759) considered an exposure limit of 100 ppm to be sufficiently low to prevent narcotic effects and, perhaps, significant liver or kidney damage. OSHA received no comments (other than NIOSH's) on this substance.

The Agency is establishing an 8-hour TWA of 50 ppm for methylcyclohexanol. OSHA concludes that this limit will protect workers against the significant risks of hepatic and renal damage and narcosis, which constitute material health impairments and are associated with exposures to this substance at levels above the revised PEL. The Agency finds that the revised limit will substantially reduce these risks.

OCTACHLORONAPHTHALENE

CAS: 2234–13–1; Chemical Formula: $C_{10}Cl_8$
H.S. No. 1295

OSHA formerly had a limit of 0.1 mg/m³ TWA, with a skin notation, for octachloronaphthalene. The Agency proposed to retain the 8-hour TWA and to add a STEL of 0.3 mg/m³, also with a skin notation, for this substance, and NIOSH (Ex. 8–47, Table N1) concurred. These limits are established in the final rule. Octachloronaphthalene is a nonflammable, pale yellow, waxy solid containing 70 percent chlorine.

Inhalation toxicity data for octachloronaphthalene fumes or dust are lacking, but exposure to the chloronaphthalenes causes acne-like lesions that itch severly. Repeated exposures to the fumes of molten chlorinated naphthalenes can cause severe and sometimes fatal systemic poisoning and are especially damaging to the liver (Patty 1963g/Ex. 1–845). Ingestion studies of cattle have shown different toxicities for different naphthalenes, with toxicity increasing with the compound's degree of chlorination (Sikes, Wise, and Bridges 1952/Ex. 1–804). However, these data are controverted by another report in which octachloronaphthalene was found to be less toxic than the hexachloro derivative (Bell 1953/Ex. 1–951). This divergence in the data may be due to differing methods of administration (suspension versus solution), or may reflect the soluble form's greater capacity for absorption (ACGIH 1986/Ex. 1–3, p. 447). NIOSH was the only submitter of comments specifically relating to octachloronaphthalene.

In the final rule, OSHA is retaining the 8-hour TWA PEL of 0.1 mg/m³ and adding a STEL of 0.3 mg/m³, with a skin notation, for octachloronaphthalene. The Agency concludes that this combined limit will protect workers against the significant risks of serious liver damage and dermal lesions, which constitute material health impairments and are associated with exposure to this substance at the elevated levels permitted by an 8-hour limit alone. The skin notation is retained because of octachloronaphthalene's demonstrated ability to cause systemic toxicity by percutaneous absorption.

PROPYLENE DICHLORIDE

CAS: 78–87–5; Chemical Formula: $CH_3CHClCH_2Cl$
H.S. No. 1341

OSHA's former limit for propylene dichloride was 75 ppm as an 8-hour TWA. The proposal retained the 75-ppm TWA and added a STEL of 110 ppm, and these are the limits being promulgated in the final rule. Propylene dichloride is a colorless, flammable, mobile liquid with an odor like that of chloroform.

The primary hazards associated with exposure to propylene dichloride are inhalation-induced toxicity to liver tissue and skin and eye irritation. Repeated inhalation exposures to 1000 ppm have been reported to kill dogs (after 24 exposures), guinea pigs (after 22 exposures), and rats (in some cases after only seven exposures); however, some animals survived more than 100 seven-hour exposures. Necropsy showed severe liver damage; the hepatotoxicity of propylene dichloride appears to be greater than that of carbon tetrachloride and less than that of ethylene dichloride (Heppel, Neal, Highman, and Porterfield 1946/Ex. 1–510). Animals of these same species (rats, dogs, and guinea pigs) survived 128 to 140 seven-hour exposures to 400 ppm propylene dichloride for five days/week without histologic effects, while mice died from similar exposures; surviving mice displayed hepatomas (Heppel, Highman, and Peake 1948/Ex. 1–605). The oral LD_{50} for rats has been reported as 1.19 ml/kg (Smyth, Carpenter, Weil et al. 1969/Ex. 1–442); the acute 8-hour

inhalation LC_{50} for rats is 3000 ppm (Pozzani, Weil, and Carpenter 1959/Ex. 1–608). NIOSH (Ex. 150A, Comments on Propylene Dichloride) noted that an NTP (1986c) bioassay showed some evidence that propylene dichloride was carcinogenic in mice and caused an increased incidence of hepatocellular adenomas; NIOSH indicated (Ex. 8–47, Table N6B) that a separate 6(b) rulemaking might be appropriate for this substance. The finding of tumors was not reproduced in rats, in that female rats showed only a marginally increased incidence of mammary adenocarcinomas, and male rats showed no response. NIOSH was the only commenter on propylene dichloride.

In the final rule, OSHA is retaining the 8-hour TWA PEL for propylene dichloride of 75 ppm and adding a 15-minute STEL of 110 ppm. The Agency concludes that this combined limit will protect workers against the significant risks of serious hepatotoxic effects, which constitute material impairments of health, that are associated with exposures at the elevated levels permitted by the absence of a short-term limit. OSHA finds that the TWA and short-term PELs will act together to reduce this risk substantially.

1,1,2,2-TETRACHLOROETHANE

CAS: 79–34–5; Chemical Formula: $CHCl_2CHCl_2$
H.S. No. 1385

OSHA's former PEL for 1,1,2,2-tetrachloroethane was 5 ppm as an 8-hour TWA, with a skin notation; a 1-ppm 8-hour TWA, also with a skin notation, was the level proposed by OSHA. NIOSH considers 1,1,2,2-tetrachloroethane to be a potential carcinogen but concurred with the limit proposed (Ex. 8–47, Table N6A). The final rule establishes a PEL of 1 ppm. TWA and retains the skin notation for this colorless, nonflammable, heavy, mobile liquid with a sweet, chloroform-like odor.

One study by Jeney, Bartha, Kondor, and Szendrei (1957, as cited in ACGIH 1986/Ex. 1–3, p. 561) revealed identifiably adverse effects on the liver, including hepatitis, in humans exposed to concentrations of tetrachloroethane ranging from 1.5 to 247 ppm; liver damage was still evident after exposures were reduced to 15 ppm. An animal study by Schmidt, Binnewies, Gohlke, and Rothe (1972/Ex. 1–222) found "barely detectable" fatty infiltration of the liver in rats exposed to 2 ppm tetrachloroethane for 11 months.

The ACGIH (1986/Ex. 1–3, p. 561) cites some early studies that show that tetrachloroethane penetrates human skin; one fatality has been attributed to excess skin absorption. The New Jersey Department of Public Health (Ex. 144) urged OSHA to set the PEL for this substance on the basis of EPA's IRIS data. The use of IRIS data is discussed in Section VI.A.

Based on this evidence, OSHA concludes that the former permissible exposure limit does not protect exposed workers against fatty infiltration of the liver or against more serious liver damage; these health consequences clearly constitute material health impairments and thus pose a significant occupational risk. OSHA finds that reducing the 8-hour TWA for 1,1,2,2-tetrachloroethane to 1 ppm will substantially reduce this significant risk, and in the final rule, OSHA is therefore establishing a 1 ppm 8-hour TWA, with a skin notation, for 1,1,2,2-tetrachloroethane.

1,2,3-TRICHLOROPROPANE

CAS: 96–18–4; Chemical Formula: $CH_2ClCHClCH_2Cl$
H.S. No. 1407

OSHA's former PEL for 1,2,3-trichloropropane was 50 ppm as an 8-hour TWA, and the proposed limit was a 10 ppm TWA with a skin notation. NIOSH (Ex. 8–47, Table N6A) concurred with the proposed limit but indicated that it considers this substance to be a potential human carcinogen. The final rule establishes the 10 ppm TWA but does not include a skin notation. 1,2,3-Trichloropropane is a colorless to straw-colored, combustible liquid with an odor similar to that of chloroform.

1,2,3-Trichloropropane is not irritating to intact skin; it is also not readily absorbed through the skin. The dermal LD_{50} in rabbits is 1770 mg/kg (*Dangerous Properties of Industrial Materials*, 7th ed., p. 173, Sax and Lewis 1989). However, 1,2,3-trichloropropane is highly irritating to the eyes (Smyth, Carpenter, Weil et al. 1962/Ex. 1–441). Five of six rats exposed to 1000 ppm died after four-hour exposures. Rats and guinea pigs exposed at 800, 2100, or 5000 ppm for 30 minutes showed central nervous system depression, which progressed, at the higher exposure levels, to narcosis and convulsions (Lewis 1979, as cited in ACGIH 1986/Ex. 1–3, p. 601). Several mice exposed for 20 minutes to 5000 ppm died, some as long as several days later, from liver damage. Daily 10-minute exposures at 2500 ppm for 10 days killed 7 of 10 mice (McOmie and Barnes 1949, as cited in ACGIH 1986/Ex. 1–3, p. 601). Animals exposed once for four hours to 1,2,3-trichloropropane at concentrations of 125, 340, 700, or 2150 ppm showed dose-related signs of irritation, which included, at 700 or 2150 ppm, labored respiration, inactivity, and eye and nose irritation; at autopsy, however, no organ or other damage was apparent (McOmie and Barnes 1949, as cited in ACGIH 1986/Ex. 1–3, p. 601).

Drew, Patel, and Lin (1978/Ex. 1–313) noted changes in liver enzymes after a single four-hour exposure to 500 ppm, and Russian studies indicate that morphologic changes and metabolic lesions of the liver, kidney, and lungs occurred in mice exposed continuously to 1,2,3-trichloropropane concentrations of 0.007 to 0.3 ppm (Sidorenko, Tsulaya, Bonashevskaya, and Shaipak 1979/Ex. 1–669; Sidorenko, Tsulaya, Koreneveskaya, and Bonashevskaya 1976/Ex. 1–668; Tsulaya, Bonashevskaya, Zykova et al. 1977/Ex. 1–450.

A National Toxicology Program (NTP) prechronic study, in which rats were gavaged daily with 1,2,3-trichloropropane at 8, 16, 32, 63, 125, and 250 mg/kg body weight for 120 days, showed good survival in all but the highest dose group (NTP 1983a, as cited in ACGIH 1986/Ex. 1–3, p. 602). Statistically significant changes in the liver and kidneys, as well as necrosis and irritation of the nasal passages, occurred in the 63- and 125-mg/kg dose groups. Decreases in red blood cell counts and hematocrits were also seen, even in the 16-mg/kg dose group. 1,2,3-Trichloropropane did not affect testicular weight, sperm count, or morphology. The NTP found this substance to be genetically active in three bioassays. Hardin, Bond, Sikov et al. (1981/Ex. 1–699) did not find 1,2,3-trichloropropane to be fetotoxic or teratogenic.

Human volunteers found exposure to 1,2,3-trichloropropane objectionable because of eye and upper respiratory tract irritation, and many found 50 ppm an unacceptable level for a full-shift exposure (Silverman, Schulte, and First 1946/Ex. 1–142).

The Agency has determined that 1,2,3-trichloropropane's dermal toxicity is not such as to warrant a skin notation; OSHA's reasoning in regard to skin notations is discussed in Section VI.C.18 of this preamble.

In the final rule, OSHA is establishing a 8-hour PEL of 10 ppm for 1,2,3-trichloropropane. The Agency concludes that the 10-ppm 8-hour TWA limit is necessary to protect workers against the significant risks of liver and kidney damage and eye and throat irritation, all of which constitute material health impairments that are potentially associated with exposures to this

substance at levels above the revised PEL.

Kidney Toxicity

Introduction

Kidney damage is the basis for revising the PELs for five of the compounds in this group. These compounds, their CAS and HS numbers, and their former, proposed, and final rule PELs, are shown in Table C4–2. Three of these substances will be regulated by OSHA for the first time, and in the other two cases, the 8-hour TWA will be reduced. In one of the latter cases, a STEL will also be added.

Description of the Health Effects

The precise mechanism by which these chemicals damage the kidneys is unknown. Typically, these compounds are selectively toxic to cells in the renal tubules, perhaps because impaired transport causes the chemical to collect in these cells. In addition to its function in the excretion of wastes, the kidney plays an important role in the regulation of total body homeostasis. This organ regulates extracellular volume, controls electrolyte and acid-base balance, and forms several hormones that control systemic metabolism. Depending on their particular site of action, nephrotoxicants can interfere with hydration, the proper excretion of the body's wastes, electrolytic balance, metabolism, or the maintenance of correct acid-base balances.

Like the hepatotoxic effects previously described, the least severe lesions caused by nephrotoxic compounds are graded and reversible. The earliest changes are usually alterations in the activities of specific enzymes in the tubular cells. These changes may be accompanied by minor morphological alterations of the cells that are visible only with an electron microscope. Higher doses or more sustained exposures are required to cause cellular necrosis that might be visible with light microscopy. Because of the reserve capacity of the kidneys, a significant degree of tubular cell necrosis must occur before it is reflected by measurable alterations in kidney function. Thus, indicators of impaired renal function that can be measured in humans, such as proteinuria, glucosuria, and increased BUN, are relatively insensitive indicators of kidney damage. Other indicators of significant kidney damage include increased kidney weight, swelling of the tubular epithelium, fatty degeneration of tubular epithelium, and the presence of tubular casts in the urine.

Dose-Response Characteristics

Kidney damage, like liver damage, is progressive; only at the earlier stages are nephrotoxic effects reversible. With continued exposure, the damage becomes more extensive, until it reaches the point at which it cannot be repaired. The toxicity of the kidney-damaging chemicals included in this group also increases as dose increases. For most nephrotoxins, there appears to be a NOEL. Workplace exposures to concentrations of these substances at levels at or below the revised limits are unlikely to cause kidney effects in most workers. OSHA has determined that the nephrotoxic risks being protected against are significant at the former PELs; kidney damage constitutes a material health impairment within the meaning of the Act.

1,3-DICHLOROPROPENE

CAS: 542–75–6; CHEMICAL FORMULA: $CHCl=CH-CH_2Cl$
H.S. No. 1129

OSHA formerly had no limit for 1,3-dichloropropene. The Agency proposed an 8-hour TWA of 1 ppm, with a skin notation, for this straw-colored, clear liquid with a chloroform-like odor. NIOSH (Ex. 8–47, Table N6A) concurred with the proposed limit, which is established in the final rule. This compound occurs in two forms: cis- and trans-isomers.

In male and female rats, the acute oral LD_{50}s for a 92-percent mixture of the cis- and trans-isomers of 1,3-dichloropropene were 713 and 470 mg/kg, respectively; postmortem examination showed liver and kidney damage and evidence of possible lung injury (Torkelson and Oyen 1977/Ex. 1–532). The dermal LD_{50} in rabbits for a 92-percent undiluted mixture was 504 mg/kg, but a 10-percent solution administered by gavage at a dose of 125 or 250 mg/kg was lethal to some of the animals (Torkelson and Oyen 1977/Ex. 1–532). Contact with the liquid was irritating to the eyes and skin of rabbits (Torkelson and Oyen 1977/Ex. 1–532).

Inhalation exposures to 1,3-dichloropropene vapor concentrations above 2700 ppm produced eye and nasal irritation and severe lung, nasal, kidney, and liver damage in rats (Torkelson and Oyen 1977/Ex. 1–532). Exposure to 1000 ppm caused eye and nasal irritation, lacrimation, and, if prolonged, unconsciousness; rats exposed to 1000 ppm for two hours died, but those exposed for one hour survived (Torkelson and Oyen 1977/Ex. 1–532). Guinea pigs exposed to 400 ppm for a single seven-hour period died, while rats exposed similarly survived but had obvious lung congestion (Torkelson and Oyen 1977/Ex. 1–532). Rats, rabbits, guinea pigs, and dogs were exposed seven hours/day, five days/week for six months to 1-ppm or 3-ppm concentrations of 1,3-dichloropropene (Torkelson and Oyen 1977/Ex. 1–532). No adverse effects were observed in any of the animals exposed at 1 ppm. Of the animals exposed at 3 ppm, only male rats showed adverse effects; these animals had reversible cloudy swelling of the renal tubular epithelium (Torkelson and Oyen 1977/Ex. 1–532).

In humans, acute exposures to 1,3-dichloropropene cause skin, eye, and respiratory irritation (Torkelson and Oyen 1977/Ex. 1–532). There are no data on the effects in humans of chronic exposure to this substance. NIOSH (Ex. 8–47, Table N6A; Tr. 3–96 to 3–97) concurs with the limits being established by OSHA but notes that 1,3-dichloropropene could be classified as a potential occupational carcinogen. The New Jersey Department of Public Health urged OSHA to derive a PEL for this substance based on EPA's IRIS data. The use of such data is discussed in Section VI.A.

OSHA is establishing an 8-hour TWA limit of 1 ppm, with a skin notation, for 1,3-dichloropropene. The Agency concludes that this limit will protect workers against the significant risks of eye and mucous membrane irritation and lung, kidney, and liver damage, all of which constitute material health impairments that are associated with exposure to this substance. A skin notation is established to protect against 1,3-dichloropropene's ability to cause systemic toxicity when absorbed through the skin.

DICYCLOPENTADIENE

CAS: 77–73–6; Chemical Formula: $C_{10}H_{12}$
H.S. No. 1132

OSHA had no former limit for dicyclopentadiene (DCPD); the proposed limit was a TWA of 5 ppm, and NIOSH (Ex. 8–47, Table N1) concurred with this limit. The final rule establishes a 5-ppm 8-hour TWA PEL for this substance, which is consistent with the ACGIH's limit. DCPD is a solid at room temperature and has a disagreeable odor.

The health effects associated with exposure to DCPD include mild eye, skin, and respiratory irritation, as well as renal damage and possible pulmonary damage. By the oral and intrapertioneal routes, DCPD is extremely toxic, with an oral LD_{50} value of 0.35 ml/kg and an intrapertioneal LD_{50} value of 0.31 ml/kg in rats; rat fatalities occurred within 60 minutes of exposure to an unspecified

concentration of the saturated vapor (Kinkead, Pozzani, Geary, and Carpenter 1971/Ex. 1–606). However, Gage (1970/Ex. 1–508) regards approximately 660 ppm as the 4-hour LC_{50} in rats and reports that 10 six-hour daily exposures to DCPD at a concentration of 250 ppm were survived only by three of four rats; when the animals were subjected to a concentration of 100 ppm for 15 similar exposures, all survived (Gage 1970/Ex. 1–318). Although other species were less susceptible than mice to the effects of DCPD exposure, they exhibited eye irritation, incoordination, and convulsions preceding death (Kinkead, Pozzani, Geary, and Carpenter 1971/Ex. 1–606).

Kinkead and associates (1971/Ex. 1–606) report that rats exposed repeatedly for 10 days survived concentrations of 72 or 146 ppm but succumbed at the 332-ppm level, with convulsions, lung hemorrhage, and blood in the intestines; female rats also suffered hemorrhage of the thymus. Mice similarly exposed succumbed at all three concentration levels (Kinkead, Pozzani, Geary, and Carpenter 1971/Ex. 1–606). Chronic exposures of seven hours/day for 89 days produced kidney damage and some pulmonary effects in rats exposed at levels of 35 and 74 ppm; the no-effect level for these endpoints in rats was determined to be below 19.7 ppm. Dogs exposed at concentrations of 9, 23, or 32 ppm on the same regimen exhibited only minimal effects (Kinkead, Pozzani, Geary, and Carpenter 1971/Ex. 1–606).

Human sensory response tests resulted in findings of mild eye and throat irritation within seven minutes' exposure to 1 ppm DCPD vapor, and of olfactory fatigue within 24 minutes; a 30-minute exposure to 5.5 ppm produced no olfactory fatigue (ACGIH 1986/Ex. 1–3, p 194). Subjective complaints of headache during the first two months of occupational exposure disappeared during the following three months of exposure, suggesting a developed tolerance for this substance (ACGIH 1986/Ex. 1–3. p. 194). No comments (other than NIOSH's) on this substance were received.

OSHA is establishing an 8-hour TWA PEL of 5 ppm TWA for dicyclopentadiene. The Agency concludes that this limit will protect workers against the significant risks of kidney injury, pulmonary effects, and irritation, which constitute material health impairments that are associated with workplace exposure to DCPD at levels above the new PEL.

ETHYL SILICATE

CAS: 78–10–4 Chemical Formula: $Si(OC_2H_5)_4$

H.S. No. 1166

OSHA's former permissible exposure limit for ethyl silicate was 100 ppm as an 8-hour TWA. The proposal included a limit of 10 ppm TWA for this colorless, flammable liquid with a faint odor; NIOSH (Ex. 8–47, Table N1) agreed with the selection of this limit. In the final rule, a PEL of 10 ppm is established for ethyl silicate; this limit is consistent with that of the ACGIH.

Ethyl silicate has been reported to cause both irritation and systemic toxicity. In guinea pigs and rats, a 60-minute exposure of 2000 ppm was reported as the maximal duration/concentration that did not cause serious disturbances; 500 ppm was the maximal no-effect exposure level for an exposure of several hours' duration (Smyth and Seaton 1940b/Ex. 1–376). Thirty-day exposures to 400 ppm ethyl silicate for seven hours/day caused significant mortality in rats and damage to the lungs, liver, and kidney in the surviving animals. Exposures of rats, guinea pigs, and mice to 88, 50, or 23 ppm for 90 days (seven hours/day, five days/week) resulted only in decreased kidney weights in mice exposed at the 88-ppm level (Pozzani and Carpenter 1951/Ex. 1–166). In another study, Kasper, McCord, and Fredrick (1937/Ex. 1–1155) showed that animals exposed to 164 ppm ethyl silicate for 17 eight-hour days showed less weight gain than did controls. Rowe and associates (1948/Ex. 1–359) reported that three 7-hour exposures at 1000 ppm were fatal to 4 of 10 rats; similar exposures to 500 ppm caused pronounced kidney changes and slight lung irritation. Four to 10 similar exposures at 250 ppm caused slow weight loss and some lung and renal changes; at 125 ppm, slight to moderate kidney damage was observed (Rowe, Spencer, and Bass 1948/Ex. 1–359). Smyth and Seaton (1940b/Ex. 1–376) reported that exposure to a concentration of 1200 ppm causes lacrimation in humans and that 250 ppm causes eye and nose irritation. Only NIOSH submitted comments to the rulemaking record on ethyl silicate.

OSHA is establishing a PEL for ethyl silicate of 10 ppm as an 8-hour TWA. The Agency concludes that this limit is required to protect workers from the significant risk of renal damage, which constitutes material health impairment, that is associated with exposures to this substance at concentrations above the revised PEL. OSHA finds that this reduced limit will substantially reduce this risk.

HEXACHLOROBUTADIENE

CAS: 87–68–3; Chemical Formula: $CCl_2=CCl-CCl=CCl_2$

H.S. No. 1195

OSHA had no former limit for hexachlorobutadiene (HCBD); the proposal included a PEL of 0.02 ppm and a skin notation for this substance. NIOSH (Ex. 8–47, Table N6A) supported the selection of this limit. the ACGIH recommends a TLV–TWA of 0.02 ppm with a skin notation and classifies this substance as a suspected human carcinogen (A2). The final rule establishes an 8-hour TWA of 0.02 ppm but does not include a skin notation. Hexachlorobutadiene is a heavy, clear liquid.

Hexachlorobutadiene has a moderate-to-high acute oral toxicity. The LD_{50}s reported for mice, rats, and guinea pigs are 87, 350, and 90 mg/kg, respectively (Murzakev 1963, as cited in ACGIH 1986/Ex. 1–3, p. 298). Gul'ko and co-workers (1964/Ex/ 1–1082) reported LD_{50} values of 116 mg/kg for mice and 270 mg/kg for rats (Gul'ko, Zimina, and Shroit 1964/Ex. 1–1082). The dermal LD_{50} in rabbits is 1211 kg/mg *Dangerous Properties of Industrial Materials*, 6th ed., p. 2145, Sax 1984). A single exposure of 133 to 150 ppm via inhalation has been fatal in rats when the exposure lasts for four to seven hours. All rats survived exposures at 161 ppm for 0.88 hour or 34 ppm for 3.3 hours; similar exposure of guinea pigs and cats to the same concentrations resulted in the death of most animals (Kociba, Schwetz, Keyes et al. 1977/Ex. 1–494). Another inhalation study in rats showed eye and nose irritation, respiratory difficulty, and damage to kidney tissue and the adrenal cortex after two 4-hour exposures at 250 ppm; twelve 6-hour exposures to 100 ppm caused eye and nose irritation, respiratory difficulty, weight loss, anemia in the female animals, and kidney and adrenal damage; fifteen 6-hour exposures at 25 ppm caused retarded weight gain in females, respiratory difficulty, and kidney damage; fifteen 6-hour exposures at 10 ppm caused retarded weight gain in females but no systemic injury; and fifteen 6-hour exposures at 5 ppm resulted in no adverse effects (Gage 1970/Ex. 1–318).

Reproductive studies in male and female rats demonstrated multiple toxicological effects, including kidney damage in both sexes and increased liver weight in males, at the high-dose level of 20 mg/kg/day. Dietary administration of 20, 2, or 0.2 mg/kg daily had no effect on conception percentages, gestational survival, neonatal survival, neonatal sex ratios, neonatal morphology, or neonatal body weights (except for the high-dose

neonates) (Schwetz, Smith, Humiston et al. 1977/Ex. 1–368). Results of lifetime dietary studies suggest that the no-effect level for hexachlorobutadiene in rats is 0.2 mg/kg/day, that a clear dose-response relationship exists for HCBD-induced toxicity affecting primarily the kidney, and that carcinogenic effects (i.e., renal neoplasms) result from ingestion of 20 mg/kg/day (Kociba, Schwetz, Keyes et al. 1977/Ex. 1–494). These authors also reported that HCBD-induced neoplasms occur only at HCBD doses higher than those causing discernible renal injury. The ACGIH states that "HCBD would seem to qualify as a carcinogen of intermediate potency" (ACGIH 1986/Ex. 1–3, p. 299). NIOSH (Ex. 8–47, Table N6A) concurs with the limit being established by OSHA and notes that this substance could be classified as a potential occupational carcinogen.

OSHA is not including a skin notation in the final rule. This decision is based on the Agency's policy in the matter of skin notations (see Section VI.C.18 of the preamble for a discussion of this issue). OSHA is establishing an 8-hour TWA limit of 0.02 ppm for this hazardous substance. Assuming a 10-m^3 per day breathing volume per 8-hour workshift and a 70-kg body weight for humans, this limit corresponds to a daily hexachlorobutadiene intake of approximately 0.03 mg/kg, which is about 10 times below the observed no-effect level in rats fed hexachlorobutadiene. The Agency concludes that this 0.02-ppm limit will protect workers exposed to HCBD from the significant risks of kidney damage; eye, skin, and pulmonary irritation; and renal neoplasms, all of which constitute material health impairments that are associated with exposure to HCBD at levels above the new limit.

HEXONE (METHYL ISOBUTYL KETONE)

CAS: 108–10–1; Chemical Formula: $CH_3COCH_2CH(CH_3)_2$
H.S. No. 1203

OSHA's former 8-hour TWA limit for hexone (methyl isobutyl ketone), or MIBK, was 100 ppm. The ACGIH has established a TLV-TWA of 50 ppm and a 15-minute STEL of 75 ppm for this substance. NIOSH recommends a TWA of 50 ppm for MIBK, which is a clear liquid with a characteristic ketone odor. OSHA proposed a 50-ppm 8-hour TWA and a 75-ppm STEL, and the final rule establishes these limits. NIOSH (Ex. 8–47, Table N1) concurred with the Agency's selection of these limits.

A four-hour exposure to 4000 ppm MIBK killed all exposed rats, but a similar exposure to 2000 ppm was not fatal to these animals (Smyth, Carpenter, and Weil 1951/Ex. 1–439). Guinea pigs exposed to a MIBK concentration of 10,000 ppm immediately showed signs of irritation (Specht, Miller, Valaer, and Sayers 1940/Ex. 1–1179).

MacEwen, Vernot, and Haun (1971/Ex. 1–194) exposed rats, mice, dogs, and monkeys to 100 or 200 ppm MIBK for two weeks and noted no signs of intoxication; however, rats exposed to 100 ppm had heavier kidneys and higher kidney-to-body-weight ratios, and, at 200 ppm, livers were heavier as well. Postmortem examination revealed nephrosis of the proximal tubules.

The same authors (MacEwen, Vernot, and Haun 1971/Ex. 1–194), exposed rhesus monkeys, dogs, and rats continuously for 90 days to MIBK concentrations of 100 ppm. These authors observed no significant changes in clinical chemistry or blood test results, although the rats had heavier kidneys and livers, reversible hyaline droplet degeneration of the proximal tubules of the kidneys, and some necrosis of the tubules.

Silverman, Schulte, and First (1946/Ex. 1–142) determined that the maximum dose of MIBK tolerable to human volunteers for eight hours was 100 ppm; at 200 ppm, these subjects found the odor of MIBK objectionable and the vapor irritating. Linari and co-workers (1964/Ex. 1–1159) reported that more than half of all workers exposed to 500 ppm of MIBK for 20 to 30 minutes daily, and perhaps to 80 ppm for the remainder of the shift, experienced weakness, loss of appetitie, headache, burning eyes, nausea, vomiting, and sore throat; several of these workers also reported insomnia, somnolence, heartburn, and intestinal pain. Some workers had enlarged livers and others had colitis. Clinical test results on these workers were normal (Linari, Perrelli, and Varese 1964/Ex. 1–1159).

In a follow-up study on this same group of centrifuge operation workers; Armeli and co-workers (1968/Ex. 1–1028) determined that reduction of MIBK levels (during the 15 to 30 minutes of centrifuge operation) to 100 to 105 ppm, and (for the remainder of the shift) to 50 ppm had also significantly reduced the symptomatology reported earlier by these workers. However, liver enlargement persisted in two workers, and a few workers continued to report gastrointestinal and nervous system effects (Armeli, Linari, and Martorano 1968/Ex. 1–1028).

Elkins (1959/Ex. 1–734) noted that exposure to 100 ppm during boot-waterproofing operations caused workers to develop headache and nausea; another similarly exposed group experienced only irritation at 100 ppm.

The AFL–CIO (Ex. 194) commented on MIBK. The AFL–CIO supports the limits OSHA has established for this substance in the final rule.

In the final rule, OSHA is establishing an 8-hour TWA of 50 ppm and a 15-minute STEL of 75 ppm for hexone. The Agency concludes that these limits will work together to protect workers from the significant risks of headache, nausea, and irritation, as well as the potential kidney and liver effects that constitute material health impairments that are associated with exposures to hexone above the revised PELs.

Conclusion for Both Liver and Kidney Toxins

The health effects associated with occupational exposures to the hepato- and nephrotoxins shown in Tables C4–1 and C4–2 can be acute or chronic, reversible or irreversible, temporarily disabling or threatening to life. Workers experiencing chemically induced hepatotoxic or nephrotoxic effects may have enlarged livers, high blood pressure, hormonal imbalances, and/or organ necrosis, all of which constitute material impairments of health or functional capacity within the meaning of the Act. In addition, exposure to the substances in this grouping is associated with a host of other adverse health effects, ranging from pulmonary irritation to cancer, and OSHA concludes that the new or revised limits will substantially reduce the risk of these effects as well.

5. Substances for Which Limits Are Based on Avoidance of Ocular Effects

Introduction

Five of the chemicals for which OSHA is establishing limits have the potential to cause serious ocular effects in the workplace setting. Certain chemicals in this group are also sensory irritants and have been classified separately from the other irritants only because of their ability to cause permanent damage to the corneas, lenses, or optic nerves of exposed individuals.

Table C5–1 lists these five chemicals, along with OSHA's former, proposed, and final rule PELs, and each chemical's CAS number and HS number. For N-ethyl morpholine, the former 8-hour TWA of 20 ppm has been reduced to 5 ppm; the skin notation has been retained. For methyl alcohol and naphthalene, OSHA has retained its former 8-hour TWA and added a STEL (in the case of methyl alcohol, a skin notation has been added as well). For methyl silicate, the Agency has promulgated a new 8-hour PEL, while for hydrogen sulfide, the former STEL of 20 ppm and ceiling of 50 ppm have been replaced with a 10-ppm 8-hour TWA, supplemented by a 15-ppm short-term exposure limit.

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Table C5-1. Substances for Which Limits Are Based on Avoidance of Ocular Effects

H.S. Number/ Chemical Name	CAS No.	Former OSHA PEL	Proposed PEL	Final Rule PEL*
1172 N-Ethylmorpholine	100-74-3	20 ppm TWA, Skin	5 ppm TWA, Skin	5 ppm TWA, Skin
1209 Hydrogen sulfide	7783-06-4	20 ppm STEL 50 ppm Ceiling	10 ppm TWA 15 ppm STEL	10 ppm TWA 15 ppm STEL
1252 Methyl alcohol	67-56-1	200 ppm TWA	200 ppm TWA 250 ppm STEL, Skin	200 ppm TWA 250 ppm STEL, Skin
1266 Methyl silicate	681-84-5	--	1 ppm TWA	1 ppm TWA
1282 Naphthalene	91-20-3	10 ppm TWA	10 ppm TWA 15 ppm STEL	10 ppm TWA 15 ppm STEL

* OSHA's TWA limits are for 8-hour exposures, its STELs are for 15 minutes unless otherwise specified, and its ceilings are peaks not to be exceeded for any period of time.

Description of the Health Effects

Damage to the eye caused by exposure to the chemicals in this group can occur in the form of corneal, lens, retinal, ganglion cell layer, or optic nerve effects. Depending on the severity of the exposure, individual susceptibility, and the particular chemical and circumstances involved, this damage may be transient, temporarily disabling, or permanently blinding.

Corneal effects. The cornea and conjunctiva are the outer surfaces of the eye and are thus directly exposed to external insults. Since the cornea must maintain transparency to remain functional, scar formation after injury to the cornea can destroy visual function completely. Recent evidence suggests that the transparency of the cornea is maintained by thin inner and outer boundary layers and that the death of these layers leads to loss of transparency (Potts 1986/Ex. 1–174). The corneal epithelium (outer layer) sometimes regenerates, depending on the depth of the burn or insult and the nature of the toxicant.

Some chemicals, including methyl silicate, produce painful corneal epithelial injuries that have a delayed symptom onset. These substances can continue to cause pain and loss of corneal epithelial cells for several hours after exposure. Typically, there is no discomfort during the actual exposure, but several hours later, the eyes begin to burn, vision blurs, and conjunctival hyperemia, tearing, photophobia, and squinting occur (Grant 1986/Ex. 1–975). Possible mechanisms of action are enzyme inhibition, denaturing of other proteins, alteration of the DNA, and interference with the mitotic process; after a period of exposure, the affected cells die. Although the damaged epithelium sometimes regenerates after this type of injury, the damage can also involve the corneal stroma and endothelium, leading to scarring, vascularization, opacity, and loss of vision. The poor warning properties characteristic of these substances (i.e., their failure to cause an immediate response) make the establishment of protective exposure limits particularly important.

Exposure to the vapors of some of the substances in this group produces painless edema of the corneal epithelium, which can be accompanied by the delayed onset of visual haloes. A chemical that produces these effects is N-ethylmorpholine, a catalyst used to manufacture urethane foam. Painless edema generally occurs in workers who have been exposed for several hours to levels that do not produce discomfort during the exposure itself. The visual effect produced by such exposures consists of the appearance of colored haloes around lights, an effect that is caused by the diffraction of light through the swollen epithelial cells of the eye. Visual haloes are severely distracting and restrict activity substantially, and the mechanism underlying this effect is not well understood (Grant 1986/Ex. 1–975).

Lens effects. The lens is a transparent, avascular tissue surrounded by a thin, collagenous capsule. The major portion of the lens is composed of long, thin fibers that form closely packed, onion-like layers. Transparency is dependent on several factors: a highly ordered cellular arrangement; fiber size, shape, and uniformity; molecular structure; and regularity of fiber packing (Potts 1986/Ex. 1–174). Interference with lens metabolism, with transport across cell boundaries, or with the integrity of the lens capsule itself can cause a loss of lens transparency and lead to decreased visual acuity (Potts 1986/Ex. 1–174). All such changes in lens transparency are referred to as cataracts.

Retinal effects. The retina is a compact neural structure that is responsible for converting the ocular light image to neural impulses. Because the retina is an internal structure, it is not generally affected by exposure to dust, splashes of liquids, or vapors. However, exposure to certain internally absorbed substances, such as methyl alcohol, may cause changes or lesions in the retina, including retinal edema or hemorrhage. Exposure to a few of these substances can cause acute narrowing of the retinal arteries themselves, which can lead, in turn, to damage of the optic nerve and loss of vision.

Effects on ganglion cell layer and optic nerve. Below the retinal surface layer lies the ganglion cell layer, which is composed of the cell bodies of neurons that extend to the midbrain via the optic nerve. Ganglion cells may be damaged directly when the chemical acts on the cell bodies themselves or secondarily when the toxin destroys the

optic nerve. Depending on the severity of the exposure, loss of visual acuity or vision may ensue.

Dose-Response Relationships and Ocular Effects

For most of the chemicals on this list, limits have been established on the basis of health surveys and case reports of occupationally exposed populations. These studies indicate that exposures to concentrations of these substances at levels above the NOE level cause damage or pain to the eyes of exposed workers. In some cases only limited human data are available, and evidence from animal studies or knowledge of a chemical's structural analogy to another chemical known to have ocular effects provides the basis for the exposure limit. Animal models are generally good predictors of ocular effects in humans because the eyes of rodents, especially those of guinea pigs and rabbits, closely resemble human eyes. Thus, animal studies of the effects of exposure on the eye can be relied on to predict accurately how the chemicals that produce these effects in animals will behave in workers exposed in industrial situations. For the five chemicals in this group, the available toxicologic data, the record evidence, and OSHA's final determinations as to their limits are described below.

N-ETHYLMORPHOLINE

CAS: 100-74-3; Chemical Formula: $C_6H_{13}NO$
H.S. No. 1172

OSHA's former 8-hour TWA PEL for N-ethylmorpholine was 20 ppm, with a skin notation. The proposed permissible exposure limit was 5 ppm as an 8-hour TWA, also with a skin notation, and the final rule establishes this limit and retains the skin notation, which is consistent with the limits of the ACGIH. NIOSH (Ex. 8-47, Table N1) agrees with the selection of this limit. N-Ethylmorpholine is a colorless, flammable liquid with an ammonia-like odor; this substance is a severe eye irritant.

Prolonged exposure to fairly low concentrations of this substance causes corneal edema, blue-gray vision, and colored haloes. Typically, vision becomes misty and haloes appear a few hours after workers have been exposed to the vapors for a period of hours. Distortion of vision can occur even at levels considerably lower than those that cause irritation (Mastromatteo 1965/Ex. 1-146).

Reversible corneal edema has been observed in workers exposed to 40 ppm or more of N-ethylmorpholine for several hours (Dernehl 1966a/Ex. 1-62). Workers routinely exposed to 3- to 4-ppm concentrations but never to concentrations above 11 ppm complained of haloes and foggy vision as well as drowsiness (ACGIH 1986/Ex. 1-3, p. 263). The irritant effects of N-ethylmorpholine were also seen in a controlled-exposure experiment on volunteer subjects. Ten subjects exposed for 2.5 minutes to a concentration of 100 ppm experienced irritation of the eyes, nose, and throat; those exposed for 2.5 minutes to 50 ppm experienced slight irritation; and no irritation was reported after exposure for 2.5 minutes to 25 ppm (ACGIH 1986/Ex. 1-3, p. 263). N-ethylmorpholine is also readily absorbed through the skin (Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1-440).

OSHA's former 20-ppm PEL for N-ethylmorpholine did not protect exposed workers against the occurrence of corneal edema. Because corneal edema is painless as it is developing and symptoms have a delayed onset, workers are especially likely not to be aware of the danger of exposure. This is particularly hazardous because the effects on visual function of repeatedly exposing the eyes to substances that cause corneal edema are not known. The Agency received no comments on the health effects or revised exposure limits for N-ethylmorpholine, with the exception of NIOSH's submission.

OSHA concludes that reducing the PEL to 5 ppm as an 8-hour TWA (and retaining the skin notation) is necessary to protect occupationally exposed workers from ethylmorpholine's injurious ocular effects. The new, lower PEL will reduce the significant risk of material health impairment, which is manifested as corneal edema, visual distortion, and impaired vision, that is associated with exposure to this substance at concentrations above the revised PEL.

HYDROGEN SULFIDE

CAS: 7783-06-4; Chemical Formula: H_2S
H.S. No. 1209

OSHA's former limits for hydrogen sulfide were a 20-ppm STEL (10-minute maximum duration) and a 50-ppm

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ceiling limit. The proposed and final rule for this substance are 10 ppm as an 8-hour TWA and 15 ppm as a STEL. These limits are consistent with those of the ACGIH. NIOSH has a REL for hydrogen sulfide of 10 ppm as a 10-minute ceiling. Hydrogen sulfide is a colorless, flammable gas with the odor of rotten eggs. It is widely used as an agricultural disinfectant, chemical intermediate, analytical reagent, and in the manufacture of heavy water in the utilities sector. However, occupational exposure to hydrogen sulfide occurs most frequently when it is encountered in natural oil or gas deposits or as a by-product in chemical reactions.

The 1986 ACGIH *Documentation* (Ex. 1–3, p. 318) cites several reports (Brieger 1964; Kranenburg and Kessener 1935; Masure 1950; Elkins 1950a/Ex. 1–953) of the occurrence of adverse ocular effects, including conjunctivitis, associated with exposure to 20 ppm or less of hydrogen sulfide. A study by Poda and Aiken (1966/Ex. 1–115) reports that the adoption of a voluntary limit of 10 ppm in two heavy-water plants eliminated exposure problems. An early study by Flury and Zernik (1931f, as cited in ACGIH 1986/Ex. 1–3, p. 318) reports that the conjunctivitis caused by exposure to 10 to 15 ppm of hydrogen sulfide for six hours endured for several days; however, OSHA is unaware of cases in which this substance caused irreversible eye damage. The National Institute for Occupational Safety and Health (NIOSH) relied essentially on the studies discussed above (Poda and Aiken 1966/Ex. 1–115; Flury and Zernik 1931f, as cited in ACGIH 1986/Ex. 1–3, p. 318) when recommending its limit for hydrogen sulfide of 10 ppm for 10 minutes; NIOSH (Ex. 8–47, Table N7) continues to recommend this ceiling for hydrogen sulfide (this issue is discussed further below).

OSHA received several comments related to the health effects and proposed limits for hydrogen sulfide (Exs. 3–1163, 3–216, 8–37, 8–47, 129; Tr. XI, pp. 114, 225). The Atlantic Electric Company (Ex. 3–1163) pointed out an error in the proposal, which listed the short-term exposure limit for hydrogen sulfide as 5 ppm rather than 15 ppm. The Edison Electric Institute (EEI) (Tr. XI, p. 225) explained that utility workers are exposed to hydrogen sulfide when they enter utility manholes and vaults that are located near coastal areas, where this gas seeps into underground spaces. The EEI reports that utility workers use respirators and ventilate these spaces before entering. The Montana Sulphur and Chemical Corporation (Ex. 3–216), a small-business manufacturer, handler, and shipper of hydrogen sulfide, commented that, in its opinion, "the evidence presented for significantly tightening the existing standards is not at all compelling." According to Montana Sulphur, the studies cited by OSHA in the proposal to support the revised limits of 10 ppm (TWA) and 15 ppm (STEL) for hydrogen sulfide involved concurrent exposures "to other pollutants or stressors peculiar to the incident involved" (Ex. 3–216, p. 2). In addition, Montana Sulphur objects to OSHA's reliance on a study by Poda and Aiken (1966/Ex. 1–115) showing that voluntary compliance with an internal standard of 10 ppm at a facility in the heavy-water industry eliminated complaints of eye irritation among hydrogen-sulfide-exposed workers at this facility (Ex. 3–216). Montana Sulphur and Chemical reports that, in its long experience of manufacturing and handling this "notoriously toxic" substance, it has never had a case of eye irritation that required medical treatment; it urges OSHA to promulgate a STEL for hydrogen sulfide in the range of 25 to 30 ppm rather than the proposed 15 ppm (Ex. 3–216).

OSHA appreciates this commenter's thoughtful and thorough discussion of his company's experience in dealing with hydrogen sulfide in the workplace. However, OSHA's revised 8-hour TWA for this substance is based on the best available evidence (i.e., data on a level of occupational exposure that has been shown not to produce the health effect of concern). The eye irritation potential of hydrogen sulfide at levels below 20 ppm is widely recognized; the comment from Montana Sulphur (Ex. 3–216) acknowledges that reduction of the 8-hour TWA to 10 to 12 ppm is warranted. OSHA finds that a STEL of 15 ppm is justified by reports of eye irritation caused by short-term exposures to levels below 15 ppm (ACGIH 1986/Ex. 1–3, p. 318). OSHA is also aware that conditions in industry often involve simultaneous exposures to more than one hazardous substance and that such mixed exposures may increase the severity of the effects experienced by workers. However, the Agency must establish exposure limits based on the best available evidence for each individual substance to be regulated; it cannot attempt to set different limits for substances on the basis of the enormous number of other substances with which they could potentially be associated in actual use.

OSHA also received a comment on hydrogen sulfide from NIOSH (Ex. 8–47, Table N7). NIOSH recommends a single 10-ppm 10-minute ceiling for this substance. The United Paperworkers International Union (Ex. 8–37) also recommends adoption of the NIOSH 10-minute ceiling of 10 ppm. The Agency believes that the protection provided by NIOSH's 10-ppm short-term limit is essentially equivalent to that provided by OSHA's combined TWA–STEL limits, and that the combination of a 10-ppm 8-hour TWA and a 15-ppm STEL established in the final rule will provide broader protection in workplaces characterized either by short-term or by steady-state exposures.

The New Jersey Department of Health (Ex. 144) urged OSHA to base its limits for hydrogen sulfide on EPA's IRIS data. OSHA discusses this approach and New Jersey's comment in Section VI.A of this preamble.

OSHA concludes that the former 20-ppm (10-minute) short-term limit and 50-ppm ceiling limit did not adequately protect workers against the adverse ocular effects associated with exposure to concentrations of hydrogen sulfide below 20 ppm, as reported in several studies. OSHA finds that the eye irritation and conjunctivitis associated with such exposures represent a significant risk of material health impairment to workers, who may be forced to seek medical treatment after such exposure and who may also be unable to work during the period of recovery. OSHA has accordingly established an 8-hour TWA limit for hydrogen sulfide of 10 ppm and a short-term limit of 15 ppm. These levels have been demonstrated to be effective in preventing irritation and conjunctivitis in the workplace (Poda and Aiken 1966/Ex. 1–115). The Agency finds that this dual limit will provide protection both in continuous steady-state exposure situations and in those characterized by sharp peaks and will do so more effectively than a single, short-term limit such as that recommended by NIOSH.

METHYL ALCOHOL

CAS: 67–56–1; Chemical Formula: CH_3OH
H.S. No. 1252

OSHA's former 8-hour TWA limit for methyl alcohol was 200 ppm. The proposed limits were an 8-hour TWA of 200 ppm, a STEL of 250 ppm, and a skin notation. The final rule establishes these limits, which are consistent with those of the ACGIH. NIOSH previously recommended exposure limits for this substance of 200 ppm as a TWA and 800 ppm as a STEL; however, after reviewing the health evidence for methyl alcohol, NIOSH concurs with OSHA's final rule PELs for this substance (Ex. 8–47, Table N1). Methyl alcohol is a mobile, highly polar,

flammable liquid that is widely used as an industrial solvent.

As stated in the proposal (53 *FR* 21061), workers exposed to concentrations of methyl alcohol between 200 and 375 ppm experience severe recurrent headaches, and at levels between 1200 and 8300 ppm, studies by Kingsley and Hirsch (1954/Ex. 1–212) report that the visual capacities of exposed individuals are diminished. OSHA finds that a 250-ppm STEL is necessary because an 8-hour PEL of 200 ppm alone does not protect workers from exposure to short-term peaks at levels that cause eye irritation and severe, recurrent headaches in exposed workers. Although the skin LD_{50} in rabbits is 20 g/kg, OSHA is adding a skin notation for methyl alcohol in the final rule (see Section VI.C.18 for a discussion of the Agency's policy on skin notations). The Agency's reason for establishing a skin notation for methyl alcohol despite this high dermal LD_{50} in rabbits is that a dermal LD_{Lo} of 500 mg/kg has been reported for this substance in monkeys (*Dangerous Properties of Industrial Materials,* 7th ed., Sax and Lewis 1989, p. 1377).

Several commenters submitted information to the record on methyl alcohol (Exs. 150 (Comments on Methyl Alcohol), 194, 3–661, 3–902, and 3–896). The Motor Vehicle Manufacturers Association (MVMA) (Ex. 3–902) presented no substantive comment with regard to methyl alcohol; instead, the MVMA listed 41 chemicals, including methyl alcohol, that, in the opinion of the MVMA, require "more review * * * to allow OSHA and industry additional time to properly assess * * * [the technological and economic] consequences" of revising the limit. Both the Eastman Kodak Company (Ex. 3–661) and the Chevron Corporation (Ex. 3–896) submitted specific comments on OSHA's proposal to add a STEL of 250 ppm to the existing 8-hour TWA of 200 ppm. Representing Chevron, Stanley Dryden stated:

> We do not believe that the proposed 250-ppm STEL is justified by the discussion in [OSHA's] preamble (Ex. 3–896, p. 10).

According to Kodak, the study by Kingsley and Hirsch (1954/Ex. 1–212) that was cited by OSHA in support of the STEL involved exposures to a duplicating machine fluid that contained between 5 and 98 percent methyl alcohol and 2 to 9 percent of an unidentified fluid(s). Kodak is of the opinion that the severe headaches experienced by exposed employees may have been related to the unidentified components of the fluid rather than to methyl alcohol, and further that these exposures may not have been the result of short-term exposures (Ex. 3–661).

OSHA finds Chevron's and Eastman Kodak's comments unpersuasive, for several reasons. First, the measured airborne levels of methyl alcohol reported in the Kingsley and Hirsch study (1954/Ex. 1–212) ranged from 200 to 375 ppm when employees were using direct process duplicating fluids; other studies also report that exposure to methyl alcohol at these levels causes headaches (Henson 1960, as cited in ACGIH 1986/Ex. 1–3, p. 372). Thus, the effects cited in the Kingsley and Hirsch study (1954/Ex. 1–212) are biologically plausible and consistent with those reported in other studies of the effects of this substance. Second, OSHA believes that a 250-ppm STEL is needed to ensure that workers are not exposed, even for short periods, to the elevated levels that have been shown to cause these effects. NIOSH has reevaluated the toxicological evidence for a STEL for methyl alcohol and concurs with the 250-ppm limit OSHA is establishing in the final rule. According to NIOSH (Ex. 150, Comments on Methyl Alcohol):

> [T]here appears to be no justification for a ceiling of 800 ppm [the ceiling level formerly recommended by NIOSH]. It appears that data are more supportive of the OSHA and ACGIH STEL of 250 ppm * * * it seems reasonable to update the NIOSH recommended ceiling (Ex. 150).

Thus, OSHA has determined that the addition of a STEL is necessary to reduce the significant risk of disturbed vision and headaches to which workers could be and have been exposed in the absence of a limit on short-term exposures. As discussed above, NIOSH concurs with OSHA that a short-term limit of 250 ppm is appropriate for methyl alcohol; NIOSH described a recent study (Frederic et al. 1984, as cited in Ex. 150, Comments on Methyl Alcohol) that found that teachers' aides exposed to 80 to 3080 ppm of methyl alcohol while using duplicating machines experienced blurred vision, headaches, dizziness, and skin problems. The AFL–CIO (Ex. 194, p. A–12) supports the addition of a STEL and a skin notation for methyl alcohol.

The final rule promulgates an 8-hour TWA of 200 ppm, a STEL of 250 ppm, and a skin notation for methyl alcohol. OSHA concludes that the 8-hour TWA and 15-minute STEL will work together to reduce substantially the significant risk of headaches and blurred vision presented by short-term occupational exposures to methyl alcohol at concentrations above 250 ppm. The Agency finds that the headache, blurred vision, and other ocular effects associated with exposure to methyl alcohol constitute material impairments of health.

METHYL SILICATE

CAS: 681–84–5; Chemical Formula: $(CH_3O)_4Si$
H.S. No. 1266

OSHA did not formerly have a limit for methyl silicate; the Agency proposed the adoption of a 1-ppm 8-hour TWA for this substance, and NIOSH (Ex. 8–47, Table N1) concurred with this selection. The final rule establishes this limit, which is consistent with that of the ACGIH. Methyl silicate exists in the form of colorless needles.

Methyl silicate damages the cornea and is associated with a delayed onset of symptoms. In many cases of methyl silicate exposure, the eyes recover completely, but there are reports of damage to the deep layers of the cornea that caused permanent opacification and, in one worker, loss of vision in one eye (Grant 1986/Ex. 1–975). It is estimated that exposing humans to methyl silicate at concentrations of 200 to 300 ppm for 15 minutes will produce lesions, and that exposure to 1000 ppm for this period will produce injury requiring hospitalization (ACGIH 1986/Ex. 1–3, p. 409).

Rabbits exposed to 1000 ppm of methyl silicate in dry air experienced delayed eye burns (ACGIH 1986/Ex. 1–3, p. 409). Exposure of these animals to approximately 15,000 ppm for five minutes caused eye burns, but exposure to this level for four minutes caused no appreciable effect. Guinea pigs showed maximum no-effect levels of 135 ppm for 15 minutes, 90 ppm for one hour, and 20 ppm for 8 one-hour periods. The latency period for ocular changes was 16 hours for serious effects and up to three days for mild involvement (ACGIH 1986/Ex. 1–3, p. 409). Only NIOSH commented on this substance.

Because the onset of response to this toxin is delayed, because exposure in the workplace could have a duration substantially greater than that in the animal bioassays, and because of interspecies variability, it is necessary to establish a PEL considerably below the NOE level in animals to reduce the significant risk of ocular damge to employees. The Agency is therefore establishing a 1-ppm 8-hour TWA limit for methyl silicite to reduce the significant risk of severe ocular effects associated with the uncontrolled exposures formerly possible in the absence of a PEL. The Agency concludes that this limit will substantially reduce this significant risk by protecting workers from the ocular effects of methyl silicate exposure, which

constitute material impairments of health.

NAPHTHALENE

CAS: 91-20-3; Chemical Formula: $C_{10}H_8$
H.S. No. 1282

OSHA's former exposure limit for naphthalene was 10 ppm as an 8-hour TWA. The final rule retains this TWA and adds a short-term limit of 15 ppm for this substance, which occurs as a colorless to brown solid and has the odor of mothballs. The ACGIH also has a 10-ppm 8-hour TWA and a 15-ppm STEL for naphthalene. NIOSH (Ex. 8-47, Table N1) concurs with the PELs selected for this substance.

The oral LD_{50} for naphthalene in rats is 1760 mg/kg (Flury and Zernik 1931g/Ex. 1-995). In humans, the inhalation of naphthalene vapor causes headache, loss of appetite, and nausea (Flury and Zernik 1931g/Ex. 1-995; Patty 1949b, as cited in ACGIH 1986/Ex. 1-3, p. 420). These authors also report that exposure causes optical neuritis, corneal damage, and kidney injury. Eight of 21 workers exposed for five years to unspecified levels of naphthalene developed opacities of the lens of the eye (Ghetti and Mariani 1956/Ex. 1-739). Ingestion of large amounts of naphthalene causes severe hemolytic anemia and hemoglobinuria (Stokinger and Mountain 1963/Ex. 1-765). The lethal dose in humans has been reported as 50 mg/kg (NIOSH 1977i/Ex. 1-1182). Concentrations somewhat above 15 ppm are reported to cause marked eye irritation (Robbins 1951/Ex. 1-799).

Only the American Iron and Steel Institute (AISI) and NIOSH commented on naphthalene. The AISI (Exs. 129 and 188) believes that a STEL for naphthalene is not warranted by the evidence. However, the Robbins (1951/Ex. 1-799) study discussed above clearly shows that excursions to 15 ppm cause severe eye irritation, and OSHA thus finds the STEL both necessary and appropriate.

In the final rule, the Agency is retaining the 8-hour TWA of 10 ppm and adding a 15-minute STEL of 15 ppm for naphthalene. This STEL is designed to protect against the eye irritation observed in workers at elevated levels (Robbins 1951/Ex. 1-799). The Agency concludes that these limits will protect workers from the significant risks of eye irritation and serious ocular effects, which constitute material health impairments that are potentially associated with exposure to levels above the 8-hour limit.

Conclusions for This Group of Ocular Toxins

OSHA finds that promulgation of the final rule's limits for this group of chemicals, which have the potential to cause adverse ocular effects ranging from transient discomfort to permanent blindness, will substantially reduce the significant risk of visual impairment associated with occupational exposure to these substances. The toxicological basis for the final rule's limits include evidence derived from occupationally exposed workers and results obtained in animals that have been shown to be excellent predictors of human responses. The risks being protected against have serious consequences, both in terms of material impairment of health and interference with the functional capacity of those workers who are themselves exposed and the safety and well-being of these workers and their co-workers. Thus, OSHA finds that the limits established by the final rule are necessary to reduce these significant occupational risks, which constitute material health impairments of health within the meaning of the Act.

6. Substances for Which Limits Are Based on Avoidance of Respiratory Effects Introduction

Limits are being established for a total of 35 substances or materials for which exposure has been shown to cause adverse respiratory effects. The chemicals in this group cause acute pulmonary edema, alveolar damage, or chronic respiratory damage through the general mechanisms of cellular damage or fibrosis. At sufficient doses, these effects can be permanent, disabling, and life-threatening.

Some of the materials in this group are composites of naturally occurring minerals, and, for these, the Agency is establishing limits based on the most hazardous component. For several materials (coal dust, crystalline tripoli, silica, and graphite), OSHA is requiring the TWA to be measured as the respirable quartz fraction of the dust, because it is exposure to this fraction that presents the greatest risk to exposed workers.

Table C6-1 lists the 35 substances in this group, along with the former, proposed, and final rule PELs, and CAS and HS Numbers. There was no former OSHA PEL for 12 of these substances. For one substance, OSHA is establishing a ceiling limit to replace an existing 8-hour TWA, and for ten substances, a lower TWA and/or STEL are being established. In three instances, OSHA is establishing a STEL to augment its former TWA-PELs. For nine substances, OSHA is changing only the form in which the limit is being expressed.

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C6-1. Substances for Which Limits Are Based on Avoidance of Respiratory Effects

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1017 Aluminum (pyro powders)	7429-90-5	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1034 Bismuth telluride (Se-doped)	1304-82-1	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1080 Chlorine dioxide	10049-04-4	0.1 ppm TWA	0.1 ppm TWA 0.3 ppm STEL	0.1 ppm TWA 0.3 ppm STEL
1093 Chromium metal	7440-47-3	1 mg/m^3 TWA	0.5 mg/m^3 TWA	1 mg/m^3 TWA
1096 Coal dust, @ 5% quartz	None	2.4 mg/m^3 TWA[a]	2 mg/m^3 TWA[a]	2 mg/m^3 TWA[a]
1097 Coal dust, # 5% quartz	None	$\frac{10\ mg/m^3}{\%\ SiO_2+2}$	0.1 mg/m^3 TWA[b]	0.1 mg/m^3 TWA[b]
1161 Ethyl acrylate	140-88-5	25 ppm TWA, Skin	5 ppm TWA 25 ppm STEL, Skin	5 ppm TWA 25 ppm STEL, Skin
1177 Ferrovanadium dust	12604-58-9	1 mg/m^3 TWA	1 mg/m^3 TWA 3 mg/m^3 STEL	1 mg/m^3 TWA 3 mg/m^3 STEL
1178 Fibrous glass	None	15 mg/m^3 TWA	5 mg/m^3 TWA	See text

C6-1. Substances for Which Limits Are Based on Avoidance of Respiratory Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1190 Grain dust (oat, wheat, barley)	None	--	4 mg/m^3 TWA	10 mg/m^3 TWA
1191 Graphite, natural, respirable @ 1% quartz	7782-42-5	15 mppcf TWA	2.5 mg/m^3 TWA	2.5 mg/m^3 TWA
1213 Indium & compounds	7440-74-6	--	0.1 mg/m^3 TWA	0.1 mg/m^3 TWA
1215 Iron oxide (dust and fume)	1309-37-1	10 mg/m^3 TWA	10 mg/m^3 TWA	5 mg/m^3 TWA
1272 Methylene bis (4-Cyclohexyliso-cyanate)	5124-30-1	--	0.01 ppm Ceiling	0.01 ppm Ceiling
1276 Mica, respirable dust containing 1% quartz	12001-26-2	20 mppcf TWA	3 mg/m^3 TWA	3 mg/m^3 TWA
1277 Mineral wool fiber	None	15 mg/m^3 TWA	10 mg/m^3 TWA	See text
1289 Nitrogen dioxide	10102-44-0	5 ppm Ceiling	1 ppm STEL	1 ppm STEL
1300 Oxygen difluoride	7783-41-7	0.05 ppm TWA	0.05 ppm Ceiling	0.05 ppm Ceiling

C6-1. Substances for Which Limits Are Based on Avoidance of Respiratory Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1301 Ozone	10028-15-6	0.1 ppm TWA	0.1 ppm TWA 0.3 ppm STEL	0.1 ppm TWA 0.3 ppm STEL
1303 Paraquat, respirable dust	4685-14-7	0.5 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA, Skin
1354 Silica, crystalline cristobalite (as respirable quartz dust)	14464-46-1	1/2 value calculated for quartz	0.05 mg/m^3 TWA	0.05 mg/m^3 TWA
1355 Silica, crystalline quartz, respirable	14808-60-7	$\frac{10\ mg/m^3}{\%\ SiO_2+2}$	0.1 mg/m^3 TWA	0.1 mg/m^3 TWA
1356 Silica, crystalline tridymite (as respirable quartz dust)	15468-32-3	1/2 value calculated for quartz	0.05 mg/m^3 TWA	0.05 mg/m^3 TWA
1357 Silica, crystalline tripoli (as respirable quartz dust)	1317-95-9	$\frac{10\ mg/m^3}{\%\ SiO_2+2}$	0.1 mg/m^3 TWA	0.1 mg/m^3 TWA
1358 Silica, fused	60676-86-0	$\frac{10\ mg/m^3}{\%\ SiO_2+2}$	0.1 mg/m^3 TWA	0.1 mg/m^3 TWA

C6-1. Substances for Which Limits Are Based on Avoidance of Respiratory Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1363 Soapstone, total dust	None	20 mppcf TWA	6 mg/m^3 TWA	6 mg/m^3 TWA
1363A Soapstone, respirable dust	None	--	3 mg/m^3 TWA	3 mg/m^3 TWA
1375 Sulfur dioxide	7446-09-5	5 ppm TWA	2 ppm TWA 5 ppm STEL	2 ppm TWA 5 ppm STEL
1378 Sulfur tetrafluoride	7783-60-0	--	0.1 ppm Ceiling	0.1 ppm Ceiling
1381 Talc (containing no) asbestos)	14807-96-6	20 mppcf TWA	2 mg/m^3 TWA	2 mg/m^3 TWA
1395 Tin oxide	7440-31-5	--	2 mg/m^3 TWA	2 mg/m^3 TWA
1409 Trimellitic anhydride	552-30-7	--	0.005 ppm TWA	0.005 ppm TWA
1430A Wood dust, hard	None	--	1 mg/m^3 TWA	5 mg/m^3 TWA 10 mg/m^3 TWA
1403B Wood dust, soft	None	--	5 mg/m^3 TWA	5 mg/m^3 TWA 10 mg/m^3 TWA

C6-1. Substances for Which Limits Are Based on Avoidance of Respiratory Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1430C Wood dust, allergenic (Western Red Cedar)	None	--	--	2.5 mg/m^3 TWA

[a] For coal dust, respirable fraction less than 5 percent SiO_2.
[b] For coal dust, respirable fraction more than 5 percent SiO_2.
* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

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Description of the Health Effects

The respiratory system is a major route of occupational exposure for toxic substances. Because of the vital nature of pulmonary function, respiratory toxicants present a serious health hazard both from acute and chronic exposures. Acute respiratory disease can be life threatening.

Chronic pulmonary disease can result from long-term exposure to respiratory toxicants and is potentially crippling because it greatly reduces the quality of life and the productivity of its victims. In addition, the onset of respiratory disease can be insidious, because it may be indicated only by the gradual development of a few nonspecific signs (Petersdorf et al., *Harrison's Principles of Internal Medicine,* 10th ed., 1983).

The difficulties of detecting irreversible respiratory effects complicate the prevention of pulmonary disease. Pulmonary function can be evaluated with a variety of tests, including measurements of the vital capacity and of the resting and forced expiratory volumes. However, certain conditions, including emphysema and fibrosis, are difficult to diagnose even with such tests. In addition, these same diseases often continue to progress even after the affected individual has recognized the problem and obtained medical assistance. Furthermore, these diseases may continue to progress even after exposure has ceased, which makes prevention even more vital.

In addition to the threat posed to the general occupational population by respiratory toxins, certain subpopulations, such as persons with impaired lung function caused by asthma, bronchitis, emphysema, and pulmonary fibrosis, are at special risk from the adverse effects of respiratory toxins. Tobacco smoking can cause or aggravate all of the respiratory conditions discussed above and can interact additively or synergistically with respiratory toxins to increase their adverse effects on the pulmonary system. For example, tobacco smoking acts additively with coal dust to diminish pulmonary function. Because tobacco smoke contains nitrogen oxides, cadmium, and ammonia, occupationally exposed workers who smoke have an additional source of exposure to these respiratory toxins.

Two general categories of lung injuries are relevant to the group of substances under consideration:

- Damage to cells lining the airways, which results in necrosis (localized areas of dead cells), increased permeability, and edema.
- Production of fibrosis, which may become massive and greatly reduce lung capacity.

Cellular damage resulting in edema and emphysema. A number of substances cause damage to cells lining the airways. This can result in increased permeability of cell membranes and subsequent edema, hemorrhage, and localized necrosis (areas of dead cells). Chronic inhalation of certain chemicals causes destruction of the alveolar septa and results in emphysema. Cellular damage may be either localized or diffuse, depending on the distribution of the toxicant in the lung.

Edema is the release of fluid into the lumen (open spaces of the airways) or alveoli. Serious edema can take several hours to develop so that, in some cases, life-threatening or even fatal exposures can take place without the individual's being aware at the time of exposure of the extent of the damage. Ozone, nitrogen dioxide, and paraquat all cause localized cellular damage leading to edema (Klaassen, Amdur, Doull et al. 1986/Ex. 1-99). Fatalities from pulmonary edema have resulted from exposures to concentrations of nitrogen dioxide of about 200 ppm (*Dangerous Properties of Industrial Materials,* 6th ed., Sax 1984). Paraquat is unusual in that it can cause delayed pulmonary damage following exposure, even when exposure occurs via routes other than inhalation (Klaassen, Amdur, Doull et al. 1986/Ex. 1-99).

Necrotic changes can reduce the functional surface area of the lung. One type of lesion often noted in persons exposed to respiratory toxins is benign granulomas, which are localized masses formed when the immune system attempts to sequester a foreign object. Depending on the extent of the damage, these masses may reduce the functional capacity of the lung. Exposure to selenium-doped bismuth telluride has been associated with the production of benign granulomas without fibrosis (Wagner, Madden, Zimber, and Stokinger 1974, as cited in ACGIH 1986/Ex. 1-3, p. 59).

Emphysema is caused by a gradual destruction of the cells of the alveolar septa, which causes a loss of elasticity in the lung. A slight degree of emphysema is present in much of the adult population and does not cause any functional impairment. As the disease progresses, however, serious and life-threatening reductions in functional capacity can occur. Once the disease has advanced to the point of serious functional impairment, it is, for the most part, irreversible (Petersdorf et al. 1983). There is evidence that a number of the substances in this group cause emphysema, including sulfur tetrafluoride (ACGIH 1986/Ex. 1-3), ozone, and nitrogen dioxide (Klaassen, Amdur, Doull et al. 1986/Ex. 1-99).

Fibrotic changes. Pulmonary fibrosis was one of the earliest recognized forms of occupational disease. Fibrosis should be distinguished from pneumoconiosis, although these terms are often used interchangeably. Pneumoconiosis is a more general term indicating the presence of a foreign substance in the lungs, as determined by radiographic (X-ray) analysis. This definition encompasses a variety of conditions and does not by itself necessarily indicate functional damage (Petersdorf et al. 1983). In contrast, fibrosis is a seriously debilitating disease. One type of fibrosis is interstitial fibrosis, which is a kind of pneumoconiosis characterized by deposition of fibrous tissue in the interstitial spaces between the alveolar membrane and the pulmonary capillary membrane. Interstitial fibrosis greatly reduces the diffusing capacity of the lung and thus causes oxygen deprivation in the body (Guyton 1981/Ex. 1-1002). Like emphysema, fibrosis is largely irreversible; it sometimes progresses even in the absence of further exposure (Petersdorf et al. 1983).

Silicosis is a form of interstitial fibrosis that is caused by exposure to respirable silica particles (Klaassen, Amdur, Doull et al. 1986/Ex. 1-99). Exposure to coal dust causes a pneumoconiosis with fibrosis that can be severely debilitating (Petersdorf et al. 1983). In addition, exposure to graphite, mica, and grain dust have all been associated with fibrosis in workers (ACGIH 1986/Ex. 1-3).

Dose-Response Relationships and Respiratory Effects

For most of the substances in this group, permissible exposure limits have been based on health surveys and case reports of occupationally exposed populations. In some cases, animal studies provide the evidence of a substance's toxicity. As is the case for most of the substances for which OSHA is establishing new, reduced, or revised limits, the dose-response curve for respiratory irritants tends to be S-shaped.

Table C6–2 presents dose-response data on the adverse pulmonary effects of representative chemicals in this group, the populations exposed, and the endpoints observed. The following discussions describe the record evidence, present OSHA's findings for all the substances on Table C6–1, and describe the nature of the risks faced by workers exposed to them.

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TABLE C6-2. Summary of Dose-Response Evidence for Adverse Respiratory Effects

H.S. Number/ Chemical Name	CAS No.	FORMER PEL	FINAL RULE PEL*	Dose-Response Data: Dose/Duration Associated With Observed Effects	Species	Comments
1034 Bismuth telluride (Se-Doped)	1304-82-1	--	5 mg/m^3 TWA	15 mg/m^3 1 year	Dogs Rats Rabbits	Granulomatous lesions in lungs seen after 6 months of exposure.
1096 Coal Dust, 5% quartz	None	2.4 mg/m^3 TWA	2 mg/m^3 TWA	4 mg/m^3 35 years	Humans	Calculated estimate of 10 percent probability of developing pneumoconiosis with fibrosis after 35 years of exposure to coal dust. (Quartz content not identified.)
1097 Coal Dust, ‖ 5% quartz	None	$\frac{10\ mg/m^3}{\%\ SiO_2+2}$	0.1 mg/m^3 TWA			
1190 Grain Dust (oat, wheat, barley)	None	--	10 mg/m^3 TWA	# 10 mg/m^3	Humans	Chronic bronchitis, shortness of breath, reduced pulmonary function, increased incidence of respiratory symptoms.
				@ 10 mg/m^3	Humans	Increased incidence of respiratory symptoms.
					Humans	Fibrosis and mottling, pneumoconiosis.

TABLE C6-2. Summary of Dose-Response Evidence for Adverse Respiratory Effects (continued)

H.S. Number/ Chemical Name	CAS No.	FORMER PEL	FINAL RULE PEL*	Dose-Response Data: Dose/Duration Associated With Observed Effects	Species	Comments
1191 Graphite, natural, respirable	7782-42-5	15 mppcf TWA	2.5 mg/m^3 TWA	N/A	Humans	Anthracosilicosis, similar to that seen in coal miners.
1213 Indium & compounds	7440-74-6	--	0.1 mg/m^3 TWA	24-97 mg/m^3	Rats	Widespread alveolar edema following exposure to In_2O_2.
1276 Mica	12001-26-2	20 mppcf TWA	3 mg/m^3 TWA	N/A	Humans	Signs and symptoms resembling silicosis and pneumoconiosis in 8 of 57 workers.
1289 Nitrogen dioxide	10102-44-0	5 ppm Ceiling	1 ppm STEL	N/A	Humans	Fatal pulmonary edema.
				0.4-2.7 ppm chronic	Humans	Change in pulmonary vital capacity.
1300 Oxygen difluoride	7783-41-7	0.05 ppm TWA	0.05 ppm Ceiling	0.5 ppm two 7-hr exposures	Lab. Animals	Lethal to a wide variety of laboratory species, causing pulmonary edema and hemorrhage after several hours of exposure.

TABLE C6-2. Summary of Dose-Response Evidence for Adverse Respiratory Effects (continued)

H.S. Number/ Chemical Name	CAS No.	FORMER PEL	FINAL RULE PEL*	Dose-Response Data: Dose/Duration Associated With Observed Effects	Species	Comments
1301 Ozone	10028-15-6	0.1 ppm TWA	0.1 ppm TWA 0.3 ppm STEL	1.5 ppm 3 hrs/day	Humans	Significant reduction in pulmonary vital capacity.
				1 ppm 1 day	Mice	Damage to alveolar tissue.
1303 Paraquat, respirable dust	4685-14-7	0.5 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA Skin	N/A	Humans	69 accidental deaths from pulmonary injury reported through 1972.
1354 Silica, crystalline cristobalite	14464-46-1	1/2 value for quartz	0.05 mg/m^3 TWA	0.5 mg/m^3 (as total dust) 2.5 years	Dogs	Cellular infiltration of lung and fibrotic nodules in pulmonary lymph nodes.
1355 Silica, crystalline quartz, respirable	14808-60-7	$\frac{10 \text{ mg/m}^3}{\% \text{ SiO}_2+2}$	0.1 mg/m^3 TWA	0.1 mg/m^3 chronic	Humans	Accelerated loss of pulmonary function over effects of aging alone.
1356 Silica, crystalline tridymite	15468-32-3	1/2 value for quartz	0.05 mg/m^3 TWA	N/A	Rats	Most active form of free silica when administered by intratracheal injections.

TABLE C6-2. Summary of Dose-Response Evidence for Adverse Respiratory Effects (continued)

H.S. Number/ Chemical Name	CAS No.	FORMER PEL	FINAL RULE PEL*	Dose-Response Data: Dose/Duration Associated With Observed Effects	Dose-Response Data: Species	Dose-Response Data: Comments
1357 Silica, crystalline tripoli	1317-95-9	$\frac{10\ mg/m^3}{\%\ SiO_2+2}$	0.1 mg/m^3 TWA	N/A	Lab. Animals	Progressive nodular fibrosis.
1375 Sulfur dioxide	7446-09-5	5 ppm TWA	2 ppm TWA 5 ppm STEL	1 ppm	Humans	Accelerated loss of pulmonary function
1378 Sulfur tetrafluoride	7783-60-0	--	0.1 ppm Ceiling	4 ppm 4 hrs/day/ 10 days	Rats	Emphysema, marked clinical signs of respiratory impairment.
1409 Trimellitic anhydride	552-30-7	--	0.005 ppm TWA		Rats	Intra-alveolar hemorrhage. (No exposure duration indicated.)

* OSHA's TWA limits are for 8-hour expcsures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

N/A = Not available.

BILLING CODE 4510-26-C

ALUMINUM (PYRO POWDERS)

CAS: 7429-90-5; Chemical Formula: Al
H.S. No. 1017

OSHA previously had no permissible exposure limit for aluminum pyro powders. The ACGIH has an 8-hour TLV-TWA of 5 mg/m^3. The proposed and final rules have a PEL of 5 mg/m^3 for the aluminum pyro powders; NIOSH (Ex. 8-47, Table N1) concurs with this limit. Powders and flake aluminum are flammable and can form explosive mixtures in air.

Aluminum pyro powders have a higher reported toxicity than aluminum metal dusts (Stokinger 1981a/Ex. 1-1133). Several British studies have examined the effects of exposure to this finely flaked aluminum on workers in paints and pyrotechnics plants. Their findings revealed that pulmonary fibrosis may result from exposure to pyro powders, although epidemiologic evidence indicates that additives used to prevent oxidation and agglomeration may have contributed to the incidence and nature of the disease (Edling 1961/Ex. 1-733; Jordan 1961/Ex. 1-559; Mitchell, Manning, Molyneux, and Lane 1961/Ex. 1-564). Exposures that have previously caused lung changes in workers are presumed to have been extremely high (ACGIH 1986/Ex. 1-3, p. 22). No comments, other than NIOSH's, were received on these powders.

OSHA concludes that the permissible exposure limit of 5 mg/m^3 TWA for aluminum pyro powders will prevent the significant risk of lung changes in workers exposed at the concentrations formerly permitted by the absence of an OSHA limit. The Agency has determined that these lung changes constitute material impairment of health.

BISMUTH TELLURIDE (DOPED)

CAS: 1304-82-1; Chemical Formula: Bi_2Te_3
H.S. No. 1034

OSHA had no former limit for doped bismuth telluride (Bi_2Te_3). The ACGIH has a TLV-TWA of 5 mg/m^3 for Bi_2Te_3 that has been doped with selenium sulfide. The proposed PEL was 5 mg/m^3 as an 8-hour TWA; NIOSH (Ex. 8-47, Table N1) concurs with this limit, and the final rule establishes it. Bismuth telluride appears as gray, hexagonal platelets; it is also available as ingots or single crystals.

Wagner, Madden, Zimber, and Stokinger (1974, as cited in ACGIH 1986/Ex. 1-3, p. 59) conducted a one-year study in which rabbits, dogs, and rats were exposed for six hours/day, five days/week to doped bismuth telluride dust (containing 80.04 mol % Bi_2Te_3 and 0.20 mol % SnTe, plus a small stoichiometric excess of Te) of 1.04 um particle diameter at a mean concentration of 15 mg/m^3. Small, granulomatous lesions without fibrosis appeared in the lungs of dogs at six months. In dogs that were sacrificed four months after an eight-month exposure, the lesions had regressed, and the affected lymph nodes were without cellular reaction. Rabbits exhibited similar histologic effects, but with decreased numbers of pulmonary macrophages, no fibrous tissue proliferation, and no cellular or fibrous tissue reaction around the dust deposits in the lymph nodes. The rats showed fewer granulomas but some areas of epithelialization of the alveolar walls. As was true for the other species, the rats showed neither fibrosis nor cellular reaction in the lymph nodes, despite accumulation of the intermetallic dust (Wagner, Madden, Zimber, and Stokinger 1974, as cited in ACGIH 1986/Ex. 1-3, p. 59). Only NIOSH commented on this substance.

In the final rule, an 8-hour PEL of 5 mg/m^3 TWA is established for Se-doped bismuth telluride to prevent the occurrence of the pulmonary lesions seen in experimental animals. OSHA concludes that this limit will substantially reduce the significant risk of these pulmonary effects.

CHLORINE DIOXIDE

CAS: 10049-04-4; Chemical Formula: ClO_2
H.S. No. 1080

Previously, OSHA had an 8-hour TWA limit of 0.1 ppm for chlorine dioxide. The ACGIH recommends the same time-weighted average and a 15-minute STEL of 0.3 ppm. The proposal retained the same TWA and added a 15-minute STEL of 0.3 ppm, and NIOSH (Ex. 8-47, Table N1 concurred with these limits, which are established in the final rule. Chlorine dioxide is a red-yellow gas at ordinary temperatures.

Rats exposed to 0.1-ppm concentrations of chlorine dioxide for 10 weeks at five hours daily showed no adverse effects from exposures. Other data in animals are not available (Dalhamn 1957/Ex. 1-307).

Data on human exposures indicate that marked irritation occurs on inhalation of 5 ppm (no time specified), and that one death occurred at 19 ppm (Elkins 1959b, as cited in ACGIH 1986/Ex. 1-3, p. 118). Repeated exposures in humans have been linked to bronchitis and and pronounced emphysema (Petry 1954/Ex. 1-1163). Clinical studies conducted by Gloemme and Lundgren (1975/Ex. 1-323) revealed that the majority of workers who had been exposed for five years to average concentrations of chlorine dioxide below 0.1 ppm, in combination with about 1.0 ppm chlorine, experienced eye and respiratory irritation and slight bronchitis. Some gastrointestinal irritation was also observed in these workers. Gloemme and Lundgren (1957/Ex. 1-323 attributed all of these effects to elavated short-term exposures involving excursions above the 0.1 ppm level. Ferris, Burgess, and Worcester (1967/Ex. 1-316) have shown that concentrations occasionally ranging as high as 0.25 ppm were associated with respiratory effects in workers concomitantly exposed to chlorine. The United Paperworkers International Union (UPIU) supported the development of comprehensive standards for irritant gases such as chloride dioxide.

In the final rule, OSHA is retaining the 0.1-ppm 8-hour TWA and adding a 15-minute STEL of 0.3 ppm for chlorine dioxide. The Agency concludes that both of these limits are necessary to protect workers against the significant risk of respiratory, skin, and eye irritation known to occur as a result of short-term exposures above the TWA of 0.1 ppm. OSHA has determined that these adverse effects constitute material impairment of health.

CHROMIUM, METAL

CAS: 7440-47-3; Chemical Formula: Cr
H.S. No. 1093

OSHA formerly had an 8-hour TWA of 1 mg/m^3 for chromium metal. The proposed PEL was 0.5 mg/m^3 NIOSH (Ex. 8-47, Table N1) concurred with the proposed limit. The ACGIH has established an 8-hour TWA of 0.5 mg/m^3 for chromium, which is a steel-grey metal. In the final rule, OSHA is retaining the former 8-hour TWA PEL of 1 mg/m^3 for chromium metal.

According to the ACGIH, a 0.5-mg/m^3 TLV-TWA for chromium "should be adequate to prevent pulmonary disease or other toxic effects" (ACGIH 1986/Ex. 1-3, p. 139). Many commenters objected to the proposed 0.5-mg/m^3 PEL for chromium metal (Exs. 3-236, 3-829, 3-902, 3-1095, 3-1123, 129, 145, and 188; Tr. pp. 11-136 to 11-137). These commenters argued that there was no health basis for lowering the PEL for chromium metal and questioned the studies described in the health effects discussion for this substance. For example, Peter Hernandez, Vice President for Employee Relations at the American Iron and Steel Institute (AISI), commented in several submissions that one of the studies (Mancuso and Hueper (1951/Ex. 1-215) relied on by OSHA, which was performed for the Indian government in 1951, found "exaggerated pulmonary markings" on the X-rays of exposed workers but failed to demonstrate that

these markings constituted a health risk to these workers (Ex. 188, p. 18). The AISI also criticized the results of another study relied on by OSHA, the work of Princi et al. (1962, as cited in ACGIH 1986/Ex. 1–3, p. 139), which detected pulmonary disease in workers exposed to chromium at levels of 0.27 mg/m³ (Princi, Miller, Davis, and Cholak 1962, as cited in ACGIH 1986/Ex. 1–3, p. 139). According to the AISI, "the results of this study are highly questionable * * * because other dust and fumes besides chromium were present, including 36.7 percent silica" (Ex. 188, p. 19).

In response to these comments, OSHA has further reviewed the toxicological literature on chromium metal. The Agency finds that the metallic form of chromium, in its pure state, does not present a significant risk to exposed workers at levels below 1 mg/m³, OSHA's former 8-hour TWA PEL for this substance. This view of chromium metal's toxicity is shared by several toxicologists. For example, Proctor, Hughes, and Fischman (*Chemical Hazards of the Workplace*, 2nd ed., 1988, p. 155) state, "Chromium metal is relatively nontoxic * * * OSHA finds that the markings associated with exposure to chromium metal (which were not suggestive of alteration of the architecture of the lung) and reported in the Mancuso and Hueper (1951/Ex. 1–215) study do not present a risk of material impairment of health because they do not presage any decrement in pulmonary function or interfere with the functional capacity of exposed workers.

OSHA also agrees with the AISI that [A] major problem [in] defining the health effects which may be associated with exposure to metallic chromium is the frequent co-existence of the metallic form with both trivalent and hexavalent salts (Tr. p. 11–136).

The Princi et al. study (1962, as cited in ACGIH 1986/Ex. 1–3, p. 139) reflects the problem of confounding exposures to which the AISI alludes. In this study, ferrochrome alloy workers were exposed to several toxic contaminants simultaneously, including chromium, salts, silica, iron oxide, and chromium metal. OSHA believes it likely that exposure to the other contaminants present, which included a high percentage of silica, accounts for the development of pulmonary disease in these workers. The ACGIH (1986/Ex. 1–3, p. 139) stated, after reviewing the Mancuso and Hueper (1951/Ex. 1–215) and the Princi et al. (1962, as cited in ACGIH 1986/Ex. 1–3, p. 139) studies, that "[e]xposure to chromium metal does not give rise to pulmonary fibrosis or pneumoconiosis."

Thus, after a reanalysis of the toxicological data and the record evidence, OSHA concludes that there is no health basis for reducing the Agency's former limit of 1 mg/m³ for chromium metal. OSHA finds that the 1-mg/m³ PEL provides appropriate worker protection from the toxic effects of exposure to chromium metal.

Accordingly, in the final rule, OSHA is retaining the former 8-hour TWA limit of 1.0 mg/m³ for chromium metal. The Agency concludes that this limit protects workers against the significant risk of pulmonary effects potentially associated with exposure to the metallic form of chromium.

COAL DUST, < 5% QUARTZ

COAL DUST, > 5% QUARTZ

CAS: None; Chemical Formula: None
H.S. Nos. 1096 and 1097

OSHA's former limits for coal dust included a formula limit of 10 mg/m³/% $SiO_2 + 2$ for coal dust containing a respirable quartz fraction greater than 5 percent and a 2.4-mg/m³ limit for coal dust containing a respirable quartz fraction of less than 5 percent. The ACGIH has a TLV-TWA of 0.1 mg/m³ for the respirable quartz fraction of coal dust containing more than 5 percent quartz, and 2 mg/m³ for the respirable dust fraction of coal dust containing less than 5 percent quartz. OSHA proposed 8-hour TWA limits of 0.1 mg/m³ for the respirable quartz fraction of coal dust containing more than 5 percent quartz and 2 mg/m³ for the respirable dust fraction containing less than 5 percent quartz; the final rule establishes these limits. OSHA's proposed and final rule limits do not represent an actual change in the value of the limits for coal dust containing more than 5 percent respirable quartz; instead, they do away with the Agency's previous and cumbersome formula limit. Coal is a dark brown to black solid formed from fossilized plants.

Because OSHA is not lowering the limits for coal dust or considering the health effects evidence for these limits but is merely changing the form in which the limits are expressed, no discussion of the health evidence is included in the final rule. The Gulf Power Company (Exs. 3–938 and 3–1144) believed that OSHA was proposing to change the value of the coal dust limits rather than the form in which those limits were being expressed. In the final rule, OSHA has clarified this fact by emphasizing it in the beginning and end of this discussion. Lawrence Hecker, Corporate Director of Industrial Hygiene and Toxicology for Abbott Laboratories, requested that both Z-table entries for coal dust in the final rule specifically indicate that it is the "respirable quartz fraction" that is to be measured (Ex. 367f, p. 9). In response to this comment, OSHA has so identified the measurable fraction in the final rule's Table Z–1–A.

NIOSH (Ex. 8–47, Table N2; Tr. p. 3–86) believes that the limit for quartz-bearing coal dust should be reduced to 0.05 mg/m³ as an 8-hour TWA on the basis of the potential carcinogenicity of respirable crystalline silica. OSHA is aware of some recent studies (NIOSH 1986b; Hurley and Maclaren 1987; IARC 1987) on the health effects of exposure to coal dust, and the Agency is monitoring this literature to assess the need for a reevaluation of this limit.

In the final rule, OSHA is establishing an 8-hour TWA PEL of 0.1 mg/m³, measured as the respirable dust fraction, for coal dust having a respirable quartz fraction of more than 5 percent quartz, and an 8-hour TWA PEL of 2 mg/m³ TWA, measured as the respirable dust fraction, for coal dust having a respirable quartz fraction of less than 5 percent quartz. The Agency's previous formula limit for silica containing more than 5 percent quartz (respirable fraction) is equivalent to the 0.1-mg/m³ limit in terms of airborne concentration. Thus, the final rule's limit is intended to simplify the units used to measure and express the limit; it does not represent an actual change in the value of the limit (see discussion for crystalline silica-quartz later in this section). OSHA believes that this revision will simplify employee exposure monitoring.

ETHYL ACRYLATE

CAS: 140–88–5; Chemical Formula: $CH_2=CHCOOC_2H_5$
H.S. No. 1161

OSHA formerly had an 8-hour TWA limit for 25 ppm for ethyl acrylate, with a skin notation. The ACGIH has a TLV–TWA of 5 ppm, a TLV–STEL of 25 ppm, and a skin notation for ethyl acrylate, which is a colorless liquid. The proposed PEL was an 8-hour TWA of 5 ppm and a 15-minute STEL of 25 ppm, with a skin notation; the final rule establishes these limits.

Ethyl acrylate produces irritation of the skin, eyes, mucous membranes, gastrointestinal tract, and respiratory system (Dreisbach 1974/Ex. 1–896). The oral LD_{50} in rats fed this substance is 1020 mg/kg, and the 4-hour inhalation LC_{50} for these animals ranges between 1000 ppm and 2000 ppm. In rabbits, the dermal LD_{50} is 1790 mg/kg (Pozzani, Weil, and Carpenter 1949/Ex. 1–925), and the minimum oral LD_{50} is 280 to 420 mg/kg (Treon, Sigmon, Wright, and Kitzmiller 1949/Ex. 1–769). Animal

studies also indicate that severe chronic effects may result from exposure to this substance. Rats exposed to levels of 70, 300, or 540 ppm of ethyl acrylate for up to 30 days showed accelerated mortality and pathologic changes in the lungs, liver, and kidneys. In those animals that developed pneumonia, renal and hepatic lesions were also seen. In a parallel study, rats, rabbits and guinea pigs who were subjected to ethyl acrylate concentrations in excess of 75 ppm for 50 seven-hour inhalation periods exhibited pulmonary edema; degenerative changes in the heart, liver, and kidneys; and death (Treon, Sigmon, Wright, and Kitzmiller 1949/Ex. 1–769). Miller et al. (1980, as cited in ACGIH 1986/Ex. 1–3, p. 240) reported that rats and mice exposed to 75 or 225 ppm, six hours per day for 30 days, developed nasal lesions and other degenerative inflammatory changes in the nasal structure. In other studies, rats and mice administered 100 or 200 mg/kg ethyl acrylate by gavage five times per week for 103 weeks developed inflammation and hyperplasia of the forestomach in addition to squamous cell carcinomas and papillomas in the same area (NTP 1983b, as cited in ACGIH 1986/Ex. 1–3, p. 240). Based on a study by Miller et al. (1980, as cited in ACGIH 1986/Ex. 1–3, p. 240), in which rats and mice exposed to 25 or 75 ppm ethyl acrylate for six hours per day, five days per week for 27 months developed lesions in the nasal cavity even at the lowest dose, the ACGIH (1986/Ex. 1–3, p. 240) concurs with the American Industrial Hygiene Association (1966/Ex. 1–1195) that a 25-ppm limit for ethyl acrylate is too high to prevent irritating effects in exposed humans.

In a study by Nemec and Bauer (1978, as cited in ACGIH 1986/Ex. 1–3, p. 240), human volunteers experienced drowsiness, headache, and nausea after prolonged inhalation exposures at 50 to 75 ppm. Opdyke (1975/Ex. 1–922) reported that the application of a 4-percent concentration of ethyl acrylate produced skin-sensitization reactions in 10 out of 24 volunteers.

NIOSH (Ex. 8–47, Table N6B; Tr. pp. 3–97 to 3–98) believes that a full Section 6(b) rulemaking is needed for this potential occupational carcinogen. A comment from Basic Acrylic Monomer Manufacturers (Ex. 184) urges OSHA not to adopt values still on the ACGIH *Notice of Intended Changes*. As discussed in Section IV, OSHA is not adopting these limits.

In the final rule, OSHA is establishing an 8-hour TWA of 5 ppm and a 15-minute STEL of 25 ppm for ethyl acrylate; the skin notation is being retained. The Agency concludes that these limits will protect workers from the significant risk of severe eye, nose, and skin irritation associated with exposure to this substance at the levels permitted by OSHA's former limit. The Agency considers these adverse effects material impairments to health.

FERROVANADIUM DUST

CAS: 12604–58–9; Chemical Formula: FeV
H.S. No. 1177

OSHA formerly had a limit of 1 mg/m^3 for ferrovanadium dust. The ACGIH has a TLV–TWA limit of 1 mg/m^3 with a TLV-STEL of 3 mg/m^3; the NIOSH-recommended exposure limit for metallic vanadium is 1 mg/mg^3 as a 10-hour TWA. The proposed PEL was 1 mg/m^3, with a STEL of 3 mg/m^3. NIOSH (Ex. 8–47, Table N1) concurred with these limits, which are established by the final rule. Ferrovanadium dust exists as dark, odorless, solid particles.

Soviet studies in animals showed ferrovanadium dust to be less toxic than vanadium pentoxide. Roshchin (1952/Ex. 1–1166) reported that no acute intoxication occurred in animals exposed to ferrovanadium dust at concentrations as high as 10,000 mg/m^3; however, serious chronic pulmonary changes were observed after short-term exposures (one hour) on alternate days for two months to concentrations in the 1000- to 2000-mg/m^3 range. These pulmonary changes consisted of chronic bronchitis and chronic lung inflammation. Only NIOSH commented on this substance.

OSHA is establishing a PEL of 1 mg/m^3 TWA and a STEL of 3 mg/m^3 for ferrovanadium dust to reduce the significant risk of chronic pulmonary damage shown to be associated with exposures to this substance at the elevated short-term levels formerly permitted by the TWA limit alone. OSHA considers the pulmonary damage caused by exposure to ferrovanadium dust to be material impairments of health. The Agency concludes that the combined TWA limit and STEL will substantially reduce this risk.

FIBROUS GLASS

CAS: None. Chemical Formula: None
H.S. No. 1178

The Agency proposed a PEL of 5 mg/m^3 (the TLV established by the ACGIH) for total fibrous glass. NIOSH (1977d/Ex. 1–261) has recommended that employee exposures to fibrous glass dust not exceed 5 mg/m^3 as an 8-hour TWA (as total dust) or 3 fiber/cc for fibers greater than 10 um long.

Extensive evidence was submitted to the record regarding the proposed PEL for fibrous glass. Because of the conflicting nature of some of the evidence and the complexity of the issues raised, OSHA has not yet been able to reach a final conclusion. Therefore, OSHA is temporarily delaying a final decision regarding the establishment of a separate PEL for fibrous glass; however, OSHA will make this final decision within a reasonable period of time.

GRAIN DUST (OAT, WHEAT, AND BARLEY)

CAS: None; Chemical Formula: None
H.S. No.: 1190

A decision by the Occupational Safety and Health Review Commission (*Secretary of Labor* v. *Krause Milling Company*, OSAHRC Docket No. 78–2307, April 22, 1986) has held that there was no former OSHA PEL for grain dust. Based on the ACGIH recommendation, OSHA proposed to establish a 4-mg/m^3 8-hour TWA PEL for dust generated from wheat, oats, and barley, and NIOSH (Ex. 8–47, Table N1) supported the proposal. However, in the final rule the Agency is establishing an 8-hour TWA limit of 10 mg/m^3 for these dusts. Grain dusts is a complex mixture of husk particles, cellulose hairs and spikes, starch granules, spores of fungi, insect debris, pollens, rat hair, and approximately 5 percent mineral particles. The mean particle size of the airborne dusts may be less than 5 um. A substantial amount of information was submitted to the record addressing the health evidence and feasibility of attaining a 4-mg/m^3 TWA limit in the feed industry (Exs. 3–751, 3–752, 3–755, 8–55, 104, 109, 118, 180, 185, and 198; Tr. pp. 6–247 to 6–319). OSHA has carefully reviewed this evidence and has determined that an exposure limit for grain dust is necessary to reduce the significant risk of adverse respiratory effects associated with exposure to this material. OSHA's review of the health evidence, described below, shows that grain workers will experience adverse respiratory symptoms upon exposure to grain dust levels exceeding the current nuisance dust limit of 15 mg/m^3 TWA; this observation was not disputed in the record. Respiratory symptoms are also prevalent among grain dust workers exposed to levels below 10 mg/m^3 TWA, as total dust, although these symptoms are diminished compared with those associated with exposure to higher dust levels. Because of uncertainties in establishing a clear threshold exposure level for respiratory effects and in determining the feasibility of the proposed 4-mg/m^3 limit (see Section VII, Summary Economic Impact and Regulatory Flexibility Analysis), OSHA

is establishing a 10-mg/m³ limit as an 8-hour TWA for wheat, oat, and barley dust to reduce the risk of respiratory disease.

The adverse effects of inhaling grain dust have been known for at least two-and-one-half centuries, dating back to Rammazini who, in 1713, described the respiratory hazards associated with exposure to cereal grain dust. More recently, several epidemiological studies conducted over the past few decades (cited by ACGIH 1986/Ex. 1–3 and Rankin et al. 1986) have demonstrated that exposure to grain dust causes "grain fever," wheezing, chest tightness, productive cough, eye and nasal irritation, and symptoms of chronic respiratory disease. Grain dust may also induce asthmatic reactions via an allergic mechanism, particularly in individuals who are predisposed to developing allergies (i.e. atopic individuals). Thus, OSHA believes that the need for an occupational limit on exposure to grain dust is clear.

The basis for OSHA's proposed 4-mg/m³ limit was a NIOSH-sponsored study of grain workers by Rankin et al. (*Study of the Prevalence of Chronic, Non-Specific Lung Disease and Related Health Problems in the Grain Handling Industry*, DHHS (NIOSH) Pub. No. 86–117, 1986). A 1980 draft of this study by Rankin and do Pico (Ex. 1–1193) formed the basis for the ACGIH-recommended limit of 4 mg/m³ TWA. This study evaluated the health status of 310 grain handlers in Wisconsin and Minnesota. The grain handlers were selected from eight elevator companies, from state grain inspection agencies, and from longshoring companies. Health status was determined by questionnaire and by physical examination, which included an assessment of pulmonary function, immunologic evaluation, blood and urine chemistries, and chest roentgenograms. The comparison group that served as controls consisted of 239 city workers who spent the majority of their workdays outside.

From the questionnaires, Rankin et al. (1986) found that the grain handlers had a higher prevalence of respiratory symptoms than did the city workers. The prevalence of respiratory symptoms was highly significant (Rankin et al. 1986, Table 13), and was independent of smoking status. The symptoms reported by grain handlers represented both acute and chronic airways reactions (occupational asthma and chronic bronchitis). Wheezing and/or chest tightness generally started within two hours of beginning the work shift. Episodes of grain fever occurred infrequently; this was attributed by the workers to improved working conditions over the previous three years. Acute recurrent conjunctivitis and rhinitis were reported to occur among most grain workers.

Lung function tests showed that exposure to grain dust had a highly significant adverse effect on pulmonary function (Rankin et al. 1986, Table 30). There was, however, no correlation between reduced pulmonary function and job category, length of employment, or place of work. The lung function decrement observed among grain handlers was not related to smoking history alone; grain handlers who were smokers or ex-smokers showed significant declines in pulmonary function when compared to city workers who were smokers or ex-smokers.

Grain workers who reported symptoms had lower values of ventilatory function than did workers without symptoms. The prevalence of chronic bronchitis symptoms with measured airways obstruction was higher in grain workers than in controls, regardless of smoking history. Chronic bronchitis with airways obstruction was also related to length of employment. Rankin et al. (1986) concluded that these findings "suggest that chronic grain dust exposure may result in chronic obstructive pulmonary disease" (p. 26).

Rankin et al.'s (1986) study also included a work-shift study in which 248 grain workers and 192 city workers were sampled for grain dust exposure during a work shift. Symptoms occurring during the shift were recorded and pulmonary function readings were taken before and after the shift. Only 14 percent of grain workers were exposed to an 8-hour TWA level exceeding 5 mg/m³ total grain dust; 7 percent were exposed above 10 mg/m³. Rankin et al. (1986) reported that grain workers showed a significant excess of cough and expectoration during a work shift in which dust concentrations were below 5 mg/m³. At dust levels between 10 and 15 mg/m³, there was a significantly increased prevalence of wheezing and dyspnea during the shift among grain workers as compared with controls (Rankin et al. 1986, Table II–156). Workers with pre-existing airways obstruction experienced significant pre- to post-shift declines in ventilatory function at dust levels below 10 mg/m³. However, the changes observed in pre- to post-shift pulmonary function did not correlate with the presence of symptoms during the shift.

Rankin et al. (1986) also conducted a short-term (three-year) follow-up study of lung function among grain workers. Their results showed no greater declines in FEV or FVC over the three-year period than could be accounted for by age alone. However, there was a significant decline in other measures of lung function (MMF, V_{max50}, V_{max75}) among both smoking and nonsmoking grain workers. The authors concluded that, although a grain-dust-related decline in these measures was observed, the long-term effects of smoking on lung function were probably greater than those caused by grain dust.

The ACGIH (1986/Ex. 1–3) recommended the 4-mg/m³ TLV based largely on the following conclusion by Rankin et al. (1986):

> The incidence of respiratory symptoms was higher among grain workers exposed to mean total airborne dust (time-weighted average concentration) of 13.9 mg/m³ when compared to grain workers exposed to 4 mg/m³ or less. In the latter group of grain workers the incidence of symptoms was similar to that found among controls (Rankin et al. 1986, p. 51).

This conclusion by Rankin et al. (1986) was derived by correlating the incidence of respiratory symptoms with workers' subjective estimations of dust levels encountered during the work-shift study: workers who judged their dust exposures during the shift to be "more than average" were exposed to mean dust levels of 13.9±12 mg/m³ TWA and had significantly higher incidences of respiratory symptoms than did workers who judged their exposures to be "average" (mean TWA dust exposures of 4±8.6 mg/m³). From this observation, the ACGIH (1986/Ex. 1–3) interpreted 4 mg/m³ to be a no-observed-effect level.

This interpretation of Rankin et al.'s (1986) results was heavily criticized by rulemaking participants. For example, the National Grain and Feed Association (NGFA) (Ex. 8–55) argued:

> OSHA states that the study found that acute bronchial symptoms did not appear among workers exposed at or below 4 mg/m³. This figure is in fact an average estimated exposure of 4.21 ± 8.62 mg/m³ and . . . was based on workers' arbitrary interpretation[s] of 'average' exposure. The researchers grossly overstated their results by implying that a specific level of 4 mg/m³ was an absolute limit below which the incidence of symptoms among workers was similar to [that among] controls (Ex. 8–55, p. 28).

Although it is true that reliance on employees' subjective impressions of the magnitude of dust exposure during a shift is not as precise as taking quantitative samples of dust exposure, it must be emphasized that Rankin et al. (1986) did find a significant excess of respiratory symptoms among grain workers whose TWA exposures were objectively determined, by air sampling,

to be less than or equal to 10 mg/m³ TWA; an excess incidence of wheezing and dyspnea were also reported among grain workers exposed to levels of between 10 and 15 mg/m³ TWA.

The NGFA also criticized the Rankin et al. (1986) study for failing to address potential biases in the design and administration of the health questionnaire (Ex. 8–55, p. 25). In Appendix C of its submission, the NGFA cites a discussion of questionnaire biases by Gamble and Battigelli (in *Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., vol. 1, pp. 129–32, Clayton and Clayton 1981) and states that "questionnaires provide a large source of error that must be guarded against," particularly when the questionnaire is self-administered (Ex. 8–55, Appendix C, pp. 3–4). OSHA believes that, although such biases are possible, Rankin et al. (1986) took measures to reduce such biases. First, their study population derives from many workplaces, including eight grain elevators, state grain inspection agencies, and longshoring companies; it thus appears unlikely that the overall results obtained from the questionnaires would be substantially biased as a result of employee dissatisfaction with the working conditions of a particular worksite. Second, Rankin et al. (1986) did rely on trained interviewers to review all questionnaires for completeness and to assist in the completion of a questionnaire when necessary. The use of trained interviewers, according to Gamble and Battigelli (Ex. 8–55, Appendix C, p. 3), may correct such biases. In addition, Rankin et al. (1986) found a correlation between symptoms reported on questionnaires and exposure levels, which suggests that the questionnaire results were not heavily biased.

Despite some of the criticisms of the Rankin et al. (1986) study, these authors' results are consistent with some other published studies of grain workers. Dr. Roy Buchan, Chief of the Occupational Health and Safety Section, College of Veterinary Medicine and Biomedical Sciences at Colorado State University, performed a study of the general health of 31 grain handlers (submitted as part of Ex. 3–751). A total of 204 personal TWA dust samples were taken, of which only six exceeded 10 mg/m³. Dr. Buchan found that neither age of facility, smoking history, nor past exposure to grain dust had any significant effect on symptom responses. There was a statistically significant association between grain dust exposure levels and sysmptom responses. The reported symptoms included nasal and throat irritation, chest discomfort, and phlegm production. Dr. Buchan concluded that, "although the association was mathematically weak but statistically significant, it would rationally be expected that symptom severity would become more pronounced as dust concentrations increase, since dust exposures in this invesitgation were suprisingly low (mean = 0.7 mg/m³ TWA)." In a larger study of 390 Canadian grain workers, Cotton, Graham, Li et al. (1983, submitted as part of Ex. 3–751) also reported a significant excess incidence of respiratory symptoms among grain workers despite total dust concentrations generally below 10 mg/³.

Although these studies show a consistent pattern of increased prevalence of respiratory symptoms among grain handlers exposed below 10 mg/m³, the association between low-level exposure to grain dust and the development of chronic pulmonary disease remains open to interpretation. Several studies, including Rankin et al. (1986), Chan-Yeung, Giclas, and Henson (1980/Ex. 1–474), and Broder, Corey, Davies et al. (1985, as cited in Ex. 3–751) have generally not found decrements in pulmonary function associated with long-term exposure to grain dust. In addition, chest roentgenograms have found no evidence of lung scarring of fibrosis (Rankin et al. 1986) among grain handlers. However, symptoms of chronic bronchitis have been frequently noted among grain handlers, including those who have never smoked (Rankin et al. 1986; Cotton, Graham, Li et al. 1983). According to Cotton et al. (1983, as cited in Ex. 3–751, p. 139), "The significance of the increase in chronic bronchitis and cough in workers and wheezing in nonsmoking workers in terms of eventual respiratory disability remains uncertain but the nuisance and discomfort of these symptoms for workers must also be considered."

Because of the conflicting evidence for an association between exposure to grain dust and the development of chronic lung damage, the NGFA (Exs. 8–55 and 180) and the American Feed Industry Association (AFIA) (Ex. 185) take the position that grain dust has been shown to be a nuisance dust. For example, in its posthearing brief, the AFIA stated:

> [F]eed industry workers are generally healthy, and experience no unique adverse health effects resulting from current levels of grain dust exposure. Therefore, setting a PEL for grain dust is unwarranted and unnecessary.
>
> The studies relied on by OSHA . . . fail to show that grain dust, at current levels, is a "harmful physical agent". . . Granted, grain dust may have *some* effect on some individuals' health; however, nothing in the record demonstrates that these effects, at typical current levels, are anything more than reversible and non-serious (Ex. 180, p. 14).

OSHA does not concur with this view. In the studies described above, as well as in others in the record, grain workers have consistently reported an excess prevalence of respiratory symptoms, including chronic bronchitis, at low levels of exposure to grain dust. OSHA believes that these symptoms, even in the absence of definitive evidence of irreversible lung damage, constitute material impairment of health and interfere with the well-being of workers. This was attested to at the informal hearing by Deborah Berkowitz, Director of Safety and Health for the Food and Allied Trades Department, AFL–CIO:

> I want to make it clear that study after study documents a very real acute hazard to grain workers. Living with chronic bronchitis is not a hazard that should go unchecked. In fact, study after study point to the possibility of very real long-term damage from chronic cumulative effects of exposure to grain dust. But even without the possibility of long-term disability, acute hazards clearly pose significant risk[s] to workers (Tr. pp. 6–306 to 6–307)

OSHA concludes that employees are placed at significant risk of respiratory symptoms, including chronic bronchitis, as a result of exposure to grain dust. It is clear that such symptoms occur at grain dust levels exceeding OSHA's former limit for dusts and particulates (15 mg/m³ TWA); in addition, workers have reported symptoms of wheezing and dyspnea upon exposure to dust levels between 10 and 15 mg/m³ TWA. Increases in respiratory symptoms have also been reported to occur among grain workers exposed generally to less than 10 mg/m³, although symptoms are diminished at these lower levels. At this time, it is difficult to identify the threshold at which adverse respiratory effects are likely to occur. This uncertainty is reflected in a posthearing submission by the NGFA (Ex. 118) in which Dr. George Bardwell of the University of Denver performed a statistical analysis of the FEV measurements reported by Chan-Yeung, Giclas, and Henson (1980/Ex. 1–474) in grain workers. Dr. Bardwell estimated that the threshold for reduced FEV is 6.41 mg/m³, with a 95-percent confidence interval of between 0 and 24.4 mg/m³.

In addition, considerable information was entered into the record addressing the technological feasibility of achieving the proposed 4-mg/m³ grain dust PEL (Exs. 3–751, 3–752, 3–755, 8–55. 109, 118,

180, 185, and 198). These data are conflicting, particularly with regard to smaller grain elevators. In light of these uncertainties, OSHA is establishing a 10-mg/m³ 8-hour TWA limit for grain dust, measured as total dust. OSHA finds that establishing this limit will substantially reduce the risk of adverse respiratory effects that occur at higher levels of exposure. OSHA has also concluded that a 10-mg/m³ TWA limit is technologically feasible (see Section VII).

The American Feed Industry Association (EX. 185) objected to OSHA's inclusion of oat and barley dust in the definition of grain dust, stating that the studies relied on by OSHA in the NPRM pertaining to oat and barley dust (Darke, Knowelden, Lacey, and Ward 1976; Cockcroft et al. 1983) were not relevant to addressing the effects of exposure to oat and barley dust at levels below 15 mg/m³. However, Rankin et al. (1986) reported in their study, which involved exposure to much lower levels of grain dust, that the types of dust most likely to bring on or aggrevate symptoms of cough and/or expectoration were durum wheat and barley, followed by spring wheat, rye, and oat. Least likely were corn, soybean, sunflower, and others. In addition, Mr. George Talley and Mr. Michael Garcia, industrial hygienists at Los Alamos National Laboratory, commented that, according to their personal experience, barley beards are more irritating than wheat dust (Ex. 3–1095). Therefore, OSHA finds that there is sufficient evidence to include oat and barley in the definition of grain dust.

At the informal hearing, Ms. Berkowitz raised the question as to whether OSHA intended to apply the grain dust limit to flour mills and bakeries (Tr. 6–310). To support this position, she submitted several reports describing asthma occurring among bakers; bakers' asthma has been attributed to flour dust exposure (Ex. 3–751). As with all other substances included in this rulemaking, OSHA intends the new limit for grain dust to apply to all workplaces, including flour mills and bakeries where there is the potential for exposure to grain dust.

In the final rule, OSHA is establishing an 8-hour TWA limit of 10 mg/m³ for grain dust, measured as total dust. Grain dusts other than oat, wheat, and barley are regulated under OSHA's generic "particulates not otherwise regulated" PEL of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction). The Agency concludes that this limit will substantially reduce the significant risk of acute and chronic respiratory symptoms and disease associated with exposure to grain dust at the levels formerly permitted by the absence of an OSHA limit. The Agency has determined that the respiratory effects caused by exposure to grain dust represent material impairments of health.

GRAPHITE, NATURAL

CAS: 7782–42–5; Chemical Formula: None
H.S. No. 1191

The former OSHA limit for natural graphite (total dust) was 15 million particles per cubic foot (mppcf), which is equivalent to 2.5 mg/m³ as respirable dust (assuming that respirable mass is one-half total particle mass). The proposed PEL was 2.5 mg/m³ for respirable natural graphite dust containing less than 1 percent quartz; NIOSH (Ex. 8–47, Table N1) concurred with this limit, and the final rule promulgates it. The ACGIH has a graphite TLV of 2.5 mg/m³ for respirable dust containing less than 1 percent quartz. Graphite is a mineral substance that is best known for its use as the "lead" in pencils.

Early reports established that graphite deposited in the lungs of occupationally exposed workers caused pneumoconiosis (Koopman 1924/Ex. 1–131). Subsequent research described the condition produced by exposure to graphite as anthracosilicosis, a pulmonary condition similar to that seen in coal miners, based on radiographic and histologic examinations in exposed individuals (Harding and Oliver 1949/Ex. 1–71). The fibrotic changes seen in graphite workers appear to be related to the silica content of the graphite; experimental animals that were administered graphite that did not contain silica did not develop fibrotic changes (Ray, King, and Harrison 1951/Ex. 1–46), while another study found that graphite containing only a small amount of silica produced fibrotic changes in exposed animals (Ottowicz and Paradowski 1961/Ex. 1–190). Radiologic changes were also observed among graphite mine and production workers exposed to graphite containing from 3.6 to 10 percent silica (Pendergrass, Vorwald, Mishkin et al. 1967/Ex. 1–77). OSHA received no comments on this substance except for those from NIOSH.

In the final rule, OSHA is revising its former limit of 15 mppcf to a limit of 2.5 mg/m³ for the respirable fraction of graphite containing less than 1 percent quartz; this change represents a change only in the units used to express or measure the limit, not a change in the value of the limit. OSHA is revising its limit to simplify the monitoring of employee exposures, because the use of impingers and microscopic analyses are not required to measure exposures that are expressed in mg/m³ rather than in mppcf.

INDIUM AND COMPOUNDS

CAS: 7440–74–6; Chemical Formula: In
H.S. No. 1213

There was no former OSHA limit for indium and compounds; however, the proposed and final rule PEL is 0.1 mg/m³ as an 8-hour TWA. NIOSH (Ex. 8–47, Table N1) concurred with this limit. The ACGIH recommends that exposures to indium not exceed 0.1 mg/m³ over an 8-hour shift. Indium metal is silver-white, shiny, and ductile.

Although there is no direct human evidence of the effect of indium compounds, severe effects have been produced by indium exposures in experimental animals. Rats that inhaled the sesquioxide form of indium at airborne concentrations ranging from 24 to 97 mg/m³ daily for a total of 224 hours developed widespread alveolar edema; these histologic lesions did not change over a 12-week post-exposure period (Leach, Scott, Armstrong et al. 1961, as cited in ACGIH 1986/Ex. 1–3, p. 322). Exposure of animals to indium reduces alveolar clearance and may be associated with chronic respiratory insufficiency, recurrent acute pneumonitis, and death (Jones 1960, as cited in ACGIH 1986/Ex. 1–3, p. 322). NIOSH was the only commenter on this substance.

Because of the severity of indium-induced injury and the persistence of such injuries, OSHA concludes that, in the absence of any exposure limit, exposed employees are at significant risk of developing chronic lung function impairment. The Agency is establishing an 8-hour TWA limit of 0.1 mg/m³ for indium and compounds to substantially reduce this risk.

IRON OXIDE (DUST AND FUME)

CAS: 1309–37–1; CHEMICAL FORMULA: Fe_2O_3
H.S. No. 1215

OSHA formerly had an 8-hour TWA limit of 10 mg/m³ for iron oxide fume. The ACGIH has established a limit of 5 mg/m³, measured as iron, total particulate. The proposed PEL was 5 mg/m³, and NIOSH (Ex. 8–47, Table N1) supported the proposed limit. However, the final rule retains OSHA's former limit of 10 mg/m³ for this substance. The fume of iron oxide is red-brown in color.

Animals exposed to iron oxide or to iron oxide mixed with less than 5 percent silica by inhalation or by intratracheal injection did not develop

pulmonary fibrosis (Naeslund 1940/Ex. 1–650; Harding, Grout, Durkan et al. 1950, as cited in ACGIH 1986/Ex. 1–3, p. 325). Inhalation of iron oxide dust also did not produce lung cancer in mice (Muller and Erhardt 1956/Ex. 1–648).

The evidence of iron oxide's toxicity in humans is conflicting. Drinker, Warren, and Page (1935/Ex. 1–315) concluded that exposures to iron oxide fume should be maintained below 10 mg/m^3, and a U.S. Department of Labor study (1941, as cited in ACGIH 1986/Ex. 1–3, p. 325) found that exposures below 30 mg/m^3 were without adverse effect. There are several studies, on the other hand, that report chest X-ray abnormalities in miners, welders, silver polishers, electrolytic iron oxide workers, foundry workers, and boiler scalers (Doig and McLaughlin 1936/Ex. 1–626; Stewart and Faulds 1934/Ex. 1–764; Doig and McLaughlin 1948/Ex. 1–627; McLaughlin, Grout, Barrie, and Harding 1945/Ex. 1–642; Davidson 1951, as cited in McLaughlin 1951/Ex. 1–727; Pendergrass and Leopold 1945/Ex. 1–653; Dunner and Hermon 1944/Ex. 1–731) exposed to iron oxide dust or fume. Some of these workers developed disabling pneumoconiosis; however, the exposures of many of these workers were mixed and in some cases included exposure to varying amounts of silica.

McLaughlin (1951/Ex. 1–727), whose opinion on the subject is widely accepted, believes that the presence of iron oxide dust or fume in the lung causes a pigmentation (termed siderosis) that is responsible for the changes seen in exposed individuals' chest X-rays. Siderosis is believed not to progress to fibrosis, and 6 to 10 years of exposure to about 15 mg/m^3 iron oxide dust is required before this condition develops (Fawcett 1943/Ex. 1–736; Fleischer, Nelson, and Drinker 1945/Ex. 1–1051; Hamlin and Weber 1950/Ex. 1–698). However, no studies are available that correlate exposure levels with X-ray changes.

Dr. Stuart M. Brooks (NIOSH 1986b, p. 425) notes that "[m]ore sophisticated physiologic testing, including measurement of the lung's mechanical properties, is required to better document lung function changes that may occur following inhalation of iron-containing dusts. *In vitro* studies or animal experimentation might be helpful in determining dose-response relationships, understanding lung clearance mechanisms for iron, and elucidating any fibrogenic properties of various ferrous compounds."

Some studies have shown that workers with exposures to iron oxide and such other substances as silica, radon gas, diesel exhaust, corn oils, and the thermal decomposition products of synthetic resins (Faulds 1957/Ex. 1–635; Dreyfus 1936/Ex. 1–897; Bidstrup 1959/Ex. 1–1030; Boyd, Doll, Faulds, and Leiper 1970/Ex. 1–716; Braun, Guillerm, Pierson, and Sadoul 1960/Ex. 1–1141; Monlibert and Roubille 1960/Ex. 1–647; Jorgensen 1973/Ex. 1–1023; Muller and Erhardt 1956/Ex. 1–648; Koskela, Hernberg, Karava et al. 1976/Ex. 1–744; Gibson, Martin, and Lockington 1977/Ex. 1–1053) have a greater risk of developing lung cancer. However, OSHA agrees with the ACGIH that, "at this time, it is not generally accepted that exposure to iron oxide dust or fume causes cancer in man" (ACGIH 1986/Ex. 1–3, p. 325). Stokinger (1984/Ex. 1–672) concluded that exposure to iron oxide dust and fume *per se* was not carcinogenic.

Several industry commenters (Exs. 8–22, 3–349, 3–829, 129, and 188; Tr. XI, pp. 137–138) objected to the proposed reduction in the PEL for iron oxide on the grounds that exposure to this substance does not cause fibrosis or pulmonary impairment, but rather siderosis, which is a benign pneumoconiosis. The American Iron and Steel Institute (Ex. 129, pp. 12–13) described siderosis as "simply a description of a condition that appears on radiographs." OSHA disagrees with Mr. Hernandez' assessment of the health effects potentially associated with exposure to iron oxide because the Agency believes that any occupational exposure that causes foreign substance to lodge in body tissues is undesirable. However, the Agency concurs with NIOSH's Dr. Brooks (NIOSH 1986b, p. 425) that additional research is necessary to determine why the lung is unable to clear iron-containing dusts after inhalation.

Accordingly, OSHA finds it appropriate to retain the Agency's former PEL for iron oxide dust and fume of 10 mg/m^3, measured as total particulate. The Agency concludes, based on the evidence currently available, that this limit will protect workers from developing of siderosis, a benign pneumoconiosis that occurs after many years of exposure to levels of iron oxide dust or fume in excess of 15 mg/m^3, and accumulation of iron dust in the lungs associated with ferric oxide exposure.

METHYLENE BIS-(4-CYCLOHEXYLISOCYANATE)

CAS: 5124–30–1; Chemical Formula: $C_{15}H_{22}N_2O_2$
H.S. No. 1272

OSHA had no former limit for methylene bis-(4-cyclohexylisocyanate). Prior to 1988, the ACGIH had a TLV ceiling of 0.01 ppm for this alicyclic diisocyanate compound. OSHA proposed a ceiling of 0.01 ppm, and NIOSH (Ex. 8–47, Table N1) supported the proposal. The final rule establishes that limit. OSHA Notes that ACGIH adopted a new limit for this substance in 1988 of 0.005 ppm TWA. The NIOSH RELs for methylene bis-(4-cyclohexylioscyanate) are a 0.005-ppm 10-hour TWA and a 0.02-ppm 10-minute ceiling.

Methylene bis-(4-cyclohexylisocyanate) is a pulmonary, skin, and eye irritant. The oral LD_{50} in rats is 9.9 g/kg. A 5-percent solution applied to the skin of guinea pigs produced strong erythema and edema, and rabbits treated with 0.1 mg showed severe skin reactions (Younger Laboratories 1965, as cited in ACGIH 1986/Ex. 1–3, p. 392).

Rats inhaling a lethal concentration of 20 ppm for five hours exhibited marked respiratory irritation, tremors, and convulsions during exposure, and their lungs revealed severe congestion and edema after death (E.I. du Pont de Nemours and Co. Inc. 1978, as cited in ACGIH 1986/Ex. 1–3, p. 392). Repeated inhalation exposure at 0.4 ppm produced initial weight loss in rats; exposure at 1.2 ppm caused respiratory irritation and decreased growth (E.I. du Pont de Nemours and Co. Inc. 1978, as cited in ACGIH 1986/Ex. 1–3, p. 392). Guinea pigs exposed to 0.12 ppm and mice exposed to 0.65 ppm did not exhibit dermal sensitivity (Stadler and Karol 1984/Ex. 1–612). Unlike toluene diisocyanate, which is a sensory irritant, methylene bis(4-cyclohexylisocyanate) depressed respiration by producing pulmonary irritation for example, an exposed mouse showed a 50-percent decrease in respiration rate, along with lung irritation, when exposed to 3.7 ppm of this substance (Weyel and Schaffer 1985/Ex. 1–581).

Human exposures to this compound have resulted in skin sensitization but only infrequently in pulmonary sensitization (Emmett 1976/Ex. 1–552; Israeli, Smirnov, and Sculsky et al. 1981/Ex. 1–701).

NIOSH (Ex. 150, Comments on Methylene Bis-(4-Cyclohexylisocyanate)) notes that both the REL and TLV for this substance have been based on the toxicological properties of toluene diisocyanate (TDI) and that "a recent study by NTP (1986a) of chronic effects in animals has produced evidence that cancer is associated with exposure to commercial grade TDI . . . and to a TDI hydrolysis product, 2,4-TDA . . . treatment of rats and mice of both sexes by gavage to

commercial grade TDI resulted in tumor induction, primarily in the pancreas and liver in male and female rats, and in female mice. The tumorigenic responses observed in both rats and mice treated with TDI meet the criteria of the OSHA cancer policy (29 CFR 1990) for classifying a substance as a potential occupational carcinogen." NIOSH suggests that the recommended RELs (0.005 ppm TWA and 0.02 ppm 10-minute ceiling) be considered as an interim level to be applied to methylene bis-(4-cyclohexylisocyanate) until adequate testing information is available. The AFL–CIO (Ex. 194) supported OSHA's proposed ceiling limit for this substance.

OSHA believes that a ceiling limit of 0.01 ppm is as protective as a 0.005-ppm TWA; the Agency therefore is establishing a ceiling limit of 0.01 ppm for methylene bis-(4-cyclohexylisocyanate). The Agency concludes that this limit will protect workers against the significant risk of eye, skin, and pulmonary irritation potentially associated with occupational exposures to this substance at the levels formerly permitted by the absence of an OSHA limit. The Agency considers these irritant effects caused by exposure to methylene bis-(4-cyclohexylisocyanate) to be material impairments of health.

MICA

CAS: 12001–26–2; Chemical Formula: $K_2Al_4(Al_2Si_6O_{20})(OH)_4$
H.S. No. 1276

OSHA formerly had a PEL of 20 mppcf TWA for mica containing less than 1 percent crystalline silica; this limit is equivalent to a 3-mg/m³ limit. The ACGIH recommends a limit of 3 mg/m³ TWA for the respirable dust of mica containing less than 1 percent quartz. OSHA proposed, and the final rule establishes, an 8-hour TWA limit of 3 mg/m³ for the respirable dust of mica containing less than 1 percent quartz. NIOSH (Ex. 8–47, Table N1) agreed with this decision.

Mica is a colorless, odorless, nonflammable, nonfibrous, water-insoluble silicate occurring in plate form and containing less than 1 percent quartz; it includes nine different species.

The final rule establishes an 8-hour TWA limit of 3 mg/m³ for respirable mica dust containing less than 1 percent quartz; this limit corresponds to the existing 20-mppcf PEL and is in keeping with the Agency's decision to delete mppcf values in favor of respirable dust values expressed in mg/m³. The Agency has decided to express this and other similar limits as mg/m³ to facilitate employee exposure monitoring.

MINERAL WOOL FIBER

CAS: None. Chemical Formula: None
H.S. No. 1277

OSHA proposed a limit of 10 mg/m³ TWA for mineral wool fiber, measured as total particulate containing less than 1 percent quartz; this was the same limit recommended by the ACGIH (1986/Ex.1–3). NIOSH recommends a 5-mg/m³ (8-hour TWA) limit, measured as total dust, as well as a 3-fiber/cc limit for fibers greater than 10 um long.

Extensive evidence was submitted to the record regarding the proposed PEL for mineral wool. Because of the conflicting nature of some of the evidence and the complexity of the issues raised, OSHA has not yet been able to reach a final conclusion. Therefore, OSHA is temporarily delaying a final decision regarding the establishment of a separate PEL for mineral wool fiber; however, OSHA will make this final decision within a reasonable period of time.

NICKEL (SOLUBLE COMPOUNDS)

CAS: 7440–02–0; Chemical Formula: Varies
H.S. No. 1283

The former OSHA PEL for all forms of inorganic nickel (as Ni) was 1 mg/m³ TWA. Based on the ACGIH recommendation, OSHA proposed revising this limit to 0.1 mg/m³ TWA; this limit is established in the final rule. NIOSH recommends that exposure to any form of inorganic nickel be maintained at or below 0.015 mg/m³.

A variety of toxic effects results from exposure to nickel compounds. Soluble nickel salts cause contact dermatitis in sensitized individuals and eye irritation (ACGIH 1986/Ex. 1–3, p. 422). Cases of asthmatic lung disease have been reported among nickel-plating workers (EPA 1986a/Ex. 1–1132).

OSHA's proposal to lower the PEL for soluble nickel compounds to 0.1 mg/m³ was based primarily on evidence that exposure to soluble nickel at low levels and for relatively short durations causes pathological changes in the lungs of experimental animals. In addition, OSHA reviewed several animal and human studies designed to investigate the carcinogenic potential of soluble nickel compounds. Three soluble nickel compounds have been tested for their carcinogenic potential: nickel chloride, nickel sulfate, and nickel acetate. Some sparingly soluble compounds, nickel carbonate and nickel hydroxide, have also been studied.

The results of animal studies suggest that some soluble nickel compounds are potentially carcinogenic; however the data are derived predominately from injection studies and results are conflicting. Results from occupational studies on soluble nickel compounds are also conflicting and are confounded by the presence of several types of nickel compounds in the facilities studied.

In the proposal, OSHA made a preliminary finding that exposure to soluble nickel compounds presented a potential cancer mortality risk to workers. Since publication of the proposal, however, OSHA has reviewed all of the record evidence, including an additional epidemiologic study, and has determined that further analysis is necessary before any definitive findings can be made with regard to the carcinogenic potential of the soluble nickel compounds. OSHA wishes to emphasize, however, that this determination does not negate the evidence that exposure of experimental animals to low levels of soluble nickel causes pathological changes in the lung. Accordingly, OSHA is establishing the 0.1-mg/m³ TWA PEL in the final rule, as proposed, but is basing this limit on the respiratory toxicity of these compounds. OSHA's findings on the evidence on soluble nickel compounds is presented below.

Bingham, Barkley, Zerwas et al. (1972/Ex. 1–204) exposed rats by inhalation to 0.1 mg/m³ nickel chloride for 12 hours per day for two weeks. Animals showed evidence of pulmonary irritation and damage in the form of marked mucous secretion, hyperplasia, and accumulations of alveolar macrophages. Fluid obtained by lung lavage appeared very cloudy and viscous due to the presence of free alveolar cells. Rats and guinea pigs exposed daily to 1.0 mg/m³ (as Ni) nickel chloride for six months showed increased lung weight, which is an indication of pulmonary damage and hyperplasia (Clary 1977, as cited in ACGIH 1986/Ex. 1–3, p. 422); exposed rats also developed signs of interstitial fibrotic lesions. Rabbits inhaling 0.3 mg/m³ (as Ni) nickel chloride aerosol for 30 days showed a doubling in alveolar cell number and volume of alveolar epithelial cells, as well as nodular accumulation of macrophages and laminated structures (Johansson, Curstedt, Robertson, and Camner 1983/Ex. 1–273). These studies clearly show that exposure at or below the former OSHA PEL of 1.0 mg/m³ for soluble nickel, even for durations considerably less than a working lifetime, is associated with increased cell turnover and pathological changes in the lung. These pathological changes, in particular the appearance of fibrotic lesions, observed in animals exposed to low levels of soluble nickel salts indicate that lung damage has occurred

and suggests that significant decrements in lung function may result from prolonged exposure to these low levels. Furthermore, the appearance of hyperplasia is indicative of abnormal cell growth and suggests the presence of pre-cancerous lesions.

Nickel chloride has been reported to be mutagenic in *Salmonella typhimurium* and *Cornebacterium*, but negative in *E. coli* (EPA 1986a/Ex. 1–1132). The positive studies are not considered conclusive, however, because the *S. typhimurium* report is an abstract lacking detailed data and *Cornebacterium* is not the usual species used in these tests. Amacher and Paillet (1980/Ex. 1–286) reported that nickel chloride was mutagenic in mouse lymphoma cells and demonstrated a dose-response relationship for this endpoint.

Some *in vitro* studies using soluble nickel compounds report finding chromosomal aberrations (EPA 1986a/Ex. 1–1132). These studies do not demonstrate a dose-response relationship or statistical significance, which weakens their findings. Several *in vivo* studies have failed to detect chromosomal aberrations (EPA 1986a/Ex. 1–1132). However, several *in vitro* studies on nickel sulfate and nickel chloride have reported findings of sister chromatid exchanges (EPA 1986a/Ex. 1–1132).

Some animal studies on soluble nickel compounds suggest that these compounds are carcinogenic in animals. Strain A mice receiving intraperitoneal injections of nickel acetate had an increased rate of lung adenomas and adenocarcinomas that was statistically significant in the high-dose group (Stoner, Shimkin, Troxell et al. 1976/Ex. 1–203). The animals were injected three times per week for eight weeks at 72, 180, or 360 mg/kg.

EPA (1986a/Ex. 1–1132) reported a study in which rats were given monthly intramuscular injections of 35 mg/kg nickel acetate for four to six months (Haro, Furst, and Falk 1968/Ex. 1–1022). Twenty-two percent of the treated rats developed sarcomas. Payne (1964/Ex. 1–200) observed tumor responses in rats after intramuscular implantation of 7 mg nickel acetate, nickel sulfate, nickel chloride, or nickel carbonate. Implant-site sarcomas developed in one of 35 rats exposed to nickel acetate, one of 35 rats exposed to nickel sulfate, none of 35 rats exposed to nickel chloride, and four of 35 rats exposed to nickel carbonate.

Results of other studies on nickel sulfate have been negative. Three studies used intramuscular injection in rats and reported that no tumors developed in the treated group (Gilman 1962/Ex. 1–205; Gilman 1966, as cited in EPA 1986/Ex. 1–1132; Kasprzak, Gabryel, and Jaraczewska 1983/Ex. 1–201). An ingestion study also repoted no tumors among treated rats or dogs (Ambrose, Larson, Borzelleca et al. 1976/Ex. 1–211).

Gilman (1966, as cited in EPA 1986a/Ex. 1–1132) administered 5 mg nickel hydroxide to rats by intramuscular injection in each thigh. Nineteen out of 40 injection sites developed sarcomas. Kasprzak, Gabryel, and Jaraczewska (1983/Ex. 1–201) gave rats intramuscular injections of nickel hydroxide in gel, crystalline, or colloidal form. Five out of 19 animals receiving the gel developed sarcomas (two with metastasis to the lung), three out of 20 receiving the crystalline form developed sarcomas (one with metastasis to the lung), and none of 13 rats receiving the colloid developed tumors.

Inco United States, Inc. (with its subsidiary, Inco Ltd.) (Exs. 3–915 and 167) and the Nickel Producers Environmental Research Association (NiPERA), Inc. (Ex 3–668) discussed the limitations of the animal data. For example, both of these commenters noted that soluble nickel compounds have produced tumors in animals only by injection and that the results among studies were conflicting. In the NPRM and in the discussion above, OSHA recognized many of these limitations of the data. Although it is true, as Inco pointed out (Exs. 3–915 and 167), that EPA (1986a/Ex. 1–1132) concluded that the animal data are "too limited to support any definitive judgment regarding * * * [the] carcinogenic potential [of soluble nickel compounds]" (EPA 1986a/Ex. 1–1132, p. 8–229), EPA also concluded that:

> The observation of pulmonary tumors in strain A mice from the administration of nickel acetate by intraperitioneal injections and the ability of nickel acetate to transform mammalian cells in culture and to inhibit RNA and DNA synthesis provides limited evidence for the carcinogenicity of nickel acetate and supports a concern for the carcinogenic potential of other soluble nickel compounds (EPA 1986a/Ex. 1–1132, p. 8–229).

OSHA agrees with EPA's assessment that, although some studies are suggestive of a carcinogenic effect and an ability of soluble nickel to transform cells, overall the animal data are too equivocal at this time to support any firm conclusions that soluble nickel compounds do or do not cause cancer in experimental animals.

In addition to the animal evidence described above, OSHA reviewed studies conducted on workers exposed to soluble nickel compounds. Electrolysis workers at a refinery in Kristiansand, Norway, experienced a higher lung cancer risk than employees from the same facility who worked in three other job categories, including roasting and smelting workers (Magnus, Andersen, and Hogetveit 1982/Ex. 1–241). Electrolysis workers were exposed to an aerosol composed predominantly of nickel sulfate, which was estimated to contain soluble nickel at a concentration of 0.2 mg/m^3 (EPA 1986a/Ex. 1–1132); these workers also had higher plasma and urine levels of nickel than did roasting and smelting workers, who were predominately exposed to insoluble nickel subsulfides and oxides. However, exposure to nickel subsulfide and oxides may have occurred in the electrolysis buildings, and the electrolysis workers may also have worked in other process departments (Grandjean, Andersen, and Nielsen 1988/Ex 1–207). Roasting and smelting workers were exposed to an estimated average of 0.5 mg/m^3 (as Ni) of roasting dust.

The standardized mortality ratios (SMRs) for lung cancer were 550 for electrolysis workers, 390 for other process workers, and 360 for roasting and smelting workers. The pattern of SMRs for nasal cancer, which is a rare form of cancer in humans, was different among these groups: 2600 for electrolysis workers, 2000 for other process workers, and 4000 for roasting and smelting workers. The results seem consistent with studies that show that roasting and smelting workers have the highest concentrations of nickel in the nasal mucosa, presumably because of the relatively larger particles resulting from roasting. Conversely, electrolysis workers, who showed a larger lung cancer risk than roasting and smelting workers, have higher plasma and urine levels of nickel, suggesting that nickel aerosolized by this process pentrates to the deep lung (EPA 1986a/Ex. 1–1132).

In the NPRM, OSHA presented quantitative estimates of the cancer risk believed to be associated with exposure to soluble nickel; these estimates were based on the Magnus at al. (1983/Ex. 1–241) study of electrolysis workers. During the rulemaking proceeding, OSHA re-evaluated the underlying exposure data and now believes that, because the electrolysis workers may have been concurrently exposed to some insoluble forms of nickel, the data from the Magnus et al. (1982/Ex. 1–241) study may not be appropriate to use to develop a quantitative estimate of the cancer risk associated with exposure to the insoluble forms on nickel.

In contrast to the study of Norwegian nickel refinery workers, a study of 4,288

refinery workers at Port Colborne, Ontario, failed to find an increased lung or nasal cancer mortality rate among electrolysis workers (Roberts et al. 1982; Roberts et al. 1984). Excess incidences of larynx and kidney cancer deaths were reported to be elevated among electrolysis workers, but the numbers of observed deaths were small (two deaths observed for each cause of death). The Roberts et al. studies did report substantially increased incidences of lung and nasal cancer deaths among sinter plant workers exposed to insoluble forms of nickel, a finding consistent with that of Magnus et al. (1982/Ex./1–241) for the Norwegian workers and with many other studies (EPA 1986a/Ex./1–1132).

The stark contrast between these two studies is difficult to explain. According to Inco (Ex./3–915, p. 5), exposures to soluble nickel at the Ontario facility, where no excess risk was found among electrolysis workers, were probably similar to those at the Norwegian facility, where cancer mortality was increased. Exposure data taken during the late 1970s at the Ontario facility (Ex./3–915, Table 1c) indicate that, in most job categories, electrolysis workers were exposed to both soluble and insoluble forms of nickel; this is evidenced by the higher reported employee sampling results for total nickel than for soluble nickel. Thus, concurrent exposure to both soluble and insoluble forms of nickel existed at both the Ontario and Norwegian facilities. The size of the cohort at the Ontario facility was approximately twice that of the Norwegian study; thus, the Ontario study has sufficient power to detect the sizable increases in the incidences of nasal and lung cancer that were reported in the Norwegian study. It is possible, as EPA (1986a/Ex./1–1132) has suggested, that quantitative or qualitative differences in the conditions of exposure between the two cohorts accounts for the discrepant results; however, no information contained in the Ontario or Norwegian reports suggest that there were substantial differences in exposure to soluble nickel. Given the magnitude of the difference in the reported cancer mortality for these two groups of electrolysis workers, it is clear that additional investigation is required to identify the risk factors that account for the different mortality patterns observed in Ontario and Norway. Therefore, OSHA concludes that, at this time, the available human data do not permit any definitive conclusion to be made linking occupational exposure to the soluble forms of nickel with an elevated cancer mortality risk in humans.

The primary impetus to revise the PEL for soluble nickel was the finding that exposure of animals for relatively short periods of time to soluble nickel aerosols at levels equal to or below the former PEL of 1 mg/m^3 produced increased cellular growth and pathological changes that reflect the lung's defense against chemical insult; this finding is consistent across three animal studies conducted in several species (Bingham, Barkley, Zerwas et al. 1972/Ex./1–204; Clary 1977, as cited in ACGIH 1986/Ex./1–3, p. 422; Johansson, Curstedt, Robertson, and Camner 1983/Ex./1–273). Furthermore, these observations were made in animals that were exposed for as short a duration as two weeks and for no more than six months; thus, the consequences of continued, low-level exposure for a full lifetime are unknown. Both Inco (Exs. 3–915 and 167) and NiPERA, Inc. (Ex./3–668) agree that these studies provide an appropriate basis for establishing a 0.1-mg/m^3 PEL for soluble nickel. NIOSH (Ex. 8–47, Table N6B) does not concur with the selection of this limit and believes that a full 6(b) rulemaking is appropriate for the soluble (or inorganic) compounds of nickel.

OSHA concludes that these studies, one of which demonstrated pathological and perhaps precancerous changes following exposure to 0.1 mg/m^3, clearly demonstrate that exposure to the former PEL of 1.0 mg/m^3 presents a significant risk to workers of lung irritation accompanied by pathological changes that may presage cancer. OSHA has determined that these effects constitute material impairments of health and functional capacity. OSHA also concludes that the final rule's reduction in the PEL will substantially reduce these significant risks. Accordingly, OSHA is establishing a revised 8-hour TWA PEL of 0.1 mg/m^3 (as Ni) for the soluble nickel compounds in the final rule.

NITROGEN DIOXIDE

CAS: 10102–44–0; Chemical Formula: NO_2
H.S. No. 1289

Both the ACGIH and NIOSH have recommended occupational limits for nitrogen dioxide. The current ACGIH recommendation is for a 3-ppm TWA and a 5-ppm STEL. The NIOSH REL is 1 ppm as a 15-minute short-term limit. OSHA's former PEL was 5 ppm as a ceiling value. The Agency proposed, and the final rule establishes, a permissible exposure limit for nitrogen dioxide of 1 ppm as a 15-minute STEL. NIOSH (Ex. 8–47, Table N1) agreed with the selection of this PEL. Nitrogen dioxide is a reddish-brown gas.

The previous ACGIH TLV of 5 ppm as a ceiling concentration (the basis for the former OSHA limit) was based primarily on the animal studies of Gray, MacNamee, and Goldberg (1952/Ex.1–154), Gray, Goldberg, and Patton (1954/Ex. 1–165), and Wagner, Duncan, Wright, and Stokinger (1965/Ex. 1–102). Gray, MacNamee, and Goldberg (1952/Ex. 1–154), and Gray, Goldberg and Patton (1954/Ex. 1–165) demonstrated lung injury among rats exposed for eight or more weeks to an 8-ppm concentration of a mixture of NO_2 and nitric acid, but these authors did not see such lesions in rats exposed for six months to 4-ppm concentrations of this mixture. Wagner, Duncan, Wright, and Stokinger (1965/Ex. 1–102) reported transient, mild, acute effects but no adverse chronic effects in rats exposed to 1 ppm, 5 ppm, or 25 ppm pure NO_2 for 18 months. The ACGIH's recommendation that the 5-ppm TLV be defined as a ceiling rather than as an 8-hour TWA was based on reports that NO_2 accelerated lung tumor development among lung-tumor-susceptible mice; in the late 1960s, the ACGIH believed that a TLV-ceiling value would minimize the risk of accelerating lung tumor development.

The current ACGIH TLVs for NO_2 are a 3-ppm 8-hour TWA and a 5-ppm STEL, and they are based on human studies that indicate that normal respiratory function may be compromised at exposures below the current OSHA ceiling limit of 5 ppm NO_2. In particular, Kosmider, Ludyga, Misiewicz et al. (1972/Ex. 1–224) reported a slight reduction in vital capacity and maximum respiratory volume in 70 men exposed to 0.4- to 2.7-ppm concentrations of the oxides of nitrogen six to eight hours daily for four to six years. These authors also reported an unspecified number of cases of chronic bronchitis among men in this group. Another study by Vigdortschik, Ancheeva, Matussevistch et al. (1937/Ex. 1–49) reported possible cases of chronic bronchitis and emphysema among 127 workers generally exposed below 2.8 ppm NO_2; these workers were also believed to be exposed to sulfuric acid mist at levels sufficient to cause dental erosion.

The NIOSH REL for NO_2 of 1 ppm as a 15-minute STEL is based on the two human studies discussed above, as well as some human studies involving short-term exposure. Abe (1967/Ex. 1–98) found a 40-percent decrease in effective lung capacity among healthy adult males 30 minutes after a 10-minute

exposure to 4- to 5-ppm NO_2. Expiratory and inspiratory maximum viscous resistance also increased by 92 percent after exposure. NIOSH (1976j/Ex. 1-265) concluded that Abe's results "document a definite and undesirable effect" at exposures approaching the former OSHA limit. A significant decrease in carbon monoxide diffusing capacity was observed by Von Nieding, Krekeler, Fuchs et al. (1973/Ex. 1-770) in healthy adults exposed to 5 ppm for 15 minutes. NIOSH also cites the work of Von Nieding, Wagner, Krekeler et al. (1971/Ex. 1-1204) and by Von Nieding and Krekeler (1971/Ex. 1-1175), who reported significant increases in airway resistance among 88 chronic bronchitis patients after a 15-minute exposure to a concentration of NO_2 as low as 1.5 ppm. NIOSH (1976j/Ex-1-265) concluded that the specific concentration of NO_2 required to produce pulmonary changes in normal, healthy adults is unknown, but "is likely to be about the same or perhaps a slightly higher concentration than the one inducing pulmonary changes in humans with existing chronic bronchitis" (1.5 ppm). Therefore, NIOSH recommended a 1-ppm 15-minute short-term limit for nitrogen dioxide. To provide additional support for a short-term rather than a TWA limit, NIOSH cites several animal studies that indicate that the toxic effects associated with exposure to NO_2 are primarily determined by peak, and not average, concentrations of exposure.

In its posthearing submission, NIOSH (Ex. 150, Comments on Nitrogen Dioxide) reported on a recent study by Mohsenin (1988, as cited in Ex. 150) in which no significant pulmonary function changes were noted among 18 healthy subjects exposed to NO_2 for one-hour periods. NIOSH (Ex. 150) noted that, in 1984, the World Health Organization, after an independent review of cross-sectional occupational health surveys, recommended a short-term occupational exposure limit of 1.8 mg/m^3 (0.9 ppm) for NO_2 and 8-hour TWA limit of 0.9 mg/m^3 (0.45 ppm). NIOSH also reviewed studies that suggest that NO_2 is mutagenic and is embryotoxic and teratogenic in rats.

The AFL-CIO (Ex. 194) supported OSHA's proposed limit for NO_2. However, several commenters (Exs. 3-349, 3-670, 3-739, 3-666, 3-1144, 133, and 133A) objected to OSHA's proposal to establish the NIOSH REL for NO_2 in the final rule, believing that the ACGIH TLVs of 3 ppm TWA and 5 ppm STEL were sufficiently protective. For example, David L. Van Lewen, Manager of Industrial Hygiene for BASF, referred to the Von Nieding et al. (1971/Ex. 1-1204) study as evidence that a 1-ppm short-term limit was not necessary:

> The Von Nieding study (1971/Ex. 1-1204) of chronic bronchitis patients . . . showed increased airway resistance when exposed to concentrations of nitrogen dioxide between 1.5 and 5.0 ppm. Lower concentrations had no significant effect. When this sensitive population does not show significant effects at concentrations below 1.5 ppm, it is not reasonable to set a workplace limit at a STEL of 1.0 ppm (Ex. 3-666).

Mr. Lawrence J. Ogden, representing the Interstate Natural Gas Association of America (INGAA) (Ex. 3-739), and Mr. Vincent D. Lajiness of the American Natural Resources Company (ANR) (Ex. 3-670) criticized the studies described in the NPRM, and in particular the Von Nieding et al. (1971/Ex. 1-1204) study; both rulemaking participants indicated that the data base developed by EPA to establish EPA's ambient air quality limit for NO_2 is superior. Mr. Ogden stated that

> [a] far more extensive body of studies about NO_2 health effects is available than is cited by OSHA in the proposed rulemaking. Much of this literature has been pulled together by the Environmental Protection Agency (EPA). The EPA review and assessment of scientific studies on the health effects of NO_2 exists in the EPA NO_2 Criteria Document and the Staff Memorandum, which have been provided to the record. . . .
>
> EPA's action should be addressed in the OSHA proposal because it represents a more recent evaluation than NIOSH, a far more concentrated Agency evaluation by research and regulatory personnel, and an extensive scientific peer review process. As a result of its evaluation, EPA decided in 1982 that evidence was insufficient that a short-term air standard for NO_2 was needed. This conclusion has been re-examined annually by EPA and checked against the latest health studies related to NO_2 effects (Ex. 3-739, pp. 7, 10).

Mr. Ogden also referred OSHA to the 1979 National Academy of Science's Committee on Toxicology report on the health evidence for NO_2.

The EPA staff memorandum referred to by Mr. Ogden is the 1982 Office of Air Quality Planning and Standards (OAQPS) Staff Paper on the assessment of scientific information on NO_2 (EPA/450/5-82/002, Ex. 3-2e). This document summarizes the findings expressed in EPA's *Air Quality Criteria for Oxides of Nitrogen* (EPA/600/8-82/026, Ex. 3-2f). Based on these reports, EPA issued a final rule retaining its 1971 ambient air quality standard for NO_2, which is 0.053 ppm (100 mg/m^3) as averaged over a one-year period.

The EPA Staff Paper concludes that the 1971 Von Nieding et al. (Ex. 1-1204) study "provides convincing evidence that chronic bronchitics exposed to NO_2 concentrations of 1.6 ppm or greater for approximately 3 minutes experience increases in airway resistance" (Ex. 3-2e, p. 18). A number of other studies were cited by EPA in which healthy adults were exposed to NO_2 concentrations in the range of 0.5 to 2.5 ppm. Folinsbee, Horvath, Bedi, and Delehunt (1978, as cited in Ex. 3-2e) reported no significant physiological changes in healthy adults exercising for up to one hour during a two-hour exposure to 0.6 ppm NO_2. Suzuki and Ishikawa (1965, as cited in Ex. 3-2e) reported a 50-percent increase in inspiratory flow resistance in healthy adults 10 minutes after a 10-minute exposure to an NO_2 concentration between 0.7 and 2 ppm.

Small changes in pulmonary function and a slight increase in the prevalence of respiratory symptoms occurred among healthy adults exposed to 1 ppm NO_2 for two hours (Hackney, Thiede, Linn et al. 1978, as cited in Ex. 3-2e). Beil and Ulmer (1976, as cited in Ex. 3-2e) reported a statistically significant increase in airway resistance among healthy adults following exposure to 2.5 ppm NO_2 for two hours, but not following exposure to 1 ppm. Based on their review of these data, the EPA staff paper concluded:

> [T]he lowest level of NO_2 exposure that credible studies have associated with measureable impairment of pulmonary function appears to be in the range of 1.0-1.6 ppm. . . . Several CASAC members have expressed concern that a standard designed to prevent relatively small changes in pulmonary function (such as those observed in the Suzuki and Ishikawa (1965) and Von Nieding et al. (1971) studies) from occurring more than once per year would be unnecessarily stringent. The CASAC members indicated that they were more concerned about the health implications of repeated exposures to the peak concentrations observed in the two studies than the effects associated with a single exposure (Ex. 3-2e, p. 18).

EPA also reviewed research reports that have become available since publication of the EPA Criteria Document and Staff Paper, in particular the reports by Linn and Hackney (1983 and 1984) that reported finding no pulmonary effects among exercising healthy adults and asthmatics exposed to 4 ppm NO_2. EPA concluded that these studies present "mixed and conflicting results," and that a more complete assessment of these studies was not possible because "many * * * have yet to be published in the peer-reviewed scientific literature" (50 *FR* 25535/Ex. 3-2d).

Regarding EPA's decision not to issue a short-term ambient-air-quality limit for

NO_2, a review of the preamble to EPA's final rule shows that EPA addressed this issue only with regard to existing *ambient* short-term levels of NO_2. EPA reported that, under its current 0.053-ppm annual average limit, the vast majority of metropolitan areas would be expected to have fewer than two days with a daily maximum hourly value of 0.2 ppm or greater (50 *FR* 25536/Ex. 3-2d). Because of the uncertainties regarding the evidence for adverse effects at NO_2 concentrations below 1 ppm, EPA concluded that the current annual average limit would "provide some measure of protection against possible short-term health and welfare effects" (50 *FR* 25537/Ex. 3-2d). It is also worth noting that, since 1971, EPA has designated a 2-ppm (one-hour average) level for NO_2 as representing a "significant harm level" requiring an emergency response. Thus, OSHA finds that EPA's recent actions and reasoning regarding a short-term ambient limit for NO_2 supports the establishment of 1 ppm as a STEL.

OSHA has also reviewed the most recent analysis of NO_2 toxicity conducted by the National Academy of Science's (NAS) Committee on Toxicology for the Department of Defense (*Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants*, Vol. 4, pp. 83-96, National Academy Press 1985); the earlier 1979 review was cited by Mr. Ogden of the INGAA. In its more recent review, the NAS concluded that exposures to NO_2 at levels between 0.5 and 1.5 ppm have demonstrated "little or no persistent change in pulmonary function" (NAS 1985, p. 89). The NAS Committee on Toxicology recommended short-term public emergency guidance levels (SPEGLs) for NO_2 of 1 ppm, averaged over a 60-minute period, and 0.12 ppm as an 8-hour average.

OSHA concludes that the evidence reviewed by the EPA and the NAS and the several studies referenced by EPA and NAS reaffirm the conclusion expressed by NIOSH in its 1976 criteria document (NIOSH 1976j/Ex. 1-265) that "humans with normal respiratory function may be acutely affected by exposure [to NO_2] at or below . . . [5 ppm]. Furthermore, the conditions of workers with chronic respiratory diseases, such as chronic bronchitis, may be aggravated by exposure to nitrogen dioxide at a concentration of approximately one-third of the current Federal standard" (NIOSH 1976j/Ex. 1-265, p. 117). In addition to the studies by Von Nieding et al. (1971/Ex. 1-1204) and Abe (1967/Ex. 1-98) described in the NPRM, both EPA (Ex. 3-2e) and the NAS (1985) cite a number of other published reports that show that exposure to NO_2 at concentrations below 5 ppm causes increased airway resistance in both healthy adults and chronic bronchitis; these reports include the studies of Suzuki and Ishikawa (1965), Rokaw et al. (1968), Streseman and Von Nieding (1970), and Beil and Ulmer (1976). Furthermore, these and other studies cited by EPA (Ex. 3-2e) and the NAS (1985) generally indicate that exposure to 1 ppm NO_2 is not normally associated with significant airway resistance, even among workers with already-compromised respiratory function.

Thus, OSHA concludes that the former 5-ppm ceiling limit for NO_2 is not sufficient to protect employees from experiencing increased airway resistance, and that establishing the ACGIH TLVs of 3 ppm TWA and 5 ppm STEL, as suggested by rulemaking participants (Exs. 3-349, 3-670, 3-739, 3-666, and 3-1144), would not provide sufficient protection. OSHA also concludes that the risk of increased airway resistance would be substantially reduced by promulgation of a 1-ppm short-term limit for NO_2; a short-term limit is clearly indicated for NO_2 since all of the studies cited above demonstrate that increased airway resistance is associated with exposure to NO_2 for durations of between three minutes and two hours. OSHA considers the increased airways resistance caused by exposure to NO_2 to be a material impairment of health. Therefore, to reduce the significant risk associated with short-term exposure to NO_2, the agency is establishing a 1-ppm limit, averaged over a 15-minute period, for nitrogen dioxide in the final rule.

OXYGEN DIFLUORIDE

CAS: 7783-41-7; Chemical Formula: OF_2
H.S. No. 1300

The former PEL for oxygen difluoride was 0.05 ppm as an 8-hour TWA. The ACGIH has established a limit of 0.05 ppm as a ceiling value. The revision of the TLV for oxygen difluoride from an 8-hour TWA to a ceiling value reflects the general position of the ACGIH that ceiling TLVs are more appropriate for chemicals that cause acute but not chronic health effects. OSHA proposed a permissible exposure limit of 0.05 ppm ceiling for oxygen difluoride. NIOSH (Ex. 8-47, Table N1) concurred with the selection of this limit, and it is established in the final rule. Oxygen difluoride is an unstable, colorless gas with a foul odor.

Oxygen difluoride is a substance having extremely high acute toxicity; it is an acute irritant and causes fatal pulmonary edema and hemorrhage in animals exposed to 0.5 ppm for a few hours (ACGIH 1986/Ex. 1-3). A single exposure to 0.1 ppm also had an effect on the lungs, as evidenced by development in animals of a tolerance to the acute effects of this substance after an isolated exposure. Animals acutely exposed to oxygen difluoride have also exhibited gross changes in the kidneys and internal genitalia (LaBelle, Metcalf, Suter, and Smith 1945, as cited in ACGIH 1986/Ex. 1-3, p. 452; Lester and Adams 1965/Ex. 1-963). Only NIOSH commented on this substance.

Because of the extreme acute toxicity of this compound and the effects noted at 0.1 ppm, the former TWA-PEL of 0.05 ppm was not sufficiently protective of workers; this former limit would still permit the brief periods of high exposure that have been associated with severe lung damage, which the Agency has determined represents a material impairment of health. Therefore, to reduce the significant risk of acute lung damage associated with brief excursion exposures to oxygen difluoride, OSHA is establishing a ceiling limit of 0.05 ppm for this substance.

OZONE

CAS: 10028-15-6; Chemical Formula: O_3
H.S. No. 1301

The former OSHA PEL for ozone was 0.1 ppm TWA. In the interval since this limit was adopted in 1971, the ACGIH has recommended that 15-minute short-term exposures to ozone not exceed 0.3 ppm. NIOSH has no REL for ozone. OSHA proposed, and the final rule establishes, permissible exposure limits of 0.1 ppm TWA and 0.3 ppm STEL for ozone. The Agency notes that the ACGIH has placed ozone on its *1988-89 Notice of Intended Changes* and is proposing a new TLV of 0.1 ppm as a ceiling value. Ozone is a liquid or an explosive gas.

Ozone is highly injurious and lethal in experimental animals at concentrations as low as a few parts per million (Stokinger 1957/Ex. 1-97). A study in which young mice were exposed to 1 ppm ozone for one or two days reported damage to alveolar tissue (Bils 1970/Ex. 1-58). Human populations chronically exposed to lower concentrations of ozone have been observed to have changes in lung function. In one study, human volunteers exposed to 0.5 ppm ozone for three hours per day, six days per week, for 12 weeks showed significant changes in lung function (Jaffe 1967/Ex. 1-101). Other authors reported a 20-percent reduction in timed vital capacity in persons exposed to average concentrations of ozone of 1.5 ppm (range not indicated) for two hours

(Griswold, Chambers, and Motley 1957/Ex. 1–128). Welders exposed to maximal ozone concentrations of 9 ppm were observed to have pulmonary congestion (Kleinfeld and Giel 1956/Ex. 1–120).

OSHA received a number of comments on the proposed PEL for ozone. The Edison Electric Institute (EEI) (Ex. 133A, pp. 22–23) stated that the studies by Bils (1970/Ex. 1–58), Jaffe (1967/Ex. 1–101), and Griswold et al. (1957/Ex. 1–128), cited above, do not provide substantial evidence for the proposed PEL. With regard to Bils' (1970/Ex. 1–58) finding of damaged alveolar tissue in mice exposed to a 1-ppm concentration of ozone for one or two days, EEI notes that "OSHA does not explain how these data can be translated to humans in the workplace" (Ex. 133A, p. 22). In addition, EEI is concerned that "OSHA neither critically evaluates . . . nor explains why the changes in lung function reported by [the Jaffe (1967/Ex. 1–101)] study represent a significant risk . . . ," and OSHA has not presented a substitute for a STEL of 0.3 ppm. Finally, EEI questions the relevance of the study by Griswold et al. (1957/Ex. 1–128) to the formulation of the proposed PEL (Ex. 133A, p. 23). The Agency believes that these three studies point to the short-term effect (i.e., less than eight hours) of ozone exposure; the Bils (1970/Ex. 1–58) data demonstrate that the lung is the target organ; the Jaffe (1967/Ex. 1–101) data point to an effect level of 0.5 ppm and show that a STEL of 0.3 ppm will afford protection; and the Griswold et al. (1957/Ex. 1–128) data provide further evidence of reduced lung function as a result of short-term, acute exposure, rather than chronic exposure.

In addition, EEI commented that "OSHA's health assessment and feasibility analysis with respect to the facilities of the electric utility industry are deficient. Thus, EEI recommends that OSHA consider explaining that its ozone proposal does not apply to that industry" (Ex. 133A, p. 22). This same concern was reflected in the submission of the second commenter, Gulf Power Company (Ex. 3–938, p. 3). In response to these comments, OSHA emphasizes that the standards established in this rulemaking are based on the evidence of adverse health effects associated with exposure to toxic substances in the workplace. These effects would be the same, regardless of industry sector, if the exposure levels were the same. If, as EEI and Gulf Power Company contend, ozone exposures in power plants pose no significant risk to workers' health because they are controlled at or below the permissible exposure limits being promulgated in this rulemaking, then the electric utility industry is already in compliance and will not be impacted by the new PELs. The Agency has determined that the scientific evidence establishes the need for a short-term limit to substantially reduce the significant risk of pulmonary dysfunction that exists as a result of acute or chronic intermittent exposure to ozone.

The Gulf Power Company also expressed its belief that the 0.3-ppm short-term limit proposed by OSHA is unsubstantiated:

> Exposing someone to 1 ppm of ozone for 15 minutes may be just as valid a ceiling limit as 0.3 ppm. . . . We think that it is arbitrary to select a value of 0.3 ppm without further study (Ex. 3–938, pp. 3–4; see also Ex. 3–1144).

The Agency notes, again, that an effect level of 0.5 ppm is demonstrated by the Jaffe (1967/Ex. 1–101) data. Further justification for a STEL of 0.3 ppm is found in Proctor, Hughes, and Fischman *(Chemical Hazards of the Workplace,* 2nd ed., 1988), who report that, "except for one report, the threshold for effects in humans appears to be between 0.2 and 0.4 ppm" (Menzel 1984, and cited in Proctor, Hughes, and Fischman 1988, p. 388). The selection of 0.3 ppm as a short-term limit was neither invalid nor arbitrary, but rather, was based on the best available scientific evidence.

NIOSH (Ex. 8–47, Table N2) believes that ozone's toxicity requires an even more stringent limit. According to NIOSH, "Ozone is a chemical capable of inducing serious adverse health effects at low exposure concentrations, tenths of a part per million. . . ." The AFL-CIO (Ex. 194) agrees with NIOSH's assessment. OSHA agrees that ozone's health effects require a protective limit, and it is for this reason that the final rule promulgates TWA and STEL limits for ozone.

In the final rule, OSHA is retaining the 8-hour TWA limit of 0.1 ppm and establishing a 15-minute STEL of 0.3 ppm for ozone based on observations that significant declines in pulmonary function can result from repeated intermittent exposures or even from a single short-term exposure (Bils 1970/Ex. 1–58; Jaffe 1967/Ex. 1–101; Griswold, Chambers and Motley 1957/Ex. 1-128). OSHA believes that, in the absence of a STEL, employees will continue to be at significant risk of material impairment in pulmonary functional capacity associated with short-term exposures that could occur if exposures are controlled only by an 8-hour TWA. Thus the Agency concludes that it is necessary to supplement the former PEL with a STEL of 0.3 ppm to substantially reduce this risk.

PARAQUAT

CAS: 4685–14–7; Chemical Formula:
H.S. No. 12303

OSHA's former limit for paraquat was 0.5 mg/m^3 as an 8-hour TWA, with a skin notation. The ACGIH has established a limit of 0.1 mg/m^3 as an 8-hour TWA. The Agency proposed, and the final rule establishes, a permissible exposure limit of 0.1 mg/m^3 TWA for this substance; the skin notation is retained. NIOSH (Ex. 8–47, Table N1) concurs. Paraquat refers to a group of compounds that are odorless, yellow solids. The principal compounds are: 1,1'-dimethyl-4,4'-bipyridinium; 1,1'-dimethyl-4,4'-bipyridinium bis (methyl sulfate); and 1,1'-dimethyl-.4,4'-bipyridinium dichloride.

The Toxicity of these compounds depends on the compound's cationic moiety. Acute oral toxicity is reported as 30 mg/kg ion as cation for guinea pigs and 127 mg/kg ion for female rats, while the dermal LD_{50} in rabbits is 240 mg/kg ion (Clark 1964, as cited in ACGIH 1986/Ex. 1–3, p. 456; Clark, McElligott, and Hurst 1966/Ex. 1-503; McElligott 1965, as cited in ACGIH 1986/Ex. 1–3, p. 456). Paraquat can penetrate broken skin after it has broken down the skin's usual barriers (Swan 1969/Ex. 1–576; Clark, McElligott, and Hurst 1966/Ex. 1–503). By inhalation or intratracheal injection, paraquat is very toxic because of its irritant properties (Gage 1968/Ex. 1–508). Rats exposed once for six hours to a concentration of 1 mg/m^3 died if the aerosol contained particles with diameters of 3 to 5 microns (Gage 1968/Ex. 1–508). Rats exposed six hours/day for three weeks to the same aerosol at 0.4 mg/m^3 exhibited signs of pulmonary irritation; no effects were observed for the same exposure regimen at 0.1 mg/m^3 (Gage 1968/Ex. 1–508).

When the diameter of the particles in the aerosol are not of respirable size, toxicity is greatly reduced. The 4-hour LC_{50} for rats is 6400 mg/kg, and dogs, rats, and guinea pigs tolerated three weeks of daily exposures to 100 mg/m^3 without apparent pulmonary effect (although nosebleeds were observed) (Palazzolo 1965, as cited in ACGIH 1986/Ex. 1–3, p. 456).

Dietary administration, for 90 days, of doses ranging from 300 to 700 ppm showed dose-related effects ranging from pulmonary edema to intraalveolar hemorrhage and death (Kimbrough and Gaines 1970/Ex. 1–560).

Paraquat's teratogenic potency in mice is low (Bus and Gibson 1975/Ex. 1-539), although 100 ppm administered in

the drinking water of pregnant rats increased postnatal mortality significantly (Bus and Gibson 1975/Ex. 1–539).

In humans, 69 accidental deaths and 81 suicides were attributed to the effects of paraquat exposure up to 1972 (Chipman Chemicals 1972, as cited in ACGIH 1986/Ex. 1–3, p. 456). Bouletreau, Ducluzeau, Bui-Xuan et al. (1977/Ex. 1–538) reported 31 cases of renal insufficiency, and a spray applicator was killed when he absorbed a lethal dose of inadequately diluted paraquat through the skin (Jaros 1978/Ex. 1–513). Workers using a 0.05- to 1-percent solution of paraquat developed skin and mucous membrane irritation but experienced no symptoms of systemic poisoning (Howard 1978/Ex. 1–512). Fugita, Suzuki, and Ochiai (1976, as cited in ACGIH 1986/Ex. 1–3, p. 456) reported five cases of reversible keratoconjunctivitis, with corneal injury, after one month of exposure to paraquat. Only NIOSH commented on paraquat.

OSHA is establishing an 8-hour TWA limit of 0.1 mg/m^3 for paraquat, with a skin notation. The Agency concludes that this limit will protect workers from the significant risk of skin, eye, and pulmonary irritation observed in animals exposed to aerosols of respirable size at levels below OSHA's former PEL for paraquat. The Agency considers the irritant effects of paraquat to be material impairments of health. OSHA is retaining the skin notation for this substance because of its capacity to penetrate the skin.

SILICA, CRYSTALLINE—CRISTOBALITE

CAS: 14464–46–1; Chemical Formula: SiO_2
H.S. No. 1354

The former OSHA PEL for respirable cristobalite was one-half the value calculated from the mass formula for quartz, measured as respirable dust. This limit corresponds to a range of 0.04 to 0.05 mg/m^3, measured as silica, for dusts containing 10 to 100 percent quartz. The ACGIH recommends an 8-hour TWA limit of 0.05 mg/m^3, measured as respirable silica dust. Although expressed differently, the current ACGIH and former OSHA limit for cristobalite are comparable. The ACGIH's mg/m^3 limit, adopted in 1985, does not reflect a re-evaluation of cristobalite's toxicity but was adopted merely to simplify the monitoring of cristobalite dust concentrations. The ACGIH limit is based on a study by Gardner (1938, as cited in ACGIH 1986/Ex. 1–3, p. 522) that was confirmed by King, Mohanty, Harrison. and Nagelschmidt (1953/Ex. 1–85). Experimental animals injected with cristobalite showed a more severe response than that produced by quartz, and the fibrosis that followed was diffuse rather than nondular. OSHA proposed, and the final rule establishes, a permissible exposure limit of 0.05 mg/m^3 TWA for cristobalite, measured as respirable silica dust. Cristobalite, one of the three major forms of silicon dioxide, is transparant, tasteless, and stable at high temperatures.

The final rule replaces OSHA's former limit for cristobalite, which is expressed, as described above, with a numerically equivalent limit of 0.05 mg/m^3; the Agency is establishing this time-weighted average limit to simplify employee exposure monitoring. NIOSH (Ex. 8–47, Table N6A; Tr. pp. 3–96 to 3–97) concurred with the selection of this limit but recommended that cristobalite be designated as a potential human carcinogen. OSHA's discussion of this and other rulemaking issues appears in the following entry describing the record evidence on quartz dust.

SILICA, CRYSTALLINE—QUARTZ

CAS: 14808–60–7; Chemical Formula: None
H.S. No. 1355

The former OSHA limit for silica-containing dusts is a respirable dust limit expressed as the following formula:

(10 mg/m^3)/(% respirable quartz+2).

At one time, the ACGIH also expressed its silica limit in terms of this formula. However, the current ACGIH TLV is 0.1 mg/m^3, measured as respirable quartz dust. OSHA proposed, and the final rule establishes, a permissible exposure limit of 0.1 mg/m^3 TWA, as respirable quartz. Quartz is a colorless, odorless, noncombustible solid.

The ACGIH does not see this change in the value of its limit for occupational exposure to silica as significant; instead, the ACGIH made this change to conform its limit for this dust to its TLVs for other dusts. If the former OSHA formula is used to calculate a limit for a dust containing 100 percent quartz, the limit would be 0.098 mg/m^3, a value that is not appreciably different from the ACGIH's revised limit of 0.1 mg/m^3 for respirable quartz dust. For quartz dusts containing less than 100 percent free silica, the former OSHA formula would yield a limit of, for example, 0.83 mg/m^3 for respirable dust containing 10 percent quartz. This result is somewhat more stringent than the ACGIH's TLV of 0.1 mg/m^3. For cristobalite and tridymite, the former OSHA formula and the ACGIH limits yield approximately the same results: both are approximately one-half the limit established by these two entities for quartz dust (see the discussions below).

Occupational exposure to free silica has been known for many years to produce silicosis, a chronic, disabling lung disease characterized by the formation of silica-containing nodules of scar tissue in the lungs. Simple silicosis, in which the nodules are less than 1 cm in diamater (as measured on chest X-ray films) is generally asymptomatic but can be slowly progessive, even in the absence of continued exposure. Complicated silicosis (i.e., with nodules greater than 1 cm in diameter) is more often associated with disability and can also progress in the absence of continuing exposure.

The health basis underlying the ACGIH's limit for crystalline silica is the work of Russell et al. (1929/Ex. 1–156), which suggested that a limit of 10 mppcf would protect workers from the effects of exposure to granite dust; a study by Ayer (1969/Ex. 1–129) demonstrated that 10 mppcf of granite dust is approximately equal to 0.1 mg/m^3 of respirable quartz dust (ACGIH 1986/Ex. 1–3).

NIOSH has recommended an exposure limit of 0.05 mg/m^3 as respirable free silica for all crystalline forms of silica. As applied to cristobalite and tridymite, the NIOSH REL is 0.05 mg/m^3, the same as the ACGIH TLV, but NIOSH's 0.05-mg/m^3 REL for quartz dust is one-half the value of the ACGIH TLV for quartz dust. To support its more stringent REL for quartz dust, NIOSH cites the work of Hosey, Ashe, and Trasko (1957, as cited in ACGIH 1986/Ex. 1–3, p. 524), which reported that no new cases of silicosis occurred in workers in Vermont granite sheds who were generally exposed to 0.05 mg/m^3 or less of granite dust. The recommendation was also partly based on studies by Theriault, Burgess, DiBerardinis et al. (1974/Ex. 1–94a); Theriault, Peters, and Fine (1974/Ex. 1–110); and Theriault, Peters, and Johnson (1974/Ex. 1–94b), which found that annual declines in pulmonary function and abnormal chest X-rays occurred among 192 granite shed workers exposed to an average quartz concentration of 0.05 mg/m^3. NIOSH noted that the exposure estimates reported in the Theriault et al. (1974/Exs. 1–94a, 1–94b, and 1–110) studies failed to account for the higher exposures that probably occurred in the years before exposure sampling was initiated and, therefore, that the Theriault et al. (1974) exposure data may have understated average exposures to quartz. Thus, NIOSH believes that the exposures responsible for the declines in pulmonary function were actually above 0.05 mg/m^3. The

ACGIH (1986/Ex. 1-3) found NIOSH's reasoning unpersuasive, citing a report by Graham, O'Grady, and Dubuc (1981/Ex. 1-172), who measured the pulmonary function of the same group of workers studied by Theriault et al. (1974/Exs. 1-94a, 1-94b, and 1-110), and found, in contrast to Theriault, that these workers experienced "an overall increase in FVC and FEV" (ACGIH 1986/Ex. 1-3).

Although OSHA did not propose a significant change in the exposure limit, there were several comments that focused on two issues: (1) the adequacy of the proposed 0.1 mg/m³ respirable quartz limit in reducing the risk of silicosis; and (2) recent evidence describing the potential carcinogenicity of silica dust.

With regard to the first issue, Dr. Philip Landrigan of the Mount Sinai School of Medicine, representing the American Public Health Association, testified as follows at the informal hearing:

> Numerous epidemiologic studies have been undertaken in this century, which have established a dose-response relationship between occupational exposure to silica dust and the development of silicosis. These studies have shown clearly that there is a positive dose-response relationship between chronic silica exposure and the development of silicosis.
>
> The most recent of these reviews which have examined that relationship is presented in the 1986 NIOSH text on occupational respiratory diseases, a most authoritative book in the field, widely read by medical scientists in this country and abroad. The data which was summarized in that chapter indicate quite clearly that the dose-response relationship between silica exposure and silicosis is present in people with lifetime exposure to silica below the current . . . standard of 100 micrograms per cubic meter. Indeed, the data suggests that the dose-response relationship extends downward even to levels of exposure below the current NIOSH recommended standard of 50 micrograms per cubic meter.
>
> And against the authoritative NIOSH review . . . OSHA has cited one short three-page article . . . [Graham et al. 1981/Ex. 1-172] to indicate that the dose-response relationship between silica and silicosis does not extend downward to below 100 micrograms per cubic meter (Tr. pp. 3-277 to 3-278).

Several commenters (Exs. 3-678, 3-733, 130, 138, 139, 147, 161, and 126) disagreed with Dr. Landrigan's assessment. For example, Frederick A. Renninger of the National Stone Association (Ex. 139) cited Dr. John Peters, the author of the chapter in the NIOSH reference referred to by Dr. Landrigan. In his chapter, Dr. Peters concluded as follows:

> All of the studies described in this section provide evidence for adverse pulmonary effects at levels of exposure above 10 mppcf or 0.1 mg/m³. Some showed that foundry workers exposed to the equivalent of 0.05 mg/m³ of quartz developed silicosis while those with less exposure did not. . . . All the Vermont findings were seen with an average exposure of around 0.05 mg/m³ of quartz. It is possible, however, that since this was the average exposure, individuals whose exposure exceeded this level accounted for the noted effects. (The "no effect" level was probably below 0.05 mg/m³, but the available data did not allow accurate determinations.) (Peters, J.M., "Silicosis." In: *Occupational Respiratory Diseases*, p. 229, J.S. Merchant, ed. DHHS (NIOSH) Pub. No. 86-102, NIOSH 1986b).

Mr. Renninger also points to the difficulty in equating impinger sampling results, which were used in the Vermont granite shed studies, to gravimetric (mg/m³) measures of respirable dust. He cited Dr. Peters as reporting that "gravimetric and impinger sampling are known to be poorly correlated" (Ex. 139, p. 5). Mr. Renninger also pointed out that the conversion between mppcf and mg/m³ measurements for silica will vary with the industry, thus adding another level of uncertainty in interpreting the health data.

OSHA's decision to propose a 0.1-mg/m³ PEL for respirable silica dust, rather than the NIOSH REL of 0.05 mg/m³, was partly based on the report by Dr. William Graham et al. (Graham, O'Grady, and Dubuc 1981/Ex. 1-172) discussed above. In a posthearing submission, Dr. Graham discussed the findings of Theriault and co-workers (1974/Exs. 1-94a, 1-94b, and 1-110), which heavily influenced the decision by NIOSH to issue a REL of 0.05 mg/m³ (Ex. 147). Dr. Graham discussed three limitations of the Theriault et al. (1974) studies. First, the X-ray films were interpreted by a single reader who was neither certified nor a chest physician; Dr. Peters points out that it is generally accepted that X-ray films must be read by three experienced readers. Second, there was no attempt to study workers hired after 1938 and exposed to low dust levels separately from workers exposed to higher dust levels prior to 1938. Third, there was a group of workers who were judged to have abnormal X-ray findings despite a reported lack of exposure to dust, which raises the question about the accuracy of interpretations.

Dr. Graham also interpreted his own findings of granite shed workers as showing that the loss in pulmonary function predicted to occur among these workers by Theriault et al. (1974/Exs. 1-94a, 1-94b, and 1-110) had, in fact, not occurred. One explanation offered by Dr. Graham is the possibility that technical difficulties arose during the Theriault et al. (1974) studies in the administration of spirometric tests, and may have resulted in spuriously low values for pulmonary function. Dr. Graham discussed a continuation of his own work in which he has found neither pulmonary function losses nor high prevalences of abnormal chest X-rays among granite shed workers who were employed after 1938-1940, when lower dust levels prevailed (Ex. 147, pp. 8-9). However, the analysis of quartz content in the dust samples collected has not yet been completed (Ex. 147, p. 8).

In addition to the evidence on the dose-response relationship for silicosis, rulemaking participants discussed at length recent data suggesting that silica may be carcinogenic (Exs. 147, 161, 194, 138, 3-1159, 3-1060, and 139; Tr. p. 3-94, Tr. p. 7-80, Tr. p. 11-104). NIOSH (Ex. 8-47, Table N6B) believes that the data on silica are such that the Agency should consider a separate 6(b) rulemaking for this substance. Dr. Frank Mirer, Director of the Health and Safety Department of the United Auto Workers, summarized the evidence on silica's potential carcinogenicity at the hearing:

> The most prominent study [on the health effects of silica exposure is] by Holland and coworkers * * * [it] provided really clear evidence that silica was carcinogenic in rats by inhalation. Non-malignant pulmonary effects were also observed. There is a considerable line of other work in rats and hamsters, in the development of both lung tumors and lymphatic tumors from exposure to silica.
>
> In epidemiology, there's ample evidence that crystalline silica is carcinogenic and that it is hazardous at levels below the proposed PEL. The IARC monograph reviewed the data available in 1986 and described a considerable body of evidence. Despite the methodological limitations pointed out by IARC, the sheer number and consistency of the findings is most persuasive (Tr. pp. 7-80 to 7-81).
>
> Studies [exist] of workers in a variety of industries where high exposure of silica-containing dusts have revealed high lung cancer risks. These results include ten positive studies among mine workers, four in ceramics and glass industries, [and] four in the foundry industry. We also bring to your attention at least four additional studies published since the IARC criteria document was completed. These, in particular, we think create an iron-clad case for the problems presented by this material (Tr. pp. 7-80 to 7-81).

In a posthearing submission by the Refractories Institute, Dr. John Craighead of the University of Vermont reviewed the human and animal data and concluded as follows:

> I find the experimental evidence in animals, suggesting a possible role of silica in

the pathogenesis of bronchogenic carcinomas, to be faulty and incomplete. I also conclude that the epidemiological studies in humans provide inadequate evidence to conclude that man is at increased risk of developing carcinoma of the lung as a result of silica dust exposure. My comments in no way exclude from consideration silica as a cause of bronchogenic carcinoma, but only point out the inadequacies of the scientific information and emphasize the need for additional, carefully designed systematic studies (Ex. 161A, p. 5)

In similar attachments to the Refractories Institute's submission, Dr. Marvin Kushner, Professor of Pathology at the State University of New York at Stony Brook, pointed to the lack of similarity between the pulmonary lesions found in exposed rats and silicosis lesions in humans; he suggested that the carcinomas seen in rats may be due to a "non-specific" effect that is not a direct result of silica inducing malignant transformation (Ex. 161C). Dr. Carl Shy, Professor of Epidemiology at the University of North Carolina, reviewed the epidemiological evidence and concluded that "the role of occupational silica exposure in causing lung cancer remains undetermined" (Ex. 161D, p. 8).

OSHA believes that the issues raised above deserve a careful and thorough scientific evaluation of the literature. The evidence that silica may present a carcinogenic hazard has been developing over the past few years and is continuing to receive considerable attention by investigators. OSHA will continue to monitor with great interest emerging developments in this area. At this time, however, OSHA believes that the record evidence leaves many questions unanswered regarding the need to reduce the PEL for silica. Therefore, in the final rule, OSHA is establishing an 8-hour TWA PEL of 0.1 mg/m^3 for quartz, measured as the respirable silica fraction. This limit represents no substantial change from OSHA's former formula limit, but will simplify sampling procedures, as indicated in the NPRM.

SILICA, CRYSTALLINE—TRIDYMITE

CAS: 15468-32-3; Chemical Formula: SiO_2
H.S. No. 1356

The former OSHA PEL for respirable tridymite was expressed as one-half the value of the mass formula for quartz dust. This formula corresponds to a range of 0.04 to 0.05 mg/m^3, measured as silica, for dusts containing 10 to 100 percent tridymite. The Agency proposed, and the final rule establishes, a PEL of 0.05 mg/m^3 TWA for tridymite. The ACGIH recommends an 8-hour TWA limit of 0.05 mg/m^3, measured as silica dust. The ACGIH limit is based on a study conducted by King, Mohanty, Harrison, and Nagelschmidt (1953/Ex. 1-85) that found tridymite to be the most active of the free silica forms when injected intratracheally into rats. Tridymite is a transparent, tasteless form of free silica.

Although expressed in different units, the current ACGIH and former OSHA limits for tridymite are comparable. The ACGIH's mg/m^3 limit, adopted in 1985, does not reflect a re-evaluation of tridymite's toxicity but was adopted merely to simplify the monitoring of tridymite dust concentrations. NIOSH (Ex. 8-47, Table N6A) concurs with the selection of this limit but recommends that tridymite be designated as a potential occupational carcinogen. No other comments were received on tridymite.

OSHA is replacing its former limit for tridymite, which is described above, with a numerically equivalent limit of 0.05 mg/m^3, measured as respirable silica dust; the final rule establishes this change to simplify employee exposure monitoring.

SILICA, CRYSTALLINE—TRIPOLI

CAS: 1317-95-9; Chemical Formula: SiO_2
H.S. No. 1357

Tripoli is a colorless microcrystalline form of quartz. Although OSHA's Table Z-2 did not specifically indicate a limit for tripoli, OSHA formerly specified a limit for crystalline quartz based on the formula measured as total respirable dust: 10 $mg/m^3/\%SiO_2+2$. Expressed as mg/m^3, this limit corresponds to a limit in the range of 0.08 to 0.1 mg/m^3 for respirable dust containing from 10 to 100 percent silica. The 8-hour TWA ACGIH limit for tripoli is 0.1 mg/m^3, measured as respirable silica dust. This limit was adopted by the ACGIH in 1985 to simplify the monitoring of quartz dust concentrations. Thus, this revision does not represent a re-evaluation of toxicity data for tripoli. NIOSH (Ex. 8-47, Table N6B) does not concur with the final rule's limit and recommends a separate 6(b) rulemaking for tripoli, which NIOSH considers a potential occupational carcinogen. (see section above on Crystalline Quartz for OSHA's discussion of the record evidence on the carcinogenicity of silica). No other comments were received on tripoli.

OSHA is replacing its limit for quartz, which is expressed as the formula presented above, with a numerically equivalent limit of 0.1 mg/m^3 TWA as respirable silica dust; the final rule establishes this limit for tripoli.

SILICA, FUSED

CAS: 60676-86-0; Chemical Formula: SiO_2
H.S. No. 1358

Fused silica is a colorless, odorless solid that is a form of quartz. As such, it was formerly covered by OSHA's limit for quartz (Table Z-3). Exposure to fused silica has long been known to cause the fibrogenic lung disease, silicosis. OSHA's former limit for quartz dust was the formula 10 $mg/m^3/\%SiO_2+2$, measured as total respirable dust. This limit correspnds to a respirable quartz concentration ranging from 0.08 to 0.1 mg/m^3 measured as free silica. The ACGIH recommends an 8-hour TWA limit of 0.1 mg/m^3, measured as free silica; the ACGIH adopted this limit in 1985 to simplify the monitoring of quartz dust concentrations. Thus, this revision does not represent a re-evaluation of the toxicity data for fused silica. NIOSH (Ex. 8-47, Table N6B) does not concur with the final rule's limit and recommends a separate 6(b) rulemaking for fused silica, which NIOSH considers a potential occupational carcinogen.

OSHA is replacing its limit for fused silica, which is expressed as the formula presented above, with a numerically equivalent limit of 0.1 mg/m^3 as total respirable silica dust; the Agency is establishing this limnit to simplify employee exposure monitoring.

SOAPSTONE, TOTAL DUST

SOAPSTONE, RESPIRABLE DUST

CAS: None; Chemical Formula: 3 MgO-4 SiO_2-H_2O
H.S. No. 1363 (total dust)
H.S. No. 1363A (respirable dust)

OSHA's former exposure limit for soapstone, total dust, was 20 mppcf (6 mg/m^3), and the Agency had no separate limit for the respirable fraction. The ACGIH has established individual TLV-TWAs for these two forms of soapstone: 6 mg/m^3 for total dust, and 3 mg/m^3 for the respirable fraction, both measured as total dust or respirable dust containing less then 1 percent quartz. Because the ratio of total dust mass to the mass of the respirable fraction is 2:1 (ACGIH 1984, p. 480), the 6-mg/m^3 total dust limit automatically implies a 3-mg/m^3 limit for the respirable fraction. OSHA proposed, and the final rule establishes, permissible exposure limits of 6 mg/m^3 TWA (total dust) and 3 mg/m^3 TWA (respirable dust) for soapstone. NIOSH (Ex. 8-47, Table N1) concurred with this determination.

A study by Dreessen and DallaValle (1935/Ex. 1-588) of mill workers exposed to soapstone showed lung changes in these workers, but it is believed that the dusts involved in these exposures were actually steatite talc, which had a tremolite content of 10

percent. Experiments by Miller and Sayers (1941/Ex. 1-595) showed no measurable toxic effects in guinea pigs injected intraperitoneally with various samples of soapstone. No comments were received on soapstone other than those submitted by NIOSH.

The final rule expresses the limit for soapstone as total dust in mg/m^3, rather than mppcf, to simplify employee sampling and analysis. The total dust limit being established, 6 mg/m^3, is equivalent to the previsious limit of 20 mppcf, and the new limit of 3 mg/m^3 for respirable dust is actually implicit in the total dust limit.

SULFUR DIOXIDE

CAS: 7446-09-5; Chemical formula: SO_2
H.S. No. 1375

OSHA's former limit for sulfur dioxide (SO_2) was 5 ppm as an 8-hour TWA. The Agency proposed to revise this limit to 2 ppm as an 8-hour TWA and to supplement this limit with a 15-minute STEL of 5 ppm. Although NIOSH recommends a limit of 0.5 ppm for sulfur dioxide, NIOSH did concur (Ex. 8-47, Table N1) with the proposed limits. The ACGIH has a TLV-TWA of 2 ppm and a TLV-STEL of 5 ppm. In the final rule, OSHA is establishing a 2-ppm 8-hour TWA and a 5-ppm 15-minute STEL for SO_2. Sulfur dioxide is a colorless, nonflammable gas or liquid with a suffocating odor.

OSHA has studied the effects of occupational exposure to SO_2 for several years. The Agency's 5-ppm limit for this substance was established in 1971 on the basis of the 1968 ACGIH TLV-TWA. In 1975, OSHA proposed to revise this limit downward to 2 ppm and held public hearings to gather information on industrial exposures to SO_2. In response to shifting priorities within the Agency, OSHA did not promulgate a final standard at that time. The following discussion summarizes the record evidence relevant to SO_2 both from the earlier (1975-1976) record and from the record of the present rulemaking.

Workplace exposure to sulfur dioxide causes both acute and chronic effects. The chronic effects of exposure include permanent pulmonary impairment, which is caused by repeated episodes of bronchoconstriction. A number of human and animal studies demonstrate this effect (Skalpe 1964/Ex. 1-438; Smith, Peters, Reading, and Castle 1977/Ex. 1-805; Archer and Gillam 1978/Ex. 1-711; Ministry of Health (Canada) 1976/Ex. 1-1208; Lewis, Campbell, and Vaughan 1969, as cited in ACGIH 1986/Ex. 1-3, p. 542).

Kehoe, Machle, Kitzmiller, and LeBlanc (1932/Ex. 1-339) studied two groups of male refrigeration workers with long-term (average of four years) exposures to average SO_2 concentrations of 20 to 30 ppm, with a range of exposures from 10 to 70 ppm. These workers were believed to have been exposed prior to 1927 to SO_2 levels considerably higher and averaging from 80 to 100 ppm. This study showed that SO_2 exposure caused an increased incidence of nasopharyngitis, shortness of breath on exertion (dyspnea), and chronic fatigue (Kehoe, Machle, Kitzmiller, and LeBlanc 1932/Ex. 1-339).

In a study of Norwegian paperpulp mill workers, Skalpe (1964/Ex. 1-438) reported that average SO_2 concentrations were believed to range from 2 to 36 ppm. Results showed a significantly higher frequency of respiratory disease symptoms, including coughing, expectoration, and dyspnea, among workers less than 50 years of age (i.e., those with the shortest exposure). Workers older than 50, however, did not display symptomatology different from that of controls.

More recently, Smith, Peters, Reading, and Castle (1977/Ex. 1-805) studied a group of smelter workers exposed, on average, to less than 2 ppm SO_2 but concurrently exposed to respirable particulate at levels generally less than 2 mg/m^3. These workers showed a decrement in forced vital capacity (FVC) and forced expiratory volume (FEV_1) of 4.8 percent when compared with controls. These authors concluded that workers exposed to SO_2 levels above 1 ppm had an accelerated loss of pulmonary function. This study has been criticized on the grounds that the control population itself may have been exposed to respiratory toxins and that other contaminants, such as iron sulfites, may have contributed to the pulmonary decrement seen in these smelter workers. On average, 60 percent more of the workers exposed to greater than 1 ppm SO_2 reported symptoms of chronic cough than did workers who were exposed to SO_2 at a concentration below 1 ppm. The prevalence of chronic sputum production was elevated for workers who had never smoked and who were exposed above 1 ppm.

Archer and Gillam (1978/Ex. 1-711) studied workers at the same smelter facility and obtained results similar to those of Smith, Peters, Reading, and Castle (1977/Ex. 1-805). Significant reductions in FVC and FEV_1 were found to be associated with chronic exposures to 0.4 to 3 ppm SO_2 (TWA) with concomitant exposure to particulate. These authors also found a corresponding increase in some symptoms of respiratory disease (chronic bronchitis) that was not attributable to smoking. Tomono and coworkers (1961, as cited in ACGIH 1986/Ex. 1-3, p. 542) found that 1.6 ppm was the lowest concentration that produced bronchoconstriction in 46 healthy male subjects.

OSHA's June 7, 1988 proposal also discussed the basis for NIOSH's recommendation of a 0.5-ppm 8-hour TWA limit for SO_2. In addition to the studies by Archer and Gillam (1977/Ex. 1-711) and Smith, Peters, Reading, and Castle (1977/Ex. 1-805) described above, NIOSH relied on a third study (Ministry of Health (Canada) 1976/Ex. 1-1208) of smelter workers exposed to SO_2 levels of 2.5 ppm for 10 or more years, which showed an increased incidence of respiratory disease in these workers. A fourth study cited by NIOSH (NIOSH 1977m, as cited in ACGIH 1986/Ex. 1-3, p. 542) reported that 10,000 workers exposed to SO_2 at levels of 0.35 ppm showed no adverse exposure-related effects.

Alarie and co-workers (1970 and 1972, as cited in ACGIH 1986/Ex. 1-3, p. 542) found that guinea pigs exposed to SO_2 by inhalation showed no decrement in pulmonary function at SO_2 levels of 5 ppm; monkeys exposed to 1.3 ppm for 78 weeks also showed no deficit (Alarie, Ulrich, Busey et al. 1970 and 1972, both as cited in ACGIH 1986/Ex. 1-3, p. 542). However, in another study, dogs exposed continuously to 5 ppm for 225 days showed increased pulmonary flow resistance and a decrease in lung compliance (Lewis, Campbell, and Vaughan 1969, as cited in ACGIH 1986/Ex. 1-3, p. 542). In addition, rats exposed to 10 ppm SO_2 daily for six weeks developed a thickening of the mucous layer that interfered with effective particle clearance (Dalhamn 1956, as cited in ACGIH 1986/Ex. 1-3, p. 542).

The acute effects of SO_2 exposure have been recognized for years in industrial settings; symptoms of acute overexposure include upper respiratory tract irritation, rhinorrhea, choking, and coughing. These symptoms are so disagreeable that most persons will not tolerate exposure for longer than 15 minutes. Within 5 to 15 minutes of the onset of exposure, workers develop temporary reflex broncho-constriction and increased airway resistance. Short-term exposure causes measurable bronchoconstriction (Frank, Amdur, Worcester, and Whittenburger 1962, as cited in ACGIH 1986/Ex. 1-3, p. 542; Weir, Stevens, and Bromberg 1972/Ex. 1-401); the ACGIH (1986/Ex. 1-3, p. 542) reports that this bronchoconstriction is dose-related and is manifested as an increase in pulmonary flow resistance.

Efforts have been made to quantify the acute no-adverse-effect level for

SO_2-induced increased airway resistance. Frank, Amdur, Worcester, and Whittenberger (1962, as cited in ACGIH 1986/Ex. 1-3, p. 542) reported that, at SO_2 concentrations of 1 ppm, one in 11 healthy subjects developed pulmonary flow resistance; at concentrations of 5 or 13 ppm, there was a 39- and 72-percent increase, respectively, in such resistance. Weir, Stevens, and Bromberg (1972/Ex. 1-401) noted a statistically significant but reversible increase in small-airway resistance and a decrease in lung compliance at a concentration of 3 ppm; however, Burton et al. (1969) reported no effects, even among smokers, at a level of 2.1 ppm.

N.R. Frank, Professor of Medicine at the University of Washington State, commented during the 1977 hearing (NIOSH 1977m) that sulfur dioxide may not by itself be hazardous to the lungs but that an aerosol of sulfur dioxide and water or SO_2 oxidized to sulfate particulate may increase the toxic potential of SO_2 (Ex. 40, Docket H-039). Dr. Frank also presented evidence showing that a single short-term exposure to very high SO_2 levels (200 to 1000 ppm) can produce lung damage (Ex. 40, Docket H-039).

In the current generic rulemaking, participants such as the American Iron and Steel Institute (AISI) (Exs. 3-1123 and 188) and the Corn Refiners Association (Exs. 8-65 and 177) raised issues similar to those raised during OSHA's 1977 rulemaking on SO_2. These included:

- Lack of evidence that long-term exposure to SO_2 causes chronic respiratory disease; and
- The potentiation of SO_2's adverse effects by the formation of sulfates or higher sulfur oxides from interactions between SO_2 and water or SO_2 and particulate matter.

Regarding the first point, the Corn Refiners Association (CRA) referred OSHA to studies and testimony on the effects of SO_2 exposure on employees in corn wet-milling from the earlier rulemaking (Ex. 66, Docket H-039). The CRA reported that the chronic respiratory disease and pulmonary impairment seen in SO_2-exposed smelter workers did not occur in corn milling plant employees (Ex. 66-1, Docket H-039). The CRA sponsored a study performed by Drs. Ferris and Essex from the Harvard School of Public Health (Ex. 66-3, Docket H-039). Fifty corn wet-milling workers involved in the early, SO_2-using stage of the wet-milling process were studied. Exposures (8-hour TWAs) in this group ranged from 0.5 to more than 5 ppm SO_2, particulates ranged from 0.0 to 0.17 mg/m^3, and water-soluble sulfates ranged from 0.0 to 40.0 mg/m^3. Results of this study showed that, at levels of about 3 ppm SO_2, acute symptoms such as coughing developed, but chronic, irreversible symptoms were not seen at exposure levels below 5 ppm (Ex. 66-1, Docket H-039). These authors concluded:

> Taken as a whole, the results suggest that no *significant chronic* respiratory impairments occurred at exposure levels under 5 ppm. The lack of association between the most serious symptoms of respiratory disease and exposure levels below 5 ppm also suggests that the atmosphere in question is quite distinct from that found in the *copper smelter studies* (Ex. 66-3, Docket H-039).

In addition, the studies by Smith, Peters, Reading, and Castle (1977/Ex. 1-373) and Archer and Gillam (1978/Ex. 1-711) were criticized in OSHA's earlier rulemaking for not taking into consideration the impact on the studied workers' health of the higher SO_2 levels to which these employees had been exposed in prior years. Arthur D. Little, Inc. (Ex. 95, Docket H-044) also criticized these studies, noting that their observation periods were too short to derive reliable data on chronic effects.

These criticisms and the lack of chronic effects observed in animals at levels below 5 ppm (Alarie, Ulrich, Busey et al. 1970 and 1972, as cited in ACGIH 1986/Ex. 1-3, p. 542) caused commenters to question whether chronic lung disease results from long-term exposure to SO_2 below the current 5-ppm PEL. Dr. Alarie appeared at the 1977 hearing and testified on animal studies conducted by him and others on sulfur dioxide (NIOSH 1977m, as cited in ACGIH 1986/Ex. 1-3, p. 542). He testified that, in his opinion, the long-term studies in animals support the establishment of a ceiling value for SO_2 but do not indicate that benefits would be gained by reducing the time-weighted average from 5 to 2 ppm. OSHA agrees with Dr. Alarie that a STEL is necessary to minimize high short-term exposures to SO_2; however, OSHA does not agree that no effects have been seen in animals at levels at or below 5 ppm. For example, Lewis, Campbell, and Vaughan (1969, as cited in ACGIH 1986/Ex. 1-3, p. 542) showed that beagles exposed to 5 ppm SO_2 exhibited decreased dynamic compliance and increased flow resistance. In addition, NIOSH (1974b/Ex. 1-235) has reported:

> [M]an is considered to be more sensitive than other mammals to the effects of sulfur dioxide in ranges commonly employed experimentally . . . (Ex. 1-235).

It is therefore not surprising that humans have also been shown to develop respiratory effects, including bronchoconstriction, coughing, and sputum production, at levels below 5 ppm (Smith, Peters, Reading, and Castle 1977/Ex. 1-805; Archer and Gillam 1978/Ex. 1-711; Frank, Amdur, Worcester, and Whittenburger 1962, as cited in ACGIH 1986/Ex. 1-3, p. 542; Weir, Stevens, and Bromburg 1972/Ex. 1-401).

Many rulemaking participants (Exs. 3-1123, 8-57, 86, 86A, 117, 177, and 188) were of the opinion that the lack of chronic effects demonstrated that exposure to SO_2 did not cause material impairment of health at levels below 5 ppm. For example, the Edison Electric Institute (EEI) (Ex. 133) criticized the Ferris et al. (1967/Ex. 1-316) study as being too old to be relevant. According to the EEI, the finding that the control group in the Ferris et al. (1967/Ex. 1-316) study also had an elevated incidence of disease and that there was no statistically significant difference in the extent of the respiratory disease incidence between the controls and the SO_2-exposed group invalidates this study's finding of a serious pulmonary effect in the SO_2-exposed workers. OSHA does not agree with this interpretation of the Ferris et al. (1967/Ex. 1-316) study. OSHA believes that a more accurate interpretation of the results of this study would be that both groups of workers were occupationally exposed to respiratory toxins; this is a very likely occupational scenario because the SO_2-exposed workers in this study were pulpmill workers, while those in the control group worked in a papermill, an occupational environment also recognized as hazardous.

Taken together, the evidence from all of the studies described in this subsection clearly shows that exposure to SO_2 below 5 ppm does cause respiratory symptoms, including repeated episodes of bronchoconstriction. The studies by Smith, Peters, Reading, and Castle (1977/Ex. 1-373), Archer and Gillam (1978/Ex. 1-711), and Frank, Amdur, Worcester, and Whittenberger (1962, as cited in ACGIH 1986/Ex. 1-3, p. 542) consistently demonstrate that persons exposed to concentrations of SO_2 below 5 ppm have an accelerated loss of pulmonary function and exhibit adverse pulmonary symptoms.

OSHA believes that these effects constitute material impairments of health and are significant. In addition, OSHA does not agree that these studies demonstrate the absence of chronic effects at low SO_2 exposure levels; long-term exposure to SO_2 has produced pulmonary function changes in dogs, and daily exposures of rats to 10 ppm (only twice the former PEL) for six

weeks produced a thickened mucous layer and reduced the effectiveness of particle clearance from the trachea (Dalhamn 1956, as cited in ACGIH 1986/Ex. 1-3, p. 542).

The second point raised by commenters concerned the formation of other toxic and irritating products from the interaction between SO_2 and water or between SO_2 and particles. Some of the participants in the earlier rulemaking, such as Dr. Colucci of the Corn Refiners Association, testified that it would be more protective to identify and limit exposure to each of these by-products, rather than to regulate SO_2 alone. OSHA disagrees with this approach; since these products are all formed from sulfur dioxide, limiting exposure to SO_2 will concurrently limit exposure to these SO_2 by-products. This approach is more straightforward and easier to implement than attempting to identify the myriad decay products that may be formed in different industrial settings. Furthermore, the studies discussed above clearly establish a relationship between airborne SO_2 levels and adverse effects; no quantitative relationship on which to base a PEL has been established for the decay products of SO_2 reactions. Therefore, to reduce the significant risk of respiratory symptoms among exposed workers, OSHA finds that limiting exposure to SO_2 will be effective.

After considering all of the relevant evidence from both the 1977 and the present dockets, OSHA concludes that a TWA of 2 ppm and a STEL of 5 ppm are necessary to reduce the significant risk of adverse respiratory effects that have been demonstrated to occur in workers exposed to SO_2 above these levels. Accordingly, OSHA is establishing these limits in the final rule. The Agency finds that the coughing, increase in sputum production, and bronchoconstriction observed in workers exposed to SO_2 at the levels permitted by the former limit constitute material impairments of health and functional capacity, and must be protected against. This discussion is also a final statement of reasons for the 1977 rulemaking.

Some evidence has been submitted by the steel and nonferrous metal industries that the STEL cannot be regularly achieved with engineering and work-practice controls in specific operations in SIC 33. These involve furnace areas in nonferrous metal smelters, blast furnace operations, and the sulfur plant. There is no evidence to the contrary in the record.

OSHA will, therefore, permit more flexibility in the use of respirators for these operations. The burden of proof will not be on employers to demonstrate that compliance with engineering and work-practice controls are infeasible in a compliance action for these operations in SIC 33 as related to meeting the requirements of the STEL.

There may be a few other operations in this category, and for the TWA, where the record is unclear for SIC 33. Based on an appropriate showing pursuant to the OSH Act, OSHA would favorably consider requests for variances for specific operations in Sector 33 on methods of compliance for the STEL and for the TWA. Of course, all requests for variances or any matters will be considered based on their merits.

SULFUR TETRAFLUORIDE

CAS: 7783-60-0; Chemical Formula: SF_4
H.S. No. 1378

OSHA's former Z tables had no exposure limits for sulfur tetrafluoride. The proposed PEL was 0.1 ppm as a ceiling; NIOSH (Ex. 8-47, Table Nl) concurs with this limit, and the final rule establishes it. This limit is consistent with that of the ACGIH. Sulfur tetrafluoride is a colorless, noncombustible gas.

On contact with moisture, sulfur tetrafluoride produces sulfur dioxide and hydrogen fluoride (HF) (Lester 1971, as cited in ACGIH 1986/Ex. 1-3, p. 546), and it is the release of HF that is primarily responsible for sulfur tetrafluoride's toxic effects (Zapp 1971, as cited in ACGIH 1986/Ex. 1-3, p. 546). A du Pont (1961, as cited in ACGIH 1986/Ex. 1-3, p. 546) study of rats exposed for four hours to 4 ppm sulfur tetrafluoride over a period of 10 days reported that the animals demonstrated nasal discharge, difficulty in breathing, and weakness. Autopsies of these animals revealed evidence of emphysema, but those rats surviving exposure and given a two-week rest period after exposure showed no significant pathological changes. In the same study by du Pont (1961, as cited in ACGIH 1986/Ex. 1-3, p. 546), a four-hour exposure to 20 ppm sulfur tetrafluoride proved lethal to one of two rats. In a study by Clayton (1962/Ex. 1-409), irregular breathing and signs of irritation were observed following exposures to concentrations of 20 ppm and lower; animals receiving lethal amounts of sulfur tetrafluoride showed pulmonary edema on autopsy, and those with sublethal exposures demonstrated no pathologic changes 14 days later.

In the final rule, OSHA is establishing a 0.1-ppm ceiling limit for this highly toxic gas. The Agency concludes that establishing this limit for this previously unregulated chemical will reduce the significant risk of chronic respiratory effects potentially associated with exposure to sulfur tetrafluoride at the levels permitted by the absence of any OSHA limit. OSHA considers the chronic respiratory effects caused by exposure to sulfur tetrafluoride to be material impairments of health. NIOSH was the only commenter to the rulemaking record on this substance.

TALC (CONTAINING NO ASBESTOS)

CAS: 14807-96-6; Chemical Formula: H_2O_3Si $^3/_4Mg$
H.S. No. 1381

The former OSHA PEL for nonasbestiform talc was 20 million particles per cubic foot of air (mppcf) as an 8-hour TWA; when expressed as mg/m^3, this is comparable to 3 mg/m^3. The ACGIH has a TLV-TWA of 2 mg/m^3 (15 mppcf) for talc, measured as respirable dust, and this is the limit proposed by OSHA and included in the final rule. NIOSH (Ex. 8-47, Table Nl) concurred that this limit is appropriate. Talc is a fine powder that is white to gray-white in color; it is found as a mineral, and the main component is a crystalline hydrated silicate of magnesium that is usually in the form of plates but occasionally may be in the form of fibers.

The health-effects evidence for talc is complicated by the fact that talcs contain amphiboles and other minerals, in addition to platiform talc crystals; adverse health effects appear to be related to the nonplatiform content (that is, to the fiber content) of the talc in question (ACGIH 1986/Ex. 1-3, p. 550). There are conflicting views regarding the extent to which the fibrous constituents are asbestos; however, no health effects information is available that is specifically related to fibrous talc (ACGIH 1986/Ex. 1-3, p. 550).

Numerous epidemiological studies have documented the effects on workers of long-term exposures to talc. In 1942, Porro et al. (1942, as cited in Stokinger 1981b/Ex. 1-1127) published a report in which 15 cases of talc pneumoconiosis, including five postmortem examinations, showed that asbestotic bodies were almost always present in fibrotic areas of the lungs of those workers with talcosis. Siegal and colleagues (1943, as cited in Stokinger 1981b/Ex. 1-1127) noted that the incidence of advanced fibrosis in a group of 221 talc miners and millers was 14.5 percent. These workers were primarily exposed to fibrous talc, which was believed to be responsible for the pathology of the asbestos-like lung lesions. A study by McLaughlin et al. (1949, as cited in Stokinger 1981b/Ex. 1-1127) revealed that talc-induced pneumoconiosis was caused by the fibrous varieties of talc; in animal

studies by Schepers and Durkan (1955, as cited in Stokinger 1981b/Ex. 1–1127), the degree of fibrosis in the lung tissue was found to be a function of the length of the talc fibers, rather than of the composition of the talc itself. A paper by Kleinfeld, Giel, Majeranowski, and Messite (1963, as cited in Stokinger 1981b/Ex. 1–1127) reported that postmortem examinations on six talc industry workers showed that the asbestotic bodies found in the lung bronchioles or embedded in fibrous tissue were indistinguishable from the asbestos bodies seen in cases of asbestosis.

Kleinfeld, Messite, Kooyman, and Zaki (1967/Ex. 1–704) later conducted a cohort study of 220 workers who had been employed in a mine that produced talc that had a tremolite and anthophyllite content. Of the 91 deaths in this group, 10 resulted from respiratory cancer and 28 were attributed to pneumoconiosis. The proportional mortality rate from respiratory cancer was four times the expected rate. In 1974, when Kleinfeld, Messite, and Zaki (Ex. 1–705) performed a follow-up study of this group (which at that time consisted of 260 workers [108 deaths]), they found significant differences between the expected and observed mortality in the period 1950 to 1954, but not during 1960 to 1969. These investigators attributed this finding to the reduction in talc dust counts (from averages of 25 to 73 mppcf (approximately 4 to 12 mg/m^3) in the years 1948 to 1965 to averages of 9 to 43 mppcf (approximately 1.5 to 6.5 mg/m^3) in the period 1966 to 1969). This study also showed a decrease of greater than 50 percent in deaths due to pneumoconiosis in the 1965-to-1969 time period.

Studies by NIOSH (Dement and Zumwald 1978, as cited in ACGIH 1986/Ex. 1–3, p. 552) of 398 white male workers employed between 1947 and 1959 in the talc industries found that 74 of these men had died, and that bronchogenic cancer was the cause of death in nine men; only 3.3 deaths from this cause would have been expected. Nonmalignant respiratory disease (NMRD) exclusive of influenza, pneumonia, and tuberculosis accounted for three deaths; 1.5 would have been expected. From these data, NIOSH concluded that a significant increase in mortality due to bronchogenic cancer and NMRD had occurred as a result of occupational exposure to talc dust. NIOSH's report also included a morbidity study of 12 talc industry workers, currently employed, in which chest X-rays, lung function tests, and questionnaires were used. This study concluded that a higher prevalence of cough, phlegm, dyspnea, and irregular opacities in chest X-rays existed in these workers than in potash miners; instances of pleural thickening and calcification were greater than in coal and potash miners; and the pulmonary function of talc workers overall was reduced in comparison with that of coal and potash miners employed for the same length of time. The reductions in pulmonary function among the talc workers were dose- and duration-related.

The ACGIH (1986/Ex. 1–3, p. 552) concludes that serious health effects have been associated in the past (i.e., prior to 1945) with exposures to amphibole-containing talc. However, the ACGIH believes that the introduction of mining improvements has all but eliminated "the excess of death rates from pneumoconiosis and lung cancer" (ACGIH 1986/Ex. 1–3, p. 552).

Two recent studies of the health effects associated with talc exposures (Rubino, Scansetti, Piolatto, and Romano 1976/Ex. 1–801; Selevan, Dement, Wagoner, and Froines 1979/Ex. 1–989) are available. The Rubino, Scansetti, Piolatto, and Romano (1976/Ex. 1–801) study found that miners and millers exposed to an average of 849 to 8470 mppcf-years (miners) or 76 to 651 mppcf-years (millers) showed no increase in the number of observed (compared to expected) deaths from causes other than silicosis. These authors concluded that the disease-causing factor in these workers was silica rather than talc (Rubino, Scansetti, Piolatto, and Romano 1976/Ex. 1–801).

The Selevan, Dement, Wagoner, and Froines (1979/Ex. 1–989) study of 392 workers exposed to talc in five mines found nonmalignant respiratory deaths for millers to be almost eight times the expected rate, while miners experienced more than three times the expected mortality rate for NMRD. The ACGIH (1986/Ex. 1–3, p. 552) believes that the Selevan et al. (1979/Ex. 1–989) study is incomplete because confounding factors were not adequately identified and controlled for.

With regard to NIOSH's findings (Dement and Zumwald 1978, as cited in ACGIH 1986/Ex. 1–3, p. 552) of excess cancer deaths among talc workers, OSHA is currently reviewing the scientific and toxicological data describing the effects of exposure to the nonasbestiform varieties of mineral fibers that are found in talc deposits. OSHA is considering a separate rulemaking to address this issue.

OSHA received few comments regarding its proposed revision to the PEL for respirable talc. John W. Kelse, Corporate Industrial Hygienist for R.T. Vanderbilt, Inc. (Ex. 3–108), supported the proposed 2-mg/m^3 respirable talc PEL. Mr. Kelse also recommended that OSHA revise its Table Z–3 entry for "Talc (nonasbestiform)" to "Talc (not containing asbestos)" and the entry for "Talc (fibrous)" to "Talc (containing asbestos)." These changes were suggested because of the potentially ambiguous meanings of the term "fibrous" and "asbestiform." OSHA concurs with this suggestion and has accordingly revised the respective entries in Tables Z–1–A and Z–3 in this rulemaking. In response to a suggestion by Richard Bidstrup, representing the Rubber Manufacturers Association (Ex. 173, p. 9), OSHA has also revised the entry for talc to clarify that the PEL is measured as respirable dust.

On a related issue, Mr. F.A. Renninger, Senior Vice President of the National Stone Association (Ex. 3–528), suggested that OSHA delete or clarify its current Table Z–3 entry for "Tremolite (see talc fibrous)" since it suggests that all forms of tremolite are considered to be asbestos. As Mr. Renninger points out, the applicability of OSHA's asbestos standard to the nonasbestiform varieties of tremolite, actinolite, and anthophyllite is currently under administrative stay, and OSHA is presently examining the health evidence for these mineral varieties. However, during this period of administrative stay, exposure to the nonasbestiform varieties of these minerals is covered by OSHA's comprehensive standard, which appears at 29 CFR 1910.1101. OSHA has therefore revised the entry for tremolite in Table Z–3 to refer to the standard at 29 CFR 1910.1101.

OSHA is establishing an 8-hour TWA limit of 2 mg/m^3 for the respirable dust of talc containing no asbestos fibers and less than 1 percent silica. The Agency concludes that this limit will protect workers from the significant risk of nonmalignant respiratory effects associated with exposure to talc dust; OSHA considers these effects material impairments of health. According to the ACGIH (1986/Ex. 1–3), talc may, at times, occur in a fibrous form. At this time, OSHA has not made any determinations with regard to the possible health consequences resulting from exposure to talc fibers.

TIN OXIDE

CAS: 7440–31–5; Chemical Formula: SnO
H.S. No. 1395

OSHA formerly had no exposure limit for tin oxide. The ACGIH has an exposure limit of 2 mg/m^3 as an 8-hour TWA. The proposed PEL was 2 mg/m^3 as an 8-hour TWA PEL; NIOSH (Ex. 8–47, Table N1) concurs, and this limit is established by the final rule. Tin oxide may be a white or yellow-brown powder.

Injection of tin dust intraperitoneally into guinea pigs resulted in a nonspecific, well-vascularized chronic granulomatous reaction (Oyanguren, Haddad, and Maass 1958/Ex. 1–652). Chronic exposure to tin oxide fume and dust results in stannosis, a form of pneumoconiosis. The fume of tin oxide is considered to be a more important source of stannosis than the dust (Dundon and Hughes 1950/Ex. 1–732), but other authorities consider the quality of the dust and the duration of exposure equally important (Robertson and Whittaker 1955/Ex. 1–987). The onset of the symptoms of stannosis may be delayed for years; the appearance of the condition is signalled by difficulty in breathing. One worker who had been exposed to unspecified tin oxide levels for 22 years was tested for stannosis and registered a vital breathing capacity 70 percent of normal and a maximal breathing capacity 61 percent of the predicted value (Spencer and Wycoff 1954/Ex. 1–611).

More than 150 cases of stannosis have been reported in the world literature (Robertson and Whittaker 1955/Ex. 1–987), and five cases were reported in the United States before 1954. No cases of massive fibrosis caused by exposure to tin oxide dust or fume have been reported (ACGIH 1986/Ex. 1–3, p. 574). Only NIOSH commented on tin oxide.

In the final rule, OSHA is establishing an 8-hour TWA of 2 mg/m^3 for tin oxide dust and fume. The Agency concludes that this limit will protect workers from the significant risks of reduced pulmonary capacity and stannosis, which are considered material impairments of health, associated with exposure to this substance at the levels permitted by the absence of an OSHA limit.

TRIMELLITIC ANHYDRIDE (TMAN)

CAS: 552–30–7; Chemical Formula: $C_9H_4O_5$
H.S. No. 1409

OSHA previously had no exposure limit for trimellitic anhydride. In 1981, the ACGIH set 0.005 ppm (0.04 mg/m^3) as the 8-hour TWA limit for this substance. The proposed PEL was 0.005 ppm as an 8-hour TWA, and the final rule promulgates this limit. NIOSH (Ex. 8–47, Table N1) concurs with this limit. Trimellitic anhydride is a colorless solid.

Exposure to trimellitic anhydride (TMAN) causes irritation of the eyes, nose, skin, and pulmonary tract. NIOSH (1978n, as cited in ACGIH 1986/Ex. 1–3, p. 606) reported in a current intelligence bulletin that trimellitic anhydride should be considered an extremely toxic workplace hazard, because exposure to it can cause noncardiac pulmonary edema and immunological sensitization, as well as upper respiratory tract irritation.

Pulmonary edema has occurred in workers exposed to TMAN at unreported air concentrations; the development of pulmonary edema in these workers without upper respiratory tract irritation suggests that TMAN is a sensitizer (Rice, Jenkins, Gray, and Greenburg 1977/Ex. 1–358). Zeiss, Patterson, Pruzansky, and colleagues (1977/Ex. 1–501) described TMAN-related illnesses among a group of workers synthesizing TMAN. These authors believe there are three separate syndromes associated with TMAN exposure: rhinitis/asthma; a flu-like condition; and irritation of the upper respiratory tract. Another case of TMAN-related occupational sensitization occurred in a worker exposed during the application of an epoxy resin coating (Fawcett, Taylor, and Pepys 1977/Ex. 1–636).

At levels averaging 1.5 and 2.8 mg/m^3 in two processes, NIOSH reported that employees reported eye and nose irritation, shortness of breath, coughing, nausea, headache, skin irritation, and throat irritation (NIOSH 1974c/Ex. 1–1181). Pulmonary hemorrhage and hemolytic anemia have been reported in workers exposed to TMAN at unspecified levels (Ahmad, Morgan, Patterson et al. 1979/Ex. 1–460).

Rats have shown intraalveolar hemorrhage after TMAN exposures to concentrations of 0.01 ppm (Amoco Chemical Corporation 1978, as cited in ACGIH 1986/Ex. 1–3, p. 606).

Based on this study, in the final rule OSHA is revising the PEL for trimellitic anhydride to an 8-hour TWA level of 0.005 ppm. The Agency concludes that this limit will protect workers from the severe pulmonary effects, sensitization, and skin and upper respiratory tract irritation observed in workers exposed to this extremely toxic substance. The Agency has determined that these effects constitute material impairments of health. OSHA finds that this limit will substantially reduce these significant risks, which were formerly not controlled because of the absence of any OSHA PEL.

WOOD DUST

CAS: None; Chemical Formula: None
H.S. No. 1430A (Hard Wood)
H.S. No. 1430B (Soft Wood)
H.S. No. 1430C (Western Red Cedar)

Before 1980, OSHA regulated wood dust under its nuisance dust standard of 15 mg/m^3 (29 CFR 1910.1000, Table Z–3). However, in a 1985 enforcement proceeding before the Occupational Safety and HealthReview Commission, wood dust was held not to be covered by the nuisance dust standard, an inert mineral dust, and the Agency did not regulate this substance after this decision (12 OSHC 1785). Consequently, OSHA had no PEL for wood dust when this generic rulemaking was undertaken. The ACGIH has a TLV–TWA of 1 mg/m^3 for hard wood dust, and a TLV–TWA of 5 mg/m^3 and STEL of 10 mg/m^3 for soft wood dust. OSHA proposed a 1–mg/m^3 8-hour TWA for hard wood dust and a 5-mg/m^3 8-hour TWA for soft wood dust. In the final rule, OSHA is establishing a single 8-hour TWA of 5 mg/m^3 and a STEL of 10 mg/m^3 for all hard wood and soft wood dusts except Western red cedar. For Western red cedar, a highly allergenic species of soft wood, the Agency is establishing an 8-hour TWA limit of 2.5 mg/m^3. Wood dust is defined as any wood particles arising from the processing or handling of woods. Hard woods derive from the deciduous broad-leaved flowering species of trees, and soft woods include the coniferous species that do not shed their leaves in the winter.

Exposure to wood dust has long been associated with a variety of adverse health effects, including dermatitis, allergic respiratory effects, mucosal and nonallergic respiratory effects, and cancer. The toxicity data in animals are limited, particularly with regard to exposure to wood dust alone; there are, however, a large number of studies in humans. The discussion below first describes some of the relevant toxicological studies and then presents the record evidence on wood dust.

Animal Studies

Groups of male guinea pigs were injected intratracheally with suspensions containing 75 mg of sheesham or mango wood dust or of hemp or bagasse fibers, or 20 mg of jute fiber (Bhattacharjee, Dogra, Lal, and Zaidi 1979/Ex. 1–463; Bhattacharjee and Zaidi 1982/Ex 1–464). Animals were sacrificed serially at intervals up to 90 days after injection. Lung examination revealed that, at 90 days, Grade I fibrosis of the lungs had occurred in the guinea pigs injected with mango or jute, while those treated with sheesham or hemp had developed Grade II pulmonary fibrosis.

In another experiment involving guinea pigs, animals were exposed by inhalation to average respirable dust concentrations of 1143 mg/m³ for 30 minutes/day, 5 days/week for 24 weeks (McMichael, DiPalma, Blumenstein et al. 1983, Ex. 1–644). Histopathological examination showed lung changes, described by the authors as moderate to severe, in all exposed guinea pigs. The changes seen included an increase in septal connective tissue components and aggregation of lymphocytes; however, no pulmonary fibrosis or extensive destruction of the parenchymal tissue occurred. The authors of this study concluded that exposure to fir bark dust may cause inflammatory changes in the lung.

Two studies examined the effect of exposing Syrian golden hamsters to beech wood dust by inhalation, with or without concurrent administration of the known carcinogen diethylnitrosamine (DEN) (Wilhelmsson, Hellquist, Olofsson, and Klintenberg 1985/Ex. 1–402; Wilhelmsson, Jernudd, Ripe, and Holmberg 1985/Ex. 1–1042; Drettner, Wilhelmsson, and Lundh 1985/Ex. 1–312). In each study, the animals were divided into four separate groups. In Study I, there were 12 animals per group. Two groups were exposed to fresh beech wood dust (a hard wood dust) at a mean total dust concentration of 15 mg/m³ for six hours/day, five days/week for 36 weeks, and one of these groups was also given 1.5 mg of DEN once a week for the first 12 weeks. The third group in Study I was given the DEN doses only (positive control), and the fourth group was given no exposure at all (negative control).

In Study II, there were 24 animals in each of four groups. Two groups of animals were exposed to fresh beech wood dust at a mean total dust concentration of 30 mg/m³ for six hours/day, five days/week for 40 weeks. The positive and negative control groups were treated as in Study I.

In Study I, none of the hamsters had lung or nasal tumors or metaplasia. Four hamsters exposed to wood dust and DEN exhibited squamous cell papillomas of the trachea, as did three animals in the positive control group and one in the negative control group. No differences in organs other than the respiratory organs were seen between the treated and control groups in Study I.

In Study II, all DEN-exposed hamsters had nasal lesions ranging from hyperplasias and dysplasias to papillomas. In addition, half of all DEN-exposed hamsters developed nasal adenocarcinomas, whether or not they had also been exposed to wood dust. Half of the DEN-exposed animals also had papillomas of the larynx and trachea. In the wood-dust-exposure-only group, two of the animals had nasal lesions, one of which was an unclassifiable malignant nasal tumor and the other of which consisted of focal metaplasia with mild dysplasia. The authors concluded that exposure to wood dust did not increase the tumor incidence in DEN-exposed animals but did affect the respiratory tract of all exposed animals.

Human Studies

Dermatitis. There are a large number of case reports, epidemiological studies, and other data on the health effects of wood dust exposure in humans. Dermatitis caused by exposure to wood dusts is common, and can be caused either by chemical irritation, sensitization (allergic reaction), or both of these together. As many as 300 species of trees have been implicated in wood-caused dermatitis.

The chemicals associated with allergic reactions are generally found in the inner parts of a tree, e.g., the heartwood, and the workers most prone to these reactions are those involved in secondary wood processing (e.g., carpenters, joiners, and finishers).

The symptoms of sensitization are redness, scaling, and itching, which may progress to vesicular dermatitis and, after repeated exposures, to chronic dermatitis. The parts of the body most often affected are the hands, forearms, eyelids, face, neck, and genitals. This form of dermatitis generally appears after a few days or weeks of contact.

Allergic respiratory effects. Allergic respiratory responses are mediated by the immune system, as is also the case with allergic dermatitis. Many authors have reported cases of allergic reactions in workers exposed to wood dust (Sosman, Schlueter, Fink, and Barboriak 1969/Ex. 1–444; Greenberg 1972/Ex. 1–482; Pickering, Batten, and Pepys 1972/Ex. 1–655; Eaton 1973/Ex. 1–478; Booth, LeFoldt, and Moffitt 1976/Ex. 1–466; Chan-Yeung, Ashley, Corey et al. 1978/Ex. 1–622; Edwards, Brooks, Henderson, and Apol 1978/Ex. 1–950; Innocenti and Angotzi 1980/Ex. 1–1036; Bush and Clayton 1983/Ex. 1–469; Cartier, Chan, Malo et al. 1986/Ex. 1–472). Asthma is the most common response to wood dust exposure, and the allergic nature of such reactions has been demonstrated by the presence of IgE antibodies and positive skin reactions on patch testing. The best-studied of the allergic reactions to wood dust is Western red cedar (WRC) asthma; it is estimated that 5 percent of the workers handling this species are allergic to it. However, only one study is available that relates exposure level to ventilatory function. In that study, exposure to concentrations of 2 mg/m³ of WRC dust caused significant decreases in forced vital capacity and forced expiratory volume (Vedal, Chan-Yeung, Enarson et al. 1986/Ex. 1–397). These authors also found that exposures to concentrations above 3 mg/m³ produced eye irritation.

Mucosal and nonallergic respiratory effects. This section discusses changes in the structure and function of the nasal mucosa and respiratory tract that are caused by exposure to wood dust. These changes include nasal dryness, irritation, bleeding, and obstruction; coughing, wheezing, and sneezing; sinusitis; and prolonged colds. These symptoms have been observed even at wood dust concentrations below 4 mg/m³.

Bellion, Mattei, and Treves (1964, as cited in NIOSH 1987a/Ex. 1–1005) found that 97 of 225 workers (carpenters, sawmill workers, woodworkers) exposed from 3 to 24 years to the dust of several different hard woods showed radiologic evidence of pulmonary abnormalities. Black, Evans, Hadfield et al. (1974/Ex. 1–299) studied nine woodworkers from a woodworking factory in England. In all of these workers, mucociliary movement was markedly depressed, leading these authors to conclude that exposure to wood dust in the furniture industry for 10 years or more can impair mucociliary clearance. These findings were confirmed in a Danish study involving furniture makers (Solgaard and Andersen 1975/Ex. 1–443; Andersen, Solgaard, and Andersen 1976/Ex. 1–297; Andersen, Andersen, and Solgaard 1977/Ex. 1–296); compared with controls, the mucociliary transport rate was also significantly impaired in these woodworkers, and dose-response effects were noted.

A respiratory survey conducted by Chan-Yeung, Giclas, and Henson (1980/Ex. 1–474) in pulp and paper mill workers in British Columbia showed that workers exposed to wood dust at a mean total dust concentration of 0.5 mg/m³ had a slight but statistically significant decrease in pulmonary function values compared with controls. The authors concluded that the chemical preservatives used to treat the wood could also have been responsible for these adverse effects.

In a cross-sectional survey of 1,157 American woodworkers (both hard and soft wood), Whitehead, Ashikaga, and Vacek (1981/Ex. 1–454) found that exposure to higher (10+ mg-years/m³), as compared with lower (0 to 2 mg-

years/m^3), dust concentrations was associated with a statistically significant and higher incidence of decreased pulmonary function. However, dose-response effects were observed only for soft wood (i.e., pine) dusts. A later study by Beckman, Ashikaga, and Whitehead (1980, as cited in NIOSH 1987a/Ex. 1-1005) examined subgroups of the workers studied by Whitehead and found no correlation between years of exposure to pine wood dust and pulmonary function.

In a pilot study of 55 workers in a North Carolina hardwood furniture plant, Goldsmith (1983, as cited in NIOSH 1987a/Ex. 1-1005) found that, at mean area wood dust concentrations of 2 mg/m^3 or below, peak ventilatory flow correlated significantly with cumulative person-years of exposure. Goldsmith interpreted this finding to mean that inhalation of wood dust may impair large-airway function.

A study of Italian woodworkers showed that the number of wood-dust-exposed workers who had developed anosmia (loss of smell) was significantly higher than in a control group of nonexposed workers (Innocenti, Valiani, Vessio et al. 1985/Ex. 1-1037). Amoore (1986/Ex. 1-1029) confirmed this finding in other workers exposed to hardwood dusts.

Summary of mucosal and nonallergic respiratory effects. A large number of studies have demonstrated that occupational exposure to wood dust causes both statistically significant and nonsignificant increases in respiratory symptoms at exposure levels as low as 2 mg/m^3. These symptoms range from irritation to bleeding, wheezing, sinusitis, and prolonged colds. In addition, chronic wood dust exposure causes mucociliary stasis (i.e., the absence of effective clearance) in the nose and, in some workers, also causes changes in the nasal mucosa. Several studies have demonstrated decreased pulmonary function among wood-dust-exposed workers, although other studies have not confirmed these findings.

Carcinogenicity

The association between occupational exposure to wood dust and various forms of cancer has been explored in many studies and in many countries. In 1987, the International Agency for Research on Cancer (IARC) classified furniture manufacturing in Category I (confirmed human carcinogen) and carpentry in Category 2B (suspected human carcinogen). NIOSH (Ex. 8-47) considers both hard and soft wood dust to be potentially carcinogenic in humans; for soft wood dust, NIOSH recommends a separate 6(b) rulemaking (Ex. 8-47, Table N6B). NIOSH concurred, however, with the proposed PEL of 1 mg/m^3 TWA for hard wood dust (Ex. 8-47, Table N6A).

The discussion below focuses on selected U.S. studies.

Nasal and sinus cavity cancer. The earliest U.S. study of wood dust exposure and nasal cancer was conducted by Brinton, Stone, Blot, and Fraumeni (Ex. 1-468) in 1976. These authors analyzed cancer death rates between 1950 and 1969 in 132 U.S. counties having at least 1 percent of their population employed in furniture and wood-fixture manufacturing. This study revealed that the age-adjusted mortality rate for cancer of the nasal cavity and sinuses among white males in the "furniture" counties was significantly higher than in nonfurniture counties.

In a later case-control study, these authors (Brinton, Blot, Becker et al. 1984/Ex. 1-467) analyzed cases of nasal and sinus cancers occurring in North Carolina and Virginia between 1970 and 1980. This study identified a significantly elevated risk of adenocarcinomas in males working in the furniture manufacturing industry, but no increased risk among lumber, carpentry, or construction workers. There was no significant increase in the risk of squamous cell carcinoma in workers from any other wood-related industry.

In a study sponsored by the Inter-Industry Wood Dust Task Force, Viren, Vogt, and Dixon (1982, as cited in NIOSH 1987a/Ex. 1-1005) described a death certificate case-control study of nasal cancer deaths for 1963 to 1977 in North Carolina, Mississippi, Washington, and Oregon. Findings of this study included a relative nasal cancer risk of 1.95 for industries involving lumber and wood products; however, no significant relative risk of nasal cancer was seen for workers in the furniture-manufacturing industry.

Imbus and Dyson conducted a study of nasal cancer and North Carolina furniture workers (1985, as cited in NIOSH 1987a/Ex. 1-1005). This study found: (1) that there was a statistically significant increase of nasal cancer among furniture workers; (2) that the nasal cancer rates among North Carolina furniture workers were much lower than those reported for English furniture workers; (3) that the number of nasal cancer deaths among North Carolina furniture workers decreased between 1956 and 1977; and (4) that a slight excess in nasal cancer may have existed among North Carolina furniture workers but is currently either declining or nonexistent.

At present, the National Cancer Institute is conducting a cohort mortality study of 36,622 workers employed in the wood, metal, and plastic furniture manufacturing industries (Miller et al. 1988, as cited in NIOSH 1987a/Ex. 1-1005). Results are too preliminary to be described at this time.

Summary of evidence for nasal and sinus cavity cancers. NIOSH (1987a/Ex. 1-1005) concluded that the literature clearly demonstrates an association between occupational wood dust exposure and nasal cancer. English studies first identified this link by showing a 10- to 20-times-greater incidence of nasal adenocarcinoma among woodworkers in the furniture industry than among other woodworkers and 100 times greater than in the general population. In the United States, three studies have reported a fourfold risk of nasal cancer or adenocarcinoma in furniture workers, and another study noted a similar relationship between nasal cancer and wood dust exposure. One other study failed to find such an association for furniture workers, but did find an increase among logging and timber industry workers.

Pulmonary cancer. A number of studies investigating the association between wood dust exposure and the development of lung cancer have been conducted. Milham (1974/Ex. 1-943) found a significant excess of malignant tumors of the bronchus and lung in workers who had belonged to the AFL-CIO United Brotherhood of Carpenters and Joiners of America. Only construction workers showed a statistically significant increase in lung cancer rate.

In a study of lung cancer in Florida residents, Blot, Davies, Brown et al. (1982/Ex. 1-465) found that an elevated risk of lung cancer that was statistically significant existed among workers in the lumber and wood industry and in construction; however, smoking may have been a confounding factor in these results.

Summary of evidence for pulmonary cancer. The association between lung cancer and occupational wood dust exposure is inconclusive, although several epidemiological studies have reported increases in lung cancer among wood-dust-exposed workers.

Hodgkin's disease. The data on the relationship between exposure to wood dust and the development of Hodgkin's disease are conflicting. Milham (1967/Ex. 1-750) and Milham and Hesser (1967/Ex. 1-645) concluded, on the basis of a case-cohort study of 1,549 white males dying of this disease between 1940-1953 and 1957-1964, that there was

an association between Hodgkin's disease and exposure to wood dust.

Another study (Spiers 1969/Ex. 1-445) concluded that men working in the wood industries in the eastern United States were at special risk for Hodgkin's disease, and suggested that pine pollen exposure might be responsible for the increase.

A Washington State epidemiological study (Petersen and Milham 1974/Ex. 1-654) also found that woodworkers had an increased risk of Hodgkin's disease, and the work of these authors was supported by the results of another study (Grufferman, Duong, and Cole 1976/Ex. 1-484), which showed a nonsignificant increase in the relative risk for Hodgkin's disease among woodworkers.

Summary of evidence for Hodgkin's disease. Although the data are conflicting, several epidemiological studies of U.S. workers do report increases in the incidence of Hodgkin's disease among woodworkers. This excess is particularly apparent among carpenters.

Other cancers. NIOSH (1987a/Ex. 1-1005) concluded that the data on the relationship between occupational exposure to wood dust and the development of cancers other than nasal, Hodgkin's disease, or lung cancers are insufficient and inconclusive.

Record Evidence

Many participants submitted comments to the record pertaining to wood dust (see, for example, Exs. 8-34, 3-748, 3-233, 3-349, 3-362, 3-626, 3-682, 3-824, 3-836, 3-859, 3-899, 3-955, 3-1160, 3-917, 115, 127, 131, 141, 155, 168, 183, 191, 194, 3-1453, 195, 196, 189, 82, 80, and 3-911; Tr. 12, pp. 144 to 455). These commenters described their facilities and woodworking processes, employee safety and health programs, and concerns about the impact of the proposed rule's limits for wood dust on their industries. The issues raised by these participants concerned the following topics:

(1) The technological and economic feasibility of the proposed limits;

(2) The justification for a separate standard for soft wood and hard wood;

(3) The health effects evidence;

(4) The appropriate levels for the final rule's PELs; and

(5) The evidence for a separate limit for allergenic wood dusts.

The discussions below deal with each of these points in turn.

Representatives from many affected segments of the wood industry stated that achieving the proposed limits of 1 mg/m^3 for hard wood and 5 mg/m^3 for soft wood would be technologically or economically infeasible or extremely difficult (Exs. 8-34, 3-917, 168, 183, 191, 80, and 3-911). OSHA has determined that, at the present time, the health evidence suggests that a single PEL of 5 mg/m^3 is appropriate for both hard and soft wood dust, with the exception of Western red cedar, for which a PEL of 2.5 mg/m^3 is being set. These revised PELs have been determined to be feasible (see the detailed discussion of these issues in the Technological Feasibility and Economic Impact sections of this preamble).

OSHA proposed separate permissible exposure limits for soft wood (5 mg/m^3) and hard wood (1 mg/m^3). The Agency received comments on this topic from many participants; these commenters were unanimously opposed to the setting of separate limits for these two types of wood dust (Exs. 8-34, 3-748, 3-682, 3-859, 3-899, 3-917, 191, 196, 80G, 80L, 80N, and 3-911; Tr. XII, pp. 12-290, 12-326, and 12-331). These participants stated that there was no health basis for making a distinction between hard wood and soft wood dusts (Exs. 33-899, 3-955, 3-917, and 191; Tr. 12, pp. 326-331; Tr. 12, p. 290). According to Dr. Harold Imbus, speaking for the Inter-Industry Wood Dust Coordinating Committee (Tr. pp. 12-58, 12-60), the distinction between the two woods derived from the fact that the early studies showing an increased cancer incidence in woodworking employees involved British furniture makers, who predominantly used hard wood; this association caused investigators to attribute greater toxicity to hard wood dust.

Commenters were of the opinion that this distinction was no longer warranted by the evidence; in fact, Dr. Lawrence Whitehead, certified industrial hygienist and a professor at the University of Texas School of Public Health (Tr. p. 12-331), stated that his own work suggested that some soft wood dust exposures might actually produce stronger adverse effects than equivalent exposures to some hard wood dusts.

Other commenters reported that it is not possible to distinguish soft wood from hard wood dust except by chemical analysis (Ex. 8-34, p. 28), that most facilities in the wood industries use both hard and soft woods (Exs. 3-682, 3-859, and 3-899), and that the distinction between the two types of woods is inappropriate (Ex. 3-917). For example, Joseph Gerard, Vice President of the American Furniture Manufacturers Association (Ex. 3-917) stated:

> The distinction between hard woods and soft woods is purely botanical. Many so-called "softwoods" are actually hard (i.e., Douglas fir as a softwood is harder than the hardwood birch) and one of the softest woods in existence (balsa) is botanically a hardwood (Ex. 3-917, p. 2).

Jamie Cohen, speaking for the United Petitioners, a coalition of labor unions (Tr. 12, p. 294), believes that a bifurcated standard for the two types of dusts would place an undue burden on employers and could lead to compliance problems. The posthearing brief submitted by the United Brotherhood of Carpenters and Joiners of America (Ex. 196) reiterated these points by stating: "Given the frequent intermixture of wood types in the workplace, this [setting two separate standards] would render OSHA's compliance efforts virtually worthless" (Ex. 196, p. 7).

After a review of this record evidence, OSHA has determined that the health evidence for the toxicity of wood dust cannot be separately distinguished for soft wood and hard wood. In addition, the Agency is convinced by the many comments from wood industry employers that most operations involve both kinds of wood and are performed on the same machines and equipment and in the same facility. Thus, any controls installed to reduce exposures would of necessity need to be sufficient to reduce airborne dust levels to the lower of the two limits (i.e., to the proposed wood dust limit of 1 mg/m^3). According to the Inter-Industry Wood Dust Coordinating Committee:

> [I]mposition of a limit of 1 mg/m^3 for hardwood dust and 5 mg/m^3 for softwood dust effectively imposes a limit of 1 mg/m^3 on a large number of plants, including those where only small amounts of hardwood are used (Ex. 3-748, p. 3).

Many commenters took exception to the review of the health effects evidence for wood dust presented by OSHA in the preamble to the proposed rule. Objections were raised by the Inter-Industry Wood Dust Coordinating Committee (Exs. 8-34, 3-748, and 168), the Appalachian Hardwood Manufacturers (Ex. 3-626), the American Furniture Manufacturers Association (Exs. 3-917 and 191), the Georgia-Pacific Corporation (Exs. 3-955 and 183), the Hardwood Plywood Manufacturing Association (Ex. 3-911), and others.

These participants criticized many of the individual studies described by OSHA; some commenters found fault with several of these studies on the grounds that they involved British or other non-U.S. woodworkers (see, for example, Exs. 8-34, 191, 3-626, and 3-917), involved only a small number of subjects (see, for example, Exs. 8-34, 168, and 191), had inconsistent results

(see, for example, Ex. 8–34), or failed to demonstrate a dose-response relationship between wood dust exposure and the health effect of concern (see, for example, Exs. 8–34, 3–626, 3–917, and 191). The Inter-Industry Wood Dust Coordinating Committee (IWDCC) stated:

[T]he observations in the European studies are not representative of conditions in U.S. workplaces, especially under modern conditions. . . .

The English and other European experience does not provide an accurate predictive model for the incidence of nasal cancer. . . . The excesses of nasal cancer observed in the European studies simply have not been observed in the United States at any time . . . (Ex. 3–748, pp. 2, 52).

OSHA agrees with the IWDCC that the *incidence* of nasal cancer seen in the United States is substantially lower than that seen in other countries, particularly in Great Britain. However, the Agency does not agree that excesses in nasal cancers, and particularly of nasal adenocarcinomas, have not been observed in American woodworkers. Several U.S. studies have reported excesses in nasal cancer risks among employees in the wood industries (Brinton, Stone, Blot, and Fraumeni 1976/Ex. 1–468; Brinton, Blot, Becker et al. 1984/Ex. 1–467; Viren, Vogt, and Dixon 1982, and Imbus and Dyson 1985, both as cited in NIOSH 1987a/Ex. 1–1005).

In response to those commenters who argued that none of the studies described by OSHA presented sufficient dose-response data to be used as a basis for establishing a limit, the Agency emphasizes that it is not relying on any single study to determine that wood dust presents a significant risk of material health impairment. Instead, OSHA is making this determination on the basis of the findings in the dozens of studies reporting on the respiratory, irritant, allergic, and carcinogenic properties of wood dust. The Agency finds the results of these studies biologically plausible and their findings reproducible and consistent. It is true that some of these studies, like all human studies, have limitations of sample size, involve confounding exposures, have exposure measurement problems, and often do not produce the kind of dose-response data that can be obtained when experimental animals are subjected to controlled laboratory conditions. What the large group of studies being relied upon by OSHA to establish the significance of the risk associated with exposure to wood dust do show is that the overall weight of evidence that such exposures are harmful and cause loss of functional capacity and material impairment of health is convincing beyond a reasonable doubt.

The industry strongly supported a single 5-mg/m^3 standard for both hard wood and soft wood dusts (Exs. 8–34, 3–626, 3–682, 3–824, 3–899, 3–1160, 3–917, 168J, 183, 191, 80 and attachments, and 3–911); some commenters (Exs. 3–859, 194, and 196) argued for a 1-mg/m^3 limit for all wood dust, while others (Exs. 3–955, 155, and 183) were of the opinion that the nuisance dust limit of 10 mg/m^3 was appropriate for wood dust. The United Petitioners (Tr. p. 12–294) strongly endorsed a 1-mg/m^3 standard for wood dust of all types on the grounds that the available health evidence clearly supports this limit.

OSHA finds that the health evidence in the record as a whole does not support a PEL of 1 mg/m^3 for all wood dusts. In addition, the Agency believes that a 1-mg/m^3 limit would present serious problems of feasibility for affected parties (see Section VII, Summary Economic Impact and Regulatory Flexibility Analysis). The Agency also finds that the health evidence clearly indicates that occupational exposure to wood dust poses a significant risk of material health impairment at the 10-mg/m^3 (or particulate) level. OSHA concludes that establishing an 8-hour PEL of 5 mg/m^3 and a 15-minute STEL of 10 mg/m^3 for all wood dusts (except Western red cedar) will substantially reduce this significant risk.

The final rule establishes an 8-hour TWA PEL of 2.5 mg/m^3 for Western red cedar wood dust, based on its widely recognized ability to cause immune-system-mediated allergic sensitization. Evidence in the record demonstrates the seriousness of this effect. A study by Brooks, Edwards, Apol, and Edwards (1980) that was submitted by the United Petitioners (Ex. 82D) reports that

a high prevalence of occupational asthma was observed among workers exposed to WRC wood dust (Ex. 82D, p. 315).

At the hearing, Dr. Brooks described occupational asthma as follows:

[T]here are spasms of the bronchial tubes, there is reduced air flow on expiration . . . [the extent of which depends] on the extent of the exposure, and also . . . on the duration of the exposure . . . as a consequence of this sensitization and airway injury from the sensitization and the asthmatic reaction and the various biochemical and cellular changes that occur, there develops an associated process . . . the airways develop an increased sensitivity and an increased bronchospastic responsiveness to many different non-specific stimuli. So such things as cold air, dust, fumes, gases that are non-specific and wouldn't normally . . . [affect] most individuals [will affect] the individual with occupational asthma. And it's [such] hyper-reactive airways that cause individuals to continue to have disability and to continue to have symptoms once they leave the work place. . . . They develop this non-specific bronchial hyper-reactivity which may last the rest of their life (Tr. pp. 12–339 to 12–343).

Some commenters (Exs. 8–34, 183, and 191) opposed the establishment of a separate PEL for Western red cedar. These participants argued that a lower PEL "for wood dust generally would be necessary or appropriate to address allergic symptoms" (Ex. 8–34, Health Effects Comments, p. 8, footnote 6). According to the Inter-Industry Wood Dust Coordinating Committee (IIWDCC):

[P]revention of allergic reactions is best achieved by good housekeeping measures directed specifically at the allergenic species (Ex. 8–34, p. 8).

Among the work practices recommended by these commenters were maintaining clean work spaces, wearing protective clothing, and avoiding skin contact with the allergenic species (Ex. 8–34, Health Effects Comments, p. 17).

Although OSHA endorses training, good work practices, and the use of appropriate protective clothing, the Agency does not agree that a reduced PEL for Western red cedar (WRC) is unnecessary. The health effects associated with occupational exposure to WRC are too severe not to be cause for concern. In addition, there is good evidence in the record of the dose-response relationship between occupational exposure to WRC dust and woodworkers' asthma. A study by Vedal, Chan-Yeung, Enarson et al. (1986/Ex. 1–397) shows such a relationship, with asthma beginning at a WRC level of 3.4 mg/m^3 and a statistically significant reduction in forced respiratory capacity noted in workers exposed to 2 mg/m^3 WRC dust or more. Harold Imbus, a physician representing the IIWDCC, stated:

This study, small though it may be, tends to support dose response, and a threshold level between 2 and 3.4 mg/m^3 for the protection of effects of WRC (Ex. 8–34, p. 7).

The 1980 study by Brooks, Edwards, Apol, and Edwards found a dose-related relationship between total WRC dust level and prevalence of asthma in employees in jobs with the greatest dust exposures. The Brooks et al. study found asthma in zero percent of WRC workers exposed at 0.5 mg/m^3; however, at 3.56 mg/m^3, this percentage rose to 5 percent.

The United Petitioners submitted a 1988 paper by Goldsmith and Shy that

found that there is a clearly defined asthma syndrome produced by WRC (Ex. 3–362). OSHA finds these studies convincing evidence of WRC's allergenic potential; in addition, the Agency believes that a threshold for occupational asthma exists and lies between 2 and 3.4 mg/m³. Based on this evidence, OSHA concludes that an 8-hour PEL of 2.5 mg/m³ is necessary to protect workers from the significant and often permanent effects of immune-mediated occupational asthma associated with exposure to WRC dust at levels above this limit. Several record comments agree that a separate PEL for WRC dust is warranted and that the threshold level is as described above (see, for example, Exs. 8–34 (Imbus review, p. 6), 168, and 191; Tr. p. 12–292; Tr. pp. 12–317, 12–318, and 12–320).

Some commenters (Tr. p. 12–316) were of the opinion that many other woods, such as Douglas fir, pine, red and white oak, redwood, walnut, spruce, boxwood, cocobolo, teak, mahogany, and others, should also be designated by OSHA as allergenic in this rulemaking. However, OSHA finds that, as Dr. Imbus of the IIWDCC notes, "it is unlikely that species other than WRC are responsible for large numbers of cases of respiratory allergies" (Ex. 8–34, Imbus review, p. 6). The authors of the Goldsmith and Shy (1988) paper concur:

> Other commonly used woods such as oak, birch, redwood, pine, teak, alder, and hemlock, produce pulmonary effects that are less well described than the asthma responses to Western red cedar (Ex. 3–362, p. 13).

The IIWDCC contends that, at the present time, there is "no consensus even as to which species should be considered allergenic" (Ex. 168). OSHA concludes that other species are somewhat allergenic. The evidence in the literature does not indicate that any other species is nearly as allergenic as WRC or would cause nearly as high a proportion of allergic reactions among exposed workers. However, the Agency will monitor the literature on these other potentially allergenic species so that other woods with demonstrably allergenic properties can be identified in the future.

Based on the evidence presented above, OSHA is establishing a PEL of 5 mg/m³ as an 8-hour TWA and 10 mg/m³ as a 15-minute STEL for hard and soft wood dust, with the exception of Western red cedar, for which a PEL of 2.5 mg/m³ (8-hour TWA) is being established. OSHA concludes that promulgation of these exposure limits will substantially reduce the significant risk of material impairment in the form of pulmonary dysfunction (including changes in peak flow, interference with mucociliary clearance, respiratory symptoms, and chronic effects) that is associated with exposure to wood dust at the higher levels that would be permitted in the absence of any limit.

Conclusions For All Respiratory Toxicants

As Table C6–2 and the discussions above show, limits for the respiratory toxins have been established to control employee exposures to or below the airborne concentrations of these substances that have been associated with the development of acute or chronic respiratory effects. For most of these substances, the evidence is sufficient to identify the NOE or low-effect levels that are related to these effects in humans or animals. Accordingly, OSHA concludes that maintaining employee exposures at or below these limits will greatly decrease the likelihood that employees will be at significant risk of respiratory effects when they are exposed to these substances in the workplace. Because the chronic pulmonary disease caused by exposure to toxic dusts is often incapacitating, such exposures can effectively end the working life of severely affected individuals. Less-serious pulmonary disease can result in lost workdays, both as a result of the associated symptoms themselves and as a consequence of increased susceptibility to respiratory infections. The effects of exposure to acute pulmonary toxins, such as ozone or trimellitic anhydride, range from reduced lung function to life-threatening pulmonary edema. OSHA has determined that these adverse pulmonary effects constitute material impairments of health. Lowering the Agency's former limits or establishing limits where none previously existed will substantially reduce these significant occupational risks.

7. Substances For Which Limits Are Based on Avoidance of Cardiovascular Effects

Introduction

For seven chemicals, OSHA is revising or establishing limits based on their adverse effects on the cardiovascular system. Table C7–1 lists the former, proposed, and final Z-table limits for these substances, along with their CAS numbers and HS numbers. OSHA is revising its current ceiling limits for two substances (ethylene glycol dinitrate and nitroglycerin) by replacing them with lower short-term limits. OSHA is reducing the TWA–PEL for carbon disulfide to 4 ppm and adding a STEL of 12 ppm. For one other substance (fluorotrichloromethane), OSHA is replacing its current TWA–PEL with a ceiling value; for 1,1,2-trichloro-1,2,2-trifluoroethane, OSHA is adding a STEL to its existing 8-hour TWA. The Agency is establishing new limits for two cardiovascular toxins, chloropentafluoroethane and sodium azide.

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TABLE C7-1. List of Substances for Which Limits Are Based on Avoidance of Cardiovascular Effects

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1070 Carbon disulfide	75-15-0	20 ppm TWA 30 ppm STEL (30 min) 100 ppm Ceiling	1 ppm TWA 10 ppm STEL	4 ppm TWA 12 ppm STEL, Skin
1087 Chloropentafluoroethane	76-15-3	--	1000 ppm TWA	1000 ppm TWA
1170 Ethylene glycol dinitrate	628-96-6	1 mg/m^3 Ceiling, Skin	0.1 mg/m^3 STEL (20 min)	0.1 mg/m^3 STEL, Skin
1180 Fluorotrichloromethane	75-69-4	1000 ppm TWA	1000 ppm Ceiling	1000 ppm Ceiling
1290 Nitroglycerin	55-63-0	1 mg/m^3 Ceiling, Skin	0.1 mg/m^3 STEL (20 min)	0.1 mg/m^3 STEL, Skin
1364 Sodium azide	26628-22-8	--	0.1 ppm Ceiling	0.1 ppm Ceiling, Skin
1403 1,1,2-Trichloro-1,2,2-trifluoroethane	76-13-1	1000 ppm TWA	1000 ppm TWA 1250 ppm STEL	1000 ppm TWA 1250 ppm STEL

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

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Description of the Health Effects

Although the cardiovascular system can be adversely affected in many different ways by exposure to toxic substances, the adverse effects caused by exposure to the seven chemicals in Table C7–1 are limited to three categories: (1) cardiac sensitization; (2) vasodilation; and (3) atherosclerosis. Because these effects can have potentially disabling or life-threatening outcomes, OSHA has determined that these effects clearly constitute material impairments of health and functional capacity.

Cardiac sensitization is not mediated by the immune system and does not cause an allergic reaction. Instead, this form of sensitization occurs when a chemical "sensitizes" the heart to the effects of a class of biological compounds called sympathomimetic amines. The physiological action of sympathomimetic amines is to stimulate the heart to beat faster. The hormone adrenaline, also called epinephrine, is an example of a sympathomimetic amine. Adrenaline is normally secreted into the bloodstream when the body anticipates an increase in physical exertion, such as occurs when someone is frightened. A concentration of epinephrine equal to or higher than the no-effect level for this substance is necessary to increase the heartbeat rate in exposed individuals. The effect of a cardiac sensitizer is to lower the no-effect level so that the heartbeat rate is stimulated by a lower concentration of adrenaline. The region of the heart that becomes sensitized is the pacemaking and conduction system, which determines the rhythm and rate of the heartbeat. Unregulated or unnecessary interference with this region of the heart can result in arrhythmia, an abnormality in the rhythm or rate of the heartbeat (Levy 1985/Ex. 1–210). The clinical consequences of an arrhythmia vary among individuals, e.g., a young person with a healthy heart may not be adversely or seriously affected by an arrhythmia. However, fatal arrhythmias have occurred in healthy young people and, in older people or in individuals whose cardiovascular systems have already been compromised, arrhythmias can cause symptoms of cerebral or myocardial ischemia, shock, or congestive heart failure.

Vasodilators are compounds that cause blood vessels to expand, resulting in a decrease in blood pressure (hypotension) and a decrease in the amount of blood reaching the organs. Acute hypotension is a common cause of shock (*Harrison's Principles of Internal Medicine*, 10th ed., Petersdorf et al. 1983). Chronic hypotension may result in a number of symptoms, including lethargy, weakness, easy fatigability, and dizziness or faintness.

Atherosclerosis is a serious disease produced by a degenerative process in the arteries. Plaques containing lipids, complex carbohydrates, blood products, and calcium form on the inside walls of arteries, usually on major blood vessels. These plaques are also called atheromas; their presence makes arteries narrower. Depending on which arteries in the body contain atheromas, different clinical consequences may result; these include renal hypertension, stroke, and myocardial ischemia (inadequate circulation of blood to the myocardium) (Balazs, Hanig, and Herman 1986/Ex. 1–176). Some chemicals can enhance or accelerate the formation of atheromas and thereby encourage the development of atherosclerosis, a major cause of coronary heart disease.

Dose-Response Relationships and Cardiac Effects

For four of the chemicals in Table C7–1 (carbon disulfide, ethylene glycol dinitrate, nitroglycerin, and sodium azide), the final rule's limits are based primarily on health surveys and case reports indicating that occupationally exposed workers subjected to concentrations above a no-adverse-effect level experience these cardiovascular effects. However, human data for the other three chemicals (chloropentafluoroethane, fluorotrichloromethane, and 1,1,2-trichloro-,1,2-trifluoro-ethane) are scarce. For these chemicals, limits are based on the results of studies in laboratory animals.

Chemically induced cardiovascular disease occurs in a pattern that corresponds to a typical effect-level dose-response relationship; that is, an exposure level and exposure duration exist below which the substance appears unlikely to exert an adverse effect. Thus, the limits for substances in this group are designed to maintain exposures below this apparent no-adverse-effect level.

The following discussions describe the record evidence and OSHA's findings for some substances in this group and point to the seriousness of the cardiovascular effects associated with exposure to these substances.

CARBON DISULFIDE

CAS: 75–15–0; Chemical Formula: CS_2
H.S. No. 1070

OSHA's former limits for carbon disulfide were 20 ppm as an 8-hour TWA, a 30-minute STEL of 30 ppm, and a ceiling limit of 100 ppm that was never to be exceeded. OSHA proposed revising these limits to 1 ppm as an 8-hour TWA and 10 ppm as a 15-minute STEL, and NIOSH (Ex. 8–47, Table N1) supported these proposed limits. OSHA has evaluated all of the evidence and testimony presented in the record and has determined that a 4-ppm 8-hour TWA limit, a 12-ppm STEL, and a skin notation are necessary to reduce the risk of cardiovascular disease and reproductive effects among carbon-disulfide-exposed workers, and the Agency is establishing these limits for carbon disulfide in the final rule. The need for a lower limit is based on evidence that exposure to carbon disulfide presents risks of cardiovascular, fetotoxic, and neurological material impairment of health.

OSHA's decision to promulgate a 4-ppm limit rather than the proposed 1-ppm limit is principally based on the feasibility evidence available to OSHA (see Section VII, Technological Feasibility and Economic Impact Assessment). A skin notation has been added because there is evidence that carbon disulfide can cause systemic toxicity via the dermal route. Carbon disulfide is a clear, colorless, or faintly yellow liquid with a strong, disagreeable odor.

OSHA's proposal to reduce the limits for carbon disulfide was based on a number of human studies reviewed by the ACGIH (1986/Ex. 1–3) and NIOSH (1977b/Ex. 1–260) that suggested that exposure to carbon disulfide levels between 10 and 40 ppm was associated with an excess risk of coronary heart disease and of adverse neurological effects. These reports comprise a series of studies carried out on carbon-disulfide-exposed workers in Great Britain (Tiller, Schilling, and Morris 1968/Ex. 1–92) and Finland (Seppalainen and Tolonen 1974/Ex. 1–100; Tolonen et al. 1975/Ex. 1–392; Tolonen, Nurminen, and Hernberg 1979/Ex. 1–158). The British cohort was recently followed up by Sweetnam et al. (1987), and the Finnish workers have been followed up by Nurminen and Hernberg (1985).

The study by Tiller et al. (1968/Ex. 1–92) of British rayon workers was the first to relate exposure to carbon disulfide to the development of coronary heart disease. These authors found that, among men employed for more than 10 years in the rayon industry and followed from 1950 to 1964, those exposed to carbon disulfide had death rates from coronary heart disease more than twice the rate in other rayon workers. Thus, the Tiller et al. (1968/Ex. 1–92) study

demonstrated that 10 years or more of exposure to carbon disulfide was associated with a significantly elevated risk of coronary disease.

The United Kingdom's threshold limit value for carbon disulfide, which had been 20 ppm in the 1960s, was subsequently reduced to 10 ppm in the 1970s. To examine the effect of this reduced limit on occupational risk, Sweetnam et al. (1987) conducted a follow-up study on the cohort first described by Tiller et al. (1968/Ex. 1-92). The health status and cause of death for 2,848 members of this cohort were ascertained up to the end of 1982. Exposure scores representing cumulative exposure to carbon disulfide were developed for each cohort member, based on an analysis of personal and area sampling results, job category, and time spent in each job category. Sweetnam et al. (1987) found the pattern of mortality similar to that found by Tiller, Schilling, and Morris (1968/Ex. 1-92): among spinner operators, who had the highest CS_2 exposures of any job category, 73 deaths from ischemic heart disease (IHD) were identified, compared with 42.5 expected deaths (SMR=172), a finding that was statistically significant. A statistically significant trend was found between cumulative exposure since first exposure and incidence of IHD mortality, which indicates a dose-related effect. A second (and perhaps most important) finding of this study was that recent (or current) exposure to carbon disulfide, as well as total cumulative exposure, were both risk factors for IHD. The authors established this association by examining the relationship between IHD mortality risk and each worker's total CS_2 exposure in the two years preceding death or the end of the study. The third result of this study was that workers with current CS_2 exposure also had significantly higher risk than workers who had ceased exposure. The dose-related relationship between increased IHD mortality risk and recent exposure to carbon disulfide suggested to the authors of this study that the effect of carbon disulfide on the cardiovascular system was direct and reversible.

Thus, the Sweetnam et al. (1987) follow-up determined that there is a relationship between the risk of IHD mortality and increased cumulative exposure to CS_2. Among workers who terminated exposure, this risk declined to non-statistically-significant levels after one year of no exposure. However, risk continued to be elevated among workers who continued to be exposed or who had not been exposure-free for a full year. OSHA interprets the findings of this important study to indicate that cumulative CS_2 dose from time of first exposure is a risk factor for IHD, and that this elevated risk continues unless exposure is terminated. That is, OSHA finds that workers who have been exposed to CS_2 in the past continue to be at increased risk as long as they are exposed to CS_2, even when their recent exposure is to lower levels (approximately 10 ppm, the current U.K. TLV).

This finding was confirmed by Nurminen and Hernberg (1985) in their follow-up study of 343 Finnish rayon workers who had been exposed to carbon disulfide for at least five years. Health status data were obtained for these workers for the period 1967 to 1982. In 1972, a preventive program had been instituted that included establishing a 10-ppm exposure limit and removing workers at high risk of coronary disease from continued exposure to carbon disulfide. Median exposure levels (largely from area samples) for the period 1975 to 1980 did not exceed 5 to 6 ppm, and third-quartile exposure levels did not exceed 10 ppm. These levels were about half those reported for the period 1967 to 1975.

Nurminen and Hernberg (1985) reported a 4.7-fold increase in IHD mortality incidence for the period 1967 to 1972, prior to the establishment of the protective measures described above. Five years after these measures were instituted, only 19 percent of the cohort continued to be exposed to carbon disulfide (compared to 53 percent of the cohort exposed in 1972). The relative risk for the first seven years of follow-up (1967 to 1974) was 3.2, compared to a relative risk of 1.0 for the last eight years (1974 to 1982). The excess risk of IHD mortality thus declined steadily throughout the follow-up period; this trend was statistically significant. The authors concluded that "* * * the cardiotoxic effects of CS_2 are reversible in the sense that the cessation of, or a radical decrease in, exposure reduces the risk of cardiovascular mortality to background levels" (Nurminen and Hernberg 1985, p. 34). Thus, the Nurminen and Hernberg (1985) study shows that reducing exposure levels below 10 ppm (combined, in their case, with a rigorous medical removal program to terminate exposure for employees who had developed signs or symptoms of coronary heart disease) can reduce the significant risk of IHD mortality to baseline levels.

In addition to NIOSH (Exs. 8-47 and 193), the AFL-CIO (Ex. 194) and Dr. James Melius, Director of the Division of Occupational Health and Environmental Epidemiology of the New York Department of Health (Ex. 152), supported OSHA's proposed 1-ppm PEL for carbon disulfide. However, several rulemaking participants criticized the studies relied on by OSHA, primarily on the grounds that the cohorts in which excess deaths from cardiovascular disease had been seen included workers who, these participants argued, were exposed for many years to levels of carbon disulfide much higher than the 10- to 40-ppm levels generally reported in these studies (Exs. 3-747, 3-1158, 8-19, 8-45, 31, 125, and 174; Tr. pp. 4-74 to 4-107). For example, Dr. Ernest Dixon, a toxicology consultant for the Inter-Industry Committee on Carbon Disulfide, testified as follows on these studies, which were also relied on by NIOSH to determine NIOSH's recommended standard:

> The NIOSH document presents a recitation of the toxic reviews, neurotoxic effects, and the various cardiovascular studies from chiefly Scandinavia, largely epidemiologic studies which attempted to determine whether or not ischemic or other cardiovascular abnormalities caused an excess of deaths among workers exposed to elevated levels of CS_2. Essentially, all of these were from the viscose manufacturing industry.
>
> Air sampling for carbon disulfide in the period prior to a decade ago was cumbersome, costly and took a long time for chemical analysis. As cited in numerous other reports, the practices of that period were to obtain area rather than personal samples. Work practices examined in the studies were such that the area sample results relied upon are believed to have significantly underestimated [both] the actual exposures and [the fact] that there were substantially higher exposures than have existed in more recent years.
>
> Accordingly, many of the workers in such studies had encountered many years of greatly higher exposure, especially for the earliest period of their exposure (Tr. p. 4-77).

In discussing the Tiller, Schilling, and Morris (1968/Ex. 1-92) study, Dr. Dixon emphasized that the coronary mortality risk of viscose production workers was not reported in this study to have been elevated, despite the fact that 17 percent of samples taken in production areas exceeded 20 ppm. However, there was a substantial excess in mortality from cardiovascular disease among spinners, where 50 percent of area samples exceeded 20 ppm (Tr. p. 4-80). In addition, Dr. Dixon pointed out that the populations studied by Vigliani (1954/Ex. 1-103) and by Seppalainen and Tolonen (1974/Ex. 1-100) were likely to have been exposed during high-viscose-production periods at the time of World

War II, when exposures were higher than in later periods.

As discussed above, OSHA believes that both cumulative exposure and current exposure are risk factors for IHD among CS_2-exposed workers; the Agency has also determined that excess risk continues for exposed workers as long as exposure continues. As to Dr. Dixon's point about area samples, OSHA does not agree that it is possible to infer that earlier area samples underestimate exposures. It is common industrial hygiene practice to measure problem areas in a facility to determine where additional control is needed. In addition, there is no way of determining, without actually taking both personal and area samples, whether the results of personal sampling would in fact be higher or lower than area samples taken in the same facility; whether breathing zone samples are higher or lower than area samples depends on a host of factors, including the positioning of the area sample in relation to the source of emissions, the location of the worker in relation to the same source, and the amount of time the worker spends in the vicinity of the emisssion source.

The Inter-Industry Committee on Carbon Disulfide submitted to the record a recent epidemiologic study by MacMahon and Monson (1988/Ex. 125). The study cohort consisted of 10,418 men employed between 1957 and 1979 in the four principal U.S. viscose rayon plants. The mortality status of the cohort was ascertained up to mid-1983. Cohort members were placed into general exposure categories according to job title; these categories were highest, intermediate, variable, least, and none. The authors found no significant increase in overall mortality in the 4,448 employees with the highest potential for CS_2 exposure compared with the mortality among 3,311 employees with no CS_2 exposure. However, there was a statistically significant excess of arteriosclerotic heart disease (ASHD) among the most heavily exposed workers (242 deaths versus 195.6 expected). No clear relationship was observed between exposure duration or latency and excess ASHD mortality; however, the data suggested that the risk was higher among employees exposed to CS_2 for 15 or more years and among employees hired prior to 1960.

In addition, MacMahon and Monson (1988/Ex. 125) found a statistically significant increase in the SMR (SMR=150) for ASHD among members of the cohort who had been exposed to CS_2 the year immediately preceding the date of death or the termination date of the study (Ex. 125, Attachment B, Table 7, p. 702); however, there was no general pattern of increased SMRs among cohort members whose time since last exposure exceeded one year. This finding is consistent with the results of the British studies, which also found an increased risk of heart disease among recently exposed employees but not among employees who had left their jobs.

The Inter-Industry Committee on Carbon Disulfide interpreted the MacMahon and Monson (1988/Ex. 125) study to mean that U.S. workers employed since 1960 were not at risk of ASHD (Tr. 4–96), and NIOSH (Ex. 193, Comments on Carbon Disulfide) noted that the study lacked exposure data. However, OSHA finds the results of the MacMahon and Monson (1988/ Ex. 125) study supportive and consistent with those of the British and Finnish studies discussed above. First, all of the studies clearly demonstrate a positive association between exposure to carbon disulfide and increased risk of mortality from heart disease. Second, studies from all three countries link the excess risk to cumulative CS_2 exposure. Third, the studies from all three countries demonstrate that significant risk can be substantially reduced or eliminated by reducing or stopping exposure, even after a considerable CS_2 dose has accumulated; both the U.S. and British studies report a significantly increased risk of death from heart disease among workers who were recently exposed. However, no increased risk was seen among workers whose exposures had ended one year or longer prior to death or the end of the study. Moreover, the Finnish study reported steady declines in heart disease mortality among workers after exposure levels were reduced to below 10 ppm and a rigorous medical screening and removal program was instituted. These findings clearly demonstrate that current or continued exposure to carbon disulfide at the levels presently encountered in these facilities is as important a risk factor for heart disease mortality as cumulative exposure.

In addition to evidence that carbon disulfide is a cardiovascular toxin, there is a substantial body of evidence that exposure to carbon disulfide presents a fetotoxic hazard and that this substance may also be a teratogen. Some of the early (pre-1977) animal data on reproductive effects were reviewed in the NIOSH (1977b/Ex. 1–260) criteria document on carbon disulfide. In its posthearing submission, NIOSH (Ex. 193) mentions two relevant reports. One by Cai and Bao (1981, as cited in Ex. 193) reported increased incidences of menstrual disturbances and of pregnancy toxemia, a potentially lethal condition, in rayon workers. These authors also presented evidence that CS_2 can cross the placental barrier and be secreted into mothers' milk. The second report cited by NIOSH (Hemminki and Niemi 1982, as cited in Ex. 193) found a significantly elevated incidence of spontaneous abortions among women employed in viscose rayon facilities in Finland; however, data on the specific CS_2 exposure levels were generally lacking.

The Rohm and Haas Company submitted a summary (Ex. 10–5) of the evidence on the reproductive toxicity of carbon disulfide to the OSHA docket; this information shows that carbon disulfide has caused fetal deaths and malformations in CS_2-exposed laboratory animals. Rohm and Haas cite a series of abstracts by Tabacova and others in which oral administration of CS_2 to female rats during gestation produced both teratogenic and fetotoxic effects. These effects were magnified in the F_2 offspring of the prenatally exposed F_1 generation, which suggests that CS_2 has a multigenerational effect that continues to cause malformations in successive generations.

Jones-Price et al. (1984, NTIS/PB84–192343) found both maternal and fetal toxicity in CD rats exposed orally to 200, 400, or 600 mg/kg/d CS_2 during days 6 through 15 of gestation. No dose-related increases in the incidence of teratogenicity were observed. In another report, Jones-Price et al. (1984, NTIS/PB84–192350) found significant dose-related increases in percent resorptions/litter, percent non-live (dead or resorbed)/litter, and percent of fetuses affected (non-live and malformed)/litter among New Zealand White rabbits exposed orally to 25, 75, or 150 mg/kg/d during days 6 through 19 of gestation. The percentage of malformed fetuses per litter increased with dose and was statistically significant at the highest dose tested.

In an inhalation study, Hardin, Bond, Sikov et al. (1981/Ex. 1–699) exposed rats and rabbits to 20 or 40 ppm CS_2 for 6.5 hours per day during days 1 through 19 (rats) or 1 through 24 (rabbits) of gestation. No embryotoxic or fetotoxic effects were noted, indicating that 40 ppm is a no-effect level for these effects in rats and rabbits. According to the analysis by Rohm and Haas, the lowest-reported-effect level (25 mg/kg/d) documented by Jones-Price et al. (1984) for rabbits corresponds to an equivalent airborne exposure of 58 ppm; this lowest-reported-effect level is in close agreement with the no-effect level

reported by Hardin et al. (1981/Ex. 1-699) for the same species.

OSHA believes that this evidence, which shows that consistent fetotoxic and teratogenic effects are associated with exposure to carbon disulfide, warrants considerable concern. OSHA is particularly alarmed at the multigenerational effect of CS_2 exposure that has been demonstrated to occur in rats. This risk of reproductive effects, combined with the previously recognized risk of cardiovascular disease, have convinced OSHA that a substantial reduction in the PEL for carbon disulfide is clearly justified.

Several foreign governments and standards-setting organizations have already established 8-hour TWA exposure limits for carbon disulfide that range from 1 to 10 ppm. For example, NIOSH has recommended a 1-ppm TWA limit for this substance, and Rohm and Haas established an internal limit of 4 ppm as an 8-hour TWA (Ex. 10-5). Several foreign countries, including West Germany, Italy, Japan, Sweden, and Switzerland, currently have 10-ppm limits. The ACGIH has established a 10-ppm TLV for CS_2; however, the ACGIH limit does not consider any of the evidence of CS_2's fetotoxic or teratogenic effects.

Based on the evidence in the record and the toxicological literature, OSHA concludes that 4 ppm is a reasonable and prudent level at which to establish a revised 8-hour TWA PEL for carbon disulfide. This limit should provide for a substantial reduction in the significant risk both of cardiovascular disease and adverse reproductive effects associated with CS_2 exposures; clearly, these effects constitute material impairments of health and functional capacity. In addition, because of the seriousness of the effects associated with exposure to carbon disulfide, and in accordance with the policy described in Section VI.C.17 on short-term exposure limits, OSHA finds that a STEL is necessary to ensure that the 8-hour TWA limit is not exceeded during operations characterized by intermittent exposures to elevated levels of CS_2. Rohm and Haas (Ex. 10-5) has established an internal guideline of 12 ppm as a short-term limit to ensure that the 8-hour TWA limit is not exceeded, and NIOSH also recommends a short-term limit to ensure that full-shift exposures are maintained under good control. In the final rule, OSHA is accordingly establishing a 12-ppm STEL to supplement the 4-ppm TWA PEL.

OSHA's assessment of the feasibility of this limit indicates that, under normal operating conditions, a 4-ppm TWA PEL and a 12-ppm STEL are generally achievable by using engineering and work practice controls; respiratory protection may be required during certain operations in rayon and sausage-casing production, such as maintenance tasks or opening of the production lines (see Section VII). Specific operations for which OSHA will accept the use of respirators include the following:

- Opening windows and hoods to change spinerettes (in SIC 28);
- Opening windows and hoods to remove filament bundles (in SIC 28);
- Effecting product-line changes (in SIC 28);
- Unloading xanthate from the baratte (in SIC 30);
- Aligning strands in the extrusion cabinet (in SIC 30); and
- Manually puncturing casings at the extrusion nozzles in the cellulosic food casing industry (in SIC 30).

Thus, OSHA finds that the TWA and STEL limits being established in the final rule are feasible.

CHLOROPENTAFLUOROETHANE

CAS: 76-15-3; Chemical Formula: $ClCF_2CF_3$
H.S. NO. 1087

OSHA previously had no limit for chloropentafluoroethane (FC-115). The proposed PEL for this substance was 1000 ppm as an 8-hour TWA, and NIOSH (Ex. 8-47, Table N1) supported the proposal. The final rule establishes this limit. The ACGIH has a TLV-TWA of 1000 ppm for this colorless, odorless gas.

Chloropentafluoroethane is an asphyxiant at high concentrations. In dogs and rats, gastrointestinal absorption following intragastric intubation has been shown to be minimal (Terrill 1974/Ex. 1-1070; Clayton, Hood, Nick, and Waritz 1966/Ex. 1-952). Rats exposed to 800,000 ppm FC-115 with 20 percent oxygen for four hours showed no clinical or histopathologic effects (Clayton, Hood, Nick, and Waritz 1966/Ex. 1-952). Rats and guinea pigs showed no adverse clinical effects at inhalation levels of 600,000 ppm FC-115 in oxygen for two hours (Weigand 1971/Ex. 1-1102), and guinea pigs exposed to 200,000 ppm FC-115 in air for varying intervals up to two hours also exhibited no adverse signs (Breen and Wallis 1963, as cited in ACGIH 1986/Ex. 1-3, p. 133). Rats, mice, rabbits, and dogs have tolerated six-hour daily exposures of 100,000 ppm FC-115 for 90 days without adverse effects (Clayton, Hood, Nick, and Waritz 1966/Ex. 1-952), and laboratory animals have tolerated doses of 200,000 ppm for 3.5 hours daily, five days per week, for four weeks (Weigand 1971/Ex. 1-1102). FC-115's potential for cardiac sensitization caused one of 13 unanesthetized dogs to develop cardiac sensitization after exposure to 150,000 ppm intravenously (Trochimowicz, Azar, Terrill, and Mullin 1974/Ex. 1-448). Several other studies indicate that unanesthetized dogs, rats, and monkeys receiving dosages of between 100,000 ppm and 200,000 ppm may show increased blood pressure, accelerated heart rate, myocardial depression, or altered pulmonary effects under certain conditions (Belej and Aviado 1975/Ex. 1-462; Friedman, Cammarato, and Aviado 1973/Ex. 1-416; Aviado and Belej 1975/Ex. 1-616). There were no reports of mutagenic, teratogenic, or carcinogenic toxicities in these studies. The Agency received no comments addressing chloropentafluoroethane, other than those submitted by NIOSH.

OSHA is establishing an 8-hour TWA permissible exposure limit of 1000 ppm for chloropentafluoroethane. The Agency concludes that this limit will protect workers from the significant risk of cardiac effects, which constitutes material impairment of health and functional capacity, at the high levels formerly permitted by the absence of an OSHA limit.

ETHYLENE GLYCOL DINITRATE

CAS: 628-96-6; Chemical Formula: $CH_2NO_3CH_2NO_3$
H.S. No. 1170

NITROGLYCERIN

CAS: 55-63-0; Chemical Formula: $CH_2NO_3CHNO_3CH_2NO_3$
H.S. No. 1290

The former OSHA PELs for ethylene glycol dinitrate (EGDN) and nitroglycerin (NG) were ceilings of 1 mg/m^3 with skin notations. The proposed PELs for these substances were 20-minute STELs of 0.1 mg/m^3, and NIOSH (Ex. 8-47, Table N1) supported the proposal (which was based on NIOSH's recommended limits). The ACGIH (1986/Ex. 1-3) has established a TLV-TWA of 0.05 ppm (0.3 mg/m^3) for EGDN and a TLV-TWA of 0.05 ppm (0.5 mg/m^3) for NG, both with skin notations. In the final rule, OSHA is establishing 15-minute STELs of 0.1 mg/m^3 for EGDN and NG and retaining the skin notations for these substances. EGDN is a yellowish, oily, explosive liquid, and NG is a pale yellow, viscous liquid.

Most occupational exposures to EGDN actually involve mixtures of EGDN and NG. Because EGDN is 160 times more volatile than nitroglycerin and most of the mixtures of these two substances used in industry consist of 60 to 80 percent EGDN, the adverse effects associated with the inhalation of the vapors from such mixtures can be attributed primarily to EGDN.

Trainor and Jones (1966/Ex. 1–107) reported that exposure to EGDN:NG at a level of 0.7 mg/m³ for 25 minutes was sufficient to produce decreased blood pressure and a slight headache in humans. These authors also reported that workers at a munitions plant developed headaches when exposed to EGDN:NG concentrations between 0.1 and 0.53 mg/m³ (0.36 mg/m³ average). Morikawa, Muraki, Ikoma et al. (1967/Ex. 1–55) found that workers in an explosives plant exposed to low concentrations of EGDN:NG (0.066 ppm (approximately 0.5 mg/m³) was the highest average level) had a much higher incidence of abnormal pulse waves than did controls (143 out of 1,271 versus 0 out of 175). Abnormal pulse waves often indicate a clinically significant defect in the functioning of the heart and/or circulatory system (Braunwald 1978/Ex. 1–1001).

In its criteria document for NG and EGDN, NIOSH (1978h/Ex. 1–234) refers to a report of a dynamite worker who died when exposed to EGDN:NG concentrations between 0.3 and 1.4 mg/m³, as well as to another report of two workers who died suddenly following exposure to EGDN:NG at concentrations ranging from 1.7 to 2.7 mg/m³. NIOSH (1978h/Ex. 1–234) observed that skin absorption may have contributed significantly to the exposures causing these deaths.

OSHA received several comments on EGDN and NG (Exs. 3–661, 8–66, 121, 190, and 154). These commenters raised two issues: the technological and economic feasibility of the proposed limits, and the strength of the evidence and significance of the adverse effects associated with exposure to EGDN and NG.

In regard to the issue of technological and economic feasibility, which was raised by ICI Americas, Inc. (Ex. 154) and the Institute of Makers of Explosives (IME) (Ex. 121), OSHA has reviewed the record and has concluded that explosive manufacturers will be able to meet these limits through a combination of equipment improvements and respiratory protection. OSHA believes that, if compliance cannot be achieved via engineering controls and/or process improvements, air-supplied respirators with quick-release couplings could be used to achieve the final rule's limits. The Agency's reasoning is discussed in detail in the Technological Feasibility section of the preamble (Section VII).

On the second point addressed by commenters, the meaning of the health effects observed to occur in connection with exposure to EGDN:NG, the IME states that:

> [T]he NIOSH Criteria Document relied upon in the . . . [proposal] was based on outdated and irrelevant information. Its findings are based on exposure conditions and data that, because of industry-initiated improvements, was not reflective of the improved conditions in NG/EGDN-manufacturing plants . . . in 1978, and is not reflective of the greatly improved conditions prevailing in plants at the present time . . . industry hygiene programs . . . [have] eliminated the bulk of . . . workplace exposure[s] (Ex. 190, p. 4).

According to the IME, OSHA's proposal did not "identify any significant health risk" of EGDN:NG exposure at the former PEL; the IME asserts that "headaches are transitory phenomena which pose no significant health risk" (Ex. 190, p. 5).

OSHA does not share the IME's view of the significance of chemically induced headaches. The Agency believes that such headaches impair performance, cause pain and suffering, affect the safety of the victim and his or her co-workers, and contribute to absenteeism. In the case of EGDN:NG-induced headaches, however, headaches have a greater meaning: they are an early warning of vasodilation, an indicator of systemic toxicity. OSHA also finds the report of an EGDN:NG-induced death in an explosives manufacturing facility both convincing and troubling. The Agency continues to be persuaded by the evidence in the Trainor and Jones (1966/Ex. 1–107) study, the NIOSH criteria document (1978h/Ex. 1–234), and the Morikawa, Muraki, Ikoma et al. (1967/Ex. 1–55) study that the health effects associated with exposure to very low levels of EGDN:NG (i.e., in the range of 0.1 to 1.4 mg/m³) are acute, may occur after brief exposures, and have been shown to be lethal.

According to NIOSH (Ex. 150, Comments on EGDN:NG), the 15-minute 0.1-mg/m³ limits being established in the final rule will protect against "angina pectoris, other signs and symptoms of cardiac ischemia or heart damage, and against sudden death . . . since all of these . . . seem to be related to compensatory vasoconstriction induced by repeated exposure to NG or EGDN" (Ex. 150). NIOSH also reports that a preliminary study of mortality resulting from heart disease and other causes among NG workers by Reeve, Bloom, Rinsky, and Smith (1983a and 1983b, as cited in Ex. 150) suggests an association between NG exposure and cardiovascular disease mortality; at the facilities where this increase in cardiovascular disease occurred, exposures were being maintained near or below 0.02 ppm (0.2 mg/m³) (Ex. 150).

Hypotension and headache have been observed in populations exposed to EGDN:NG at levels below 0.5 mg/m³ for brief periods (25 minutes), and fatalities have occurred after EGDN:NG exposures at concentrations between 0.3 and 1.4 mg/m³, in one instance, and between 1.7 and 2.7 mg/m³, in another. OSHA's former standard was 1.0 mg/m³; since worker deaths have occurred at or near this level, OSHA is establishing short-term limits for EGDN and NG of 0.1 mg/m³ and retaining the skin notations for these substances in the final rule. OSHA concludes that these limits are necessary to prevent fatalities and to protect against the significant risks of vasodilation and cardiac effects associated with exposures to EGDN:NG in the workplace. The Agency has determined that the cardiovascular effects caused by EGDN:NG represent material impairments of health. Because EGDN:NG is readily absorbed through the skin and can produce systemic effects by this exposure route (Tr. pp. 9–149 to 9–150), OSHA is retaining the skin notations for both substances.

FLUOROTRICHLOROMETHANE (TRICHLOROFLUOROMETHANE)

CAS: 75–69–4; Chemical Formula: CCl_3F
H.S. No. 1180

Fluorotrichloromethane (trichlorofluoromethane), also known as FC–11, is a member of a large family of chemicals, the chlorofluorocarbons. The former OSHA PEL was an 8-hour TWA of 1000 ppm. The proposed PEL was a ceiling of 1000 ppm and NIOSH (Ex. 8–47, Table N1) supported the proposal. The final rule establishes this limit. At ordinary temperatures, FC–11 is a noncombustible, colorless liquid or gas.

Inhalation of large doses of FC–11 has caused cardiac sensitization and death in humans. Experimental mice that inhaled aerosol containing 10 percent FC–11 exhibited cardiac arrhythmias. In the same study, dogs that inhaled aerosol containing 2.5 percent FC–11 had decreased myocardial function; monkeys that inhaled an aerosol containing 5 percent FC–11 developed tachycardia and hypotension (*Drinking Water and Health*, National Research Council 1977).

Exposure to 5000 ppm FC–11 has induced cardiac sensitization and arrhythmia in dogs that were intravenously injected with epinephrine (Reinhardt, Azar, Maxfield, Smith, and Mullin 1971/Ex. 1–78). Jenkins, Jones, Coon, and Siegel (1970/Ex. 1–95) found that four species of animals (monkeys, dogs, rats, and guinea pigs) suffered no ill effects after 90 days of continuous exposure to 1000 ppm of FC–11. Other

than those submitted by NIOSH, OSHA received no comments on FC-11.

The cardiac sensitization exhibited by FC-11-exposed animals is an acute effect. OSHA's former 1000-ppm TWA PEL would permit workers to be exposed to short-term concentrations of FC-11 that are sufficiently high to sensitize the heart to sympathomimetic amines; OSHA considers this effect to be a material impairment of health. Accordingly, OSHA concludes that, at the former limit, workers are at significant risk of experiencing arrhythmia. Revising this limit to a 1000-ppm ceiling limit will substantially reduce this significant risk of cardiac sensitization.

SODIUM AZIDE

CAS: 26628-22-8; Chemical Formula: NaN_3
H.S. No. 1364

There was no former OSHA PEL for sodium azide. The proposed PELs were a ceiling of 0.1 ppm as hydrazoic acid vapor (HN_3) and a ceiling of 0.3 mg/m^3 as sodium azide (NaN_3); NIOSH (Ex. 8-47, Table N1) concurred with the Agency's selection. The final rule establishes this limit. In addition, a skin notation is being added to the limit in the final rule. The ACGIH (1986/Ex. 1-3) has ceiling limits for sodium azide of 0.1 ppm (as hydrazoic acid vapor) and 0.3 mg/m^3 (as NaN_3). Sodium azide is a colorless, crystalline solid.

Sodium azide is known to produce hypotension in laboratory animals and humans. An intravenous dose of 1 mg/kg was reported to lower blood pressure in cats (Graham 1949/Ex. 1-109). In the 1950s, the medicinal usefulness of sodium azide as a hypotensive agent was tested in 30 hypertensive patients. Their hypertension was reduced, but observed side effects included headaches; in addition, 20 of 30 patients developed increased sensitivity to sodium azide, necessitating a reduction in the dose (Black, Zweifach, and Speer 1954/Ex. 1-163). Hicks (1950, cited in ACGIH 1986/Ex. 1-3, p. 533) reported that repeated intraperitoneal injections of 5 to 10 mg/kg in rats caused demyelination of nerve fibers of the CNS. Alben and Fager (1972, cited in ACGIH 1986/Ex. 1-3, p. 533) showed that sodium azide formed strong complexes with hemoglobin and blocked oxygen transport in the blood.

Acute inhalation by humans of hydrazoic acid vapor (which forms when sodium azide contacts water) results in lowered blood pressure, eye irritation, bronchitis, headache, weakness, and collapse (Fairhall et al. 1943/Ex. 1-130; Graham 1949/Ex. 1-109). The exposure levels that produce these effects were not reported by these authors. Haas and Marsh (1970/Ex. 1-121) reported that exposure to concentrations of hydrazoic acid vapor as low as 0.5 ppm "cause[d] some discomfort to laboratory personnel." Dr. Hecker of Abbott Laboratories (Ex. 3-678) commented that the limit for sodium azide should include a skin notation, and Sax and Lewis (*Dangerous Properties of Industrial Materials*, 7th ed., 1989) report the dermal LD_{50} in rabbits to be 20 mg/kg, demonstrating that sodium azide readily penetrates the skin and causes systemic poisoning. Grace Ziem, an independent occupational physician, also supported a skin notation for sodium azide (Ex. 46). In the final rule, OSHA is therefore adding a skin notation for sodium azide.

Because of its hypotensive effect in humans, OSHA concludes that ceiling limits of 0.1 ppm (as HN_3) and 0.3 mg/m^3 (as NaN_3) should be established for sodium azide to reduce the significant risk of cardiovascular and irritation effects posed to workers at the levels formerly permitted by the absence of an OSHA limit. The Agency considers the effects associated with exposure to sodium azide as material impairments of health. To reduce this significant risk substantially, OSHA is establishing these ceiling limits for sodium azide in the final rule. In addition, OSHA is adding a skin notation to the PEL to alert employers to the fact that sodium azide readily penetrates intact skin and that dermal exposure can contribute significantly to overall worker exposure.

1,1,2-TRICHLORO-1,2,2-TRIFLUOROETHANE

CAS: 76-13-1; Chemical Formula: CCl_2FCClF_2
H.S. No. 1403

1,1,2-Trichloro-1,2,2-trifluoroethane (FC-113) is a member of the chlorofluorocarbon family. The former OSHA PEL was an 8-hour TWA of 1000 ppm. The Agency proposed to retain this limit and to add a STEL, and NIOSH (Ex. 8-47, Table N1) concurred that these limits are appropriate. The final rule retains the 8-hour TWA of 1000 ppm and supplements it with a 1250-ppm STEL. The ACGIH has an 8-hour TLV-TWA of 1000 ppm and a 15-minute STEL of 1250 ppm for FC-113. FC-113 is a colorless, noncombustible liquid.

Cardiac sensitization following the administration of epinephrine is the most significant effect observed after exposure to FC-113. Reinhardt, Mullin, and Maxfield (1973/Ex. 1-114) observed that 10 out of 29 dogs exposed to 5000 ppm FC-113 for 5 minutes and simultaneously injected with epinephrine developed serious arrhythmias. Similar experiments, in which the dogs were exposed to 2000 to 2500 ppm of this substance for longer periods of time (from 30 minutes to 6 hours) and simultaneously administered epinephrine, resulted occasionally in arrhythmia (Aviado 1975, as cited in ACGIH 1986/Ex. 1-3, p. 603). However, when the experiment was repeated using four 6-hour exposures to 1000 ppm in conjunction with an injection of epinephrine, no arrhythmias were observed.

A study by Stopps and McLaughlin (1967/Ex. 1-122) of human volunteers revealed that exposure to 2500 ppm FC-113 for 1.5 hours resulted in impairment of psychomotor performance (described as lethargy and inability to concentrate). This effect was not observed at concentrations below 2500 ppm. Within the first one-half to one hour of exposure to 2500 ppm or more, subjects reported subjective sensations including loss of concentration, a tendency to somnolence, and a feeling of "heaviness" in the head. Dr. Lawrence Hecker of Abbott Laboratories (Ex. 3-678) commented that there was no basis for a STEL for FC-113. OSHA does not agree with Dr. Hecker's assessment because the results of the Stopps and McLaughlin (1967/Ex. 1-122) study described above demonstrate that FC-113 can induce subjective effects in humans on short-term exposure. Thus, OSHA finds that a STEL is necessary to prevent these effects. The UAW (Tr. pp. 7-67 to 7-69) and the AFL-CIO (Ex. 194) supported short-term or ceiling limits for FC-113 lower than the proposed STEL.

The evidence described above demonstrates that FC-113 can exert toxic effects at levels of exposure comparable to the levels that were formerly permitted by excursions above the former OSHA TWA limit of 1000 ppm; such levels thus pose a significant risk of cardiac sensitization to exposed workers. The Agency considers cardiac sensitization induced by FC-113 as material impairment of health and functional capacity. OSHA concludes that a STEL of 1250 ppm will provide a wider margin of safety against cardiac sensitization and will reduce the risk of impaired psychomotor performance by limiting the potentially high, short-term exposures formerly permitted by the 8-hour TWA limit alone. The final rule establishes limits of 1000 ppm TWA and 1250 ppm STEL for 1,1,2-trichloro-1,2,2-trifluoroethane to substantially reduce the significant risks associated with exposure to this substance.

Conclusions

Of all the physiological systems, the cardiovascular system is especially

vulnerable to occupational hazards because cardiovascular diseases are already so prevalent in our society. According to Levy (1985/Ex. 1–210), "an estimated 40 million Americans have some form of cardiovascular disease." The major risk factors, as revealed by epidemiology, are age, male sex, hypertension, cigarette smoking, the existence of low-density and high-density plasma lipoproteins, cholesterol, and diabetes (Levy 1985/Ex. 1–210). Many American workers exposed to the chemicals grouped on the basis of their cardiovascular effects have one or more of these risk factors and are therefore particularly susceptible to exposure to cardiovascular toxicants. Although the precise interactions among these risk factors and exposures to cardiovascular toxins are difficult to demonstrate with accuracy, few would argue that they do not occur.

OSHA concludes that the potential for cardiovascular system damage associated with exposure to these cardiac sensitizers, vasodilators, and atherosclerosis-causing substances poses a significant risk to employees in a broad range of workplaces. The effects experienced by exposed workers include arrhythmia, low blood pressure, stroke, and blockage of the flow of blood to the myocardium. OSHA has demonstrated that these effects clearly constitute material impairment of health and functional capacity. Revising or establishing exposure limits for these cardiovascular toxins will substantially reduce these significant risks.

8. Substances for Which Limits Are Based on Avoidance of Systemic Toxicity

Introduction

For a number of substances, OSHA's revised limits are based primarily on evidence that exposure is associated with general systemic toxicity. This group of substances is unique among the groupings discussed in this preamble in that no single organ system can be identified as the target of low-dose exposure to these chemicals. Instead, these substances have been shown either to affect several organ systems simultaneously or to cause a variety of nonspecific adverse signs and symptoms that are indicative of general toxicity.

The 34 substances belonging to this group and their CAS numbers, HS numbers, and former, proposed, and final rule PELs are shown in Table C8–1. OSHA is establishing exposure limits for 17 substances in this group that were not formerly regulated and retaining the former PELs for eight substances to which STELs are being added. For six substances, OSHA is lowering its former 8-hour TWA PELs. For two substances that formerly had 8-hour TWA PELs, OSHA is deleting the full shift limit and replacing it with a short-term limit or a ceiling. For one substance, OSHA is establishing an 8-hour TWA in place of a former ceiling limit.

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TABLE C8-1. Substances for Which Limits Are Based on Avoidance of Systemic Toxicity

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1005 Acetonitrile	75-05-8	40 ppm TWA	20 ppm TWA, Skin	40 ppm TWA 60 ppm STEL
1006 Acetylsalicylic acid (Aspirin)	50-78-2	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1019 Aluminum (Welding fumes)	7429-90-5	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1046 2-Butoxyethanol	111-76-2	50 ppm TWA, Skin	25 ppm TWA, Skin	25 ppm TWA, Skin
1052 n-Butyl glycidyl ether	2426-08-6	50 ppm TWA	25 ppm TWA	25 ppm TWA
1067 Captan	133-06-2	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1088 beta-Chloroprene	126-99-8	25 ppm TWA, Skin	10 ppm TWA, Skin	10 ppm TWA, Skin
1109 Cyclohexylamine	108-91-8	--	10 ppm TWA	10 ppm TWA
1112 Cyhexatin	13121-70-5	--	5 mg/m^3 TWA	5 mg/m^3 TWA

TABLE C8-1. Substances for Which Limits Are Based on Avoidance of Systemic Toxicity (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1113 Dichlorodiphenyl-trichloroethane (DDT)	50-29-3	1 mg/m^3 TWA, Skin	1 mg/m^3 TWA, Skin	1 mg/m^3 TWA, Skin
1120 2-N-Dibutylamino-ethanol	102-81-8	--	2 ppm TWA, Skin	2 ppm TWA
1139 Diglycidyl ether	2238-07-5	0.5 ppm Ceiling	0.1 ppm TWA	0.1 ppm TWA
1159 Ethanolamine	141-43-5	3 ppm TWA	3 ppm TWA 6 ppm STEL	3 ppm TWA 6 ppm STEL
1167 Ethylene chlorohydrin	107-07-3	5 ppm TWA, Skin	1 ppm Ceiling, Skin	1 ppm Ceiling, Skin
1189 Glycidol	556-52-5	50 ppm TWA	25 ppm TWA	25 ppm TWA
1198 Hexafluoroacetone	684-16-2	--	0.1 ppm TWA, Skin	0.1 ppm TWA, Skin
1207 Hydrogen cyanide	74-90-8	10 ppm TWA, Skin	4.7 ppm Ceiling (10 min)	4.7 ppm STEL, Skin
1210 Hydrogenated terphenyls	61788-32-7	--	0.5 ppm TWA	0.5 ppm TWA

TABLE C8-1. Substances for Which Limits Are Based on Avoidance of Systemic Toxicity (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1223 2-Isopropoxyethanol	109-59-1	--	25 ppm TWA	25 ppm TWA
1227 Isopropyl glycidyl ether	4016-14-2	50 ppm TWA	50 ppm TWA 75 ppm STEL	50 ppm TWA 75 ppm STEL
1273 4,4'-Methylene bis (2-chloroaniline)	101-14-4	--	0.02 ppm TWA, Skin	0.02 ppm TWA, Skin
1317 Phenylhydrazine	100-63-0	5 ppm TWA, Skin	5 ppm TWA 10 ppm STEL, Skin	5 ppm TWA 10 ppm STEL, Skin
1318 Phenylphosphine	638-21-1	--	0.05 ppm Ceiling	0.05 ppm Ceiling
1321 Phosphine	7803-51-2	0.3 ppm TWA	0.3 ppm TWA 1 ppm STEL	0.3 ppm TWA 1 ppm STEL
1330 Piperazine dihydrochloride	142-64-3	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1340 n-Propyl nitrate	627-13-4	25 ppm TWA	25 ppm TWA 40 ppm STEL	25 ppm TWA 40 ppm STEL
1366 Sodium fluoroacetate	62-74-8	0.05 mg/m^3 TWA, Skin	0.05 mg/m^3 TWA 0.15 mg/m^3 STEL, Skin	0.05 mg/m^3 TWA 0.15 mg/m^3 STEL, Skin

TABLE C8-1. Substances for Which Limits Are Based on Avoidance of Systemic Toxicity (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1412 Trimethyl benzene	25551-13-7	--	25 ppm TWA	25 ppm TWA
1416 Tungsten Compounds (insoluble)	7440-33-7	--	5 mg/m^3 TWA 10 mg/m^3 STEL	5 mg/m^3 TWA 10 mg/m^3 STEL
1417 Tungsten Compounds (soluble)	7440-33-7	--	1 mg/m^3 TWA 3 mg/m^3 STEL	1 mg/m^3 TWA 3 mg/m^3 STEL
1428 Vinylidene chloride	75-35-4	--	5 ppm TWA 20 ppm STEL	1 ppm TWA
1430 Welding fumes (Total particulate)	--	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1437 Zinc oxide (Fume)	1314-13-2	5 mg/m^3 TWA	5 mg/m^3 TWA 10 mg/m^3 STEL	5 mg/m^3 TWA 10 mg/m^3 STEL
1439 Zirconium compounds	7440-67-7	5 mg/m^3 TWA	5 mg/m^3 TWA 10 mg/m^3 STEL	5 mg/m^3 TWA 10 mg/m^3 STEL

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

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Description of the Health Effects

For each substance included in this grouping, limits have been established to protect against a variety of adverse exposure-related effects that are manifested at multiple target organ sites. In some instances, the nature of the toxic effects associated with exposure is well-defined and clearly understood (for example, CNS depression, histological organ changes, embryo-toxicity, methemoglobinemia, conjunctivitis, liver and kidney damage, testicular damage). The effects of exposure to other substances in this group, however, have been demonstrated only by such nonspecific indicators as dizziness, respiratory irritation, hematuria, chest tightness, weight loss or decreased rate of weight gain, lethargy, loss of appetite, nervousness, or gastrointestinal disturbances. Although the specificity of the systemic effect caused by exposure to the substances in this group may vary, all of these substances have been shown to be biologically active in mammalian species, to interfere significantly with biological processes, and to impair normal organ function.

Table C8–2 summarizes the toxic effects reported in humans and experimental animals that support the establishment of limits for these substances. This table shows the variety of adverse health effects that adoption of the final rule's limits will minimize or prevent. The table also shows that, for the vast majority of substances in this group, the risks of exposure have been defined in studies of humans or animals and are known to include respiratory effects, neurological effects, adverse effects on the reproductive system, organ damage, hematopoietic effects, sensitization, and mucosal irritation. All of these effects are indicative of generalized systemic effects rather than localized effects occurring at the site of chemical contact.

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TABLE C8-2. Summary of Adverse Health Effects Reported for Substances Producing General Systemic Toxicity

H.S. Number/ Chemical Name	Effects Reported in Humans	Effects Reported in Animals
1005 Acetonitrile	Tightness in chest; flushing of face	Embryotoxicity; teratogenicity at maternally toxic doses; liver, blood count changes
1006 Acetylsalicylic acid	Mucosal irritation; respiratory sensitization; internal bleeding	Teratogenicity at high doses
1019 Aluminum welding fumes	No data	Respiratory effects
1046 2-Butoxyethanol	Mild sensory irritation	Severe hemoglobinuria; lung, kidney, liver changes; hemolytic anemia; increased osmotic fragility in erythrocytes
1052 n-Butyl glycidyl ether	Dermatitis; skin sensitization	Delirium; depression
1067 Captan	Recurrent urticaria	Decreased fertility index in males; polyploid carcinoma of duodenum

TABLE C8-2. Summary of Adverse Health Effects Reported for Substances Producing General Systemic Toxicity (continued)

H.S. Number/ Chemical Name	Effects Reported in Humans	Effects Reported in Animals
1088 Chloroprene	CNS depression; lung, liver, kidney injury; conjunctivitis; necrosis of cornea; lowering of blood pressure	Minimal systemic effects
1109 Cyclohexylamine	Acute toxicity; sensitization	Mutagenic and reproductive effects
1112 Cyhexatin	No data	Microscopic changes in liver, kidneys, adrenal glands
1113 Dichlorodiphenyl-trichloroethane (DDT)	Mild poisoning	Cancer of liver, lungs, lymph system
1120 2-N-Dibutylamino-ethanol	No data	Weight loss; elevated liver- and kidney-to-body-weight ratios
1139 Diglycidyl ether	Mucosal irritation	CNS depression, clouding of cornea; respiratory irritation; hematopoietic effects

TABLE C8-2. Summary of Adverse Health Effects Reported for Substances Producing General Systemic Toxicity (continued)

H.S. Number/ Chemical Name	Effects Reported in Humans	Effects Reported in Animals
1159 Ethanolamine	No data	Pulmonary, hepatic, and renal lesions; decreased alertness; temporary weight loss
1167 Ethylene chlorohydrin	Liver and brain damage; mucosal irritation; gastrointestinal disturbances	Respiratory depression; liver and kidney damage
1189 Glycidol	No data	Pneumonitis; emphysema
1198 Hexafluoroacetone	No data	Renal dysfunction; increased lung weight; testicular damage; hematopoietic effects; fetotoxicity
1210 Hydrogenated terphenyls	No data	Decreased weight gain; liver, kidney damage; lung changes, bronchopneumonia
1207 Hydrogen cyanide	Cyanide poisoning; weakness; mucosal irritation; colic; nervousness; enlargement of thyroid	None reported

TABLE C8-2. Summary of Adverse Health Effects Reported for Substances Producing General Systemic Toxicity (continued)

H.S. Number/ Chemical Name	Effects Reported in Humans	Effects Reported in Animals
1223 2-Isopropoxyethanol	No data	Anemia; hemoglobinuria; lung congestion
1227 Isopropyl glycidyl ether	Mucosal irritation	Reduced weight gain; hemoglobin increase; emphysematous changes in lungs; CNS depression
1273 4,4'-methylene-bis (2-chloroaniline)	Bladder cancer; hematuria	Cyanosis; methemoglobinemia; liver, lung tumors
1317 Phenylhydrazine	Skin sensitization	Anemia; irregular growth; general weakness; blood vessel tumors
1318 Phenylphosphine	No data	Mild hemolytic anemia; testicular degeneration; hind leg tremor; nausea; loss of appetite; hypersensitivity to sound and touch

TABLE C8-2. Summary of Adverse Health Effects Reported for Substances Producing General Systemic Toxicity (continued)

H.S. Number/ Chemical Name	Effects Reported in Humans	Effects Reported in Animals
1321 Phosphine	Pulmonary edema; gastrointestinal disturbances; dizziness	Respiratory irritation
1330 Piperazine dihydrochloride	Skin burns; sensitization; asthma	No data
1340 n-Propyl nitrate	No data	Cyanosis; methemoglobinemia; hypotension; respiratory depression
1366 Sodium fluoroacetate	No data	Fluctuation in growth rate; tissue changes
1412 Trimethylbenzene	Nervousness, tension, anxiety; asthmatic bronchitis; hypochromic anemia	CNS depression Lymphopenia; neutrophilia
1416 Tungsten compounds (insoluble)	No data	Gross changes in liver and spleen; lung tissue changes

TABLE C8-2. Summary of Adverse Health Effects Reported for Substances Producing General Systemic Toxicity (continued)

H.S. Number/ Chemical Name	Effects Reported in Humans	Effects Reported in Animals
1417 Tungsten compounds (soluble)	No data	Generalized cellular asphyxiation; colic; incoordination; dyspnea
1428 Vinylidene chloride	No data	Nasal irritation; liver cell degeneration; retarded weight gain; embryotoxicity; kidney adenocarcinoma
1430 Welding fumes (total particulate)	Pulmonary irritation	Pulmonary irritation
1437 Zinc oxide (fume)	Metal fume fever; gastritis	No data
1439 Zirconium compounds	No data	Toxic effects from zirconium tetrachloride due to liberation of hydrochloric acid

BILLING CODE 4510-26-C

Dose-Response Relationships and Systemic Effects

As Table C8–2 shows, adverse toxic reactions have been reported to occur in humans for 19 of the 34 substances in this group; thus, for more than half of these substances, it has been established conclusively that exposure is associated with adverse health effects in humans. Experimental animal data comprise the principal evidence for the toxicologic action of the remaining substances. As is the case for many substances for which limits are being established, apparent no-observed-effect levels, supplemented by the use of appropriate margins of protection, provide the basis for setting limits. The systemic effects caused by exposure to substances in this group appear to follow an NOE dose-response pattern. That is, as intensity and/or duration of exposure decreases, the severity of the effect on organ systems also decreases until a point is reached (the NOE level) where there is no detectable effect, at least at observable levels, on organ systems. No-effect exposure levels have been identified in humans and animals for several of the substances in this group; where no-effect levels have been identified (i.e., for diglycidyl ether and phenylphosphine), they have provided the primary basis for the new limits.

In instances where no-effect levels have not been reported (e.g., for n-butyl glycidyl ether, trimethylbenzene, and acetylsalicylic acid), OSHA has used safety factors and expert judgment to derive an NOE value.

The following discussions describe the record evidence and OSHA's findings for these systemic toxicants and present a summary of the material impairments of health associated with exposure to these substances.

ACETONITRILE

CAS: 75–05–8; Chemical Formula: CH_3CN
H.S. No. 1005

Acetonitrile is most widely used in industry as a specialty solvent and chemical intermediate. OSHA's former occupational exposure limit for acetonitrile was a 40-ppm 8-hour TWA. The ACGIH has a 40-ppm TLV–TWA with a 60-ppm TLV–STEL, in addition to a skin notation. OSHA proposed to reduce the former 8-hour TWA PEL to 20 ppm with a skin notation; this was the NIOSH REL, and NIOSH concurred with the proposed limit (Ex. 8–47, Table Nl). However, after a thorough evaluation of the record evidence, OSHA has concluded that the ACGIH limits for this substance will provide appropriate protection against acetonitrile's systemic toxicity Accordingly, the final rule establishes an 8-hour TWA of 40 ppm and a STEL of 60 ppm, without a skin notation, for acetonitrile.

In animal studies, acetonitrile has been found to be embryotoxic and teratogenic in rodents exposed to levels sufficiently high to cause maternal toxicity (Berteau, Levinskas, and Rodwell 1982/Ex. 1–179; Willhite 1983/Ex. 1–43). A 13-week inhalation study conducted by the National Toxicology Program (Hazleton Laboratories, Inc. 1983, as cited in ACGIH 1986/Ex. 1–3, p. 8) found pathological changes in the liver and some blood changes in mice and rats exposed to concentrations of 400 ppm acetonitrile.

The human evidence describing the toxic effects associated with exposure to acetonitrile consists of a report by Pozzani, Carpenter, Palm et al. (1959/Ex. 1–106), who exposed human subjects to acetonitrile vapor, and a case report by Amdur (1959/Ex. 1–143), who described a poisoning incident involving acetonitrile. None of three subjects exposed to 40 ppm for four hours reported any adverse responses during the exposure period, but one subject experienced tightness of the chest a few hours after termination of exposure, as well as a cooling sensation in the lungs the following day. None of the subjects had elevated blood cyanide levels; one subject showed a slightly elevated urinary thiocyanate level. Pozzani et al. (1959/Ex. 1–106) also exposed two subjects to 80 ppm and 160 ppm of acetonitrile for four hours. When exposed to 80 ppm, subjects reported no adverse response; however, at 160 ppm, one subject experienced slight flushing of the face and chest tightness a few hours after exposure (Pozzani, Carpenter, Palm et al. 1959/Ex. 1–106).

In addition to the Pozzani et al. (1959/Ex. 1–106) study, NIOSH (1978g/Ex. 1–262) cites a report by Amdur (1959/Ex. 1–143), who investigated an incident in which 16 painters became ill (with one death) after using an acetonitrile-containing material in a confined space. Amdur (1959/Ex. 1–143) reported no further incidents after adequate ventilation was installed and acetonitrile levels were maintained at about 17 ppm. NIOSH concluded that exposure to 40 ppm may have produced minimal effects and that no observable effects were produced at 17 ppm (NIOSH 1978g/Ex. 1–262, p. 97). Therefore, NIOSH recommended that exposure not exceed 20 ppm as a 10-hour TWA. Other than the comment by NIOSH (Ex. 8–47), no comments were received on this substance.

OSHA has carefully reevaluated the evidence of acetonitrile's toxicity to determine the appropriate permissible exposure limits to establish in the final rule. The Agency concludes that the evidence in humans suggests that no adverse effects are experienced at long-term exposures of 40 ppm and that a short-term limit of 60 ppm will provide protection against the facial flushing and chest tightness experienced by workers exposed for several hours to levels above these concentrations. In addition, in accordance with the policy on skin notations enunciated in Section VI.C.18, OSHA is not including a skin notation for acetonitrile in the final rule (the dermal LD_{50} in rabbits is 1250 mg/kg).

In the final rule, OSHA is therefore retaining its existing 8-hour TWA for acetonitrile and adding a STEL of 60 ppm to protect against this substance's systemic effects. The Agency concludes that these limits will prevent the significant risk of acute illness (and, in one case, death) observed in workers exposed to excessive short-term exposures of acetonitrile; the Agency finds that these health effects clearly constitute material impairments of health. In the proposal, OSHA specifically requested information on the feasibility of achieving the proposed limit; no comments were received, and OSHA accordingly assumes that the final rule's limits, which are higher than the limit proposed, are feasible.

ACETYLSALICYLIC ACID (ASPIRIN)

CAS: 50–78–2; Chemical Formula: $CH_3COOC_6H_4COOH$
H.S. No. 1006

There was no former OSHA exposure limit for acetylsalicylic acid. The ACGIH has a TLV of 5 mg/m^3 as an 8-hour TWA. The proposed PEL was 5 mg/m^3 as an 8-hour TWA. NIOSH (Ex. 8–47, Table Nl) concurs with this limit, and this is the limit established by the final rule. Acetylsalicylic acid is a white crystal or powder that is essentially odorless and has a slightly bitter taste.

The work of O'Brien (1968/Ex. 1–47) reports that a normal therapeutic dose of 600 mg aspirin will interfere with platelet aggregation in subjects exposed for a period of five days or more. Hart (1947/Ex. 1–137) also reported that 150 mg is the smallest oral dose of acetylsalicylic acid that will produce this effect. Unpublished data from the Dow Chemical Company (cited in ACGIH 1986/Ex. 1–3, p. 10) indicate that aspirin concentrations exceeding 100 mg/m^3 are tolerated except for occasional skin irritation. However, no data are available on the long-term effects on organ systems of inhalation exposure to aspirin. Secondary sources report that aspirin is an acute irritant to

the gastric mucosa and respiratory tract. No comments other than that by NIOSH (Ex. 8–47) were received on this substance.

In the final rule, OSHA is establishing an 8-hour TWA of 5 mg/m³ for acetylsalicylic acid. The Agency concludes that this reduced limit will protect workers from experiencing the adverse blood effects and gastric and respiratory irritation, which constitute material impairments of health that are potentially associated with exposure to this substance at the previously uncontrolled levels.

ALUMINUM (WELDING FUMES)

CAS: 7429–90–5; Chemical Formula: Al
H.S. No. 1019

OSHA formerly had no permissible exposure limit for aluminum welding fumes. The proposed PEL was 5 mg/m³, which is consistent with the ACGIH limit. The final rule promulgates an 8-hour TWA for aluminum welding fumes of 5 mg/m³, measured as aluminum. NIOSH (Ex. 8–47, Table N1) concurs with this limit.

OSHA received two comments pertaining to aluminum welding fumes. The first commenter (Ex. L3–1330) sought clarification as to whether the term "aluminum welding fumes" refers to aluminum fumes or to the gases and fumes usually associated with aluminum welding, such as ozone, nitrous gases, carbon monoxide, and carbon dioxide. The second commenter, the Specialty Steel Industry of the United States (Ex. 3–829), objected to the establishment of a permissible exposure limit for aluminum welding fumes because, in this commenter's opinion, no scientific evidence was cited in the proposal to indicate that exposure resulted in deleterious effects (Ex. 3–829, p. 6).

The PEL addresses the aluminum fume that is released in the welding process; this limit is being established to keep the total aluminum particulate concentrations low enough to prevent aluminum particle accumulation in the lungs. However, to the extent either that other toxic substances or materials are released in the welding process or that conditions are conducive to the formation of toxic gases, employers must pay attention to the permissible exposure limits for these substances as well. For example, in Appendix B of the 1987–88 *Threshold Limit Values and Biological Exposure Indices* (ACGIH 1987/Ex. 1–16), the ACGIH states that "reactive metals and alloys such as aluminum and titanium are arc-welded in a protective, inert atmosphere such as argon. These arcs create relatively little fume, but an intense radiation which can produce ozone" (ACGIH 1987/Ex. 1–16, Appendix B, p. 42). In such an instance, employers would be required to meet the ozone limits established in this rulemaking (0.1 ppm TWA and 0.3 ppm STEL) as well as the PEL for aluminum welding fumes.

The ACGIH states that "most welding, even with primitive ventilation, does not produce exposures inside the welding helmet above 5 mg/m³. That which does * * * should be controlled" (ACGIH 1987/Ex. 1–16, Appendix B, p. 43). In those rare instances where internal helmet exposures do exceed 5 mg/m³, employees are at risk from the irritant effects of hot metal fumes, which enter the lung deeply and accumulate.

Because workers exposed to arc welding fumes have previously not been protected by a permissible exposure limit, OSHA is establishing a PEL of 5 mg/m³ TWA for these fumes (measured in the breathing zone of the welder); the details of the appropriate positioning of the sampler should be determined on the basis of guidance in the *Field Operations Manual* (OSHA 1984). This is consistent with a past OSH Review Commission decision (8 OSHRC 1049). The Agency concludes that this limit will protect welders and other workers in the vicinity of the welding from experiencing the significant irritation potentially associated with inhalation of these fumes; OSHA finds the respiratory irritation caused by exposure to these fumes constitutes a material health impairment.

2-BUTOXYETHANOL

CAS: 111–76–2; Chemical Formula: $C_4H_9OCH_2CH_2OH$
H.S. No. 1046

OSHA's former permissible exposure limit for 2-butoxyethanol, one of the family of substances known as the glycol ethers, was 50 ppm as an 8-hour TWA, with a skin notation. The ACGIH has a limit of 25 ppm TWA, also with a skin notation, for this colorless liquid with a mild ether odor. The proposed PEL was 25 ppm as an 8-hour TWA, and this limit is established by the final rule. The skin notation is retained. NIOSH (Ex. 8–47, Table N1) concurs with the 25-ppm limit for 2-butoxyethanol.

2-Butoxyethanol has long been known to be toxic, with early studies indicating that a single seven-hour exposure to 700 ppm was lethal to laboratory animals (Werner, Mitchell, Miller, and von Oettingen 1943a, as cited in ACGIH 1986/Ex. 1–3, p. 71). Exposures near the lethal level caused systemic toxicity in the form of hemoglobinuria and lung, kidney, and liver changes. Carpenter, Pozzani, Weil, and associates (1956/Ex. 1–303) reported hemolytic anemia and increased fragility of the red blood cells in rats repeatedly exposed to 2-butoxyethanol at 320 ppm for five weeks. However, repeated exposure for 12 weeks at 400 ppm was only slightly injurious to dogs (Werner, Mitchell, Miller, and von Oettingen 1943b, as cited in ACGIH 1986/Ex. 1–3, p. 71).

Humans appear to be less susceptible to butoxyethanol poisoning than experimental animals. In humans, several single 8-hour exposures at levels of 200 ppm and 100 ppm caused urinary excretion of butoxyacetic acid; these subjects experienced irritation and discomfort after these exposures (Carpenter, Pozzani, Weil et al. 1956/Ex. 1–303). A recent study has confirmed that the increased erythrocyte osmotic fragility observed in rats exposed to many of the glycol ethers is a very sensitive indicator of toxicity and correlates with the development of hemoglobinuria at higher exposure levels (Moffett, Linnett, and Blair 1976, as cited in ACGIH 1986/Ex. 1–3, p. 71). These findings indicate that the no-effect level in animals is approximately 25 ppm. The ACGIH suggests that 2-butoxyethanol's toxicity may be more likely to occur as a result of skin absorption than as a consequence of inhalation (ACGIH 1986/Ex. 1–3, p. 71).

The Independent Lubricant Manufacturers (Ex. 3–830) objected to the establishment of a PEL for 2-butoxyethanol on the basis of a 25-ppm no-effect level in animals, particularly when the evidence suggests that humans may be less susceptible than animals to the effects of this substance (Ex. 3–830, p. 5). In response to this comment, OSHA notes that *Patty's Industrial Hygiene and Toxicology* (3rd rev. ed., Clayton and Clayton 1982) states that "the lowest concentration of ethylene glycol butyl ether vapor considered to be unpleasant and therefore disagreeable was 40 ppm" (Vol. 2C, p. 3939). This level is below OSHA's former PEL of 50 ppm, and the Agency thus believes that its former standard for 2-butoxyethanol was too high.

OSHA concludes that the former PEL of 50 ppm was insufficiently protective against the risk of 2-butoxyethanol's irritant, hematological, and other potential systemic effects, which constitute material health impairments. The limit of 25 ppm included in the final rule will reduce this significant risk to a level below that at which these toxic effects have been observed in animals and humans. This lower limit will also prevent the discomfort experienced by workers at exposure levels of 40 ppm. The skin notation is retained because of 2-butoxyethanol's ability to be absorbed dermally in toxic quantities (2-

butoxyethanol's dermal LD_{50} in rabbits is 490 mg/kg [RTECS 1988]).

n-BUTYL GLYCIDYL ETHER

CAS: 2426-08-6; Chemical Formula: $C_4H_9OCH_2CH_2OH$
H.S. No. 1052

The former OSHA limit for n-butyl glycidyl ether was 50 ppm as an 8-hour TWA. The ACGIH-recommended TLV is 25 ppm; NIOSH has recommended that occupational exposure to n-butyl glycidyl ether not exceed 5.6 ppm as a 15-minute short-term level. The proposed PEL was 25 ppm as an 8-hour TWA, and the final rule promulgates this limit. n-Butyl glycidyl ether is a clear, colorless liquid.

OSHA's former PEL of 50 ppm, which was adopted from the ACGIH's 1968 TLV list, was based on a Dow Chemical Company report (cited in ACGIH 1986/Ex. 1-3) that showed that repeated applications of n-butyl glycidyl ether to the skin of humans caused irritation and sensitization; at the time, the ACGIH concluded that a limit of 50 ppm would prevent these irritant responses. Subsequently, the ACGIH reduced the TLV to 25 ppm, noting that the 50-ppm limit was only 13 times lower than the 8-hour LC_{50} (670 ppm) reported for this chemical in rats, and that a wider margin of protection was desirable.

The NIOSH limit of 5.6 ppm was recommended in the Institute's June 1978 criteria document on glycidyl ethers (NIOSH 1978d/Ex. 1-232). This limit was based, in large part, on mutagenic studies conducted in microbial and mammalian test systems, as well as on some evidence for other members of the glycidyl ether family showing that exposure is associated with testicular atrophy and hematopoietic abnormalities in laboratory animals. After publication of its Criteria Document, NIOSH received a confidential report prepared for the Shell Development Company by Anderson et al. (1957, as cited in ACGIH 1986/Ex. 1-3, p. 81), who had conducted a rat inhalation study. In this research, rats were exposed to 38 ppm, 75 ppm, 150 ppm, or 300 ppm n-butyl glycidyl ether for seven hours daily, five days per week for 10 weeks. Atrophic testes were found in 5 of 10 rats exposed to 300 ppm, very small testes were found in 1 of 10 rats exposed to 150 ppm, and patchy atrophy was found in the testes of 1 of 10 rats exposed to 75 ppm. No effects were observed in rats exposed at 38 ppm. Based on this additional evidence, NIOSH reaffirmed its REL for n-butyl glycidyl ether in a current intelligence bulletin (NIOSH 1978p, as cited in ACGIH 1986/Ex. 1-3, p. 81).

The Workers Institute for Safety and Health (WISH) and the AFL-CIO submitted posthearing comments on n-butyl glycidyl ether (Exs. 116, 194). These commenters opposed OSHA's proposal to adopt the ACGIH TLV for this substance on the basis of the reproductive study published in a NIOSH CIB (discussed above) which shows testicular atrophy in exposed laboratory animals. According to WISH:

> OSHA's review of this substance in the proposal attempts to state that the 25 ppm ACGIH level is protective against these reproductive effects because a no-effect level of 38 ppm was observed (Ex. 116).

WISH found this conclusion unjustifiable because of the short exposure period (10 weeks) used in the study establishing the NOEL for reproductive effects and because considerable uncertainty always surrounds no-effect-level studies. In addition, WISH pointed out that "fertility in rats is less sensitive to certain testicular effects than human fertility" and, therefore, that this animal is not the best predictor of human reproductive effects (Ex. 116). In response to these comments, OSHA wishes to clarify that the Agency did not intend to imply in the proposal that the 25-ppm limit would protect against *all* risk of possible reproductive effects. In fact, the proposal merely noted that 25 ppm was below the no-effect level for reproductive effects in rats. The Agency agrees with WISH that the use of a longer exposure period in the Anderson et al. (1957, as cited in ACGIH 1986/Ex. 1-3, p. 81) reproductive study might have established a lower NOEL.

However, based on the existing evidence for reproductive effects linked to n-butyl glycidyl ether exposure, OSHA concludes that reducing the PEL from 50 ppm to 25 ppm will substantially reduce the significant risk of these reproductive effects and will also protect workers against the irritation and sensitization effects, all of which constitute material health impairment caused by exposure to this chemical. The Agency notes that NIOSH's REL of 5.6 ppm (15-minute STEL) is based on the result of *in vitro* testing in both microbial and mammalian systems; extensive extrapolation is required to predict effects in humans on these bases. The final rule establishes a permissible exposure limit of 25 ppm TWA for n-butyl glycidyl ether.

CAPTAN

CAS: 133-06-2; Chemical Formula: $C_9H_8Cl_3NO_2S$
H.S. No. 1067

OSHA did not formerly regulate captan. The ACGIH has a TLV-TWA of 5 mg/m^3 for this substance, which is a white, crystalline, odorless solid. The proposed PEL was an 8-hour TWA of 5 mg/m^3, and the final rule promulgates this limit.

Skin applications of 900 mg/kg captan produce skin irritation in experimental animals. Long-term feeding studies did not reveal adverse effects in dogs fed captan in the diet at levels of 100 mg/kg/day for 66 weeks or in rats fed 1000 mg/kg/day for two years (Martin 1971/Ex. 1-1161; Spencer 1968, as cited in ACGIH 1986/Ex. 1-3, p. 98). Male mice showed decreased fertility at levels of 50 or 100 mg/kg/day for five days (Collins 1972/Ex. 1-893).

Studies on the mutagenicity of captan indicate that the substance acts as an alkylating agent and induces chromosome rearrangements in rats and point mutations in *Neurospora crassa* (Epstein and Legator, as cited in ACGIH 1986/Ex. 1-3, p. 98). Legator and colleagues (1969, as cited in ACGIH 1986/Ex. 1-3, p. 98) reported that captan concentrations of 10 μg/ml inhibited DNA in human embryo cells, and concentrations of 1.5 μg/ml produced chromosomal aberrations in the somatic and germ cells of kangaroo rats. Animal evidence concerning the carcinogenicity of captan is contradictory, although high doses caused significant incidences of polyploid carcinoma of the duodenum and adenomatous polyps in mice (NCI 1977a, as cited in ACGIH 1986/Ex. 1-3, p. 98).

Some captan-exposed individuals experience skin irritation (Spencer 1968, as cited in ACGIH 1986/Ex. 1-3, p. 98). A case of recurrent urticaria caused by captan exposure has been reported and confirmed (Croy 1973/Ex. 1-894), and captan caused high reactivity when administered in a battery of patch tests (Rudner 1977/Ex. 1-967).

NIOSH (Ex. 8-47, Table N6A) concurs with the limit being established, but notes that captan could be classified as a potential occupational carcinogen. No other comments were received on this substance.

In the final rule, OSHA is establishing a PEL of 5 mg/m^3 TWA to protect workers exposed to captan from the significant risk of exposure-related skin irritation, reproductive effects, mutagenicity, and, perhaps, carcinogenicity, all of which constitute material health impairments. The Agency concludes that this limit will substantially reduce these significant risks.

CHLOROPRENE

CAS: 126-99-8; Chemical Formula: $CH_2{:}CClCH{:}CH_2$
H.S. No. 1088

The former OSHA limit for beta-chloroprene was an 8-hour TWA of 25 ppm, with a skin notation. The ACGIH has a 10-ppm TLV-TWA, with a skin notation, and NIOSH (1977c/Ex. 1-277) recommended a limit of 1 ppm, measured over a 15-minute period. The proposed PEL was an 8-hour TWA of 10 ppm, and the final rule establishes this limit and retains the skin notation. NIOSH (Ex. 8-47, Table N1) concurs that this limit is appropriate. Chloroprene is a colorless, highly flammable liquid.

The ACGIH recommended a reduction in the TLV for chloroprene from 25 ppm to 10 ppm in 1981, based on a review of the world literature by Trochimowicz, who prepared the 1980 ACGIH documentation, and by Reinhardt (1980, as cited in ACGIH 1986/Ex. 1-3, p. 135). Reinhardt concluded that there was no evidence indicating that the former 25-ppm PEL was not protective, but OSHA believes the systemic effects (i.e., growth retardation) seen in rats and hamsters exposed to 39 ppm chloroprene for four weeks or to 50 ppm for a lifetime suggest that the 25-ppm PEL is not sufficiently protective.

In recommending a 1-ppm 15-minute exposure limit for chloroprene, NIOSH (1977c/Ex. 1-277) cited three reports on facilities in the Soviet Union. Katsova (1973, as cited in ACGIH 1986/Ex. 1-3, p. 135) reported finding a significant excess of chromosomal abnormalities in the blood of workers exposed to approximately 5 ppm chloroprene. Volkova, Fomenko, Bagdinov et al. (1976/Ex. 1-1025) reported similar findings in a plant where chloroprene levels ranged from 0.8 to 1.95 ppm. In the third study, Sanotskii (1976/Ex. 1-662) reported abnormal sperm morphology among workers exposed at levels of from 0.28 to 1.94 ppm; a threefold increase in the rate of spontaneous abortion among wives of these workers was also found. In addition, NIOSH (1977c/Ex. 1-277) cited a study by Davtian, Fomenko, and Andreyeva (1973/Ex. 1-1032) that reported a significant excess of embryonic mortality in female rats that were mated to male rats exposed to 1 ppm chloroprene. These investigators also found chromosomal aberrations in the bone marrow cells of exposed male rats. NIOSH (1977c/Ex. 1-277) also cited a number of reports showing chloroprene to be mutagenic in a variety of test systems. NIOSH concluded that it was prudent to reduce exposure to 1 ppm over a 15-minute period, to reduce the risk of genetic abnormalities being transmitted to subsequent generations. This exposure represents the lowest concentration that can be measured reliably over a 15-minute period.

The Workers Institute for Safety and Health (WISH) and the AFL-CIO commented on OSHA's proposed limit for chloroprene (Ex. 116; Tr. VII, pp. 130-131; Ex. 194). WISH raised questions about the adequacy of the ACGIH documentation for this substance, which is critical of the Soviet literature that served as the basis for the issuance of the first NIOSH *Current Intelligence Bulletin on Chloroprene* (1975c). OSHA notes that sizable discrepancies exist between the findings from the Russian studies and results from other studies that were undertaken to confirm the Soviet claims. Torkelson and Rowe (1981c, in *Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., Vol. 2B, Clayton and Clayton 1981) offer two possible explanations for these discrepancies:

> beta-Chloroprene is a very unstable compound, which, unless handled with extreme care, . . . [epoxidizes] and polymerizes to toxic compounds. This might explain the alleged effects in animals. Alleged effects in humans may be due to this same cause or to the use of different chemical processes which produce different types of impurities. Many other causes can be postulated, but in our opinion more credence must be given to animal studies in which the sample is known to have been handled with extreme care and to the results of experience in U.S. industry where the method of handling has been reported (Torkelson and Rowe 1981c, p. 3578).

These authors report that when the purity of the sample was carefully controlled, repeated exposures to 25 ppm or less of the vapor have caused no reproductive, teratological, or embryotoxic effects in rats: "Despite frank clinical toxicity in exposed pregnant rats, fetuses showed no teratogenic effects at beta-chloroprene levels as high as 175 ppm" (Trokelson and Rowe 1981c, pp. 3579-80).

WISH also expressed concern about the "unscientific" use by the ACGIH of uncertainty factors with regard to this substance. WISH notes that the ATSDR protocol for uncertainty factors would require a TLV of 0.05 ppm based on lowest effect level data on growth retardation (Ex. 116). (See OSHA's discussion of the use of safety factors in establishing occupational exposure limits in Section VI.A. of this preamble.)

The 1-ppm (15-minute STEL) value recommended by NIOSH is based on studies reported in the Soviet literature; in addition, this limit is set at the analytical limit of detection. OSHA's 10-ppm PEL is based on a 1981 critical review of the world literature (Trochimowicz 1980, as cited in ACGIH 1986/Ex. 1-3, p. 135) and on the observation that only mild systemic effects are observed at 38 ppm. In the final rule, OSHA is establishing an 8-hour TWA PEL of 10 ppm, with a skin notation, to substantially reduce the significant risk of reproductive and systemic effects, which constitute material health impairments that are potentially associated with exposure to chloroprene. The Agency concludes that this limit will substantially reduce this significant risk.

CYCLOHEXYLAMINE

CAS: 108-91-8; Chemical Formula: $C_6H_{13}N$
H.S. No. 1109

OSHA had no former limit for cyclohexylamine. The ACGIH has a TLV-TWA of 10 ppm. OSHA proposed an 8-hour TWA PEL of 10 ppm; NIOSH (Ex. 8-47, Table N1) concurred with the proposed limit, and the final rule promulgates this limit. Cyclohexylamine is a liquid with a strong, fishy, amine odor.

Data concerning the acute toxicity of cyclohexylamine were reported by Eastman Kodak in 1958 (ACGIH 1986/Ex. 1-3, p. 161). In rats, the oral LD_{50} of a 5-percent solution in water was between 400 and 800 mg/kg; mice fed a diet of the 1-percent aqueous solution or the undiluted amine had LD_{50}s of between 200 and 400 mg/kg. Injection of the 5-percent aqueous solution in rats produced LD_{50}s of between 5 and 25 mg/kg, while mice injected intraperitoneally with the 1-percent solution had LD_{50}s of between 5 and 10 mg/kg. In guinea pigs, the dermal LD_{50} of undiluted cyclohexylamine is reported to be between 1 and 5 ml/kg. Edema, necrosis, and eschars were reported as a consequence of these dermal exposures. In rabbits, one drop of a 50-percent solution caused complete destruction of the eye. Six-hour inhalation exposures at a vapor concentration of 12,000 ppm caused deaths in rats, but exposure to 1000 ppm caused neither toxic effects nor deaths.

Legator, Palmer, Green, and Petersen (1969/Ex. 1-496) considered cyclohexylamine to be a potential carcinogen, mutagen, or teratogen on the basis of dose-dependent chromosomal abnormalities observed in rats injected intraperitoneally with cyclohexylamine. Khera, Stolz, Gunner et al. (1971/Ex. 1-343) noted adverse effects on rat fertility, and Becker and Gibson (1970/Ex. 1-298) reported embryotoxic effects in mice intraperitoneally injected with cyclohexylamine. In contrast, Kennedy, Sanders, Weinberg et al. (1969, as cited in ACGIH 1986/Ex. 1-3, p. 161) reported no effects of exposure to

cyclohexylamine on rabbit and rat fertility, reproduction, embryogenesis, or perinatal and postnatal development.

In general, there is agreement concerning the moderate to severe toxicity of cyclohexylamine and its potential for intense skin irritation and moderate skin sensitization (Sax 1968b, as cited in ACGIH 1986/Ex. 1-3, p. 161). The chemical is well known to be pharmacologically active, having sympathomimetic activity (Barger and Dale 1910/Ex. 1-1104). However, Litchfield and Swan (1971/Ex. 1-346) report that human dietary levels of 5 g/day for seven to eight days produced no pharmacologically active levels in the tissues; furthermore, no changes were detected in blood pressure, heart rate, or electrocardiograms of exposed subjects. Chronic experimental toxicity data are lacking, but Watrous and Schulz (1950/Ex. 1-940) have reported that exposure to 4 to 10 ppm of cyclohexylamine caused no symptoms of any kind in acutely exposed workmen. No comments other than those of NIOSH (Ex. 8-47) were received on this substance.

In the final rule, OSHA is establishing an 8-hour TWA PEL of 10 ppm for cyclohexylamine. The Agency concludes that limiting workplace exposures to this previously unregulated substance to the 10-ppm level will protect workers from the significant risk of severe skin and eye irritation and sensitization, all material health impairments that are associated with exposure to cyclohexylamine. OSHA has determined that this limit will substantially reduce these significant occupational risks.

CYHEXATIN

CAS: 13121-70-5; Chemical Formula: $(C_6H_{11})_3SnOH$
H.S. No. 1112

Previously, OSHA had no limit for cyhexatin. The ACGIH has a TLV-TWA of 5 mg/m³. The proposed PEL was an 8-hour TWA of 5 mg/m³. NIOSH (Ex. 8-47) concurred with the proposed limit and this is the limit established by the final rule. At room temperature, cyhexatin exists in the form of white crystals.

Cyhexatin has oral LD_{50}s of 500, 700, and 654 mg/kg for rabbits, guinea pigs, and chickens, respectively. The intraperitoneal LD_{50} for the rat is 13 mg/kg (NIOSH 1977i/ Ex. 1-1182), and the oral LD_{50} for rats has been reported to be 190 mg/kg (ACGIH 1974, as cited in ACGIH 1986/Ex. 1-3, p. 165). Skin exposure to a 1- to 2-percent solution of cyhexatin in goats and cattle caused mild effects; sheep showed mild effects after application of a 0.5-percent solution. One of five sheep died from multiple skin applications of a 1-percent suspension (Johnson, Younger, Witzel, and Radeleff 1975/Ex. 1-336).

The toxicity of cyhexatin is considered to be moderate, although it is greater than the toxicity of most other organic tin compounds. Long-term feeding in rats produced no behavioral changes, mortality, tissue changes, or hematologic or biochemical changes in response to two years of dosing at 12 mg/kg per day; however, dosed animals were smaller than controls. After daily doses by gavage of 24 mg/kg per day for two weeks, rats showed microscopic changes in the liver, kidneys, and adrenal glands at autopsy. Six mg/kg is considered to be the no-effect level in rats, and in dogs, the no-effect feeding level is reported to be 3 mg/kg. Rats fed 4 to 6 mg/kg, and rabbits fed 3 mg/kg, showed no ill effects on indices for fertility, gestation, viability, or lactation (Dow Chemical Company 1973d, as cited in ACGIH 1986/Ex. 1-3, p. 165). No inhalation data on animals are available, and there are no human data. Other than the comment by NIOSH (Ex. 8-47), no comments were received on this substance.

In the final rule, OSHA is establishing an 8-hour TWA limit of 5 mg/m³ for cyhexatin. OSHA concludes that a PEL of 5 mg/m³ will protect workers against the significant risk of skin and respiratory irritation, as well as other possible adverse effects associated with exposure to this tin compound in the absence of a current limit. The Agency considers eye and respiratory irritation to be material health impairments within the meaning of the Act.

DICHLORODIPHENYLTRICHLOROETHANE (DDT)

CAS: 50-29-3; Chemical Formula: $C_{14}H_9Cl_5$
H.S. No. 1113

OSHA's existing limit for dichlorodiphenyltrichloroethane (DDT) is 1 mg/m³ TWA as an 8-hour TWA, with a skin notation. The ACGIH has the same 8-hour TWA limit for DDT, without a skin notation. NIOSH has a REL of 0.5 mg/m³ for DDT. The Agency proposed to retain both the skin notation for DDT and the existing 8-hour TWA limit. The final rule retains the skin notation and the Agency's 8-hour TWA PEL. DDT is a noncombustible, colorless to white powder with a slightly aromatic odor.

The U.S. Public Health Service (Neal, von Oettingen, Smith et al. 1944, as cited in ACGIH 1986, p. 168) reports that six daily exposures of one hour each to 423 mg/m³ DDT was without effect in human volunteers. Barnes (1953, as cited in ACGIH 1986, p. 168/Ex. 1-3) reported that a review of the world literature revealed no illness among workers from many countries who applied DDT as an insecticide. At chronic exposure levels of 35 mg/person/day, no adverse health effects are observed in humans, but DDT does accumulate in the fatty tissues of the body and it is possible that delayed effects might occur after many years (ACGIH 1986, p. 168/Ex. 1-3). OSHA received no comments on DDT except those from NIOSH (Ex. 8-47, Table N6B), which urged regulation of DDT as a potential occupational carcinogen. The dermal LD_{50} in rabbits is 400 mg/kg (*Dangerous Properties of Industrial Materials*, 7th ed., Sax and Lewis 1989), indicating a significant degree of percutaneous absorption that justifies the skin notation.

Based on a review of the evidence of the health effects of exposure to DDT, OSHA concludes that the existing PEL of 1.0 mg/m³ is adequate to protect workers from the significant risk of bioaccumulation of DDT in adipose tissue, which may have the potential to produce delayed ill effects in later years. The Agency finds that the existing limit, with its skin notation, provides appropriate protection against DDT's systemic effects.

2-N-DIBUTYLAMINOETHANOL

CAS: 102-81-8; Chemical Formula: $(C_4H_9)_2NCH_2CH_2OH$
H.S. No. 1120

OSHA formerly had no limit for 2-N-dibutylaminoethanol (DBAE). The ACGIH has a TLV-TWA of 2 ppm, with a skin notation, for this colorless, combustible liquid, which has a faint, amine-like odor. The proposed PEL was 2 ppm as an 8-hour TWA. NIOSH (Ex. 8-47) concurred with this limit, and this is the limit established by the final rule. The proposed skin notation is not retained in the final rule.

In rats, 2-N-dibutylaminoethanol has a single-dose oral LD_{50} of 1.7 g/kg and a corresponding intraperitoneal LD_{50} of 0.14 g/kg; these values are approximately analogous to the oral and intraperitoneal LD_{50}s for diethanolamine (Hartung and Cornish 1968/Ex. 1-328). The LD_{50} for skin absorption in rabbits is 1.68 g/kg (Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1-440). In male rats, the lowest five-week drinking water dose tolerated without weight loss was 0.13 g/kg/day. Rats that ingested a dose of 0.43 g/kg/day showed elevated kidney-to-body-weight ratios but no histologic changes at autopsy (Cornish, Dambrauskas, and Beatty 1969/Ex. 1-411). In inhalation studies of rats, 6-hour exposures at 70 ppm for five days killed one rat; the surviving rats showed a 57-percent average body weight loss, as

well as a doubling of kidney-to-body-weight ratios, a tenfold increase in serum bilirubin, a slight increase in clotting time, and an elevated hematocrit. Inhalation of 33 ppm for one week caused a 3-percent body weight loss and a slight increase in clotting time, but no significant changes in the other variables observed. Twenty-seven weeks of exposure to 22 ppm resulted in no differences between exposed rats and controls in the variables measured (Cornish, Dambrauskas, and Beatty 1969/Ex. 1-411). 2-N-dibutylaminoethanol is a more potent inhibitor of acetylcholinesterase *in vitro* than is diethylamine (DEA) (Hartung and Cornish 1968/Ex. 1-328). NIOSH was the only commenter to the rulemaking record for DBAE.

In the final rule, OSHA is establishing an 8-hour TWA PEL of 2 ppm for 2-N-dibutylaminoethanol. The Agency concludes that this limit will protect workers from the significant risk of metabolic effects associated with inhalation exposure at the levels permitted in the absence of any OSHA limit. OSHA has determined that this substance does not present a significant risk of systemic toxicity via percutaneous absorption (2-N-dibutylaminoethanol's dermal LD_{50} in rabbits is 1.68 g/kg) and therefore, that no skin notation is required. Accordingly, the skin notation proposed for DBAE is not retained in the final rule.

DIGLYCIDYL ETHER

CAS: 2238-07-5; Chemical Formula: $C_6H_{10}O_3$
H.S. No. 1139

The former OSHA limit for diglycidyl ether (DGE) was 0.5 ppm as a ceiling concentration, and the ACGIH-recommended TLV is 0.1 ppm as an 8-hour TWA. NIOSH recommends a limit of 0.2 ppm for DGE as a 15-minute ceiling. OSHA proposed an 8-hour TWA of 0.1 ppm, and this limit is established in the final rule.

Both the previous ACGIH 0.5-ppm TLV and that organization's current TLV are based on the results of an animal study reported by Hine and Rowe (1963b, as cited in ACGIH 1986/Ex. 1-3, p. 202) in which rats were administered repeated 4-hour exposures of 20, 3, or 0.3 ppm DGE. Rats exposed to 20 ppm of DGE showed respiratory irritation, loss of body weight, decreased leukocyte count, involution of the spleen and thymus, and hemorrhagic bone marrow. Residual hematopoietic effects were observed among rats exposed to 3 ppm, and no observed effects were noted among rats exposed to 0.3 ppm, even after as many as 60 exposures. The ACGIH's previous TLV of 0.5 ppm as a ceiling value was based on the no-observed-effect level of 0.3 ppm reported in the Hine and Rowe (1963b, as cited in ACGIH 1986/Ex. 1-3, p. 202) study and on industrial experience. In 1979, the ACGIH reconsidered its limit for DGE, noting that, "in view of the seriousness of some of the effects produced [in the rat study], a TLV below the no-ill-effect level [of 0.3 ppm] would normally be adopted" (ACGIH 1986/Ex. 1-3). The ACGIH consequently revised the TLV to 0.1 ppm as an 8-hour TWA.

NIOSH concurs with this limit but notes that DGE may be a potential occupational carcinogen (Ex. 8-47), and the Workers Institute for Safety and Health (Ex. 116) objected to the establishment of a ceiling limit. No other comments were received on this substance.

In the final rule, OSHA concludes that the revised 8-hour TWA limit of 0.1 ppm will protect workers against the significant risk of hematopoietic and irritant effects, which constitute material health impairments and to which they were potentially exposed at OSHA's former PEL. The risks of DGE exposure range from respiratory irritation to bone marrow effects. The final rule's limit for DGE will reduce this risk substantially.

ETHANOLAMINE

CAS: 141-43-5; Chemical Formula: $NH_2CH_2CH_2OH$
H.S. No. 1159

OSHA formerly had an 8-hour TWA limit of 3 ppm for ethanolamine. The ACGIH has the same TWA limit, along with a 15-minute STEL of 6 ppm. OSHA proposed to retain the 8-hour TWA PEL of 3 ppm and to supplement this limit with a 6-ppm STEL; NIOSH (Ex. 8-47, Table N1) concurred with the proposed limits, and the final rule establishes them. Ethanolamine is a colorless liquid with a mild smell like that of ammonia.

The health hazards associated with exposures to ethanolamine include irritation and necrosis of the skin and central nervous system depression. The oral LD_{50} in rats is reported as 3.32 g/kg, and the intraperitoneal LD_{50} in rats is 981 mg/kg (Hartung and Cornish 1968/Ex. 1-328). The dermal toxicity of ethanolamine is considerably higher, with an LD_{50} of 1 mg/kg reported in the rabbit. Dermal application of the undiluted liquid also caused redness, swelling, and burns comparable to mild first-degree burns (Union Carbide Corporation, as cited in ACGIH 1986/Ex. 1-3, p. 235). The eye injury potential of ethanolamine is just slightly less than that of undiluted ammonia (Carpenter and Smyth 1946/Ex. 1-859). Rats fed 0.5 percent (320 mg/kg/day) ethanolamine in their food for 90 days (Smyth, Carpenter, and Weil 1951/Ex. 1-439) showed no adverse effects, but at 1.28 g/kg/day, fatalities occurred. Treon, Cleveland, Stemmer, and associates (1957/Ex. 1-1172) reported lung, liver, and kidney damage in various species exposed to high concentrations of the vapor and mist. In tests of various species, Weeks and co-workers (1960/Ex. 1-941) reported marked dermal effects from continuous exposures (24 hours/day, seven days/week, for from 24 to 90 days) at various concentrations of the vapor; at 12 to 26 ppm, dermal effects were less severe, but at 5 ppm, skin irritation was still evident. After 90 days of exposure to 5 ppm, dogs also experienced a slight and temporary weight loss as well as decreased activity and alertness (Weeks, Downing, Musselman et al. 1960/Ex. 1-941). Luck and Wilcox (1953/Ex. 1-917) demonstrated that a portion of low doses of ethanolamine is not excreted and is presumably retained in the body of cats, rats, and rabbits.

In studies of anesthetized dogs, Priddle (1954, as cited in ACGIH 1986/Ex. 1-3, p. 235) reported that sublethal doses of ethanolamine cause central nervous system stimulation, while lethal doses cause CNS depression. Ethanolamine's irritant and necrotic effects on the skin are not related to its alkalinity (Hinglais 1947/Ex. 1-909). OSHA received no comments, other than the one by NIOSH (Ex. 8-47), on this substance.

In the final rule, OSHA is establishing a PEL of 3 ppm as an 8-hour TWA and a 15-minute STEL of 6 ppm for ethanolamine. The Agency concludes that both of these limits are required to protect workers against the significant risk of irritation and neuropathic effects, which constitute material health impairments that are potentially associated with exposure to ethanolamine at levels permitted above the 8-hour TWA limit. The Agency has determined that these limits will substantially reduce this significant risk.

ETHYLENE CHLOROHYDRIN

CAS: 107-07-3; Chemical Formula: $ClCH_2CH_2OH$
H.S. No. 1167

OSHA formerly had an 8-hour TWA limit of 5 ppm, with a skin notation, for ethylene chlorohydrin. The ACGIH has a ceiling limit of 1 ppm, also with a skin notation. The proposed PEL was a ceiling of 1 ppm, with a skin notation. NIOSH (Ex. 8-47) concurred with the proposed limit, and the final rule establishes this limit and retains the skin notation. Ethylene chlorohydrin is a

colorless liquid with a faint, ethereal odor.

A broad range of serious health hazards are associated with exposure to this substance; these include central nervous system effects, cardiovascular effects, liver damage, kidney damage, gastrointestinal effects, skin irritation, eye irritation, and mutagenic effects. OSHA considers that all of these effects constitute material health impairments. The oral LD_{50} for rats is 72 mg/kg, and the intraperitoneal LD_{50} in the same species is 56 mg/kg (Goldblatt and Chiesman 1944/Ex. 1–980). In guinea pigs, the intraperitoneal LD_{50} is 98 mg/kg, and the percutaneous LD_{50} is 205 mg/kg (Wahlberg and Boman 1978/Ex. 1–938).

The skin absorption rate for ethylene chlorohydrin is high; Semenova and associates (1978/Ex. 1–932) determined that the LD_{50} must be reduced to one-fifth of its original value if ethylene chlorohydrin is administered daily for 20 days (Semenova, Kazanina, Fedyanina et al. 1978/Ex. 1–932).

The inhalation toxicity of ethylene chlorohydrin is also high. Ambrose (1950/Ex. 1–888) reported that a single one-hour exposure at 7.5 ppm and repeated one-hour exposures at 2 ppm can be fatal to rats. Exposures of 15 minutes daily at concentrations of from 900 to 1000 ppm were fatal to rats within a few days (Goldblatt and Chiesman 1944/Ex. 1–980).

In subacute and chronic studies, rats have died from a daily dietary dose of 67.5 mg/kg (Oser, Morgareidge, Cox, and Carson 1975/Ex. 1–923). Semenova and associates (1980, as cited in ACGIH 1986/Ex. 1–3, p. 248) reported a four-month no-effect inhalation level of 0.0033 ppm; at 0.017 ppm, slight CNS changes and alterations in the urinary secretion of nitrogen were observed after four months. These investigators also observed increased chromosomal aberrations in bone marrow in rats exposed at the 0.22-ppm level for four months (Semenova, Kazanina, Fedyanina et al. 1980, as cited in ACGIH 1986/Ex. 1–3, p. 248).

Voogt and Vet (1969/Ex. 1–1205) tested ethylene chlorohydrin in *Klebsiella pneumoniae* and found it strongly mutagenic. This finding was confirmed by the Ames test in *Salmonella typhimurium;* ethylene chlorohydrin reacts with DNA, since it inhibits the growth of DNA-deficient bacteria (Rosenkranz and Wlodkowski 1974/Ex. 1–1201). A dose-related increase of liver protein and depletion in glutathion was observed in rats after a single dose of ethylene chlorohydrin of from 10 to 50 mg/kg (Friedman, Scalera, Balazs et al. 1977/Ex. 1–1198).

One fatal and several nonfatal cases of poisoning in industrial workers have been reported from exposure (for unspecified periods of time) to ethylene chlorohydrin at levels of between 300 and 500 ppm. An autopsy of the worker who died revealed severe damage to the liver and brain, as well as effects in other organs. The survivors experienced nausea, vomiting, and irritation of the eyes, nose, and lungs (Bush, Abrams, and Brown 1949/Ex. 1–1196). Dierker and Brown (1944/Ex. 1–1197) reported that a two-hour inhalation exposure to 300 ppm was fatal in one accidental exposure. OSHA received no comments, other than that of NIOSH (Ex. 8–47), on this substance.

In the final rule, OSHA is establishing a ceiling limit of 1 ppm for ethylene chlorohydrin and is retaining the skin notation. The Agency concludes that this limit will substantially reduce the significant risk of central nervous system and other systemic effects associated with workplace exposures at the levels permitted by the TWA limit alone. The skin notation is retained because ethylene chlorohydrin is readily absorbed through the skin.

GLYCIDOL (2,3-EPOXY-1-PROPANOL)

CAS: 556–52–5; Chemical Formula: $C_3H_6O_2$
H.S. No. 1189

Previously, OSHA had an 8-hour TWA limit of 50 ppm TWA for glycidol. The ACGIH has a limit of 25 ppm TWA for this colorless liquid. The proposed PEL was an 8-hour TWA of 25 ppm. NIOSH (Ex. 8–47) concurred with this limit, and the final rule promulgates this PEL.

Glycidol causes eye, respiratory, and pulmonary irritation. Hine and associates (1956/Ex. 1–331) conducted a study of animal toxicity caused by glycidol exposure and reported that glycidol is irritating to the lungs, with mice and rats exhibiting pneumonitis and emphysema resulting from vapor inhalation. The LC_{50} reported for mice is 450 ppm for a four-hour exposure; the 8-hour LC_{50} for rats is 580 ppm (Hine, Kodama, Wellington et al. 1956/Ex. 1–331). A single dermal application was only mildly irritating (Draize score 4.5); however, repeated daily skin applications were severely irritating after four days. One drop of pure glycidol in the rabbit eye caused severe but reversible corneal injury (Hine, Kodama, Wellington et al. 1956/Ex. 1–331). In rats, chronic exposures to 400 ppm (seven hours/day for 50 days) did not cause systemic toxicity, but eye irritation and respiratory distress were observed after the first few exposures (Hine, Kodama, Wellington et al. 1956/Ex. 1–331). A study to determine glycidol's tumorigenic potential on the skin of mice showed negative results (Van Duuren, Langseth, Goldschmidt, and Orris 1967/Ex. 1–1203). OSHA received no comments, other than that of NIOSH (Ex. 8–47), on this substance.

In the final rule, OSHA is establishing an 8-hour TWA limit of 25 ppm TWA for glycidol. The Agency concludes that this limit will protect workers against the significant risk of eye, respiratory, and pulmonary irritation potentially associated with exposures to this substance. The Agency has determined that this limit will substantially reduce these significant risks.

HEXAFLUOROACETONE

CAS: 684–16–2; Chemical Formula: C_3F_6O
H.S. No. 1198

Previously, OSHA had no limit for hexafluoroacetone. The ACGIH has a TLV–TWA of 0.1 ppm, with a skin notation, for this colorless, nonflammable, highly reactive gas. The proposed PEL was an 8-hour TWA of 0.1 ppm, with a skin notation. NIOSH (Ex. 8–47) concurred with these limits, which are established by the final rule.

Inhalation studies of hexafluoroacetone in animals have shown varied systemic toxicities, including injury to the liver, kidney, testes, thymus, and bone marrow. In rats and dogs exposed six hours/day, five days/week for 13 weeks at concentrations of about 0.1, 1.0, or 12 ppm, no effects (other than increased lung weights in dogs) were observed in either species at 0.1 ppm. However, the 12-ppm exposures produced severe effects in both species, including marked but reversible testicular damage and slight hypoplasia of the spleen, thymus, and lymph nodes (E.I. du Pont de Nemours & Co., Inc. 1971, as cited in ACGIH 1986/Ex. 1–3, p. 303). Reversible kidney damage in rats and increased lung weights in dogs occurred during the 1.0-ppm exposures. An earlier four-hour acute exposure of rats demonstrated that 300 ppm was a lethal concentration (E.I. du Pont de Nemours and Co., Inc. 1971, as cited in ACGIH 1986/Ex. 1–3, p. 303).

In rats, two-week dermal exposures of 65, 130, or 250 mg/kg resulted in numerous adverse effects, including testicular damage and corresponding changes in lipid metabolism (Kennedy, Henry, Chen, and Dashiell 1982/Ex. 1–1038). A dermal dose of 13 mg/kg produced no adverse effects in rats (Lee and Gillies 1984/Ex. 1–561). An injected dose of radiolabeled hexafluoroacetone was, for the most part, rapidly excreted in the urine in unmetabolized form; this material also did not accumulate in rat

testes (Gillies and Rickard 1984/Ex. 1–322). Brittelli and co-workers (1979/Ex. 1–300) reported that hexafluoroacetone was fetotoxic in rats. Dermal application of 90 mg/kg/day to pregnant rats resulted in maternal toxicity. Fetal toxicity occurred at maternal doses of 25 mg/kg, and fetal size was reduced at maternal doses of 5 and 25 mg/kg; however, 1 mg/kg produced no fetal effect. Although soft-tissue damage and external abnormalities were observed, teratogenicity could not be demonstrated definitively (Brittelli, Culik, Dashiell, and Fayerweather 1979/ Ex. 1–300). Other than the comment by NIOSH (Ex. 8–47), OSHA received no comments on this substance.

The final rule establishes an 8-hour TWA PEL of 0.1 ppm TWA and a skin notation for hexafluoroacetone. The Agency concludes that these limits, taken together, will protect workers from the significant risk of systemic injuries at multiple organ sites, reproductive effects, kidney damage, and fetotoxic effects, all of which constitute material health impairments that are associated with exposure to hexafluoroacetone at levels above the new PEL.

HYDROGEN CYANIDE

CAS: 74–90–8; Chemical Formula: HCN
H.S. No. 1207

The former OSHA limit for hydrogen cyanide was a 10-ppm 8-hour TWA, with a skin notation. The ACGIH has a 10-ppm ceiling limit, also with a skin notation. NIOSH (1976e/ Ex. 1–240) has recommended that workplace exposures to hydrogen cyanide not exceed 4.7 ppm (5 mg/m^3) as a 10-minute ceiling. OSHA proposed a 10-minute ceiling of 4.7 ppm for hydrogen cyanide, and the final rule establishes this limit as a 15-minute STEL. The skin notation is retained. NIOSH (Ex. 8–47, Table N1) concurs with the selection of this PEL. Hydrogen cyanide is a colorless gas at room temperature.

The ACGIH (1986/Ex. 1–3) has summarized the extensive body of human evidence on the adverse effects resulting from exposure to hydrogen cyanide. The *Documentation* notes that exposure to levels of 45 to 54 ppm hydrogen cyanide can be tolerated for one hour with no immediate or delayed effects, but that 18 to 36 ppm produces "slight" symptoms after several hours of exposure. The ACGIH also cites Grabois (1954/ Ex. 1–1150), who reported that workers in apricot kernel processing plants experienced no ill effects when exposed to hydrogen cyanide at a concentration of approximately 10 ppm.

The NIOSH recommendation of 4.7 ppm as a 10-minute ceiling limit is based largely on an epidemiologic study by El Ghawabi et al. (1975/Ex. 1–632) that showed an increase in symptoms of headache, weakness, throat irritation, vomiting, dyspnea, lacrimation, colic, and nervousness among workers exposed to cyanide for an average of 7.5 years. The 36 male workers that were studied were employed in three electroplating factories. Breathing zone samples (15 minutes in duration) were collected and ranged from 4.2 to 12.4 ppm. Cyanide levels at two of the three plants did not exceed 9.6 ppm. El Ghawabi et al. (1975/Ex. 1–632) also reported that two workers in one plant suffered from psychotic episodes; these conditions were reported to be similar to cases that occurred during the therapeutic use of thiocyanate. Mean values of urinary thiocyanate in the 36 workers correlated well with air concentrations of cyanide (El Ghawabi, Gaafar, El-Saharti et al. 1975/Ex. 1–632).

Symptoms resulting from chronic exposure to cyanide were also reported by Radojicic (1973, as cited in NIOSH 1976e/Ex. 1–240) among workers exposed to HCN levels between 5.4 and 12.3 ppm, and by Saia, DeRosa, and Galzigna (1970, as cited in NIOSH 1976e/Ex. 1–240). NIOSH (1976e/Ex. 1–240) interpreted the significance of these studies as follows:

> Colle (1972) . . . advanced the belief that these symptoms of headache, dyspnea, epigastric burning, vertigo, tinnitus, nausea, vomiting, tremor, and precordial pain represent a true clinical entity and that they are sufficiently documented and characteristic of chronic cyanide exposure to be grouped into a true syndrome. . . .
>
> Chaumont (1960) . . . also stated that there is no clinical evidence to deny that cyanides can cause this type of occupational intoxication. He apparently found the debate on whether this intoxication is truly chronic or whether it involves repeated subacute symptoms to be semantic in nature and opted for the admission that chronic intoxication caused by HCN and the cyanide salts is a true occupational disease. . . .
>
> Thus, one might describe chronic cyanide poisoning as a slow deterioration of resistance, and, therefore, an intensified sensitivity, due to inadequate time between exposures for replacement of damaged tissues, enzyme systems and metabolic stores, the elimination of detoxication products, and the regeneration of homeostatic mechanisms (NIOSH 1976e/Ex. 1–240, pp. 90–91).

OSHA received a few comments, in addition to that made by NIOSH (Ex. 8–47), on its proposal to revise the PEL for HCN to 4.7 ppm (5 mg/m^3) as a short-term limit. Dr. Lawrence Hecker, representing Abbott Laboratories (Ex. 3–678), recommended that OSHA retain its former skin notation for HCN; OSHA's intention to do so was inadvertently omitted from the discussion of hydrogen cyanide in the NPRM. There is ample evidence that cyanide penetrates the skin in sufficient quantities to cause systemic effects (NIOSH 1976e/Ex. 1–240).

Accordingly, OSHA is retaining its skin notation for HCN in the final rule. BP America (Ex. 8–57; Tr. 9–127) urged OSHA to establish the ACGIH TLV rather than the NIOSH REL for HCN, and the New Jersey Department of Health urged use of EPA's IRIS data to set a PEL for this substance (Ex. 144). In response to these commenters, OSHA notes that the ACGIH is not, in the Agency's opinion, sufficiently protective. Use of the IRIS data is discussed in Section VI.A.

OSHA concludes that a variety of symptoms are associated with exposure to hydrogen cyanide at levels less than 10 ppm. This shows that neither the former PEL nor the ACGIH TLV is sufficiently protective. In the final rule, OSHA is therefore establishing a 4.7-ppm 15-minute STEL as the PEL. The Agency finds that the final rule's short-term limit will protect workers from the significant risk of headache, weakness, colic, and nervousness, which together constitute material impairment of health; these effects have been observed in individuals exposed at the 10-ppm level over a full working shift. OSHA concludes that this limit will substantially reduce these significant risks.

HYDROGENATED TERPHENYLS

CAS No.: 61788–32–7; Chemical Formula: None
H.S. No. 1210

Previously, OSHA did not regulate the hydrogenated terphenyls. The ACGIH has a TLV–TWA of 0.5 ppm (approximately 5 mg/m^3) TWA for these complex mixtures of ortho-, meta-, and para-terphenyls in various stages of hydrogenation. The proposed PEL was 0.5 ppm as an 8-hour TWA; NIOSH (Ex. 8–47) concurred with the proposed limit, and the final rule establishes that limit.

Acute exposure to the hydrogenated terphenyls poses a risk of potential lung, eye, and skin damage. Chronic exposure presents a risk of systemic toxicity involving injury to the liver, kidneys, and blood-forming organs, as well as possible metabolic disturbances and cancer (ACGIH 1986/Ex. 1–3, p. 311).

Early studies of unhydrogenated terphenyl isomers determined that the LD_{50} in rats is low, i.e., 1900 mg/kg for the ortho isomer, 2400 mg/kg for the meta isomer, and 10,000 mg/kg for the para isomer (Cornish, Bahor, and Ryan 1962/Ex. 1–410). Thirty-day oral

administration of 500 mg/kg/day in the diet of rats indicated possible liver and kidney damage, which was suggested by increases in the liver- and kidney-to-body-weight ratios and decreases in the rate of weight gain (Cornish, Bahor, and Ryan 1962/Ex. 1–410). Other studies have demonstrated nephrotoxicity and liver damage in rats fed 33 mg/kg or more of unirradiated terphenyl isomers (Petkau and Hoogstraaten 1965/Ex. 1–432; Young, Petkau, and Hoogstraaten 1969/Ex. 1–459). Inhalation studies showed that bronchopneumonia is associated with exposure at 88 to 356 ppm to the ortho and meta isomers, but not to the para isomer at 103 ppm (Haley, Detrick, Komesu et al. 1959/Ex. 1–326). The work of Cornish, Bahor, and Ryan (1962/Ex. 1–410) showed that none of the isomers caused skin irritation in rabbits following a 24-hour dermal application. For terphenyls that are approximately 40-percent hydrogenated, the acute oral LD_{50} in rats is reported as 17,500 mg/kg; in mice, it is 12,500 mg/kg (Adamson and Weeks 1973/Ex. 1–295). This study also demonstrated that an irradiated hydrogenated terphenyl mixture is three times more acutely toxic by ingestion than is a nonirradiated mixture. This finding was confirmed in 16-week chronic ingestion studies (Adamson, Bowden, and Wyatt 1969/Ex. 1–293); these authors found that 1200 mg/kg of an irradiated mixture was lethal to mice, while the same dose in nonirradiated form produced only an irreversible interstitial nephritis. In the same study, no effects were observed for either mixture at a dose level of 250 mg/kg.

Eight-day inhalation studies in mice showed some pathologic changes in lung tissue after 500 mg/m^3 (50 ppm) exposures to nonirradiated hydrogenated terphenyls; eight-week exposures at 2000 mg/m^3 (200 ppm) resulted in the same lung damage, as well as in some proliferation of the smooth endoplastic reticulum in the liver (Adamson, Bowden, and Wyatt 1969/Ex. 1–293; Adamson and Weeks 1973/Ex. 1–295). Carcinogenesis in mice has been reported from 8-week skin exposures to the irradiated mixture (Henderson and Weeks 1973/Ex. 1–784). The significance of the changes observed by Adamson and Furlong (1974/Ex. 1–294) in the mouse lung after eight weeks of inhalation exposure to the irradiated mixture is difficult to interpret in terms of the potential of the hydrogenated terphenyls to cause pulmonary cancer; particles were found to clear the lungs rapidly but to accumulate and clear more slowly in the intestine, kidney, and liver. No comments other than those of NIOSH (Ex. 8–47) were received on this substance.

In the final rule, OSHA is establishing a 0.5-ppm 8-hour TWA for the complex mixtures of ortho-, meta-, and para-terphenyls (either irradiated or nonirradiated) in various stages of hydrogenation. The Agency concludes that this limit will protect workers from the significant risks of eye, skin, and lung damage and of systemic toxicity to the liver, kidneys, and blood-forming organs, all material health impairments that are potentially associated with exposure to these substances at levels above the new PEL.

2-ISOPROPOXYETHANOL

CAS: 109–59–1; Chemical Formula: $(CH_3)_2CHOCH_2CH_2OH$
H.S. No. 1223

OSHA had no former limit for 2-isopropoxyethanol. The ACGIH has a TLV–TWA of 25 ppm for this mobile liquid. The proposed PEL was 25 ppm as an 8-hour TWA, and the final rule establishes this limit.

2-Isopropoxyethanol has been demonstrated to produce systemic toxicity in laboratory animals. In studies of rats, 15 six-hour exposures at 1000 ppm caused hemoglobinuria, anemia, and lung congestion, but no fatalities (Gage 1970/Ex. 1–318). At 300 ppm, Gage reported transient hemoglobin and MCHC decreases and lung congestion after 15 exposures. Exposure at the 100-ppm level produced no effect (Gage 1970/Ex. 1–318). Another study reported a significant increase in the osmotic fragility of erythrocytes in female rats after a four-hour inhalation exposure to 62 ppm, but no effect was observed at 32 ppm (Carpenter, Pozzani, Weil et al. 1956/Ex. 1–303). Studies of four species exposed at concentrations of 200, 50, or 25 ppm for six hours/day for 26 weeks resulted in hematologic changes only in rats; increased osmotic fragility of erythrocytes was marked at 200 ppm, slight at 50 ppm, and minimal at 25 ppm (Moffett, Linnett, and Blair 1976, as cited in ACGIH 1986/Ex. 1–3, p. 235).

NIOSH (Ex. 8–47) did not concur with OSHA's proposed limit of 25 ppm, noting that 25 ppm represented an effect level. Although "slight" increases in osmotic fragility were reported in animals subchronically exposed (Moffett, Linnett, and Blair 1976, as cited in ACGIH 1986/Ex. 1–3, p. 235), OSHA notes that a marked reaction did not occur until exposure was increased eightfold. Therefore, at this time, OSHA judges the 25-ppm PEL to be sufficiently protective.

OSHA is establishing an 8-hour TWA PEL of 25 ppm for 2-isopropoxyethanol in the final rule. The Agency concludes that this limit will substantially reduce the significant risk of hemolytic effects, which are material health impairments that are associated with exposure to this substance at levels above the new PEL.

ISOPROPYL GLYCIDYL ETHER

CAS: 4016–14–2; Chemical Formula: $C_6H_{12}O_2$
H.S. No. 1227

OSHA's former limit for isopropyl glycidyl ether (IGE) was 50 ppm as an 8-hour TWA. The ACGIH has an 8-hour TWA of 50 ppm and a 15-minute STEL of 75 ppm for IGE. NIOSH (Ex. 8–47, Table N7) recommends a limit of 50 ppm as a 15-minute ceiling. OSHA proposed an 8-hour TWA of 50 ppm and a 15-minute STEL of 75 ppm for IGE, and these limits are established in the final rule. IGE is a colorless, volatile liquid.

The 4-hour LC_{50} for IGE in mice was 1500 ppm and the 8-hour LC_{50} in rats was 1100 ppm (Hine, Kodama, Wellington et al. 1956/Ex. 1–331). The intragastric LD_{50}s in mice and rats were 1.30 and 4.2 g/kg, respectively; in rabbits, the dermal LD_{50} was 9.65 g/kg (Hine, Kodama, Wellington et al. 1956/Ex. 1–331). Fifty daily seven-hour exposures of rats to 400 ppm caused a reduced rate of weight gain, an increase in hemoglobin, a decrease in peritoneal fat, and, in some animals, emphysematous lungs and mottling of the liver (Hine, Kodama, Wellington et al. 1956/Ex. 1–331). Animals in this study also exhibited signs of ocular irritation and respiratory distress.

In humans, eye, nose, and upper respiratory irritation occurred in the technicians handling the animals in the Hine and co-workers (1956/Ex. 1–331) study; exposure levels were not specified. Dermatitis has also been reported in workers exposed to other glycidyl ethers during manufacture, and one such case involved IGE exposure (ACGIH 1986/Ex. 1–3, p. 340).

In the final rule, OSHA is retaining the 8-hour TWA of 50 ppm and adding a 15-minute STEL of 75 ppm for IGE. The Agency concludes that both the TWA and STEL are necessary to reduce the risk to workers of chronic organ effects, such as those demonstrated to occur in animals (Hine, Kodama, Wellington et al. 1956/Ex. 1–331), and the significant risk of eye, skin, and upper respiratory tract irritation associated with short-term IGE exposures at the levels permitted in the absence of a short-term limit. OSHA considers sensory irritation, dermatitis, and chronic organ effects to be material impairments of health.

4,4'-METHYLENE BIS(2-CHLOROANILINE)

CAS: 101-14-4; Chemical Formula: $CH_2(C_6H_4ClNH_2)_2$

H.S. No. 1273

Previously, OSHA had no limit for 4,4-methylene bis (2-chloroaniline), or MBOCA, although in 1974, OSHA did issue a standard for MBOCA as part of the Agency's "14 Carcinogens" rulemaking; however, the reviewing court set the MBOCA standard aside on procedural grounds. The ACGIH has a limit of 0.02 ppm (0.22 mg/m^3) TWA, with a skin notation, and classifies MBOCA as a suspected human carcinogen (A2). NIOSH recommends a TWA limit of 3 μg/m^3 for MBOCA, which NIOSH considers a potential occupational carcinogen. OSHA proposed an 8-hour TWA of 0.02 ppm TWA for MBOCA, with a skin notation; the final rule establishes these limits. MBOCA is a tan-colored solid.

MBOCA is highly toxic, causing cyanosis, kidney irritation, methemoglobinemia, and cancer. It is similar in effect to the other aromatic amines (Hosein and van Roosmalen 1978/ Ex. 1-1054; Mastromatteo 1965/Ex. 1-146).

Steinhoff and Grundmann (1969/Ex. 1-762) demonstrated that feeding MBOCA at unspecified levels to rats on a protein-deficient diet caused a high incidence of liver cancer. Russfield, Homburger, Boger and associates (1975/ Ex. 1-929) reported liver and lung tumors in rats fed MBOCA while on a standard diet. Dogs fed MBOCA at a dose of 100 mg/day, five days/week showed no hepatic cancer, but malignant nodules in the bladder occurred in a dog fed MBOCA for nine years (Stula et al. 1977, as cited in ACGIH 1986/Ex. 1-3, p. 392.4).

In industry, reversible hematuria has been reported among MBOCA-exposed workers, but precise concentration data are lacking (Mastromatteo 1965/Ex. 1-146). An early study of workers exposed for as long as 18 years to MBOCA showed no adverse effects, although the substance and its metabolites were detected in the urine of these subjects (Linch, O'Connor, Barnes et al. 1971/Ex. 1-791). Hosein and van Roosmalen (1978/Ex. 1-1054) reported an industrial accident in which molten MBOCA was splashed in a worker's face; urinary levels of 3.6 mg/L MBOCA, as well as protein, were detected in the urine, and the subject experienced nausea. However, this worker recovered quickly.

A recent NIOSH retrospective study involving 370 workers employed in a MBOCA-manufacturing plant evaluated the carcinogenicity of this substance, which is structurally similar to benzidine. This study found two cases of bladder cancers in very young workers (less than 30 years of age), both of whom were nonsmokers.

The Polyurethane Manufacturers Association (PMA) expressed its support for establishing a 0.02-ppm TWA for MBOCA, stating that the proposal "will significantly assist in assuring that any exposure to the chemical is appropriately controlled while imposing a regulation which can be feasibly complied with by employers" (Ex. 3-683, p. 4). In addition, the PMA indicated that, with currently applied engineering and work practice controls, MBOCA "can be used with no or very limited employee exposure" (Ex. 3-683, p. 5). The PMA also supported establishment of a PEL for MBOCA "to provide OSHA with a chemical-specific enforcement capability to deal with any isolated instances where a user of the chemical also disregards recognized industry practices and fails to reasonably control employee exposure to the chemical" (Ex. 3-683, p. 7). The PMA supported the addition of a skin notation for MBOCA, identifying dermal contact as a "principal potential route for employee exposure" (Ex. 3-683, p. 7).

NIOSH (Ex. 8-47, Table N6B) did not concur with OSHA's proposed PEL and recommended instead that the Agency undertake a separate 6(b) rulemaking for MBOCA. OSHA is aware of the two bladder cancer cases reported by NIOSH, and will continue to monitor the toxicologic evidence on MBOCA in the future to determine whether the evidence warrants a further reduction in the exposure limit. The AFL-CIO (Ex. 194) urged OSHA to promulgate ancillary limits for MBOCA; however, as discussed in Section IV.D., the Agency is not at this time promulgating such provisions because of the size and scope of this rulemaking.

In the final rule, OSHA is establishing an 8-hour TWA limit of 0.02 ppm for MBOCA, with a skin notation. The Agency concludes that this limit will protect workers against the significant risks of cyanosis, methemoglobinemia, kidney irritation, and bladder cancer, all material health impairments potentially associated with exposure to this substance. A skin notation is established to protect against the percutaneous absorption and systemic toxicity demonstrated by this substance in industrial accidents.

PHENYLHYDRAZINE

CAS: 100-63-0; Chemical Formula: $C_6H_5NHNH_2$

H.S. No. 1317

OSHA's former limit for phenylhydrazine was 5 ppm TWA as an 8-hour, with a skin notation. The ACGIH has a TLV-TWA of 5 ppm with a STEL of 10 ppm, and a skin notation. NIOSH (1978e/Ex. 1-263) recommends that workplace exposures not exceed 0.14 ppm as measured over a two-hour period. OSHA proposed to retain the PEL of 5 ppm as an 8-hour TWA and to add a STEL of 10 ppm, with a skin notation, and these limits are established in the final rule. Phenylhydrazine may be either yellow crystals or an oily liquid that darkens on exposure to air and light.

No data are available on the effects of phenylhydrazine resulting from inhalation. The ACGIH limits are based on the high acute toxicity of the compound when administered orally or subcutaneously to animals; single doses on the order of 20 mg/kg have resulted in the death of dogs within 22 days (Hesse, Franke, and Hering 1935/Ex. 1-785) and produced a marked decrease in erythrocyte count in rodents (von Oettingen and Deichmann-Greubler 1936/Ex. 1-771). Anemia and hemolysis are the characteristic responses seen in animals fed or injected with phenylhydrazine.

In its criteria document on the hydrazines, NIOSH (1978e/Ex. 1-263) reviewed four studies on the carcinogenicity of phenylhydrazine in mice. One study (Toth and Shimizu 1976/Ex. 1-675) found significant increases in blood vessel tumors. Another study (Clayson, Biancifiori, Milia, and Santilli 1966, as cited in ACGIH 1986/Ex. 1-3, p. 477) reported increased incidences of lung adenomas and adenocarcinomas. Two other studies (Roe, Grant, and Millican 1967/ Ex. 1-659; Kelly, O'Gara, Yancy et al. 1969/Ex. 1-703) were negative. NIOSH concluded that phenylhydrazine should be considered a potential human carcinogen and recommended that exposures not exceed 0.14 ppm over a two-hour sampling period, which represents the lowest level that can be detected reliably. The ACGIH (1986/Ex. 1-3) has placed phenylhydrazine on its A2 (suspected human carcinogens) list.

NIOSH (Ex. 8-47, Table N6B; Tr. 3-97 to 3-98), the Workers Institute for Safety and Health (WISH) (Ex. 116), the AFL-CIO (Ex. 194), the Oil Chemical and Atomic Workers (Tr. 9-218), and the American Industrial Hygiene Association (Ex. 8-16) were of the opinion that OSHA's proposed revision of the PEL for phenylhydrazine was not sufficiently protective. NIOSH (Ex. 8-47) indicated that phenylhydrazine may be a suitable candidate for an individual 6(b) rulemaking. Typical of the views of these commenters was the statement of

WISH (Ex. 116), which commented that the ACGIH had, at one time, considered reducing its 5-ppm TLV-TWA, and cited a 1974 study in which rabbits given intravenous injections of phenylhydrazine showed blood and liver effects. The evidence of phenylhydrazine's possible carcinogenicity was also cited by WISH as additional support for a more stringent limit. In response, OSHA notes that the Agency is also concerned about the evidence for these adverse effects of phenylhydrazine exposure and will continue to monitor and evaluate the toxicologic literature on phenylhydrazine to determine whether there is a need in the future for a further reduction in the occupational exposure limit.

However, at the present time, OSHA is retaining the 5-ppm 8-hour TWA and adding a 10-ppm STEL for phenylhydrazine; the skin notation is also retained. The Agency concludes that these two limits will work together to keep workplace exposures well controlled and will reduce the significant health risks associated with exposure to this substance. These risks include acute blood-related toxicity and may also include cancer; these effects clearly constitute material impairments of health. OSHA finds that the TWA and STEL limits established in the final rule will substantially reduce these significant risks.

PHENYLPHOSPHINE

CAS: 638-21-1; Chemical Formula: $C_6H_5PH_2$
H.S. No. 1318

OSHA had no former requirement for limiting worker exposure to phenyl phosphine; NIOSH also has no REL for this substance. The ACGIH has recommended a ceiling limit of 0.05 ppm for this solid. The proposed PEL was a ceiling of 0.05 ppm; NIOSH (Ex. 8-47, Table NI) concurred with the proposed limit, and this limit is established in the final rule.

A 90-day inhalation study conducted by the du Pont Company, in which rats and beagle dogs were exposed to average concentrations of 0.6 ppm or 2.2 ppm phenylphosphine for six hours per day, five days per week, showed that rats exposed to 2.2 ppm had significant hematologic changes and testicular degeneration (E.I. du Pont de Nemours & Co., Inc. 1970, as cited in ACGIH 1986/Ex. 1-3, p. 479). These effects were not noted among rats exposed to 0.6 ppm, but rats exposed at the lower level did show hypersensitivity to sound and touch and mild hyperemia. The dogs tolerated the higher exposure level better than the rats in that some regeneration of testicular damage occurred in dogs during a one-month recovery period. Dogs exposed to 0.6 ppm exhibited intermittent nausea, diarrhea, lacrimation, and hind leg tremor (ACGIH 1986/Ex. 1-3). The ACGIH considered 0.6 ppm to be an NOE level for severe effects in animals and recommended a 0.05-ppm ceiling TLV to provide a tenfold safety margin to protect workers against the changes exhibited by the test animals at the 0.6-ppm level. No comments other than that from NIOSH (Ex. 8-47) were received by OSHA.

OSHA concludes that workers formerly exposed to uncontrolled levels of phenylphosphine were at significant risk of experiencing the nausea, irritation, and CNS effects found to be associated with such exposures in animals. OSHA finds that these effects constitute material health impairments. The Agency concludes that the final rule's ceiling of 0.05 ppm will reduce these significant risks substantially.

PHOSPHINE

CAS: 7803-51-2; Chemical Formula: PH_3
H.S. No. 1321

OSHA formerly had a PEL of 0.3 ppm TWA for phosphine. The ACGIH recommends a TLV-TWA of 0.3 ppm and a TLV-STEL of 1.0 ppm. The proposal retained the 8-hour TWA of 0.3 ppm and added a STEL of 1 ppm. NIOSH (Ex. 8-47) concurred with this proposal. These limits are established in the final rule. Phosphine is a colorless gas with a disagreeable, garlic-like odor.

Early studies reported that laboratory animals could tolerate phosphine in four-hour-daily exposures of 5 ppm for two months, but fatalities were observed from seven similar exposures at 10 ppm (Muller 1940/Ex. 1-919). In 1975, Waritz and Brown (Ex. 1-451) reported a 4-hour LC_{50} of 11 ppm in rats; these lethal exposures caused effects typical of respiratory irritation.

Prior to 1958, numerous cases of phosphine-related occupational poisonings and deaths were reported, including a fatality caused by pulmonary edema that was attributed to an exposure of 8 ppm for two hours daily (Harger and Spolyar 1958/Ex. 1-327). Sublethal symptoms (without chronic effects) occurred at phosphine exposures averaging 10 ppm or less, with excursions of up to 35 ppm; recorded symptoms included diarrhea, nausea, vomiting, respiratory distress, and dizziness (Jones, Jones, and Longley 1964/Ex. 1-420). The literature contains no documented reports of chronic poisoning caused by prolonged exposure to phosphine, although several authorities have asserted that this is a possibility (Henderson and Haggard 1943e/Ex. 1-1086; Fairhall 1957h, as cited in ACGIH 1986/Ex. 1-3, p. 883; Johnstone and Miller 1960/Ex. 1-1114; Patty 1963f, as cited in ACGIH 1986/Ex. 1-3, p. 883; American Industrial Hygiene Association (AIHA) 1964/Ex. 1-407).

Joel Carr, Health and Safety Research Director for the American Federation of Grain Millers Union, testified on the toxicology of and employee exposures to phosphine in grain elevators and flour mills (Ex. 8-1; Tr. pp. 7-240 to 7-259). Mr. Carr described a report of a group of industrial hygiene studies published by NIOSH (Zaebst 1986; Zaebst, Blade, Morelli-Schroth et al. 1987; Zaebst, Blade, Burroughs et al. 1988), in which applicators of phosphine were found to be exposed above the proposed TWA PEL and STEL; nonapplicator workers also become exposed while working near fumigated grain, while loading or transferring fumigated grain, or while working in elevators and mills.

Mr. Carr also cited additional health studies, including a report of chronic neurological problems following an acute episode of phosphine poisoning (Kurzbauer and Keise 1987), animal data indicating that phosphine inhibits catalase activity (Price and Walter 1987), and studies showing phosphine to be mutagenic both *in vitro* and *in vivo* (*Occupational/Environmental Pathology Review* 1988) (Tr. p. 7-246; Ex. 45A). He cited another NIOSH report (*Studies of the Prevalence of Chronic, Non-Specific Lung Disease and Related Health Problems in the Grain Handling Industry,* Rankin et al. 1986) that identified several symptoms associated with phosphine exposure, including headaches, dizziness, diarrhea, nausea, and dyspnea, as well as palpable abdomen (Tr. p. 7-247). Mr. Carr also mentioned the preliminary results of an NCI mortality study of grain workers in which elevated relative risks were found for non-Hodgkin's lymphoma (Tr. p. 7-254). Mr. Carr urged OSHA to adopt a short-term limit of 0.3 ppm, which is consistent with EPA's Maximum Concentration Limit for phosphine applicators (Tr. p. 7-250); in addition, he recommended that OSHA establish provisions for exposure and medical monitoring, training, and respiratory protection for phosphine.

OSHA appreciates the information supplied by Mr. Carr on phosphine toxicity and awaits completion of the ongoing studies discussed by him at the hearing. In response to Mr. Carr's request that OSHA establish other requirements in addition to the PEL, OSHA notes that the Agency is currently conducting rulemaking activities to develop generic standards

for respiratory protection, medical surveillance, and exposure monitoring, but that the sole purpose of this rulemaking is to revise OSHA's outdated exposure limits.

In the final rule, OSHA is retaining the 8-hour TWA PEL for phosphine of 0.3 ppm and adding a 15-minute STEL of 1 ppm. The Agency concludes that both of these limits are required to substantially reduce the significant risk of lung damage, diarrhea, and nausea, all material health impairments associated with elevated short-term and long-term exposure to this gas.

PIPERAZINE DIHYDROCHLORIDE

CAS: 142-64-3; Chemical Formula: $C_4H_{10}N_2$ 2 HCl
H.S. No. 1330

Previously, OSHA had no limit for piperazine dihydrochloride. The ACGIH recommends a TLV-TWA limit of 5 mg/m^3. The proposed PEL was an 8-hour TWA of 5 mg/m^3; NIOSH (Ex. 8-47) concurred with the proposed PEL, and this limit is established in the final rule. Piperazine dihydrochloride is a solid.

Piperazine dihydrochloride is a water-soluble solid with low systemic toxicity and mild irritant properties; the compound is biologically active. The oral LD_{50} for rats has been reported as 4.9 g/kg (NIOSH 1984, as cited in ACGIH 1986/Ex. 1-3, p. 491).

Eye and skin irritation have been reported as a result of human exposures to high (not further specified) levels of piperazine dihydrochloride; subjects experienced mild to moderate skin burns and sensitization. Inhalation of the dust has been associated with asthmatic reactions (Dow Chemical Company 1977h, as cited in ACGIH 1986/Ex. 1-3, p. 491). OSHA received no comments other than that from NIOSH (Ex. 8-47) on this substance.

In the final rule, OSHA is establishing a limit of 5 mg/m^3 as an 8-hour TWA for piperazine dihydrochloride. The Agency concludes that this limit will reduce the significant risks of sensitization and eye and skin irritation, which constitute material health impairments and are potentially associated with exposures to this substance at levels above the new limit.

n-PROPYL NITRATE

CAS: 627-13-4; Chemical Formula: $CH_3CH_2CH_2ONO_2$
H.S. No. 1340

OSHA formerly had an 8-hour TWA limit of 25 ppm for n-propyl nitrate. The ACGIH has a 25-ppm TWA and a 15-minute STEL of 40 ppm; these limits were proposed by OSHA. NIOSH (Ex. 8-47) concurred with these proposed limits, and these limits are established in the final rule. n-Propyl nitrate is a pale yellow liquid with a sickly sweet odor.

Rats inhaling propyl nitrate vapor for four hours at a concentration of 10,000 ppm exhibited cyanosis and methemoglobinemia before they died (Hood 1953, as cited in ACGIH 1986/Ex. 1-3, p. 505). The intravenous LD_{50} in unanesthetized rabbits has been reported to be between 200 and 250 mg/kg; in anesthetized dogs and cats, intravenous doses of between 100 and 200 mg/kg were usually fatal (Murtha, Stabile, and Wills 1956/Ex. 1-649). Murtha and associates (1956/Ex. 1-649), who conducted these studies, concluded that n-propyl nitrate exerts a direct action on the vascular smooth muscle and that the ensuing cardiac effects and respiratory depression contribute to the compound's hypotensive action (Murtha, Stabile, and Wills 1956/Ex. 1-649). Inhalation trials in mice, rats, hamsters, guinea pigs, and dogs have established 4-hour LC_{50} values ranging from 9000 to 10,000 ppm for rats, 6000 to 7000 ppm for mice, and 2000 to 2500 ppm for dogs. Dogs survived repeated exposures (six hours/day, five days/week) at 260 ppm for six months, although slight clinical signs were observed during the first two weeks of exposure (Rinehart, Garbers, Greene, and Stoufer 1958/Ex. 1-524). The percutaneous toxicity of n-propyl nitrate is low but may cause inflammation and thickening of the skin after repeated exposures; these effects are sometimes transient (ACGIH 1986/Ex. 1-3, p. 505). To protect against cardiovascular and respiratory depressant effects requires both TWA and STEL limits. NIOSH (Ex. 8-47) was the only commenter to the rulemaking record for this substance.

In the final rule, OSHA is retaining the PEL of 25 ppm TWA and adding a STEL of 40 ppm for n-propyl nitrate. The Agency concludes that this combined PEL-STEL limit will protect workers against the significant risk of cyanosis, methemoglobinemia, and hypotension, all material health impairments are potentially associated with exposure to n-propyl nitrate at levels above the 8-hour TWA PEL.

SODIUM FLUOROACETATE

CAS: 62-74-8; Chemical Formula: $CH_2FCOONa$
H.S. No. 1366

The former OSHA standard for sodium fluoroacetate was 0.05 mg/m^3 as an 8-hour TWA, with a skin notation. The ACGIH has established exposure limits of 0.05 mg/m^3 TLV-TWA and 0.15 mg/m^3 TLV-STEL, with a skin notation. The proposal retained the former 8-hour TWA PEL and added a STEL of 0.15 mg/m^3, with a skin notation; NIOSH (Ex. 8-47) concurred with this proposal, and these limits are established in the final rule. The skin notation is retained. Sodium fluoroacetate is a fine white powder, which is sometimes dyed black for commercial use.

Sodium fluoroacetate causes vomiting, convulsions, and ventricular fibrillation. It is highly toxic by inhalation, ingestion, or via absorption through the skin (*Occupational Health Guidelines for Chemical Hazards*, NIOSH/OSHA 1981). The ACGIH calculated and set the threshold limit of 0.05 mg/m^3 based on studies of rats indicating an oral LD_{50} of 1.7 mg/kg (Lehman 1951/Ex. 1-790). Tissue changes in rats were noted in a later study by the same author in which the animals were fed 0.25 mg sodium fluoroacetate/kg/day (Lehman 1952, as cited in ACGIH 1986/Ex. 1-3, p. 534); the equivalent level in humans would be 17 mg/person/day. A further study by Miller and Phillips (1955, as cited in ACGIH 1986/Ex. 1-3, p. 534) examined growth rates in rats fed various dosages of sodium fluoroacetate. Rats who received 10 ppm in their diet experienced a transient fluctuation in growth rate. At 20 ppm (approximately 2 mg/kg in young rats), the growth rate declined markedly the first week; the rats survived and resumed growth at the normal rate in three to four weeks. Tolerance for the chemical lasted less than two weeks, and those rats who had adjusted to sodium fluoroacetate showed a second retardation of growth when returned to a dietary level of 20 ppm after a two-week interval of eating a normal diet. Miller and Phillips (1955, as cited in ACGIH 1986/Ex. 1-3, p. 534) noted that rats conditioned to a dietary level of 20 ppm were then able to adjust to a level of 40 ppm (a dose that is greater than the single LD_{50} dose per day). The comment from NIOSH (Ex. 8-47) was the only one made to the record on sodium fluoroacetate.

In the final rule, OSHA is retaining the 8-hour TWA of 0.05 mg/m^3 and adding a STEL of 0.15 mg/m^3 for sodium fluoroacetate; the skin notation is also retained. The Agency concludes that the 8-hour TWA and short-term exposure limits, with a skin notation, will reduce the risk of systemic effects possible as a result of short-term exposures above the 8-hour TWA of 0.05 mg/m^3.

TRIMETHYLBENZENE

CAS: 25551-13-7; Chemical Formula: $(CH_3)_3C_6H_3$
H.S. No. 1412

OSHA formerly had no exposure limit for trimethylbenzene. The ACGIH TLV for all isomers of trimethylbenzene is 25

ppm as an 8-hour TWA. The proposed PEL was 25 ppm as an 8-hour TWA; NIOSH (Ex. 8–47, Table N1) concurred with the 25-ppm TWA limit, and the final rule establishes this limit for this liquid.

A study by Battig, Grandjean, and Turrian (1957/Ex. 1–104) provides the basis for the final rule's limit; this work reports symptoms among 27 workers exposed to a solvent containing 30 percent 1,3,5-trimethylbenzene and 50 percent 1,2,3-trimethylbenzene. A "significant number" of these workers were reported to have experienced symptoms of nervousness, tension and anxiety, and asthmatic bronchitis. The peripheral blood of these workers "showed a tendency to hypochromic anemia" and a somewhat abnormal clotting ability. This group of workers had been occupationally exposed to total hydrocarbon concentrations ranging from 10 to 60 ppm for several years. The authors of the study recommended maintaining employee exposures below 35 ppm (Battig, Grandjean, and Turrian 1957/Ex. 1–104). No comments other than that from NIOSH (Ex. 8–47) were received on this substance.

In the final rule, OSHA is establishing a 25-ppm 8-hour PEL to reduce the significant risks of bronchitis and blood effects reported to occur in exposed workers.

TUNGSTEN AND COMPOUNDS (INSOLUBLE)

CAS: 7440–33–7; Chemical Formula: W
H.S. No. 1416

Previously, OSHA had no exposure limits for insoluble tungsten and its compounds. The ACGIH has established 5 mg/m^3 as an 8-hour TWA and 10mg/m^3 as a short-term exposure limit for these substances. NIOSH recommends a limit of 5 mg/m^3 as a 10-hour TWA. The proposed PEL for this group of substances was 5 mg/m^3 as an 8-hour TWA and 10 mg/m^3 as a 15-minute STEL. NIOSH (Ex. 8–47) concurred with OSHA's proposed limits. The final rule promulgates a 5-mg/m^3 8-hour TWA and a 10-mg/m^3 5-minute STEL, measured as tungsten. Tungsten is a gray, hard metal.

Rats fed a diet containing 0.5 percent insoluble tungsten compounds died, and another group of rats fed 0.1 percent of these compounds suffered noticeable weight loss (Kinard and Van de Erve 1941/Ex. 1–492). Studies in rats fed tungsten at 2, 5, or 10 percent of their diet showed that females in all dose groups had a 15-percent reduction in weight gain (Kinard and Van de Erve 1943/Ex. 1–493). The intraperitoneal LD_{50} for tungsten metal powder in rats was 5 g/kg body weight; survivors showed minor liver and spleen changes at necropsy (Fredrick and Bradley 1946, as cited in ACGIH 1986/Ex. 1–3, p. 614). Studies of the tissues of guinea pigs intratracheally injected with tungsten metal and tungsten carbide revealed moderate interstitial cellular proliferation and no changes, respectively. However, Soviet studies involving similar intratracheal injections showed proliferation of the intra-alveolar septa (Kaplun and Mezentseva 1960, as cited in ACGIH 1986/Ex. 1–3, p. 614). The NIOSH criteria document for tungsten (1977h, as cited in ACGIH 1986/Ex. 1–3, p. 614) reports that Russian investigators found a 9- to 11-percent incidence of pulmonary fibrosis in workers exposed to tungsten (Kaplun and Mezentseva 1959/Ex. 1–961; and Mezentseva 1967, as cited in ACGIH 1986/Ex. 1–3, p. 614). NIOSH (1977h) recommended that the standard for tungsten and its insoluble compounds be set at 5 mg/m^3 to protect against pulmonary effects.

Stokinger (in *Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., Vol. 2A, Clayton and Clayton 1981) reported on several epidemiological studies of workers in the "hard metal industry," in which tungsten carbide is machined. These studies describe a condition known as hard metal disease, which may be accompanied by pulmonary fibrosis. The disease is characterized by a moderate incidence of cough, dyspnea, and wheezing, a high incidence of minor radiological abnormalities with a few instances of marked abnormalities, and development of hypersensitivity asthma in some workers (which may be due to exposure to the cobalt that is used as a binding agent). The disease is progressive and potentially lethal. Stokinger (in *Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., Vol. 2A, Clayton and Clayton 1981, p. 1992) reported that, unlike other lung diseases produced by inorganic dust, there is no correlation between onset of symptoms, length of exposure, and the development of interstitial fibrosis. Analysis of the lung of one worker who had clinical signs and radiological changes showed the presence of large amounts of tungsten with much smaller amounts of other metals.

Mr. H.K. Thompson, Corporate Industrial Hygiene Manager for Caterpillar, Inc. (Ex. 3–349), questioned the need for a STEL for tungsten. OSHA believes that, given the potential seriousness of hard metal disease and the uncertainties regarding the relationship between exposure and response, a short-term limit for tungsten will provide additional assurance that the 8-hour TWA PEL is not exceeded. Therefore, in accordance with OSHA's policy for establishing STELs in this rulemaking (see Section IV.C.17, OSHA finds that a STEL for tungsten is necessary.

In the final rule, OSHA is establishing an 8-hour TWA of 5 mg/m^3 and a STEL of 10 mg/m^3 for tungsten and its insoluble compounds, measured as tungsten. The Agency concludes that these limits will substantially reduce the significant risk of pulmonary fibrosis and other lung effects, which constitute material impairments of health that are associated with exposure to this metal and its insoluble compounds at levels above the new PELs.

TUNGSTEN AND COMPOUNDS (SOLUBLE)

CAS: 7440–33–7; Chemical Formula: W
H.S. No. 1417

OSHA had no former limit for exposure to tungsten and its soluble compounds. The ACGIH limit is 1 mg/m^3 TWA, with a 3-mg/m^3 STEL, measured as tungsten. NIOSH recommends a 1–mg/m^3 10-hour TWA for tungsten and its soluble compounds. OSHA proposed an 8-hour TWA PEL of 1 mg/m^3 and a 15-minute STEL of 3 mg/m^3; NIOSH (Ex. 8–47, Table N1) concurred with the addition of a STEL to the 1-mg/m^3 TWA limit. The final rule establishes limits of 1 mg/m^3 as an 8-hour TWA and 3 mg/m^3 as a 15-minute STEL, measured as tungsten. Tungsten is a grey, hard metal.

Animal studies have shown that the LD_{50} for soluble sodium tungstate when injected subcutaneously in rats ranges from 140 to 160 mg/kg (Kinard and Van de Erve 1940/Ex. 1–788). Soluble tungsten's lethal effects are the result of systemic poisoning that occurs as the compound is absorbed by multiple organs; this is followed by cellular asphyxiation (International Labour Office [ILO] 1934c, as cited in ACGIH 1986/Ex. 1–3, p. 614). Karantassis (1924, as cited in ACGIH 1986/Ex. 1–3, p. 614) also observed a systemic response in guinea pigs given soluble sodium tungstate or pure soluble tungsten either orally or intravenously; the animals developed anorexia, colic, trembling, and difficulty in breathing prior to death. Rats fed a diet containing 0.5 percent tungsten as soluble sodium tungstate or tungsten oxide died from this dose. Dietary doses of 0.1 percent tungsten oxide and the sodium salt caused weight loss in rats, but no deaths (Kinard and Van de Erve 1941/Ex. 1–492). Tungsten is believed to act by antagonizing the action of molybdenum (Higgins, Richert, and Westerfield 1956/Ex. 1–487). In its criteria document for

tungsten (1977h, as cited in ACGIH 1986/Ex. 1-3, p. 614), NIOSH states that information on the effects of exposure to soluble tungsten compounds in the working population is not available. The ACGIH (1986/Ex. 1-3, p. 614) recommends a lower TLV for the soluble, as compared to the insoluble, compounds of tungsten because of the former's greater systemic toxicity. No comments other than those of NIOSH (Ex. 8-47) were received on this substance.

In the final rule, OSHA is establishing an 8-hour TWA of 1 mg/m³ and a STEL of 3 mg/m³ for tungsten and its soluble compounds, measured as tungsten. The Agency concludes that these limits will protect workers against the significant risks of systemic toxicity, anorexia, colic, incoordination, trembling, and dyspnea, all of which constitute material health impairments that are associated with exposure to these compounds at levels above the new PELs.

VINYLIDENE CHLORIDE (1,1-DICHLOROETHYLENE)

CAS: 75-35-4; Chemical Formula: $CH_2 = CCl_2$ H.S. No. 1428

Previously, OSHA's Z tables did not include a limit for vinylidene chloride (VDC). The ACGIH has established 5 ppm as an 8-hour TWA and 20 ppm as a 15-minute STEL. NIOSH and OSHA, in 1978, jointly recommended that employee exposure to VDC be reduced to the lowest feasible level on the basis of VDC's carcinogenicity (NIOSH/OSHA 1978/Ex. 1-1119). OSHA proposed a PEL of 5 ppm (8-hour TWA) and a STEL of 20 ppm. However, in response to record comments, the final rule promulgates a 1-ppm limit as an 8-hour TWA. Vinylidene chloride is a colorless liquid that polymerizes readily.

The acute oral LD_{50} for male rats is 2500 mg/kg (Jenkins, Trabulus, and Murphy 1972/Ex. 1-960). The LC_{50} for rats exposed to a single four-hour exposure of VDC vapor was reported as 6350 ppm in one study (Siegel, Jones, Coon, and Lyon 1971/Ex. 1-371) and 32,000 ppm in an earlier study (Carpenter, Smyth, and Pozzani 1949/Ex. 1-722). Liquid VDC causes transient irritation to the eyes of rats but has little effect on exposed skin if the VDC is allowed to evaporate (Torkelson and Rowe 1981b, as cited in ACGIH 1986/Ex. 1-3, p. 628).

Prendergast and co-workers (1967/Ex. 1-926) exposed rats, rabbits, guinea pigs, and monkeys eight hours/day, five days/week for six weeks to 395 mg/m³ (100 ppm); these authors saw no visible signs of toxicity while the exposure was in process, but rabbits and monkeys lost weight. These same species were exposed continuously to VDC concentrations of 5, 15, 25, or 47 ppm for 90 days; only the animals exposed to 5 ppm showed no increases in mortality (Prendergast, Jones, Jenkins, and Siegel 1967/Ex. 1-926).

Nasal irritation, liver cell degeneration, and retarded weight gain were reported in rats following 20 six-hour exposures to 500 ppm VDC (Gage 1970/Ex. 1-318); at 200 ppm, only nasal irritation occurred. Studies by Torkelson and Rowe (1981b, as cited in ACGIH 1986/Ex. 1-3, p. 628) in which rats, rabbits, guinea pigs, and dogs were exposed to 25, 50, or 100 ppm VDC for eight hours per day, five days per week for six months revealed injury of the kidneys and liver in all animals at all levels of exposure. Maltoni (1977/Ex. 1-985) and Maltoni, Cotti, Morisi, and Chieco (1977/Ex. 1-1090) conducted an evaluation of VCD's carcinogenicity in which mice, rats and hamsters were exposed to levels from 10 to 150 ppm for four hours per day, five days per week for 52 weeks, with results reported through week 98 of the study. In those mice exposed to 25 ppm VCD, 21 percent of the males and 1.5 percent of the females developed kidney adenocarcinomas; these tumors were not seen in rats exposed to amounts of VDC up to 150 ppm. Exposures of 100 or 150 ppm in rats did produce a significant increase in mammary adenocarcinomas, and this response was dose-related (Maltoni 1977/Ex. 1-985; Maltoni, Cotti, Morisi, and Chieco 1977/Ex. 1-1090). Overt toxicity and mortality occurred early in the studies after four-hour exposures at levels of 50 ppm in mice and 200 ppm in rats; hamsters exposed to 20 ppm VDC showed no increase in tumor incidence (Maltoni 1977/Ex. 1-985; Maltoni, Cotti, Morisi, and Chieco 1977/Ex. 1-1090).

A study by Murray, Nitschke, Rampy, and Schwetz (1979/Ex. 1-920) investigated the embryotoxic, fetotoxic, and teratogenic effects of inhaled and ingested VDC (in rats) and inhaled VDC (in rabbits). In the inhalation studies, rats were exposed to 20, 80, or 160 ppm VDC for seven hours per day. VDC was toxic to both the adults and their embryos at levels of 80 and 160 ppm among the rats, and at 160 ppm in rabbits. At exposure levels of 20 ppm in rats and 80 ppm in rabbits, neither maternal toxicity nor effects on embryonic or fetal development were noted. In the ingestion study with rats, drinking water containing 200 ppm VDC caused no toxic effects in either the rats or their offspring.

Two strains of rats exposed to 75 or 100 ppm VDC for five days/week, six hours/day for 12 months did not show a significant increase in tumors (Viola and Caputo 1977/Ex. 1-937). Other investigators exposed rats to 25 or 75 ppm by inhalation for six hours/day, five days/week for 18 months, or to 60, 100, or 200 ppm VDC in their drinking water for two years, and found no increase in tumor incidence in these animals (Rampy, Quast, Humiston et al. 1977, as cited in ACGIH 1986/Ex. 1-3, p. 628). In mice, VDC was not active either as a whole mouse skin carcinogen or by subcutaneous injection.

In other studies, VDC proved mutagenic in both *E. coli* and *S. typhimurium* strains (Greim, Bonse, Radwan et al. 1975/Ex. 1-904; Bartsch, Malaveille, Montesano, and Tomatis 1975/Ex. 1-889). VDC has been implicated as a tumor initiator in a carcinogenesis bioassay by Van Duuren, Goldschmidt, Loewengart et al. (1979/Ex. 1-936). Studies by Reitz, Watanabe, McKenna et al. (1980/Ex. 1-927) suggest that VCD's tumorigenicity is a result of its ability to initiate cell injury, rather than of its ability to alter the genetic material of an injured cell. However, VDC has been shown to alkylate DNA *in situ* and increase the rate of DNA repair to a small extent in mice (Norris and Reitz 1984/Ex. 134B). The actual cell injury is caused by VDC metabolites, which are highly reactive and cytotoxic (Maltoni 1977/Ex. 1-985; Hathway 1977/Ex. 1-906; Henschler and Bonse 1977/Ex. 1-908).

A cohort study of 138 VCD-exposed workers did not identify any VCD-related health effects in these workers (Ott, Fishbeck, Townsend, and Schneider 1976/Ex. 1-924). The cohort was too small to provide any evidence that VDC is not likely to be carcinogenic.

The Chemical Manufacturers Association submitted the results of an NTP gavage study of VDC in mice and rats (NTP 1982/Ex. 134B). The only observed significant increase in tumor incidence occurred in low-dose female mice; this increase was not considered to be related to VDC administration because similar effects were not observed in high-dose female mice, male mice, or rats. The NTP (1982/Ex. 134B) concluded that VDC was not carcinogenic in mice or rats exposed by gavage, but cautioned that a maximum tolerated dose had not been demonstrated and that previously reported studies had shown that carcinogenicity is associated with VDC inhalation by animals.

Based on the carcinogenicity evidence described above, NIOSH (Ex. 8-47, Table N6B) indicated that VDC is a suitable candidate for an individual 6(b)

rulemaking. However, the CMA (Ex. 165) was of the opposite opinion, stating that the demonstrated lack of tumor response in most studies, coupled with evidence that VDC metabolism is species-specific, "demonstrates that VDC is unlikely to pose an oncogenic risk to humans" (Ex. 165, p. 42). CMA also objected to the statement by NIOSH and OSHA in the joint *Current Intelligence Bulletin on VDC* (NIOSH/OSHA 1978/Ex. 1–1119) that VDC be considered a potential carcinogen because of its structural similarity to vinyl chloride; the CMA considered this statement inappropriate, given the toxicity data available.

Matthew Gillen and Scott Schneider of the Workers Institute for Safety and Health (WISH) commented that the proposed 5-ppm PEL and 20-ppm STEL for VDC would not provide sufficient protection from systemic effects (Ex. 116). They pointed out that the study by Prendergast et al. (1967/Ex. 1–926) found 15 ppm to be the lowest effect level for increased mortality in animals, and that the Torkelson and Rowe (1981b, as cited in ACGIH 1986/Ex. 1–3, p. 628) study found liver and kidney injury in animals. These commenters stated that the "ACGIH TLV cannot be considered to provide adequate protection for this substance. Given this fact, OSHA should consider the NIOSH REL of 1 ppm as an interim value until further risk assessment studies can be carried out" (Ex. 116).

OSHA has re-examined the health evidence in light of the comment by WISH, and has determined that the proposed 5-ppm TWA PEL for VDC does not afford workers sufficient protection from systemic effects. Although it is questionable, in the Prendergast et al. (1967/Ex. 1–926) study, that the observed deaths at lower exposure levels were compound-related, histopathologic examination of animals exposed to 47 ppm showed treatment-related liver and kidney damage. Using an exposure regimen similar to occupational exposure (i.e., eight hours/day, five days/week), Torkelson and Rowe (1981b, as cited in ACGIH 1986/Ex. 1–3, p. 628) demonstrated kidney and liver toxicity in four species of animals after exposure to VDC levels as low as 25 ppm were administered for only six months.

OSHA believes that these studies clearly demonstrate that VDC can cause adverse liver and kidney damage at airborne concentrations as low as 25 to 50 ppm and suggest that VDC is a potential occupational carcinogen. Liver and kidney damage and cancer clearly constitute material health impairments within the meaning of the Act. Therefore, OSHA concludes that the proposed limits of 5 ppm as an 8-hour TWA and 20 ppm as a STEL will not sufficiently protect workers from the significant risk of organ damage, and that a further reduction in the PEL is warranted. Accordingly, OSHA is establishing a 1-ppm 8-hour TWA limit for vinylidene chloride in the final rule.

WELDING FUMES

CAS: None; Chemical Formula: Not available
H.S. No. 1430

OSHA formerly had no limit for exposure to welding fumes, which are defined as fumes that are generated by the manual metal arc or oxy-acetylene welding of iron, mild steel, or aluminum. The ACGIH has set an 8-hour TWA of 5 mg/m^3 for these welding fumes, measured as total particulate in the welder's breathing zone. OSHA proposed an 8-hour TWA of 5 mg/m^3 for these fumes; this limit is established in the final rule. This limit applies to the total fume concentration generated during the welding of iron, mild steel, or aluminum; the fumes generated by the welding of stainless steel, cadmium, or lead-coated steel, or other metals such as copper, nickel, or chrome are considerably more toxic and should be kept at or below the levels required by their respective PELs. Welding fumes consist of metallic oxides generated by the heating of metal being welded, the welding rod, or its coatings.

Although these types of welding generally produce fumes consisting of aluminum, iron, or zinc oxides, other toxic gases may also be produced in large amounts (Ferry and Ginther 1952/Ex. 1–900; Ferry 1954/Ex. 1–782; Silverman 1956/Ex. 1–1169; Homer and Mohr 1957/Ex. 1–787). The welding of iron metals may give off fumes of manganese, silicate, and various organic binders. Aluminum welding may generate fumes consisting of fluorine, arsenic, copper, silicon, and beryllium (NIOSH 1975h and American Welding Society 1974, both as cited in ACGIH 1986/Ex. 1–3, p. 634). Eighteen different substances, including fluoride, manganese, silicon, titanium, and sodium and potassium silicates, have been measured in the fumes resulting from the welding of mild steel (ACGIH 1986/Ex. 1–3, p. 634).

Excessive exposure to welding fume can cause a variety of disorders, most notably metal fume fever. It has been estimated that 30 to 40 percent of all welders have experienced metal fume fever at some time (Abraham 1983, in *Environmental and Occupational Medicine*, W.N. Rom, ed., p. 146). This disorder, which results from exposure to freshly formed metal fume, results in the appearance of delayed, flu-like symptoms, including dyspnea, coughing, pains in muscles and joints, fever, and chills. Recovery usually requires one or two days of time away from work. In addition to fume fever, exposure to welding fume may damage the small airways, causing interstitial pneumonia (Abraham 1983).

Several commenters, the American Iron and Steel Institute (Exs. 129, 188), the Abbott Laboratories (Tr. 9–155 to 9–156), and the American Welding Society (Ex. 3–860), were of the opinion that OSHA's discussion of welding fumes in the NPRM was not clear with regard to whether the limit applied to exposure samples taken inside or outside of the welding helmet. OSHA wishes to clarify that welding fume is to be measured in the breathing zone of the welder; the specific details of the appropriate positioning of the sampler should be determined on the basis of guidance in the *Field Operations Manual* (OSHA 1984). This is consistent with a past OSH Review Commission decision (8 OSHRC 1049).

NIOSH (Ex. 8–47) stated at the hearing that welding fumes should be designated as a carcinogen. This view was also endorsed by Dr. James Melium, of the New York State Department of Health (Tr. p. 11–104). In response to these commenters, OSHA notes that there are few data sufficient to establish a-dose-response for the fumes. Accordingly, OSHA believes it would be premature to identify these fumes as potential occupational carcinogens.

OSHA concludes that a PEL for welding fumes is needed to protect workers involved in the welding of aluminum, iron, or mild steel from the significant risk of metal fume fever and respiratory irritation associated with the generation of welding fumes. In the final rule, OSHA is establishing a TWA of 5 mg/m^3 for these particular types of welding fumes, measured as total particulate inside the welder's breathing zone. The Agency finds that this limit will substantially reduce the significant risk of material health impairment to which manual metal arc or oxy-acetylene welders of iron, mild steel, or aluminum were previously exposed in the absence of any OSHA limit.

ZINC OXIDE (FUME)

CAS: 1314–13–2; Chemical Formula: ZnO
H.S. No. 1437

OSHA's former exposure limit for zinc oxide fume was 5 mg/m^3 as an 8-hour TWA. The ACGIH recommends a 5-mg/m^3 TWA and also has a STEL of 10 mg/m^3. NIOSH recommends a 5-mg/m^3 10-

hour TWA limit with a 15-minute ceiling of 15 mg/m³. OSHA proposed to retain the 5-mg/m³ 8-hour TWA and to add a STEL of 10 mg/m³, and NIOSH (Ex. 8–47, Table Nl) concurs with this proposal. The final rule establishes these limits. When heated, zinc oxide produces a white fume.

The most prevalent toxic effect of zinc oxide fume is a condition known as "metal fume fever," whose symptoms include chills, fever, muscular pain, nausea, and vomiting (Turner and Thompson 1926/Ex. 1–1124). Studies in the workplace have shown that welders exposed to zinc oxide fume at concentrations of 320 to 580 mg/m³ reported nausea, with the development of chills, shortness of breath, and severe chest pains 2 to 12 hours later. Most workers took approximately 4 days to recover, and some eventually developed pneumonia (Hammond 1944/Ex. 1–981). Other studies have reported the frequent occurrence of chills in workers exposed to zinc oxide at levels as low as 5 mg/m³ (Hickish 1963 and Wall 1970, both as cited in ACGIH 1986/Ex. 1–3, p. 645). Hammond (1944/Ex. 1–981) reported that workers exposed to 8 to 12 mg/m³ of zinc oxide fume did not suffer from metal fume fever.

Zinc oxide exposures of guinea pigs that lasted only an hour caused a drop in body temperature, followed 6 to 18 hours later by an increase above normal levels (Turner and Thompson 1926/Ex. 1–1124). The animals in the high-exposure group (2500 mg/m³ for three to four hours) died after exposure.

Early studies (Drinker, Thomson, and Finn 1927/Ex. 1–356) suggested that metal fume fever was unlikely to occur at concentrations below 15 mg/m³, but subsequent experience shows that exposures even at 5 mg/m³ can cause this syndrome (Hickish 1963 and Wall 1970, both as cited in ACGIH 1986/Ex. 1–3, p. 646).

NIOSH's criteria document (1975d, as cited in ACGIH 1986/Ex. 1–3, p. 645) reported that the development of metal fume fever was unlikely at levels as low as 5 mg/m³, but the Institute stated that exposures to the fume at this level could cause chronic respiratory effects. Dr. Lawrence Hecker, representing Abbott Laboratories (Ex. 3–678), objected to a STEL for zinc oxide fume. However, in both its criteria document (1975d) and post-hearing testimony (Ex. 150, Comments on Zinc Oxide Fume), NIOSH indicated that a short-term limit is necessary to "prevent pathological tissue changes in the lung from acute exposure." Therefore, OSHA finds that a STEL for zinc oxide fume is necessary to prevent or minimize these effects.

In the final rule, OSHA is retaining the 5-mg/m³ 8-hour TWA and adding a STEL of 10 mg/m³. The Agency concludes that both of these limits will protect workers from the significant risk of metal fume fever, which constitutes a material health impairment that is associated with acute and chronic exposure to zinc oxide fumes.

ZIRCONIUM COMPOUNDS

CAS: 7440–67–7; Chemical Formula: Zr
H.S. No. 1439

The former OSHA limit for zirconium compounds was an 8-hour TWA of 5 mg/m³, measured as zirconium. The ACGIH has established a TLV–TWA of 5 mg/m³, supplemented by a 10-mg/m³ STEL, (as Zr). The proposal retained the 8-hour TWA but added a STEL of 10 mg/m³; these limits are promulgated by the final rule. Zirconium compounds may be either bluish-black powders or grayish-white lustrous metals.

The toxic effects of inhalation exposures to zirconium compounds include the formation of granulomas, both in the lungs and on the skin. Sax (*Dangerous Properties of Industrial Materials*, 6th ed., 1984) reports cases of pulmonary granulomas in workers exposed to zirconium aerosols. In laboratory animals, oral toxicity is low (NIOSH 1972b, as cited in ACGIH 1986/Ex. 1–3, p. 647), and inhalation studies conducted for one year at levels of 3.5 mg zirconium/m³ dust and mist resulted in limited toxicity (Stokinger 1981c/Ex. 1–1134).

NIOSH (Ex. 8–47) recommended that zirconium tetrachloride should not be included among the compounds for which the proposed zirconium PEL is applied. NIOSH cites an animal study by Spiegl et al. (1956, as cited in ACGIH 1986/Ex. 1–3, p. 647), in which a 60-day exposure to zirconium tetrachloride at a concentration of 6 mg/m³ (six hours/day, five days/week) resulted in increased mortality in rats and guinea pigs and a decrease "of borderline significance" in blood hemoglobin and red blood cell levels in dogs. Given that the observed effect level for mortality of 6 mg/m³ is close to the proposed 5-mg/m³ limit, NIOSH (Ex. 8–47) stated that a separate PEL should be considered for zirconium tetrachloride.

At this time, OSHA is establishing the PELs as proposed for all zirconium compounds, including zirconium tetrachloride. There are no reports, other than the one cited by NIOSH, that indicate that exposure to zirconium compounds causes severe toxicity at levels near the proposed 5-mg/m³ TWA PEL; in addition, the toxic reaction of dogs exposed to 6 mg/m was of borderline significance.

OSHA concludes that the 5-mg/m³ TWA and 10-mg/m³ STEL limits for the zirconium compounds, measured as zirconium, will protect workers from the significant risk of pulmonary effects potentially associated with the short-term exposures permitted by the 8-hour TWA alone. The Agency has determined that these effects constitute material health impairments.

Conclusions for This Group of Systemic Toxicants

For the group of systemic toxicants shown on Table C8–1, OSHA concludes that the risks associated with occupational exposures are significant. As Table C8–2 shows, the systemic effects caused by such exposures include cancer, liver and kidney damage, testicular damage, fetal poisoning, central nervous system depression, and asthma, each of which constitutes material impairment of health within the meaning of the Act. Affected employees may experience dizziness, nausea, generalized weakness, respiratory irritation, blood in the urine, chest tightness, hives, and necrosis of the cornea. These effects represent significant impairments of health and functional capacity, and reducing the limits for these systemic toxins will substantially reduce these significant risks.

9. Substances for Which Limits Are Based on No-Observed Adverse-Effect Levels

Introduction

For a group of 23 toxic substances, OSHA is establishing limits based on evidence that these substances cause toxic responses at higher levels but have been shown not to produce adverse effects in animals or exposed populations at the permissible exposure limits being established. These substances are shown in Table C9–1, along with their CAS numbers, H.S. numbers, and former, proposed, and final rule limits. OSHA is establishing limits for 17 chemicals in this group that have not formerly been regulated by the Agency. The Agency is retaining its 8-hour TWA PEL and adding a STEL for two substances, reducing the 8-hour TWA and adding a STEL in the case of uranium (insoluble compounds), reducing the 8-hour TWA for one substance (petroleum distillates), and retaining the existing 8-hour TWA for two chemicals.

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Table C9-1. Substances for Which Limits Are Based On A No-Observed-Adverse-Effect Level

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1029 Atrazine	1912-24-9	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1041 Bromacil	314-40-9	--	1 ppm TWA	1 ppm TWA
1056 p-tert-Butyltoluene	98-51-1	10 ppm TWA	10 ppm TWA 20 ppm STEL	10 ppm TWA 20 ppm STEL
1085 Chlorodifluoromethane	75-45-6	--	1000 ppm TWA 1250 ppm STEL	1000 ppm TWA
1090 o-Chlorotoluene	95-49-8	--	50 ppm TWA 75 ppm STEL	50 ppm TWA
1110 Cyclonite	121-82-4	--	1.5 mg/m^3 TWA 3 mg/m^3 STEL, Skin	1.5 mg/m^3 TWA, Skin
1117 2,6-Di-tert-butyl-p-cresol	128-37 0	--	10 mg/m^3 TWA	10 mg/m^3 TWA
1134 Diethanolamine	111-42 2	--	3 ppm TWA	3 ppm TWA
1136 Diethyl phthalate	84-66 2	--	5 mg/m^3 TWA	5 mg/m^3 TWA

Table C9-1. Substances for Which Limits Are Based On A No-Observed-Adverse-Effect Level (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1144 Dinitolmide	148-01 6	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1147 Diphenylamine	122-39-4	--	10 mg/m^3 TWA	10 mg/m^3 TWA
1153 Diuron	330-54-1	--	10 mg/m^3 TWA	10 mg/m^3 TWA
1249 Methyl acetate	79-20-9	200 ppm TWA	200 ppm TWA 250 ppm STEL	200 ppm TWA 250 ppm STEL
1275 Metribuzin	21087-64-9	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1297 Oil mist (mineral)	8012-95-1	5 mg/m^3 TWA	5 mg/m^3 TWA 10 mg/m^3 STEL	5 mg/m^3 TWA
1312 Petroleum distillates (naphtha)	8002-05-9	500 ppm TWA	400 ppm TWA	400 ppm TWA
1327 m-Phthalodinitrile	626-17 5	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1332 Platinum, metal	7440-06-4	--	1 mg/m^3 TWA	1 mg/m^3 TWA
1346 Resorcinol	108-46-3	--	10 ppm TWA 20 ppm STEL	10 ppm TWA 20 ppm STEL

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Table C9-1. Substances for Which Limits Are Based On A No-Observed-Adverse-Effect Level (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1382 Tantalum, metal dust and oxide	7440-25-7	5 mg/m^3 TWA	5 mg/m^3 TWA 10 mg/m^3 STEL	5 mg/m^3 TWA
1410 Trimethyl phosphite	121-45-9	--	2 ppm TWA	2 ppm TWA
1415 Triphenyl amine	603-34-9	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1418 Uranium (insoluble compounds)	7440-61-1	0.25 mg/m^3 TWA	0.2 mg/m^3 TWA 0.6 mg/m^3 STEL	0.2 mg/m^3 TWA 0.6 mg/m^3 STEL

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

Description of the Health Effects

The substances included in this group cause a wide range of adverse health effects in both animals and humans. Unlike most of the other groupings described in this preamble, these toxicants do not affect the same target organ or system: some are central nervous system depressants, several are upper respiratory tract irritants, and still others have their primary effect on the heart, liver, and/or kidney.

The commonality among these otherwise diverse substances is that apparent no-observed-adverse-effect levels (NOAELs) have been defined for all of them; that is, there are data demonstrating that overt toxic effects caused by exposure to these substances at higher levels do not occur below a certain "no-observed-adverse-effect" level. Permissible exposure limits have been developed for these chemicals on the basis of these "no-observed-adverse-effect" levels. Table C9-2 shows the health effects observed in animals and observed or likely to occur in humans exposed to these substances.

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TABLE C9-2. Health Effects Associated With Substances for Which Limits are Based on No-Observed-Adverse-Effect Levels

H.S. Number/ Chemical Name	CAS No.	Health Effects Observed in Animals	Health Effects Observed/ Projected in Humans
1029 Atrazine	1912-24-9	Ataxia, dyspnea, convulsions	Systemic effects
1041 Bromacil	314-40-9	Irritation, thyroid damage	Thyroid effects, irritation
1056 p-tert-Butyl-toluene	98-51-1	CNS depression, respiratory tract irritation, liver and kidney changes	Nasal irritation, nausea, headache, weakness
1085 Chlorodifluoromethane	75-45-6	Cardiac sensitization	CNS effects, cardiac sensitization
1090 o-Chlorotoluene	95-49-8	Weakness, vasodilation, incoordination, convulsions, irritation	Neuropathic effects, irritation
1110 Cyclonite	121-82-4	Death	Nausea, vomiting, convulsions, unconsciousness, death
1117 2,6-Di-tert-butyl-p-cresol	128-37-0	Growth rate decrease, increase in liver weight	Systemic effects
1134 Diethanolamine	111-42-2	Impaired vision, skin irritation	Visual effects, irritation
1136 Diethyl phthalate	84-66-2	Polyneuritis, disturbance of balance	Pain, numbness, transient irritation, polyneuritis
1144 Dinitolmide	148-01-6	Liver changes	Hepatic effects

TABLE C9-2. Health Effects Associated With Substances for Which Limits are Based on No-Observed-Adverse-Effect Levels (continued)

H.S. Number/ Chemical Name	CAS No.	Health Effects Observed in Animals	Health Effects Observed/ Projected in Humans
1147 Diphenylamine	122-39-4	Liver, kidney, spleen changes	Tachycardia, bladder symptoms, hypertension, eczema
1153 Diuron	330-54-1	Anemia, methemoglobinemia	Anemia, methemoglobinemia
1249 Methyl acetate	79-20-9	—	Eye, mucous membrane irritation, chest tightness, narcosis, destruction of optic nerve
1275 Metribuzin	21087-64-9	CNS depression, thyroid and liver changes	Neuropathic effects, thyroid and liver damage
1297 Oil mist (mineral)	8012-95-1	Lung irritation	Lung irritation, pneumonitis, scrotal and skin cancer
1312 Petroleum distillates (naphtha)	8030-30-6	Motor incoordination, convulsions	Neuropathic effects, eye, throat irritation
1327 m-Phthalodinitrile	626-17-5	Skin irritation	Irritation, systemic effects
1332 Platinum, metal	7440-06-4	Tumorigen by implantation	—
1346 Resorcinol	108-46-3	Eye, skin irritation; mutagenicity; hemolytic effects	Irritation, systemic effects (methemoglobinemia, cyanosis)

TABLE C9-2. Health Effects Associated With Substances for Which Limits are Based on No-Observed-Adverse-Effect Levels (continued)

H.S. Number/ Chemical Name	CAS No.	Health Effects Observed in Animals	Health Effects Observed/ Projected in Humans
1382 Tantalum, metal dust and oxide	7440-25-7	Bronchitis, pneumonitis, hyperemia	Pulmonary effects
1410 Trimethyl phosphite	121-45-9	Teratogenicity, ocular irritation	Lung, skin, eye irritation, reproductive effects
1415 Triphenyl amine	603-34-9	Skin irritation	Irritation
1418 Uranium (insoluble compounds)	7440-61-1	Kidney damage, blood disorders	Kidney damage, blood effects

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Dose-Response Relationships and No-Observed-Adverse-Effect Levels

The concept of setting limits based on a NOAE level assumes that there is a concentration at which repeated and prolonged exposure to a toxic substance causes no observable adverse effect in the majority of workers. A similar concept is widely used by a variety of Federal agencies, for example the Food and Drug Administration and the Environmental Protection Agency, to set contaminant tolerances, acceptable daily intake values, and other limits.

All of the limits for these substances have been set at a no-observed-adverse-effect or minimal effect level, regardless of the specific health endpoint being protected against. At least in part, the exposure limits for the 23 substances listed in Table C9–1 are based on data indicating that these limits are already being maintained in work environments and that these levels are both feasible and not likely to be associated with adverse health effects or symptoms in employees. These limits will also protect against any effects these substances cause at higher concentrations. Even at relatively low exposure concentrations, many of the substances in this group cause effects that can be severe and irreversible.

The following discussions describe OSHA's findings and the record evidence for the substances in this group and illustrate the material impairments of health faced by workers exposed to these toxicants in the workplace.

ATRAZINE

CAS: 1912–24–9: Chemical Formula: $C_8H_{14}ClN_5$
H.S. No. 1029

Formerly, OSHA had no limit for atrazine; an 8-hour TWA of mg/m³ was proposed. The final rule establishes this limit, which is consistent with that of the ACGIH. NIOSH (Ex. 8–47, Table N1) concurs with this limit for atrazine. Atrazine is a stable, white, crystalline compound.

Animal studies indicate that the oral toxicity of the s-triazine herbicides, of which atrazine is the best known, is relatively low. However, the ingestion of high doses can cause ataxia, dyspnea and convulsions in animals (ACGIH 1986/Ex. 1–3, p. 44). Rats, dogs, horses, or cattle fed dietary levels of more than 25 ppm of atrazine for extended periods did not exhibit adverse effects. The s-triazine herbicides are apparently excreted in urine and feces within relatively short periods of time (Bakke, Larson, and Price 1972/Ex. 1–950). The s-triazines appear to interfere with carbohydrate metabolism by blocking the production of sugars (Gysin 1962/Ex. 1–740; Gast 1958, as cited in ACGIH 1986/Ex. 1–3, p. 44).

There have not been reports of atrazine poisoning in exposed people (ACGIH 1986/Ex. 1–3, p. 44). Because there are no reports of human reactions to atrazine that can be correlated with airborne concentrations, the 5-mg/m³ limit was set on the basis of animal studies. Long-term feeding studies in dogs have established 3.75 mg/kg as the highest no-adverse-effect level (EPA 1979, as cited in ACGIH 1986/Ex. 1–3, p. 44). Assuming that lung absorption is less than 50 percent and applying a safety factor would yield an 8-hour TWA limit for humans of 5 mg/m³ (Zielhuis and van der Kreek 1979/Ex. 1–613).

Wayne Bellinger, Corporate Safety Director of ConAgra, Inc., objected to the establishment of permissible exposure limits on the basis of a "no-adverse-effect" level (Ex. 3–635). In support of this position, ConAgra referred to the proposed limit for atrazine; according to ConAgra, PELs should not be set "where there are no reports of human reactions that can be attributed to air concentrations" (Ex. 3–635, p. 2).

OSHA believes that ConAgra has misunderstood the phrase "no-observed-adverse-effect level" as it is used in toxicology. As discussed in the Description of the Health Effects section, above, this term simply means a level below which overt toxic effects have not been observed and above which they have. The use of a no-observed-adverse-effect level to establish "acceptable" exposure levels, intake values, etc. is common, both in the health effects literature and in public health agencies; this approach is widely used with substances that have threshold effects. In addition, it is standard toxicological practice to rely on animal data when human data are sparse or nonexistent, as is the case for atrazine. OSHA has reviewed the health effects evidence for this substance and finds the proposed rule's limit both appropriate and necessary to protect against significant workplace risk.

In the final rule, OSHA is establishing an 8-hour TWA PEL of 5 mg/m³ for atrazine. The Agency concludes that this limit will protect employees from the significant risk of neuropathic and metabolic effects, which constitute material health impairments that are likely to occur at levels above the new PEL.

BROMACIL

CAS: 314–40–9; Chemical Formula: $C_9H_{13}BrN_2O_2$
H.S. No. 1041

OSHA had no former permissible exposure limit for bromacil. The Agency proposed an 8-hour TWA PEL of 1 ppm for this substance, which is consistent with the ACGIH's TLV–TWA for bromacil. The final rule establishes a PEL of 1 ppm for this substance: NIOSH concurs with OSHA's determination of a PEL for bromacil (Ex. 8–47, Table N1). Bromacil is a white crystalline solid.

In two-year feeding studies in rats, no-observed-adverse-effect dietary concentrations were determined to be 12.55 mg/kg/day (1.25 ppm) for rats and 1250 ppm for dogs; the oral LD_{50} for male rats is 5200 mg/kg (Sherman and Kaplan 1975/Ex. 1–572). Inhalation studies in rats have shown that all rats tolerate a four-hour exposure to concentrations equivalent to 4800 mg/m³. Studies of guinea pigs showed no skin sensitization but displayed skin irritation after exposures at unspecified levels. Rabbits showed no clinical signs of toxicity when bromacil was applied to the skin at a dose of 5000 mg/kg (ACGIH 1986/Ex. 1–3, p. 64).

OSHA received a comment on the proposed limit for bromacil from ConAgra, Inc. (Ex. 3–635), which is of the opinion that bromacil's exposure effects do not warrant the establishment of a PEL. OSHA notes, however, that rats fed 1250 ppm (125 mg/kg/day) exhibited damage to the thyroid; the Agency finds that this evidence requires the establishment of a PEL at the 1-ppm level to protect workers exposed to this herbicide from experiencing this and other potentially adverse systemic effects.

OSHA is therefore establishing an 8-hour TWA permissible exposure limit for 1 ppm for bromacil. The Agency concludes that this limit protect employees against the significant risk of thyroid damage and irritation, which together constitute material health impairments that are potentially associated with exposure to bromacil at levels above the new PEL.

p-tert-BUTYLTOLUENE

CAS: 98–51–1; Chemical Formula: $(CH_3)_3C—C_6H_4CH_3$
H.S. No. 1056

OSHA formerly had an 8-hour TWA PEL of 10 ppm TWA for p-tert-butyltoluene; the Agency proposed to retain this limit and to supplement it with a 20-ppm STEL. The ACGIH has a TLV–TWA of 10 ppm and a TLV–STEL of 20 ppm for this substance. The final rule adopts a STEL of 20 ppm to supplement OSHA's 10-ppm 8-hour TWA PEL for p-tert-butyltoluene. NIOSH (Ex. 8–47, Table N1) concurs with the selection of these limits. p-tert-Butyltoluene is a

colorless liquid with an aromatic, gasoline-like odor.

p-tert-Butyltoluene has been shown to have varying degrees of toxicity, depending on route of administration. It is slightly toxic on ingestion, and minimally toxic through skin exposure, but moderately toxic when inhaled (Hine, Ungar, Anderson et al. 1954/Ex. 1–983). Repeated exposures in animals have shown liver and kidney changes and microscopic degenerative hemorrhages in the spinal cord and brain, even at relatively low concentrations. The chief acute effects in animals are central nervous system depression and respiratory irritation; in rats exposed for one to seven hours daily over a 26-week period, 25 ppm daily appeared to be the no-observed-adverse-effect level (Gerarde 1960a, as cited in ACGIH 1986/Ex. 1–3, p. 85).

In humans, Hine, Ungar, Anderson et al. (1954/Ex. 1–983) observed nasal irritation, nausea, malaise, headache, and weakness associated with exposure to p-tert-butyltoluene at unspecified levels. These authors also noted cardiovascular effects, as well as effects on the central nervous system, the skin, and the respiratory tract. Half of the subjects exposed to p-tert-butyltoluene developed tremor and anxiety, and 25 percent of exposed individuals showed evidence of chemical contact irritation of the respiratory tract (Hine, Ungar, Anderson et al. 1954/Ex. 1–983).

OSHA is retaining its 8-hour TWA of 10 ppm and adding a STEL of 20 ppm for p-tert-butyltoluene. The Agency concludes that a STEL as well as a TWA will protect workers against the significant risks of central nervous and cardiovascular system effects, as well as those of irritation and nausea, all of which constitute material impairments to health that are potentially associated with short-term (one to seven hours) exposures to this substance at levels above the 8-hour TWA.

CHLORODIFLUOROMETHANE

CAS: 75–45–6: Chemical Formula: $CHClF_2$ H.S. No. 1085

OSHA formerly had no limit for chlorodifluoromethane (Freon 22). The Agency proposed an 8-hour TWA PEL of 1000 ppm, supplemented by a STEL of 1250 ppm. NIOSH (Ex. 8–47, Table N1) supported this proposal. The ACGIH has a TLV–TWA of 1000 ppm for this substance. In the final rule, OSHA is establishing an 8-hour TWA for chlorodifluoromethane of 1000 ppm; the Agency has decided not to establish a STEL for chlorodifluoromethane (see Section VI.C.17) for a discussion of OSHA's rationale with regard to STELs in this rulemaking).

Chlorodifluoromethane is a colorless, nearly odorless, nonflammable gas.

Exposure to very high atmospheric levels of Freon 22 causes stimulation and then depression of the central nervous system, followed by asphyxiation. Rats and guinea pigs exposed to concentrations of 75,000 to 100,000 ppm over a two-hour period exhibited excitation and disequilibrium; narcosis was observed at 200,000 ppm and mortality at 300,000 and 400,000 ppm (Weigand 1971/Ex. 1–1102). In mice, similar exposures to 320,000 ppm were the maximum tolerated, and the minimum lethal dose was 370,000 ppm (Karpov 1963, as cited in ACGIH 1986/Ex. 1–3, p. 127). In rabbits, the minimum concentration altering reflex responses was 11,000 to 20,000 ppm (Karpov 1963, as cited in ACGIH 1986/Ex. 1–3, p. 127). Studies of guinea pigs reported no fatalities as a result of exposure for two hours at 200,000 ppm, but mild clinical changes were observed at 50,000 ppm and minimal effects at 25,000 ppm (Underwriters' Laboratories, Inc. 1940, as cited in ACGIH 1986/Ex. 1/3, p. 127). Thirty-minute exposures at 500,000 ppm were lethal to guinea pigs (Booth and Bixby 1932/Ex. 1–1079). Karpov (1963, as cited in ACGIH 1986/Ex. 1–3, p. 127) also reported the results of a 10-month study of inhalation effects in rats, guinea pigs, dogs, and cats. Six-hour inhalation exposures to 14,000 ppm or 2000 ppm for five days/week were studied, and alterations in weight, endurance, blood chemistry, and pathology of the lungs, central nervous system, heart, liver, kidney, and spleen were seen at the 14,000-ppm level in rats, mice, and rabbits. At the 2000-ppm daily inhalation level, rats and mice showed no effects (Karpov 1963, as cited in ACGIH 1986/Ex. 1–3, p. 127). In dogs, cardiac sensitization was not observed at the 25,000-ppm level but did occur at the 50,000-ppm level (Reinhardt, Azar, Maxfield, Smith, and Mullin 1971/Ex. 1–78). No data have been published concerning the carcinogenicity, mutagenicity, or teratogenicity of this substance. OSHA received a few comments on Freon 22, from NIOSH (Ex. 8–47, Table N1), the American Industrial Hygiene Assocation, the du Pont Company (Ex. 3–660), and Dr. Grace Ziem (Ex. 46). du Pont and the AIHA stated that OSHA should not adopt limits (short-term, ceiling, or skin notations) for substances for which the ACGIH has deleted, or is on record as intending to delete, such limits (Exs. 8–16, 3–600). Discussions of OSHA's policy on STELs and skin notations in this rulemaking can be found in Sections VI.C.17 and VI.C.18, respectively. OSHA notes that cardiac sensitization does not occur in animals until levels reach 25 to 50 times the 1000–TWA limit and, therefore, a STEL that is 1.25 times that limit is unwarranted. Dr. Ziem (Ex. 46) reported that Freon 22's effects on heart rhythm have been seen at the 300-ppm level.

The Agency is establishing an 8-hour TWA limit of 1000 ppm for chlorodifluoromethane. OSHA concludes that this limit will provide protection against the CNS effects, asphyxiant effects, and cardiac sensitization effects (which together constitute material health impairments) that could occur as the result of exposure to Freon 22 at levels above the new PEL. The Agency finds that the new limit will substantially reduce these significant risks.

o–CHLOROTOLUENE

CAS: 95–49–8; Chemical Formula: C_7H_7Cl H.S. No. 1090

Formerly, OSHA had no limit for o-chlorotoluene. The Agency proposed an 8-hour TWA of 50 ppm (consistent with the ACGIH's TLV–TWA) and a 75-ppm, STEL for this substance, a colorless liquid. NIOSH (Ex. 8–47, Table N1) supported this proposal. In the final rule, OSHA is establishing a 50-ppm 8-hour TWA for o-chlorotoluene. The Agency has decided not to establish a STEL (see Section VI.C.17 for a discussion of OSHA's rationale in regard to STELs for this rulemaking).

The oral LD_{50} in rats for o-chlorotoluene is greater than 1600 mg/kg. When the undiluted material was administered orally in doses ranging from 50 to 100 mg/kg, the animals experienced weakness and vasodilation at the higher dose levels, but all survived and were gaining weight two weeks later (ACGIH 1986/Ex. 1–3, p. 137). When the undiluted liquid was applied to the skin of guinea pigs in doses of 1 ml. or 10 ml/kg for 24 hours, moderately severe skin irritation occurred at both dose levels. The guinea pigs lost weight over the two-week period following application, indicating percutaneous absorption of this substance; however, no dermal LD_{50} has been established for o-chlorotoluene. One drop of undiluted material in the eyes of rabbits produced a delayed erythema of the conjunctiva, although this effect cleared after 14 days (Ely 1971, as cited in ACGIH 1986/Ex. 1–3, p. 137). Rats exposed to an atmosphere of 21 mg/L (or about 4000 ppm) for six hours exhibited loss of coordination within 1.5 hours, prostration at 1.75 hours, and tremors at 2 hours. At 14,000 ppm, rats showed loss of coordination, vasodilation, labored respiration,

narcosis, and eye tearing. Rats exposed at 4000 and 14,000 ppm survived. At 175,000 ppm, one of three rats died (Ely 1971, as cited in ACGIH 1986/Ex. 1-3, p. 137). In another study, mice, rats, and quinea pigs were exposed to o-chlorotoluene at a concentration of about 4400 ppm. Mice showed gasping and convulsions within 30 minutes, and guinea pigs and rats exhibited gasping, hyperpnea, ataxia, and convulsions in 45 minutes. All animals were comatose within 60 minutes, and, except for two guinea pigs that continued to survive at 14 days, all of the animals died (Hazleton Laboratories, Inc. 1966, as cited in ACGIH 1986/Ex. 1-3 p. 137).

In rabbits, the 24-hour patch test resulted in moderate skin irritation; albino rabbits displayed conjunctival irritation from a single instillation of 0.1 ml of undiluted o-chlorotoluene, but no corneal damage was observed seven days later (Hazleton Laboratories, Inc. 1966, as cited in ACGIH 1986/Ex. 1-3, p. 137).

Data concerning human exposures are lacking; no cases of dermatitis or poisoning have been reported as a result of occupational exposure. Personal communications from several occupational health experts have recommended limits for o-chlorotoluene ranging from 25 to 200 ppm TWA (Hopton 1962, Mastromatteo 1971, Elkins 1972, Torkelson 1972, all as cited in ACGIH 1986/Ex. 1-3, p. 137). These limits were recommended on the basis of analogy with similar compounds, such as the chlorinated benzenes. OSHA received comments on o-chlorotoluene from NIOSH (Ex. 8-47, Table N1), the du Pont Company (Ex. 3-660), and the American Industrial Hygiene Association (Ex. 8-16). du Pont and the AIHA stated that OSHA should not adopt limits (short-term, ceiling, or skin notations) for substances for which the ACGIH has dropped or is on record as intending to drop such limits (Exs. 8-16 and 3-660). OSHA agrees with this view in many cases (see Sections VI.C.17 and VI.C.18 for discussions of OSHA's policy on STELs and skin notations in this rulemaking).

OSHA is establishing an 8-hour TWA PEL of 50 ppm for o-chlorotoluene. The Agency concludes that this limit will protect workers from the significant risks of eye and skin irritation and systemic poisoning, all material impairments of health that may occur following exposure to this substance at levels above the new PEL.

CYCLONITE

CAS: 121-82-4; Chemical Formula: $C_3H_6N_6O_6$
H.S. No. 1110

OSHA has not previously had a permissible exposure limit for cyclonite. The Agency proposed an 8-hour TWA of 1.5 mg/m^3, a STEL of 3 mg/m^3, and a skin notation, and NIOSH (Ex. 8-47, Table N1) concurred with the selection of these PELs. The ACGIH has an 8-hour TWA limit of 1.5 mg/m^3 for this substance. In the final rule, OSHA is establishing a 1.5-mg/m^3 8-hour TWA PEL for cyclonite, with a skin notation; the Agency has decided not to establish a STEL for this substance (see Section VI.C.17 for a discussion of OSHA's rationale in regard to STELs). Cyclonite exists in the form of orthorhombic crystals.

Cyclonite, an explosive and a rat poison, has not been shown in animal studies to be acutely toxic. In industry, reports of poisonings as a result of occupational exposures to cyclonite were widespread as late as 1962 (Kaplan, Berghout, and Peczenik 1965/Ex. 1-338). Exposure causes central nervous system effects, including nausea, vomiting, convulsions, and unconsciousness. These clinical signs result from repeated gastrointestinal and respiratory exposures and from skin absorption (Sunderman et al. 1944, as cited in ACGIH 1986/Ex. 1-3, p. 162; von Oettingen, Donahue, Yagoda et al. 1949/Ex. 1-398). In an epidemiological study, Hathaway and Buck (1977/Ex. 1-418) reported that 8-hour TWA exposures ranging up to 1.57 mg/m^3 and averaging 0.28 mg/m^3 caused no identifiable abnormalities attributable to cyclonite exposure. The American Industrial Hygiene Association (Ex. 8-16) urged OSHA to drop the STEL for this substance.

OSHA is establishing an 8-hour TWA limit of 1.5 mg/m^3 TWA and a skin notation for cyclonite. The Agency concludes that establishing these limits for this previously unregulated chemical will protect workers from the significant risk of neuropathic effects, which constitute material health impairments that are associated with inhalation or percutaneous exposure to cyclonite.

2,6-Di-tert-BUTYL-p-CRESOL

CAS: 128-37-0; Chemical Formula: $C_{15}H_{24}O$
H.S. No. 1117

OSHA previously had no limit for 2,6-di-tert-butyl-p-cresol (DBPD). The Agency proposed an 8-hour TWA of 10 mg/m^3 for DBPD, and this limit is adopted in the final rule. NIOSH (Ex. 8-47, Table N1) concurred with the selection of this limit. The ACGIH has a TLV-TWA of 10 mg/m^3 for this white crystalline compound, which is prepared from p-cresol and isobutylene. DBPD is widely used as a food preservative.

DBPD has a low order of toxicity; in extensive animal studies, ingestion has not been associated with toxic effects (ACGIH 1986/Ex. 1-3, p. 227). Deichmann and associates (1955/Ex. 1-505) reported oral LD_{50} values of 10.7 g/kg for guinea pigs, 1.7 and 1.97 g/kg for male and female rats, respectively, and ranges of between 0.94 and 2.1 g/kg for cats and between 2.1 and 3.2 g/kg for rabbits. One year of daily oral administration of 0.17 to 0.9 g/kg in dogs produced no effects, nor did a 24-month oral administration of 0.2, 0.5, or 0.8 percent DBPD in rats (Deichmann, Clemmer, Rakoczy, Bianchine et al. 1955/Ex. 1-505). Other studies have confirmed these overall results, although some growth rate decreases and liver weight increases were demonstrated in rats fed 0.01 to 0.5 percent DBPD, total daily diet (Brown, Johnson, and O'Halloran 1959/Ex. 1-621; Creaven, Davies, and Williams 1966/Ex. 1-547).

The estimated human intake of DBPD in the United States does not exceed a few milligrams daily (perhaps no more than 0.2 mg/kg body weight) (Gilbert and Golberg 1965/Ex. 1-902). These authors also observed that the no-effect dietary level for DBPD in rats is 25 mg/kg.

OSHA is establishing an 8-hour TWA limit of 10 mg/m^3 for 2,6-di-tert-butyl-p-cresol. The Agency concludes that this limit will protect workers against the significant risk of material health impairments in the form of acute or chronic effects that may potentially be associated with occupational exposure to this substance at the levels permitted by the absence of any OSHA PEL.

DIETHANOLAMINE

CAS: 111-422-2; Chemical Formula: $HO(CH_2)_2NH(CH_2)_2OH$
H.S. No. 1134

OSHA formerly had no limit for diethanolamine. The proposed limit was 3 ppm (8-hour TWA), and this is also the limit adopted in the final rule. NIOSH (Ex. 8-47, Table N1) concurred with the selection of this limit. The ACGIH has established an 8-hour TWA limit of 3 ppm for this substance. Diethanolamine exists as a solid or a liquid at room temperature.

The oral LD_{50} of diethanolamine for both rats and guinea pigs has been reported to be about 2 g/kg (Dow Chemical Company 1977g, as cited in ACGIH 1986/Ex. 1-3, p. 197). Acute toxicity studies have shown that direct contact may impair vision and denature the skin if exposure is repeated. Dietary studies in rats showed no ill effects after 90 days of feeding at 20 mg/kg/day (Smyth, Carpenter, and Weil 1951/Ex. 1-

439). NIOSH (Ex. 8–47, Table N1) was the only commenter on this substance.

OSHA is establishing an 8-hour PEL of 3 ppm TWA for diethanolamine. The Agency concludes that this limit will protect workers from the significant risks of eye damage and skin irritation, material health impairments that are associated with exposure to diethanolamine at levels above the new PEL.

DIETHYL PHTHALATE

CAS: 84–66–2; Chemical Formula: C_6H_4—$(COOC_2H_5)_2$
H.S. No. 1136

OSHA had no previous limit for diethyl phthalate. The proposed rule contained an 8-hour TWA exposure limit for this substance of 5 mg/m³, and this limit is adopted in the final rule. NIOSH (Ex. 8–47, Table N1) supported the Agency's determination. The ACGIH has a TLV–TWA of 5 mg/m³ for this stable, colorless, odorless, oily liquid with a bitter taste.

Diethyl phthalate exposure may cause polyneuritis and disturbance in vestibular function. By most routes of administration, this substance has low acute toxicity in laboratory animals. Oral LD_{50} values in the rat range between 9.5 and 31 g/kg (Shibko and Blumenthal 1973/Ex. 1–934); the intraperitoneal LD_{50} for the rat is 5.08 ml/kg (Singh, Lawrence, and Autian 1972/Ex. 1–436) and, for the mouse, 2.8 g/kg (Calley, Autian, and Guess 1966/Ex. 1–890). Chronic feeding studies lasting six or more weeks resulted in no-effect levels of 2.5 g/kg/day for the rat and 1.25 g/kg/day for the dog, with no specific lesions attributable to diethyl phthalate and no unusual incidence of tumors (Shibko and Blumenthal 1973/Ex. 1–934).

A study of workers exposed to a mixture of diethyl phthalate, dibutyl phthalate, and di-2-ethyl hexyl phthalate vapors in air at concentrations of 8 to 53 mg/m³ resulted in findings of no phthalates in the blood (before or after the exposure) and no peripheral polyneuritis (Raleigh, personal communication, as cited in ACGIH 1986/Ex. 1–3, p. 200). Fassett (1963a, as cited in ACGIH 1986/Ex. 1–3, p. 200) reported transient nasal and throat irritation produced by exposure to the heated vapors of diethyl phthalate, but no cumulative effects have been noted. A Russian study of workers (employed for between 0.5 and 19 years) who were exposed to several phthalate plasticizers (e.g., butyl phthalate, the higher aryl phthalates, dioctyl phthalate, and benzyl butyl phthalate), as well as the sebacates, adipates, and tri-o-cresyl phosphate at concentrations ranging from 1.7 to 66 mg/m³ reported that there were complaints of pain, numbness, and spasms in the upper and lower extremities. These complaints were related to the duration of exposure and usually began after the sixth or seventh year of employment (Milkov, Aldyreva, Popova et al. 1973/Ex. 1–646). These investigators reported polyneuritis in 32 percent of the 47 persons examined for this health effect; of 81 persons evaluated for vestibular dysfunction, 78 percent showed depression of vestibular receptors (Milkov, Aldyreva, Popova et al. 1973/Ex. 1–646).

OSHA is establishing an 8-hour TWA PEL of 5 mg/m³ for diethyl phthalate. The Agency concludes that this limit will protect workers against the significant risks of polyneuritis and vestibular dysfunction, which constitute material health impairments that are potentially associated with occupational exposure to this substance at levels above the new PEL.

DINITOLMIDE (3,5–DINITRO–O–TOLUAMIDE)

CAS: 148–01–6; Chemical Formula: $C_8H_7N_3O_5$
H.S. No. 1144

OSHA previously had no limit for dinitolmide. The proposed 8-hour TWA PEL was 5 mg/m³, and the final rule adopts this limit. NIOSH (Ex. 8–47, Table N1) agreed with the selection of this PEL. The ACGIH has an 8-hour limit of 5 mg/m³ TWA for this yellowish solid.

In rats, the oral LD_{50} for males is 560 mg/kg, and for females, 650 mg/kg; the ACGIH (1986/Ex. 1–3, p. 213) concludes that dinitolmide has moderate oral toxicity in rats. Two-year dietary studies of rats fed 62.5 ppm (or 3 mg/kg/day) dinitolmide reported no ill effects. Rats of both sexes fed 6 mg/kg/day showed slight fatty changes in the liver; female rats also exhibited slight liver weight increases. Dogs fed 10 mg/kg/day showed no effects after one year. A three-generational study of rats fed 3 or 6 mg/kg/day revealed no effects on fertility, gestation, viability, or lactation (Dow Chemical Company 1973e, as cited in ACGIH 1986/Ex. 1–3, p. 213). There are no inhalation data for dinitolmide.

OSHA is establishing an 8-hour TWA PEL for dinitolmide of 5 mg/m³. The Agency concludes that this limit will protect workers from the significant risk of material impairment in the form of the hepatic changes that are potentially associated with exposure to this substance at levels above the new PEL.

DIPHENYLAMINE

CAS: 122–39–4; Chemical Formula: $(C_6H_5)_2NH$
H.S. No. 1147

OSHA formerly had no limit for diphenylamine. The proposed PEL was 10 mg/m³, as an 8-hour TWA, and the final rule adopts this limit; NIOSH (Ex. 8–47, Table N1) has indicated its concurrence. The ACGIH recommends a TLV of 10 mg/m³ TWA. Diphenylamine exists as monoclinic crystalline leaflets that discolor when exposed to light.

Acute oral toxicity data for diphenylamine are limited. A single report describes a study in which a dietary dose of 1500 mg/kg killed 2 of 20 rats within 30 days of ingestion (Griswold, Casey, Weisburger et al. 1966/Ex. 1–483). This suggests that diphenylamine is significantly less toxic than aniline (Hamblin 1963/Ex. 1–1085). Dietary studies of rats fed 0.025, 0.1, 0.5, 1.0, or 1.5 percent diphenylamine for 226 days demonstrated nonmalignant renal cysts at the three highest doses (Thomas, Cox, and Deeds 1957/Ex. 1–873). However, rats given diphenylamine crystals encapsulated in collodion developed bladder papillomas within 125 days (Yoshida, Shimauchi, and Kin 1941, as cited in ACGIH 1986/Ex. 1–3, p. 220). Exposure to diphenylamine dust has been linked to liver, spleen, and kidney changes in experimental animals (Robert, Dervilée, and Collet 1937/Ex. 1–928).

A report of industrial diphenylamine poisoning in France described bladder symptoms, tachycardia, hypertension, and eczema (Fairhall 1957g, as cited in ACGIH 1986/Ex. 1–3, p. 220).

OSHA is establishing an 8-hour PEL of 10 mg/m³ TWA for diphenylamine. The Agency concludes that this limit will protect workers against the significant risks of liver, kidney, cardiovascular, and other systemic effects, all of which constitute material health impairments that are potentially associated with exposures to this substance at levels above the new PEL.

DIURON

CAS: 330–54–1; Chemical Formula: $C_9H_{10}C1_2N_2O$
H.S. No. 1153

OSHA previously had no limit for diuron. The Agency proposed an 8-hour TWA PEL of 10 mg/m³ for diuron, and this limit is established by the final rule. NIOSH (Ex. 8–47), Table N1) agrees that this limit is appropriate. The ACGIH has a TLV of 10 mg/m³ TWA for this white crystalline solid.

Hodge and Associates (1967/Ex. 1–911; 1968/Ex. 1–912) have reported that diuron has a low order of acute and chronic toxicity. For male rats, the oral LD_{50} is 3400 mg/kg. In two-year feeding studies of rats and dogs, the no-effect levels were reported to be 250 and 125 ppm, respectively. A concentration of 125 ppm in the diet did not cause

reproductive or carcinogenic effects in a three-generational study of rats (Hodge, Downs, Panner et al. 1967/Ex. 1-911; Hodge, Downs, Smith et al. 1968/Ex. 1-912); 1400 ppm did not have carcinogenic effects in mice (Innes, Ulland, Valerio et al. 1969/Ex. 1-270). Skin irritation and sensitization test findings in guinea pigs have been negative (ACGIH 1986/Ex. 1-3, p. 228). However, repeated doses of this pre-emergence herbicide produced anemia in rats and methemoglobinemia after hydrolysis to dichloroaniline in the body (*Condensed Technical Information*, du Pont 1961).

OSHA is establishing an 8-hour TWA limit of 10 mg/m^3 for diuron. OSHA concludes that this limit will protect workers from the significant risks potentially associated with workplace exposure to this substance at the levels permitted in the absence of any OSHA PEL. These risks include anemia and methemoglobinemia, both of which constitute material impairments of health. The final rule's 10-mg/m^3 PEL will substantially reduce these risks.

METHYL ACETATE

CAS: 79-20-9; Chemical Formula: CH_3COOCH_3
H.S. No. 1249

OSHA's former 8-hour TWA limit for methyl acetate was 200 ppm; the Agency proposed to retain this limit and to add a STEL of 250 ppm. NIOSH (Ex. 8-47, Table N1) concurred with this proposal. The ACGIH has established an 8-hour TWA limit of 200 ppm and a TLV-STEL of 250 ppm. In the final rule, OSHA is establishing an 8-hour TWA of 200 ppm and a 15-minute STEL of 250 ppm. Methyl acetate is a highly volatile, colorless liquid with a pleasant odor.

Methyl acetate is mildly narcotic and is a known irritant to the mucous membranes of the eyes and respiratory passages. Occupational exposure to this substance by vapor inhalation at unreported levels resulted in inflammation of the eyes, nervous irritation, and a sensation of tightness in the chest (Duquenois and Revel 1934/Ex. 1-779; Fairhall 1957f, as cited in ACGIH 1986/Ex. 1-3, p. 367). Duquenois and Revel (1934/Ex. 1-779) suggested that, like methyl alcohol, methyl acetate may produce atrophy of the optic nerve.

Other researchers have suggested that the methanol formed by hydrolysis in the body may be responsible for the toxicity of methyl acetate and, on this basis, have recommended a limit of 250 ppm in the occupational setting (Henderson and Haggard 1943j, as cited in ACGIH 1986/Ex. 1-3, p. 367). However, Lehmann and Flury (1943d, as cited in ACGIH 1986/Ex. 1-3, p. 367) have attributed toxic effects (e.g., blood changes, weight loss, lung irritation), as well as some deaths, to chronic exposures to methyl acetate at 6600 ppm.

The ACGIH 1986/Ex. 1-3, p. 367) reports that "no cases of irritation or systemic injury have been reported from industrial exposures to methyl acetate below 200 ppm." There were no record comments on methyl acetate, except for the concurrence from NIOSH (Ex. 8-47, Table N1).

OSHA is establishing an 8-hour TWA PEL of 200 ppm TWA and a 15-minute STEL of 250 ppm for methyl acetate. The STEL is necessary to ensure that exposures do not exceed 250 ppm even for a short time because effects have been reported (ACGIH 1986/Ex. 1-3, p. 367) above 250 ppm. The Agency concludes that both of these limits will protect workers from the significant risk of narcosis, eye and skin irritation, and pulmonary irritation, all of which constitute material health impairments.

METRIBUZIN

CAS: 21087-64-9; Chemical Formula: $C_8H_{14}N_4OS$
H.S. No. 1275

OSHA has not formerly regulated exposure to metribuzin. The proposed PEL was 5 mg/m^3. NIOSH (Ex. 8-47, Table N1) concurred with the proposal, and the final rule adopts this limit. The ACGIH has recommended a TLV-TWA of 5 mg/m^3 for this substance. Metribuzin is a crystalline solid.

Metribuzin is a herbicide that has a low order of acute toxicity; single exposures to high concentrations produce central nervous system depression, and repeated high doses affect the thyroid and liver function (*Deutsche Forschungsgemeinschaft* 1981, as cited in ACGIH 1986/Ex. 1-3, p. 411). The oral LD_{50} in rats has been reported to be 2000 mg/kg; in cats and rabbits, the LD_{50} is as high as 500 mg/kg. A four-hour aerosol exposure at concentrations of between 860 and 892 mg/m^3 was tolerated by rats and mice; no skin or eye irritation was observed in rabbits. No sensitizing effects were seen in guinea pigs, and a skin application of the 70-percent wettable powder of 1000 mg/kg per day for three weeks produced no effects in rats (*Deutsche Forschungsgemeinschaft* 1981, as cited in ACGIH 1986/Ex. 1-3, p. 411).

Inhalation studies have shown no adverse effects in rats exposed to 31 mg/m^3 of the aerosol for six hours/day, five days/week during a three-week period (Bayer 1981, as cited in ACGIH 1986/Ex. 1-3, p. 411). No carcinogenic effects were observed in rats and mice fed 20. 800, or 3200 ppm for two years (Kimmerle 1982a, as cited in ACGIH 1986/Ex. 1-3, p. 411). A no-effect level of 100 ppm was observed in a two-year dietary study of rats and dogs (*Deutsche Forschungsgemeinschaft* 1981, as cited in ACGIH 1986/Ex. 1-3, p. 411); these same investigators observed no teratogenic, embryotoxic, or reproductive effects in rats or rabbits. In Chinese hamsters and mice, no mutagenic activity was observed (Siebert and Lemperle 1974/Ex. 1-689).

No human poisonings caused by metribuzin have been reported. In oral long-term studies, the highest no-observed-effect levels (NOELs) were 2.5 to 5 mg/kg per day (ACGIH 1986/Ex. 1-3, p. 411). Single and repeated patch tests in humans did not cause irritation or sensitization (*Deutsche Forschungsgemeinschaft* 1981, as cited in ACGIH 1986/Ex. 1-3, p. 411). Except for NIOSH's concurrence with this limit (Ex. 8-47, Table N1), no comments were received on metribuzin.

In the final rule, OSHA is establishing an 8-hour TWA PEL of 5 mg/m^3 TWA for metribuzin. The Agency concludes that this limit will protect workers against the significant risks of metabolic and central nervous system effects, which are material impairments of health that are potentially associated with workplace exposure to metribuzin at the levels permitted by the absence of any OSHA limit.

OIL MIST (MINERAL)

CAS: 8012-95-1; Chemical formula: None
H.S. No. 1297

OHSA formerly had a limit of 5 mg/m^3 as an 8-hour TWA for oil mist. The Agency proposed to retain mg/m^3 as an 8-hour TWA PEL and to add 10 mg/m^3 as a 15-minute STEL; however, the final rule retains the former 8-hour TWA but does not add a STEL. The ACGIH has a 5-mg/m^3 TLV-TWA limit and a 10-mg/m^3 TLV-STEL for oil mist (mineral), which refers to the airborne mist of petroleum-based cutting oils or of white petroleum oil; the odor of this substance is described as similar to that of burned lubrication oil.

Studies in animals have shown that repeated six-hour daily exposures to 5 mg/m^3 caused no adverse effects (Wagner, Wright, and Stokinger 1964, as cited in ACGIH 1986/Ex. 1-3, p. 449). At 100 mg/m^3, slight changes, including lung effects, were observed in exposed animals (Lushbaugh, Green, and Redemann 1950/Ex. 1-792). It has been suggested that heat-decomposed oil fumes are irritating to the lungs (Wagner, Dobrogorski, and Stokinger 1961/Ex. 1-773).

OHSA received a number of comments on the proposed STEL for oil mist (Exs. 3–829, 3–830, 3–856, 3–1115, 188, and 194; Tr. pp. 7–47 to 7–53). For example, William Fladung, Manager of Environmental Control for the Timken Company, believes that the limits for oil mist presented in the proposed rule are not justified by the evidence discussed in the preamble to the proposal (Ex. 3–856). According to this commenter, "the only health effect observed in animals is 'lung irritation.' No health effect has been observed in humans" (Ex. 3–856). This view was shared by representatives of the independent Lubricant Manufacturers Association (Ex. 3–830), the Specialty Steel Industry of the United States (Ex. 3–829), and the Anti-Friction Bearing Manufacturers Association (Ex. 3–1115).

In response to these comments, OSHA has reviewed the toxicological evidence for oil mist. Proctor, Hughes, and Fischman (*Chemical Hazards of the Workplace*, 2nd ed., 1988) report a single case of lipoid pneumonitis in a worker repeatedly exposed to high concentrations of oil mist, and these authors also note that some mineral oils (i.e., those containing additives and impurities) have been linked to cancers of the skin and scrotum. NIOSH submitted comments to the record noting that certain types of oils and/or their additives may present a carcinogenic hazard (Ex. 150, Comments on Oil Mist.) The United Auto Workers and the AFL–CIO (Tr. pp. 7–47 to 7–53) urged OSHA to adopt a lower PEL on the basis of oil mist's carcinogenic effects. According to the UAW, oil mist "has been known for many years . . . [to] cause skin cancer, particularly scrotal cancer among exposed workers" (Tr. p. 7–50). The UAW also believes that oil mist exposure increases the risk of primary malignancies of the respiratory and upper digestive systems (Tr. p. 7–50). However, OSHA believes that these carcinogenic effects may be attributable to contaminants in the oil, such as polycyclic aromatic hydrocarbons and certain additives. OSHA also notes that modern refining techniques have generally eliminated these hazardous substances from mineral oils.

After a review of the record evidence, OSHA fnds that the toxicological data on this substance do not support the addition of a STEL at this time. Accordingly, the final rule retains the 8-hour TWA PEL of 5 mg/m^3 but does not add a 15-minute STEL of 10 mg/m^3 for mineral oil mist. The Agency concludes that the existing 8-hour TWA limit will protect exposed employees against the significant risks of eye and respiratory tract irritation potentially associated with exposures to mineral oil mist. OSHA finds that these eye and lung effects constitute material impairments of health.

PETROLEUM DISTILLATES (NAPHTHA)

CAS: 8002–05–9; Chemical Formula: None
H.S. No. 1312

For petroleum distillates (naphtha), also identified as rubber solvent, OSHA proposed to reduce its former 8-hour limit of 500 ppm to 400 ppm. The final rule establishes an 8-hour TWA PEL of 400 ppm for petroleum distillates. The ACGIH has a TLV-TWA of 400 ppm, and NIOSH recommends a TWA of 87 ppm and a 15-minute ceiling of 450 ppm for these substances.

A study performed by Carpenter, Kinkead, Geary et al. (1975b/Ex. 1–53) exposed rats to between 2800 and 24,200 ppm of naphtha. Motor incoordination occurred at 5300 ppm, and convulsions and death occurred in all animals at 24,200 ppm. Animals exposed to 480 ppm for 63 days showed no signs of toxicity (Carpenter, Kinkead, Geary et al. 1975b/Ex. 1–53).

NIOSH (1977g, as cited in ACGIH 1986/Ex. 1–3, p. 516) noted that rubber solvent (naphtha) is composed primarily of C_5–C_8 alkanes and, thus, that the limit of 350 mg/m^3 (85 ppm) recommended for C_5–C_8 alkanes should apply to naphtha. This recommendation presumes that all C_5–C_8 alkanes possess equivalent neurotoxicity; however, as discussed above in Section V (Summary of Commenters' Responses to NPRM Questions), OSHA has concluded that not all of the C_5–C_8 alkanes are neuropathic agents.

In establishing the 400-ppm TLV-TWA for petroleum distillates, the ACGIH relied on observations showing that slight irritation occurs in humans exposed to 430 ppm and that no signs of toxicity occur in animals exposed to 480 ppm. The NIOSH-recommended 85-ppm ceiling limit is based on the assumption that all C_5–C_8 alkanes possess equivalent neuropathic properties. As discussed above, OSHA has rejected this hypothesis and is therefore reducing the PEL for petroleum distillates to an 8-hour TWA limit of 400 ppm to protect workers against the significant risk of irritation, which constitutes a material health impairment that is associated with exposure to these substances. OSHA has determined that this limit will substantially reduce this risk.

m-PHTHALODINITRILE

CAS: 626–17–5; Chemical Formula: $C_8H_4N_2$
H.S. No. 1327

OSHA has no previous limit for m-phthalodinitrile. The proposed PEL was 5 mg/m^3 as an 8-hour TWA, and this limit is established in the final rule. NIOSH (Ex. 8–47, Table N1) concurred with the selection of this PEL. The ACGIH has a TLV–TWA of 5 mg/m^3 for this substance. meta-Phthalodinitrile exists in the form of needles obtained from solutions containing either water or ligroin as the solvent.

In rabbits, slight skin reactions have been reported from dermal applications of m-phthalodinitrile to intact or abraded skin for six hours/day, five days/week over a three-week period. The doses applied were 0.5, 1.0, and 2.0 g/kg; at the two higher dose levels, some changes in organ (unspecified) size, without histopathologic changes, were observed. Female rabbits exposed at the highest dose lost weight (Owen 1972, as cited in ACGIH 1986/Ex. 1–3, p. 488).

A 15-year review of industrial experience revealed no reports of adverse effects from exposure to m-phthalodinitrile (Zeller, Hofmann, Thiess, and Hey 1963, as cited in ACGIH 1986/Ex. 1–3, p. 488). Williams (1959/Ex. 1–1176) attributes this absence of exposure effects to the fact that the aromatic nitriles, of which m-phthalodinitrile is one, do not liberate cyanide in the body, as is the case with the aliphatic nitriles. No comments other than NIOSH's were received by OSHA on this substance.

OSHA is establishing an 8-hour TWA limit for m-phthalodinitrile of 5 mg/m^3. The Agency concludes that this limit will protect exposed workers from the significant risk of skin irritation, a material health impairment that exists at m-phthalodinitrile levels about the new limit.

PLATINUM (METAL)

CAS: 7440–06–4; Chemical Formula: Pt
H.S. No. 1332

OSHA had no former limit for platinum metal. The proposed PEL was 1 mg/m^3, and this limit is established in the final rule. NIOSH (Ex. 8–47, Table N1) agreed that this PEL is appropriate. The ACGIH has a limit of 1.0 mg/m^3 TWA for platinum metal dust. Platinum is a silver-gray, lustrous, malleable, ductile precious metal.

Based on the TLV for platinum soluble salts and the absence of any severe health effects associated with exposure to the metal dust, the ACGIH recommended a TLV of 1.0 mg/m^3 for platinum metal dust. This limit reflects good industrial hygiene practice and acknowledges that heavy metal dusts are more toxic than nuisance dusts (which are controlled to 10 mg/m^3). No

comments (other than that of NIOSH) were received.

OSHA is establishing an 8-hour TWA limit of 1.0 mg/m³ for platinum metal. The Agency concludes that this limit will protect workers against the significant risk of adverse health effects potentially associated with workplace exposures to this substance at the levels permitted by the absence of any OSHA PEL.

RESORCINOL

CAS: 108-46-3; Chemical Formula: $C_6H_4(OH)_2$
H.S. No. 1346

OSHA formerly had no limit for resorcinol. The proposed limit was an 8-hour TWA of 10 ppm and a 15-minute STEL of 20 ppm; the final rule establishes these limits. NIOSH (Ex. 8-47, Table N1) supports the selection of these PELs. The ACGIH has an 8-hour TWA limit of 10 ppm and a TLV-STEL of 20 ppm. Resorcinol occurs in the form of sweet-tasting white crystals that may turn pink on exposure to air and light or on contact with iron.

Resorcinol has been reported to be less toxic by ingestion or skin penetration than either catechol or phenol (von Oettingen 1949 and Koppers Company 1974, both as cited in ACGIH 1986/Ex. 1-3, p. 511). The oral LD_{50} in rats is 301 mg/kg (NIOSH 1977i/Ex. 1-1182). Daily six-hour exposures at 8 ppm for two weeks produced no ill effects in rats, guinea pigs, and rabbits. Acute inhalation exposures to a resorcinol-water aerosol at concentrations as high as 7800 mg/m³ for one hour and 2800 mg/m³ for eight hours caused no toxic effects in laboratory animals (Koppers Company 1974, as cited in ACGIH 1986/Ex. 1-3, p. 511).

In humans, the cutaneous application of solutions or salves containing from 3 to 25 percent of this compound may result in local hyperemia, itching, dermatitis, edema, corrosion, and the loss of the superficial layers of the skin. If these damages are severe, they may be associated with some or all of the following effects: enlargement of regional lymph glands, restlessness, methemoglobinemia, cyanosis, convulsions, tachycardia, dyspnea, and death (*Patty's Industrial Hygiene and Toxicology*, Vol. 2A, p. 2588, Clayton and Clayton, 1981). An epidemiologic study of rubber workers exposed to a hexamethylene-tetramine-resorcinol rubber system revealed no specific symptoms caused by resorcinol; the concentrations in air were less than 0.3 mg/m³. In another study, there were no reports of irritation or discomfort by workers when concentrations were 10 ppm or less for periods of at least 30 minutes (*Patty's Industrial Hygiene and Toxicology*, Vol. 2A, p. 2588, Clayton and Clayton, 1981). Dr. Grace Ziem (Ex. 46) notes that resorcinol exposure is also associated with renal and hepatic effects and with methemoglobinemia.

In the final rule, OSHA is establishing a PEL of 10 ppm TWA and a STEL of 20 ppm for resorcinol. The Agency concludes that this combined limit will protect workers against the significant risks of irritation, methemoglobinemia, and other adverse effects, all material impairments of health that are associated with exposure to this substance at levels above the new PELs.

TANTALUM (METAL DUST AND OXIDE)

CAS: 7440-25-7; Chemical Formulas: (Tantalum metal)TA; (Tantalum oxide)Ta_2O_5
H.S. No. 1382

OSHA's former PEL for tantalum is 5 mg/m³. The Agency proposed to retain this limit and to supplement it with a 15-minute STEL of 10 mg/m³, and NIOSH (Ex. 8-47, Tale N1) concurred with this proposal. The final rule retains an 8-hour TWA for tantalum metal dust and oxide but does not adopt the proposed STEL for these substances (see Section XI.C.17 for a discussion of OSHA's rationale in regard to STELs). The ACGIH has a 5-mg/m³ TWA but has recently deleted its former 15-minute STEL of 10 mg/m³. Tantalum dust is a black powder and tantalum oxide is a white, microcrystalline powder.

Animal studies by Miller, Davis, Goldman, and Wyatts (1953/Ex. 1-40) have not implicated tantalum as a cause of pneumoconiosis, although an exposure to 100 mg tantalum oxide produced "soft white circumscribed pigmented dust lesions" in the lungs of these animals (ACGIH 1986/Ex. 1-3, p. 554). Additionally, this particular study demonstrated transient bronchitis, interstitial pneumonitis, and hyperemia at the 100-mg exposure level. Tantalum oxide has been used as a dressing for burns (Olsen 1944/Ex. 1-651), and the use of tantalum gauze in surgical repair produced no long-term adverse effects (Dales and Kyle 1958/Ex. 1-587). No adverse health effects have been associated with industrial exposures to tantalum or its compounds (Cochran, Doull, Mazur, and DuBois 1950/Ex. 1-586). A single oral dose of 6500 mg/kg oxide was virtually nontoxic to rats (ACGIH 1986/Ex. 1-3, p. 554).

OSHA concludes that the existing 5-mg/m³ TWA for these compounds should be retained to protect workers from the respiratory effects of exposure, which constitute material health impairments. The final rule retains the Agency's former PEL of 5 mg/m³ for tantalum (metal dust and oxide).

TRIMETHYL PHOSPHITE

CAS: 121-45-9; Chemical Formula: $(CH_3O)_3P$
H.S. No. 1410

OSHA previously had no limit for trimethyl phosphite. The proposed PEL was an 8-hour TWA of 2 ppm and NIOSH (Ex. 8-47, Table N1) supported this proposal. The final rule establishes this limit. The ACGIH limit for this substance is a 2-ppm 8-hour TWA. Trimethyl phosphite is a colorless liquid with a pungent odor.

Trimethyl phosphite's toxic effects include lung, skin, and eye irritation. In a chronic inhalation study of rats, Levin and Gabriel (1973/Ex. 1-746) found that exposure to trimethyl phosphite at concentrations of 500 ± 75 ppm for 7.5 hours daily, five days/week for eight weeks caused an adverse effect on body weight and, at necropsy, revealed evidence of severe pulmonary and cutaneous pathology. At exposures of 600 ppm for six hours/day, five days/week for four weeks, 70 percent of the rats died, and 10 percent of those exposed even at 300 ppm on the same regimen died (Mobil Oil Corporation 1979, as cited in ACGIH 1986/Ex. 1-3, p. 609).

Rats exposed at 100 ppm showed signs of eye irritation, and at 300 to 600 ppm, mild to severe cataracts developed. At doses of 164 mg/kg, trimethyl phosphite caused gross abnormalities in the offspring of treated rats (Mobil Oil Corporation 1979, as cited in ACGIH 1986/Ex. 1-3, p. 609). Skin contact with trimethyl phosphite produced severe skin irritation in rabbits, and instillation in the eyes of rabbits caused temporary swelling and irritation but no permanent effects (Fassett 1963c/Ex. 1-1148).

In a group of 179 workers exposed to average concentrations of trimethyl phosphite of between 0.3 and 4 ppm, no ocular changes were observed (Mobil Chemical Company 1980, as cited in ACGIH 1986/Ex. 1-3, p. 609).

OSHA is establishing an 8-hour TWA PEL of 2 ppm for trimethyl phosphite. The Agency concludes that this limit will protect workers against the significant risk of eye damage, skin irritation, and upper respiratory tract irritation, all of which constitute material health impairments that are potentially associated with exposures to this substance at levels above the new PEL.

TRIPHENYL AMINE

CAS: 603-34-9; Chemical Formula: $(C_6H_5)_3N$
H.S. No. 1415

OSHA formerly had no exposure limit for triphenyl amine. The proposed PEL was 5 mg/m³ as an 8-hour TWA, and the

final rule adopts this limit. NIOSH (Ex. 8–47, Table N1) agrees with the selection of this PEL. The ACGIH has a 5-mg/m³ 8-hour TWA limit for this substance. Triphenyl amine takes the form of colorless monoclinic prisms.

Animal studies conducted by the Eastman Kodak Company (Roudabush 1973, as cited in ACGIH 1986/Ex. 1–3, p. 612) showed an oral LD_{50} in rats of 3200 to 6400 mg/kg and an oral LD_{50} in mice of 1600 to 3200 mg/kg. The LD_{50} by intraperitoneal administration for both rodent species exceeded 6400 mg/kg. Skin and eye sensitivity tests in both rabbits and guinea pigs were essentially negative, except that application of 5 to 20 ml/kg occlusively for four hours produced slight erythema (Roudabush, 1973, as cited in ACGIH 1986/Ex. 1–3, p. 612).

OSHA is establishing a 5-mg/m³ TWA limit for triphenyl amine. The Agency concludes that this limit will protect workers against the significant risk of skin irritation, a material health impairment that is potentially associated with exposure to this substance at levels above the new PEL.

URANIUM (INSOLUBLE COMPOUNDS)

CAS: 7440–61–1; Chemical Formula: U
H.S. No. 1418

OSHA's former PEL for insoluble uranium compounds is 0.25 mg/m³. The proposed limits were 0.2 mg/m³ as an 8-hour TWA and 0.6 mg/m³ as a 15-minute STEL, based on the ACGIH recommendation. These limits are being established in the final rule. Uranium is a silver-white radioactive metal.

OSHA's former limit for the insoluble compounds of uranium was based on several early studies of uranium's toxic effects in animals; these effects included kidney damage and blood changes (Voegtlin and Hodge 1953, as cited in ACGIH 1986/Ex. 1–3, p. 617). In the intervening years, a considerable body of evidence has accumulated based on the actual occupational exposures of uranium plant workers over periods as long as 25 years. This evidence shows that, before 1950, workers were often exposed to uranium levels between 0.2 and 1.5 mg/m³, but that after 1950, only about 6 percent were exposed at 0.05 mg/m³ or above; despite these relatively high early exposures, the incidence of all diseases, whether or not linked to radiation exposure, has been no higher than is the case for workers in the general population (ACGIH 1986/Ex. 1–3, p. 617). However, there is also evidence that several workers were exposed to brief excursions during which exposure levels reached a concentration as much as five times the TLV (Wing, Heatherton, and Quigley 1963, as cited in ACGIH 1986/Ex. 1–3, p. 617).

NIOSH (Ex. 8–47, Table N6A) indicated that uranium compounds may present a carcinogenic hazard, but concurred with the proposed limits. OSHA has reviewed the scientific evidence on insoluble uranium compounds and notes the results of a five-year inhalation toxicity study of natural uranium dioxide (UO_2), which involved monkeys, dogs, and rats (Leach, Hodge, Wilson et al. 1970, as cited by H.E. Stokinger in *Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., Vol. 2A, p. 2002, Clayton and Clayton 1981). This study found that the two major sites of uranium accumulation, the lungs and tracheobronchial lymph nodes, accounted for over 90 percent of the uranium found in the body. Fibrotic changes suggestive of radiation injury were seen occasionally in the tracheobronchial lymph nodes of both dogs and monkeys, as well as in the lungs of monkeys after exposure periods longer than three years. The lung and lymph node data obtained in this study show that the animal body can accumulate sufficient uranium, from prolonged exposures to insoluble uranium dust at 5 mg/m³, to create potential radiological hazards. The lung and tracheobronchial lymph node radiation values were high enough, "in fact, to anticipate radiation hazards in these tissues from exposures at or lower than the occupational TLV (200 μg U/m³) recommended by the ACGIH . . ." (*Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., Vol. 2A, pp. 2002–2003, Clayton and Clayton 1981).

Laurence Hecker, representing Abbott Laboratories (Ex. 3–678), commented that there was no health basis for the proposed STEL for uranium. OSHA believes that the findings from the study discussed above illustrate the importance of maintaining employee TWA exposures at or below the 0.2-mg/m³ PEL. Therefore, in accordance with the policy described in Section VI.C.17, OSHA is establishing a STEL for insoluble uranium compounds to ensure that adequate process control is achieved to maintain exposure at or below the TWA PEL.

In the final rule, OSHA is establishing an 8-hour TWA PEL of 0.2 mg/m³ and a STEL of 0.6 mg/m³ for the insoluble forms of uranium. The Agency concludes that these limits are required to protect workers exposed to uranium from the significant risks of kidney or blood disorders and radiological damage potentially associated with both full-shift and excursion exposures to these compounds. The Agency considers these adverse effects material impairments of health. OSHA finds that these limits will substantially reduce these risks.

Conclusions for This Group of Substances

For the group of substances shown in Table C9–1, OSHA concludes that workplace exposures cause a broad range of adverse health consequences in exposed individuals; these effects include central nervous system depression, respiratory irritation, liver and kidney damage, cardiac sensitization, and hepatocellular cancer; OSHA considers all of these effects material impairments of health. For the substances in this group, few comments were received on the new or revised limits being proposed. In addition, NIOSH (Exs. 8–47 and 150) concurred with OSHA's proposed revisions in the great majority of cases. The Agency has determined, based on a thorough review of all of the evidence in the record, that the new or revised limits established in the final rule are necessary to reduce the significant risk of material health impairment associated with workplace exposures to systemic toxins.

10. Substances for Which Limits Are Based on Avoidance of Physical Irritation and Other Effects

Introduction

OSHA is establishing or revising the permissible exposure limits for a large group of substances that cause a variety of irritant and other adverse effects; in addition, the Agency is retaining its former generic limit of 15 mg/m³ total particulate [1] and its generic limit of 5 mg/m³ respirable particulate for several of the substances in this category. In the final rule, OSHA has separated this group of physical irritants into two groups, based on the evidence available on their toxic effects.

For 18 of these substances (one "Particulates Not Otherwise Regulated" which applies to all particulates not identified in Table Z–1–A), OSHA has retained the Agency's former 15-mg/m³ generic total particulate limit as an 8-hour TWA. Workers exposed to these 18 substances are subject to the physical-irritant effects traditionally associated with excessive particulate exposures in the workplace. These effects include eye irritation, interference with vision, upper-respiratory-tract irritation, and deposition of particulate in the eyes,

[1] Because the term particulate applies to dusts, aerosols, and mists, OSHA uses this term generically in this section to apply to all of these states of matter.

ears, nose, and mouth. OSHA believes that these effects may cause safety problems among exposed workers, who are more likely than nonexposed workers to have accidents or safety mishaps because they are distracted and physically irritated by the presence of these substances in the workplace. However, after a thorough analysis of the available literature on these 18 substances, OSHA has concluded that retention of the former total-particulate PEL of 15 mg/m^3 will provide protection against the exposure effects currently known to be associated with these substances. The 18 substances for which the former 15 mg/m^3 total particulate limit has been retained are shown in Table C10–1.

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TABLE C10-1. Substances for Which the Exposure Limit is Based on the Avoidance of Physical Irritation and Other Effects

H.S. Number/ Chemical Name	CAS No.	OSHA's Former Generic Total Particulate Limit*	Proposed PEL*	Final Rule PEL for Total Particulate**; Respirable Fraction
1014 alpha-Alumina	1344-28-1	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1016 Aluminum metal dust	7429-90-5	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1024 Ammonium sulfamate	7773-06-0	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1031 Barium sulfate	7727-43-7	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1032 Benomyl	17804-35-2	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1035 Bismuth telluride (undoped)	1304-82-1	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1039 Boron oxide	1303-86-2	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1057 Calcium carbonate	1317-65-3	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1061 Calcium silicate	1344-95-2	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1062 Calcium sulfate	7778-18-9	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1076 Cellulose	9004-34-6	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1082 2-Chloro-6-trichloro-methyl pyridine	1929-82-4	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA

TABLE C10-1. Substances for Which the Exposure Limit is Based on the Avoidance of Physical Irritation and Other Effects (continued)

H.S. Number/ Chemical Name	CAS No.	OSHA's Former Generic Total Particulate Limit*	Proposed PEL*	Final Rule PEL for Total Particulate**; Respirable Fraction
1095 Clopidol	2971-90-6	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1102 Crag herbicide (sesone)	136-78-7	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1133 Dicyclopentadienyl iron	102-54-5	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1155 Emery	112-62-9	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1176 Ferbam	14484-64-1	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1188 Glycerin (mist)	56-81-5	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1191A Graphite, synthetic	--	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1192 Gypsum	7778-18-9	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1230 Kaolin	—	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1232 Limestone	1317-65-3	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1233 Magnesite	546-93-0	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1234 Magnesium oxide fume	1309-48-4	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA

TABLE C10-1. Substances for Which the Exposure Limit is Based on the Avoidance of Physical Irritation and Other Effects (continued)

H.S. Number/ Chemical Name	CAS No.	OSHA's Former Generic Total Particulate Limit*	Proposed PEL*	Final Rule PEL for Total Particulate**; Respirable Fraction
1235 Malathion	121-75-5	15 mg/m^3 TWA, Skin	10 mg/m^3 TWA, Skin	10 mg/m^3 TWA; 5 mg/m^3 TWA, Skin
1239 Marble	1317-65-3	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1246 Methoxychlor	72-43-5	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1278 Molybdenum (insoluble compounds)	7439-98-7	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1294 Particulates (not otherwise regulated)	--	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1305 Pentaerythritol	115-77-5	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1310 Perlite	--	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1328 Picloram	1918-02-1	15 mg/m^3 TWA	10 mg/m^3 TWA 20 mg/m^3 STEL	10 mg/m^3 TWA; 5 mg/m^3 TWA
1331 Plaster of Paris	7778-18-9	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1333 Portland cement	65997-15-1	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1351 Rouge	--	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA

TABLE C10-1. Substances for Which the Exposure Limit is Based on the Avoidance of Physical Irritation and Other Effects (continued)

H.S. Number/ Chemical Name	CAS No.	OSHA's Former Generic Total Particulate Limit*	Proposed PEL*	Final Rule PEL for Total Particulate**; Respirable Fraction
1359 Silicon	7440-21-3	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1360 Silicon carbide	409-21-2	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1369 Starch	9005-25-8	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1374 Sucrose	57-50-1	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1383 Temephos	3383-96-8	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1391 4,4'-Thiobis (6-tert-butyl-m-cresol)	96-69-5	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1396 Titanium dioxide	13463-67-7	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1423 Vegetable oil mist	—	15 mg/m^3 TWA	10 mg/m^3 TWA	15 mg/m^3 TWA; 5 mg/m^3 TWA
1434 Zinc stearate	557-05-1	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA
1438 Zinc oxide	1314-13-2	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA; 5 mg/m^3 TWA

* OSHA did not propose to revise the Agency's generic 5-mg/m^3 respirable-fraction PEL for particulates and therefore did not mention the 5-mg/m^3 respirable-fraction limit in the proposed rule.

** OSHA's TWA limits are for 8-hour exposures.

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For the remaining 27 substances in this category, OSHA has reviewed the available toxicological literature and has determined that the former generic total particulate limit is not sufficiently protective. For these substances, the adverse health effects associated with exposure include, in addition to physical irritation, chronic pulmonary disease, cancer, and mutagenic, reproductive, and teratogenic effects. In the past, many of these substances were designated as "nuisance" dusts or particulates; however, recent developments in toxicology have increasingly shown that exposure to these substances has led to serious health effects. Thus, as applied to these substances, the term nuisance is a misnomer, because the hazards these substances pose in the workplace are real, widespread, and potentially serious. NIOSH shares OSHA's concern about this trend in the toxicology of particulates and has already either designated several of these substances, which were formerly considered "inert," as potential occupational carcinogens or labeled them as causing other target-organ effects. Examples of substances in this category that the recent toxicological literature has suggested may cause more serious effects are: benomyl (reproductive effects); kaolin (pulmonary fibrosis); methoxychlor (cancer); picloram (liver and kidney damage); synthetic graphite (pneumoconiosis); and titanium dioxide (cancer) (Ex. 8-47).

In addition to these diseases, toxicologists have recently expressed concern over the identification of a condition known as pulmonary alveolar proteinosis. This condition, which is apparently caused by the physical effects of particulate exposure, can be fatal if not properly diagnosed, although treatment with lung lavage is effective (NIOSH-ILO 1988). Pulmonary alveolar proteinosis has occurred in workers exposed to several particulates, including the so-called inert dusts. The onset of this condition may occur within months after first exposure to dust.

For the 27 physical irritants determined by OSHA to have identified adverse health effects in the literature, the final rule establishes an 8-hour TWA total particulate limit of 10 mg/m^3 and retains the former respirable fraction limit of 5 mg/m^3. The 10 mg/m^3 limit is consistent with the ACGIH's total particulate limit for these substances.

OSHA previously had no substance-specific limits for these 44 individual physical irritants; the Agency's former generic limit for particulates was 15 mg/m^3 as total particulate and 5 mg/m^3 as respirable particulate (see Table Z-3 of 29 CFR 1910.1000). OSHA proposed to reduce the limit for all substances falling within this category and for many specific particulates to 10 mg/m^3, measured as total particulate, to retain the 5-mg/m^3 respirable particulate limit, and to list many of these particulates individually on the Z tables. However, in the final rule, OSHA finds it appropriate to retain the former limits of 15-mg/m^3 as total particulate and 5-mg/m^3 as the respirable fraction limits for the 17 substances (and particulates not otherwise regulated) in this category for which there is little or no evidence of specific health effects. OSHA is listing these substances individually on the Z-1-A table in the final rule. As noted above, the Agency has also determined that worker protection requires that the total particulate limits for the remaining 27 substances, which have been shown to cause serious and potentially life-threatening health effects, be set at 10 mg/m^3. The 5-mg/m^3 respirable particulate limit is retained for all substances in the physical-irritant category.

There were several general comments on the substances in this category. NIOSH (Ex. 8-47) asked for clarification of the fact that OSHA did not mention in the proposal the limits for the respirable fraction of the total particulates in this category. NIOSH urged OSHA to include a respirable fraction limit because, "for substances that typically become airborne in the workplace as respirable particulates, a PEL based on the respirable fraction of the substance would be warranted" (Ex. 8-47, pp. 9-10). OSHA agrees entirely with NIOSH and wishes to clarify that its former 5-mg/m^3 limit for respirable particulate is being retained for all of the substances in this category; the preamble to the proposed rule did not discuss the respirable particulate limit specifically because the Agency did not intend to revise this component of its generic limit for particulates.

Several commenters (Exs. 3-661 and 3-726) raised the issue of the interaction between the individual listing of particulates in the Z tables and the requirements of OSHA's Hazard Communication Standard (HCS) (29 CFR 1910.1200). These commenters are concerned that particulates that were formerly considered nonhazardous under the HCS will be considered "hazardous" if OSHA lists these substances separately on the Z tables. On August 8, 1988 (53 *FR* 29822), OSHA published a proposal to modify the HCS. The coverage of nuisance particulates was specifically raised as an issue in that proposal, and the Agency will make a determination regarding that coverage in the final HCS rule.

Description of the Health Effects

The adverse exposure effects caused by the 18 substances in this group for which the 15-mg/m^3 limit is being retained include: interference with vision; deposition of these substances in the eyes, ears, nasal passages, and upper respiratory tract; and skin and corneal irritation. For the group of 27 substances for which a limit of 10 mg/m^3 is being established, the additional exposure effects include pulmonary alveolar proteinosis, reproductive effects, irreversible pulmonary effects, liver and kidney effects, systemic poisoning, and cancer. As discussed above, these latter effects are increasingly being associated with exposure to some of these substances, many of which were formerly considered biologically inert.

Thus, workers exposed to excessive airborne concentrations of any of these physical irritants may have difficulty seeing, may cough uncontrollably, may develop conjunctivitis or dermatitis, or may develop disabling or even life-threatening disease. In addition to these primary effects, workers distracted by physical-irritant effects may be more likely than nonexposed workers to have accidents and thus to endanger both themselves and others. (These adverse health effects also clearly have substantial productivity impacts.)

Many commenters opposed any reduction in the PELs for these substances on the grounds that inadequate evidence was provided to support the contention that exposure leads to material impairment of health or that a reduced limit would protect against a significant risk (see, for example, Exs. 3-1123, 3-726, 3-755, 3-887, 3-898, 3-939, 3-1012, 3-1016, and 8-22). Typical of the comments submitted on this subject are those made by the American Feed Industry Association (AFIA):

> AFIA believes an arbitrary choice by a non-government entity [i.e., the ACGIH], which is not supported by even a scintilla of scientific evidence, has very limited validity, and should not be used by OSHA as a basis for promulgating a regulation (Ex. 3-755, p. 19).

Arguing along the same lines, Peter Hernandez of the American Iron and Steel Institute stated that, in his opinion, the effects of exposure to these substances are "short-term and immaterial" (Ex. 8-22, pp. 29-30). OSHA is not persuaded by these arguments, for several reasons. First, the ACGIH

represents the opinion of professional industrial hygienists with experience and expertise as to what constitutes sound industrial hygiene and public health practice. Second, practical experience has shown that even the so-called "inert" dusts represent a danger to health; the International Labour Organization states:

[T]he biological effects of these inert dusts are of a long-term nature and are neither fibrogenic nor carcinogenic, toxic or allergenic. In excessive quantities they will overcharge the protective and scavenging mechanisms, thereby leading to respiratory disease. The extent to which any type of dust represents a health risk thus depends on exposure, which includes the nature of the dust, its concentration and the duration of exposure, as well as upon individual factors such as the general constitution and state of health of the person concerned, including the functional state of the upper respiratory tract, the lung function and its structure, the general immunological status and specific immunological reactivity, and the biochemical reactivity. All these factors will play a part in the onset of disease (ILO 1983, *Encyclopedia of Occupational Health and Safety*, Vol. I, p. 680).

In addition, the Agency notes that a particulate standard of 10 mg/m^3 or less (measured as total particulate) is the official standard in a great many countries, including Finland, Denmark, Norway, Sweden, the United Kingdom, Japan, Poland, Czechoslovakia, and the Republic of China (Cook 1987/Ex. 1–187, pp. 234–241).

In addition, as discussed above, OSHA notes with concern the trend in the toxicology of these substances, which is to find increasingly that substances formerly believed to be inert are in fact associated with serious and sometimes life-threatening effects. When exposures to the substances shown in Table C10–1 are kept under good industrial hygiene control in the workplace, OSHA believes that exposures are not likely to result in significant organic disease or irreversible toxic effects.

The following discussions describe the record evidence and OSHA's findings for the physical irritants included in this group. In addition, the health effects potentially associated with exposures to these substances are reviewed.

ALPHA-ALUMINA

CAS: 1344–28–1; Chemical Formula: Al_2O_3
H.S. No. 1014

OSHA formerly had no specific limit for alpha-alumina, although OSHA's general limit of 15 mg/m^3 total particulate (5 mg/m^3 for the respirable fraction) applied to this substance. The ACGIH has an 8-hour TWA of 10 mg/m^3, measured as total dust, for alpha-alumina. OSHA proposed an 8-hour TWA of 10 mg/m^3 for this substance, and this is the limit established by the final rule. The 5-mg/m^3 respirable-fraction limit is retained. Alpha-alumina, also called aluminum oxide, is a white powder that is widely used as an abrasive grinding material.

A study by Miller and Sayers (1941/Ex. 1–595) determined that alumina particles with diameters of less than 40 microns produced no reaction in laboratory animals. The results of a study by Stacy, King, Harrison et al. (1959/Ex. 1–761) confirmed the findings of Miller and Sayers; these authors found a-alumina to be nearly inert when injected into the lungs of rats (Stacy, King, Harrison et al. 1959/Ex. 1–761). Inhalation of fine aluminum powders at unspecified levels did not cause fibrosis in rats, guinea pigs, or hamsters (Gross, Harley, and deTreville 1973/Ex. 1–696).

In 1923, shortly after a-alumina replaced sandstone as the industrial abrasive of choice, Macklin and Middleton (1923, as cited in ACGIH 1986/Ex. 1–3, p. 21) reported that workers exposed to aluminum oxide dust using the new, synthetic abrasive had much less pulmonary disease than had workers using sandstone abrasives. Other studies (Sutherland, Meiklejohn, and Price 1937/Ex. 1–674; Meiklejohn and Posner 1957/Ex. 1–1060; Meiklejohn and Jones 1948/Ex. 1–964) reported that workers exposed to aluminum oxide dust in the chinaware industry and in aluminum production showed no evidence of pneumoconiosis. However, some early studies (Clark and Simmons 1925/Ex. 1–725; Clark 1929/Ex. 1–1048) reported that workers engaged in aluminum oxide production and exposed to dust levels generally between 50 and 100 mppcf showed X-ray evidence of pulmonary fibrosis; these workers are likely also to have been exposed to silica. Workers exposed during World War II to bauxite fumes containing both alumina and silica developed pulmonary fibrosis and emphysema; the authors believe that silica fume was involved in the development of these diseases (Shaver and Riddell 1947/Ex. 1–666). The ACGIH (1986/Ex. 1–3, p. 21) states that alpha-alumina acts as an inert material. However, NIOSH (Ex. 8–47, Table N4) reports that two studies in animals (Stacy, King, Harrison et al. 1959/Ex. 1–761; Stanton, Laynard, Tegeris et al. 1981, as cited by NIOSH in Ex. 8–47) have found that exposure to alpha-alumina is associated with the development of respiratory effects. For this reason, NIOSH does not concur with OSHA's limit for this substance, urging instead that OSHA establish a lower limit. However, OSHA believes that additional evidence is needed to support an additional reduction in the PEL for this substance. No other comments on alpha-alumina were submitted to the record.

OSHA is establishing 8-hour TWA limits of 10 mg/m^3 total particulate and 5 mg/m^3 respirable particulate for alpha-alumina, the limits being established for all physical irritants having identified health effects. The Agency concludes that these limits will protect workers from the significant risk potentially associated with exposures to alpha-aluminum dust in the workplace. OSHA finds that skin, eye, and upper respiratory irritation and other possible respiratory effects constitute material health impairments.

ALUMINUM METAL DUST

CAS: 7429–90–5; Chemical Formula: Al
H.S. No. 1016

OSHA formerly had no specific permissible exposure limit for aluminum metal dust, although the Agency's generic 15 mg/m^3 TWA limit for total particulate applied. The ACGIH has an 8-hour TWA limit of 10 mg/m^3 as total dust for this substance. OSHA proposed a PEL of 10 mg/m^3 (total particulate) and 5 mg/m^3 (respirable fraction) for aluminum metal dust; however, in the final rule, OSHA is retaining its former 15-mg/m^3 total particulate limit for this substance. In its elemental form, aluminum is a white, malleable, ductile metal.

Aluminum metal dust has been shown to present a minimal health hazard, according to results from the McIntyre Foundation's 27-year study for aluminum oxide dust (discussed in Stokinger 1981a, in *Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., Vol. 2A, pp. 1500–1503). No deleterious lung or systemic effects were observed as a result of exposure to aluminum metal dust having a particle size of 1.2 um at calculated concentrations equivalent to 2 mg/m^3 over an 8-hour workshift. Even much higher concentrations (not further specified) over 10- or 20-minute periods produced no adverse effects (ACGIH 1986/Ex. 1–3, p. 22). A comment submitted by the Reynolds Aluminum Company endorses OSHA's classification of aluminum metal dust under the general dust and particulate heading (Ex. 3–135). NIOSH (Ex. 8–47, Table N4) did not conduct an in-depth review of the health evidence for this substance.

OSHA has concluded that aluminum metal dusts are appropriately controlled by retaining the Agency's PELs of 15

mg/m³ TWA, as total particulate, and 5 mg/m³, as the respirable fraction. OSHA has determined that these limits will provide protection against the significant risk of physical irritation.

AMMONIUM SULFAMATE

CAS 7773-06-0; Chemical Formula: $NH_4SO_3NH_2$
H.S. No. 1024

OSHA formerly regulated ammonium sulfamate under its generic limit of 15 mg/m³ as total particulate. The ACGIH has established a limit of 10 mg/m³ for this substance as an 8-hour TWA. The final rule establishes a limit of 10 mg/m³ (and 5 mg/m³ for the respirable fraction) for ammonium sulfamate, which is the limit the Agency proposed. NIOSH (Ex. 8-47, Table N4) concurs with this limit. Ammonium sulfamate is a noncombustible, white, crystalline substance.

Lehman (1951/Ex. 1-790) found oral LD_{50}s of 3900, 5700, and 3000 mg/kg in rats, mice, and quail, respectively. He also reported that no effects were noted in rats administered 10,000 ppm ammonium sulfamate in the diet for 105 days. The hazards associated with exposure to ammonium sulfamate include eye and nose irritation, interference with vision, and the danger of accidents caused by the distraction and avoidance reactions typical of workers overexposed to dusts in the workplace. Only NIOSH commented on ammonium sulfamate.

OSHA is establishing a PEL of 10 mg/m³ TWA, total particulate, and retaining the 5-mg/m³ TWA PEL for respirable particulate for ammonium sulfamate. The Agency concludes that these limits will protect workers against the significant risk of material health impairment in the form of physical and other irritation that is associated with exposure to this substance.

BARIUM SULFATE

CAS: 7727-43-7; Chemical Formula: $BaSO_4$
H.S. No. 1031

OSHA formerly had no specific limit for barium sulfate, although OSHA's generic 15-mg/m³ total particulate limit previously applied; the ACGIH has a TLV-TWA of 10 mg/m³, total dust, for this substance. The proposal included a 10-mg/m³ TWA PEL for barium sulfate (total particulate), and the final rule establishes this limit and additionally retains the 5-mg/m³ PEL for the respirable fraction. Barium sulfate is a white or yellowish, odorless, tasteless powder.

Einbrodt, Wobker, and Klippel (1972/Ex. 1-1020) exposed rats to a concentration of 40 mg/m³ for two months and concluded that barium sulfate is not toxic. As an inert dust of the noncollagenous type, however, barium sulfate has the potential to cause pneumoconiosis through tissue reactions to accumulated dust in the lung (Anonymous, *British Medical Journal* 1972, as cited in ACGIH 1986/Ex. 1-3, p. 48). Barium sulfate has not been known to cause adverse effects in industrial workers exposed over periods of several years. (Doig 1976/Ex. 1-551). NIOSH did not conduct an in-depth review of the health evidence for barium sulfate (Ex. 8-47, Table N4); no other comments on this substance were submitted to the record.

In the final rule, OSHA is establishing a 8-hour TWA PEL for barium sulfate of 10 mg/m³ (total particulate) and retaining the 5-mg/m³ 8-hour TWA (respirable particulate). The Agency concludes that these limits will protect workers against the significant risks of material health impairment in the form of eye, nose, and upper-respiratory-tract irritation and, perhaps, of pneumoconiosis that are associated with exposure to barium sulfate.

BENOMYL

CAS: 17804-35-2; Chemical Formula: $C_{14}H_{18}N_4O_3$
H.S. No. 1032

OSHA formerly regulated benomyl under its generic total particulate limit of 15 mg/m³. The ACGIH has established a total dust TLV-TWA of 10 mg/m³ for this substance. OSHA proposed a PEL of 10 mg/m³ as total particulate for benomyl, and the final rule establishes this limit and retains the Agency's existing 5-mg/m³ respirable fraction limit. Benomyl is a white crystalline solid; exposures to this substance occur in its particulate form.

Studies of rats and rabbits indicate that the oral and skin absorption LD_{50}s are greater than 10,000 mg/kg, and studies of guinea pigs show a very low risk of skin irritation. Application to the shaved intact skin of ten male guinea pigs (as aqueous suspensions containing 5, 12.5, and 25 percent benomyl) resulted in slight irritation; one of ten guinea pigs had mild erythema two days after application of the high concentration (E.I. du Pont de Nemours and Co., Inc. 1974, as cited in ACGIH 1986/Ex. 1-3, p. 49). In another study, instillation of 10 mg of dry 50-percent powder or of 0.1 ml of 10-percent suspension in mineral oil caused only temporary mild conjunctival irritation (E.I. du Pont de Nemours and Co., Inc., unpublished data, as cited in ACGIH 1986/Ex. 1-3, p. 49). NIOSH notes that benomyl exposure may cause adverse reproductive effects (Ex. 8-47, p. 12); no other comments on this substance were submitted.

In the final rule OSHA is establishing 10 mg/m³, total particulate, and 5 mg/m³, respirable particulate, for this substance as 8-hour TWA limits. The Agency concludes that these limits will protect workers from the significant risks of benomyl's effects, which include irritation and erythema, and the possibility that exposure to benomyl may cause reproductive effects. OSHA finds that these health effects constitute material impairments of health. OSHA will also continue in the future to monitor the scientific evidence on the health effects associated with exposure to benomyl to determine whether a further reduction in the PEL is warranted.

BISMUTH TELLURIDE (UNDOPED)

CAS: 1304-82-1; Chemical Formula: Bi_2Te_3
H.S. No. 1035

OSHA had no former limit for undoped bismuth telluride, although OSHA's generic total particulate limit of 15 mg/m³ formerly applied. The ACGIH has a total-dust TLV-TWA of 10 mg/m³ for the undoped form of this substance. The proposed PELs for bismuth telluride in the undoped form were 10 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate); however, in the final rule, OSHA is retaining the total particulate limit of 15 mg/m³ for this substance, as well as the 5-mg/m³ respirable-fraction limit. Bismuth telluride appears as gray, hexagonal platelets; it is also available as ingots or single crystals.

An eleven-month inhalation study of dogs, rabbits, and rats exposed to pure undoped bismuth telluride dust at 15 mg/m³ showed the pulmonary responses typical of exposures to inert dusts (Wagner, Madden, Zimber, and Stokinger 1974, as cited in ACGIH 1986/Ex. 1-3, p. 59). NIOSH has not evaluated the evidence for undoped bismuth telluride in depth (Ex. 8-47, Table N4). No other comments on this substance were submitted.

OSHA is retaining its permissible exposure limits of 15 mg/m³ TWA, as total particulate, and 5 mg/m³, as the respirable fraction, for pure undoped bismuth telluride. The Agency concludes that these limits protect workers from the significant risks associated with workplace exposures to bismuth telluride.

BORON OXIDE

CAS: 1303-86-2; Chemical Formula: B_2O_3
H.S. No. 1039

OSHA formerly regulated boron oxide under its generic total particulate limit

of 15 mg/m³ (5 mg/m³ for the respirable fraction), and the ACGIH recommends a total dust TLV–TWA of 10 mg/m³. The proposed total particulate PEL was 10 mg/m³, and this limit is established in the final rule; the 5-mg/m³ PEL for the respirable fraction is retained. NIOSH (Ex. 8–47, Table N4) concurs with these limits. Boron oxide occurs as either a white powder or a granular solid, and it has a bitter taste.

Animal studies indicate that eye and skin irritation were caused by the ocular instillation and the topical application, respectively, of boron oxide to the skin and eyes of rabbits. Aerosol administration at various exposure levels for varying time periods caused mild irritation and an increase in urine acidity and creatinine coefficient in dogs and rats (Wilding, Smith, Yevich, et al. 1959/Ex. 1–599). Young rats that were force-fed a 10-percent slurry to boron oxide in water for three weeks showed no growth retardation or other effects (Wilding, Smith, Yevich et at. 1959/Ex. 1–599).

Garabrant and co-workers (1984/Ex. 1–555) determined the prevalence of eye and respiratory irritation among boron oxide-exposed workers; those exposed to boron oxide concentrations ranging from 1.2 to 8.5 mg/m³ were then compared with controls. Workers exposed at an average concentration of 4.1 mg/m³ reported significant increases in productive cough; eye, nose, and throat irritation; dryness of the mouth; and sore throats (Garabrant, Bernstein, Peters, and Smith 1984/Ex. 1–555).

The ACGIH believes that a total dust TLV–TWA of 10 mg/m³ will provide protection against boron oxide's irritant effects (ACGIH 1986/Ex. 1–3). However, OSHA specifically noted in the preamble to the proposed rule that irritation of the upper respiratory tract and eyes occurs among occupationally exposed workers at levels below 10 mg/m³, and the Agency solicited additional information on the boron oxide exposure levels associated with adverse health effects in workers.

U.S. Borax (Exs. 3–744, and 8–49; Tr. pp. 9–11 to 9–120) submitted comments to the record on the health effects of exposure to the borates and boron oxide. John C. Middleton, Manager of Product Safety for U.S. Borax Research Corporation, opposed the reduction in the PEL for boron oxide from 15 mg/m³ to 10 mg/m³ on the grounds that such a reduction was not "supportable" (Tr. p. 9–112). Mr. Middleton urged OSHA to "delay action" on boron oxide until a large epidemiological study being sponsored by U.S. Borax is completed; the American Mining Congress (Ex. 3–876) supported U.S. Borax's request for a delay.

In response to these commenters, OSHA notes that boron oxide dust is not an inert substance; it causes eye and upper respiratory tract irritation as well as skin irritation. Although OSHA will follow the progress of the U.S. Borax study with great interest, the Agency does not find it appropriate to delay further in reducing the PEL for boron oxide.

Accordingly, the final rule establishes permissible exposure limits of 10 mg/m³ TWA, as total particulate, and 5 mg/m³ TWA, as the respirable fraction, for boron oxide. The Agency concludes that these limits will protect workers against the significant risk of upper-respiratory-tract and eye irritation associated with exposure to this substance. OSHA finds that these health effects constitute material impairments of health.

CALCIUM CARBONATE

CAS: 1317–65–3; Chemical Formula: $CaCO_3$
H.S. No. 1057

OSHA formerly regulated calcium carbonate under the Agency's generic 15-mg/m³ total particulate limit. The ACGIH has a TLV–TWA of 10 mg/m³ for this substance, measured as total dust. The proposed total particulate PEL was 10 mg/³ as an 8-hour TWA; NIOSH (Ex. 8–47, Table N4) concurred with this limit. In the final rule, however, OSHA is retaining its 8-hour total particulate limit for calcium carbonate of 15 mg/m³. The Agency's former 5-mg/m³ PEL for the respirable fraction is also being retained. Calcium carbonate is an odorless, tasteless powder or crystal that is found in limestone, chalk, marble, plant ashes, bones, and shells.

Calcium carbonate is a moderate skin irritant and a severe eye irritant (*Dangerous Properties of Industrial Materials,* 7th ed., p. 677, Sax and Lewis 1989). Rabbits exposed dermally for 24 hours or ocularly for the same period developed moderate and severe irritation, respectively. The oral LD_{50} in rats is 6450 mg/kg (Sax and Lewis 1989, p. 677).

In the final rule, OSHA is retaining both the 8-hour TWA PEL of 15 mg/m³ for calcium carbonate (total particulate) and the 5-mg/m³ respirable particulate limit, to protest workers against the significant risk of physical irritation associated with exposure to calcium carbonate in the workplace.

CALCIUM SILICATE

CAS: 1344–95–2; Chemical Formula: None
H.S. No. 1061

OSHA formerly had no specific limit for calcium silicate; the Agency regulated this substance under its generic 8-hour TWA limit for particulates of 15 mg/m³ (total particulate). The ACGIH classifies calcium silicate as a nuisance dust and has an 8-hour limit of 10 mg/m³ for this white powder. The proposed total particulate PEL was 10 mg/m³; however, OSHA has determined that it is appropriate to retain the former 15-mg/m³ total particulate limit for calcium silicate. The 5-mg/m³ limit for the respirable fraction is also retained. Calcium silicate is a white powder.

There are no reported health effects in humans or animals as a result of exposure to calcium silicate. Calcium silicate is thus without long-term adverse health effects if exposures are kept under reasonable control. NIOSH (Ex. 8–47, Table N4) has not conducted an in-depth evaluation of calcium silicate, and no other comments on this substance were received.

OSHA is retaining its 8-hour TWA limits of 15 mg/m³, total particulate, and 5 mg/m³, as the respirable fraction, for calcium silicate. The Agency concludes that these limits protect workers from the significant risk of physical irritation in the workplace.

CALCIUM SULFATE

CAS: 7778–18–9; Chemical Formula: $CaSO_4$
H.S. No. 1062

OSHA formerly regulated calcium sulfate under its generic total particulate limit of 15 mg/m³. The ACGIH has a TLV–TWA of 10 mg/m³ (total dust) for this crystalline or powdery substance. OSHA proposed an 8-hour TWA PEL of 10 mg/m³ for calcium sulfate; however, the final rule retains the former limits of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction) for calcium sulfate.

Calcium sulfate dust is reported not to have specific irritant properties (ACGIH 1986/Ex. 1–3, p. 93). One report has indicated that no lung diseases are associated with exposure to calcium sulfate in miners (Hunter 1975, as cited in ACGIH 1986/Ex. 1–3, p. 93). Calcium sulfate appears to produce no adverse effects beyond those associated with general physical irritation. NIOSH (Ex. 8–47, Table N4) has not thoroughly evaluated the evidence for calcium sulfate; no other comments were submitted to the rulemaking record.

OSHA is retaining its 8-hour TWA permissible exposure limits for calcium sulfate of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate); the Agency concludes that these limits are sufficient to prevent the significant risk of eye, skin, and other physical irritation.

CELLULOSE

CAS: 9004-34-6; Chemical Formula: $(C_6H_{10}O_5)_n$
H.S. No. 1076

OSHA formerly regulated cellulose under the Agency's generic 8-hour TWA total particulate limit of 15 mg/m³. The ACGIH has a TLV-TWA of 10 mg/m³ (total dust) for this substance. The proposed total particulate PEL was 10 mg/m³ as an 8-hour TWA; however, the final rule retains the Agency's former total particulate limit of 15 mg/m³ and the 5-mg/m³ limit for the respirable fraction. Technical cellulose refers to that portion of the plant cell wall derived exclusively from glucose; it resembles cotton cellulose in its physical and chemical properties (ACGIH 1986/Ex. 1-3, p. 113).

Inhalation of cellulose dust is not irritating or toxic in exposed humans if exposures are properly controlled (Schreiber 1974/Ex. 1-1096). In industry, cellulose dust occurs in combination with other substances, such as quartz dust, wood, cotton, flax, jute, and hemp fibers, and these substances have demonstrated toxicities that are unrelated to their cellulose content (ACGIH 1986/Ex. 1-3, p. 113). NIOSH, the only commenter on cellulose dust, has not conducted an in-depth review of the health effects associated with exposure to this substance (Ex. 8-47, Table N4).

OSHA is retaining its former 8-hour TWA PELs for this substance of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) for cellulose dust containing less than 1 percent quartz. The Agency concludes that these limits protect exposed workers from the significant risks of eye, skin, and other physical irritation.

2-CHLORO-6-TRICHLOROMETHYL PYRIDINE (NITRAPYRIN)

CAS: 1929-82-4; Chemical Formula: $C_6H_3C1_4N$
H.S. No. 1082

OSHA formerly had no specific limit for nitrapyrin, although the Agency's generic total particulate limit of 15 mg/m³ TWA applied. The ACGIH has a TLV-TWA of 10 mg/m³ and a TLV-STEL of 20 mg/m³ for nitrapyrin. The proposed PEL was 10 mg/m³; however, in the final rule, OSHA is retaining its former total particulate limit of 15 mg/m³ and the respirable particulate limit of 5 mg/m³. NIOSH concurred with the proposed limit (Ex. 8-47, Table N4). Nitrapyrin is a crystalline substance.

Nitrapyrin's very low vapor pressure makes hazardous inhalation exposures unlikely. Torkelson (as cited in ACGIH 1986/Ex. 1-3, p. 428) has reported feeding dogs and rats a dosage of 15 mg/kg daily for 93 days. He observed no adverse effects in appearance, behavior, growth, food consumption, body and organ weight, mortality, or blood chemistry, and no tissue or organ changes. In the proposal, OSHA asked for comment on the need for a 20-mg/m³ 15-minute STEL for nitrapyrin. The Agency received no comments on this issue. Because OSHA has not determined that short-term exposures to nitrapyrin pose a significant risk to workers, no STEL is included in the final rule (see Section VI.C.17 for a discussion of the Agency's policies on STELs).

In the final rule, OSHA is retaining 8-hour TWA PELs of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) for this dust; OSHA finds that these limits are protective against the significant risk of physical irritation.

CLOPIDOL (COYDEN)

CAS: 2971-90-6; Chemical Formula: $C_7H_7C1_2NO$
H.S. No. 1095

OSHA formerly had no specific limit for clopidol; however, OSHA's generic total particulate limit of 15 mg/m³ TWA applied. The ACGIH has a TLV-TWA of 10 mg/m³ and a TLV-STEL of 20 mg/m³ for clopidol, which is a solid. The proposed total particulate PEL was an 8-hour TWA of 10 mg/m³; NIOSH (Ex. 8-47, Table N4) agreed that this limit is appropriate. However, in the final rule, OSHA is retaining the former 15-mg/m³ limit (total particulate) and 5-mg/m³ limit (respirable fraction), both 8-hour TWAs.

The oral LD_{50} for clopidol in rats, rabbits, and guinea pigs is greater than 8 g/kg (Dow Chemical Company 1973c, as cited in ACGIH 1986/Ex. 1-3, p. 141). Long-term (two-year) studies of rats and dogs fed at levels of 15 mg/kg and 5 mg/kg per day, respectively, showed no adverse effects. Similarly, there were no adverse effects on fertility, gestation, viability, or lactation in rats and rabbits, and no increase in teratogenicity (Dow Chemical Company 1973c, as cited in ACGIH 1986/Ex. 1-3, p. 141). The chronic toxicity of clopidol is reported to be low (ACGIH 1986/Ex 1-3, p. 141).

In the proposal, OSHA solicited comment on the need for a STEL for clopidol, but no comments were received on this issue. Because OSHA finds no evidence to suggest that short-term exposures pose a significant risk to workers, the final rule contains no STEL (see Section VI.C.17 for a discussion of the Agency's policies on STELs).

In the final rule, OSHA is retaining 8-hour TWA PELs of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) for clopidol. OSHA concludes that these limits will protect workers from the significant risk of eye, skin, and other physical irritation.

CRAG HERBICIDE (SESONE)

CAS: 136-78-7; Chemical Formula: $C_8H_7C1_2NaO_5S$
H.S. No. 1102

OSHA formerly applied a TWA limit of 15 mg/m³ for the total particulate of crag herbicide; this was the Agency's generic total particulate limit for all particulates. The ACGIH has a total-dust TLV-TWA of 10 mg/m³ for this colorless, odorless, noncombustible solid. The proposed PEL for crag herbicide was 10 mg/m³ (total particulate), and the final rule promulgates this limit; the 5-mg/m³ limit for the respirable fraction is retained. NIOSH (Ex. 8-47, Table N4) concurs with OSHA in the selection of these limits.

An early study reported an oral LD_{50} in rats of 1500 mg/kg for this herbicide (Smyth 1956/Ex. 1-759). At high concentrations, crag herbicide is a gastrointestinal irritant (NIOSH 1984, as cited in ACGIH 1986/Ex. 1-3, p. 519). Rats fed a diet containing 60 mg sesone/100 mg of diet experienced minor liver damage; when fed 20 mg sesone/100 gm of diet for two years, rats showed no adverse effects (ACGIH 1986/Ex 1-3, p. 519). In 1984, NIOSH reported the oral LD_{50} in rats to be 730 mg/kg (NIOSH 1984, as cited in ACGIH 1986/Ex 1-3, p. 519). There are no reported incidents of human poisoning associated with the use of sesone. NIOSH submitted the only comment on this substance.

OSHA is reducing the 8-hour TWA PEL for crag herbicide (total particulate) to 10 mg/m³ and retaining the 5-mg/m³ (respirable particulate) limit. OSHA concludes that these limits will protect workers from eye, skin, gastrointestinal, and other forms of irritation caused by exposure to crag herbicide.

DICYCLOPENTADIENYL IRON (FERROCENE)

CAS: 102-54-5; Chemical Formula: $C_{10}H_{10}Fe$
H.S. No. 1133

OSHA formerly regulated dicyclopentadienyl iron (ferrocene) under its generic total particulate limit of 15 mg/m³. The ACGIH has a TLV-TWA of 10 mg/m³ for this bright orange crystalline solid that smells like camphor. The proposed and final-rule PEL for dicyclopentadienyl iron is 10 mg/m³ (total particulate) as an 8-hour TWA. The 5-mg/m³ PEL for the respirable fraction is retained. NIOSH (Ex. 8-47, Table N4) supports the selection of these PELS.

Available evidence in animals suggests that dicyclopentadienyl iron

has a moderate order of oral toxicity but a high order of intravenous and intraperitoneal toxicity. In mice, the oral LD_{50} has been reported as 600 mg/kg (Madinaveitia 1965/Ex. 1–862). In rats, 1000 mg/kg has been reported as the lethal dose, but subacute oral toxicity tests have shown no fatalities when 10 feedings of 200 mg/kg were given over a two-week period (E.I. du Pont de Nemours and Co., Inc. 1955, as cited in (ACGIH 1986/Ex 1–3, p. 195). Ferrocene has been found to be mutagenic in bioassays involving several species (*Dangerous Properties of Industrial Materials*, 7th ed., Sax and Lewis 1989). NIOSH was the only commenter to the rulemaking record on this substance.

In the final rule, OSHA is establishing 8-hour TWA limits of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction) for dicyclopentadienyl iron. The Agency concludes that these limits will substantially reduce the significant risk of material health impairments, in the form of mutagenic and other effects, that are associated with occupational exposure to this substance.

EMERY

CAS: 112–62–9; Chemical Formula: $A1_2O_3$
H.S. No. 1155

OSHA formerly regulated emery under the Agency's generic 15-mg/m³ total particulate limit for all particulates. The ACGIH has a limit of 10 mg/m³ TWA, total dust, for emery containing less than 1 percent quartz. The proposed total particulate PEL was 10 mg/m³ as an 8-hour TWA, and the final rule establishes this limit and retains the 5-mg/m³ PEL for the respirable fraction. Emery is impure corundum (aluminum oxide), and is found in certain mineralogical deposits.

Emery dust inhalation is believed to have contributed to a case of pneumoconiosis in France, although it is questionable whether this incident was caused by emery dust alone or by the silica impurities in the dust (*Archives des Maladies Professionelles de Medecin du Travail et de Securité Sociale* 1970, as cited in ACGIH 1986/Ex. 1–3, p. 229). Exposure to emery dust containing less than 1 percent silica produces little, if any, effect on the health of exposed workers; it does not affect the lungs or produce organic disease at commonly encountered levels (ACGIH 1986/Ex. 1–3, p. 229).

NIOSH (Ex. 8–47) notes that rats exposed to aluminum oxide developed lipoid pneumonia (Stacy, King, Harrison et al. 1959/Ex. 1–761) and that humans so exposed have reported skin and respiratory tract irritation. Based on these data, NIOSH (Ex. 8–47, Table N4) does not concur with OSHA in the establishment of this PEL for emery. No other comments on this substance were received by the Agency.

In the final rule, OSHA is establishing a PEL of 10 mg/m³ TWA, total particulate, and retaining the PEL of 5 mg/m³, respirable particulate, for emery. OSHA concludes that these limits will prevent the significant risk associated with exposures to emery in the workplace; these risks include skin and upper-respiratory-tract irritation and, perhaps, other respiratory effects, all of which constitute material health impairments.

FERBAM

CAS: 14484–64–1; Chemical Formula: $[(CH_3)_2NCS_2]_3Fe$
H.S. No. 1176

OSHA formerly applied its generic particulate limit of 15 mg/m³ as an 8-hour TWA (total particulate) to ferbam. The ACGIH has a TLV–TWA of 10 mg/m³ for this odorless black solid. OSHA proposed an 8-hour TWA total particulate PEL of 10 mg/m³ for ferbam, and this limit is established in the final rule. The 5-mg/m³ PEL for the respirable fraction is retained. NIOSH (Ex. 8–47, Table N1) concurs with these limits.

Ferbam, which is a fungicide, has been reported to have an oral LD_{50} of more than 17 mg/kg in rats, but rabbits and guinea pigs demonstrated less sensitivity to this substance (Hodge, Maynard, Downs, and Blanchet 1952/Ex. 1–861). Thirty-day dietary studies of rats showed no effect at ferbam doses of 0.01 percent, with fatalities occurring at 0.5 percent. Dogs showed no adverse effects when fed 25 mg/kg of ferbam daily for six months. Inhalation of ferbam affects the upper respiratory tract in humans, in the manner typical of airborne exposures to workplace dusts (Hodge, Maynard, Downs, and Blanchet 1952/Ex. 1–861). NIOSH submitted the only comment on ferbam.

In the final rule, OSHA is reducing the total particulate PEL for ferbam to a 10-mg/m³ 8-hour TWA; the 5-mg/m³ TWA limit for the respirable fraction is retained. The Agency concludes that this reduction is necessary to prevent the significant health and safety risks associated with workplace exposures to ferbam. These risks include skin, eye, and upper respiratory tract irritation, which together constitute material health impairments.

GLYCERIN (MIST)

CAS: 56–81–5; Chemical Formula: $CH_2OHCHOHCH_2OH$
H.S. No. 1188

OSHA formerly had no specific limit for glycerin mist, although this substance was previously regulated at 15 mg/m³ under the generic total particulate limit. The ACGIH has a TLV-TWA of 10 mg/m³ (total particulate) for glycerin. OSHA proposed a total particulate PEL of 10 mg/m³, and the final rule promulgates this limit and retains the 5-mg/m³ limit for the respirable fraction. Glycerin is an oily, hygroscopic liquid with a warm, sweet taste.

Glycerin was long considered to be nontoxic; however, there are indications that the mist may be injurious to the kidneys at very high exposure levels (Campanacci 1965/Ex. 1–1047). NIOSH (Ex. 8–47) states that, at high concentrations, exposure may cause hemolysis, hemoglobinuria, and renal failure. Ackermann, Bässler, and Wagner (1975, as cited in ACGIH 1986/Ex. 1–3, p. 286) have reported that glycerin mist is easily metabolized and excreted. In the adult human of average weight, 2 grams of glycerol can be metabolized and excreted in an 8-hour workday. At this metabolic and elimination rate, the ACGIH believes that no ill effects are likely to occur as a result of exposure at or below 10 mg/m³ as an 8-hour TWA (ACGIH 1986/Ex. 1–3, p. 286).

NIOSH, the only commenter on this substance, does not agree that the final rule's limit of 10 mg/m³ is appropriate for glycerin mist because a recent study by Wiebe and Barr (1984, as cited in Ex. 8–47) found reproductive effects in rats injected intratesticularly with glycerin mist (Ex. 8–47).

OSHA is establishing an 8-hour TWA limit of 10 mg/m³ (total particulate) and retaining the 5-mg/m³ (respirable particulate) limit for glycerin mist. The Agency concludes that these limits will provide protection against the significant risks of glycerin exposure, which include kidney damage and, perhaps, testicular effects. OSHA finds that these health effects constitute material health impairments, and the Agency intends to monitor the literature on glycerin in the future.

GRAPHITE, SYNTHETIC

CAS: None; Chemical Formula: None
H.S. No. 1191A

OSHA formerly had no specific limit for synthetic graphite, although it was covered under the Agency's generic total particulate limit. OSHA's proposed 8-hour TWA PEL for synthetic graphite was 10 mg/m³ (total particulate), and this limit is established by the final rule; the 5-mg/m³ limit for the respirable fraction is retained. The ACGIH also has a TLV-TWA limit of 10 mg/m³ for graphite as total dust. Synthetic graphite

is a crystalline form of carbon made from the high-temperature treatment of coal or petroleum products; it has the same properties as natural graphite.

Meiklejohn reported in 1958 that synthetic graphite injected intraperitoneally in mice produced effects characteristic of those of the inert dusts (Meiklejohn 1958, as cited in ACGIH 1986/Ex. 1-3, p. 291).

In humans, exposure to natural graphite has long been associated with the development of pneumoconiosis (Koopman 1924/Ex. 1-131; Ruttner, Bovet, and Aufdermauer 1952/Ex. 1-661; Pendergrass, Vorwald, Mishkin et al. 1967/Ex. 1-77). Lister (1961/Ex. 1-422) and Lister and Wimborne (1972/Ex. 1-423) reported fibrotic changes in the lungs of a worker who had been engaged for 17 years in the production and milling of synthetic graphite. Other reports of lung injury caused by exposure to graphite have not distinguished between the form of the graphite (i.e., natural or synthetic) causing the injury; in addition, exposures to impurities, such as quartz silica, were involved in many of the reported cases (ACGIH 1986/Ex. 1-3, p. 291). NIOSH (Ex. 8-47) does not believe that it is appropriate to distinguish between the natural and synthetic forms of graphite and notes that the Lister and Wimborne (1972/Ex. 1-423) study described above suggests that synthetic graphite dust exposure "is capable of producing pneumoconiosis." NIOSH believes that a 2-mg/m^3 8-hour TWA PEL is appropriate for synethetic graphite because this is the limit set for coal dust (respirable) to protect against pneumoconiosis (Ex. 8-47). OSHA received no responses other than NIOSH's to a question raised in the proposal about synthetic graphite-related occupational disease. OSHA intends to continue to evaluate any new evidence on synthetic graphite exposures, such as the study on carbon/graphite fibers submitted by NIOSH (Zumwalde and Harmison 1980, as cited in Ex. 8-47), to determine whether further action to reduce the PEL is warranted in the future.

At present, however, OSHA is reducing the 8-hour TWA total particulate limit for synthetic graphite from 15 mg/m^3 to 10 mg/m^3 and retaining the 5-mg/m^3 limit for the respirable fraction to protect against the significant health risks associated with graphite exposure in the workplace. OSHA concludes that these limits will substantially reduce the risks of granite-induced respiratory disease, which constitutes a material impairment of health.

GYPSUM

CAS: 7778-18-9; Chemical Formula: $CaSO_4H_2O$
H.S. No. 1192

The former OSHA limit for gypsum was an 8-hour TWA of 15 mg/m^3; the ACGIH has a TLV-TWA of 10 mg/m^3, measured as total particulate, for gypsum. The proposed PEL was 10 mg/m^3 (total particulate). However, in the final rule, OSHA is retaining both the 8-hour TWA PEL of 15 mg/m^3 (total particulate) and the 5-mg/m^3 respirable particulate limit for gypsum. Gypsum is found either as colorless or white crystals.

The ACGIH (1986/Ex. 1-3) states that gypsum does not "produce significant organic disease or toxic effect when exposures are kept under reasonable control." Exposures in excess of the recommended limit may result in reduced visibility, deposits of gypsum dust in the eyes, ears, and nasal passages, and skin irritation. NIOSH, the only commenter on this substance, has not thoroughly reviewed the effects of gypsum exposure (Ex. 8-47, Table N4).

In the final rule, OSHA is retaining the Agency's limit for gypsum (total particulate) of 15 mg/m^3 as an 8-hour TWA and the 5-mg/m^3 limit for the respirable fraction of this substance. The Agency concludes that these limits protect workers from the significant risk of eye, skin, and other forms of physical irritation caused by gypsum exposure.

KAOLIN

CAS: None; Chemical Formula: $H_2Al_2Si_2O_8O$ H_2O
H.S. No. 1230

OSHA's former limit for kaolin was 15 mg/m^3, measured as total particulate; this was the Agency's generic total particulate limit for all dusts and particulates. The ACGIH has a TLV-TWA of 10 mg/m^3, measured as total dust. The Agency proposed an 8-hour TWA PEL of 10 mg/m^3 for kaolin, and the final rule establishes this limit; the 5-mg/m^3 limit for the respirable fraction is retained. Kaolin may be a white powder, or a white or yellow-white, earthy mass.

Exposure to excess amounts of kaolin dust may cause injury to the skin or mucous membranes (ACGIH 1986/Ex. 1-3). Although NIOSH (Ex. 8-47, Table N4) has not conducted an in-depth review of kaolin dust exposure, it notes that exposure to kaolin dust has been associated with respiratory effects (Lapenas and Gale 1983, as cited in Ex. 8-47). OSHA intends to monitor the developing toxicological literature on kaolin in the future. No other comments on this substance were received.

At this time, however, OSHA is establishing PELs of 10 mg/m^3 (total particulate) and 5 mg/m^3 (respirable particulate) as 8-hour TWA limits for kaolin. The Agency concludes that these limits will protect workers from the significant health risks associated with exposure to this substance. These risks include skin and mucous membrane injury, and, perhaps, irreversible respiratory effects, all of which constitute material health impairments.

LIMESTONE

CAS: 1317-65-3; Chemical Formula: $CaCO_3$
H.S. No. 1232

The former OSHA PEL for limestone was an 8-hour TWA of 15 mg/m^3, measured as total particulate. The ACGIH has a 10-mg/m^3 TWA for limestone (total particulate). OSHA proposed 10 mg/m^3 as the 8-hour TWA PEL for total limestone particulate and 5 mg/m^3 TWA for the respirable fraction; however, the final rule retains the 8-hour total particulate limit of 15 mg/m^3 and the respirable particulate limit of 5 mg/m^3. Limestone is rock formed by the accumulation of organic remains that consist of calcium carbonate and, less often, magnesium carbonate.

Direct contact with limestone dust at unspecified levels has been associated with the development of severe eye irritation and moderate skin irritation (*Dangerous Properties of Industrial Materials*, 7th ed., p. 677, Sax and Lewis 1989). The application of 500 mg limestone to the skin of rabbits for 24 hours produced moderate irritation, and 750 mg instilled into the eyes of rabbits caused severe irritation. The oral LD_{50} in rats is 6450 mg/kg (Sax and Lewis 1989, p. 677). The American Iron and Steel Institute (Exs. 3-1123 and 8-22) argued that limestone dust produces effects that are "short-term and immaterial" (Ex. 8-22, pp. 29-30); however, OSHA does not agree that the physical irritant effects caused by exposure to dusts and particulates are not material impairments; such irritation involves the skin, eyes, nose, upper respiratory tract and mucous membranes.

In the final rule, OSHA is retaining 8-hour TWA limits of 15 mg/m^3 (total particulate) and 5 mg/m^3 (respirable particulate) for limestone. The Agency concludes that these limits protect workers from the significant risk of eye and skin irritation, which may be experienced by employees exposed to limestone in the workplace.

MAGNESITE

CAS: 546-93-0; Chemical Formula: $(MgCO_3)_4$ $Mg(OH)_2$ $5H_2O$(approx)
H.S. No. 1233

OSHA's former PEL for magnesite was 15 mg/m³, measured as total particulate; this was the Agency's generic limit for all dusts and particulates. The ACGIH has a TLV-TWA of 10 mg/m³, also measured as total particulate. The proposed PELs for magnesite were 8-hour TWAs of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction). In the final rule, however, OSHA is retaining its former total particulate limit of 15 mg/m³ for magnesite. Magnesite occurs as a white powder.

Magnesite is considered by both OSHA and the ACGIH to be one of the dusts that "do not produce significant organic disease or toxic effect when exposures are kept under reasonable control" (ACGIH 1986/Ex. 1-3). Exposure to excess levels of magnesite in the workplace causes skin or mucous membrane irritation resulting either from contact with the magnesite itself or from the rigorous cleansing procedures necessary for removing the dust. NIOSH, the only commenter on this substance, has not substantively reviewed the effects of exposure to magnesite (Ex. 8-47, Table N4).

OSHA is retaining its 8-hour TWA PEL of 15 mg/m³ TWA for magnesite, measured as total particulate; the 5-mg/m³ TWA limit for the respirable fraction is also being retained. The Agency concludes that these limits protect workers from the significant risk of skin, mucous membrane, and other physical irritation.

MAGNESIUM OXIDE (FUME)

CAS: 1309-48-4 Chemical Formula: MgO
H.S. No. 1234

OSHA's former limit for magnesium oxide (as fume) was 15 mg/m³ as an 8-hour TWA, the Agency's generic limit for particulates. The ACGIH has a TLV-TWA limit of 10 mg/m³ for the fume of this white, odorless, very fine powder. OSHA proposed 8-hour TWA PELs of 10 mg/m³ (total particulate) and 5 mg/m³ (respirable particulate) for magnesium oxide fume, and the final rule establishes these limits.

Slight reactions (not further specified) have been reported in human subjects after exposures of less than 10 minutes to freshly generated MgO fume at concentrations of from 400 to 600 mg/m³ (Drinker, Thomson, and Finn 1927/Ex. 1-356). Animal and human studies of magnesium oxide fume exposure have shown toxicities less marked than but similar to those attributable to zinc oxide fume (Drinker and Drinker 1928/Ex. 1-314). The symptoms of exposure include those of metal fume fever (fever, chills, muscular pain, nausea, and vomiting) and leukocytosis, symptoms analogous to those caused by exposure to zinc oxide fume. NIOSH does not concur with the final rule's limit for this fume. NIOSH notes that exposure to magnesium oxide may also cause chronic respiratory disease (Ex. 8-47, p. 12); no other comments on this substance were received.

In the final rule, OSHA is setting a PEL of 10 mg/m³ TWA (total particulate) and retaining the 5-mg/m³ TWA limit for the repirable fraction of magnesium oxide fume. OSHA concludes that these limits will substantially reduce the significant risks of metal fume fever, leukocytosis, and, perhaps, chronic respiratory disease associated with exposure to magnesium oxide fume in the workplace. OSHA finds that these health effects constitute material health impairments.

MALATHION

CAS: 121-75-5; Chemical Formula: $C_{10}H_{19}O_6PS_2$
H.S. No. 1235

OSHA formerly had a 15-mg/m³ total particulate limit for malathion, with a skin notation; the ACGIH TLV for this substance is 10 mg/m³ as a TWA, also with a skin notation, and the NIOSH REL is 15 mg/m³. The proposed PEL was 10 mg/m³ (total particulate), with a skin notation. The final rule establishes a 10-mg/m³ TWA limit for total malathion particulate and includes a skin notation; the 5-mg/m³ TWA limit for the respirable fraction is retained. NIOSH (Ex. 8-47, Table N4) concurs with the selection of these limits. Malathion is a noncombustible, yellow to deep brown liquid with a skunk-like odor.

Malathion is a widely used organophosphorus insecticide having relatively low level of toxicity; some authors have determined that malathion is approximately 1/100th as toxic as parathion (Johnson, Fletcher, Nolan, and Cassaday 1952/Ex. 1-149). Rats fed malathion at a concentration of 100 ppm for two years exhibited no toxic effects (Hazleton and Holland 1953/Ex. 1-126). Several occupational and research exposures involving scientists or human volunteers produced no changes in blood cholinesterase or other effects (Rider, Mueller, Swader et al. 1969/Ex. 1-189; Hayes, Mattson, Short, and Witter 1960/Ex. 1-90; Culver, Caplan, and Batchelor 1956/Ex. 1-177). Fatalities have been reported in the Japanese and Indian literature, but these deaths have always involved extremely high doses of malathion (Chabra 1970/Ex. 1-151; Horiguchi 1973/Ex. 1-221). The symptoms of malathion overexposure include headache, lacrimation, vomiting, tremors, and convulsions.

The Agency received, in addition to NIOSH's comment, two record comments on malathion. ConAgra, Inc. (Ex. 3-635) questioned the classification of this substance as a nuisance dust "since most malathion-containing pesticides are liquids and are available over the counter for public use in retail stores" (Ex. 3-635, p. 2). OSHA points out that this health effects category includes all particulates (i.e., can include aerosols and mists generated by the handling of liquid materials).

Another commenter, Lawrence H. Hecker, Director of Corporate Industrial Hygiene and Toxicology for Abbott Laboratories, questioned the need for a skin notation for a substance with a dermal LD_{50} of 200-mg/kg or less in animal tests when there was no evidence of systemic effects in humans as a result of skin contact (Ex. 3-678, p. 3). Dr. Hecker stated that this 200-mg/kg cutoff would be consistent with OSHA's Hazard Communication Standard (29 CFR 1910.1200). OSHA agrees with Dr. Hecker that a consistent policy in regard to skin notations is appropriate but does not agree that the 200-mg/kg level is an appropriate cutoff point. The Agency finds that a dermal LD_{50} in rabbits of 1000 mg/kg is a better indicator of dermal toxicity; this is the Hazard Communication Standard's upper cutoff for a toxic, rather than highly toxic, substance administered by the dermal route (see Section VI.C.18 of this preamble for a discussion of OSHA's reasoning on this issue). In addition, OSHA believes that evidence that a substance has caused systemic toxicity in humans exposed via the dermal route sufficient reason to retain a skin notation; in the case of malathion, OSHA has received reports of exposed workers whose blood cholinesterase levels were reduced after dermal exposure to this substance. OSHA is therefore retaining the skin notation for malathion in the final rule.

In the final rule, OSHA is establishing PELs of 10 mg/m³ TWA (total particulate) and 5 mg/m³ TWA (respirable particulate) for malathion, with a skin notation. The Agency finds that exposure to malathion poses a significant risk of material health impairment in the form of cholinesterase inhibition.

MARBLE

CAS: 1317-65-3; Chemical Formula: None
H.S. No. 1239

OSHA formerly had no specific limit for marble dust, but regulated this substance under the generic total particulate limit of 15 mg/m³. The ACGIH has established an 8-hour TLV-

TWA of 10 mg/m³ for marble dust containing less than 1 percent quartz (measured as total dust). Marble dust, a metamorphic form of calcium carbonate dust, is an odorless and tasteless powder or crystal. OSHA proposed an 8-hour TWA limit of 10 mg/m³ for marble dust as total particulate containing less than 1 percent quartz and 5 mg/m³ TWA for the respirable fraction of this dust. NIOSH (Ex. 8-47, Table N4) did not specifically evaluate the effects of marble dust exposure, and no one else commented on marble.

In the final rule, OSHA is retaining its former total particulate limit for marble of 15 mg/m³, as well as the respirable particulate limit of 5 mg/m³. OSHA finds that these limits protect exposed workers against the significant risk posed by physical-irritant properties of marble.

METHOXYCHLOR

CAS: 72-43-5; Chemical Formula: $C_{16}H_{15}Cl_3O_2$
H.S. No. 1246

OSHA formerly applied its generic 15-mg/m³ TWA limit for particulates to methoxychlor. The ACGIH recommends a limit of 10 mg/m³ TWA for this white crystalline solid. This is the limit that was proposed for the total particulate of methoxychlor, and the final rule establishes this limit; the 5-mg/m³ limit for the respirable particulate is retained. NIOSH (Ex. 8-47, Table N4) concurs with the selection of these limits.

The reported oral LD_{50} for methoxychlor in rats is 6000 mg/kg (Lehman 1954, as cited in ACGIH 1986/Ex. 1-3, p. 364). Lehman also determined that 100 ppm for two years is the lowest dietary level producing no effect in rats; this corresponds to a level of 350 mg/man/day (Lehman 1954, as cited in ACGIH, 1986/Ex. 1-3, p. 364). Results of another dietary study indicated that rats fed 200 ppm methoxychlor for two years were not affected in terms of growth or survival (Hodge, Maynard, and Blanchet 1952/Ex. 1-488). Tegeris and co-workers (1966/Ex. 1-389) reported that dogs fed 1 g/kg daily for six months showed weight loss; most animals died within nine weeks when the dietary level was increased to 2 g/kg daily (Tegeris, Earl, Smalley, and Curtis 1966/Ex. 1-389). Morgan and Hickenbottom (1978/Ex. 1-351) reported that male Holtzman rats fed 10, 40, 160, or 640 mg/kg for 24 hours showed no liver abnormalities. Extrapolating from animal data, Lehman (1954) estimated the dose levels that would produce toxic effects in humans as follows: the fatal oral dose would be 450 grams; adverse health effects would occur at 6430 mg/kg orally; and 2414 mg/kg is the level at which dermal effects would be predicted to occur (Lehman 1954, as cited in ACGIH 1986/Ex. 1-3, p. 364).

NIOSH concurs with these limits (Ex. 8-47, Table N4) but recommends that methoxychlor also be designated as a potential occupational carcinogen. However, OSHA notes that both IARC and NCI find the evidence for the carginogenicity of methoxychlor in animals to be inadqequate (*Dangerous Properties of Industrial Materials*, 7th ed., p. 1326, Sax and Lewis 1989). The Agency will continue to monitor the scientific evidence for this substance in the future. No other comments on methoxychlor were received by OSHA.,

In the final rule, OSHA is reducing the existing 15-mg/m³ 8-hour TWA limit for methoxychlor to 10 mg/m³ (total particulates) and retaining the 5-mg/m³ TWA limit for the respirable fraction to reduce the significant health risks of systemic toxicity, which constitutes a material impairment of health. OSHA also notes that cancer may be an exposure effect of methoxychlor.

MOLYBDENUM (INSOLUBLE COMPOUNDS)

CAS: 7439-98-7; Chemical Formula: Insoluble compounds (as Mo)
H.S. No. 1278

OSHA formerly had a limit of 15 mg/m³ TWA for the insoluble compounds of molybdenum, which include molybdenum metal dust and the dioxide; this was the Agency's generic limit for all particulates. The ACGIH recommends a TLV-TWA of 10 mg/m³, measured as molybdenum, for these substances. The proposed PELs were 10 mg/m³ TWA (total particulate) and 5 mg/m³ (respirable particulate), measured as molybdenum, and these are the limits established in the final rule. Molybdenum is a silver-white metal or a dark gray or black powder.

Mogilvskaya (1950, as cited in ACGIH 1986/Ex. 1-3, p. 415) reported that the dust of molybdenum metal and molybdenum dioxide caused irritation of mucosal surfaces in white mice after an intensive dusting for one hour; in a similar 30-day exposure, the metal and the dioxide proved minimally poisonous.

NIOSH (Ex. 8-47, Table N4) notes that reviews of molybdenum's toxicity have been published by Browning (1961b), Friberg and Lener (1986), and Stokinger (1981d). NIOSH states that, although these reviewers generally agree that the insoluble compounds of molybdenum have a low order of toxicity, there is some evidence that respiratory effects have been caused by exposure to these compounds. NIOSH recommends that the toxicological literature on molybdenum be evaluated on a continuing basis. No other comments on this substance were received by OSHA.

OSHA is establishing PELs for the insoluble compounds of molybdenum of 10 mg/m³ TWA (total particulate) and 5 mg/m³ TWA (respirable particulate), measured as molybdenum. The Agency concludes that these limits will protect workers from the significant health risks of exposure to the insoluble compounds of molybdenum, which include eye, nose, and skin irritation, and, perhaps, chronic respiratory effects. OSHA finds that these effects constitute material health impairments.

PARTICULATES (NOT OTHERWISE REGULATED)

CAS: None; Chemical Formula: None
H.S. No. 1294

OSHA formerly covered all otherwise unregulated particulates under a single 8-hour TWA PEL of 15 mg/m³ (measured as total particulate) and 5 mg/m³ (measured as the respirable fraction). The ACGIH has a TLV-TWA of 10 mg/m³ (as total dust) for particulates having a quartz content of less than 1 percent. OSHA's proposed total particulate PEL for these physical irritants was 10 mg/m³; NIOSH (Ex. 8-47, Table N4) concurred with the proposed limit. In the final rule, OSHA is retaining its 15-mg/m³ total particulate PEL for particulates that are not specifically identified in the Z tables; OSHA is also retaining its 5-mg/m³ respirable particulate limit for these substances. The Agency has decided to retain its existing limits for particulates that are not specifically identified in the Z tables because this group of physical irritants consists of substances, both inorganic and organic, for which substance-specific toxicologic data are not available. For those physical irritants for which specific toxicologic data are available, OSHA has separately identified the substance in Table Z-1-A and has promulgated a 10-mg/m³ 8-hour TWA (measured as total particulate) and a 5-mg/m³ 8-hour TWA PEL (measured as the respirable fraction) in the final rule.

The 8-hour TWA limits of 15 mg/m³ (total particulate) and 5 mg/m³ (respirable fraction) apply to all not-otherwise-regulated particulates (i.e., to those irritants that are not specifically identified in the Z tables). For example, OSHA's limits for corn dust, a particulate not identified in the Z tables or otherwise regulated, are 15 mg/m³ (total dust) and 5 mg/m³ (respirable dust). The Agency believes that other particulates that present physical irritant hazards in the workplace should also be regulated under the final rule's

generic total particulate limit of 15 mg/m³.

OSHA believes that good industrial hygiene practice requires that exposures to these particulates be controlled in the workplace to or below the 15-mg/m³ level as an 8-hour TWA to protect workers from the broad range of adverse effects associated with exposure to these substances. In the past, these particulates were often called "nuisance" or "inert" substances. These terms are misleading, however, because exposures to these substances in the workplace may cause serious and sometimes disabling effects. Further, good industrial hygiene and public health practice require that workplace exposure to particulates be maintained below the level associated with physical irritation, accidents, and respiratory effects.

Several commenters (see, for example, Exs. 3–661, 3–755, 3–1012, 3–1112, and 8–22) submitted comments on OSHA's proposed generic total particulate limit. Most of these participants argued that the proposed reduction in the 8-hour TWA PEL from 15 mg/m³ to 10 mg/m³ was unwarranted because there was, in the opinion of these commenters, no evidence of adverse health effects associated with exposure to these particulates (Exs. 3–755, 3–1012, 3–1112, and 8–22). According to Peter Hernandez of the American Iron and Steel Institute (Ex. 8–22), the effects of such exposures are "short-term and immaterial."

OSHA has responded to these commenters in the final rule by establishing a lower 8-hour TWA total-dust limit of 10 mg/m³ for all particulates having identified health effects in the toxicological literature, and retaining the former 15-mg/m³ total particulate limit for those particulates not specifically linked to health effects other than physical irritation. OSHA finds that good industrial hygiene practice demands, and prudent public health policy supports, effective workplace control over exposure to all particulates. The effects associated with overexposure to particulates in the workplace constitute material impairments of health and functional capacity and include upper respiratory tract irritation, skin injury, eye irritation, and other forms of physical irritation.

The 15-mg/m³ 8-hour TWA total particulate PEL applies to all particulates not otherwise regulated, not just to inorganic dusts. The OSHA Review Commission interpreted the Agency's former generic dust standard as applying only to mineral dusts, primarily because this limit was entered on the Z tables under the heading of "mineral dust." The ACGIH and OSHA both had intended this limit to apply to all particulates, organic and inorganic. Exposure to organic particulates at high levels also causes material health impairment, such as throat, skin, and eye irritation, upper-respiratory-tract problems, and the safety hazards caused by distraction in the workplace.

In the final rule, OSHA establishes an 8-hour TWA limit of 15 mg/m³, measured as total particulate, and retains the 5-mg/m³ limit for respirable particulates for all particulates not otherwise regulated. The Agency concludes that these limits will protect workers against the significant safety and health risks associated with exposure to excessive concentrations of these substances, which include reduced visibility, deposits in the eyes, ears, and nasal passages, throat and eye irritation, upper-respiratory-tract problems, skin injury, and other forms of physical irritation. The change in terminology from nuisance dusts to particulates not otherwise regulated clarifies OSHA's intent and also more accurately reflects the fact that exposure to all particulates at levels higher than those being established in this final rule causes material impairment of health and functional capacity in workers experiencing these exposures.

PENTAERYTHRITOL

CAS: 115–77–5; Chemical Formula: $C(Ch_2OH)_4$
H.S. No. 1305

OSHA formerly had no separate limit for pentaerythritol, but this substance was regulated at 15 mg/m³ TWA, the Agency's generic total particulate limit. The ACGIH has a TLV–TWA of 10 mg/m³ for total pentaerythritol dust containing less than 1 percent quartz. The proposed PEL was 10 mg/m³ TWA (total particulate), and this is the limit established in the final rule; the Agency's 5-mg/m³ respirable particulate TWA limit is being retained. Pentaerythritol is an odorless, white crystalline solid.

Rats exposed to pentaerythritol at 11,000 mg/m³ for six hours were reported to show no ill effects from a single exposure, and rats, dogs, and guinea pigs exposed six hours daily for 90 days also showed no effects (Keplinger and Kay 1964/Ex. 1–743). The oral LD_{50}s in guinea pigs and mice were 11.3 and 22.5 g/kg, respectively; rats survived oral doses as high as 16 g/kg. At higher doses, animals displayed diarrhea, tremors, ataxia, and loss of righting reflex (Keplinger and Kay 1964/Ex. 1–743). Daily applications of a saturated aqueous solution of technical pentaerythritol to rabbit skin produced no significant irritation; a single application of 10 g/kg aqueous paste on intact or abraded rabbit skin produced no evidence of percutaneous absorption (Keplinger and Kay 1964/Ex. 1–743; Hercules, Inc., as cited in ACGIH 1986/Ex. 1–3, p. 462). Instillation of a 50-percent aqueous suspension into the conjunctival sac of rabbits' eyes resulted in slight transient irritation (Hercules, Inc., as cited in ACGIH 1968/Ex. 1–3, p. 462).

Human volunteers are reported to have eliminated 85 percent of dietary pentaerythritol unchanged in the urine within 30 hours. A slight and transient increase in apparent blood sugar that was proportional to the ingested dose appeared in these subjects soon after administration (Berlow, Barth, and Snow 1958, as cited in ACGIH 1986/Ex. 1–3, p. 462). NIOSH (Ex. 8–47, Table N4) has not conducted an extensive review of this substance. No other comments were submitted to the record.

The final rule promulgates an 8-hour PEL of 10 mg/m³ TWA (total particulate) for pentaerythritol, and the 5-mg/m³ respirable fraction PEL is retained. The Agency concludes that these limits will protect employees from the significant risks of physical irritation potentially associated with exposure to pentaerythritol at higher levels. OSHA finds that physical irritation constitutes a material impairment of health within the meaning of the Act.

PERLITE

CAS: None; Chemical Formula: None
H.S. No. 1310

OSHA formerly regulated perlite under its generic total particulate limit of 15 mg/m³. The ACGIH has a TLV–TWA of 10 mg/m³ for perlite as total dust containing less than 1 percent quartz. The proposed PELs were 10 mg/m³ (total particulate) and 5 mg/m³ TWA (respirable particulate); however, the final rule retains the 15-mg/m³ TWA PEL for perlite as total particulate containing less than 1 percent quartz. The respirable fraction limit of 5 mg/m³ is also retained. Perlite is a natural volcanic glass; it is essentially an amorphous mineral consisting of fused sodium-potassium-aluminum silicate.

Perlite is reported to have a free-silica content varying from zero to 3 percent (Anderson, Selvig, Baur et al. 1956 and the Perlite Institute, both as cited in ACGIH 1986/Ex. 1–3, p. 467). In its processed crude and expanded forms, perlite is reported to have a measurable quartz content of 0.4 percent quartz and 0.2 percent cristobalite (Sheckler 1977, as cited in ACGIH 1986/Ex. 1–3, p. 467). There are no published reports of

adverse physiologic effects from exposure to perlite dust. NIOSH, the only commenter on perlite, has not reviewed the evidence for this substance in depth (Ex. 8–47, Table N4).

OSHA finds that perlite is nontoxic when airborne total particulate concentrations are maintained at levels of 15 mg/m^3 or below and when its quartz content is limited to a level below 1 percent crystalline silica. For these reasons, the final rule establishes an 8-hour PEL of 15 mg/m^3 TWA for total perlite dust containing less than 1 percent quartz and retains the 5-mg/m^3 TWA PEL for the respirable fraction of perlite dust. OSHA concludes that these limits protect workers from the significant risk of eye, skin, and other forms of physical irritation.

PICLORAM

CAS: 1918–02–1; Chemical Formula: $C_6H_3Cl_3N_2O_2$
H.S. No. 1328

OSHA formerly had no limit for picloram, but regulated it at the generic total particulate limit of 15 mg/m^3 for all particulates. The ACGIH has a TLV–TWA of 10 mg/m^3 and a TLV–STEL of 20 mg/m^3 (both as total dust) for this white powder, which has an odor like that of chlorine; these were the limits proposed for picloram. The final rule promulgates the 10-mg/m^3 TWA limit for total particulate but does not include a STEL.

Picloram has low acute oral toxicity, with LD_{50} values of 3.75 g/kg for rats, 1.5 g/kg for mice, and 2.0 g/kg for rabbits (NIOSH 1979b, as cited in ACGIH 1986/Ex. 1–3, p. 489). Two-year feeding studies showed no ill effects in albino rats and beagle dogs from ingestion of does up to and including 150 mg/kg/day (McCollister and Leng 1969, as cited in ACGIH 1986/Ex. 1–3, p. 489). At 225 mg/kg/day, rats displayed moderate liver and kidney changes and, in females, slight body weight loss after 90 days. These authors (McCollister and Leng 1969, as cited in ACGIH 1986/Ex. 1–3, p. 489) also reported no fertility, reproduction, or lactation effects in albino rats fed at levels of up to 3000 ppm (0.3 percent) in a three-generational study. Although maternal toxicity in rats was reported at dietary levels of 750 and 1000 mg/kg administered during days 6 through 15 of gestation, neither teratogenic nor neonatal effects were observed when subtoxic or maternally toxic doses of picloram were administered during organogenesis (Thomson et al. 1972, as cited in ACGIH 1986/Ex. 1–3, p. 489). The National Cancer Institute (NCI) (1977d, as cited in ACGIH 1986/Ex. 1–3, p. 489) found a dose-related increase in benign liver tumors in female rats only and concluded that "under the conditions of the bioassay, the findings are suggestive of the ability of the compound to induce benign tumors in the livers of female Osborne-Mendel rats." Based on these results, NIOSH (Ex. 8–47, Table N4) concludes that "picloram is not a nuisance particulate and is not without toxic effects." OSHA notes that picloram must therefore be added to the list of substances formerly believed to be inert but subsequently shown to be toxic. No other comments on picloram were submitted to the rulemaking record.

At the present time, however, OSHA is establishing 8-hour TWA limits of 10 mg/m^3 (total particulate) and 5 mg/m^3 (respirable fraction) for picloram. The final rule does not include a short-term limit; in accordance with the policy described in Section VI.C.17 for short-term limits, OSHA has reviewed the evidence and has concluded that there is no basis for establishing a STEL for picloram, as proposed. The Agency concludes that these total and respirable particulate limits will minimize the significant risk of material health impairment in the form of systemic effects, such as liver and kidney damage, that are potentially associated with exposure to this substance at higher levels. OSHA intends to monitor the health effects literature on picloram in the future.

PLASTER OF PARIS

CAS: 7778–18–9; Chemical Formula: $CaSO_4$
H.S. No. 1331

OSHA's former Z–3 table listed an 8-hour TWA exposure limit of 15 mg/m^3 (total particulate) for Plaster of Paris. The ACGIH has a 10-mg/m^3 TWA for Plaster of Paris, measured as total dust, and this is the limit that was proposed. The final rule retains 15 mg/m^3 as the 8-hour TWA PEL for the total particulate of Plaster of Paris; the 5-mg/m^3 limit for the respirable fraction is also retained. Plaster of Paris is a fine, white powder.

Where occupational exposures to Plaster of Paris have been limited, no toxic effects or organic diseases of the lungs have occurred. Exposure to excessive levels of dust in the workplace may result in reduced visibility or injury to the skin or mucous membranes from the dust itself, or in damage to the skin from the rigorous skin-cleansing procedures required to remove the dust (ACGIH 1986/Ex. 1–3). Only NIOSH commented on this substance; NIOSH stated that it had not substantively reviewed the effects of Plaster of Paris exposure (Ex. 8–47, Table N4).

OSHA is retaining both the 8-hour TWA of 15 mg/m^3 (total particulate) and the 8-hour TWA PEL of 5 mg/m^3 (respirable particulate) for Plaster of Paris. The Agency concludes that these limits will protect against the significant risks of skin, eye, and other forms of physical irritation.

PORTLAND CEMENT

CAS: 65997–15–1; Chemical Formula: None
H.S. No. 1333

OSHA formerly had a limit of 50 mppcf (approximately 15 mg/m^3) for Portland cement containing less than 1 percent crystalline silica. The ACGIH has a TLV/TWA of 10 mg/m^3 for Portland cement as total dust containing less than 1 percent quartz. The proposed PEL was 10 mg/m^3, measured as total particulate, and this is the limit established in the final rule; the 5-mg/m^3 respirable fraction limit is retained. Portland cement refers to a class of hydraulic cements that are odorless gray powders containing less than 1 percent crystalline silica. Portland cement is insoluble in water and contains tri- and dicalcium silicate, in addition to varying amounts of alumina, tricalcium aluminate, and iron oxide.

Intraperitoneal injection of Portland cement in guinea pigs produced an absorptive reaction, which is an effect typical of inert particulates. Portland cement is eventually eliminated from the tissue and is generally not considered harmful when ingested (Miller and Sayers 1941/Ex. 1–595).

In a study of industrial exposures, Gardner and associates (1939/Ex. 1–589) found no evidence of Portland-cement-related pneumoconiosis in 2,278 workers who had been heavily exposed to this substance for prolonged periods of time (Gardner, Durkan, Brumfiel, and Sampson 1939/Ex. 1–589). Conflicting reports of pneumoconiosis (Parmeggiani 1951, as cited in ACGIH 1986/Ex. 1–3, p. 494; Prosperi and Barsi 1957/Ex. 1–1093) are attributed to the presence of silica in the inhaled dust rather than to exposure to Portland cement itself (ACGIH 1986/Ex. 1–3, p. 494). Cement dermatitis does occur among exposed workers, however, as a consequence of the alkaline, abrasive, and hygroscopic properties of the wet cement, which cause irritation of the skin (Schwartz, Tulipan, and Birmingham 1957a/Ex. 1–1168). NIOSH, the only commenter on this substance, reported that it has not thoroughly reviewed the health effects for Portland cement (Ex. 8–47, Table N4).

OSHA is establishing 8-hour TWA PELs of 10 mg/m^3 (total particulate) and 5 mg/m^3 (respirable fraction) for Portland cement containing less than 1

percent quartz. The Agency concludes that these limits will protect workers against the significant risks associated with on-the-job exposures to Portland cement dust. These risks include eye, skin, and mucous membrane irritation, and may include more severe respiratory effects, all of which constitute material health impairments. In addition, revising the total particulate limit to 10 mg/m³ as an 8-hour TWA will simplify employee exposure monitoring for Portland cement, since gravimetric rather than impinger methods can then be used.

ROUGE

CAS: None; Chemical Formula: None
H.S. No. 1351

OSHA formerly had no specific limit for rouge but regulated this substance under the Agency's generic total particulate standard of 15 mg/m³ as an 8-hour TWA. The ACGIH has an 8-hour TWA limit of 10 mg/m³ for rouge as total dust containing less than 1 percent quartz, and this is the limit that was proposed. The final rule establishes 10 mg/m³ as the 8-hour TWA PEL for the total particulate of rouge and retains the 5-mg/m³ 8-hour TWA for the respirable fraction of rouge dust. Rouge is a high-grade red pigment, composed mainly of ferric oxide, that is used as a polishing agent for glass, jewelry, etc.

NIOSH (Ex. 8–47, Table N4) believes that exposure to rouge should be reduced to levels below 10 mg/m³ on the basis of evidence showing that exposure to hematite dust (ferric oxide) increased the risk of lung cancer in hematite miners. According to NIOSH, this human evidence is consistent with the results of two recent animal studies: Warshawsky, Bingham, and Niemeier (1984 as cited in Ex. 8–47), which showed that intratracheal administrations of ferric oxide and exposure to benzo(a)pyrene (BaP) "enhances the metabolic activation of BaP"; and Niemeier, Mulligan, and Rowland (1986, as cited in Ex. 8–47), who found that ferric oxide has co-carcinogenic potential. OSHA shares NIOSH's concern about rouge's carcinogenicity and intends to monitor toxicological developments closely in the future to determine whether further reduction in the PEL is warranted. No other comments on rouge were received.

In the final rule, OSHA is establishing an 8-hour TWA of 10 mg/m³ for the total particulate of rouge and is retaining 5 mg/m³ as an 8-hour TWA for the respirable fraction. OSHA concludes that these limits will protect workers from the significant health risks associated with workplace exposure to higher levels of rouge. These effects include eye, nose, and upper respiratory irritation and, perhaps, other more serious chronic diseases, all of which constitute material health impairments within the meaning of the Act.

SILICON

CAS: 7440–21–3; Chemical Formula: Si
H.S. No. 1359

OSHA's former Z tables had no specific limit for silicon; however, silicon was formerly regulated under OSHA's generic particulate limits of 15 mg/m³ TWA (total particulate) and 5 mg/m³ (respirable fraction). The ACGIH has a 10-mg/m³ 8-hour TWA for silicon, measured as total dust. The proposed total particulate PEL for silicon was 10 mg/m³ as an 8-hour TWA, and this limit is established in the final rule; the 5-mg/m³ respirable fraction limit is retained. Silicon is a black to gray, lustrous, needle-like crystal that is used in the manufacture of semiconductors.

The evidence of silicon's toxicity in animals is conflicting. An early study by McCord, Fredrick, and Stolz (1937/Ex. 1–640) reported no response in guinea pigs and rats injected intraperitoneally with silicon. A more recent study (Schepers 1971/Ex. 1–570) demonstrated pulmonary lesions in rabbits administered an intratracheal dose of 25 mg silicon dust. NIOSH (Ex. 8–47, Table N4) has not throughly reviewed the health effects evidence for silicon, and no other comments on silicon were submitted to the record.

In the final rule, OSHA is establishing an 8-hour TWA limit of 10 mg/m³ (total particulate) for silicon and retaining the 5-mg/m³ (respirable fraction) limit. The Agency concludes that these limits will reduce the significant health risks potentially associated with exposure to this substance at higher levels. These risks include eye, skin, mucous membrane and other forms of physical irritation and may include chronic respiratory effects. OSHA finds that these effects constitute material health impairments.

SILICON CARBIDE

CAS: 409–21–2; Chemical Formula: SiC
H.S. No. 1360

OSHA formerly regulated silicon carbide under its generic 15-mg/m³ total particulate limit. The ACGIH has a 10-mg/m³ 8-hour TWA limit, measured as total dust. The proposed total particulate PEL for silicon carbide was 10 mg/m³, and the final rule promulgates this limit and retains the 5-mg/m³ respirable fraction limit for silicon carbide, which is a green to blue-black irridescent crystal.

An animal study (Gardner 1923/Ex. 1–737 showed that, although exposure to silicon carbide alone produced no fibrosis of the lungs, exposure of guinea pigs infected with tuberculosis to silicon carbide (six hours/day, five days/week for one year) aggravated pulmonary tuberculosis to the extent that extensive fibrosis occurred. Guinea pigs exposed to silicon carbide dust and infected with the tubercle bacteria developed tuberculopneumoconiotic lesions (Gross, Westrick, and McNerney 1959/Ex. 1–697). Miller and Sayers (1941/Ex. 1–595) observed that silicon carbide dust administered by intraperitoneal injection to guinea pigs produced no reaction.

Bruusgaard (1949/Ex. 1–1143) found that X-rays of 10 out of 32 workers exposed to average levels of 34 mppcf of silicon carbide for 15 years or more demonstrated pulmonary changes; these 10 workers were also tuberculin-positive. Miller, Davis, Goldman, and Wyatts (1953/Ex. 1–40) described three cases of pulmonary reactions and hyperglobinemia in tungsten carbide industry workers; these authors concluded that exposure to silicon carbide was not a hazard unless the exposed workers already had pulmonary tuberculosis. NIOSH (Ex. 8–47, Table N4) has not reviewed the health effects literature for silicon carbide in depth. No other comments on this substance were submitted.

In the final rule, OSHA is establishing a 10-mg/m³ TWA total particulate limit for silicon carbide and retaining the 5-mg/m³ TWA respirable fraction limit. The Agency concludes that these limits will protect workers from the significant risk of material health impairment in the form of the physical irritation that is associated with exposure to this particulate.

STARCH

CAS: 9005–25–8; Chemical Formula: $(C_6H_{10}O_5)_n$
H.S. No. 1369

The former OSHA limit for starch was 15 mg/m³ as an 8-hour TWA, the Agency's generic limit for all particulates. The ACGIH has a TLV-TWA of 10 mg/m³ for starch as total dust that contains no asbestos and less than 1 percent crystalline silica. The proposed total particulate PEL was 10 mg/m³; however, in the final rule, OSHA is retaining a total particulate limit of 15 mg/m³ for starch. Starch is a white, odorless powder.

Exposure to high concentrations of starch dust may result in impaired vision, or may cause injury to the mucous membranes or skin. Injury may also result from the vigorous skin-cleansing procedures necessary for the

complete removal of starch (ACGIH 1986/Ex. 1–3). NIOSH, the only commenter on starch, has not substantively reviewed its health effects (Ex. 8–47, Table N4).

OSHA is retaining both the 8-hour TWA total particulate PEL of 15 mg/m^3 and the 5-mg/m^3 respirable particulate limit for starch. The Agency concludes that these limits will control the significant risk of eye, skin, and other physical irritation that may result from exposure to high levels of starch in the workplace.

SUCROSE

CAS: 57–50–1; Chemical Formula: $C_{12}H_{22}O_{11}$
H.S. No. 1374

The former OSHA 8-hour TWA limit for sucrose was 15 mg/m^3 as total particulate, the Agency's generic limit for all particulates. The ACGIH includes sucrose in its grouping of particulates that "do not produce significant organic disease or toxic effect when exposures are kept under reasonable control" (ACGIH 1986/Ex. 1–3) and has a TLV-TWA limit of 10 mg/m^3 for sucrose as total particulate containing no asbestos and less than 1 percent quartz; this is also the limit OSHA proposed for this substance. The final rule, however, retains the 15-mg/m^3 total particulate and the 5-mg/m^3 respirable fraction TWA limits for sucrose, which is found in the form of white crystals.

Exposure to excess levels of sucrose dust can cause skin and eye irritation, interference with vision, and distraction from the task at hand.

OSHA is retaining the 8-hour total particulate TWA of 15 mg/m^3 for sucrose and is also retaining the 5-mg/m^3 respirable fraction limit. The Agency concludes that these limits protect exposed workers against the significant risk of physical irritation.

TEMEPHOS

CAS: 3383–96–8; Chemical Formula: $C_{16}H_{20}O_6P_2S_3$
H.S. No. 1383

The former OSHA Z tables had no specific limit for exposure to temephos, a cholinesterase-inhibiting insecticide. Temephos was formerly regulated under OSHA's generic particulate limit of 15 mg/m^3. The ACGIH limit is 10 mg/m^3 (total dust) as an 8-hour TWA. The proposed PEL was 10 mg/m^3 (total particulate), and this is the limit promulgated in the final rule; the 5-mg/m^3 limit for the respirable fraction of temephos dust is retained. NIOSH (Ex. 8–47, Table N4) concurs with the selection of these PELs. Temephos may be a white crystalline solid or a viscous brown liquid.

In rats and mice, temephos has an acute oral LD_{50} of 400 mg/kg or greater. Various animal species tolerated doses of 10 mg/kg without clinical effect and 1 mg/kg without effect on cholinesterase activity (Gaines, Kimbrough, and Laws 1967/Ex. 1–553). Laws, Morales, Hayes, and Joseph (1967/Ex. 1–562) revealed that human volunteers consuming oral doses or temephos at levels of either 256 mg/man/day for five days or 64 mg/man/day for four weeks evidenced no detectable effects on erythrocyte or plasma cholinesterase levels. Murphy and Cheever (1972/Ex. 1–567) reported that 1 mg of temephos per liter of drinking water produces no effect. These authors found that rat liver carboxylesterases were at least 30 times more sensitive to inhibition from temephos than were rat cholinesterases. Assuming that human liver carboxylesterases are proportionately more sensitive than cholinesterases, it is estimated that significant inhibition of these carboxylesterases could occur as a result of consuming 2 liters of drinking water containing 1 mg/L of temephos. Although nonspecific liver carboxylesterase is not critical for normal physiologic function, adverse effects on this enzyme could increase the susceptibility of exposed individuals to chemicals and drugs that contain carboxylesterase linkages (ACGIH 1986/Ex. 1–3, p. 557).

The ACGIH derived its limit of 10 mg/m^3 TWA for temephos from studies of malathion, which has an acute LD_{50} of 2100 mg/kg in rats, or roughly one-half that of temephos. Because humans tolerate 16 mg/day oral doses of malathion without effects on blood cholinesterase levels, the ACGIH believes the 10-mg/m^3 limit is appropriate for temephos (ACGIH 1986/Ex. 1–3, p. 557).

OSHA agrees with the ACGIH's reasoning in this matter and is establishing limits in the final rule of 10 mg/m^3 (total particulate) and 5 mg/m^3 (respirable fraction) for temephos. The Agency concludes that these limits will protect workers from the significant risk of cholinesterase inhibition and reduction in carboxylesterase activity, which together constitute material health impairments within the meaning of the Act and are potentially associated with exposure to this substance.

4,4'-THIOBIS (6-TERT-BUTLY-n-CRESOL)

CAS: 96–69–5; Chemical Formula: $C_{22}H_{30}O_2S$
H.S. No. 1391

OSHA formerly regulated 4,4'-thiobis under the Agency's generic total particulate limit of 15 mg/m^3 TWA. The ACGIH limit is 10 mg/m^3 as an 8-hour TWA, the limit established by the ACGIH for all of the nuisance dusts. OSHA proposed a 10-mg/m^3 total particulate TWA limit and a 5-mg/m^3 respirable fraction PEL for 4,4'-thiobis, and these limits are established in the final rule. NIOSH (Ex. 8–47, Table N4) concurs with the selection of these limits. 4,4'-Thiobis is a light gray to tan powder with a slightly aromatic odor.

In a 30-day study, rats fed diets of 500 ppm 4,4'-thiobis exhibited normal weight gain; those rats fed five times this amount exhibited enlarged livers and a reduced rate of weight gain (Lefaux 1968/Ex. 1–814). In a 90-day study reported by the same author, rats fed 50 ppm showed no toxic effects, but male rats fed 500 ppm ate and grew at a slightly lower rate. No pathologic changes were observed in the 500-ppm-dosed rates. A dose of 5 g/kg of 4,4'-thiobis proved lethal to rats, with the predominant symptom being gastroenteritis. NIOSH was the only commenter on this substance.

In the final rule, OSHA is establishing exposure limits of 10 mg/m^3 TWA (total particulate) and 5 mg/m^3 TWA (respirable fraction) for 4,4'-thiobis. The Agency concludes that these limits will protect workers from the significant risk of material health impairment, in the form of eye, skin, and other physical irritation, which is associated with exposure to this substance.

TITANIUM DIOXIDE

CAS: 13463–67–7; Chemical Formula: TiO_2
H.S. No. 1396

OSHA's former PEL for titanium dioxide was 15 mg/m^3 as an 8-hour TWA; this was the Agency's generic exposure limit for particulates. A 10-mg/m^3 8-hour TWA, measured as total dust, has been established by the ACGIH. The Agency proposed PELs of 10 mg/m^3 (total particulate) and 5 mg/m^3 (respirable particulate) for titanium dioxide, and these limits are established in the final rule. Titanium dioxide is a white crystalline solid.

Miller and Sayers (1941/Ex. 1–595) reported that intraperitoneal injections of titanium dioxide in guinea pigs showed a tendency to remain in the injected tissues but not to produce a proliferative response. A study by Grandjean, Turrian, and Nicod (1956/Ex. 1–638), in which rats were administered 50 mg of titanium dioxide intratracheally, showed pigmented dust deposits in the lungs. In addition, evidence of infection appeared in the alveoli of one rat and diffuse fibrosis was found in the lungs of a separate test animal. No nodule formation was observed (Grandjean, Turrian, and Nicod 1956/Ex. 1–638). Another study by

Dale (1973/Ex. 1–624) revealed thickening of the walls of the alveoli in the lungs of rabbits injected with titanium dioxide dust; however, lungs had returned to normal by three months post-treatment. Feeding studies of rats and mice at doses of 2.5 percent or 5 percent titanium dioxide for 103 weeks revealed no signs of carcinogenicity in either species (National Cancer Institute (NCI) 1979d/Ex. 1–947).

At the rulemaking hearing, NIOSH (Tr. p. 3–95) testified that exposure to this substance is associated with "a risk of cancer . . . . The incidence of tumors in animals exposed to titanium dioxide (Lee, Trochimowicz, and Reinhardt 1985) meets the . . . criteria for . . . [a] potential occupational carcinogen." Accordingly, NIOSH (Ex. 8–47, Table N4) recommends a full 6(b) rulemaking for this substance. In response to NIOSH, OSHA notes that the Agency intends to monitor the developing literature on titanium dioxide to determine whether an additional reduction in the PEL is warranted. NIOSH was the only commenter on titanium dioxide.

OSHA is establishing 8-hour TWAs of 10 mg/m^3 (total particulate) and 5 mg/m^3 (respirable particulate) for titanium dioxide in the final rule. OSHA concludes that the final rule's limits will protect workers from the significant health risks associated with exposure to titanium dioxide at higher levels. These risks include material impairments of health in the form of eye, skin, and other physical irritation, and, perhaps, of carcinogenicity.

VEGETABLE OIL MIST (EXCEPT CASTOR OIL, CASHEW NUT, OR SIMILAR IRRITANT OILS)

CAS: None; Formula: None
H.S. No. 1423

The former OSHA Z tables had no substance-specific limit for vegetable oil mist. The ACGIH has established a 10-mg/m^3 8-hour TWA for all nuisance particulates. The proposed PEL was 10 mg/m^3 (total particulate) as an 8-hour TWA and 5 mg/m^3 (respirable fraction) as an 8-hour TWA. NIOSH (Ex. 8–47, Table N4) concurs with the proposed limits. In the final rule, OSHA is establishing a 15-mg/m^3 total particulate limit for vegetable oil, which is a pale yellow, oily liquid.

One commenter (Ex. 3–1080) stated that OSHA had not, in the proposal, demonstrated that vegetable oil mist presents a health and safety hazard. OSHA finds that oil mist presents the same safety and health hazards as do all of the physical irritants. Occupational exposure to vegetable oil mist is associated with a variety of health and safety hazards, including interference with vision; eye tearing, and skin and other forms of physical irritation.

OSHA is establishing 8-hour TWA limits of 15 mg/m^3 (total particulate) and 5 mg/m^3 (respirable particulate) for vegetable oil mist (except castor oil, cashew nut, or similar irritant oils). The Agency concludes that these limits protect exposed workers against the significant risks of physical irritation described above.

ZINC STEARATE

CAS: 557–05–1; Chemical Formula: $Zn(C_{18}H_{35}O_2)_2$
H.S. No. 1434

OSHA formerly regulated zinc stearate under its generic total particuate limit of 15 mg/m^3 TWA. The proposed PEL was 10 mg/m^3 (total particulate), and the final rule promulgates this limit. NIOSH (Ex. 8–47, Table N4) agrees that this PEL is appropriate. The ACGIH has established an 8-hour TWA of 10 mg/m^3 for zinc stearate, measured as total dust. Zinc stearate is a white hydrophobic powder.

A report in *Folia Medica* (Volita and Noro 1957, as cited in ACGIH 1986/Ex. 1–3, p. 646) documented the case of a worker exposed to zinc stearate dust for 30 years who died from extensive fibrosis of the lungs. More recent studies have revealed incidences of pulmonary fibrosis associated with encephalopathy that stemmed directly from exposure to aluminum dust, which is frequently coated with stearic acid (*British Journal of Industrial Medicine* 1962, as cited in ACGIH 1986/Ex. 1–3, p. 646); the ACGIH (1986/Ex. 1–3, p. 646) is uncertain of the relevance of this report to zinc stearate exposures.

Observations of long-term worker exposures to this dust in the rubber industry revealed no adverse effects of exposure (B.F. Goodrich Rubber Company, private communication, as cited in the ACGIH 1986/Ex. 1–3, p. 646). NIOSH was the only commenter on zinc stearate.

OSHA is establishing a 10-mg/m^3 TWA limit for this particulate (measured as total particulate) and is retaining the 5-mg/m^3 TWA limit for the respirable fraction. The Agency concludes that these limits will prevent the significant health risks associated with workplace exposures to zinc stearate dust at higher levels. OSHA finds that the pulmonary effects potentially associated with exposure to zinc stearate constitute material impairments of health within the meaning of the Act.

ZINC OXIDE DUST

CAS: 1314–13–2; Chemical Formula: ZnO
H.S. No. 1438

OSHA formerly had no exposure limit specifically for zinc oxide dust. The ACGIH has a limit of 10 mg/m^3 as an 8-hour TWA for zinc oxide, measured as total dust. The proposed PEL was 10 mg/m^3, and this limit, measured as total particulate, is established by the final rule. Zinc oxide dust is a white or pale yellow powder.

According to Turner and Thompson (1926/Ex. 1–1124), exposure to finely divided zinc oxide dust can produce symptoms similar to those for metal fume fever. Beeckmans and Brown (1963/Ex. 1–775) reported that catalytically active zinc oxide dust is more toxic when treated with ultraviolet light. Aside from these considerations, the ACGIH considers zinc oxide dust to be a nuisance dust.

Two comments on zinc oxide were submitted to the rulemaking record (Exs. 3–673 and 3–675), but neither of these comments addressed the health effects associated with zinc oxide exposure. NIOSH does not concur with these limits; the NIOSH RELs for zinc oxide dust are 5 mg/m^3 TWA (respirable fraction) and 15 mg/m^3 (total dust) as 15-minute ceilings (Ex. 8–47, Table N4). NIOSH believes that exposure to zinc oxide dust causes respiratory effects and cites Gupta, Pandey, Misra, and Viswanathan (1986); Lam, Conner, Rogers et al. (1985); and NIOSH (1975d) in support of this view. OSHA will monitor developments on the toxicology of zinc oxide in the future to ensure that the PELs for this substance are protective.

In the final rule, OSHA is establishing limits of 10 mg/m^3 TWA (total particulate) and 5 mg/m^3 TWA (respirable particulate) for zinc oxide. The Agency concludes that these limits will protect workers from the significant risk of material health impairment in the form of physical irritation and, perhaps, of respiratory effects.

Conclusions

OSHA's generic 8-hour TWA particulate standard (29 CFR 1910.1000, Table Z–3) was adopted from the 1968 ACGIH TLV–TWA of 15 mg/m^3 for total dust and 5 mg/m^3 for respirable dust. At the time, the ACGIH considered the 15-mg/m^3 value to be "an acceptable limit of good hygienic practice," based on the then-prevailing "lack of knowledge" of any adverse exposure-related effects at levels below this value (*Documentation of the Threshold Limit Values and Biological Exposure Indices*, ACGIH 1966/Ex. 1–13). Shortly after OSHA adopted the ACGIH's 1968 limit, the ACGIH revised its limit downward to 10

mg/m³ for total dust and 5 mg/m³ for respirable dust. In justifying this reduction, the ACGIH noted that the lower levels would "result in appreciable improvement of working conditions in plants where the old limit of 15 mg/m³ formerly prevailed" (*Documentation of the Threshold Limit Values for Substances in Workroom Air*, 3rd ed., p. 190, ACGIH 1971).

In the final rule, OSHA has determined that it is appropriate to set a 10-mg/m³ total particulate limit for those particulates demonstrated to have, in addition to physical-irritant properties, specific adverse health effects. These substances are also being identified separately in Table Z-1-A. For the 18 substances in this section that are physical irritants but for which other health effects have not specifically been identified, OSHA is retaining the 8-hour TWA total particulate limit of 15 mg/m³. These substances are also separately identified in Table Z-1-A. For the group of Particulates not otherwise regulated (which includes all workplace particulates, both organic and inorganic) that is not separately identified in Table Z-1-A, OSHA is establishing a generic total particulate limit of 15 mg/m³. For all of the particulates in this section, the agency's former 5-mg/m³ TWA limit for the respirable fraction is being retained.

11. Substances for Which Limits Are Based on Avoidance of Odor Effects

Introduction

This category includes three substances that have a variety of toxic effects, including intolerable odors; they are grouped together in this section because their permissible exposure limits were set at levels that would prevent intolerable concentrations of these odors in the workplace. OSHA is retaining its existing 8-hour TWA limits for the three substances in this category; the Agency believes that the PELs for these substances provide adequate prevention against these odorant effects. At levels above those established by these PELs, workers are distracted from the task at hand, may be more prone to accidents, and are likely to experience considerable discomfort. OSHA is retaining its existing limits for these odorants based on the data described below, which provide information on the levels at which intolerable odor effects occur. Table C11-1 shows the substances included in this group and their former, proposed, and final rule limits, as well as their CAS and HS numbers.

BILLING CODE 4510-26-M

TABLE C11-1. Substances for Which Limits Are Based on Avoidance of Odor Effects

Chemical Name	CAS No.	Former OSHA PEL	Proposed PEL	Final Rule PEL*
1226 Isopropyl ether	108-20-3	500 ppm TWA	500 ppm TWA	500 ppm TWA
1314 Phenyl ether (vapor)	101-84-8	1 ppm TWA	1 ppm TWA	1 ppm TWA
1427 Vinyl toluene	25013-15-4	100 ppm TWA	100 ppm TWA	100 ppm TWA

* OSHA's TWA limits are for 8-hour exposures, and OSHA's STELs are for 15 minutes unless otherwise specified.

BILLING CODE 4510-26-C

Description of the Health Effects

The chemicals in this category have obnoxious odors and cannot willingly be tolerated by most workers for any period of time. Because odor detection occurs at very low concentrations for many of these chemicals, OSHA's existing permissible exposure limits were set at levels below the concentration at which the odor would become intolerable to employees.

The preamble to the proposed rule (53 *FR* 20961) asked commenters for information on the extent to which exposure to these odorant chemicals causes material impairment of health. Several commenters addressed this question (see discussion of this issue in Section V of this preamble). For example, William Prokop of the National Renderer's Association (Ex. 3–11) is of the opinion that the variability in odor threshold and response among individuals makes "the selection of a suitable limit based on odor objectionability . . . quite arbitrary." This commenter reports that "there can be a hundredfold difference in olfactory sensitivity" even within a group consisting only of 10 people (Ex. 3–11, p. 2). Because of such inter-individual variability, Mr. Prokop believes that the exposure limits should not be set on the basis of intolerable odor (Ex. 3–11). OSHA notes that it is a longstanding practice in industrial hygiene to prevent the hazards associated with obnoxious workplace odors; both the ACGIH and OSHA have had such limits for more than 20 years. In addition, the levels selected for these substances take the variability described by Mr. Prokop into account, because they are set at the level found to be unobjectionable by most exposed individuals.

According to NIOSH (Ex. 8–47):

> [T]he odors emitted by industrial chemicals often play an important role in occupational safety and health. . . . These odors may cause undue health concerns among exposed workers or may create safety hazards by distracting workers from their tasks. Strong odors in the workplace may also mask the presence of other, more toxic substances. . . . Olfactory fatigue often occurs and should be considered a functional impairment that can result in increased worker exposure . . . (Ex. 8–47, p. 41).

OSHA does not agree with NIOSH that these chemicals constitute material health impairments in situations where odor is the only adverse effect at the level of concern. OSHA's reasoning on this issue is discussed further in Section V of the preamble.

The following sections describe the record evidence on each of these chemicals and their adverse health effects in animals and humans. These effects, which range from nausea to narcosis, generally occur at levels higher than the limits for these substances; that is, the limits for these substances were set to prevent these more serious effects as well as objectionable odor effects. Because odor effects range in severity from distracting to intolerable, these limits have been set below the concentration at which the odor becomes objectionable enough to create a significant safety risk.

ISOPROPYL ETHER

CAS: 108–20–3; Chemical Formula: $(CH_3)_2CHOCH(CH_3)_2$
H.S. No. 1226

OSHA's former limit of 500 ppm (8-hour TWA) is being retained for isopropyl ether, and NIOSH (Ex. 8–47, Table N1) concurs that this limit is appropriate. The ACGIH recommends a TLV–TWA of 250 ppm and a TLV-STEL of 310 ppm for this liquid, which has a sharp, sickly sweet odor similar to that of ether.

Animal studies have shown that exposures to high concentrations of isopropyl ether cause narcosis and death (Machle, Scott, and Treon 1939/Ex. 1–348). Twenty exposures at a 1-percent vapor concentration produced intoxication and depression but no significant blood or organ weight changes. In rabbits, the minimum lethal dose has been reported to be 5 to 6.5 g/kg. The liquid is an irritant to the skin and mucous membranes and causes dermatitis on repeated exposure (Machle, Scott, and Treon 1939/Ex. 1–348).

Humans exposed for 15 minutes to isopropyl ether concentrations of 300 ppm experienced no overt irritation but complained about the objectionable odor of isopropyl ether; however, eye and nose irritation did occur as a result of five-minute exposures to 800 ppm. A 15-minute exposure to 500 ppm was not reported by volunteers to be irritating (Silverman, Schulte, and First 1946/Ex. 1–142). NIOSH was the only commenter to the record on this substance.

The final rule retains OSHA's former limit for isopropyl ether of 500 ppm as an 8-hour TWA. OSHA is retaining its former limit because the evidence suggests that, although some volunteers complained of the odor at 300 ppm, the concentration reached 800 ppm before volunteers experienced objectionable effects.

PHENYL ETHER

CAS: 101–84–8; Chemical Formula: $(C_6H_5)_2O$
H.S. No. 1314

OSHA is retaining its former 8-hour TWA limit of 1 ppm for phenylether, and NIOSH (Ex. 8–47, Table N1) supports this decision. The ACGIH recommends a TWA–TLV of 1 ppm and a 2-ppm 15-minute STEL for phenyl ether. Phenyl ether is a colorless liquid or solid with a low volatility; its vapor has a disagreeable odor.

The acute oral lethal dose is approximately 4 g/kg for rats and guinea pigs, and single doses of between 1 and 2 g/kg administered to various species have shown no liver, spleen, kidney, thyroidal, or gastrointestinal toxicities in surviving animals (Vogel, Snyder, and Schulman 1964/Ex. 1–681). Repeated inhalation studies in rats, rabbits, and dogs have shown that 20 exposures to 4.9 ppm for five days per week, seven hours per day produced no adverse effects. Eye and nasal irritation were observed in rats and rabbits exposed at 10 ppm (Hefner, Leong, Kociba, and Gehring 1975/Ex. 1–329). Skin and eye irritation have been reported only as a result of prolonged undiluted exposures. There is no evidence that skin absorption presents a health hazard (ACGIH 1986/Ex. 1–3, p. 475). The primary complaints associated with human exposures to phenyl ether vapor are of disagreeable odor and occasional nausea (Hake and Rowe 1963b, as cited in ACGIH 1986/Ex. 1–3, p. 475). NIOSH was the only commenter on this substance.

OSHA is retaining its former TWA limit of 1 ppm for phenyl ether; the Agency finds insufficient evidence to support the adoption of a STEL to complement the TWA.

VINYL TOLUENE

CAS: 25013–15–4; Chemical Formula: $CH_3C_6H_4CH{=}CH_2$
H.S. No. 1427

OSHA is retaining its limit of 100 ppm (8-hour TWA) for vinyl toluene. NIOSH (Ex. 8–47, Table N1) concurs with the retention of this limit. The ACGIH recommends a TWA of 50 ppm with a 100-ppm short-term exposure limit. Vinyl toluene is a colorless liquid with a strong, disagreeable odor.

Wolf, Rowe, McCollister et al. (1956/Ex. 1–404) noted fatty degeneration of the liver and an increase in kidney and liver weights in rats, guinea pigs, rabbits, and monkeys subjected to approximately 100 seven- to eight-hour exposures to vinyl toluene at 1250 ppm. Some deaths occurred among the rats in this group. Animals exposed to vinyl toluene at 600 ppm appeared normal and showed no blood or urine abnormalities, no gross or microscopic tissue changes, and no changes in growth rate or organ weight (Wolf, Rowe, McCollister et al. 1956/Ex. 1–404).

Human volunteers reported eye and nose irritation at 400 ppm and objectionable odor at 300 ppm. At 50 ppm, the odor of vinyl toluene was detectable, but no irritation was experienced and the odor was not intolerable (ACGIH 1986/Ex. 1–3, p. 630). NIOSH was the only commenter on this substance.

OSHA is retaining its 8-hour TWA limit of 100 ppm for vinyl toluene: the Agency finds that this level protects workers against the significant risk of intolerable odor and irritation caused by vinyl toluene exposures in the workplace. The Agency has found no health evidence to suggest that a short-term limit is necessary, and the final rule accordingly does not contain a STEL for vinyl toluene.

12. Substances for Which Limits are Based on Analogy to Related Substances

Introduction

OSHA is establishing limits for 73 substances on the basis of their toxicologic and structural similarities to other chemical substances that create significant risks of systemic toxicity, ocular effects, kidney or liver damage, and other similarly adverse health effects. For 46 of these substances, OSHA has not previously had Z-table limits. For an additional 11 substances, OSHA is reducing the 8-hour TWA limit, and in 13 cases, the Agency is retaining its 8-hour limit and adding a STEL to supplement the TWA. OSHA is deleting the 8-hour TWA limit and adding a ceiling in the case of acetic anhydride and deleting a ceiling limit and adding an 8-hour TWA for another substance. For one substance, OSHA proposed a reduction in the TWA PEL, but, after careful review of the scientific evidence and rulemaking record, the Agency has decided to retain the existing limit. Table C12–1 shows these substances, their CAS and HS numbers, and their former, proposed, and final rule limits.

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TABLE C12-1. Substances for Which Limits Are Based on Analogy to Related Substances

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1003 Acetic anhydride	108-24-7	5 ppm TWA	5 ppm Ceiling	5 ppm Ceiling
1009 Acrylic acid	79-10-7	—	10 ppm TWA	10 ppm TWA, Skin
1015 Aluminum (alkyls)	7429-90-5	—	2 mg/m^3 TWA	2 mg/m^3 TWA
1018 Aluminum (soluble salts)	7429-90-5	—	2 mg/m^3 TWA	2 mg/m^3 TWA
1040 Boron tribromide	10294-33-4	—	1 ppm Ceiling	1 ppm Ceiling
1043 Bromine pentafluoride	7789-30-2	—	0.1 ppm TWA	0.1 ppm TWA
1048 n-Butyl acrylate	141-32-2	—	10 ppm TWA	10 ppm TWA
1055 o-sec-Butylphenol	89-72-5	—	5 ppm TWA, Skin	5 ppm TWA, Skin
1059 Calcium hydroxide	1305-62-0	—	5 mg/m^3 TWA	5 mg/m^3 TWA
1060 Calcium oxide	1305-78-8	5 mg/m^3 TWA	2 mg/m^3 TWA	5 mg/m^3 TWA
1074 Carbonyl fluoride	353-50-4	—	2 ppm TWA 5 ppm STEL	2 ppm TWA 5 ppm STEL
1075 Catechol	120-80-9	—	5 ppm TWA	5 ppm TWA, Skin
1081 1-Chloro-1-nitro-propane	600-25-9	20 ppm TWA	2 ppm TWA	2 ppm TWA

TABLE C12-1. Substances for Which Limits Are Based on Analogy to Related Substances (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1098 Cobalt carbonyl, as Co	10210-68-1	—	0.1 mg/m^3 TWA	0.1 mg/m^3 TWA
1099 Cobalt hydrocarbonyl, as Co	16842-03-8	—	0.1 mg/m^3 TWA	0.1 mg/m^3 TWA
1118 Diazinon	333-41-5	—	0.1 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA, Skin
1121 1,1-Dichloro-1-nitro-ethane	594-72-9	10 ppm Ceiling	2 ppm TWA	2 ppm TWA
1125 p-Dichlorobenzene	106-46-7	75 ppm TWA	75 ppm TWA 110 ppm STEL	75 ppm TWA 110 ppm STEL
1128 Dichloromono-fluoromethane	75-43-4	1000 ppm TWA	10 ppm TWA	10 ppm TWA
1135 Diethyl ketone	96-22-0	—	200 ppm TWA	200 ppm TWA
1138 Diethylene triamine	111-40-0	—	1 ppm TWA, Skin	1 ppm TWA
1148 Dipropyl ketone	123-19-3	—	50 ppm TWA	50 ppm TWA
1150 Diquat	85-00-7	—	0.5 mg/m^3 TWA	0.5 mg/m^3 TWA
1152 Disulfoton	298-04-4	—	0.1 mg/m^3 TWA	0.1 mg/m^3 TWA, Skin

TABLE C12-1. Substances for Which Limits Are Based on Analogy to Related Substances (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1154 Divinyl benzene	108-57-6	—	10 ppm TWA	10 ppm TWA
1156 Endosulfan	115-29-7	—	0.1 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA, Skin
1181 Fonofos	944-22-9	—	0.1 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA, Skin
1182 Formamide	75-12-7	—	20 ppm TWA 30 ppm STEL	20 ppm TWA 30 ppm STEL
1186 Germanium tetra-hydride	7782-65-2	--	0.2 ppm TWA	0.2 ppm TWA
1212 Indene	95-13-6	—	10 ppm TWA	10 ppm TWA
1214 Iodoform	75-47-8	—	0.6 ppm TWA	0.6 ppm TWA
1219 Isobutyl alcohol	78-83-1	100 ppm TWA	50 ppm TWA	50 ppm TWA
1220 Isooctyl alcohol	26952-21-6	--	50 ppm TWA, Skin	50 ppm TWA, Skin
1229 n-Isopropylaniline	768-52-5	--	2 ppm TWA, Skin	2 ppm TWA, Skin
1231 Ketene	463-51-4	0.5 ppm TWA	0.5 ppm TWA 1.5 ppm STEL	0.5 ppm TWA 1.5 ppm STEL
1244 Methacrylic acid	79-41-4	—	20 ppm TWA	20 ppm TWA, Skin

TABLE C12-1. Substances for Which Limits Are Based on Analogy to Related Substances (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1247 4-Methoxyphenol	150-76-5	--	5 mg/m^3 TWA	5 mg/m^3 TWA
1250 Methyl acetylene-propadiene mixture	--	1000 ppm TWA	1000 ppm TWA 1250 ppm STEL	1000 ppm TWA 1250 ppm STEL
1256 Methyl demeton	8022-00-2	--	0.5 mg/m^3 TWA, Skin	0.5mg/m^3 TWA, Skin
1257 Methyl ethyl ketone peroxide	1338-23-4	--	0.2 ppm Ceiling	0.7 ppm Ceiling
1258 Methyl formate	107-31-3	100 ppm TWA	100 ppm TWA 150 ppm STEL	100 ppm TWA 150 ppm STEL
1259 Methyl iodide	74-88-4	5 ppm TWA, Skin	2 ppm TWA, Skin	2 ppm TWA, Skin
1260 Methyl isoamyl ketone	110-12-3	--	50 ppm TWA	50 ppm TWA
1262 Methyl isopropyl ketone	563-80-4	--	200 ppm TWA	200 ppm TWA
1265 Methyl parathion	298-00-0	-	0.2 mg/m^3 TWA, Skin	0.2 mg/m^3 TWA Skin

TABLE C12-1. Substances for Which Limits Are Based on Analogy to Related Substances (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1268 Methylcyclohexane	108-87-2	500 ppm TWA	400 ppm TWA	400 ppm TWA
1271 2-Methylcyclopenta-dienyl Mn tricarbonyl, as Mn	12108-13-3	—	0.2 mg/m^3 TWA, Skin	0.2 mg/m^3 TWA, Skin
1279 Monocrotophos	6923-22-4	--	0.25 mg/m^3 TWA	0.25 mg/m^3 TWA
1281 Morpholine	110-91-8	20 ppm TWA, Skin	20 ppm TWA 30 ppm STEL, Skin	20 ppm TWA 30 ppm STEL, Skin
1286 Nitric acid	7697-37-2	2 ppm TWA	2 ppm TWA 4 ppm STEL	2 ppm TWA 4 ppm STEL
1287 p-Nitroaniline	100-01-6	6 mg/m^3 TWA, Skin	3 mg/m^3 TWA, Skin	3 mg/m^3 TWA, Skin
'292 Nitrotoluene				
o-isomer	88-72-2;	5 ppm TWA, Skin	2 ppm TWA, Skin	2 ppm TWA, Skin
m-isomer	99-08-1;			
p-isomer	99-99-0			
1293 Nonane	111-84-2	-	200 ppm TWA	200 ppm TWA
1299 Oxalic acid	144-62-7	1 mg/m^3 TWA	1 mg/m^3 TWA 2 mg/m^3 STEL	1 mg/m^3 TWA 2 mg/m^3 STEL
1309 Perchloryl fluoride	7616-94-6	3 ppm TWA	3 ppm TWA 6 ppm STEL	3 ppm TWA 6 ppm STEL

TABLE C12-1. Substances for Which Limits Are Based on Analogy to Related Substances (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1320 Phosdrin (Mevinphos)	7786-34-7	0.1 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA 0.3 mg/m^3 STEL, Skin	0.1 mg/m^3 TWA 0.3 mg/m^3 STEL, Skin
1323 Phosphorus oxychloride	10025-87-3	—	0.1 ppm TWA 0.5 ppm STEL	0.1 ppm TWA
1324 Phosphorus pentasulfide	1314-80-3	1 mg/m^3 TWA	1 mg/m^3 TWA 3 mg/m^3 STEL	1 mg/m^3 TWA 3 mg/m^3 STEL
1326 Phthalic anhydride	85-44-9	2 ppm TWA	1 ppm TWA	1 ppm TWA
1335 Propargyl alcohol	107-19-7	—	1 ppm TWA, Skin	1 ppm TWA, Skin
1336 Propionic acid	79-09-4	—	10 ppm TWA 15 ppm STEL	10 ppm TWA
1338 n-Propyl acetate	109-60-4	200 ppm TWA	200 ppm TWA 250 ppm STEL	200 ppm TWA 250 ppm STEL
1339 n-Propyl alcohol	71-23-8	200 ppm TWA	200 ppm TWA 250 ppm STEL, Skin	200 ppm TWA 250 ppm STEL
1344 Propylene oxide	75-56-9	100 ppm TWA	20 ppm TWA	20 ppm TWA

TABLE C12-1. Substances for Which Limits Are Based on Analogy to Related Substances (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1361 Silicon tetrahydride	7803-62-5	—	5 ppm TWA	5 ppm TWA
1379 Sulfuryl fluoride	2699-79-8	5 ppm TWA	5 ppm TWA 10 ppm STEL	5 ppm TWA 10 ppm STEL
1393 Thionyl chloride	7719-09-7	—	1 ppm Ceiling	1 ppm Ceiling
1402 Tributyl phosphate	126-73-8	5 mg/m^3 TWA	2.5 mg/m^3 TWA	2.5 mg/m^3 TWA
1404 Trichloroacetic acid	76-03-9	—	1 ppm TWA	1 ppm TWA
1411 Trimethylamine	75-50-3	—	10 ppm TWA 15 ppm STEL	10 ppm TWA 15 ppm STEL
1420 n-Valeraldehyde	110-62-3	—	50 ppm TWA	50 ppm TWA
1432 m-Xylene-alpha, alpha', diamine	1477-55-0	—	0.1 mg/m^3 Ceiling, Skin	0.1 mg/m^3 Ceiling, Skin
1433 Xylidine	1300-73-8	5 ppm TWA, Skin	2 ppm TWA, Skin	2 ppm TWA, Skin

* OSHA's TWA limits are for 8-hour exposures; its SILLs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time

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Description of the Health Effects

The health effects associated with occupational exposures to the diverse group of substances shown in Table C12–1 vary widely, ranging from sensory irritation, systemic toxicity, ocular effects, and neuropathy to renal and liver damage. This variation in target organs reflects the fact that the substances in this group have not been grouped on the basis of similarity in toxic effects, target organs, or mechanism of action; instead, they are considered together because the specific limits being established for them have been set on the basis of toxic effects caused by exposure to analogous chemicals. Table C12–2 shows these substances, along with their adverse health effects and the substances with which they share structural similarities.

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TABLE C12-2. Summary of Rationale for Limits Based on Analogy to Related Substances

H.S. Number/ Chemical Name	Substance with Analogous Structure or Activity	Associated Health Effects
1003 Acetic anhydride	Acetic acid	Sensory irritation
1009 Acrylic acid	Acetic acid	Sensory irritation
1015 Aluminum (alkyls)	Welding fumes	Pulmonary irritation
1018 Aluminum (soluble salts)	Hydrogen chloride	Sensory irritation
1040 Boron tribromide	Hydrogen bromide	Sensory irritation
1043 Bromine pentafluoride	Chlorine terifluoride	Systemic injury
1048 n-Butyl acrylate	Methyl acrylate	Sensory Irritation
1055 o-sec-Butylphenol	Phenol and cresol	Respiratory, liver, and kidney effects
1059 Calcium hydroxide	Sodium hydroxide	Sensory irritation
106C Calcium oxide	Sodium hydroxide	Sensory irritation
1074 Carbonyl fluoride	Hydrolysis to hydrogen fluoride	Sensory irritation

TABLE C12-2. Summary of Rationale for Limits Based on Analogy to Related Substances (continued)

H.S. Number/ Chemical Name	Substance with Analogous Structure or Activity	Associated Health Effects
1075 Catechol	Phenol	Peripheral vaso-constriction, renal tubule degeneration
1081 1-Chloro-1-nitropropane	Nitropropane	Damage to heart muscle, liver, and kidneys
1098 Cobalt carbonyl	Nickel carbonyl	Systemic toxicity
1099 Cobalt hydrocarbonyl	Nickel carbonyl	Systemic toxicity
1118 Diazinon	Parathion	Cholinesterase inhibition
1121 1,1-Dichloro-1-nitroethane	Related compounds	Systemic toxicity
1125 p-Dichlorobenzene	o-Dichlorobenzene	Neurological effects, cataract formation
1128 Dichlorofluoromethane	Chloroform	Hepatotoxicity, cardiac sensitization
1135 Diethyl ketone	Methyl propyl ketone	Narcosis
1138 Diethylenetriamine	Ethylamine	Irritation, sensitization

TABLE C12-2. Summary of Rationale for Limits Based on Analogy to Related Substances (continued)

H.S. Number/ Chemical Name	Substance with Analogous Structure or Activity	Associated Health Effects
1148 Dipropyl ketone	Methyl isobutyl ketone	Narcosis
1150 Diquat	Paraquat	Ocular effects
1152 Disulfoton	Parathion	Cholinesterase inhibition
1154 Divinyl benzene	Styrene	Sensory irritation
1156 Endosulfan	Aldrin, Dieldrin	Neurological effects
1181 Fonofos	Ethyl parathion	Cholinesterase inhibition
1182 Formamide	Dimethyl formamide	Testicular toxicity, teratogenicity
1186 Germanium tetrahydride	Stibine	Hemolytic effects
1212 Indene	Naphthalene	Sensory irritation
1214 Iodoform	Methyl iodide	Irritation, hepatotoxicity
1219 Isobutyl alcohol	n-Butanol	Irritation, narcosis
1220 Isooctyl alcohol	Isoamyl alcohol	Sensory irritation

TABLE C12-2. Summary of Rationale for Limits Based on Analogy to Related Substances (continued)

H.S. Number/ Chemical Name	Substance with Analogous Structure or Activity	Associated Health Effects
1229 n-Isopropylaniline	Aniline, N,N-dimethylaniline	Hemolytic effects
1231 Ketene	Phosgene	Sensory irritation
1244 Methacrylic acid	Acrylic acid	Sensory irritation
1247 4-Methoxyphenol	Hydroquinone	Ocular effects
1250 Methyl acetylene-propadiene mixture	Methyl acetylene	
1256 Methyl demeton	Demeton	Ocular effects, respiratory effects, inner ear irritation
1257 Methyl ethyl ketone peroxide	Benzoyl peroxide, hydrogen peroxide	Sensory irritation
1258 Methyl formate	Methyl acetate	Sensory irritation
1259 Methyl iodide	Methyl bromide	Central nervous system effects
1260 Methyl isoamyl ketone	Methyl isobutyl ketone	Neuropathy

TABLE C12-2. Summary of Rationale for Limits Based on Analogy to Related Substances (continued)

H.S. Number/ Chemical Name	Substance with Analogous Structure or Activity	Associated Health Effects
1262 Methyl isopropyl ketone	Diethyl ketone, methyl propyl ketone	Narcosis, sensory irritation
1265 Methyl parathion	Parathion	Cholinesterase inhibition
1268 Methylcyclohexane	Heptane	Irritation
1271 Methylcyclopentadienyl manganese tricarbonyl	Tetraethyl lead	Central nervous system effects, chronic lung effects
1279 Monocrotophos	Parathion	Cholinesterase inhibition
1281 Morpholine	Ammonia	Kidney and liver degeneration, sensory irritation
1286 Nitric acid	Hydrogen chloride, sulfuric acid	Sensory irritation
1287 p-Nitroaniline	Aniline	Methemoglobin formation
1292 Nitrotoluene	Aniline	Methemoglobin formation
1293 Nonane	Octane	Narcosis

TABLE C12-2. Summary of Rationale for Limits Based on Analogy to Related Substances (continued)

H.S. Number/ Chemical Name	Substance with Analogous Structure or Activity	Associated Health Effects
1299 Oxalic acid	Sulfuric acid, phosphoric acid	Irritation, burns
1309 Perchloryl fluoride	Fluoride	Fluorosis
1320 Phosdrin (Mevinphos)	Parathion	Cholinesterase inhibition
1323 Phosphorus oxychloride	Phosphorous trichloride	Sensory irritation, respiratory effects
1234 Phosphorus pentasulfide	Phosphoric acid	Sensory irritation
1326 Phthalic anhydride	Tetrachlorophthalic anhydride, maleic anhydride	Sensory irritation
1335 Propargyl alcohol	Allyl alcohol	Sensory irritation
1336 Propionic acid	Acetic acid	Sensory irritation
1338 n-Propyl acetate	Isopropyl acetate, n-butyl acetate	Sensory irritation
1339 n-Propyl alcohol	Isopropyl alcohol	Sensory irritation

TABLE C12-2. Summary of Rationale for Limits Based on Analogy to Related Substances (continued)

H.S. Number/ Chemical Name	Substance with Analogous Structure or Activity	Associated Health Effects
1344 Propylene oxide	Ethylene oxide	Central nervous system depression, sensory irritation
1361 Silicon tetrahydride	Germane, stannane	Sensory irritation
1379 Sulfuryl fluoride	Hydrogen fluoride	Fluorosis
1393 Thionyl chloride	Hydrogen chloride	Sensory irritation
1402 Tributyl phosphate	Triphenyl phosphate	Narcosis, cholinesterase inhibition
1404 Trichloroacetic acid	2,2-Dichloropropionic acid	Sensory irritation
1411 Trimethylamine	Dimethylamine	Sensory irritation
1420 n-Valeraldehyde	Saturated aliphatic aldehydes	Sensory irritation
1432 m-Xylene-alpha,alpha', diamine	Phenylenediamine	Allergic respiratory sensitization
1433 Xylidine	Aniline	Methemoglobin formation

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The use of structural analogy is a reasonable methodology for limit-setting because of the similarities in structure and activity between each substance in this grouping and at least one other toxic substance. Industrial hygienists and toxicologists frequently use this approach when dealing with lesser-known substances either in the workplace or the laboratory. The limits for the substances in this grouping have been set based on dose-response information for other compounds that have a similar chemical structure or that are known to have a similar mechanism of action. For example, limits are being established for a number of substances that are known cholinesterase inhibitors (including diazinon, disulfoton, and monocrotophos); however, since direct dose-response data are not available for these substances specifically, OSHA has established limits that are similar to the final rule's limit for parathion, another cholinesterase inhibitor for which adequate dose-response data are available.

It is important to note that the establishment of a limit on the basis of analogy to other substances does not reflect a lack of information showing that the substance is toxic; acute animal data are available to demonstrate the toxicity of all of the substances for which limits are being established in this category, and, for several substances, there are case reports of human poisonings caused by exposure. Thus the limits established for these substances reflect much more than a theoretical consideration of chemical structure and physiologic reaction: the hazardous nature of exposure to every substance in this category has been demonstrated beyond doubt, although the precise level at which these effects will occur cannot be foretold with certainty.

The following sections describe the record evidence and OSHA's findings for the substances in this grouping. They also discuss the material health impairments likely to occur as a consequence of occupational exposure to these substances.

ACETIC ANHYDRIDE

CAS: 108-24-7; Chemical Formula: $(CH_3CO)_2O$
H.S. No. 1030

The former OSHA PEL for acetic anhydride was 5 ppm as an 8-hour TWA. The ACGIH has a TLV of 5 ppm as a ceiling, based on analogy with acetic acid's (TLV = 5 ppm ceiling) irritant potential. The proposed PEL was 5 ppm as a ceiling, and NIOSH (Ex. 8-47, Table N1) concurs with this limit, which is established by the final rule. Acetic anhydride is a colorless, mobile, strongly refractive liquid with a strong odor.

In one study, rats inhaling 1000 ppm of acetic anhydride for four hours survived, but 2000 ppm was fatal (Smyth 1956/Ex. 1-759). In human studies, eye, nose, and throat irritation has been observed, and it has been suggested that bronchial and lung injury may occur as a consequence of exposure (Henderson and Haggard 1943j, as cited in ACGIH 1986/Ex. 1-3, p. 5). Skin burns and serious corneal injury have been reported in industrial settings when workers came into contact with the liquid (McLaughlin 1946/Ex. 1-641), and acetic anhydride is a marked lacrimator (Fairhall 1949b, as cited in ACGIH 1986/Ex. 1-3, p. 5).

In light of acetic anhydride's potential for acute toxicity, OSHA is replacing the former 5-ppm 8-hour TWA with a 5-ppm ceiling. The Agency concludes that this limit will protect workers from the significant risk of ocular and respiratory effects associated with high, short-term exposures to acetic anhydride at the former level. Ocular and respiratory effects constitute material impairments of health. The final rule's limit will substantially reduce these risks among industrially exposed workers.

ACRYLIC ACID

CAS: 79-10-7; Chemical Formula: $CH_2 = CHCO_2H$
H.S. No. 1009

Previously, OSHA had no permissible exposure limit for acrylic acid. The ACGIH has an 8-hour TLV-TWA of 10 ppm. The propsed PEL was 10 ppm as an 8-hour TWA, and the final rule establishes this limit and adds a skin notation. Acrylic acid is a colorless, corrosive liquid with a distinctive acrid odor.

Acrylic acid is known to polymerize explosively with amines, ammonia, oleum, and chlorosulfonic acid, and it is incompatible with strong alkalis and pure nitrogen. Occupational exposure to acrylic acid usually occurs when the chemical is used in the form of methyl, ethyl, or butyl esters in the manufacture of acrylic resins.

Data indicate that the oral LD_{50} in rats is between 0.25 and 0.5 mg/kg (Dow Chemical Company 1977f, as cited in ACGIH 1986/Ex. 1-3, p. 14), and the skin absorption LD_{50} in rabbits is 0.95 ml/kg (Smyth, Carpenter, Weil et al. 1962/Ex. 1-441). Another study indicates that rabbits given acrylic acid orally had no ill effects at a level of 0.025 mg/kg (Klimkina et al. 1969, as cited in ACGIH 1986/Ex. 1-3, p. 14), and Gage (1970/Ex. 1-318) reports that rats exposed to 80 ppm for 6 hours daily for 20 days showed no adverse effects.

Case reports indicate that acute exposures to acrylic acid in workers have caused skin burns, eye burns, and upper respiratory effects (ACGIH 1986/Ex. 1-3, p. 14). OSHA received a comment from the Basic Acrylic Monomer Manufacturers (Ex. 184) supporting the proposed 10-ppm TWA limit. The New Jersey Department of Health (Ex. 144) discussed acrylic acid in connection with the Department's recommendation that OSHA use EPA's IRIS data as the basis for limit-setting; OSHA has discussed this approach in Section VI.A of this preamble. NIOSH (Ex. 8-47, Table N2) believes that the limit being established by OSHA for acrylic acid should be lower, based on recent studies demonstrating degeneration of the nasal mucosa, changes in pulmonary function, and skin absorption (miller, Ayres, Jersey, and Mckenna 1981 and Silver, Leith, and Murphy 1981, both as cited in ACGIH 1986/Ex. 1-3, p 14.1). OSHA is aware of the recent literature on acrylic acid and will continue to monitor it in the future.

OSHA concludes that an 8-hour TWA PEL of 10 ppm and a skin notation are necessary to protect workers from the significant risk of nasal and eye irritation, which constitute material health impairments that are potentially associated with exposure to acrylic acid at levels above the new limit. The Agency has determined that this limit will substantially reduce this risk and prevent recurrences of the burns and irritation previously associated with industrial exposures to acrylic acid.

ALUMINUM (ALKYLS)

CAS: 7429-90-5
Chemical Formula: Al
H.S. No. 1015

ALUMINUM (SOLUBLE SALTS)

CAS: 7429-90-5
Chemical Formula: Al
H.S. No. 1018

OSHA formerly had no permissible exposure limits for either the soluble salts of aluminum or the aluminum alkyls. The ACGIH has an 8-hour TLV-TWA limit of 2 mg/m ^{3} for aluminum (soluble salts) and 2 mg/m^3 for the aluminum alkyls. The proposed PELs were 2 mg/m^3 for both groups of aluminum compounds. and NIOSH (Ex. 8-47, Table N1) concurred that these limits were appropriate. The final rule establishes a 2-mg/m^3 8-hour TWA PEL for the aluminum alkyls and the soluble salts of aluminum.

The ACGIH's limits for aluminum soluble salts have been set on the basis of the amount of hydrolized acid, such as hydrochloric acid or sulphuric acid, in their acid compounds. For example,

three mols of hydrogen chloride (HCl) hydrolize from one mol of aluminum chloride; since HCl has a PEL of 5 ppm, a PEL of 2 mg/m³ for aluminum chloride (which is a soluble salt of aluminum) would provide the same degree of protection from irritation as that provided by this limit for HCl. The acute toxicity of aluminum chloride is generally representative of the toxicity of all of the soluble salts of aluminum. For the aluminum alkyls, toxicity data are sparse. However, all of the nonhalogenated alkyls decompose into aluminum oxide fume, and the halogenated alkyls are even more irritating because of acid hydrolysis.

In the final rule, OSHA is establishing an 8-hour TWA limit of 2 mg/m³, for both the soluble salts of aluminum and the aluminum alkyls. The Agency concludes that these limits will protect against the significant risk of irritation and skin burns, which constitute material health impairments that are associated with exposures at levels above the new PEL.

BORON TRIBROMIDE

CAS: 10294-33-4; Chemical Formula: BBr_3
H.S. No. 1040

OSHA formerly had no limit for exposure to boron tribromide. The ACGIH has a 1-ppm ceiling limit for boron tribromide, which is a colorless, fuming liquid that is decomposed by water and alcohol. The proposed PEL, with which NIOSH (Ex. 8-47, Table N1) concurred, was a ceiling of 1 ppm; this limit is established in the final rule.

Boron tribromide has a high potential for acute local irritation, and its potential for systemic toxicity is analogous with that of hydrogen bromide (HBr). On decomposition, one molecule of boron tribromide would be expected to produce three molecules of HBr (ACGIH 1986/Ex. 1-3, p. 62).

Animals repeatedly exposed to boron tribromide develop pneumonia, and exposure to 100 ppm caused a uniformly high mortality rate in animals from six laboratory species (Stokinger, Spiegel et al. 1953, as cited in ACGIH 1986/Ex. 1-3, p. 63). Rats, rabbits, and mice exposed at 1.5, 3.4, or 12.8 ppm boron trifluoride developed pneumonitis and dental fluorosis, although, at the lowest level tested, the evidence of pneumonitis was described as "marginal" (Torkelson, Sadek, and Rowe 1961, as cited in ACGIH 1986/Ex. 1-3, p. 63).

Based on this evidence of boron tribromide's severe pulmonary toxicity at exposure levels of 3.4 ppm, OSHA is establishing a ceiling limit of 1 ppm. The Agency concludes that this limit will protect workers from the significant risk of serious pulmonary damage, a material health impairment that is associated with exposure to this substance at levels above the new PEL.

BROMINE PENTAFLUORIDE

CAS: 7789-30-2; Chemical Formula: BrF_5
H.S. No. 1043

OSHA had no former limit for bromine pentafluoride. The ACGIH has TLV-TWA of 0.1 ppm. The proposed PEL was 0.1 ppm as an 8-hour TWA, and NIOSH (Ex. 8-47, Table N1) concurs with this limit. The final rule promulgates a 0.1-ppm 8-hour TWA PEL for bromine pentafluoride. This substance is a pale yellow liquid at temperatures below 40.3°C; above this temperature, is a colorless, pungent, corrosive gas.

Bromine pentafluoride has been shown to be acutely toxic in animals. Animals exposed to bromine pentafluoride vapor at 500 ppm exhibited immediate symptoms of gasping, swollen eyelids, clouded corneas, tearing, salivation, and acute distress; these symptoms appeared after exposure for a period as short as three minutes. Exposures to 50 ppm were fatal after 30 minutes, and chronic exposure above 3 ppm resulted in severe nephrosis (in some animals), as well as marked hepatosis and severe respiratory involvement (The Matheson Co., Inc., as cited in ACGIH 1986/Ex. 1-3, p. 66). Bromine pentafluoride is toxicologically more active than free, elemental fluorine, and its toxicity appears to be closely related to that of chlorine trifluoride (Horn and Weir 1955/Ex. 1-592; Horn and Weir 1956, as cited in ACGIH 1986/Ex. 1-3, p. 66). Chlorine trifluoride has caused severe toxicity and some fatalities in dogs and rats exposed over a period of six months to an average concentration of 1.17 ppm for six hours daily (Horn and Weir 1955/Ex. 1-592).

In the final rule, OSHA is establishing a PEL of 0.1 ppm as an 8-hour TWA to prevent the significant risk of serious systemic injury potentially associated with exposure to this substance at levels above the new limit. The Agency concludes that this limit will substantially reduce this risk of systemic toxicity, which constitutes a material impairment of health.

n-BUTYL ACRYLATE

CAS: 141-32-2; Chemical Formula: $C_7H_{12}O_2$
H.S. No. 1048

OSHA had no former limit for n-butyl acrylate. The ACGIH's Threshold Limit Value is a 10-ppm TWA. The proposed PEL, with which HIOSH (Ex. 8-47, Table N1) concurs, was 10 ppm as an 8-hour TWA, and this limit is promulgated by the final rule, n-Butyl acrylate is a colorless, flammable liquid.

n-Butyl acrylate is a skin and eye irritant and is toxic to animals. The LC_{50} for a 4-hour exposure was 1000 ppm (Carpenter, Weil, and Smith 1974/Ex. 1-304). In rabbits, the dermal LD_{50} for n-butyl acrylate is approximately 1800 mg/kg, compared with 1235 mg/kg for methyl acrylate (Smyth, Carpenter, and Weil 1951/Ex. 1-439). n-Butyl acrylate has also been found to be mildly irritating to the skin and to produce corneal necrosis in the unwashed eyes of rabbits (Holland 1974, as cited in ACGIH 1986/Ex. 1-3, p. 75).

At the rulemaking hearing, Dr. Isadore Rosenthal from Rohm and Haas was asked to submit to the docket any information on butyl acrylate that Rohm and Haas had in its possession and had not previously transmitted either to the ACGIH or to OSHA. In response. Dr. Rosenthal (Ex. 112) submitted a 1974 internal memo reporting on butyl acrylate's overall toxicity. These data report, among other things, that the dermal LD_{50} in rabbits for this substance is about 1800/ mg/kg (Ex. 112).

In the final rule, OSHA is establishing an 8-hour TWA PEL of 10 ppm for n-butyl acrylate, based on the similarity of the toxicological response of n-butyl acrylate to methyl acrylate, for which OSHA also has a 10-ppm TWA limit. The Agency concludes that this limit is necessary to reduce the significant risk of skin irritation and corneal necrosis, which constitute material health impairments.

o-sec-BUTYLPHENOL

CAS: 89-72-5; Chemical Formula: $C_2H_5(CH_3)CHC_6H_4OH$
H.S. No. 1055

OSHA had no former limit for o-sec-butylphenol. The ACGIH has a 5-ppm 8-hour TLV-TWA, with a skin notation. The proposed PEL was an 8-hour TWA of 5 ppm, with a skin notation; NIOSH (Ex. 8-47, Table N1) concurs with this limit, which is established in the final rule. o-sec-Butylphenol is a colorless liquid.

Animal studies indicate that contact with o-sec-butylphenol causes irritation of the skin, eyes, and respiratory tract, and may result in skin burns. A Dow Chemical Company study (1977i, as cited in ACGIH 1986/Ex. 1-3, p. 84) showed that the oral and skin absorption LD_{50}s for guinea pigs ranged between 0.6 and 2.4 g/kg. Prolonged contact of o-sec-butylphenol with the skin of these animals resulted in burns, whereas direct application to the eyes did not cause corneal injury. The oral LD_{50} for rats is 2700 mg/kg (*Dangerous*

Properties of Industrial Materials, 6th ed., Sax 1984), and rats exposed to saturated air levels of this chemical survived for seven hours (Dow Chemical Company 19771, as cited in ACGIH 1986/Ex. 1–3, p. 84). The intravenous LD_{50} for mice is 6 mg/kg (Sax 1984). Acute workplace exposure to o-sec-butylphenol have resulted in mild respiratory irritation and skin burns (ACGIH 1986/Ex. 1–3, p. 84).

In the final rule, OSHA is establishing an 8-hour TWA limit of 5 ppm for o-sec-butylphenol, with a skin notation. The Agency concludes that this limit is necessary to protect workers from the significant risks of eye and respiratory tract irritation and skin burns associated with exposure to this substance at the levels formerly permitted by the absence of an OSHA limit. Eye and mucous membrane irritation and skin burns constitute material health impairments within the meaning of the Act.

CALCIUM HYDROXIDE

CAS: 1305–62–0; Chemical Formula: $Ca(OH)_2$
H.S. No. 1059

OSHA formerly had no limit for calcium hydroxide; the ACGIH has a TLV–TWA of 5 mg/m³. In the proposal, the PEL was 5 mg/m³ as an 8-hour TWA, and NIOSH (Ex. 8–47, Table N1) concurred with this limit. The final rule promulgates an 8-hour TWA PEL of 5 mg/m³ for calcium hydroxide. Calcium hydroxide is a soft, white, odorless, crystalline powder with an alkaline, bitter taste.

Calcium hydroxide is a moderate to severe caustic irritant when it comes in contact with the skin, eyes, or mucous membranes of the upper respiratory tract (ACGIH 1986/EX. 1–3, p. 92; Sax and Lewis 1989, p. 682). The oral LD_{50} in rats is reported to be 7.34 g/kg (Smyth, Carpenter, Weil et al. 1969/Ex. 1–442). Industrial experience with this substance has not shown a high incidence of adverse health effects, although Sax (*Dangerous Properties of Industrial Materials,* 6th ed., 1984) reports that it is known to cause dermatitis (p. 621). Calcium hydroxide is also mutagenic (*Dangerous Properties of Industrial Materials,* 7th ed., Sax and Lewis 1989, p. 682). Calcium hydroxide has less alkalinity than the hydroxides of the alkali series, and the ACGIH has suggested that limits for exposures to calcium hydroxide should be based on its total alkalinity.

OSHA received only one comment other than NIOSH's on calcium hydroxide. The National Lime Association (NLA) (Ex. 3–890) raised several issues related to this substance. First, the NLA argues that the health evidence for calcium hydroxide does not support a PEL of 5 mg/m³ for this substance. According to the NLA, the oral toxicity study described by OSHA in the proposal has no relevance to airborne lime exposures (Ex. 3–890, p. 13). In addition, the NLA believes that calcium hydroxide should be regulated as a nuisance dust, with a PEL of 10 mg/m³. OSHA does not agree that calcium hydroxide is a biologically inert substance. The Agency agrees with Sax (1984), who reports that, "in the form of dust, it ½ calcium hydroxide ¾ is considered to be an important industrial hazard." OSHA finds that a PEL of 5 mg/m³, half that of the inert particulate limit, is appropriate for this well-known eye, skin, and upper respiratory tract irritant.

The NLA's second point is that the monitoring methods available for measuring workplace exposures to lime are inadequate to distinguish between "different compounds of calcium" because they are "element- not compound-specific" (Ex. 3–890). OSHA's decision in the final rule to establish 5-mg/m³ limits for both calcium oxide and calcium hydroxide (see the discussion below for calcium oxide) should eliminate this problem for affected employers.

In the final rule, OSHA is establishing an 8-hour TWA limit for calcium hydroxide of 5 mg/m³ to protect against the significant risk of skin, eye, and mucous membrane irritation, which are material impairments of health that are caused by exposure to this substance at levels above the new PEL. The Agency concludes that this limit will reduce these risks substantially.

CALCIUM OXIDE

CAS: 1305–78–8; Chemical Formula : CaO
H.S. No. 1060

OSHA's former 8-hour TWA permissible exposure limit for calcium oxide was 5 mg/m³, and the proposal contained a revised 8-hour TWA PEL of 2 mg/m³ for this substance. NIOSH (Ex. 8–47, Table N1) concurred with this proposal. This revised limit was consistent with the ACGIH TLV for calcium oxide, which was set on the basis of analogy with sodium hydroxide, a widely recognized sensory irritant. The final rule retains OSHA's former 5-mg/m³ 8-hour TWA PEL for calcium oxide, for the reasons discussed below.

Calcium oxide (lime) is produced when limestone is calcined to drive off carbon dioxide. Calcium oxide is used as a refractory material; as a flux in steelmaking; as a binding agent in building, pulp and paper manufacture, sugar refining, and leather tanning; as the raw material for chlorinated lime bleaching powder, and as a soil treatment in agriculture (*Encyclopedia of Occupational Health and Safety,* Vol. 2, p. 1234, International Labour Office 1983).

The amount of information that has been published specifically about calcium oxide's toxicological effects in animals or humans is limited, which accounts for the ACGIH's reliance on the similarity in action between calcium oxide and sodium hydroxide in establishing a TLV of 2 mg/m³ for calcium oxide. The National Lime Association (NLA) (Ex. 3–890) and the American Iron and Steel Institute (Tr. p. 11–130 to 11–131; Ex. 188) objected to the comparison of calcium oxide's properties with those of sodium hydroxide; according to the NLA, "no qualitative or quantitative analysis is offered [in the proposal] to support the use of this analogy." OSHA's analysis of this issue is discussed below.

In direct contact with tissues, calcium oxide can result in burns and severe irritation because of its high reactivity and alkalinity. The major complaints of workers exposed to lime consist of irritation of the skin and eyes, although inflammation of the respiratory passages, ulceration and perforation of the nasal septum, and even pneumonia have been attributed to inhalation of the dust (ACGIH 1986/ Ex. 1–3, p. 92). The Pennsylvania Department of Health reported that strong nasal irritation occurred as a consequence of exposure to a mixture of calcium-oxide-containing dusts at a concentration of approximately 25 mg/m³, but that exposure to concentrations of 9 to 10 mg/m ³ produced no observable irritation (Wands 1981a, in *Patty's Industrial Hygiene and Toxicology,* 3rd rev. ed., Vol. 2B, p. 3054). By comparison, exposure to airborne sodium hydroxide at a concentration of between 0.005 and 0.7 mg/m³ produced burning/redness of the nose, throat, or eyes in workers engaged in cleaning operations (Hervin and Cohen 1973/EX. 1–945, as cited in NIOSH 1976k/Ex. 1–965). Thus, the demonstrated effect level for sensory irritation caused by exposure to sodium hydroxide is below 1 mg/m³, while that for calcium oxide is above 9 mg/m³.

OSHA finds that analogy with sodium hydroxide is not an appropriate basis for establishing a PEL for calcium oxide, because there is nearly a tenfold difference in the no-effect levels for these two substances. Based on evidence that exposure to calcium oxide at levels above 9 mg/m³ may cause eye-tearing and mucous membrane irritation,

OSHA concludes that the Agency's former limit of 5 mg/m^3 as an 8-hour TWA continues to be appropriate for this substance. The Agency concludes that this limit protects exposed workers from the significant risk of sensory irritation known to occur at concentrations of 9 to 10 mg/m^3.

CARBONYL FLUORIDE

CAS: 353-50-4; Chemical Formula: COF_2
H.S. No. 1074

OSHA had no former limit for carbonyl fluoride. The ACGIH has an 8-hour TWA limit of 2 ppm and a 15-minute STEL of 5 ppm for this colorless and essentially odorless gas. The proposed PELs were an 8-hour TWA of 2 ppm and a 15-minute STEL of 5 ppm. NIOSH (Ex. 8-47, Table N1) concurred with these limits, and they are established in the final rule.

The 1-hour LC_{50} for rats is 360 ppm, and the 4-hour LC_{50} for the same species is 90 ppm (ACGIH 1986/Ex. 1-3, p. 111). Carbonyl fluoride hydrolyzes instantly on contact with moisture. The ACGIH (1986/Ex. 1-3, p. 14) reports that carbonyl fluoride is "about as toxic as hydrogen fluoride as a respiratory irritant gas."

Repeated exposure of animals to carbonyl fluoride is known to have metabolic effects; it inhibits the fluoride-sensitive enzyme succinic dehydrogenase via hydrolysis of carbonyl fluoride to hydrogen fluoride (Scheel, McMillan, and Phipps 1968/Ex. 1-364). Carbonyl fluoride is also a strong irritant to the eyes, skin, mucous membranes, and respiratory tract (*Dangerous Properties of Industrial Materials*, 6th ed., Sax 1984). The only comment on carbonyl fluoride came from NIOSH.

In the final rule, OSHA is establishing an 8-hour TWA limit of 2 ppm and a 15-minute 5-ppm STEL for carbonyl fluoride; these limits are based on analogy with the 3-ppm TWA limit being established for hydrogen fluoride. The Agency concludes that both a TWA and a STEL are necessary to provide protection against the significant risks of marked irritation and metabolic effects, which constitute material health impairments that are associated with exposure to carbonyl fluoride at levels above the new PELs.

CATECHOL (PYROCATECHOL)

CAS: 120-80-9; Chemical Formula: $C_6H_4(OH)_2$
H.S. No. 1075

OSHA formerly had no established limit for catechol. The ACGIH has a TLV-TWA of 5 ppm. the proposed PEL was 5 ppm as an 8-hour TWA, and NIOSH (Ex. 8-47, Table N1) concurs with this limit, which is established in the final rule. In addition, the Agency has added a skin notation for this substance, in accordance with its policy on skin designations, as discussed in Section VI.C.18. Catechol is a colorless crystalline solid that sublimes readily and thus occurs in the vapor state at room temperature.

Catechol is approximately 1.1 to 2.2 times more toxic than phenol, dependidng on the route of exposure (Industrial Bio-Test Laboratories 1974, as cited in ACGIH 1986/Ex. 1-3, p. 112). The oral LD_{50} in rats is 300 mg/kg, or approximately half that of phenol. Percutaneous toxicity for catechol in rabbits is 800 mg/kg, only slightly greater than the value for phenol. OSHA notes that phenol has a skin designation and that catechol's dermal LD_{50} in rabbits of 0.8 g/kg places this substance in the category of "toxic" by the percutaneous route of administration, as discussed in Section VI.C.18. In addition, the Agency is concerned by reports of central nervous system effects (i.e., convulsions) in humans as a result of skin absorption that are "more marked" than those produced by phenol (Deichmann and Keplinger 1981, in *Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., Vol. 2A, p. 2586). OSHA is therefore adding a skin notation to the final limit for catechol to protect workers from the serious CNS effects that may potentially occur from percutaneous absorption of this substance. Eye and nose irritation, as well as muscular spasms and tremor, have been observed in rats at a concentration of 2800 mg/m^3 catechol, indicating that the acute respiratory toxicity of catechol is approximately one-third that of phenol (Industrial Bio-Test Laboratories 1974, as cited in ACGIH 1986/Ex. 1-3, p.112). Metabolic data indicate that the urinary elimination rate of catechol in rabbits is only 10 percent that of phenol (Williams 1959/Ex.1-1176). In mice, catechol is easily absorbed through the skin and gastrointestinal tract (Forsyth and Quesnel 1957/Ex. 1-978). Additional data document a variety of dermal, respiratory, and systemic toxicities that are closely analogous to those of phenol in their metabolic actions (Harold, Nierenstein, and Roaf 1910/Ex. 1-1111; Dietering 1938/Ex. 1-1019; Cushny et al. 1940, as cited in ACGIH 1986/Ex. 1-3, p. 112).

Exposure to catechol causes an increase in blood pressure, and, at high doses, kidney damage, eczematous dermatitis, and systemic illness (Harold, Nierenstein, and Roaf 1910/Ex. 1-1111; Dietering 1938/Ex. 1-1019; Cushny et al. 1940, as cited in ACGIH 1986/Ex. 1-3, p.112). OSHA received no comments, except for those from NIOSH, on catechol.

In the final rule, OSHA is establishing a permissible exposure limit of 5 ppm as an 8-hour TWA for this substance with a skin notation. The Agency concludes that these limits will protect workers against the significant risks of dermal, upper respiratory tract, convulsions, and central nervous system effects (i.e., convulsions), all of which constitute material impairments of health that are potentially associated with exposure to catechol at levels above the new PEL.

1-CHLORO-1-NITROPROPANE

CAS: 600-25-9; Chemical Formula: $CH_3CH_2CHClNO_2$
H.S. No. 1081

OSHA's former time-weighted average limit for 1-chloro-1-nitropropane was 20 ppm. The ACGIH has a TLV-TWA of 2 ppm for this flammable liquid (ACGIH 1986/Ex. 1-3,). The proposed PEL was 2 ppm as an 8-hour TWA, and NIOSH (8-47, table N1) concurs with this limit. The final rule promulgates a 2-ppm 8-hour TWA PEL for 1-chloro-1-nitropropane.

1-Chloro-1-nitropropane is the most acutely toxic of the fungicides known as the chloronitropropanes. In an inhalation experiment, two rabbits were exposed for six hours to a concentration of 393 ppm, after which one rabbit died; at an average concentration of 2574 ppm, both rabbits died. Guinea pigs tested under the same conditions survived these exposures. The oral LD_{50} for rabbits determined in the same study was between 50 and 100 mg/kg (Machle, Scott, Treon et al. 1945/Ex. 1-349). Other members of this family of fungicides show lesser skin and lung irritation but do have higher ingestion toxicities (Patty 1963i, as cited in ACGIH 1986/Ex. 1-3, p.132). Exposure to high concentrations of 1-chloro-1-nitropropane can cause heart muscle, liver, and kidney damage (Patty 1963i, as cited in ACGIH 1986/Ex. 1-3, p. 132). OSHA received no comments on this substance, except for those from NIOSH. The ACGIH considers chloronitropropane to be more toxic than nitropropane, for which a TLV-TWA of 25 ppm has been established.

In the final rule, OSHA is establishing an 8-hour TWA PEL of 2 ppm. The Agency concludes that this limit will protect exposed employees from the significant risk of skin and upper respiratory tract irritation and of systemic toxicity, which constitute material health impairments that are potentially associated with 1-chloro-1-nitropropane exposure at the former PEL.

COBALT CARBONYL

CAS: 10210-68-1; Chemical Formula: $Co_2(CO)_4$
H.S. No. 1098

OSHA had no former limit for cobalt carbonyl. The ACGIH has a TLV-TWA of 0.1 mg/m³ (measured as cobalt) for this substance, which is a solid that decomposes at 50°C. The proposed PEL was 0.1 mg/m³ as an 8-hour TWA, and NIOSH (Ex. 8-47, Table N1) concurs with this limit, which is established by the final rule.

Sax (*Dangerous Properties of Industrial Materials,* 6th ed., 1984) reports that cobalt carbonyl has a moderate-to-high order of toxicity by the oral route. The oral LD_{50} in mice is 377.7 mg/kg; in rats, it is 753.8 mg/kg (Spiridonova and Shabalina 1973/Ex. 1-1098). The hazards of exposure to the metal carbonyls range from relatively low (for iron pentacarbonyl) to extremely serious (for nickel carbonyl) (Stokinger 1981e, in *Patty's Industrial Hygiene and Toxicology,* 3rd rev. ed., Vol 2A, pp. 1797-1806); the greater the toxicity of the metal and the more stable and volatile the carbonyl, the more hazardous the compound. Exposure to any of the metal carbonyls causes the same symptoms of nausea, dizziness, headache, substernal pain, coughing and dyspnea (Stokinger 1981e). Evidence concerning any chronic effects of long-term exposure is lacking (ACGIH 1986/Ex. 1-3, p. 145). Only NIOSH commented on this substance.

In the final rule, OSHA establishes an 8-hour TWA PEL of 0.1 mg/m³ TWA for cobalt carbonyl to protect against the significant risk of headache, nausea, and pulmonary effects, which are material impairments of health that are associated with occupational exposure to this substance at levels above the new PEL. The Agency concludes that this limit will substantially reduce these significant risks.

COBALT HYDROCARBONYL

CAS: 16842-03-8; Chemical Formula: $HCo(CO)_4$
H.S. No. 1099

OSHA had no former limit for cobalt hydrocarbonyl. The ACGIH has a TLV-TWA of 0.1 mg/m³ (measured as cobalt) for this flammable and toxic gas. The proposed PEL was an 8-hour TWA of 0.1 mg/m³; NIOSH (Ex. 8-47, Table N1) concurs with this limit. The final rule promulgates an 8-hour TWA PEL of 0.1 mg/m³ (measured as cobalt) for cobalt hydrocarbonyl.

Cobalt hydrocarbonyl is approximately half as toxic as nickel carbonyl in terms of acute effects; in animals, it produces clinical signs and symptoms very similar to those produced by nickel carbonyl (ACGIH TLV-TWA of 0.007 mg/m³) and iron pentacarbonyl (ACGIH TLV-TWA of 0.8 mg/m³) (ACGIH 1986/Ex. 1-3, p. 145). These include headache, dizziness, and, after a delay in onset, liver, brain, and lung damage. The 30-minute LC_{50} in rats is 165 mg/kg (Palmes, Nelson, Laskin, and Kuschner 1959/Ex. 1-430). There is no evidence of chronic toxicity or of carcinogenicity.

In the final rule, OSHA establishes an 8-hour TWA limit of 0.1 mg/m³ for cobalt hydrocarbonyl. The Agency concludes that this limit will protect exposed employees from the significant risk of pulmonary, brain, and liver damage, as well as that of acute effects such as headaches and dizziness, which constitute material health impairments that are associated with exposure to levels above the new PEL.

DIAZINON

CAS: 333-41-5; Chemical Formula: $C_{12}H_{21}N_2O_3PS$
H.S. No. 1118

Previously, OSHA had no limit for diazinon. The ACGIH has a TLW-TWA of 0.1 mg/m³, with a skin notation. The proposed PEL was an 8-hour TWA of 0.1 mg/m³, with a skin notation; NIOSH concurs that these limits are appropriate (Ex. 8-47, Table N1). The final rule establishes a 0.1-mg/m³ PEL, with a skin notation, for diazinon. Pure diazinon is a colorless liquid, but the technical grade is pale yellow to dark brown in color and has a faint odor.

Gaines (1960/Ex. 1-319) reports the acute oral LD_{50} for male and female rats to be 108 and 76 mg/kg, respectively. Other reports set the acute oral LD_{50}s in rats, guinea pigs, and rabbits at 76 to 150, 240 to 320, and 130 mg/kg, respectively (Association of American Pesticide Control Officials, Inc. 1969, as cited in ACGIH 1986/Ex. 1-3, p. 172). Studies from Hazleton Laboratories (1965, as cited in ACGIH 1986/Ex. 1-3, p. 172) and Radeleff (1958/Ex. 1-434) have shown much greater susceptibility to diazinon in birds and calves, with the oral LD_{50} being less than 10 mg/kg in some instances. The dermal LD_{50} in rabbits is 400 mg/kg (RTECS 1983-84). However, susceptibility to repeated doses is relatively consistent among species, with dogs showing signs of poisoning at 9.3 mg/kg per day and rats showing complete inhibition of red blood cell cholinesterase and marked inhibition of brain cholinesterase at 50 mg/kg/day (Bruce, Howard, and Elsea 1955/Ex. 1-585). Monkeys were poisoned at 5 mg/kg/day (Woodard, Woodard, and Cronin 1968/Ex. 1-458). Chronic feeding studies in rats have shown no chronic toxicity at 10, 100, and 1000 ppm. For many mammals, diazinon is less toxic than parathion (ACGIH TLV-TWA of 0.1 mg/m³, although this is not true under some circumstances (ACGIH 1986/Ex. 1-3, p. 172).

In humans, Hays (1963/Ex. 1-982) reports that two patients were poisoned by a dermal diazinon dosage of about 1.1 mg/kg; however, Gassman (1957/Ex. 1-901) reports no ill effects from an accidental ingestion of 30 mg/kg. One man received a dose of 250 mg/kg and recovered after treatment, which included gastric lavage (Bockel 1967, as cited in ACGIH 1986/Ex. 1-3, p. 172). In tests, Geigy (1966, as cited in ACGIH 1986/Ex. 1-3, p. 172), found that a series of doses of 0.05 mg/kg/day for 28 days produced plasma cholinesterase inhibition, and it has been suggested that the no-effect level for cholinesterase inhibition in humans is 0.02 mg/kg/day. Skin absorption of diazinon occurs readily, and overexposures are associated with weakness, headache, blurred vision, salivation, sweating, nausea, vomiting, diarrhea, abdominal cramps, slurred speech, and moist rales in the lungs (ACGIH 1986/Ex. 1-3, p. 172).

In the final rule, the Agency is establishing an 8-hour TWA PEL of 0.1 mg/m³, with a skin notation, for diazinon. OSHA concludes that these limits will protect exposed workers from the significant risk of cholinesterase inhibition, weakness, headache, nausea, vomiting, as well as the other symptoms and signs of diazinon poisoning, which together constitute material health impairments that are associated with exposures at levels above the new PEL.

1,1-DICHLORO-1-NITROETHANE

CAS: 594-72-9; Chemical Formula: $CH_3CCl_2NO_2$
H.S. No. 1121

OSHA formerly had a ceiling limit of 10 ppm for 1,1-dichloro-1-nitroethane. The ACGIH has a TLV-TWA of 2 ppm for this colorless liquid. The proposed PEL was an 8-hour TWA of 2 ppm, with which NIOSH (Ex. 8-47, Table N1) concurs. The final rule establishes the 2-ppm 8-hour TWA PEL for 1,1-dichloro-1-nitroethane.

Toxicity data on 1,1-dichloro-1-nitroethane are largely derived from the 1945 studies conducted by Machle and co-workers (Ex. 1-349). These scientists reported that both rabbits and guinea pigs died from inhaling vapors at 100 ppm for six hours; at a concentration of 60 ppm, the animals survived a two-hour exposure. Four-hour inhalation exposures at 34 ppm and six-hour daily exposures at 25 ppm for a total of 204 hours also did not kill rabbits or guinea

pigs. Skin and mucous membrane irritation were not produced at the 25-ppm exposure level. At survival concentrations, the primary targets of toxicity were the lungs, which showed edema, congestion, hemorrhage, and acute bronchitis. At lethal exposures, these investigators observed acute myocardial degeneration with interstitial edema, cloudy swelling of the liver with cellular degeneration, and tubular degeneration and interstitial edema of the kidney, as well as edema of the tufts of the glomeruli and kidney necrosis. The compound was also found to be a severe skin irritant when two applications were applied on two successive days (Machle, Scott, Treon et al. 1945/Ex. 1-349). The ACGIH (1986/Ex. 1-3, p. 188) states that dichloronitroethane is more toxic than the nonchlorinated nitroalkanes. The Workers Institute for Safety and Health (Ex. 116) questioned OSHA's selection of an 8-hour TWA rather than ceiling limit for this substance. In response, OSHA notes that the final rule's lower TWA limit is protective because the health effects of concern do not occur at the peak exposures that would be permitted by the revised 8-hour TWA PEL.

In the final rule, OSHA is establishing a PEL of 2 ppm TWA for 1,1-dichloro-1-nitroethane. The Agency concludes that this limit will protect workers against the significant risk of irritation, lung injury, and liver and kidney damage, all material health impairments that are associated with exposures at levels above the revised PEL.

p-DICHLOROBENZENE

CAS: 106-46-7; Chemical Formula: $C_6H_4Cl_2$
H.S. No. 1125

OSHA formerly had an 8-hour TWA limit of 75 ppm for p-dichlorobenzene. The ACGIH has a limit of 75 ppm TWA and a STEL of 110 ppm for this white crystalline material, which has a camphor-like odor. The ACGIH's limit recognizes that the para isomer is somewhat less toxic than the ortho isomer, for which the ACGIH has established a ceiling limit of 50 ppm. The proposed PEL retained the 75-ppm TWA limit and added a STEL of 110 ppm; the final rule establishes these limits.

In animal studies, an injection of 0.005 gram of p-dichlorobenzene in rats caused slight liver necrosis (Cameron, Thomas, Ashmore et al. 1937/Ex. 1-471). The intraperitoneal injection LD_{50} for rats has been reported as 2562 mg/kg (Zupko and Edwards 1949/Ex. 1-878). The oral LD_{50} in mice is 2950 mg/kg (Domenjoz 1946, as cited in ACGIH 1986/Ex. 1-3, p. 179); for rats, the oral LD_{50} is 2512 mg/kg (Varshavskaya 1970, as cited in ACGIH 1986/Ex. 1-3, p. 179). Rabbits fed a daily dietary exposure of 5 grams developed opacity of the lens in 3 weeks (Berliner 1939/Ex. 1-175); this finding was not confirmed, however, in repeated studies (Pike 1944/Ex. 1-656).

Reports of a human inhalation exposure to unspecified levels of p-dichlorobenzene describe swelling of the feet, ankles, and hands after day-long use of a mothproofing agent consisting of this substance (Clayton 1935/Ex. 1-306). Other reports describe cataracts caused by exposure to unspecified concentrations of the vapor of p-dichlorobenzene (Berliner 1939/Ex. 1-715). Petit and Champaix (1948, as cited in ACGIH 1986/Ex. 1-3, p. 179) report the case of a woman who experienced tingling of the hands, vertigo, and loss of weight from working for 18 months with a mixture of 90 parts p-dichlorobenzene and 10 parts hexachloroethane (airborne concentration not specified).

OSHA received three comments on p-dichlorobenzene: from NIOSH (Ex. 8-47), the Workers Institute of Safety and Health (WISH) (Ex. 116), and the Halogenated Solvents Industry Alliance (HSIA) (Ex. 186). WISH simply pointed out that the ACGIH *Documentation* (1986/Ex. 1-3) entry for this substance includes fewer, and different, references from those relied on by the Agency for Toxic Substances and Disease Registry (Ex. 116, Table 1), without further comment. The HSIA (Ex. 186, App. D) submitted a letter from EPA's Science Advisory Board to Lee Thomas, Administrator of EPA (3/9/88). The letter points out that there is a scientific hypothesis to the effect that, for many halogenated organics (including p-dichlorobenzene), the mechanism causing tumors in rats exposed to these substances may not be operative in humans (Ex. 186D). According to the HSIA, this hypothesis may have "important implications for human health risk assessment" (Ex. 186D, p. 2). On the other hand, NIOSH (Ex. 8-47, Table N6D) interprets the evidence for p-dichlorobenzene to mean that it is a potential human carcinogen that deserves full Section 6(b) rulemaking. OSHA will consider NIOSH's recommendation in light of the Agency's rulemaking priorities.

In the final rule, OSHA is retaining the 8-hour TWA PEL of 75 ppm TWA and adding a STEL of 110 ppm for p-dichlorobenzene. The Agency concludes that both a TWA and a STEL are necessary to protect workers from the significant risk of eye damage, vertigo, and neuropathic effects, which constitute material impairments of health that are associated with occupational exposure to p-dichlorobenzene at levels above the 8-hour TWA PEL.

DICHLOROMONOFLUOROMETHANE

CAS: 75-43-4; Chemical Formula: $CHCl_2F$
H.S. No. 1128

OSHA formerly had a limit of 1000 ppm TWA for dichloromonofluoromethane (FC-21). The ACGIH has a TLV-TWA of 10 ppm for this colorless gas; this limit is based on FC-21's similarity to chloroform in terms of hepatotoxic effects. The proposed PEL for FC-21 was 10 ppm, and NIOSH (Ex. 8-47, Table N1) concurs with this limit. The final rule promulgates an 8-hour TWA PEL of 10 ppm for FC-21.

FC-21 is considered more toxic than the related difluorinated methanes. The major health hazards associated with exposure to this substance are liver damage, cardiac sensitization, and narcosis. Freon-21 has a 4-hour LC_{50} of 49,900 ppm in rats (Tappan and Waritz 1964, as cited in ACGIH 1986/Ex. 1-3, p. 187). Within an hour, exposure to 100,000 ppm killed rats and guinea pigs (Weigard 1971/Ex. 1-1102); other tests with guinea pigs and mice demonstrated that concentrations of 50,000 ppm and higher cause unconsciousness or death (Nuckolls 1935, as cited in ACGIH 1986/Ex. 1-3, p. 187; Booth and Bixby 1932/Ex. 1-1079). The clinical signs of overexposure include loss of coordination, tremors, narcosis, and prostration, as well as possible lung and liver changes (Tappan and Waritz 1964, as cited in ACGIH 1986/Ex. 1-3, p. 187).

Two-week exposures of rats to 10,000 ppm for 6 hours daily caused hepatic failure or marked liver damage (Trochimowicz, Moore, and Chiu 1977/Ex. 1-34). A series of 90-day exposures of rats and dogs to concentrations of 1000 and 5000 ppm dichloromonofluoromethane resulted in bilateral hair loss, cirrhosis, and excessive mortality in rats in both exposure levels; dogs exhibited weight loss at both levels, but mild liver changes were observed only at the 5000-ppm level (Trochimowicz, Lyon, Kelly, and Chiu 1977, as cited in ACGIH 1986/Ex. 1-3, p. 187). Another uncompleted study reported liver pathology in rats repeatedly exposed for 90 days at 500 ppm, and probable liver pathology from similar exposures to 200 ppm; no hepatic effects were observed after exposure to 50 ppm (Allied Chemical Company 1978, as cited in ACGIH 1986/Ex. 1-3, p. 187).

Two of 12 dogs exposed to 10,000 ppm FC-21 plus intravenous epinephrine developed serious arrhythmia (Mullin,

as cited in ACGIH 1986/Ex. 1-3, p. 187). Dogs and monkeys (anesthetized) demonstrated tachycardia and hypotension after exposure to FC-21 at levels between 50,000 and 100,000 ppm; bronchoconstriction was observed at 25,000 ppm (Aviado and Smith 1975/Ex. 1-82; Belej and Aviado 1975/Ex. 1-462). Anesthetized mice exposed to a concentration of 100,000 ppm FC-21 showed arrhythmia and cardiac sensitization to epinephrine (Aviado and Belej 1974/Ex. 1-615). Preimplantation loss has been reported in pregnant rats exposed to FC-21 at 10,000 ppm on days 6 through 15 of gestation (Belej and Aviado 1975/Ex. 1-462). OSHA received no comments other than NIOSH's on FC-21.

In the final rule, OSHA is establishing a TWA limit of 10 ppm for dichloromonofluoromethane. The Agency concludes that this limit will protect workers against the significant risks of hepatotoxic effects, cardiac sensitization, and narcosis associated with expsoure to this substance. OSHA finds that these exposure-related effects constitute material impairments of health within the meaning of the Act.

DIETHYL KETONE

CAS: 96-22-0; Chemical Formula: $C_2H_5COC_2H_5$
H.S. No. 1135

Previously, OSHA had no limit for diethyl ketone. The ACGIH has a limit of 200 ppm TWA for this colorless liquid, which has an acetone-like odor. The proposed PEL was 200 ppm as an 8-hour TWA, and NIOSH (Ex. 8-47, Table N1) concurs with this limit, which is established in the final rule.

The oral LD_{50} for diethyl ketone in rats is reported to be 2.14 g/kg. Four of six rats died when exposed to diethyl ketone for four hours at 8000 ppm (Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1-440). In general, the toxicities of the methyl ketones increase with increasing molecular weight; diethyl ketone is somewhat less toxic than is methyl propyl ketone (TLV-TWA of 200 ppm) (NIOSH 1978f, as cited in ACGIH 1986/Ex. 1-3, p. 199). All of the ketones cause mucous membrane and eye and skin irritation. OSHA received no comments on diethyl ketone except those from NIOSH.

In the final rule, OSHA is establishing an 8-hour TWA PEL of 200 ppm for diethyl ketone, the same limit being proposed for methyl propyl ketone. The Agency concludes that this limit will reduce the significant risk of eye and skin irritation, which are material health impairments that are associated with exposure to diethyl ketone at levels above the new PEL.

DIETHYLENE TRIAMINE

CAS: 111-40-0; Chemical Formula: $(NH_2CH_2CH_2)_2NH$
H.S. No. 1138

Formerly, OSHA had no limit for diethylene triamine (DETA). The ACGIH has a TLV-TWA of 1 ppm, with a skin notation, for this strongly alkaline, hygroscopic, and somewhat viscous yellow liquid that smells like ammonia. The proposed PEL was 1 ppm as an 8-hour TWA, with a skin notation, and NIOSH (Ex. 8-47, Table N1) concurs with this limit. The final rule promulgates this 1-ppm 8-hour TWA for diethylene triamine; however, the skin notation is not retained (see the discussion on skin notations in Section VI.C.18 of this preamble).

The acute intraperitoneal LD_{50} values for DETA are reported to be 71 and 74 mg/kg for the mouse and rat, respectively (Hine, Kodama, Anderson et al. 1958/Ex. 1-511). In the rat, the reported oral and percutaneous LD_{50} values are the same (1080 mg/kg); the dermal LD_{50} for the rabbit is 1090 mg/kg (Smyth, Carpenter, and Weil 1949/Ex. 1-528). Exposure to 300 ppm of diethylene triamine vapor for 8 hours failed to kill any of a group of exposed rats (Savitt 1955/Ex. 1-663).

Sutton (1963/Ex. 1-1101) has reported that DETA causes severe corneal injury; solutions of 15 to 100 percent caused lasting corneal damage. If improperly controlled, the vapor and liquid cause sensitization of the respiratory tract and skin (American Industrial Hygiene Association 1960, as cited in ACGIH 1968/Ex. 1-3, p. 197). Dernehl (Ex. 1-728) demonstrated such sensitization in a study reported in 1951.

OSHA received no other comments on this substance. However, OSHA has carefully reviewed the health evidence on the percutaneous toxicity of DETA and has determined that a skin notation is not necessary for this substance (see the discussion on skin notations in Section VI.C.18). The final rule thus contains no skin notation for DETA.

In the final rule, OSHA is establishing an 8-hour TWA limit of 1 ppm for diethylene triamine. The Agency concludes that this limit will protect workers against the significant risk of skin and respiratory tract irritation and sensitization, all of which constitute material health impairments that are associated with exposure to diethylene triamine at levels above the new PEL.

DIPROPYL KETONE

CAS: 123-19-3; Chemical Formula: $(CH_3CH_2CH_2)_2CO$
H.S. No. 1148.

OSHA formerly had no limit for dipropyl ketone. The ACGIH has a TLV of 50 ppm TWA for this colorless liquid with a penetrating odor. The proposed PEL was 50 ppm as an 8-hour TWA, and NIOSH (Ex. 8-47, Table N1) concurs with this limit, which is established by the final rule.

Dipropyl ketone has a moderate oral and inhalation toxicity (*Dangerous Properties of Industrial Materials*, 6th ed., Sax 1984). In rats, the oral LD_{50} is 3.35 g/kg, and the dermal LD_{50} in rabbits is 9.5 g/kg. Tests have indicated that rats inhaling 2000 ppm for 4 hours survived, but at 4000 ppm all animals died (Carpenter, Weil, and Smyth 1974/Ex. 1-304). Methyl isobutyl ketone (MIBK) has a similar acute toxicity (ACGIH 1986/Ex. 1-3, p. 221); OSHA is establishing a 50-ppm 8-hour TWA and a 75-ppm STEL for MIBK. Only NIOSH submitted comments on dipropyl ketone.

In the final rule, OSHA is establishing an 8-hour TWA PEL of 50 ppm TWA for dipropyl ketone. The Agency concludes that this limit is necessary to protect workers from the significant risk of narcosis and irritation, both material health impairments that are associated with exposures at levels above the new PEL.

DIQUAT

CAS: 85-00-7; Chemical Formula: $C_{12}H_{12}Br_2N_2$
H.S. No. 1150

Previously, OSHA had no PEL for diquat. The ACGIH has a limit of 0.5 mg/m³ TWA for these yellow crystals. The proposed PEL was 0.5 mg/m³ as an 8-hour TWA, and NIOSH (Ex. 8-47, Table N1) concurs with this limit. The final rule establishes 0.5 mg/m³ as the 8-hour TWA PEL for diquat.

In most species, the acute oral toxicity of diquat is similar to that of paraquat and ranges from 100 to 400 mg/kg in rats, mice, rabbits, and dogs. Cows experience more severe toxic effects, with an acute oral LD_{50} or 30 mg/kg. The 24-hour percutaneous LD_{50} in rabbits is greater than 400 mg cation/kg; no skin irritation or other ill effects were demonstrated at this level (Clark and Hurst 1970/Ex. 1-135; Rowe and Wright 1965, as cited in ACGIH 1986/Ex. 1-3, p. 222). Rats fed 1000 ppm daily (about 50 mg/kg/day) for two years survived; reduced food intake and growth were the only consequences observed. At 500 ppm (about 25 mg/kg/day), the only ill effect observed was a pathologic change in the eye. A dietary level of 10 ppm (about 0.5 mg/kg/day) for two years did not induce cataract formation, but cataracts do occur at higher levels, with pathology observed at the 500-ppm level; one in four animals demonstrated complete corneal opacity in one or both lenses after six months at the 1000-ppm

level. Cataract formation requires prolonged exposure and is not induced by single high-level exposures (ACGIH 1986/Ex. 1–3, p. 222).

Unlike paraquat, diquat does not produce lung damage in exposed humans or animals. Acute poisoning may produce nonspecific respiratory distress as well as other nonspecific signs of poisoning. In humans, accidental ingestion has produced less toxic reactions than those associated with paraquat ingestion (Orepoulos and McEvoy 1969/Ex. 1–429). OSHA received no comments, other than NIOSH's, on diquat.

In the final rule, OSHA is establishing an 8-hour PEL of 0.5 mg/m^3 TWA for diquat. The Agency concludes that this limit will protect against the significant risk of ocular damage, which constitutes a material health impairment that is associated with chronic exposure at levels above the new PEL.

DISULFOTON

CAS: 298–04–4; Chemical Formula: $C_8H_{19}O_2PS_3$
H.S. No. 1152

OSHA formerly had no exposure limit for disulfoton. The ACGIH has a limit of 0.1 mg/m^3 TWA for this substance. The proposed PEL for disulfoton was 0.1 mg/m^3 as an 8-hour TWA; the final rule establishes this limit and adds a skin notation. Pure disulfoton is an oily, colorless liquid; the technical grade is a brown liquid.

The acute toxicity of disulfoton is very high by all laboratory-tested routes of administration. For weanling rats, the intraperitoneal LD_{50} is reported to be 5.4 mg/kg; for adult rats, it is 9.4 mg/kg (Brodeur and Dubois 1963/Ex. 1–718). The acute dermal LD_{50} is 6 mg/kg for adult female rats and 25 mg/kg for adult male rats (Gaines 1969/Ex. 1–320). The acute oral LD_{50}s for male and female rats are reported as 6.8 mg/kg and 2.3 mg/kg, respectively (Brodeur and Dubois 1964/Ex. 1–1015). Rats have demonstrated an acquired tolerance for disulfoton (Brodeur and Dubois 1964/Ex. 1–1015).

Metabolically, disulfoton is highly fat-soluble, and the compound apparently interferes with mixed-function oxidase activity in the same manner shown to be the case for parathion; with respect to median lethal doses, parathion and disulfoton are similar (Stevens et al. 1973, as cited in ACGIH 1986/Ex. 1–3, p. 226).

NIOSH (Ex. 8–47, Table N2) noted that OSHA had inadvertently omitted the skin notation for the proposed limit for disulfoton. NIOSH points out that the studies described above for this substance clearly demonstrate that disulfoton "is almost as toxic via the skin as when administered internally," and further, that the 1986 ACGIH *Documentation* (Ex. 1–3, p. 226) includes a skin notation for this substance. On the basis of these comments, OSHA is including a skin notation for disulfoton in the final rule. With the exception of NIOSH, no commenter submitted evidence to the record on disulfoton.

In the final rule, OSHA is establishing an 8-hour TWA PEL for disulfoton of 0.1 mg/m^3, with a skin notation. The Agency concludes that this limit will prevent the significant risk of acute toxicity and metabolic injury, which are material impairments of health that are associated with exposures at levels above the new PEL. The skin notation is included to protect workers against the dermal toxicity that has been demonstrated in animal tests.

DIVINYL BENZENE

CAS: 108–57–6; Chemical Formula: $C_6H_4(CHCH_2)_2$
H.S. No. 1154

Previously, OSHA had no limit for divinyl benzene. The ACGIH has a TLV–TWA of 10 ppm, based on this substance's similarity to styrene. The proposed PEL was 10 ppm as an 8-hour TWA, and NIOSH (Ex. 8–47, Table N1) concurs with this limit. In the final rule, an 8-hour TWA of 10 ppm is promulgated for divinyl benzene. The commercial grade of divinyl benzene is a pale-straw-colored liquid; it contains all three isomers, but the meta isomer predominates.

The oral LD_{50} for rats is reported to be 4.1 g/kg, and an acute inhalation study showed no ill effects from a single seven-hour exposure at 351 ppm. However, repeated or prolonged contact with the liquid may cause skin burns (Dow Chemical Company 1977j, as cited in ACGIH 1986/Ex. 1–3, p. 228).

Industrial experience indicates that irritation of the respiratory system, skin, and eyes can result from inhalation exposures to divinyl benzene, but there are no data concerning chronic exposures in humans. No comments, other than those of NIOSH, were received on divinyl benzene.

The final rule establishes a PEL of 10 ppm (8-hour TWA) for divinyl benzene. The Agency concludes that this limit will protect against the significant risk of irritation to the respiratory tract, eyes, and skin; such irritation constitutes a material impairment of health within the meaning of the Act.

ENDOSULFAN

CAS: 115–29–7; Chemical Formula: $C_9H_6Cl_6O_3S$
H.S. No. 1156

OSHA formerly had no permissible exposure limit for endosulfan. The ACGIH has a TLV–TWA of 0.1 mg/m^3, with a skin notation. The proposed PEL was 0.1 mg/m^3, as an 8-hour TWA, with a skin notation; NIOSH (Ex. 8–47, Table N1) concurs. The final rule establishes an 8-hour TWA PEL for endosulfan of 0.1 mg/m^3, with a skin notation. Technical endosulfan is a tan, semi-waxy solid mixture; it may have a slight odor similar to that of sulfur dioxide.

The insecticide, endosulfan, is similar in its acute oral toxicity to the related insecticides aldrin and dieldrin (TLV–TWAs of 0.25 mg/m^3), except that it is slightly more toxic than these substances in female laboratory animals. In rats, the oral LD_{50} for endosulfan is 43 mg/kg for males and 18 mg/kg for females (*Farm Chemicals Handbook* 1974/Ex. 1–1147a). The dermal LD_{50} in male and female rats are 130 mg/kg and 74 mg/kg, respectively (*Farm Chemicals Handbook* 1974/Ex. 1147a). The respiratory LC_{50} for male rats is 50 mg/kg for 4 hours of exposure (Association of American Pesticide Control Officials, Inc. 1969, as cited in ACGIH 1986/Ex. 1–3, p. 230).

In laboratory tests of chronic exposure, rats tolerated oral doses of up to 3.2 mg/kg/day for 3 months without injury (Gaines 1975, as cited in ACGIH 1986/Ex. 1–3, p. 230), and dogs tolerated doses up to 0.75 mg/kg for 1 year (Ely, MacFarlane, Galen, and Hines 1967/Ex. 1–414). A 2-year dietary level of 10 ppm (approximately 0.5 mg/kg/day) in rats was associated with a statistically insignificant decline in female survival rates and caused a reduction in testis weights in males. At 5.0 mg/kg/day, histopathologic findings showed renal tubular damage and some hydropic changes in rat livers (Czech 1958, as cited in ACGIH 1986/Ex. 1–3, p. 230).

Inhalation of endosulfan dust by humans has been associated with slight nausea, confusion, excitement, flushing, and dry mouth (State of California: Department of Industrial Relations/Ex. 1–8). Nine employees who had been working with 50-percent water-wettable endosulfan powder for only a few days had convulsions (Association of American Pesticide Control Officials, Inc. 1969, as cited in ACGIH 1986/Ex. 1–3, p. 230). With the exception of NIOSH's comments, no evidence on endosulfan was submitted to the record.

OSHA concludes that exposure to endosulfan poses a significant risk of systemic poisoning and renal and testicular damage, and the Agency therefore is establishing a PEL of 0.1 mg/m^3 TWA for endosulfan, with a skin notation; these effects constitute a

material impairment of health within the meaning of the Act. OSHA finds that this limit will substantially reduce the significant risk associated with exposure to this substance at the levels formerly permitted by the absence of an OSHA limit.

FONOFOS

CAS: 944-22-9; Chemical Formula: $C_{10}H_{15}OPS_2$
H.S. No. 1181

OSHA formerly had no limit for fonofos. The ACGIH has a limit of 0.1 mg/m³ TWA, with a skin notation, for this light-yellow liquid, which is similar to ethyl parathion and other cholinestase inhibitors. The proposed PEL was an 8-hour TWA of 0.1 mg/m³, with a skin notation; NIOSH (Ex. 8-47, Table N1) concurs with this limit. The final rule's PEL for fonofos is an 8-hour TWA limit of 0.1 mg/m³, with a skin notation.

In male rats, the average acute oral LD_{50} of technical fonofos has been reported to be 13.2 mg/kg (Stauffer Chemical Co. 1974, as cited in ACGIH 1986/Ex. 1-3, p. 275). For female rats, an average oral LD_{50} of 3 mg/kg has been reported (NIOSH 1974d). The acute dermal LD_{50}s reported for rats and guinea pigs are 147 and 278 mg/kg, respectively (Weir and Hazleton 1981/Ex. 1-1135). Weir and Hazleton reported that no localized eye irritation occurred when 0.1 ml of technical fonofos was instilled into rabbit eyes; however, death resulted in these animals within 24 hours after the instillation (1981/Ex. 1-1135). Dietary studies of rats lasting 105 weeks have shown 10 ppm (about 0.2 mg/kg) to be a no-effect level. Dogs fed fonofos for 14 weeks showed no-effect dietary levels of 8 ppm; no carcinogenic effects were observed. Rats showed reproductive effects at dietary levels of 10 ppm and 31.6 ppm (about 0.7 mg/kg) (Stauffer Chemical Co. 1974, as cited in ACGIH 1986/Ex. 1-3, p. 275).

There are no reports of human poisonings caused by fonofos, although it is known to be a cholinesterase inhibitor (ACGIH 1986/Ex. 1-3, p. 275). There were no comments, other than NIOSH's, on fonofos.

In the final rule, OSHA is establishing an 8-hour PEL of 0.1 mg/m³ TWA for fonofos to protect exposed workers from the significant risk of cholinesterase inhibition that is characteristic of exposure to this and other organic phosphate pesticides. OSHA considers cholinesterase inhibition a material impairment of health. A skin notation is also established, based on evidence in animals that fonofos can readily penetrate the skin and cause death. The Agency concludes that these limits will substantially reduce this significant risk.

FORMAMIDE

CAS: 75-12-7; Chemical Formula: CH_3NO
H.S. No. 1182

Previously, OSHA had no limit for formamide. The ACGIH has a TLV-TWA of 20 ppm and a TLV-STEL of 30 ppm for this clear, viscous, odorless liquid. The proposed PELs were an 8-hour TWA of 20 ppm and a 15-minute STEL of 30 ppm, and the final rule establishes these limits.

Formamide has an LD_{50} of approximately 6 g/kg for rats (Thiersh 1962/Ex. 1-690; Zaeva, Vinogradova, Savina, and Osipenko 1969/Ex. 1-1026). Dietary administration at 1.5 g/kg for two weeks resulted in fatalities in rats; pathologic examination revealed cumulative changes characteristic of gastritis and malnutrition (E.I. du Pont de Nemours and Company, Inc., as cited in ACGIH 1986/Ex. 1-3, p. 278). Czajkowska (1981, as cited in ACGIH 1986/Ex. 1-3, p. 278) reports the dermal LD_{50} for skin absorption in rabbits as 6 g/kg. Mild and transient irritation, but no allergic skin sensitization, occurred when formamide was applied to the skin of guinea pigs (*Dangerous Properties of Industrial Materials*, 6th ed., Sax 1984; E.I. du Pont de Nemours and Company, Inc., as cited in ACIGH 1986/Ex. 1-3, p. 278). Eye irritation tests in rabbits showed only slight, temporary irritation (Carpenter and Smyth 1946/Ex. 1-303). No signs of toxicity in rats were detected in single six-hour exposures at 3900 ppm formamide dispensed as a mist, or in six-hour daily exposures for 10 days at approximately 1500 ppm formamide vapor (equivalent to air saturated with formamide at room temperature); no indications of organ damage were seen in these animals on pathologic examination (E.I. du Pont de Nemours and Company, Inc., as cited in ACGIH 1986/Ex. 1-3, p. 278).

Gross fetal malformations were not noted following dermal applications of formamide to the skin of pregnant rats; the effects that were observed were weak and were produced at overwhelming concentrations (Stula and Krauss 1977/Ex. 1-1068). The no-observed-effect level in a rabbit developmental toxicity study was 22 mg/kg orally (Merkle and Zeller 1980/Ex. 1-683).

According to the ACGIH, there are no reports of industrial poisoning by formamide (E.I. du Pont de Nemours and Company, Inc., as cited in ACGIH 1986/Ex. 1-3, p. 278).

OSHA received comments on formamide from Grace Ziem (Ex. 46) and NIOSH (Ex. 8-47, Table N2). Dr. Ziem, an occupational physician on the staff of Johns Hopkins School of Hygiene and Public Health and the University of Maryland School of Medicine, believes that OSHA should revise the PEL for formamide to 10 ppm as an 8-hour TWA based on Grant's (1986/Ex. 1-975) statement that this substance causes Grade 4 eye irritation rather than the "mild" irritation reported by du Pont (as cited in ACGIH 1986/Ex. 1-3, p. 278). Dr. Ziem also notes that the ACGIH has dropped its STEL for formamide, lowered its 8-hour TWA PEL to 10 ppm, and added a skin notation for this substance. In addition, consistent with the Agency's policy on skin notations (discussed in Section VI.C.18 of the preamble), OSHA is not adopting the skin notation at the present time. The Agency concludes that the 30-ppm STEL should be retained to ensure that workplace exposures to formamide are not permitted to exceed the 8-hour TWA by any substantial margin. NIOSH (Ex. 8-47, Table N2) does not concur with the limits proposed and points out that formamide is a testicular toxin and has been identified in mice as a teratogen. OSHA is aware of the developing literature on both formamide and dimethyl formamide, and the Agency intends to monitor toxicological developments on these chemicals closely in the future to determine whether other action is necessary.

In the final rule, OSHA is establishing a PEL of 20 ppm TWA and a STEL of 30 ppm for formamide. The Agency concludes that this limit will not only protect workers against the significant risks of eye and skin irritation, but will substantially reduce the risks of other health effects that exist as a consequence of workplace exposure to formamide at levels above the new PELs. OSHA considers sensory irritation, testicular toxicity, and teratogenicity material impairments of health within the meaning of the Act.

GERMANIUM TETRAHYDRIDE

CAS: 7782-65-2; Chemical Formula: GeH_4
H.S. No. 1186

OSHA formerly had no limit for germanium tetrahydride. The ACGIH has a TLV of 0.2 ppm TWA for this colorless gas. The proposed PEL was an 8-hour TWA of 0.2 ppm, with which NIOSH (Ex. 8-47, Table N1) concurs. In the final rule, the 0.2-ppm 8-hour TWA is established as OSHA's PEL for germanium tetrahydride.

An early study indicated that germanium tetrahydride has a toxicity between that of tin hydride and arsine (Flury and Zernik 1931e/Ex. 1-993). In this study, a rabbit survived exposure to

100 ppm for one hour. One-hour exposures at 150 and 185 ppm caused fatalities in mice, and similar exposures involving guinea pigs resulted in sickness at the 150-ppm level and death at 185 ppm (Flury and Zernik 1931e/Ex. 1–993). On the other hand, Webster (1946/Ex. 1–399) reported that germanium tetrahydride is less toxic than both tin hydride and arsine. The effect of exposure to germanium tetrahydride is hemolysis. Data concerning chronic or subacute toxicities are not available. Based on germanium's acute toxicity, which is approximately half that of stibine, the ACGIH recommends an 8-hour TLV of 0.2 ppm TWA. OSHA received no comments, other than NIOSH's, on this substance.

In the final rule, OSHA establishes a PEL of 0.2 ppm as an 8-hour TWA for germanium tetrahydride to reduce the significant risk of hemolytic effects, which constitute material impairments of health that are associated with exposure to this substance at levels above the new PEL. The Agency concludes that implementation of this limit will substantially reduce this significant risk.

INDENE

CAS: 95–13–6; Chemical Formula: C_9H_8
H.S. No. 1212

OSHA had no former limit for indene. The ACGIH has a TLV–TWA of 10 ppm for this colorless liquid. The proposed PEL was 10 ppm as an 8-hour TWA, a limit with which NIOSH (Ex. 8–47, Table N1) concurs. The final rule promulgates an 8-hour TWA PEL for indene of 10 ppm.

Early inhalation studies of indene reported injury to the spleen, liver, and kidney of rats exposed to indene vapor concentrations of 800 to 900 ppm for six 7-hour periods (Cameron and Doniger 1939/Ex. 1–470). Some animals were found at necropsy to have severe necrosis of the liver with hemorrhage; kidney necrosis was also observed. No other organ damage was found and no deaths occurred as a result of these exposures (Cameron and Doniger 1939/Ex. 1–470). By analogy with the effects of exposure to other monoaromatic hydrocarbons, exposure to indene is likely to irritate the mucous membranes. In laboratory animals, chemical pneumonitis, pulmonary edema, and hemorrhage have resulted from the aspiration of indene liquid into the lung, and repeated skin contact has caused dermatitis as a result of the defatting properties of indene (Gerarde 1960b/Ex. 1–738b). In dermal studies of rats, one to eight applications of 0.1 ml to the shaved skin were reported to have no effect; three applications of 0.5 ml to guinea pig skin also produced no effect (Cameron and Doniger 1939/Ex. 1–470). The oral toxicity of indene appears to be moderate, with adult rabbits tolerating a single dose of 1 gram without signs of systemic toxicity (Gerarde 1960b/Ex. 1–738b). Subcutaneous injection of 1 gram, however, caused liver pathology and fatalities; high oral doses (2.5 ml of a 1.1 v/v mixture in olive oil) were uniformly fatal, with characteristic liver, lung, and gastrointestinal changes. Chronic administration of 3 mg/m^3 indene for 105 days caused catalase inhibition and stimulation of blood cholinesterase in rats, but no effects were observed in rats exposed at 0.6 mg/m^3 (Dyshinevich 1976/Ex. 1–631). No comments (other than those from NIOSH) were received on this substance.

The final rule establishes an 8-hour PEL of 10 ppm TWA for indene. OSHA concludes that this level will reduce the significant risks of irritation, pulmonary effects, and systemic toxicity which may constitute material impairments of health that are associated with exposure to levels above the new PEL.

IODOFORM

CAS: 75–47–8; Chemical Formula: CHI_3
H.S. No. 1214

OSHA had no former limit for iodoform. The ACGIH has an 8-hour TWA limit of 0.6 ppm for this yellow-green powder or crystalline solid with a pungent odor. The proposed PEL was 0.6 ppm as an 8-hour TWA; NIOSH (Ex. 8–47, Table N1) concurred with this limit, which is established by the final rule.

The subcutaneous LD_{50} for rabbits is 50 mg/kg, and the oral LD_{Lo} for iodoform in dogs is 1000 mg/kg (Kutob and Plaa 1962/Ex. 1–61). These authors also report that, on a molar basis, iodoform has an acute toxicity in mice similar to that of methyl iodide; this conclusion is based on parameters of lethality, barbiturate sleeping time, and bromsulphalein (BSP) retention time. An NCI bioassay (1978c/Ex. 1–1117) of iodoform indicates that the substance is not carcinogenic nor of high systemic toxicity, although histopathological examination of laboratory animals in this bioassay was judged by NCI to be inadequate.

No human data are available for this compound, and OSHA received no comments on this substance, other than those from NIOSH.

In the final rule, OSHA is establishing an 8-hour TWA limit of 0.6 ppm for iodoform, based on the limit being established for methyl iodide (2 ppm TWA); these limits are comparable on a molar iodine basis. OSHA concludes that this limit will protect workers from the significant risks of irritation and hepatotoxicity, both material impairments of health that are associated with exposure to iodoform. The Agency has determined that this limit will substantially reduce these significant risks.

ISOBUTYL ALCOHOL

CAS: 78–83–1; Chemical Formula: $(CH_3)_2CHCH_2OH$
H.S. No. 1219

OSHA formerly had a limit of 100 ppm as an 8-hour TWA for isobutyl alcohol. The ACGIH has a limit of 50 ppm TWA for this flammable, refractive, colorless liquid. The proposed PEL was 50 ppm as an 8-hour TWA; NIOSH (ex. 8–47, Table N1) concurs. The final rule establishes a 50 ppm 8-hour TWA PEL for isobutyl alcohol.

Limited inhalation studies have reported a somewhat higher acute toxicity for isobutyl alcohol than for n-butyl alcohol (which has a ceiling of 50 ppm) (Smyth, Carpenter, and Weil 1951/Ex. 1–439; Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1–440). A 4-hour LC_{50} of 8000 ppm has been reported in rats for isobutyl alcohol. Ingestion studies in rabbits have reported an acute oral toxicity of 3.75 g/kg for isobutyl alcohol (Smyth, Carpenter, and Weil 1951/Ex. 1–439; Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1–440). The dermal LO_{50} is 4.2 g/kg (Stokinger 1976, as cited in ACGIH 1986/Ex. 1–3, p. 331). Weese (1928/Ex. 1–1073) reported that the narcotic inhalation dose over a total of 136 hours is 6400 ppm in mice. Slight changes in the liver and kidneys were reported, but no fatalities occurred after repeated narcotizing doses (Weese 1928/Ex. 1–1073).

The effects of liquid isobutyl alcohol on the human eye appear to be comparable to those of n-butanol; no data are available on ocular exposure to the isobutyl alcohol vapor. Dermal application of isobutyl alcohol has caused slight erythema and hyperemia in humans (Schwartz and Tulipan 1939/Ex. 1–1167; Oettel 1936/Ex. 1–921).

OSHA received one comment on this substance in addition to NIOSH's; the Motor Vehicle Manufacturers Association (MVMA) (Ex. 3–902) lists isobutyl alcohol as a substance for which, in the opinion of the MVMA, rulemaking should be delayed. The MVMA provided no substantive information in support of its position.

In the final rule, OSHA is reducing the former 8-hour TWA PEL of 100 ppm to 50 ppm for isobutyl alcohol. The Agency concludes that a 50-ppm limit will reduce the significant risk of skin irritation, which is a material

impairment of health that is associated with exposure to concentrations at levels above the revised PEL.

ISOOCTYL ALCOHOL

CAS: 26952-21-6; Chemical Formula: ge $CH_3(CH_2)_3CH(C_2H_5)CH_2OH$
H.S. No. 1220

Previoulsy, OSHA had no PEL for isooctyl alcohol. The ACGIH has a TLV-TWA of 50 ppm, with a skin notation, for this colorless liquid mixture. The proposed PEL was 50 ppm, with a skin notation, and these limits are established in the final rule NIOSH (Ex. 8-47, Table N1) concurs with these limits.

The single-dose oral LD_{50}s for isooctyl alcohol reported for rats and mice are between 3.2 and 6.4 g/kg; intraperitoneal injection LD_{50}s for these species range from less than 0.4 g/kg to 1.6 g/kg (Hodge 1943/Ex. 1-700; Fassett 1951, as cited in ACGIH 1986/Ex. 1-3, p. 332). The dermal LD_{50} for the guinea pig is greater than 10 ml/kg (Fassett 1951, as cited in ACGHI 1986/Ex. 1-3, p. 332); in the rabbit, the dermal LD_{50} is 2.38 ml/kg (Smyth, Carpenter, Weil et al. 1969/Ex. 1-442). Moderate skin irritation for exposure to isooctyl alcohol has also been reported. Rats and rabbits have shown skin irritation at exposure levels ranging from 1.7 to 3.34 ml/kg (Smyth, Carpenter, Weil, et al. 1969/Ex. 1-442). Fassett (1951, as cited in ACGIH 1986/Ex. 1-3 p. 332) also reported no fatalities in rats after an 8-hour inhalation test at 235 ppm. OSHA received no comments, other than NIOSH's, on this substance.

In the final rule, OSHA is establishing an 8-hour TWA PEL of 50 ppm, with a skin notation, for isooctyl alcohol. The Agency concludes that these limits will reduce the significant risks of skin irritation, a material impairment of health that is associated with exposure to this substance at levels above the new PEL.

N-ISOPROPYLANILINE

CAS: 768-52-5; Chemical Formula: $C_6H_5NHCH(CH_3)_2$
H.S. No. 1229

OSHA formerly had no limit for N-isopropylaniline. The ACGIH recommends a TLV-TWA of 2 ppm, with a skin notation, for this liquid. The proposed PEL was 2 ppm, with a skin notation; NIOSH (Ex. 8-47, Table N1) concurs. The final rule establishes an 8-hour TWA PEL of 2 ppm, and a skin notation, for N-isopropylaniline.

The oral LD_{50} for rats exposed to N-isopropylaniline is between 0.25 and 0.5 g/kg. Slight irritation of the skin and eyes has been reported in animals as a result of direct contact with this chemical (Dow Chemical Company 1977k, as cited in ACGIH 1986/Ex. 1-3, p. 338). No other data concerning chronic toxicity or human exposure are available (ACGIH 1986/Ex. 1-3, p. 338).

Chemical analysis shows N-isopropylaniline to have toxicologic properties similar to those of its parent compound, aniline. The oral LD_{50}s for the two chemicals are approximately equal. The ACGIH has established the 2-ppm TLV-TWA for N-isopropylaniline on the basis of its structural analogy with aniline (which has a 2-ppm TLV-TWA) and N,N-dimethylaniline (which has a 5-ppm TLV-TWA and a 10-ppm STEL); exposure to these substances has been shown to cause hemolytic and central nervous system effects in animals and humans. These substances are also toxic when absorbed through the skin. OSHA received only one comment, form NIOSH, on this substance.

In the final rule, OSHA is establishing an 8-hour PEL of 2 ppm for N-isopropylaniline, with a skin notation. The Agency concludes that this limit will protect exposed workers from the significant risk of irritation and systemic and hemolytic effects, all material health impairments that are caused by inhalation, ingestion, or dermal absorption of N-isopropyl-aniline.

KETENE

CAS: 463-51-4; Chemical Formula: $CH_2=C=O$
H.S. No. 1231

OSHA's former 8-hour TWA limit for ketene was 0.5 ppm. The ACGIH has a TLV-TWA of 0.5 ppm and a TLV-STEL of 1.5 ppm for this colorless gas with a sharp, penetrating odor. The proposal retained the 8-hour TWA and added a STEL of 1.5 ppm; NIOSH (Ex. 8-47, Table N-1) concurs. The final rule retains an 8-hour TWA PEL of 0.5 ppm and adds a STEL of 1.5 ppm for ketene.

Ketene is highly irritating to the respiratory tract (Mendenhall and Stokinger 1959/Ex. 1-428), and the effects of its action are delayed (Treon, Sigmon, Kitzmiller 194/Ex. 1-769). Mendenhall and Stokinger (1959/Ex. 1-428) have reported a 10-minute LC_{50} for mice of 17 ppm. Cronic exposure at 1 ppm for six months on a schedule of six hours daily, five days per week, was tolerated by animals of several species (Mendenhall and Stokinger 1960, as cited in ACGIH 1986/Ex. 1-3, p. 341). Similar results have been reported in monkeys exposed repeatedly (55 exposures) for seven hours (Treon, Sigmon, and Kitzmiller 1949/Ex. 1-769). Evidence strongly suggests that the development of emphysema and fibrosis may occur in individuals who have developed a tolerance to the acute effects of ketene exposure (Stokinger, Wagner, and Dobrogarski 1957/Ex. 1-139. No comments other than NIOSH's were received on ketene.

In the final rule, OSHA is retaining the 8-hour TWA PEL of 0.5 ppm and adding a 15-minute STEL of 1.5 ppm for ketene. The Agency concludes that workers exposed to this highly irritating and toxic gas are at significant risk of developing respiratory irritation, pulmonary edema, and other severe plumonary effects that constitute material health impairments. OSHA finds that a TWA and STEL are required to protect against both acute and chronic health effects. The final rule's limits will substantially reduce these risks.

METHACRYLIC ACID

CAS: 79-41-4; Chemical Formula: $CH_2=C(CH_3)COOH$
H.S. No. 1244

OSHA formerly had no limit for methacrylic acid. The ACGIH has a TLV-TWA of 20 ppm for this substance. Methacrylic acid is a liquid with an acrid, disagreeable odor. The proposed PEL was 20 ppm, and NIOSH (Ex. 8-47, Table N1) concurred that this limit is appropriate. The final rule establishes an 8-hour TWA PEL of 20 ppm for methacrylic acid, with a skin notation.

The primary toxic hazard associated with exposure to methacrylic acid is irritation, although the degree of irritation from exposure to this substance is significantly less than that from acrylic acid (ACGIH 1986/Ex. 1-3, p. 362).

Direct contact of methacrylic acid with the skin or eye can cause corrosion of the skin or blindness. In rabbits, the skin absorption LD_{50} for methacrylic acid is 0.5 to 1 g/kg (Dow Chemical Company 1977m, as cited in ACGIH 1986/Ex. 1-3, p. 362). Rats exposed by inhalation to approximately 1000 ppm methacrylic acid exhibited eye irritation (Dow Chemical Company 1977m, as cited in ACGIH 1986/Ex. 1-3, p. 362). Rats exposed to 300 ppm for six hours daily for 20 days showed slight congestion of the kidneys (Gage 1970/Ex. 1-318).

Medical reports of acute exposures (at concentrations of up to 113 ppm) in an industrial setting revealed no respiratory symptoms; however, skin responses and a severe corneal burn were reported (Dow Chemical Company 1977m, as cited in ACGIH 1986/Ex. 1-3, p. 362). Only NIOSH commented on this substance.

In the final rule, OSHA is establishing a PEL of 20 ppm as an 8-hour TWA for this substance, with a skin notation. The

Agency concludes that this limit will protect workers from the significant risk of severe eye and skin irritation, which are material health impairments that are associated with exposure to methacrylic acid at levels above the new limit. The skin notation is necessary to prevent dermal absorption and systemic toxicity.

4-METHOXYPHENOL

CAS: 150-76-5; Chemical Formula: $CH_3OC_6H_4OH$
H.S. No. 1247

Previously, OSHA had no limit for 4-methoxyphenol. The ACGIH has a TLV-TWA of 5 mg/m³ for this solid substance. The proposed PEL was 5 mg/m³; NIOSH (Ex. 8-47, Table N1) concurs with this limit. In the final rule, OSHA establishes an 8-hour TWA PEL of 5 mg/m³ for methoxyphenol.

In rats the oral LD_{50} for 4-methoxyphenol is between 1 and 2 g/kg; the skin absorption LD_{50} is reported as greater than 1 g/kg in rabbits. Results of a two-month dietary study demonstrated no ill effects at 0.1 ppm (approximately 50 mg/kg/day). Direct contact of 4-methoxyphenol with the skin or eyes causes burns or moderate corneal damage (Hodge, Sterner, Maynard, and Thomas 1949/Ex. 1-41; Dow Chemical Company 1977n, as cited in ACGIH 1986/Ex. 1-3, p. 367). Only NIOSH commented on this substance.

To reduce the risk of dermal and ocular effects resulting from exposure to 4-methoxyphenol, a compound similar in chemical structure and toxicity to hydroquinone, OSHA is establishing a permissible exposure limit of 5 mg/m³ as an 8-hour TWA. The Agency concludes that this limit will protect workers against the significant risk of dermal and skin effects potentially associated with exposures to this substance at levels above the new PEL.

METHYL ACETYLENE-PROPADIENE MIXTURE (MAPP)

CAS: None; Chemical Formula: C_3H_4 isomers
H.S. No. 1250

OSHA formerly had a standard of 1000 ppm TWA for MAPP. The ACGIH also has an 8-hour TWA limit of 1000 ppm, with a TLV-STEL of 1250 ppm. OSHA proposed to retain the 8-hour PEL of 1000 ppm and to add a STEL of 1250 ppm, and the final rule establishes these limits, with which NIOSH (Ex. 8-47, Table N1) concurs. MAPP contains 58 percent of a mixture of propadiene (a colorless, unstable gas with a strong, unpleasant odor) and methyl acetylene (a colorless gas with a sweet odor); the balance of the mixture consists of paraffinic and olefinic C_3 and C_4 hydrocarbons.

Tests of rabbits, dogs, and guinea pigs exposed to an average concentration of 5000 ppm for seven hours/day, five days/week for four months resulted in no adverse health effects except decreased lung weights. No changes at all were observed in animals exposed to 1000 ppm for four months (Dow Chemical Company 1964, as cited in ACGIH 1986/Ex. 1-3, p. 368).

On the basis of these data, which show MAPP to be a chemical mixture of low toxicity in experimental animals, the Agency is retaining its 8-hour TWA PEL of 1000 ppm and adding a STEL of 1250 ppm. The Agency concludes that both of these limits are necessary to ensure that workers are protected and that good industrial hygiene practice is maintained.

METHYL DEMETON

CAS: 8022-00-2; Chemical Formula: $(CH_3O)_2PSO(CH_2)_2SC_2H_5$
H.S. No. 1256

OSHA formerly had no limit for methyl demeton. The ACGIH has a TLV-TWA limit of 0.5 mg/m³, with a skin notation. The proposed PEL was 0.5 mg/m³, with a skin notation, and NIOSH (Ex. 8-47, Table N1) concurs. The final rule establishes an 8-hour TWA of 0.5 mg/m³, and a skin notation, for methyl demeton. Methyl demeton is an oily, colorless to pale-yellow liquid with an unpleasant odor.

Methyl demeton is reported to have an oral LD_{50} value of 40 to 65 mg/kg for the thiolo isomer and 150 to 250 mg/kg for the thiono isomer. Both isomers form sulfoxide or sulfone, with an oral LD_{50} similar to that of the parent compounds (Dubois and Plzak 1962/Ex. 1-629; Heath and Vandekar 1965, Klimmer and Plaff 1955, both as cited in ACGIH 1986/Ex. 1-3, p. 388). In solution or storage, methyl demeton may form alkyl sulfonium compounds of very high intravenous toxicity and an oral LD_{50} of 10 to 20 mg/kg. Dermal toxicity is reported to be moderate, with an LD_{50} of approximately 400 mg/kg (Heath and Vandekar 1965, as cited in ACGIH 1986/Ex. 1-3, p. 388). OSHA received only one comment, from NIOSH, on methyl demeton.

In humans, methyl demeton causes changes in intraocular pressure, and acute poisonings produce nausea, headache, dizziness, vomiting, and hyperemia of the nasal mucosa. Chronic exposure causes hyperemia of the respiratory organs and inner ear irritation (Dugel'nyy 1970; Rasuleva 1970, both as cited in ACGIH 1986/Ex. 1-3, p. 388).

OSHA is establishing an 8-hour TWA for methyl demeton of 0.5 mg/m³, with a skin notation. The Agency concludes that this limit will protect workers from the significant risk of ocular and nasal irritation, pulmonary effects, and cholinesterase inhibition, all of which constitute material impairment of health and are associated with exposure to this substance at levels above the new limit.

METHYL ETHYL KETONE PEROXIDE

CAS: 1338-23-4 Chemical Formula: $C_8H_{18}O_6$
H.S. No.: 1257

OSHA did not formerly have a limit for methyl ethyl ketone peroxide (MEKP). The Agency proposed a PEL of 0.2 ppm (1.5 mg/m³) as a ceiling for this substance to protect workers against the significant risk of eye and skin irritation, as well as kidney and liver damage; this limit is consistent with that of the ACGIH. NIOSH (Ex. 8-47, Table N1) concurred with this limit. In the final rule, OSHA has determined that the data available at the present time indicate that 0.7 ppm (approximately 5 mg/m³) is an appropriate level at which to establish a PEL, and the final rule establishes this limit. MEKP is sold commercially as a colorless liquid mixture consisting of approximately 60 percent MEKP and 40 percent diluent; the diluent is added to reduce MEKP's sensitivity to shock.

The health effects data for MEKP in animals rely primarily on a study conducted in 1958 by Floyd and Stokinger (Ex. 1-783). In a series of experiments conducted in rats, mice, and rabbits to determine the toxicity of MEKP by various routes of exposure, these investigators found that inhalation exposure for four hours to a concentration of 200 ppm was fatal to 50 percent of rats, and a four-hour exposure to 170 ppm was fatal to 50 percent of mice. Inhalation of MEKP vapors produced petechial and gross hemorrhages of the lungs in rats after four-hour exposures; liver and kidney damage was also observed (Floyd and Stokinger 1958/Ex. 1-783). Two drops of a 40-percent solution of MEKP in dimethyl phthalate caused severe damage when instilled in rabbits' eyes, but at 3 percent, a moderate, transient reaction was produced. The direct application of MEKP to closely shaved rabbit skin caused no immediate discomfort but did cause a severe delayed reaction, consisting of erythema, edema, and vesiculation within two or three days; of the four organic peroxides tested (di-t-butyl peroxide, t-butyl hydroperoxide, cumene hydroperoxide, and methyl ethyl ketone peroxide), MEKP exhibited the greatest toxicity. The maximal nonirritating strength of MEKP applied dermally was 0.6 percent. In addition, rats died or

showed marked evidence of cumulative systemic effects after either oral or intraperitoneal administration of MEKP at 20 percent of the LD_{50} level for three days/week for seven weeks (Floyd and Stokinger 1958/Ex. 1–783).

OSHA received several comments on the proposed PEL for MEKP (Exs. 8–47, 8–86, 3–902, 144, 155, 181, and 3–1172; Tr. 11–265/266). Robert Schumacher, a certified industrial hygienist representing a group of six manufacturing companies (including the U.S. Marine Corporation), stated that the proposal did not adequately demonstrate occupational risk for MEKP because it relied on the findings of a single 30-year-old study that described the results of animal experiments involving "novel and unusual" exposures (Ex. 3–1172, Attachment; Exs. 86 and 155; Tr. 11–265/266). In addition, this commenter stated that information is lacking as to what concentrations of MEKP currently exist in the workplace, how to measure MEKP in the occupational environment, and the feasibility of engineering controls to regulate exposures to MEKP (Ex. 3–1172, Attachment; Exs. 8–86 and 155). The Motor Vehicle Manufacturers Association (MVMA) (Ex. 3–902) merely listed MEKP as a substance for which rulemaking should be delayed; however, the MVMA provided no details in support of this comment. The New Jersey Department of Health (Ex. 144) suggested that the limits for MEKP should be derived based on EPA's IRIS data; the use of IRIS data is discussed above, in Section VI.A of the preamble.

In response to Mr. Schumacher and the National Marine Manufacturers Association (Exs. 8–86, 155, 3–1172, and 181; Tr. 11–265/266), OSHA notes that the study of MEKP's toxicity performed by Floyd and Stokinger (1958/Ex. 1–783) was a thorough and comprehensive bioassay involving three species (mice, rats, and rabbits) and five routes of exposure inhalation, intraperitoneal, oral, dermal, and eye contact). According to the ACGIH (1986/Ex. 1–3, p. 396), this study demonstrated that MEKP was "20- to 50-fold more acutely toxic than di-ti-butyl peroxide by all routes tested." The consequences of exposure to this substance ranged from skin and eye irritation to gross hemorrhage of the lung and liver and kidney damage; OSHA notes that these effects were observed even after short-term exposures. The Floyd and Stokinger study (1958/Ex. 1–783) demonstrated that MEKP is significantly more toxic than benzoyl peroxide (TLV–TWA of 5 mg/m^3) and resembles hydrogen peroxide (TLV–TWA of 1.4 mg/m^3) in terms of its potential to cause irritation on an acute basis. A study by Moskowitz and Grabois (1950, as cited in ACGIH 1986/Ex. 1–3, p. 54) showed that exposure to 12.2 mg/m^3 benzoyl peroxide caused "pronounced irritation of the nose and throat" in workers; because MEKP is significantly more irritant than benzoyl peroxide, MEKP concentrations considerably below the 12 mg/m^3 level can be expected to cause irritation as well.

Sax and Lewis (1989, p. 2312) report that MEKP is an experimental tumorigen and note that systemic effects in humans resulting from oral exposure include changes in the structure or function of the esophagus, nausea or vomiting, and other gastrointestinal effects. NIOSH (Ex. 8–47) has carefully reviewed the health effects information and the limits proposed for MEKP by OSHA. NIOSH concurs that a ceiling limit is appropriate for MEKP to protect against this substance's severe irritant effects (see Table N1 of Ex. 8–47).

With regard to Mr. Schumacher's comments concerning concentrations of MEKP in the workplace and available controls, the record contains several NIOSH health hazard evaluations and technical assistance surveys taht were conducted in workplaces where MEKP was used as a reaction catalyst in polyester resin operations, the same type of operation of concern to Mr. Schumacher (NIOSH Health Hazard Evaluation Determination Report Nos. HE 79–132–673; HE 76–000–066; and HE 78–003–555). At two of the three sites surveyed, all personal and area MEKP air samples were below 1.5-mg/m.^{3} At the third site, a total of 20 short-term samples were taken over a three-day period to determine airborne MEKP exposures during the construction of fibrous glass reinforced products utilizing the styrene-modified polyester resin sprayup process and a MEKP catalyst; eight of these short-term samples exceeded the 1.5-mg/m^3 (0.2-ppm) level (NIOSH Health Hazard Evaluation Determination Report No. HE 78–003–555).

Mr. Schumacher (Ex. 155) was also of the opinion that OSHA had failed, in either the proposal or its supporting documents, to take into account the fact that a facility having multiple exposures would have to design its engineering controls to handle multiple chemicals; in the opinion of Mr. Schumacher, to control for multiple chemicals in a facility would be more expensive than controlling for a single substance. OSHA does not agree with Mr. Schumacher on either point. As discussed in Section VII of the preamble, OSHA's entire costing methodology is based on a process approach that assumes multiple chemical exposures and the use of a system of engineering controls designed to control the exposures of all employees involved in the process. OSHA believes this is a reasonable costing methodology because multiple chemicals are used at most processes. For example, in the sector of interest to Mr. Schumacher (SIC 37, Transportation Equipment), and particularly in boat-building, styrene, fiberglass, and MEKP are all used.

OSHA also conducted two site visits to MEKP-using facilities in connection with the present rulemaking (Exs. 136A and 136B). Both of the plants visited produced fiberglass boats, and personal samples were taken at both facilities for workers involved in gel-coat and lamination operations. One plant was a high-volume facility that produced approximately 24 boats per day, while the other plant produced only two to three boats per day. In the low-production facility, all MEKP sampling results for gel-coat and lamination workers were below 1.5-mg/m^3 for MEKP (Ex. 136A); at the high-production facility, the single MEKP sample taken on a gel-coat operator was 3.0 mg/m^3 (Ex. 136B). OSHA believes that the higher reading at the second facility is accounted for by the high rate of production at that site; controlling exposures at a high-volume facility requires the implementation of additional controls to compensate for the increase in production.

In regard to sampling and analytical methods for MEKP, OSHA notes that NIOSH has published a sampling and analytical method (PECA or 3508) for this substance and that OSHA has developed an in-house method that is available from the Agency on request; OSHA used this method without difficulty on the two site visits to MEKP-using facilities conducted for this rulemaking.

However, OSHA does find that the data in the record do not provide information that can be used to determine that 0.2 ppm (1.5 mg/m^3) represents an appropriate level at which to establish the final rule PEL for MEKP. For example, the Floyd and Stokinger study reports that MEKP is "20- to 50-fold more acutely toxic than di-t-butyl peroxide by all routes tested"; however, there is no PEL or TLV for di-t-butyl peroxide for OSHA to use as a basis for the PEL. The same study notes that MEKP is significantly more toxic than benzoyl peroxide (TLV–TWA of 5 mg/m^3) and resembles hydrogen peroxide in toxicity (TLV–TWA of 1.5 mg/m^3) but

provides no data to suggest how this "extra" toxicity might translate into a PEL. OSHA carefully reviewed the ACGIH (1986) documentation for this substance and also could find no specific basis for a ceiling of 0.2 ppm (1.5 mg/m^3).

Accordingly, OSHA has concluded that, at this time, the available data support establishing a PEL for MEKP that is at least equivalent to that for benzoyl peroxide (i.e., 5 mg/m^3, which is approximately equivalent to 0.7 ppm for MEKP). Given that MEKP is reported by Floyd and Stokinger to be more irritating than benzoyl peroxide and that irritation can result from even very brief exposures to excessive concentrations of MEKP, OSHA also concludes that a ceiling limit for MEKP is necessary and appropriate. Therefore, to reduce the significant risk of irritation to workers who are exposed to MEKP at higher levels, OSHA is establishing a 0.7 ppm ceiling PEL for MEKP.

METHYL FORMATE

CAS: 107-31-3; Chemical Formula: $HCOOCH_3$
H.S. No. 1258

OSHA had a limit of 100 ppm TWA for methyl formate. The ACGIH also has an 8-hour time-weighted average of 100 ppm, with a TLV-STEL of 150 ppm. OSHA proposed to retain the 8-hour TWA of 100 ppm for methyl formate and to add a STEL of 150 ppm; NIOSH (Ex. 8-47, Table N1) concurs that these limits are appropriate. The final rule retains the 8-hour TWA of 100 ppm and adds a 15-minute STEL of 150 ppm. Methyl formate is a flammable, colorless liquid with an agreeable odor.

Methyl formate causes nose and eye irritation, vomiting, incoordination, narcosis, and death in guinea pigs exposed at high concentrations (Schrenk, Yant, Chornyak, and Patty 1936/Ex. 1-756). A 5-percent concentration was fatal in 20 to 30 minutes, a 1.5- to 2.5-percent concentration was dangerous in 30 to 60 minutes, and a 0.5-percent concentration (5000 ppm) was considered the maximum concentration tolerable for a 60-minute period without serious consequences. Lehmann and Flury (1943b/Ex. 1-963) observed that inhalation of 1.02 percent methyl formate for two to three hours caused pulmonary edema and death in cats; a concentration of 1600 ppm resulted in lung inflammation after one hour. Guinea pigs died when exposed by inhalation to 2.5 percent methyl formate (Lehmann and Flury 1943b/Ex. 1-963).

In studies of methyl formate exposure in humans, von Oettingen (1959/Ex. 1-499) reported that exposed workers showed temporary blindness, narcosis, mucous membrane irritation, and dyspnea. Fairhall (1957c/Ex. 1-1107) has reported that methyl formate is more irritating than either methyl or ethyl acetate. Only NIOSH commented on this substance.

In the final rule, OSHA is retaining the 8-hour PEL of 100 ppm TWA and adding a STEL of 150 ppm to prevent the significant risks of irritation, narcotic effects, and pulmonary damage, which constitute material health impairments that are associated with exposure to concentrations of methyl formate even for short periods (one hour or more). The basis for this limit is analogy to the toxicity of methyl acetate. The Agency concludes that these limits will substantially reduce these significant risks.

METHYL IODIDE

CAS: 74-88-4; Chemical Formula: CH_3I
H.S. No. 1259

OSHA formerly had a limit of 5 ppm TWA, with a skin notation, for methyl iodide. The ACGIH has a TLV-TWA limit of 2 ppm, with a skin notation, for methyl iodide, and classifies it as a suspected human carcinogen (A2). NIOSH recommends reducing exposure to the lowest feasible limit, and also considers this chemical a carcinogen. The proposed PEL was an 8-hour TWA PEL of 2 ppm, with a skin notation; the final rule establishes these limits. Methyl iodide is a colorless, sweet-smelling liquid that turns yellow, red, or brown when exposed to light and moisture.

Methyl iodide has been reported to have an LD_{50} in rats of 150 to 200 mg/kg; liver damage was evident after these lethal exposures (Kutob and Plaa 1962/Ex. 1-61). Fifteen-minute exposures to 3800 ppm were fatal in rats (Chambers et al. 1950, as cited in ACGIH 1986/Ex. 1-3, p. 399), and Bachem (1927/Ex. 1-1013) has reported that methyl iodide is six times as toxic in mice as methyl bromide. Inhalation studies have shown eye irritation and depressed body weight in rats as a result of 14-week exposures to 30 and 60 ppm (Blank, Nair, Roloff, and Ribelin 1984/Ex. 1-619). The same authors observed fatalities in rats within four weeks of exposure to 143 ppm; 10 ppm was reported to be a no-effect level.

In industry, fatalities have occurred from methyl iodide poisoning in chemical workers (Garland and Camps 1945/Ex. 1-1190; Appel, Galen, O'Brien, and Schoenfeldt 1975/Ex. 1-1076). However, the exposure levels associated with these fatal overexposures are not known (ACGIH 1986/Ex. 1-3, p. 399).

In tests of carcinogenicity, methyl iodide produced local sarcomas in rats injected subcutaneously and lung tumors in mice given intraperitoneal injections (Druckrey, Kruse, Preussman et al. 1970/Ex. 1-246; Poirier, Stoner, and Shimkin 1975/Ex. 1-686). These carcinogenic effects occurred at a dosage approximately equivalent to a daily 8-hour exposure to 20 or 25 ppm for an adult human (ACGIH 1986/Ex. 1-3, p. 399). OSHA received comments on methyl iodide's health effects from the American Industrial Hygiene Association (AIHA). (Ex. 8-16; Tr. 3-309) and from NIOSH (Ex. 8-47, Table N6A). The AIHA stated that "[a] number of potentially carcinogenic substances for which PEL revisions are proposed appear to have been misclassified concerning their toxic effect" (Ex. 8-16, p. 6). The AIHA includes methyl iodide in this group of substances. As discussed in the introduction to Section VI.C, OSHA did not intend the proposal's classifications to have regulatory implications; rather, both this classification and that of the final rule are intended only to reflect the health endpoint used by the ACGIH or NIOSH as the basis for selecting a particular PEL for a given substance, and to facilitate generic rulemaking. NIOSH (Ex. 8-47, Table N6A) agreed that the methyl iodide limit established by OSHA is appropriate, but pointed out that this substance could be classified as an occupational carcinogen.

In the final rule, OSHA establishes an 8-hour TWA limit of 2 ppm, with a skin notation, for methyl iodide. The Agency concludes that these limits will protect workers from the significant risk of irritation and liver and kidney damage, which are material impairments of health that are associated with exposure to methyl iodide in the workplace. The skin notation is needed to prevent dermal absorption of toxic amounts of methyl iodide.

METHYL ISOAMYL KETONE

CAS: 110-12-3; Chemical Formula: $CH_3COCH(C_2H_5)_2$
H.S. No. 1260

OSHA formerly had no limit for methyl isoamyl ketone (MIAK). The ACGIH has established an 8-hour TLV-TWA of 50 ppm. NIOSH also recommends a 50-ppm TWA limit for MIAK. The proposed PEL was 50 ppm as an 8-hour TWA, with which NIOSH (Ex. 8-47, Table N1) concurs. The final rule establishes these limits. Methyl isoamyl ketone is a colorless, clear liquid with a pleasant odor.

The oral LD_{50} value of methyl isoamyl ketone in rats is 1.67 g/kg (Smyth,

Carpenter, Weil et al. 1962/Ex. 1–441). No data relating exposure levels to specific effects in humans have been reported. However, the ACGIH (1986/Ex. 1–3, p. 400) believes that MIAK is likely to be more irritating and a more potent narcotic than is the case for methyl isobutyl ketone.

The NIOSH criteria document on the ketones (1978f, as cited in ACGIH 1986/Ex. 1–3, p. 400) states that "because methyl isoamyl ketone contains one more carbon atom than does methyl isobutyl ketone, methyl [isoamyl] ketone might produce irritation and narcosis at concentrations at least as low as those at which methyl isobutyl ketone produces these effects," and NIOSH thus recommends a 50-ppm TWA for MIAK, corresponding to NIOSH's recommendation for methyl isobutyl ketone (NIOSH 1978f, as cited in ACGIH 1986/Ex. 1–3, p. 400). NIOSH submitted the only comments on MIAK.

In the final rule, OSHA is establishing an 8-hour TWA limit of 50 ppm for methyl isoamyl ketone. The Agency concludes that this limit will protect workers against the significant risk of narcotic and irritant effects, which constitute material health impairments that are associated with exposure to MIAK at levels above the new PEL.

METHYL ISOPROPYL KETONE

CAS: 563–80–4; Chemical Formula: $(CH_3)_2CHCOCH_3$
H.S. No. 1262

OSHA formerly had no limit for methyl isopropyl ketone (MIPK). The ACGIH has a TLV–TWA of 200 ppm. The proposed PEL was 200 ppm as an 8-hour TWA; NIOSH (Ex. 8–47, Table N1) concurs, and the final rule establishes this limit. Methyl isopropyl ketone is a colorless, flammable liquid.

Animal studies have shown MIPK to have an acute toxicity somewhat greater than that of diethyl ketone and somewhat less than that of di-n-propyl ketone or methyl-n-propyl ketone (ACGIH 1986/Ex. 1–3, p. 405). Rats exposed for four hours at a concentration of 5700 ppm died (NIOSH 1977i, Ex. 1–1182). Other data concerning the inhalation toxicity of MIPK are lacking. Dr. Grace Ziem (Ex. 46) noted that respiratory irritation, headaches, and nausea have been demonstrated to occur in humans at low levels of MIPK exposure.

OSHA establishes in the final rule a limit of 200 ppm TWA for methyl isopropyl ketone. The Agency concludes that this limit will protect workers against the significant risk of irritation, a material health impairment that is associated with exposure to this ketone at levels above the new PEL.

METHYL PARATHION

CAS: 298–00–0; Chemical Formula: $C_8H_{10}NO_5PS$
H.S. No. 1265

OSHA formerly had no limit for methyl parathion. The ACGIH has a TLV–TWA of 0.2 mg/m³, with a skin notation. NIOSH also recommends a TWA of 0.2 mg/m³, and a skin notation for methyl parathion. The proposed PEL was an 8-hour TWA limit of 0.2 mg/m³, with a skin notation; NIOSH (Ex. 8–47, Table N1) concurs with these limits, and they are established in the final rule. Methyl parathion is a tan to brown liquid with a pungent odor like that of garlic.

Methyl parathion is an acetylcholinesterase inhibitor, and excessive exposure can cause sweating, salivation, diarrhea, bradycardia, bronchoconstriction, muscle fasciculations, and coma. Methyl parathion's acute oral LD_{50} for male rats is almost identical to that of parathion, (i.e., 10 to 25 mg/kg); for female rats, the LD_{50} is 24 mg/kg, or approximately one-sixth that of parathion. By the dermal route, methyl parathion is much less toxic than parathion, with an LD_{50} of 67 mg/kg in rats of both sexes (Hayes 1963/Ex. 1–982). Erythrocyte cholinesterase activity was inhibited in dogs fed methyl parathion for 12 weeks at a rate corresponding to approximately 24 mg/day; inhibition of both plasma and erythrocyte cholinesterase activity occurred at doses of 70 mg/day, without accompanying illness (Williams, Fuyat, and Fitzhugh, 1959/Ex. 1–810). Dogs fed 6 mg/day methyl parathion for 12 weeks showed no effects from such exposures (Williams, Fuyat, and Fitzhugh 1959/Ex. 1–810). Lifetime feeding studies of rats and mice fed diets containing methyl parathion concentrations of up to 40 ppm and up to 125 ppm, respectively, produced no evidence of cancer (NCI 1979a/Ex. 1–1116).

Plasma and erythrocyte cholinesterase levels did not differ by more than 20 percent in subjects exposed at 7, 7.5, 8, or 9 mg/man/day, compared with controls (Moeller and Rider 1963/Ex. 1–565). Tiess, Wegener, and Tamme (1982/Ex. 1–774) have reported a case of protracted methyl parathion poisoning resulting from both percutaneous and inhalation exposures; Dille and Smith (1964/Ex. 1–549) attribute the long-term neuropsychiatric illness of two pilots to exposure to methyl parathion and other cholinesterase-inhibiting agents. Chronic exposure to small doses of methyl parathion have not caused chromosomal effects (de Cassia Stocco, Becak, Gaeta, and Rabello-Gay 1982/Ex. 1–540). No comments other than those from NIOSH were received on methyl parathion.

In the final rule, OSHA establishes a limit of 0.2 mg/m³ TWA for methyl parathion, with a skin notation. The Agency concludes that this limit will protect workers against the significant risk of acetylcholinesterase inhibition, which constitutes a material impairment of health that is associated with workplace exposures at levels above the new PEL. The skin notation will protect workers from the significant risk of systemic toxicity associated with percutaneous absorption of this substance.

METHYLCYCLOHEXANE

CAS: 108–87–2; Chemical Formula: C_7H_{14}
H.S. No. 1268

OSHA had an 8-hour TWA limit of 500 ppm for methylcyclohexane, and the ACGIH has a limit of 400 ppm TWA for this colorless liquid. The proposed PEL was 400 ppm; NIOSH concurred that this reduction in the TWA was appropriate (Ex. 8–47, Table N1). The final rule reduces the 8-hour TWA for methylcyclohexane from 500 ppm to 400 ppm.

Lehmann and Flury (1943e, as cited in ACGIH 1986/Ex. 1–3, p. 384) indicate that the acute toxicity of methylcyclohexane is greater than that of heptane but less than that of octane. Lazarew (1929/Ex. 1–1059) found that a two-hour exposure to a concentration of 7500 to 10,000 ppm caused prostration in mice, and exposure to 10,000 to 12,500 ppm caused death. Treon, Crutchfield, and Kitzmiller (1943b/Ex. 1–394) reported that exposure to 1200 ppm had no effect in rabbits, and prolonged exposures to 370 ppm had no effect in monkeys. Methylcyclohexane's histologic effects in animals resemble those of cyclohexane; the liver and kidney are the sites affected (ACGIH 1986/Ex. 1–3, p. 384). Only NIOSH commented on methylcyclohexane.

OSHA establishes an 8-hour TWA limit of 400 ppm for methylcyclohexane in the final rule. The Agency concludes that this limit will protect workers against the significant risk of irritation, a material health impairment that is associated with exposure to methylcyclohexane at levels above the new PEL.

2-METHYLCYCLOPENTADIENYL MANGANESE TRICARBONYL

CAS: 12108–13–3; Chemical Formula: $(CH_3)C_5H_5$–$Mn(CO)_3$
H.S. No. 1271

OSHA formerly had no limit for 2-methylcyclopentadienyl manganese tricarbonyl (Cl–2). The ACGIH has a

TLV-TWA of 0.2 mg/m³, measured as manganese, with a skin notation. The proposed PEL was 0.2 mg/m³ as an 8-hour TWA, with a skin notation; NIOSH (Ex. 8-47, Table N1) concurs. This limit, measured as manganese, is established in the final rule, along with a skin notation. Cl-2 is a dark orange liquid with a faintly pleasant odor; it is a complex organic compound containing about 25 percent manganese by weight.

2-Methylcyclopentadienyl Mn tricarbonyl is highly toxic in its concentrated form, causing adverse effects primarily on the central nervous system. It is somewhat irritating to the eyes but skin contact does not produce irritation or sensitization; however, Cl-2 is readily absorbed through the skin (ACGIH 1986/Ex. 1-3, p. 387). Animal studies indicate that Cl-2 has a toxicity similar to that of tetraethyl lead and is highly toxic by all routes of exposure (U.S. Navy Smoke Abatement Additive, as cited in ACGIH 1986/Ex. 1-3, p. 387).

The single-dose oral LD_{50} for rats is 23 or 39 mg/kg, depending on sex. The skin LD_{50} for rabbits is 1692 ± 145 mg/kg, and the 1-hour inhalation LC_{50} for rats is about 350 mg/m³ (The Ethyl Corporation, as cited in ACGIH 1986/Ex. 1-3, p. 387). Toxic exposures by all routes produce rapidly appearing symptoms of mild excitement, hyperactivity, tremors, severe clonic spasms, weakness, respiratory distress, and occasional clonic convulsions, followed by terminal coma (U.S. Navy Smoke Abatement Additive, as cited in ACGIH 1986/Ex. 1-3, p. 387).

Acute exposure causes damage to the liver, kidneys, and cerebral cortex, as well as changes in lung tissue (ACGIH 1986/Ex. 1-3, p. 387). Browning (1966/Ex. 1-1018) observed chronic bronchitis, peribronchitis, interstitial pneumonia, and lung abscesses in animals that subsequently died from long-term inhalation exposure to Cl-2; exposure to Cl-2 concentrations of approximately 12 mg/m³ for 100 days produced no deviation in weight gain patterns and no gross or microscopic changes in two dogs (Browning 1966/Ex. 1-1018). The liver and kidneys are the principal target organs associated with acute overexposures; the lungs of overexposed animals were hemorrhagic (Browning 1966/Ex. 1-1018).

In humans, skin contact should be entirely avoided. A 5- to 15-ml spill on one worker's hand and wrist was reported to have caused "thick tongue," nausea, giddiness, and headache within 3 to 5 minutes (U.S. Navy Smoke Abatement Additive, as cited in ACGIH 1986/Ex. 1-3, p. 387). NIOSH submitted the only comment on this substance.

In the final rule, OSHA establishes a PEL of 0.2 mg/m³ TWA, measured as manganese, with a skin notation, for 2-methylcyclopentadienyl manganese tricarbonyl. The Agency concludes that this limit will protect workers against the significant risk of CNS effects and systemic damage, which constitute material health impairments and are associated with exposure to Cl-2 at levels higher than the new PEL. A skin notation is established because of Cl-2's demonstrated ability to penetrate human skin rapidly and to cause systemic effects.

MONOCROTOPHOS (AZODRIN)

CAS: 6923-22-4; Chemical Formula: $C_7H_{14}NO_5P$
H.S. No. 1279

OSHA formerly had no limit for the systemic insecticide monocrotophos. The ACGIH has a TLV-TWA of 0.25 mg/m³ for this reddish-brown solid with a mild ester odor. The proposed PEL was 0.25 mg/m³ as an 8-hour TWA; NIOSH (Ex. 8-47, Table N1) concurs with this limit, which is established in the final rule.

Monocrotophos is a highly toxic, direct acting cholinesterase inhibitor that penetrates the intact skin (ACGIH 1986/Ex. 1-3, p. 416). The acute oral LD_{50} values in rats and mice range from 5.7 to 17 mg/kg in a water formulation (Brown et al. 1970, Shellenberger and Newell, both as cited in ACGIH 1986/Ex. 1-3, p. 416) and from 10 to 23 mg/kg in an oil formulation (Shellenberger and Newell, as cited in ACGIH 1986/Ex. 1-3, p. 416). These authors also report a percutaneous LD_{50} in the rabbit that ranges from 112 to 709 mg/kg, depending on the vehicle used. A two-year dietary study of rats ingesting 0, 1, 10, or 100 ppm monocrotophos revealed that both sexes in the 100 ppm group failed to gain as much weight as the controls, but autopsy showed no significant findings; plasma, erythrocyte, and brain cholinesterase decreased at the two highest dose levels but were unaffected at 1 ppm (Johnston 1966-67, as cited in ACGIH 1986/Ex. 1-3, p. 416). Another two-year feeding study in dogs administered doses of up to 16 ppm monocrotophos revealed no adverse effects at levels of 0.16 and 1.6 ppm, but serious cholinesterase reduction was observed at the 16-ppm level (Johnston 1966-67, as cited in ACGIH 1986/Ex. 1-3, p. 416). Metabolism studies in rats and goats indicate that monocrotophos is excreted rapidly in the rat and does not accumulate in the body (Bull and Lindquist 1966/Ex. 1-719; goats given labeled monocrotophos by mouth showed only traces of the material in their milk (Menzer and Casida 1965/Ex. 1-986; Potter, as cited in ACGIH 1986/Ex. 1-3, p. 416). Inhalation exposure of rats to an unknown concentration of 75 percent monocrotophos in air for one hour was not lethal; a four-hour exposure to an unknown concentration of the aerosol (0.4 and 0.75 percent) was fatal to two out of six (0.4 percent aerosol) and five out of eight rats (0.75 percent aerosol). Head-only exposure to the 0.4-percent aerosol resulted in the death of one of eight animals (Wilson, as cited in ACGIH 1986/Ex. 1-3, p. 416).

Intravenous injection of radiolabeled monocrotophos in human volunteers showed maximum excretion at 4 to 8 hours, with 67±5 percent of the material in the urine; absorption of 14 ±7 percent occurred when the radiolabeled material was applied to the forearm; 33±9 percent of the applied dose was absorbed when it was covered with a vapor-proof film for 72 hours (Maibach 1970, as cited in ACGIH 1986/Ex. 1-3, p. 416). Although gauze patches attached to the clothing and skin of field workers attested to the presence of monocrotophos, no cholinesterase inhibition was observed in post-exposure examinations at three hours and at three and seven days (Maibach, as cited in ACGIH 1986/Ex. 1-3, p. 416). Only NIOSH commented on this substance.

OSHA is establishing a PEL of 0.25 mg/m³ (8-hour TWA) for monocrotophos in the final rule. The Agency concludes that this limit will protect workers against the significant risk of cholinesterase inhibition, a material impairment of health that is associated with exposure to this substance in the workplace at levels above the new PEL.

MORPHOLINE
CAS: 110-91-8; Chemical Formula: C_4H_9NO
H.S. No. 1281

OSHA had a limit of 20 ppm, with a skin notation, for morpholine. The ACGIH has a 20-ppm TWA limit and a TLV-STEL of 30 ppm, as well as a skin notation. The proposal retained the 8-hour TWA PEL of 20 ppm and added a STEL of 30 ppm; NIOSH concurs that these limits are appropriate (Ex. 8-47, Table N1), and they are established in the final rule. The skin notation is retained. Morpholine is a colorless liquid with an amine-like odor.

Exposure to morpholine produces nasal and bronchial irritation and liver and kidney impairment in animals (Shea 1939/Ex. 1-758); the substance readily penetrates the skin and is highly irritating to the eyes (Jefferson Chemical Company, Inc. 1961, as cited in ACGIH 1986/Ex. 1-3, p. 417). The single oral LD_{50} in rats is 1.05 g/kg (range: 0.95 to 1.16 g/kg), and the single skin LD_{50} for 24-hour contact is 0.5 ml/kg (Smyth, Carpenter, Weil, and Pozzani 1954/Ex.

1–440). Neither a one-hour exposure to concentrated vapor nor an 8-hour exposure to 8000 ppm was fatal in rats (Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1–440). Rats were exposed for eight hours daily to a concentration of 18,000 ppm for a total of five days; after the first day, all animals showed severely reddened thoracic walls, and one fatality (from kidney and liver congestion) occurred. A similar fatality occurred on the third day; on day 4, a third rat died, and postmortem examination revealed degeneration of the epithelial lining of the kidney tubules. Three additional deaths occurred after the exposures had ended; autopsy revealed thickened alveoli, emphysema, and liver and kidney effects (Shea 1939/Ex. 1–758).

Reporting on his own reactions to morpholine exposure at a concentration of 12,000 ppm, Shea (1939/Ex. 1–758) complained of nose irritation (after 1 minute) and coughing (after 90 seconds); in addition, when he transferred morpholine by pipette, he experienced sore throat and mucosal irritation. All symptoms disappeared after the experiment stopped (Shea 1939/Ex. 1–758). Skin contact poses a moderately high degree of hazard, which diminishes as the product is diluted with water to less than 25 percent (Jefferson Chemical Company, Inc. 1961, as cited in ACGIH 1986/Ex. 1–3, p. 417). Respiratory irritation but no chronic effects have been reported as a result of industrial exposure (Patty 1963e/Ex. 1–858). In comparison with ammonia, morpholine has a greater potential for systemic toxicity (ACGIH 1986/Ex. 1–3, p. 417).

OSHA received a comment on morpholine from Lawrence Hecker of Abbott Laboratories (Ex. 3–678). Dr. Hecker states (Ex. 3–678, p. 8) that the STEL proposed for morpholine should not be included in the final rule because the health evidence for this substance does not warrant a STEL. OSHA does not agree with Dr. Hecker; there is evidence in the record that morpholine's effects are experienced even at elevated exposures lasting only one minute (Shea 1939/Ex. 1–758). Because morpholine has a greater potential for systemic effects than does ammonia, a STEL is needed to ensure that short-term excursions substantially above the 8-hour TWA PEL do not occur.

In the final rule, OSHA is retaining the 8-hour TWA limit for morpholine of 20 ppm TWA and the skin notation, and is adding a 15-minute STEL of 30 ppm. The Agency concludes that these limits will work together to protect workers against the significant risk of eye and respiratory tract irritation, which are material impairments of health that are associated with exposures at levels above the 8-hour TWA limit. OSHA is retaining the skin notation for morpholine because of this substance's ability to be absorbed through the skin in toxic amounts.

NITRIC ACID

CAS: 7697–37–2; Chemical Formula: HNO_3
H.S. No. 1286

OSHA had an 8-hour TWA limit of 2 ppm for nitric acid. The ACGIH has the same TWA limit and a 15-minute STEL of 4 ppm, and NIOSH recommends a TWA limit of 2 ppm. The proposal retained the 8-hour TWA PEL of 2 ppm for nitric acid and added a STEL of 4 ppm; NIOSH (Ex. 8–47, Table N1) concurred with these limits, which are established in the final rule. Nitric acid is a fuming colorless or yellowish liquid.

Rats receiving a single exposure to nitric acid mist at a concentration of 63 mg/m^3 exhibited no apparent adverse effects (Diggle and Gage 1954/Ex. 1–729). Chronic exposure to airborne nitric acid vapor or mist at unspecified levels was reported to cause chronic bronchitis, pneumonitis (Fairhall 1957i, as cited in ACGIH 1986/Ex. 1–3, p. 428), and tooth erosion (Lynch and Bell 1947/Ex. 1–793). Nitric acid's irritant potential is considered similar to that of other strong acids; it typically exists in conjunction with nitrogen dioxide, which is regarded as being more hazardous (ACGIH 1986/Ex. 1–3, p. 428). No comments, other than NIOSH's, were submitted on this substance.

OSHA is retaining the 8-hour TWA PEL of 2 ppm and adding a STEL of 4 ppm for nitric acid in the final rule. The Agency concludes that this combined limit will protect workers against the significant risk of irritation, chronic pulmonary disease, and dental corrosion, which together constitute a material impairment of health.

p-NITROANILINE

CAS: 100–01–6; Chemical Formula: $NO_2C_6H_4NH_2$
H.S. No. 1287

OSHA formerly had a limit of 1 ppm TWA (6 mg/m^3) for p-nitroaniline (PNA), with a skin notation. The ACGIH has a limit of 3 mg/m^3 TWA, with a skin notation. OSHA proposed to reduce the former 8-hour TWA of 1 ppm (equivalent to 6 mg/m^3) to 3 mg/m^3, and to retain the skin notation. NIOSH (Ex. 8–47, Table N1) concurred that these limits were appropriate, and they are established in the final rule. para-Nitroaniline usually exists in the form of yellow needles.

p-Nitroaniline is readily absorbed through the skin and is a strong methemoglobin-forming agent; prolonged exposure can cause liver damage (ACGIH 1986/Ex. 1–3, p. 430). Anderson (1946/Ex. 1–1049) reported several cases of PNA-poisoning among shipboard workers assigned to clean up a p-nitroaniline spill; one man with a history of liver disease became jaundiced and died, and the other exposed workers became cyanotic and complained of headache, sleepiness, weakness, and respiratory distress (Anderson 1946/Ex. 1–1049). It has also been reported that children who ingested p-nitroaniline that was contained in wax crayons subsequently became ill (Rieders and Brieger 1953/Ex. 1–798).

Several investigators (Anderson 1946/Ex. 1–1049; Gupta 1953, Fairhall 1957j, both as cited in ACGIH 1986/Ex. 1–3, p. 430; Linch 1974/Ex. 1–747) have concluded that the nitroanilines are more hazardous than aniline, and, on this basis, the ACGIH has recommended a TWA Limit for PNA that is lower than the limit for aniline (ACGIH 1986/Ex. 1–3, p. 430). Only NIOSH submitted comments on p-nitroaniline.

In the final rule, OSHA is establishing a PEL of 3 mg/m^3 (8-hour TWA) for p-nitroaniline and is retaining the skin notation. The Agency concludes that this limit will protect workers against the significant risk of methemoglobinemia and liver damage, both of which constitute material health impairments that are associated with exposure to PNA at levels above 3 mg/m^3. The Agency is retaining the skin notation because this substance is readily absorbed through the skin in toxic amounts.

NITROTOLUENE

CAS: 88–72–2 (o-isomer); 99–08–1 (m-isomer); 99–99–0 (p-isomer);
Chemical Formula: $CH_3C_6H_4NO_2$
H.S. No. 1292

OSHA formerly had an 8-hour TWA limit of 5 ppm, with a skin notation, for nitrotoluene. The ACGIH has a TLV-TWA of 2 ppm, also with a skin notation. The proposed PEL was 2 ppm as an 8-hour TWA, with a skin notation, and NIOSH concurred with this limit (Ex. 8–47, Table N1). The final rule establishes an 8-hour TWA PEL for nitrotoluene of 2 ppm and retains the skin notation. The ortho- and meta-isomers of nitrotoluene are yellow liquids; the para-isomer is also yellow, but exists in crystalline form.

Nitrotoluene is one of the aromatic nitrogen compounds that may cause methemoglobin formation. Linch (1974/Ex. 1–747) has studied the nitrotoluene isomers and reported that they have

relatively low emiagenic potential; he considered nitrotoluene comparable to aniline in its toxic effects (Linch 1974/Ex. 1–747). Cases of poisoning as a result of exposure to nitrotoluene are rare (von Oettingen 1941/Ex. 1–874). Only NIOSH commented on this substance.

In the final rule, OSHA establishes an 8-hour TWA limit of 2 ppm and retains the skin notation for nitrotoluene. The Agency concludes that this limit will protect workers against the significant risk of methemoglobinemia, a material health impairment that is associated with exposure to this substance; the skin notation is retained because of nitrotoluene's capacity to penetrate the skin.

NONANE

CAS: 111–84–2; Chemical Formula: $CH_3(CH_2)_7CH_3$
H.S. No. 1293

Previously, OSHA has no limit for nonane. The ACGIH has a TLV–TWA of 200 ppm for this colorless liquid. The proposed PEL was 200 ppm; NIOSH concurs that this limit is appropriate (Ex. 8–47, Table N1). The final rule promulgates an 8-hour TWA PEL for nonane of 200 ppm.

The toxicity of nonane is approximately equal to that of VM&P naphtha. Naphtha has a 4-hour inhalation LC_{50} for rats of 3400 ppm, while nonane has an LC_{50} of 3200 ppm (Carpenter, Kinkead, Geary et al. 1975a/Ex. 1–302; Carpenter, Geary, Myers et al. 1978/Ex. 1–301). These investigators found a no-effect level of 590 ppm nonane for rats exposed six hours/day, five days/week for a 65-day period; under the same exposure conditions, a no-effect level of 560 ppm was reported for rats exposed to VM&P naphtha (Carpenter, Kinkead, Geary et al. 1975a/Ex. 1–302; Carpenter, Geary, Myers et al. 1978/Ex. 1–301). Earlier studies of octane and heptane have resulted in much higher LC_{50} values for mice, i.e., 13,500 ppm and 16,000 ppm, respectively, for 30- to 60-minute exposures (Flury and Zernik 1931j/Ex. 1–994). Swann and associates (1974/Ex. 1–124) have reported similarly high LD_{50} values in mice for octane and hexane; mice died from respiratory arrest after 3 to 5 minutes of exposure to 16,000 ppm of octane or to 48,000 ppm of hexane (Swann, Kwon, and Hogan 1974/Ex. 1–124). The AFL–CIO (Ex. 194) and the United Auto Workers (Ex. 197) favor a 10 ppm PEL for all petroleum solvents and urge OSHA to consider a lower PEL.

In the final rule, OSHA establishes an 8-hour TWA limit of 200 ppm for nonane. The Agency concludes that this limit will protect workers against the significant risk of narcosis, a material impairment of health that is associated with exposure to nonane at levels above the new PEL.

OXALIC ACID

CAS: 144–62–7; Chemical Formula: $H_2C_2O_4$
H.S. No. 1299

OSHA formerly had a limit of 1 mg/m³ for oxalic acid. The ACGIH has a TLV–TWA of 1 mg/m³ and a TLV–STEL of 2 mg/m³. The proposal retained the 1-mg/m³ 8-hour TWA limit but added a STEL of 2 mg/m³; NIOSH (Ex. 8–47, Table N1) concurs with these limits. The final rule retains the 8-hour TWA PEL of 1 mg/m³ for oxalic acid and adds a STEL of 2 mg/m³. Anhydrous oxalic acid usually occurs in the form of a white powder; the dihydrate form is a colorless, odorless, crystalline substance.

Oxalic acid is known to produce severe burns of the eyes, mucous membranes, and skin (Windholz 1983d/Ex. 1–835, p. 991). There have been human fatalities from ingesting as little as 5 grams of oxalic acid. It appears that these deaths were caused by oxalic acid's ability to disturb the calcium-potassium balance in critical tissues (Klauder, Shelanski, and Gabriel 1955/Ex. 1–1057). Solutions of 5- to 10-percent oxalic acid have also been reported to irritate the skin on prolonged exposure. NIOSH was the only commenter on oxalic acid.

Because of oxalic acid's severe acute toxicity, OSHA is retaining the 8-hour TWA limit of 1 mg/m³ PEL and adding a STEL of 2 mg/m³ in the final rule. The Agency concludes that both of these limits are required to protect exposed workers from the significant risk of severe eye and skin burns and respiratory tract irritation, which are material health impairments associated with elevated short-term exposures at levels above the TWA limit.

PERCHLORYL FLUORIDE

CAS: 7616–94–6; Chemical Formula: ClO_3F
H.S. No. 1309

OSHA's former 8-hour TWA limit for perchloryl fluoride was 3 ppm. The ACGIH has a TLV–TWA of 3 ppm and a STEL of 6 ppm for this colorless gas with a sweet odor. The proposal retained the 8-hour TWA PEL of 3 ppm for perchloryl fluoride and added a STEL of 6 ppm. NIOSH (Ex. 8–47, Table N1) concurs that these limits are appropriate and they are established in the final rule.

The 4-hour LC_{50}s in rats and mice were 385 and 630 ppm, respectively. Dogs exposed for 4 hours to 220- to 450-ppm concentrations of the vapor, followed by exposure to 620 ppm for 2.5 hours, became hyperneic and cyanotic and showed increased methemoglobin. Dogs succumbing to these exposures had pigment deposition in the liver, spleen, and bone marrow; alveolar hemorrhage and collapse; and emphysema (Greene, Colbourn, Donati, and Weeks 1960, as cited in ACGIH 1986/Ex. 1–3, p. 466).

Exposure to 185 ppm for six hours/day, five days/week for seven weeks killed 18 of 20 rats, 20 of 39 mice, and all exposed guinea pigs (Greene, Colbourn, Donati, and Weeks 1960, as cited in ACGIH 1986/Ex. 1–3, p. 466). These animals had difficulty breathing, became cyanotic, and developed alveolar edema and methemoglobinemia; at autopsy, they showed fluorosis, patchy lungs, enlarged spleens, and hemosiderosis of the kidneys, spleen, and liver. When animals were exposed on a similar regimen but to a concentration of 104 ppm for six weeks, all guinea pigs but only 1 of 20 rats died (Greene, Colbourn, Donati, and Weeks 1960, as cited in ACGIH 1986/Ex. 1–3, p. 466). After a six-month exposure to 24 ppm, bone fluoride levels increased fourfold in guinea pigs, threefold in rats, and about 50 percent in dogs. Animals exposed at 24 ppm showed no signs of irritation (Greene, Colbourn, Donati, and Weeks 1960, as cited in ACGIH 1986/Ex. 1–3, p. 466). Only NIOSH commented on perchloryl fluoride.

In the final rule, OSHA is retaining the 8-hour TWA of 3 ppm and adding a STEL of 6 ppm for perchloryl fluoride. These limits are based on the fluoride content of this compound. The Agency concludes that this combined limit will protect workers from the significant risk of fluorosis and hematologic effects, which together constitute material impairments of health that are associated with exposures to perchloryl fluoride at levels above these limits.

PHOSDRIN (MEVINPHOS)

CAS: 7786–34–7; Chemical Formula: $C_7H_{13}O_6P$
H.S. No. 1320

OSHA formerly had an 8-hour TWA limit of 0.1 mg/m³, with a skin notation, for phosdrin (mevinphos). The ACGIH has a TLV–TWA of 0.01 ppm (0.1 mg/m³) and a TLV–STEL of 0.03 ppm (0.3 mg/m³), also with a skin notation. The proposal retained the 8-hour TWA of 0.1 mg/m³ and added a STEL of 0.3 mg/m³; the skin notation was retained. NIOSH (Ex. 8–47, Table N1) concurred with these limits, which are established in the final rule. Phosdrin is a colorless liquid. The commercial product is a mixture of cis- and trans-isomers that have a yellow color.

The acute oral LD_{50} of phosdrin is 4 to 8 mg/kg for male mice and 6 to 8 mg/kg for female rats (Shell Chemical Corporation 1956, as cited in ACGIH 1986/Ex. 1–3, p. 412). Phosdrin is a cholinesterase inhibitor and has been reported to cause slight plasma cholinesterase depression but no decrease in brain cholinesterase activity in rats fed 2 to 5 ppm. The compound may be absorbed dermally and by inhalation or ingestion; the action of the compound is direct and immediate (Cleveland and Treon 1961/Ex. 1–476). The dermal LD_{50} in rats has been reported to be 4.5 mg/kg (Gaines 1969/Ex. 1–320). Chronic feeding of rats demonstrated a minimal lethal dose of between 100 and 200 ppm. Cholinesterase activity decreased continually when sublethal doses were administered until a maximum reduction in RBC cholinesterase activity of 25 percent was achieved on the 27th day of the administration of 1.5 to 20 mg doses (Huelse and Federspil 1975/Ex. 1–959).

In industry, the primary hazards associated with exposure to phosdrin are absorption of phosdrin through the skin, lung, and mucous membranes, which causes liver damage (Natoff 1970/Ex. 1–966). Phosdrin intoxication is reported to occur in human, with accompanying symptoms of headache, visual distortion, weakness, cramps, diarrhea, pain, and respiratory distress. Severe exposure may cause convulsions; in one reported case, some symptoms (anxiety, depression, vertigo, and nystagmus) persisted for as long as four months (Zavon, as cited in ACGIH 1986/Ex. 1–3, p. 412). Only NIOSH commented on this substance.

In the final rule, OSHA is retaining the 8-hour TWA PEL of 0.1 mg/m^3 and adding a STEL of 0.3 mg/m^3 for phosdrin; the skin notation is retained. These limits are based on analogy to the toxicity of parathion. The Agency concludes that these limits will protect workers against the significant risk of cholinesterase inhibition and hepatic injury, which constitute material health impairments that result from the absorption of phosdrin through the skin and mucous membranes and from exposure by the inhalation and oral routes. OSHA finds that these limits will substantially reduce these significant risks.

PHOSPHORUS OXYCHLORIDE

CAS: 10025–87–3; Chemical Formula: $POCl_3$
H.S. No. 1323

OSHA had no former limit for phosphorus oxychloride. The ACGIH has a TLV–TWA of 0.1 ppm and a TLV-STEL of 0.5 ppm for this clear, colorless, fuming liquid, which has a pungent odor. The proposed PELs were 0.1 ppm as an 8-hour TWA and 0.5 ppm as a 15-minute STEL. NIOSH (Ex. 8–47, Table N1) concurred with these limits. The final rule establishes an 8-hour TWA of 0.1 ppm for this substance but, for the reasons discussed below, does not include a STEL for phosphorus oxychloride.

The primary hazards associated with inhalation of phosphorus oxychloride vapor are irritation of the eyes and respiratory tract, as well as narcotic effects, gastric irritation, pulmonary edema, and nephritis (International Technical Information Institute 1978/Ex. 1–837).

Weeks and associates (1964, as cited in ACGIH 1986/Ex. 1–3, p. 485) reported 4-hour LC_{50} values for phosphorus oxychloride of 48 ppm and 52 ppm for rats and guinea pigs, respectively. They also observed that ammonia vapor mediates the irritant effects of exposure to phosphorus oxychloride without significantly altering this LC_{50} value (Weeks, Downing, Musselman et al. 1964, as cited in ACGIH 1986/Ex. 1–3, p. 485).

Both chronic and acute occupational intoxication have been reported to occur among workers exposed to phosphorus oxychloride (Sassi 1954/Ex. 1–931).

The American Industrial Hygiene Association (AIHA) recommended that OSHA delete the STEL for phosphorus oxychloride (Tr. 3–307, Ex. 8–16) on the grounds that the ACGIH intends to delete this STEL. After a review of the available evidence for this substance, OSHA is not including a STEL for phosphorus oxychloride in the final rule. The Agency's reasoning on this issue is discussed in Section VI.C.17 of this preamble.

In the final rule, OSHA is establishing a PEL of 0.1 ppm (8-hour TWA) for phosphorus oxychloride, by analogy to the toxicity of phosphorus trichloride. The Agency concludes that this limit will reduce the significant risk of narcosis and systemic poisoning, which are material health impairments that are associated with acute and chronic exposure at levels above the new PEL.

PHOSPHORUS PENTASULFIDE

CAS: 1314–80–3; Chemical Formula: P_2S_5
H.S. No. 1324

OSHA formerly had a limit of 1 mg/m^3 as an 8-hour TWA for phosphorus pentasulfide. The ACGIH also has a limit of 1 mg/m^3 TWA but adds a 15-minute STEL of 3 mg/m^3. The proposal retained the 8-hour TWA PEL of 1 mg/m^3 and added a STEL of 3 mg/m^3; NIOSH (Ex. 8–47, Table N1) concurs with these limits. The final rule retains the 8-hour TWA PEL of 1 mg/m^3 for phosphorus pentasulfide and adds a STEL of 3 mg/m^3. Phosphorus pentasulfide is a greenish-yellow crystalline mass with an odor like that of rotten eggs.

The primary hazard associated with exposure to phosphorus pentasulfide is respiratory irritation (Smyth 1956/Ex. 1–759). In the presence of moisture, phosphorus pentasulfide is rapidly hydrolyzed to phosphoric acid and hydrogen sulfide. The ACGIH (1986/Ex. 1–3, p. 485) considers phosphorus pentasulfide to be as toxic as phosphoric acid. Only NIOSH commented on this substance.

In the final rule, OSHA is retaining the 8-hour TWA PEL of 1 mg/m^3 and adding a 15-minute STEL of 3 mg/m^3 for phosphorus pentasulfide. The Agency concludes that both of these limits are necessary to reduce the significant risk of respiratory irritation, a material health impairment that is associated with exposure to this substance at the higher concentrations permitted in the past by the TWA alone.

PHTHALIC ANHYDRIDE

CAS: 85–44–9; Chemical Formula: $C_6H_4(CO)_2O$
H.S. No. 1326

OSHA had an 8-hour TWA limit of 2 ppm for phthalic anhydride. The ACGIH has a limit of 1 ppm TWA for phthalic anhydride, which exists in the form of white crystalline needles with a mild odor. The proposed PEL was 1 ppm as an 8-hour TWA, and NIOSH (Ex. 8–47, Table N1) concurs that this limit is appropriate. The final rule establishes an 8-hour TWA PEL of 1 ppm for phthalic anhydride.

The primary exposure hazards associated with phthalic anhydride are severe skin, eye, and respiratory irritation. The substance can also produce skin and, perhaps, pulmonary sensitization (Patty 1963i, as cited in ACGIH 1986/Ex. 1–3, p. 487). Baader (1955/Ex. 1–1139) has reported irritant effects in animals exposed to 30 mg/m^3 (approximately 5 ppm) phthalic anhydride in air.

In studies of workers exposed to phthalic anhydride, symptoms of respiratory tract injury as well as bronchitis, eye irritation, and nasal bleeding have been reported. Precise exposure concentrations were not detectable by the analytic method being used, which had a limit of detection of 25 mg/m^3 (i.e., of 4 ppm or lower) (Baader 1955/Ex. 1–1139; Menschick 1955/Ex. 1–1091). Other industrial acid anhydrides (e.g., tetrachlorphthalic anhydride and maleic anhydride) are considered more irritating than phthalic

anhydride (ACGIH 1986/Ex. 1-3, p. 489). Only NIOSH commented on this substance.

OSHA is establishing an 8-hour TWA limit of 1 ppm for phthalic anhydride in the final rule. The Agency concludes that this 1-ppm limit will reduce the significant risk of respiratory irritation and skin and pulmonary sensitization, all of which constitute material impairments of health that are associated with exposure to levels above the new PEL.

PROPARGYL ALCOHOL

CAS: 107-19-7; Chemical Formula: $HC{=}CCH_2OH$
H.S. No. 1335

Previously, OSHA had no limit for propargyl alcohol. The ACGIH has established an 8-hour TWA of 1 ppm, with a skin notation, for this straw-colored liquid, which smells like geraniums. The proposed PEL was an 8-hour TWA of 1 ppm, with a skin notation. NIOSH (Ex. 8-47, Table N1) concurs with this limit, which is established in the final rule.

In rats, guinea pigs, and mice, the oral LD_{50}s are 70, 60, and 50 mg/kg, respectively; the 2-hour inhalation LC_{50} in both the rat and mouse is reported to be about 850 ppm (NIOSH 1977i/Ex. 1-1182). The dermal LD_{50} in rabbits is 88 mg/kg (RTECS).

Propargyl alcohol is a primary skin irritant, but it is not a skin sensitizer (Antara Chemicals 1952, as cited in ACGIH 1986/Ex. 1-3, p. 496). The toxicity of propargyl alcohol is estimated to be equal to that of allyl alcohol (oral LD_{50} in rats of 64 mg/kg) (NIOSH 1977i/Ex. 1182). The ACGIH limit is based on the structural and toxicological similarity of propargyl alcohol to allyl alcohol (ACGIH 1986/Ex. 1-3, p. 496). Grace Ziem, an occupational physician, commented (Ex. 46) that the ACGIH *Documentation* (1986/Ex. 1-3) for propargyl alcohol neglects to mention this substance's ability to cause "degenerative changes in liver and kidneys in [an] 89-day rat study" that is cited by Rowe and McCollister (1982) in (*Patty's Industrial Hygiene and Toxicology* 1982, Vol. 2C, p. 4673) and also overlooks the fact that propargyl alcohol is a "moderate CNS depressant." OSHA agrees with Dr. Ziem that this substance has these effects which, in OSHA's opinion, point to the need for the limits being established in this final rule.

OSHA is establishing in the final rule an 8-hour TWA for propargyl alcohol of 1 ppm, with a skin notation. The Agency concludes that these limits will protect workers against the significant risk of skin and mucous membrane irritation, CNS depression, and liver and kidney damage, all of which constitute material impairments of health that are associated with exposure to this substance at levels above the new limit.

PROPIONIC ACID

CAS: 79-09-4; Chemical Formula: CH_3CH_2COOH
H.S. No. 1336

OSHA previously had no limit for propionic acid. The ACGIH has a TLV-TWA of 10 ppm for this substance; the TLV was set on the basis of analogy with acetic acid (10 ppm 8-hour TLV). The proposed PELs were 10 ppm as an 8-hour TWA and 15 ppm as a 15-minute STEL. NIOSH (Ex. 8-47, Table N1) concurred that these limits are appropriate, and the final rule establishes an 8-hour TWA of 10 ppm but does not include a STEL. Propionic acid is a colorless, oily liquid with a pungent odor.

The primary health effects associated with exposure to propionic acid are skin burns and irritation of the eyes and respiratory system. Smyth, Carpenter, Weil, and co-workers (1962/Ex. 1-441) reported that the oral LD_{50} for rats is 4.3 g/kg; NIOSH (1977i/Ex. 1-1182) stated that the intravenous LD_{50} for mice is 625 mg/kg and the skin absorption LD_{50} for rabbits is 500 mg/kg. Inhalation of the saturated vapor for eight hours caused no fatalities in rats (ACGIH 1986/Ex. 1-3, p. 498).

Acute industrial exposures to propionic acid have been reported to cause mild to moderate skin burns, eye irritation, and, in a single incident, asthmatic cough. No irritation was observed as a consequence of exposures in humans averaging below 0.25 ppm with excursions to 2.1 ppm in an eight-hour period (Dow Chemical Company 1977o, as cited in ACGIH 1986/Ex. 1-3, p. 498).

Two commenters in addition to NIOSH commented on propionic acid. The American Industrial Hygiene Association (AIHA) (Tr. 3-307) urged OSHA to delete the STEL for propionic acid on the ground that the ACGIH has put the STEL for this substance on its List of Intended Changed (ACGIH 1988). Kodak (Ex. 661) agrees with the AIHA on the issue of a STEL, noting that, in Kodak's opinion, the 15-ppm STEL "cannot be justified on either available toxicological data or . . . ½Kodak's¾ own experience." After a review of the evidence for propionic acid's short-term effects, OSHA has determined, in accordance with the STEL policy outlined in Section VI.C.17 of this preamble, that no STEL is necessary for propionic acid. Accordingly, the final rule contains no short-term limit for this substance.

In the final rule, OSHA establishes an 8-hour TWA limit of 10 ppm (8-hour TWA) for propionic acid. The Agency concludes that this limit is required to protect workers against the significant risk of eye and respiratory tract irritation, which are material impairments of health that are associated with exposure to levels above the new PEL.

n-PROPYL ACETATE

CAS: 109-60-4; Chemical Formula: $CH_3COOCH_2CH_2CH_3$
H.S. No. 1338

OSHA previously had an 8-hour TWA limit of 200 ppm for n-propyl acetate. The ACGIH also had a 200-ppm TWA limit but adds a TLV-STEL of 250 ppm. The proposal retained the 8-hour TWA PEL of 200 ppm for n-propyl acetate and added a 15-minute STEL of 250 ppm. NIOSH (Ex. 8-47, Table N1) concurs that these limits are appropriate, and they are established by the final rule. n-Propyl acetate is a pleasant-smelling liquid.

The primary health effects associated with exposure to n-propyl acetate are narcosis and eye and respiratory irritation. The five-hour narcotic concentrations for cats and mice have been reported as 9000 ppm and 6000 ppm, respectively (Flury and Wirth 1933, as cited in ACGIH 1986/Ex. 1-3, p. 500). n-Propyl acetate's narcotic action is 1.3 times that of ethyl acetate; salivation and irritation of cats' eyes occurred at 2600 ppm (Flury and Wirth 1933, as cited in ACGIH 1986/Ex. 1-3, p. 500). A four-hour exposure at 8000 ppm killed four of six rats (Smyth 1964, as cited in ACGIH 1986/Ex. 1-3, p. 500). Only NIOSH commented on n-propyl acetate.

n-Propyl acetate appears to be more toxic than isopropyl acetate or ethyl acetate but less so than n-butyl acetate (ACGIH 1986, p. 500).

In the final rule, OSHA is retaining the 8-hour TWA PEL of 200 ppm for n-propyl acetate and adding a STEL of 250 ppm. The Agency concludes that both of these limits are required to prevent the significant risk of narcosis and eye and respiratory tract irritation, which are material impairments of health that are associated with exposures to levels above the 8-hour TWA limit alone.

PROPYL ALCOHOL

CAS: 71-23-8; Chemical Formula: $CH_3CH_2CH_2OH$
H.S. No. 1339

OSHA had a limit of 200 ppm (8-hour TWA) for n-propyl alcohol. The ACGIH has the same TWA limit but adds a 250-ppm 15-minute STEL and a skin

notation. The proposal retained the 200-ppm 8-hour TWA PEL and added a 15-minute STEL of 250 ppm and a skin notation. NIOSH (Ex. 8-47, Table N1) concurs that these limits are appropriate, and they are established in the final rule. The skin notation, however, is not retained (see the discussion of skin notations in Section VI.C.18 of this preamble). Propyl alcohol is a colorless liquid with an alcohol-like odor.

The primary health effect associated with exposure to propyl alcohol is mild narcosis. Propyl alcohol's toxicity is somewhat greater than that of isopropyl alcohol (Gleason, Gosselin, and Hodge 1963/Ex. 1-1034).

The inhalation LD_{50} for propyl alcohol in rats is reported as 1.9 g/kg (Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1-440). Starrek reported deep narcosis in mice inhaling the vapor at a concentration of 4100 ppm for 240 minutes and of 24,500 ppm for 60 minutes; ataxia appeared in 90 to 120 minutes at 3250 ppm (Starrek 1938/Ex. 1-872). These effects are almost twice as intense as those reported for exposure to the vapor of isopropyl alcohol. The dermal LD_{50} in rabbits is 5040 mg/kg (*Dangerous Properties of Industrial Materials,* 6th ed., Sax, 1984).

Nelson, Enge, Ross, and associates (1943/Ex. 1-66) reported mild eye, nose, and throat irritation in humans exposed at 400 ppm to the vapor of isopropyl alcohol, but no data exist on human sensory response to propyl alcohol vapor. The ACGIH (1986/Ex. 1-3, p. 500) reports that many industrial hygienists consider the vapor of propyl alcohol to be more irritating to the throat than the vapor of the isomer.

One comment, other than NIOSH's, was received on isopropyl alcohol. The Motor Vehicle Manufacturers Association (Ex. 3-902) asked that rulemaking for propyl alcohol be delayed, but did not provide any evidence in support of its position.

In the final rule, OSHA is retaining the 8-hour TWA PEL of 200 ppm and adding a STEL of 250 ppm for propyl alcohol; the skin notation is not included in the final rule because the LD_{50} in rabbits is 5040 mg/kg, well above the level determined by OSHA to require a skin notation (see Section VI.C.18 for a discussion of skin notations). The Agency concludes that these limits will protect workers against the significant risk of narcosis and irritation, both material impairments of health.

PROPYLENE OXIDE

CAS: 75-56-9; Chemical Formula: CH_3CHOCH_2
H.S. No. 1344

Previously, OSHA had an 8-hour TWA limit of 100 ppm for propylene oxide. The ACGIH has a limit of 20 ppm TLV-TWA. The proposed PEL was an 8-hour TWA of 20 ppm, and the final rule establishes this revised limit. Propylene oxide is a colorless, highly flammable, volatile, and ethereal liquid.

The health hazards associated with exposure to this substance are primary, skin, eye, and respiratory irritation, as well as central nervous system depression. The oral LD_{50} values reported for rats and guinea pigs are 930 mg/kg and 690 mg/kg, respectively. In mice, the inhalation LC_{50} has been reported to be at 1740 ppm for 4 hours. Dogs and guinea pigs exposed for 4 hours at 2000 and 4000 ppm, respectively, died (NIOSH 1977i/Ex. 1-1182). Although only some species tolerate daily exposures to 200 ppm, all species tested tolerated 100 ppm without ill effects (Rowe, Hollingsworth, Oyen et al. 1956/Ex. 1-609). Jacobson and associates (1956/Ex. 1-702) considered the toxic effects of propylene oxide to be one-half to one-third as intense as those of ethylene oxide (Jacobson, Hackley, and Feinsilver 1956/Ex. 1-702).

Corneal burns and skin necrosis, as well as respiratory and pulmonary irritation, have been reported in humans as a result of direct contact with the liquid or vapor (Patty 1963h/Ex. 1-857); central nervous system effects include ataxia, incoordination, and general depression.

OSHA received several comments on propylene oxide. Lawrence Birkner, Manager of Safety and Industrial Hygiene for ARCO Petroleum and Chemical Company (Tr. 3-229/3-245), reported that his company has an internal limit for propylene oxide of 20 ppm and that about "98 or 99 percent of . . . [ARCO's] exposures are [presently] below the current ACGIH TLVs" (Tr. 3-243).

Richard E. Sanderson, Director of the Office of Federal Activities for the EPA, commented that the discussion of propylene oxide's health effects in the proposal neglected to mention this substance's carcinogenicity or its ability to cause adverse reproductive effects (Ex. 3-746). NIOSH (Ex. 8-47, Table N6B) agrees with EPA that propylene oxide is a potential occupational carcinogen that warrants a full Section 6(b) rulemaking. NIOSH bases its inclusion on an NTP bioassay in rats and mice that demonstrates "some evidence" of carcinogenicity in rats and "clear evidence" of carcinogenicity in mice (Ex. 8-47). In response to these commenters, OSHA states that the Agency is aware of propylene oxide's serious health effects and is monitoring the literature on this substance closely.

OSHA establishes an 8-hour TWA limit of 20 ppm for propylene oxide in the final rule to protect workers against the significant risk of primary irritation and CNS depression, which constitute material health impairments that are associated with exposure to propylene oxide at levels above the revised PEL. The Agency concludes that this limit will substantially reduce these significant risks.

SILICON TETRAHYDRIDE

CAS: 7803-62-5; Chemical Formula: SiH_4
H.S. No. 1361

OSHA formerly had no limit for silicon tetrahydride. The ACGIH limit of 5 ppm as an 8-hour TWA was established in 1983. The proposed PEL was 5 ppm as an 8-hour TWA, a limit with which NIOSH concurs (Ex. 8-47, Table N1). The final rule establishes an 8-hour PEL for silicon tetrahydride of 5 ppm. Silicon tetrahydride, a colorless gas, is used in the manufacture of semiconductors.

Studies of rats exposed to silicon tetrahydride at levels of 126 ppm for one hour (Matheson Gas Products 1971, as cited in ACGIH 1986/Ex. 1-3, p. 528) and at 1400 ppm for six hours (Union Carbide Corporation 1980, as cited in ACGIH 1986/Ex. 1-3, p. 528) have failed to identify any systemic effects associated with exposure to this chemical. Sax (*Dangerous Properties of Industrial Materials,* 6th ed., 1984) lists the effects of acute exposure to silicon tetrahydride as moderate irritation of the eyes, skin, and mucous membranes.

In addition to NIOSH's comment on silicon tetrahydride, Grace Ziem, an occupational physician, stated that she believed OSHA's reference to Sax (1984) in the proposal's discussion of silicon tetrahydride's irritant effects was incorrect. However, OSHA notes that this notation was correct; Dr. Ziem did not realize that Sax (1984, p. 2394) has an entry for silicon tetrahydride under silane, a synonym.

OSHA is establishing an 8-hour TWA limit of 5 ppm for silicon tetrahydride in the final rule. The basis of this limit is analogy to the toxicity of silicon tetrahydride and other tetrahydrides. The Agency concludes that this limit will protect exposed workers from the significant risk of eye, skin, and upper respiratory tract irritation, which are material health impairments that are associated with exposure to this substance at levels above the new PEL.

SULFURYL FLUORIDE

CAS: 2699-79-8; Chemical Formula: SO_2F_2
H.S. No. 1379

The former OSHA limit for sulfuryl fluoride was 5 ppm as an 8-hour TWA. The ACGIH has a limit of 5 ppm as a TWA and adds a STEL of 10 ppm. The proposal retained the 8-hour TWA PEL of 5 ppm for sulfuryl fluoride and added a STEL of 10 ppm. NIOSH (Ex. 8–47, Table N1) concurred that these limits are appropriate, and they are established in the final rule. Sulfuryl fluoride is a colorless gas with a sulfide odor.

When selecting this limit, the ACGIH took into consideration the fact that, compared with hydrogen fluoride (TLV-TWA ceiling of 3 ppm), only a small portion of the inhaled gas is retained and converted to inorganic fluorides. In extensive animal studies conducted by the Dow Chemical Company (1962 and 1970, as cited in ACGIH 1986/Ex. 1–3, p. 546), sulfuryl fluoride was determined to exhibit one-half to one-third the acute inhalation toxicity of methyl bromide. Acute exposures of animals resulted in tremors that later developed into severe convulsions. Pulmonary edema was seen in laboratory animals after a single severe exposure. Repeated exposures of rats, guinea pigs, and mice to 20 ppm sulfuryl fluoride for seven hours per day produced both kidney and lung injury after six months. Some evidence of fluorosis was observed in the incisors of mice, but not in the teeth of the rats or guinea pigs (Dow Chemical Company 1962 and 1970, as cited in ACGIH 1986/Ex. 1–3, p. 546).

A report by Taxay (1966/Ex. 1–577) that examined an incident of workplace exposure to sulfuryl fluoride noted that abdominal pain, nausea, vomiting, and itching were the major symptoms. On the day following exposure, the serum of the affected worker tested positive for fluoride. No comment, other than NIOSH's, was submitted on this substance.

In the final rule, OSHA is retaining the 8-hour TWA limit of 5 ppm and adding a STEL of 10 ppm for sulfuryl fluoride; these limits are based on this substance's fluorine content. The Agency concludes that these limits will protect workers against the significant risks of kidney and lung injury and of fluorosis, which together constitute material health impairments that are associated with exposure to this substance at levels above the 8-hour TWA limit.

THIONYL CHLORIDE

CAS: 7719–09–7; Chemical Formula: Cl_2OS
H.S. No. 1393

OSHA's former Z tables had no limit for thionyl chloride. The ACGIH has established a ceiling limit of 1 ppm for this substance. The proposed ceiling was 1 ppm, and the final rule establishes this limit. NIOSH (Ex. 8–47, Table N1) concurs. Thionyl chloride is a colorless to pale yellow liquid with a suffocating odor.

Thionyl chloride vapors are skin, eye, and mucous membrane irritants, probably because they form sulfur dioxide and hydrogen chloride on contact with moisture (ACGIH 1986/Ex. 1–3, p. 572). An inhalation of 17.5 ppm proved lethal to cats within 20 minutes (Sax 1979/Ex. 1–866).

The ACGIH's exposure limit for thionyl chloride is based on the exposure limits for the decomposition products (hydrogen chloride and sulfur dioxide) of thionyl chloride when mixed with water. The reaction of one mole of thionyl chloride with water produces two moles of hydrogen chloride and one of sulfur dioxide, so that 1 ppm of thionyl chloride can be shown to produce a total irritant gas concentration of 3 ppm. The exposure limit for hydrogen chloride is 5 ppm as a ceiling limit; for sulfur dioxide, the limit is a TWA of 2 ppm. Thus, "the * * * ceiling limit of 1 ppm for thionyl chloride should prevent the irritant effects of its reaction products" (ACGIH 1986/Ex. 1–3, p. 572). No comments, other than NIOSH's, were received on this substance.

In the final rule, the Agency is establishing a ceiling limit of 1 ppm for thionyl chloride on the basis of analogy to the irritation potential of hydrogen chloride and sulfur dioxide. OSHA concludes that this limit will protect workers from the significant risk of irritation of the eyes, skin, and mucous membranes, which constitutes a material health impairment that is associated with exposure to this substance at levels above the new PEL.

TRIBUTYL PHOSPHATE

CAS: 126–73–8; Chemical Formula: $(C_4H_9)_3PO_4$
H.S. No. 1402

The former OSHA standard for tributyl phosphate was 5 mg/m³ as an 8-hour TWA. The ACGIH has a 2.5-mg/m³ TWA for tributyl phosphate, which is a clear, colorless, odorless liquid. The proposed PEL was an 8-hour TWA of 2.5 mg/m³; NIOSH (Ex. 8–47, Table N1) concurs that this limit is appropriate. The final rule establishes an 8-hour TWA PEL of 2.5 mg/m³ for tributyl phosphate.

Tributyl phosphate's toxicity affects the skin, mucous membranes, lungs, and central nervous system, and this substance is also a cholinesterase inhibitor.

A paper by Smyth and Carpenter (1944/Ex. 1–374) reported that contact with liquid tributyl phosphate caused severe eye injury and skin irritation when tested in rabbits. Chambers and Casida (1967/Ex. 1–305) found that mice injected with 1 g/kg tributyl phosphate intraperitoneally became paralyzed. A study by Vandekar (1957/Ex. 1–498) in which mice were given tributyl phosphate by gavage received that a dose of 80 mg/kg resulted in a one-hour period of anesthesia, and a dose of 100 mg/kg resulted in 8 to 10 minutes of anesthesia, followed by respiratory failure and death. Administered intraperitoneally to rats, tributyl phosphate inhibited cholinesterase activity and stimulated plasma beta-glucuronidase activity (Suzuki, Kikuchi, Kato et al. 1977/Ex. 1–1170). This substance did not exhibit mutagenic activity in bacterial or fruit fly assays (Hanna and Dyer 1975/Ex. 1–485).

Nausea and headache were reported by workers exposed to levels of 15 mg/m³ of tributyl phosphate (Mastromatteo 1964b, as cited in ACGIH 1986/Ex. 1–3, p. 591). No comments, other than NIOSH's, were received on this substance.

In the final rule, OSHA is reducing the 8-hour PEL from 5 mg/m³ to 2.5 mg/m³. OSHA concludes that this limit will protect workers against the significant risk of paralysis, anesthetic effects, and skin or eye irritation, all of which constitute material impairments of health that are associated with exposure to tributyl phosphate at levels above the new PEL.

TRICHLOROACETIC ACID

CAS: 76–03–9; Chemical Formula: CCl_3COOH
H.S. No. 1404

OSHA formerly had no exposure limits for trichloroacetic acid. The ACGIH has an 8-hour TWA of 1 ppm to protect against the corrosive effects of this substance. Trichloroacetic acid is a relatively strong acid that forms deliquescent crystals. The proposed PEL was 1 ppm as an 8-hour TWA, and NIOSH (Ex. 8–47, Table N1) concurs that this limit is appropriate. The final rule established a 1-ppm 8-hour TWA for trichloroacetic acid.

The Dow Chemical Company (1977p, as cited by the ACGIH 1986/Ex. 1–3, p. 592) reported that the oral LD_{50} for trichloroacetic acid in rats is 3.33 g/kg. Studies on mice conducted by NIOSH (1984, as cited in ACGIH 1986/Ex. 1–3, p. 592) established that the oral LD_{50} for this species is 4.97 g/kg, and that a 500-mg/kg dose was fatal when administered intraperitoneally.

Medical reports show mild to moderate skin and eye burns in workers exposed to unspecified levels of

trichloroacetic acid; although corrosive, however, trichloroacetic acid is not readily absorbed by the skin (ACGIH 1986/Ex. 1-3, p. 592). Only NIOSH commented on this substance.

In the final rule, OSHA is establishing an 8-hour TWA limit for trichloroacetic acid of 1 ppm. This limit is based on analogy to the toxicity of 2,2-dichloropropionic acid. The Agency concludes that this limit will protect exposed workers from the significant risk of skin and eye irritation, which are material impairments of health that are associated with exposure to this substance at levels above the new PEL.

TRIMETHYLAMINE

CAS: 75-50-3; Chemical Formula: $(CH_3)_3N$
H.S. No. 1411

OSHA formerly had no exposure limit for trimethylamine. The ACGIH has a 10-ppm limit as an 8-hour TWA and a 15-ppm limit as a 15-minute STEL. The proposed PELs, with which NIOSH (Ex. 8-47, Table N1) concurs, were 10 ppm as an 8-hour TWA and 15 ppm as a 15-minute STEL. These limits are established in the final rule. Trimethylamine has a pungent, fishy odor and is a gas at room temperature.

Few toxicological data are available for trimethylamine. One study reports that the intravenous LD_{50} for this substance is 90 mg/kg in mice (Dechezlepretre, Portet, and Cheymol 1967/Ex. 1-777). The ACGIH established the TLV for trimethylamine on the basis of its chemical similarity to dimethylamine, for which the current TLV-TWA is 10 ppm. Dimethylamine is a central nervous system depressant and causes methemoglobinemia. Only NIOSH commented on trimethylamine.

OSHA is establishing an 8-hour TWA limit of 10 ppm and a STEL of 15 ppm (15 minutes) for trimethylamine. Based on analogy with dimethylamine, the Agency concludes that these limits will protect workers exposed at previously unregulated levels from the significant risk of eye, mucous membrane, and upper respiratory tract irritation, which constitute material impairments of health.

n-VALERALDEHYDE

CAS: 110-62-3; Chemical Formula: $CH_3(CH_2)_3CHO$
H.S. No. 1420

OSHA formerly had no limit for n-valeraldehyde. The ACGIH limit is 50 ppm as an 8-hour TWA for n-valeraldehyde, which is a colorless liquid. The proposed PEL was 50 ppm as an 8-hour TWA, and NIOSH (Ex. 8-47, Table N1) concurs that this limit which is established by the final rule, is appropriate.

n-Valeraldehyde's toxic effects include both skin and eye irritation. Animal studies showed n-valeraldehyde's to be severely irritating when applied to guinea pig skin and to rabbits' eyes (Fassett, as cited in ACGIH 1986/Ex. 1-3, p. 619). The dermal LD_{50} for guinea pigs exceeds 20 ml/kg (Fassett, as cited in ACGIH 1986/Ex. 1-3, p. 619).

A series of studies of the relative acute inhalation toxicity of 13 aliphatic saturated and unsaturated aldehydes in mice, guinea pigs, and rabbits showed that valeraldehyde was relatively nontoxic systemically (Salem and Cullumbine 1960/Ex. 1-360). Only NIOSH commented on this substance.

In the final rule, OSHA is establishing a 50-ppm 8-hour TWA limit for this previously unregulated chemical. The Agency concludes that this limit will protect workrs from the significant risk of severe eye and skin irritation associated with exposure to this substance at levels above the new PEL.

m-XYLENE ALPHA, ALPHA'-DIAMINE

CAS: 1477-55-0; Chemical Formula: $C_6H_4(CH_2NH_2)_2$
H.S. No. 1432

OSHA formerly had no exposure limit for this substance. The ACGIH has established a limit of 0.1 mg/m^3 as a ceiling that should not be exceeded during any part of a working day, and has added a skin notation to indicate that substantial percutaneous absorption can occur through the eyes, mucous membranes, and skin. OSHA proposed a ceiling of 0.1 mg/m^3 for this substance, with a skin notation, and NIOSH (Ex. 8-47, Table N1) concurs. These limits are established by the final rule. m-Xylene alpha, alpha'-diamine (MXDA) is a colorless liquid.

Animal studies have demonstrated that MXDA is strongly irritating to the skin (Haskell Laboratory 1973, Sherwin-Williams Company 1978, both as cited in ACGIH 1986/Ex. 1-3, p. 638). Research at du Pont (Haskell Laboratory 1973, as cited in ACGIH 1986/Ex. 1-3, p. 638) showed that pure MXDA was corrosive when applied to the skin of guinea pigs, and a 50-percent MXDA solution caused severe irritation in these animals. In a separate study (Sherwin-Williams Company 1978, as cited in ACGIH 1986/Ex. 1-3, p. 638), a 10-percent mixture of MXDA caused severe skin irritation and erythema in guinea pigs. Sherwin-Williams (1978, as cited in ACGIH 1986/Ex. 1-3, p. 638) also reported that rats exposed to levels of MXDA ranging from 1.74 to 6.04 mg/liter even for one hour sustained liver, kidney, and lung damage, as determined at necropsy. One study showed mild sensitization when MXDA was applied to guinea pig skin, but this effect was not observed in a second study (Sherwin-Williams Company 1978, as cited in ACGIH 1986/Ex. 1-3, p. 638). NIOSH was the sole commenter on this substance.

OSHA concludes that a ceiling limit of 0.1 mg/m^3 and a skin notation are necessary to protect against the significant risk of skin irritation, percutaneous absorption of MXDA, and potential systemic effects, all of which constititute material impairments of health. The Agency has determined that these limits will substantially reduce this significant risk.

XYLIDINE

CAS: 1300-73-8; Chemical Formula: $(CH_3)_2C_6H_3NH_2$
H.S. No. 1433

OSHA's former Z tables included an exposure limit of 5 ppm as an 8-hour TWA for xylidine, with a skin notation. In 1982, the ACGIH reduced its TLV to 2 ppm as an 8-hour TWA and retained the skin notation. The proposed PEL was 2 ppm as an 8-hour TWA, and the skin notation was retained. NIOSH (Ex. 8-47, Table N1) concurs with these limits, and they are established by the final rule. Xylidine is a pale yellow to brown liquid. Commercial xylidine is a mixture of isomers.

Several studies indicate that the former OSHA PEL for xylidine is insufficient to protect workers against hepatotoxic and other adverse effects. A paper by von Oettingen, Neal, Sievers et al. (1947), as cited in ACGIH 1986/Ex. 1-3, p. 639) reported liver damage in dogs, rats, cats, and mice repeatedly exposed to 45 ppm xylidine for seven hours per day for a period of 20 to 40 weeks; these exposures also caused death in dogs cats, and mice. Treon, Sigmon, Wright et al. (1950/Ex. 1-533) noted cardiac, liver, and kidney damage in animals fatally exposed at the following doses: Cats, 17 ppm; guinea pigs, 50 ppm; and rabbits, 60 ppm; cyanosis was also observed in these animals. Only NIOSH commented on xylidine.

In the final rule, OSHA is reducing the existing 8-hour TWA to 2 ppm and retaining the skin notation for xylidine. The Agency concludes that these limits will protect workers from the significant risk of exposure-related cardiac, kidney, and liver damage, all of which constitute material health impairments.

Conclusions For This Group of Substances

Exposure to the 73 substances included in this category place workers at significant risk of material health impairment and functional incapacity.

The adverse health consequences of exposure to these chemicals include neuropathies, skin and respiratory tract irritation, kidney and liver damage, and gastrointestinal disorders, all of which constitute material health impairments within the meaning of the Act. OSHA concludes, based on the record evidence, that the new or revised limits for these hazardous substances will substantially reduce these significant occupational risks.

13. Substances for Which Limits Are Based on Avoidance of Biochemical/ Metabolic Effects

Introduction

One basis for establishing exposure limits is the ability of many toxic substances to interfere with the normal metabolism or biochemistry of the body. A total of 26 substances for which OSHA is establishing limits fall into this group. Table C13–1 shows these substances, their former, proposed, and final rule PELs, and their CAS and HS numbers. For four of these substances, OSHA is only lowering the 8-hour TWA; for two other substances, the Agency is retaining the 8-hour limit and adding a STEL. In one instance, OSHA is reducing the TWA and adding a ceiling. In one case (terphenyls), OSHA is reducing a ceiling limit, and for 17 substances, new limits are being established. In the case of p-nitrochlorobenzene, OSHA is retaining the former limit of 1 mg/m^3 as an 8-hour TWA.

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Table C13-1. Substances for Which Limits Are Based on Avoidance of Metabolic Effects

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*	Biochemical/ Metabolic Effect
1025 Aniline and homologs	62-53-3	5 ppm TWA, Skin	2 ppm TWA, Skin	2 ppm TWA, Skin	Methemoglobinemia
1058 Calcium cyanamide	156-62-7	—	0.5 mg/m^3 TWA	0.5 mg/m^3 TWA	Antabuse-like effect
1068 Carbofuran	1563-66-2	—	0.1 mg/m^3 TWA	0.1 mg/m^3 TWA	Cholinesterase inhibition
1069 Carbon dioxide	124-38-9	5,000 ppm TWA	5,000 ppm TWA 30,000 ppm STEL	10,000 ppm TWA 30,000 ppm STEL	Hyperventilation
1071 Carbon monoxide	630-08-0	50 ppm TWA	35 ppm TWA 200 ppm Ceiling	35 ppm TWA 200 ppm Ceiling	Carboxyhemoglobinemia
1091 Chlorpyrifos	2921-88-2	—	0.2 mg/m^3 TWA 0.6 mg/m^3 STEL, Skin	0.2 mg/m^3 TWA, Skin	Cholinesterase inhibition
1103 Crufomate	299-86-5	—	5 mg/m^3 TWA 20 mg/m^3 STEL	5 mg/m^3 TWA	Cholinesterase inhibition
1104 Cyanamide	420-04-2	—	2 mg/m^3 TWA	2 mg/m^3 TWA	Antabuse-like effect
1131 Dicrotophos	141-66-2	--	0.25 mg/m^3 TWA Skin	0.25 mg/m^3 TWA, Skin	Cholinesterase inhibition

Table C13-1. Substances for Which Limits Are Based on Avoidance of Metabolic Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*	Biochemical/ Metabolic Effect
1143 Dimethylaniline	121-69-7	5 ppm TWA, Skin	5 ppm TWA, Skin 10 ppm STEL	5 ppm TWA, Skin 10 ppm STEL	Methemoglobinemia
1146 Dioxathion	78-34-2	—	0.2 mg/m^3 TWA, Skin	0.2 mg/m^3 TWA, Skin	Cholinesterase inhibition
1151 Disulfiram (Antabuse)	97-77-8	—	2 mg/m^3 TWA	2 mg/m^3 TWA	Antabuse effects
1160 Ethion	563-12-2	—	0.4 mg/m^3 TWA, Skin	0.4 mg/m^3 TWA, Skin	Cholinesterase inhibition
1173 Fenamiphos	22224-92-6	—	0.1 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA, Skin	Cholinesterase inhibition
1174 Fensulfothion	115-90-2	—	0.1 mg/m^3 TWA	0.1 mg/m^3 TWA	Cholinesterase inhibition
1175 Fenthion	55-38-9	—	0.2 mg/m^3 TWA, Skin	0.2 mg/m^3 TWA, Skin	Cholinesterase inhibition
1245 Methomyl	16752-77-5	—	2.5 mg/m^3 TWA	2.5 mg/m^3 TWA	Cholinesterase inhibition
1280 Monomethylaniline	100-61-8	2 ppm TWA, Skin	0.5 ppm TWA, Skin	0.5 ppm TWA, Skin	Methemoglobinemia

Table C13-1. Substances for Which Limits Are Based on Avoidance of Metabolic Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*	Biochemical/ Metabolic Effect
1288 p-Nitrochloro-benzene	100-00-5	1 mg/m^3 TWA, Skin	1 mg/m^3 TWA, Skin	1 mg/m^3 TWA, Skin	Methemoglobin-emia
1319 Phorate	298-02-2	—	0.05 mg/m^3 TWA 0.2 mg/m^3 STEL, Skin	0.05 mg/m^3 TWA 0.2 mg/m^3 STEL, Skin	Cholinesterase inhibition
1337 Propoxur	114-26-1	—	0.5 mg/m^3 TWA	0.5 mg/m^3 TWA	Cholinesterase inhibition
1349 Ronnel	299-84-3	15 mg/m^3 TWA	10 mg/m^3 TWA	10 mg/m^3 TWA	Cholinesterase inhibition
1380 Sulprofos	35400-43-2	—	1 mg/m^3 TWA	1 mg/m^3 TWA	Cholinesterase inhibition
1384 Terphenyls	26140-60-3	1 ppm Ceiling	0.5 ppm Ceiling	0.5 ppm Ceiling	Mitochondrial changes
1401 m-Toluidine	108-44-1	—	2 ppm TWA, Skin	2 ppm TWA, Skin	Methemoglobinemia
1413 2,4,6-Trinitrotoluene	118-96-7	1.5 mg/m^3 TWA, Skin	0.5 mg/m^3 TWA, Skin	0.5 mg/m^3 TWA, Skin	Methemoglobinemia

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

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Description of the Health Effects

The compounds shown in Table C13–1 are further divided into the following sub-classes, based on their mechanism of action:

- Substances that are cholinesterase inhibitors;
- Substances that interferes with the oxygen-carrying capacity of blood;
- Substances with Antabuse-like effects.

The disruption of metabolic processes by toxic substances, if severe enough, results in potentially dangerous effects on the neurological, cardiovascular, and respiratory systems. The adverse health consequences caused by exposure to chemicals having cholinesterase inhibition effects range from wheezing, nausea, vomiting, and confusion to respiratory failure, coma, and death. If exposure has localized rather than systemic effects, the signs and symptoms of cholinesterase inhibition can include sweating, blurred vision, and constriction of the bronchial tubes. Substances that interfere with the ability of the blood to carry oxygen cause a broad range of symptoms, including fainting, loss of consciousness, rapid heartbeat, headache, nausea, coma, and death. Carbon monoxide (CO) is the best-known substance in this category of chemicals, and exposure to CO is common throughout industry.

The Antabuse-like effects associated with exposure to three chemicals—disulfiram, cyanamide, and calcium cyanamide—include facial flushing, nausea, and a racing heartbeat. However, these effects are manifested only if the exposed individual has ingested alcohol. The three chemicals in this subgroup cause this effect by inhibiting aldehyde dehydrogenase activity, which is involved in the biotransformation of alcohol.

For chemicals that cause systemic toxicity in animals and/or humans, the grossly observable signs and symptoms of intoxication are usually secondary to the interaction of the chemical with a molecular target. In other words, the chemical interacts with (binds with or modifies) an endogenous molecular constituent (protein, nucleic acid, lipid, etc.) in the target tissue(s). The result of the interaction is ordinarily a modification or elimination of the normal function of the specific molecular constituent which, if sufficiently severe, may lead to secondary effects within the affected cells and/or tissues. It is possible for a number of molecules to be affected by the toxic chemical without there being any overt manifestation of toxicity. In other words, there is an apparent no-effect level governing the overt manifestation of toxicity, although there are usually metabolic effects at levels below those that cause overt effects.

For chemicals for which the molecular target is known and for which methods are available to detect the altered molecular target, it is possible to use the measure of altered biochemical function as a sensitive indicator of exposure to the chemicals at levels below those that cause grossly observable signs and symptoms of poisoning. For other classes of chemicals, studies in animals and/or humans have shed light on the biochemical basis of their toxicity. For some of these classes of chemicals, it is possible to base limits of human exposure on biochemical, metabolic, or pharmacologic indicators of their interaction with molecular targets rather than on grossly visible signs and symptoms of adverse systemic effects.

Substances that are cholinesterase inhibitors. A number of organophosphate and carbamate insecticides produce acute toxicity in humans through inhibition of acetylcholinesterase at cholinergic synapses in the central and peripheral nervous systems. There are 14 substances in this group. This inhibition causes an accumulation of acetylcholine at the effector sites and elicits signs and symptoms consistent with excessive cholinergic activity. These include bronchoconstriction; increased bronchial secretions, salivation, and lacrimation; nausea; vomiting; cramps; constriction of the pupils; muscular weakness; and cardiac irregularities. If sufficiently severe, acetylcholinesterase inhibition may cause coma, irreversible CNS damage, and death.

The mechanisms by which carbamates and organophosphates inhibit acetyl-cholinesterase differ. In general, carbamates form a noncovalently bound complex with the enzyme, while most organophosphates bind covalently with the enzyme. The net result, inactivation of the enzyme, is similar for both groups. In either case, the inhibition is usually reversible. The carbamate-cholinesterase complex dissociates to regenerate the active enzyme, while cholinesterase inactivated by organophosphates is replaced by the *de novo* synthesis of active enzyme. Therefore, unless the inhibition is sufficiently severe to cause brain damage or death, the manifestations of acute toxicity are reversible, and poisoned individuals recover without sequelae. A significant proportion of endogenous cholinesterase activity may be inhibited before the overt manifestations of intoxication appear. The fraction of total cholinesterase activity that can be inhibited without there being signs and symptoms of toxicity varies from individual to individual and also appears to depend on the intensity and duration of exposure. The lack of warning signs at low levels of exposure increases the need to set exposure limits at levels that will protect those individuals who do not readily manifest the symptoms and signs of toxicity from experiencing the subclinical effects of exposure.

Substances that interfere with the oxygen-carrying capacity of the blood. Nine compounds in this section produce their immediate toxicity in humans by altering the ability of hemoglobin in the red blood cells to bind, transport, and release oxygen. Perhaps the best studied of these is carbon monoxide. Carbon monoxide binds to hemoglobin with a greater affinity than does oxygen. It also alters the dissociation characteristics for the oxygen-hemoglobin complex. The overall effect is to reduce the oxygen-carrying capacity of the blood. Also included in this overall category of compounds is a group of aromatic amines and nitro compounds that react with hemoglobin in the blood to reduce it to methemoglobin. Methemoglobin will not bind with oxygen and therefore is not an effective carrier of oxygen.

Because these compounds reduce the ability of the blood to transport oxygen, the overt signs and symptoms of acute toxicity are those of tissue anoxia, i.e., neurobehavioral disturbances, dizziness, cardiac irregularities, cyanosis, unconsciousness, and death. The severity of the symptoms is a function of the degree to which the oxygen-carrying capacity of the blood has been depleted and of the state of the exposed individual's health. In the case of carbon monoxide, individuals with pre-existing cardiovascular disease or healthy individuals engaged in physical labor may be placed at increased risk when more than 5 percent of their hemoglobin is bound to carbon monoxide.

In the cases of both carbon monoxide and the methemoglobin-forming compounds, the primary effect (i.e., formation of carboxyhemoglobin or methemoglobin) is reversible. In the absence of additional carbon monoxide exposure, carboxyhemoglobin dissociates to carbon monoxide and fully functional hemoglobin. Methemoglobin can be reoxidized to hemoglobin by endogenous mechanisms, but the major recovery mechanism is via the synthesis of new hemoglobin.

Substances with Antabuse-like effects. The ingestion of alcoholic beverages following exposure to

disulfiram, cyanamide, or calcium cyanamide results in a characteristic syndrome consisting of flushing of the face, nausea, vomiting, hypotension, and increased heart rate. If exposure is particularly severe, the reaction may trigger convulsions, cardiac arrhythmias, or heart attacks and has in some cases caused death. In the vast majority of less severe cases, the reaction is fully reversible, although the symptoms are temporarily completely disabling. Disulfiram (Antabuse) is used therapeutically in the treatment of chronic alcoholism; employees who are currently being treated with disulfiram for alcoholism are therefore at particularly high risk if they are also occupationally exposed to these substances that cause Antabuse-like effects. These compounds do not cause any signs or symptoms of toxicity in the absence of alcohol ingestion unless exposure levels are far above those that trigger the alcohol-induced response.

Dose-Response Relationships and Biochemical/Metabolic Effects

Substances that are cholinesterase inhibitors. Typically, the cholinesterase inhibition potential of a compound is assessed by measuring plasma cholinesterase activity in the treated organism. Data from experiments in animals and limited data from human clinical trials indicate that the percentage of basal plasma cholinesterase activity decreases with increasing dose and that the dose-response curve is S-shaped. Because there is inter-individual variation in this relationship, the dose-response curve for a population exposed to a cholinesterase inhibitor would be expected to be much shallower in slope and to have longer tails than the dose-response curve for any single individual.

The relationship between the dose-response curve for plasma cholinesterase inhibition and the dose-response curves for more direct indicators of clinical intoxication, such as acetylcholinesterase activity in the CNS or the actual appearance of signs of intoxication, is not known. Evidence suggests that there is considerable inter-individual variability in these relationships. Some individuals may be free of the symptoms and signs of intoxication when their plasma cholinesterase levels have been inhibited by as much as 90 percent, while others may experience symptoms after only a small decrease in plasma cholinesterase activity. Because of this variability, any exposure limit should be set with this individual variability in mind.

Substances that interfere with oxygen transport. Both carboxyhemoglobin and methemoglobin formation exhibit a classical sigmoidal dose-response relationship in relation to exposure to carbon monoxide or methemoglobin-forming compounds. The loss in the oxygen-carrying capacity of the blood is a function of the intensity and duration of exposure. As stated above, the majority of healthy individuals can tolerate some reduction in the oxygen-carrying capacity of their blood without experiencing symptoms of overt toxicity. However, there is great inter-individual variability in the degree of decreased oxygen-carrying capacity that can be tolerated without apparent ill effect. Individuals with pre-existing anemia or with high carboxyhemoglobin levels as a result of other environmental exposures (e.g., smoking) may already be at or above the level at which they will display the signs or experience the symptoms of tissue anoxia. For these individuals, even a small incremental decrease in the oxygen-carrying capacity of the blood can have serious consequences.

Substances causing Antabuse-like effects. The dose-response characteristics of disulfiram, cyanamide, and calcium cyanamide follow the usual S-shaped curve. The final rule's limits for the substances in this group have been set at levels below those associated with the Antabuse effect in workers ingesting alcohol either during or after work.

The following paragraphs describe the record evidence and the Agency's findings with respect to the substances that cause metabolic disturbances. The discussions below also illustrate the risk of material health impairment associated with exposure to these substances.

ANILINE (AND HOMOLOGS)

CAS: 62-53-3; Chemical Formula: $C_6H_5NH_2$
H.S. No. 1025

The former OSHA 8-hour TWA permissible exposure limit for aniline was 5 ppm, with a skin notation. The ACGIH-recommended 8-hour TLV is a 2-ppm TWA, with a skin notation. The proposed PEL was 2 ppm as an 8-hour TWA, and this limit is established in the final rule. The skin notation is retained. Aniline, when first distilled, is an oily, colorless liquid that darkens on exposure to air.

Occupational aniline poisoning was a relatively common occurrence in earlier years (ACGIH 1986/Ex. 1-3, p. 30). The early limits for aniline were set to guard against acute toxicity manifested as cyanosis (Henderson and Haggard 1943i, as cited in ACGIH 1986/Ex. 1-3, p. 30). Cirrhosis and chronic CNS effects were also reported (Holstein 1955/Ex. 1-913; von Oettingen 1941/Ex. 1-874). Skin absorption occurs when aniline vapor contacts the skin (Dutkiewicz 1962, as cited in ACGIH 1986/Ex. 1-3, p. 30); the dermal LD_{50} in rabbits is 820 mg/kg (*Dangerous Properties of Industrial Materials*, 7th ed., Sax and Lewis 1989, p. 262).

Early studies suggested that less than full-shift exposures of 7 to 53 ppm of aniline vapor caused mild symptoms, while one-hour inhalation exposures to concentrations in the range of 100 to 160 ppm caused severe effects (Henderson and Haggard 1943i, as cited in ACGIH 1986/Ex. 1-3, p. 30). Later studies in several species of animals found no effects, other than a slight increase in methemoglobin in the blood of rats, after the animals had been exposed to aniline concentrations of 5 ppm for six months (Oberst, Hackley, and Comstock 1956/Ex. 1-685). An early NCI aniline hydrochloride cancer bioassay in Fischer 344 rats and B6C3F1 mice demonstrated carcinogenic effects, primarily in the spleens of rats, but multiple organ sites were also involved in rats fed 0.6 percent or 0.3 percent aniline hydrochloride for 103 weeks (NCI 1978a/Ex. 1-1118).

NIOSH (Ex. 8-47, Table N6A) testified (Tr. III-96/97) that aniline and its analogs are carcinogens; NIOSH concurred that the limit established by OSHA for aniline and its homologues is appropriate. However, NIOSH is of the opinion that OSHA should designate substances suspected of having carcinogenic potential as carcinogens. This issue is discussed in Section V of the preamble. OSHA received no other comments on aniline and its homologues.

OSHA has concluded that the former limit of 5 ppm is not sufficiently protective, since systemic effects have been observed in humans exposed to levels as low as 7 ppm and in animals at levels as low as 5 ppm. Accordingly, OSHA is establishing an 8-hour TWA of 2 ppm for aniline and retaining the skin notation, which will protect against percutaneous absorption. The Agency has determined that this limit will substantially reduce the significant risk of methemoglobinemia, which constitutes a material impairment of health, seen in exposed animals at the former level. The Agency intends to continue to monitor the evidence on the carcinogenicity of aniline in the future to determine whether other action is appropriate.

CALCIUM CYANAMIDE

CAS: 156-62-7; Chemical Formula: CaNC-N
H.S. No. 1058

OSHA formerly had no limit for calcium cyanamide. The ACGIH has a TLV-TWA of 0.5 mg/m^3 for this crystalline gray material. The proposed PEL was an 8-hour TWA of 0.5 mg/m^3, and NIOSH (Ex. 8-47, Table N1) concurs with this limit, which is established in the final rule.

Data regarding the acute toxicity of calcium cyanamide are sparse. The oral LD_{50} in rabbits is 1400 mg/kg, and that for rats is 1000 mg/kg (Spencer 1973, as cited in ACGIH 1986/Ex. 1-3, p. 91).

Skin and eye irritation have been reported in rats and rabbits, with significant irritation occurring when 100 mg of calcium cyanamide is placed directly into the eyes of rabbits (Martin 1975, as cited in ACGIH 1986/Ex. 1-3, p. 91). Severe skin irritation developed in rabbits when a paste of this substance was applied to the shaved abdominal skin for 24 hours (Martin 1975, as cited in ACGIH 1986/Ex. 1-3, p. 91). Two of five animals died when the dose was 10 g/kg, but all survived a dose of 5 g/kg.

Most cases of industrial calcium cyanamide poisoning involve primary skin irritation or sensitizing dermatitis. Skin irritation develops in the form of an erythematous rash over the surfaces of the body that are exposed to the substance of those body surfaces irritated by clothing or perspiration. Some individuals develop a macular rash on exposure, and this may progress to the weeping stage. In addition, exposed workers may develop temporary vasomotor disturbances of the upper body, with susceptibility increasing with alcohol intake (Fassett 1963d, as cited in ACGIH 1986/Ex. 1-3, p. 91). Calcium cyanamide is used medically for its Antabuse-like effect, and the maintenance dose in adults is between 50 and 100 mg/day (Hald, Jacobsen, and Larson 1952/Ex. 1-905). No comments, other than NIOSH's, were received on this substance.

In the final rule, OSHA is establishing an 8-hour TWA PEL of 0.5 mg/m^3 for calcium cyanamide. The Agency concludes that this limit will substantially reduce the significant risks of material health impairment in the form of eye and skin irritation, sensitizing dermatitis, and the occurrence of Antabuse-like effects, which were possible at the levels of exposure formerly permitted by the absence of an OSHA limit.

CARBOFURAN

CAS: 1563-66-2; Chemical Formula: $C_{12}H_{15}NO_3$
H.S. No. 1068

Previously, OSHA had no limit for carbofuran. The ACGIH has a TLV-TWA of 0.1 mg/m^3 for this white crystalline solid. The proposed PEL for carbofuran was 0.1 mg/m^3 as an 8-hour TWA, and NIOSH concurred with this limit (Ex. 8-47, Table N1), which is established in the final rule.

Tobin (1970/Ex. 1-935) reports that the LC_{50} of 50-percent wettable carbofuran powder is 108 mg/m^3 for male and 133 mg/m^3 for female rats; a respiratory LC_{50} of 53 mg/m^3 for guinea pigs exposed to the 75-percent wettable powder is also reported (Tobin 1970/Ex. 1-935). Rhesus monkeys did not display cholinesterase depression at levels equivalent to 0.56 mg/m^3 of 75-percent wettable powder (Tobin 1970/Ex. 1-935). Chronic feeding studies in the rat have shown no effects at 25 ppm; in the dog, the no-effect level was 20 ppm (Gaines, unpublished data, as cited in ACGIH 1986/Ex. 1-3, p. 100). Inhibition of plasma, erythrocyte, and brain cholinesterase levels was evident at levels of 50 ppm in the diet (Tobin 1970/Ex. 1-935). Six-hour exposures at levels of 0.86 mg/m^3 caused significant cholinesterase inhibition in animals (Tobin 1970/Ex. 1-935).

Workers exposed at concentrations approaching 0.1 mg/m^3 have not shown any adverse effects (Tobin, personal communication to ACGIH TLV Committee, as cited in ACGIH 1986/Ex. 1-3, p. 100). No comments, other than NIOSH's, were received on carbofuran.

In the final rule, OSHA is establishing a permissible exposure limit of 0.1 mg/m^3 as an 8-hour TWA for this substance to protect employees from the significant risk of cholinesterase inhibition potentially associated with exposure to this previously unregulated substance. The Agency concludes that this limit will substantially reduce this significant occupational risk of material impairment of health.

CARBON DIOXIDE

CAS: 124-38-9; Chemical Formula: CO_2
H.S. No. 1069

OSHA's former limit for carbon dioxide was 5000 ppm as an 8-hour TWA. The ACGIH has a 5000-ppm TLV-TWA with a 30,000-ppm TLV-STEL, and these were the limits proposed. NIOSH has a TWA REL of 10,000 ppm with a 10-minute 30,000-ppm ceiling limit; however, NIOSH (Ex. 8-47, Table N1) concurred that the proposed limits were appropriate. After carefully reviewing the record evidence submitted in response to OSHA's proposal for carbon dioxide, the Agency has determined that exposure limits of 10,000 ppm (8-hour TWA) and 30,000 ppm (15-minute STEL) are appropriate. Carbon dioxide is a colorless, odorless, noncombustible gas.

Both the ACGIH (1986/Ex. 1-3) and NIOSH (1976a, as cited in ACGIH 1986/Ex. 1-3, p. 102) cite studies indicating that continuous exposure to between 1.5 and 3 percent carbon dioxide (15,000 to 30,000 ppm) results in few, if any, adverse effects. However, electrolyte imbalances and other metabolic changes have been associated with prolonged exposure to 10,000 to 20,000 ppm CO_2 (Schulte 1964/Ex. 1-366; Gray 1950, as cited in ACGIH 1986/Ex. 1-3, p. 102). Increases in the rate of respiration have been observed among resting subjects exposed to 39,500 ppm for periods shorter than a day and among exercising subjects exposed to airborne concentrations below 30,000 for the same period (Sinclair et al. 1969, as cited in ACGIH 1986/Ex. 1-3, p. 102).

OSHA received comments on carbon dioxide from the American Iron and Steel Institute (AISI) (Ex. 1-1123; Tr. p. 11-24) and the Corn Refiners Association (Ex. 177), among others; both organizations listed CO_2 as a substance affecting their respective industries but did not provide further information. OSHA also received comments from the Beer Institute (Exs. 49 and 142; Tr. 8/9/88, p. 9-26) and from the Anheuser-Busch Company (Ex. 199).

The Beer Institute (Exs. 49 and 142; Tr. 8/9/88, p. 9-26) and the Brewing Industry Safety Advisory Committee submitted comments to OSHA on carbon dioxide. This industry's position is that there is no health risk to employees exposed to CO_2, even at levels between 15,000 and 20,000 ppm for an 8-hour period (Ex. 49, p. 2). In support of this position, the Beer Institute testified that the 8-hour TWA limit of 5000 ppm is "unnecessarily low and restrictive" (Ex. 49; Tr. 8/9/88, p. 9-27). The Institute also submitted a study by Riley and Bromberger-Barnea (1979/Ex. 49B) on the CO_2 exposure of brewery workers. This study monitored the full-shift exposures of these workers to CO_2 and determined that they average 1.08 percent CO_2 (10,800 ppm).

The Beer Institute testified that the beer industry "is unique relative to carbon dioxide exposure and control. * * * no other industry faces the same engineering difficulties for controlling ambient carbon dioxide as the brewing industry" (Tr. 8/9/88, p. 9-26). These commenters identified two situations where exposure to CO_2 might be a problem for cellar workers (Tr. 8/9/88, p. 9-27). The first situation occurs when excessive CO_2 builds up in the large fermentation tanks used in the beer-making process and blows an escape

valve, and the second exposure situation arises when workers must enter the fermentation tanks to flush out the sludge remaining after the tank has been drained. OSHA finds that these situations involve either upset conditions (safety valve blowout) or maintenance (tank cleaning); both of these operations are considered nonroutine, and respiratory protection may be used to protect employees when these situations arise. OSHA's analysis of the technological feasibility of achieving the final rule's limits in facilities in the beer industry is presented in Section VII of this preamble.

In response to these commenters, OSHA notes that the limit to which these industry spokesmen are objecting is the CO_2 limit that has been in force since the Agency was founded in 1971. Neither the Beer Institute nor the Brewing Industry Safety Advisory Committee objects to the only change OSHA proposed in this rulemaking (i.e., the addition of a 30,000-ppm STEL for CO_2). According to Gary Nateman, Vice President of the Beer Institute (Tr. 8/9/88, pp. 9–30):

> It is appropriate in our view for OSHA to adopt the 3-percent [30,000-ppm] short-term exposure limit for carbon dioxide. There is a scientific basis for this limit and in terms of real health benefit, this is the most meaningful approach (Tr. 8/9/88, pp. 9–31).

The basis for the beer industry's objection to the retention of OSHA's 5000-ppm limit is that NIOSH recommended a higher 8-hour TWA limit of 10,000 ppm in its 1976 criteria document for carbon dioxide (NIOSH 1976a, as cited in ACGIH 1986/Ex. 1–3, p. 102).

After reviewing this evidence, OSHA is persuaded that a 10,000-ppm 8-hour TWA limit, combined with a 30,000-ppm STEL, will protect employees from the adverse effects associated with excessive exposures to CO_2. OSHA bases this conclusion on the fact that, while the evidence has not shown that prolonged exposures to 10,000-ppm are harmful, acute exposures to CO_2 concentrations in excess of 30,000-ppm have been demonstrated to cause changes in respiration rates in humans.

In the final rule, OSHA is establishing a 10,000-ppm PEL as an 8-hour TWA and a 30,000-ppm STEL to protect employees from experiencing the metabolic and respiratory changes, which constitute material health impairments, that are associated with elevated short-term CO_2 exposures. The Agency concludes that adding this limit will substantially reduce the risk associated with the high short-term exposures to CO_2 that are possible in the absence of a STEL. The former 8-hour TWA of 5000 ppm is retained.

CARBON MONOXIDE

CAS: 630–08–0; Chemical Formula: CO
H.S. No. 1071

OSHA's former limit for carbon monoxide was 50 ppm as an 8-hour TWA. The ACGIH has a TLV–TWA of 50 ppm with a TLV–STEL of 400 ppm. NIOSH (1973d/Ex. 1–237) recommends an 8-hour TWA limit of 35 ppm with a 200-ppm ceiling. The proposed PEL and ceiling were 35 ppm and 200 ppm, respectively; NIOSH (Ex. 8–47, Table N1) concurs that these limits are appropriate, and they are established in the final rule. Carbon monoxide is a flammable, colorless, practically odorless gas.

Carbon monoxide readily combines with hemoglobin to form carboxyhemoglobin (COHb). Excessive accumulations of COHb cause hypoxic stress in healthy individuals as a result of the reduced oxygen-carrying capacity of the blood. In patients with cardiovascular disease, such stress can further impair cardiovascular function. The ACGIH (1986/Ex. 1–3) cites a number of studies showing that exposure to 50 ppm TWA carbon monoxide generally results in COHb levels of 8 to 10 percent, and that such levels are not generally associated with overt signs or symptoms of health impairment in healthy individuals under nonstressful conditions. However, the ACGIH comments that a TLV of 25 ppm, which results in COHb levels of 4 percent or less, may be necessary to protect workers with cardiovascular disease, because this condition places workers at higher risk of serious cardiovascular injury (ACGIH 1986/Ex. 1–3, p. 106). The NIOSH recommendation of 35 ppm TWA is also aimed at protecting workers with chronic heart disease; NIOSH believes that such workers should not be allowed to have carboxyhemoglobin levels that approach 5 percent. The rationale for the ACGIH's recommendation of a 400-ppm TLV–STEL for CO is not entirely clear, but may be based on a study by Schulte (1964/Ex. 1–366), which stated that exposure to 10 ppm carbon monoxide for four hours is excessive.

Several commenters (Exs. 133, 188, 3–675, 3–673, L3–1330, 3–902, 3–660, 3–349, 3–1123, and 129) submitted comments on the Agency's proposed limits for carbon monoxide. Some of these commenters (Exs. 3–675 and 3–673) were concerned that the revised limits would have serious economic impacts on their industries (electric utilities, steel, and nonferrous foundries). However, OSHA has determined that it is feasible for facilities in these sectors to comply with the proposed CO limits (see Section VII of the preamble).

Many rulemaking participants questioned the health basis for lowering the former CO limit of 50 ppm as an 8-hour TWA to 35 ppm and supplementing this limit with a 200-ppm STEL (Exs. 133A, 188, 3–660, 3–349, 3–1123, and 129). These commenters pointed out that the discussion of CO's health effects in the preamble to the proposal (53 FR 21171) stated that the carboxyhemoglobin levels associated with CO exposures of 50 ppm "are not associated with toxic effects in healthy individuals." According to the American Iron and Steel Institute (Ex. 3–1123), whose remarks were typical of the views of these commenters:

> The proposed PEL should not be adopted because there is not adequate evidence that exposure to carbon monoxide at levels of 50 ppm TWA poses a significant risk to workers with heart or pulmonary disease * * * (Ex. 3–1123, p. 23).

H.K. Thompson, Corporate Industrial Hygiene Manager of Caterpillar, Inc. (Ex. 3–349), stated:

> PELs or TLVs are not set to protect individuals with chronic heart disease. In our industry we transfer people with disabilities to jobs where the risk for them is minimal (Ex. 3–349, p. 3).

In response to these commenters, OSHA quotes the ACGIH (1986/Ex. 1–3):

> Each molecule of CO combining with hemoglobin reduces the oxygen carrying capacity of the blood and exerts a finite stress on man. Thus, it may be reasoned that there is *no dose of CO* that is not without an effect on the body. Whether that effect is physiologic or harmful *depends upon the dose of CO and the state of health of the exposed individual.* The body compensates for this hypoxic stress by increasing cardiac output and blood flow to specific organs, such as the brain or the heart. When this ability to compensate is overpowered or is limited by disease, tissue injury results [emphasis added].
>
> Exposure to CO sufficient to produce COHb saturations in the 3–5% range impairs cardiovascular function in patients with cardiovascular disease and in normal subjects * * *. The primary effect of exposure to low concentrations of CO on workmen results from the hypoxic stress secondary to the reduction in the oxygen-carrying capacity of blood * * *. Workmen with significant disease, both detected and undetected, may not be able to compensate adequately and are at risk of serious injury. For such workers, a TLV of 25 ppm * * * might be necessary. Even such a concentration might be detrimental to the health of some workers who might have far advanced cardiovascular disease * * *. It would appear to the Committee that the time-

weighted TLV of 50 ppm for carbon monoxide might also be too high under conditions of heavy labor, high temperatures, or at high elevations (ACGIH 1986/Ex. 1–3, p. 106).

Thus, the ACGIH also regards a lower limit for CO as necessary to protect workers with cardiovascular or pulmonary disease or those working under stressful conditions.

NIOSH (Ex. 150, Comments on Carbon Monoxide) submitted a substantial amount of posthearing evidence demonstrating the significant risk associated with CO exposure, particularly with respect to coronary heart disease. The following studies are particularly relevant to this issue. Atkins and Baker (1985, as cited in NIOSH/Ex. 150) report the case of two workers with preexisting coronary artery disease who died after exposure to CO at work. A study of firefighters in Los Angeles (Barnard and Weber 1979, as cited in NIOSH/Ex. 150) suggests that CO exposure during firefighting may be responsible for the high incidence of heart disease in firefighters; peak exposures during fire fighting were as high as 3000 ppm CO, with 40 percent of peak values in the 100- to 500-ppm CO range. A prevalence study by Hernberg et al. (1976, as cited in NIOSH/Ex. 150) reports a clear dose-response relationship between CO exposure and angina pectoris in foundry workers. Stern and co-workers (1981, as cited in NIOSH/Ex. 150) suggest that the slight overall excess of deaths in motor vehicle examiners caused by cardiovascular disease is attributable to chronic exposure to low levels of CO (10 to 24 ppm as an 8-hour TWA). The AFL-CIO's posthearing comment (Ex. 194) agrees that the comments submitted by NIOSH are persuasive evidence of the need to reduce the 8-hour TWA for CO.

NIOSH also submitted recent data on carbon monoxide's reproductive effects and on its neurotoxic/behavioral effects. Based on a review of all of these studies, NIOSH concludes that "[t]he new data suggest a reevaluation of the REL and strongly support the inference that there is a significant risk of material impairment to health at the * * * [former] 50-ppm PEL which will be reduced by the proposed 35-ppm PEL" (Ex. 150, Comments on Carbon Monoxide).

OSHA notes that cardiovascular disease (detected or undetected) and pulmonary impairment are widespread in the general population in this country, and that workers constitute a significant part of this general population. In addition, workers regularly encounter complex and stressful situations at work, including heat stress, jobs demanding heavy exertion, and tasks requiring both judgment and motor coordination.

The AISI (Ex. 129) submitted an article (Redmond, Emes, Mazumdar et al. 1977, "Mortality of Steelworkers Employed in Hot Jobs") to OSHA which, in the opinion of the AISI, demonstrates that steelworkers who are exposed to high heat (and ostensibly also to CO) do not have coronary heart disease. Based on this article, the AISI asks that the steel industry be exempted from the revised PEL for CO. OSHA finds the article submitted by the AISI unconvincing on the point at issue; the article is not primarily concerned with CO exposures but with heat stress and, further, does not include a large enough sample to demonstrate the absence of the effect. Moreover, OSHA is establishing limits that will apply to all of general industry; the Agency does not customarily set standards based on the particular conditions prevailing in a specific operation or industry.

However, some evidence has been submitted by the AISI (Ex. 129) to the effect that the ceiling limit cannot regularly be achieved with engineering and work practice controls in specific operations in SIC 33. These operations are: blast furnace operations, vessel blowing at basic oxygen furnaces, and sinter plant operations. There is no evidence to the contrary in the record. For these operations, OSHA will therefore permit more flexibility in the use of respirators. The burden of proof will not be on employers to demonstrate that compliance with the ceiling by means of engineering and work practice controls is infeasible in any compliance action involving these operations in SIC 33.

There may be a few other operations that fall into this same category; however, the record is unclear on this point. Based on an appropriate showing pursuant to the OSH Act, OSHA will favorably consider requests for variances for specific operations in SIC 33 involving methods of compliance for the ceiling limit. Of course, all requests for variances or any other matters will be considered based on their merits.

OSHA thus finds that the reduced 8-hour TWA of 35 ppm for carbon monoxide is needed to reduce the significant risk of serious injury that has repeatedly been demonstrated to result from overexposure to CO in a host of occupational environments. The Agency concludes that a ceiling of 200 ppm is necessary to ensure that peak CO exposures do not reach levels demonstrated to be hazardous and that overall full-shift exposures remain under good control. In the absence of a ceiling, concentrations approaching the Immediately-Dangerous-to-Life-or-Health (IDLH) level of 1500 ppm could occur.

In the final rule, OSHA is establishing an 8-hour TWA of 35 ppm and a ceiling of 200 ppm as the PELs for carbon monoxide to ensure that employee COHb levels are maintained at or below 5 percent, in order to protect those workers at greater risk because of cardiovascular or pulmonary impairment. In addition, these revised limits will protect healthy workers who must work in environments involving exertion, heat stress, or other strenuous conditions. The Agency has determined that these limits will substantially reduce the significant occupational risk associated with both chronic and peak CO exposures in the workplace. OSHA concludes that the hypoxic stress associated with overexposures to carbon monoxide clearly constitutes a material impairment of health and functional capacity.

CHLORPYRIFOS

CAS: 2921–88–2; Chemical Formula: $C_9H_{11}Cl_3NO_3PS$

H.S. No. 1091

OSHA had no former limit for chlorpyrifos. The ACGIH has a TLV–TWA of 0.2 mg/m^3 and a 0.6-mg/m^3 STEL, with a skin notation, for this white, crystalline solid. The proposed PELs were an 8-hour TWA of 0.2 mg/m^3 and a 15-minute STEL of 0.6 mg/m^3, with a skin notation; NIOSH (Ex. 8–47, Table N1) concurs with these limits. The 0.2-mg/m^3 8-hour TWA and a skin notation are established in the final rule, but the proposed STEL is not retained.

Chlorpyrifos has an acute oral LD_{50} of 135 mg/kg for female rats and 163 for male rats (Windholz 1983b, pp. 309–310, as cited in ACGIH 1986/Ex. 1–3, p. 138). Other sources have reported the acute oral LD_{50} as 82 mg/kg in rats and the dermal LD_{50} as about 2000 mg/kg for rabbits (Gray 1965/Ex. 1–1151; Gaines 1969/Ex. 1–320).

Chlorpyrifos is an active inhibitor of plasma cholinesterase but has only moderate capacity to reduce red blood cell cholinesterase or to cause cholinergic symptoms and systemic injury (ACGIH 1986/Ex. 1–3, p. 138). Particle inhalation has been shown to cause mild plasma cholinesterase depression in dogs exposed for four hours at the upper end of a 140- to 280-mg/m^3 range (Spencer 1968, as cited in ACGIH 1986/Ex. 1–3, p. 138).

Dogs and rats fed 3.0 mg/kg of chlorpyrifos daily for two years showed no adverse effects (FAO/WHO (Food and Agriculture Organization/World Health Organization) 1972, as cited in

ACGIH 1986/Ex. 1-3, p. 138). Male and female rats showed no teratogenic or reproductive effects when fed 1.0 mg/kg per day (Dow Chemical Company 1972a, as cited in ACGIH 1986/Ex. 1-3, p. 138).

Workers applying chlorpyrifos as a spray were exposed to 0.5 percent chlorpyrifos emulsion and exhibited a marked decrease in plasma and red cell cholinesterase levels (Eliason, Cranmer, von Windeguth et al. 1969/Ex. 1-633). In five of seven exposed sprayers, this reduction was greater than 50 percent. However, another study showed no ill effects on cholinesterase metabolism when human volunteers were exposed to an ultra-low-volume spray (0.8 um/m^3 for three to eight minutes) (Ludwig, Kilian, Dishburger, and Edwards 1970/Ex. 1-563). Human cholinesterase levels appear to be less affected by dermal exposure than do those of rabbits (ACGIH 1986/Ex. 1-3, p. 138). However, human volunteers, administered four repeated dermal doses of 25 mg/kg, applied for 12 hours each, did exhibit depressed plasma cholinesterase levels. Human subjects ingesting 0.03 mg/kg for three weeks showed no cholinesterase effects, but subjects ingesting 0.1 mg/kg demonstrated plasma cholinesterase depression (Dow Chemical Compnay 1973f, as cited in ACGIH 1986/Ex. 1-3, p. 138).

The American Industrial Hygiene Association (Ex. 8-16; Tr. III, p. 307) urged OSHA to delete the proposed 0.6-mg/m^3 STEL for chlorpyrifos because the ACGIH has now deleted the STEL for this substance. OSHA has carefully reviewed the health evidence for a STEL for this substance and has determined, in accordance with the Agency's policy (see Section VI.C.17 of this preamble), that it is appropriate not to include a short-term limit in the final rule.

In the final rule, OSHA is establishing a PEL of 0.2 mg/m^3 as an 8-hour TWA, with a skin notation; these limits for chlorpyrifos will protect workers against the significant risk of cholinesterase inhibition caused by exposure to this previously unregulated substance. The skin notation is included in the final rule to prevent the systemic effects that have been demonstrated to occur in humans dermally exposed to chlorpyrifos. OSHA finds that the cholinesterase inhibition and systemic effects associated with exposure to chlorpyrifos constitute material impairments of health.

CRUFOMATE

CAS: 299-86-5; Chemical Formula: $C_{12}H_{19}C1NO_3P$
H.S. No. 1103

OSHA had no former limit for crufomate. The ACGIH has a TWA-TLV of 5 mg/m^3 and a STEL of 20 mg/m^3 for this substance. The proposed PELs were 5 mg/m^3 as an 8-hour TWA and 20 mg/m^3 as a 15-minute STEL, and NIOSH (Ex. 8-47, Table N1) concurs. The final rule establishes this 8-hour TWA limit but does not establish a STEL for crufomate. Pure crufomate exists as crystals, and commercial crufomate is a yellow oil.

Crufomate actively inhibits both plasma and erythrocyte cholinesterase. A study in humans showed that ingestion of 200 mg of crufomate daily for seven days caused no apparent cholinesterase inhibition in the subjects of this controlled study; however, rats and dogs receiving higher doses (5 mg/kg/day) for two years did show this effect (McCollister, Olson, Rowe et al. 1968/Ex. 1-350).

The American Industrial Hygiene Association (AIHA) testified at the hearing that, in the AIHA's opinion, OSHA should delete any STELs that the ACGIH has either deleted or indicated that it intends to delete (Ex. 8-16, Tr. p. 3-307). OSHA agrees that such limits should be reevaluated on the basis of current health information (see the discussion in Section VI.C.17); after reviewing the evidence of crufomate's toxicity in short-term exposures, OSHA has determined that it is not appropriate to include a STEL for this substance in the final rule.

Because cholinesterase inhibition is a very sensitive indicator of exposure, OSHA concludes that the final rule's 8-hour TWA limit of 5 mg/m^3 is needed to provide an appropriate margin of safety below the ingestion NOEL of 200 mg/day for humans, which corresponds approximately to an 8-hour inhalation exposure of 20 mg/m^3. The Agency finds that this PEL will protect workers from the significant risk of material health impairment in the form of cholinesterase inhibition, which was possible at the previously uncontrolled levels.

CYANAMIDE

CAS: 420-04-2; Chemical Formula: $H_2NC{=}N$
H.S. No. 1104

Previously, OSHA had no limit for cyanamide. The ACGIH has a TLV-TWA of 2 mg/m^3. The proposed PEL for cyanamide was 2 mg/m^3 as an 8-hour TWA; NIOSH (Ex. 8-47, Table N1) concurs that this limit is appropriate. An 8-hour PEL of 2 mg/m^3 is established in the final rule. Undiluted cyanamide is a deliquescent, crystalline solid.

The average oral LD_{50} for cyanamide in rats is 125 (85 to 180) mg/kg, and cyanamide has been observed to be very irritating and caustic to the skin (American Cyanamide Company Product Information Bulletin, as cited in ACGIH 1986/Ex. 1-3, p. 152). The dermal LD_{50} in rabbits is 590 mg/kg (*Dangerous Properties of Industrial Materials*, 7th ed., Sax and Lewis 1989, p. 891). Irritation occurred in the form of primary skin irritation and, following instillation into the eye, slight irritation of the conjunctival sac (American Cyanamide Company Product Information Bulletin, as cited in ACGIH 1986/Ex. 1-3. p. 152).

When cyanamide is ingested or inhaled by a person who has also consumed an alcoholic beverage, the person experiences vasodilation of the face and neck, tachycardia, tachypnea, nausea, vomiting, and hypotension. This syndrome is referred to as the Antabuse effect. Study of cyanamide's Antabuse-like effects indicate that the effect is about one-half that of an equivalent dose of tetraethylthiuram disulfide (Antabuse) and one-sixth that of tetramethyl thiuram disulfide (Hald, Jacobsen, and Larsen 1952/Ex. 1-905).

OSHA received comments on cyanamide from the American Cyanamid Company (Exs. 8-76, 3-961, and 94). Linda Dulak, Toxicology Program Manager for American Cyanamid, argued that the final rule should not promulgate limits for substances for which there are no analytical methods (Ex. 3-961, p. 13). According to Dr. Dulak:

> Without an analytical limit, the employer cannot determine whether employee exposures are being maintained below those limits * * * (Ex. 3-961, p. 13).

OSHA notes, however, that Dr. Dulak later stated (Tr. XI, p. 75) that American Cyanamid measures the airborne cyanamide level in their plants by sampling for calcium cyanamide. However, Dr. Dulak was unsure whether this method is applicable to hydrogen cyanamide. OSHA notes that a method for the sampling and analysis of cyanamide has been submitted to the docket.

According to Dr. Dulak (Ex. 3-961), OSHA also has not evaluated the technological and economic feasibility of the 2-mg/m^3 limit for cyanamide. However, OSHA notes that American Cyanamid's representatives testified at the hearing (Tr. XI, p. 76) that, although the company has no internal standard for cyanamide, it controls airborne cyanamide exposures by measuring and controlling airborne levels of calcium cyanamide. According to Dr. Dulak, her company has had "no problems" controlling cyanamide exposures (Tr. XI, p. 76).

Dr. Dulak's third point is that there is no health basis for setting an inhalation limit for cyanamide (Ex. 3-961, p. 13).

OSHA does not agree with this view and finds the evidence of cyanamide's irritant properties sufficient to warrant the establishment of an 8-hour TWA limit. Sax and Lewis (*Dangerous Properties of Industrial Materials*, 7th ed., 1989, p. 981) note that this substance is a severe eye irritant, a moderately toxic substance by skin contact, and a poison by ingestion, inhalation, and intraperitoneal injection. The ACGIH (1986/Ex. 1-3, p. 152) set its limit, which is the same as the final rule's PEL, on the basis of cyanamide's relative potential to cause irritation when compared with other irritants (ACGIH 1986/Ex. 1-3, p. 152). Thus, OSHA finds the final rule's PEL both appropriate and protective.

In the final rule, OSHA is establishing an 8-hour TWA limit of 2 mg/m^3 TWA for cyanamide. The Agency concludes that this limit will protect against the significant risks or irritation and of the Antabuse syndrome in individuals who have ingested alcohol. The Agency has determined that this limit will substantially reduce these significant risks, which constitute material health impairments.

DICROTOPHOS (BIDRIN)

CAS: 141-66-2; Chemical Formula $C_8H_{16}NO_5P$
H.S. No. 1131

Previously, OSHA had no limit for dicrotophos; the ACGIH has a TLV of 0.25 mg/m^3 TWA, with a skin notation, for this brown liquid with a mild ester odor. The proposed PEL was 0.25 mg/m^3, with a skin notation; NIOSH (Ex. 8-47, Table N1) concurs that this limit is appropriate. The final rule establishes an 8-hour TWA of 0.25 mg/m^3 and a skin notation.

Dicrotophos is a cholinesterase inhibitor (ACGIH 1986/Ex. 1-3, p. 193). The acute oral LD_{50} in rats is reported as 22 mg/kg, and the percutaneous LD_{50} in rabbits is 224 mg/kg (Stanford Research Institute 1962, as cited in ACGIH 1986/Ex. 1-3, p. 193). Another study reports the oral LD_{50} in rats as 16 to 21 mg/kg and the dermal LD_{50} in the same species as 42 mg/kg (Gaines 1969/Ex. 1-320). Two-year feeding studies in rats given 0, 1, 10, or 100 ppm dicrotophos showed no detectable effects at the 1-ppm concentration. At the higher concentrations, decreased body weights (as compared with those of controls) and cholinesterase inhibition were observed (Woodard Research Corporation 1967, as cited in ACGIH 1986/Ex. 1-3, p. 193). Dietary studies in dogs showed both plasma and erythrocyte cholinesterase inhibition at a 16-ppm concentration, but no significant ill effects at concentrations of 0, 0.16, or 1.6 ppm (Woodard Research Corporation 1967, as cited in ACGIH 1986/Ex. 1-3, p. 193). Studies of vapor inhalation in male rats have shown that transient illness occurred after a one-hour exposure to 910 mg/m^3 of technical dicrotophos, and to 2620 mg/m^3 or 2120 mg/m^3 of 38-percent dicrotophos (Kettering Laboratories 1965, as cited in ACGIH 1986/Ex. 1-3). Dicrotophos does not cause demyelinization in chickens (Tunstall Laboratory 1965 and Kettering Laboratory 1963, both as cited in ACGIH 1986/Ex. 1-3, p. 193), and it is metabolized in a fashion similar to mono-microtophos (Menzer and Casida 1965/Ex. 1-986). Only NIOSH commented on dicrotophos.

The proposed PEL was based on the data described above and, in part, by analogy with other cholinesterase-inhibiting substances. In the final rule, OSHA is establishing an 8-hour TWA permissible exposure limit of 0.25 mg/m^3, with a skin notation, for dicrotophos. The Agency concludes that this limit will protect workers from the material impairments of health, such as cholinesterase inhibition, potentially associated with inhalation, ingestion, and dermal exposure to this substance at the levels formerly permitted by the absence of a limit. OSHA has determined that these limits will substantially reduce this significant risk.

DIMETHYLANILINE

CAS: 121-69-7; Chemical Formula: $C_6H_5N(CH_3)_2$
H.S. No. 1143

OSHA's former permissible exposure limit for dimethylaniline was 5 ppm as an 8-hour TWA, with a skin notation. The ACGIH has an 8-hour TWA limit of 5 ppm, with a 15-minute STEL of 10 ppm and a skin notation. OSHA proposed to retain its 8-hour TWA PEL of 5 ppm with a skin notation and to add a STEL of 10 ppm, and NIOSH (Ex. 8-47, Table N1) concurred with these limits. The 5-ppm 8-hour TWA and skin notation are retained in the final rule, and the 10-ppm STEL is established. Dimethylaniline is a yellow to brown, oily liquid.

One of the major toxic effects of dimethylaniline exposure is methemoglobinemia, although authorities disagree concerning the level at which humans can tolerate exposure to this substance (ACGIH 1986/Ex. 1-3, p. 207).

Hamblin (1963a/Ex. 1-1084) reported that dimethylaniline is quantitatively less toxic than aniline. Dogs administered a single oral dose of 50 mg/kg exhibited methemoglobinemia, and absorption of dimethylaniline through the skin can increase the overall exposure (Hamblin 1963/Ex. 1-1085). The dermal LD_{50} in rabbits is 1770 mg/kg (*Dangerous Properties of Industrial Materials*, 7th ed., Sax and Lewis 1989, p. 1360). Mayer (1930/Ex. 1-973) reported that dimethylaniline's necrotic potential was markedly lower than that of aniline, which has a TLV-TWA of 2 ppm. However, von Oettingen (1941/Ex. 1-874) stated that dimethylaniline has a greater depressant effect on the nervous system than does aniline.

The literature on industrial experience with dimethylaniline is limited. Hamilton (1919/Ex. 1-741) reported collapse, prolonged unconsciousness, visual disturbances, and intense abdominal pain following the severe exposure of two workers. Only NIOSH commented on dimethylaniline.

In the final rule, the Agency is retaining the 8-hour TWA PEL of 5 ppm and a skin notation for dimethylaniline; a STEL of 10 ppm is also being promulgated. OSHA finds that the STEL is necessary to afford protection from the CNS depression that follows acute exposures. OSHA concludes that these limits, taken together, will provide workers with protection from the significant risks of skin absorption, methemoglobinemia, and neuropathic effects associated with exposure to this substance; the Agency finds that these effects clearly constitute material health impairments.

DIOXATHION (DELNAV)

CAS: 78-34-2; Chemical Formula: $C_{12}H_{26}O_6P_2S_4$
H.S. No. 1146

OSHA formerly had no permissible exposure limit for dioxathion. The ACGIH has a limit of 0.2 mg/m^3 as an 8-hour TWA, with a skin notation. The proposed PEL was 0.2 mg/m^3 as an 8-hour TWA, with a skin notation, and NIOSH (Ex. 8-47, Table N1) concurs with this limit, which is established in the final rule. Dioxathion is a nonvolatile, very stable, dark amber liquid.

The pesticide, dioxathion, contains both the cis- and trans-isomers of 2,3-p-dioxanedithiol; the cis-isomer is approximately four times as acutely toxic as the trans-isomer (ACGIH 1986/Ex. 1-3, p. 219). The oral LD_{50} values reported for rats range from 23 to 118 mg/kg (with most values in the 23- to 64-mg/kg portion of the range); in dogs, oral LD_{50}s range from 10 to 40 mg/kg. The LC_{50} in rats is 1398 mg/m^3; in mice, it is 340 mg/m^3 (Hercules, Inc. 1973, as cited in ACGIH 1986/Ex. 1-3, p. 219). The percutaneous LD_{50}s in rats and rabbits are reported to be 63 and 85 mg/kg, respectively (NIOSH 1983b, as cited in ACGIH 1986/Ex. 1-3, p. 219). Instillation of 0.1 ml dioxathion into the rabbit eye

produces mild, transient conjunctivitis but no corneal damage (ACGIH 1986/Ex. 1–3, p. 219).

In subacute oral toxicity studies, the no-effect dose level in rats was reported to be 0.22 mg/kg/day; in dogs, a no-effect level of between 0.075 and 0.25 mg/kg/day was indicated (Frawley, Weir, Tusing et al. 1963/Ex. 1–317). The no-effect level in multigenerational studies of reproductive effects in rats was reported to be 10 ppm (Kennedy, Frawley, and Calandra 1973/Ex. 1–340).

Human volunteers who ingested 0.075mg/kg/day of dioxathion had no symptoms related to plasma or blood cholinesterase activity, while those ingesting 0.15 mg/kg/day exhibited a slight decrease in plasma cholinesterase activity (Frawley, Weir, Tusing et al. 1963/Ex. 1–317). The World Health Organization has estimated an acceptable daily intake for man of 0.0015 mg dioxathion/kg (WHO 1967, as cited in ACGIH 1986/Ex. 1–3, p. 219). Only NIOSH commented on this substance.

In the final rule, OSHA is establishing an 8-hour TWA PEL of 0.2 mg/m^3 for dioxathion; the Agency is also establishing a skin notation for this substance. OSHA concludes that these limits will protect workers against the significant risk of metabolic effects associated with inhalation and oral exposure and with dermal penetration of this substance, which was formerly not regulated by OSHA. The Agency has determined that these limits will substantially reduce these significant risks; OSHA finds that the cholinesterase inhibition caused by exposure to dioxathion constitutes a material impairment of health.

DISULFIRAM

CAS: 97–77–8; Chemical Formula: $C_{10}H_{20}N_2S_4$
H.S. No. 1151

OSHA had no former limit for disulfiram. The ACGIH recommends a limit of 2 mg/m^3 TWA for this crystalline solid. The proposed PEL was 2 mg/m^3 as an 8-hour TWA; NIOSH concurs with this limit (Ex. 8–47, Table N1), which is established by the final rule.

Disulfiram's LD_{50} in rats is reported as 8.6 g/kg (Windholz 1983e, pp. 491–492, as cited in ACGIH 1986/Ex. 1–3, p. 225), and the oral LD_{50} for rabbits is reported to be 2.05 g/kg (Brieger 1947/Ex. 1–717). The compound is highly toxic when injected intraperitoneally, with an LD_{50} of 75 mg/kg for mice (National Technical Information Service, as cited in ACGIH 1986/Ex. 1–3, p. 225). The effects of high-dose ingestion include degenerative changes in the liver and kidneys. Very high doses can cause leukopenia and marked hypoplasia or aplasia of the bone marrow; in the most seriously afflicted animals, the blood urea nitrogen sometimes increased and the thymol turbidity test was positive (Brieger 1947/Ex. 1–717).

Adverse health effects occur in humans consuming alcohol and simultaneously exposed to disulfiram. This represents a significant concern since disulfiram, under the trade name Antabuse, is used as a medication in the treatment of chronic alcoholism. For individuals who drink alcohol and are exposed to disulfiram, the symptoms of exposure are facial vasodilation, tachycardia, tachypnea, nausea, vomiting, pallor, and hypotension. High doses of disulfiram can induce convulsions, cardiac arrhythmias, and myocardial infarction, and the compound has also been associated with polyneuropathy, peripheral neuritis, and skin eruptions (*Compendium of Pharmaceuticals and Specialties* 1968, as cited in ACGIH 1986/Ex. 1–3, p. 225). In industry, there have been reports of minimal skin irritation (Mastromatteo 1973, as cited in ACGIH 1986, p. 225) and of optic neuritis (Norton and Walsh 1972/Ex. 1–877). NIOSH submitted the only comment on this substance.

OSHA is establishing a PEL of 2 mg/m^3 as an 8-hour TWA for disulfiram. The Agency concludes that this limit will protect workers against the significant risk of Antabuse-like effects associated with exposure to airborne concentrations of disulfiram in combination with alcohol consumption. OSHA has determined that this limit will substantially reduce this significant risk and that the symptoms of the Antabuse syndrome clearly constitute material impairment of health.

ETHION (NIALATE)

CAS: 563–12–2; Chemical Formula: $C_9H_{22}O_4P_2S_4$
H.S. No. 1160

OSHA formerly had no permissible exposure limit for ethion. The ACGIH has a limit of 0.4 mg/m^3 TWA, with a skin notation. The proposed PEL was 0.4 mg/m^3 as an 8-hour TWA, with a skin notation; NIOSH (Ex. 8–47, Table N1) concurs that these limits are appropriate, and the final rule establishes them. Pure ethion is an odorless and colorless liquid; however, technical-grade ethion has a very disagreeable odor.

Ethion is an insecticide that is used in a variety of forms, including 25-percent wettable powder, 2-, 3-, and 4-percent dust, 5-percent granules, and in several oil solutions and combinations with other chemicals. As a result, the acute toxicity values reported vary considerably.

NIOSH (1974d, as cited in ACGIH 1986/Ex. 1–3, p. 236) reports an oral LD_{50}s in rats of 13 mg/kg. Other reported values for oral LD_{50}s in rats include 65 mg/kg, 96 mg/kg, and 208 mg/kg (*Farm Chemicals Handbook* 1974/Ex. 1–1147a; Association of American Pesticide Control Officials, Inc. 1969, as cited in ACGIH 1986/Ex. 1–3, p. 236; Hayes 1963/Ex. 1–982). Studies with 95 percent technical ethion report oral LD_{50}s of 87.4 ± 0.16 mg/kg for albino rats and 24.4 mg/kg for female rats (Niagara Chemical Division, FMC Corp., as cited in ACGIH 1986/Ex. 1–3, p. 236). Inhalation studies report LC_{50} values of 710 mg/m^3 for female rats exposed to 25-percent wettable powder dust for one hour, and 7200 mg/m^3 for male rats similarly exposed. Dermal exposure studies, employing technical ethion, report a median acute dermal lethal dose in rabbits of 915 mg/kg, demonstrating enthion's ability to penetrate skin; instillation of 0.05 ml ethion in the rabbit eye is immediately irritating but does not cause corneal scarring (Niagara Chemical Division, FMC Corp., as cited in ACGIH 1986/Ex. 1–3, p. 236). Dietary studies of rats fed 600, 1000, or 1500 ppm reported complete cholinesterase inhibition; 300 ppm in the diet produced marked cholinesterase inhibition (Association of American Pesticide Control Officials, Inc. 1969, as cited in ACGIH 1986/Ex. 1–3, p. 236).

Ethion poisonings have been reported in workers harvesting grapes and peaches (State of California Department of Industrial Relations, as cited in ACGIH 1986/Ex. 1–3, p. 236). Only NIOSH commented on ethion.

OSHA is establishing a PEL of 0.4 mg/m^3 for ethion as an 8-hour TWA, with a skin notation. The Agency concludes that these limits will protect exposed workers from the significant risks of organophosphate poisoning and cholinesterase inhibition formerly permitted by the absence of any OSHA limit. The Agency notes this substance's potential for dermal absorption in laboratory animals and is establishing a skin notation to protect against the risk of systemic toxicity by this route of exposure. OSHA finds that the systemic poisoning and cholinesterase inhibition caused by overexposure to ethion constitute material health impairments.

FENAMIPHOS

CAS: 22224–92–6; Chemical Formula: $C_{13}H_{22}NO_3PS$
H.S. No. 1173

OSHA formerly had no limit for fenamiphos. The ACGIH has a TLV-

TWA of 0.1 mg/m³ for this substance, with a skin notation. The proposed PEL was 0.1 mg/m³, with a skin notation; NIOSH (Ex. 8–47, Table N1) concurs, and this limit is established in the final rule, along with a skin notation. Fenamiphos is a tan-colored, waxy solid.

Fenamiphos is a cholinesterase inhibitor that produces both central and peripheral cholinergic reactions (WHO 1975, as cited in ACGIH 1986/Ex. 1–3, p. 265). The acute oral LD_{50} values reported for fenamiphos are 2 to 19 mg/kg in rats, 22 mg/kg in mice, 56 to 100 mg/kg in guinea pigs, 10 to 17 mg/kg in rabbits, and approximately 10 mg/kg in cats and dogs. Acute dermal LD_{50} values are 72 to 154 mg/kg in rats and 178 to 225 mg/kg in rabbits. One- and four-hour exposures of rats to fenamiphos aerosols resulted in LC_{50} values of 110 to 175 mg/m³ and 91 to 100 mg/m³, respectively. Rabbits exhibited no dermal or eye irritation (WHO 1975 and Loeser and Kimmerle 1971, both as cited in ACGIH 1986/Ex. 1–3, p. 265).

Rats exposed to fenamiphos aerosol at concentrations of 0.03, 0.25, or 3.5 mg/m³ of air for three weeks exhibited no symptoms. At 3.5 mg/m³, rats showed significant depression of plasma cholinesterase; 0.25 mg/m³ was the highest no-effect concentration observed (Kimmerle 1982c, as cited in ACGIH 1986/Ex. 1–3, p. 265). Two-year feeding studies of dogs (at levels of 0.5, 1.0, and 10 ppm) and rats (at levels of 3, 10, and 30 ppm) revealed no treatment-related toxic or oncogenic effects or tissue changes at a dietary level of 10 ppm; no-observed-effect levels were 3 ppm for the rat and 1 ppm for the dog (WHO 1975, as cited in ACGIH 1986/Ex. 1–3, p. 265). Studies of rabbits and rats showed no embryotoxic or teratogenic effects, and results of a three-generation study in rats showed that fenamiphos had no effect on reproduction (WHO 1975, as cited in ACGIH 1986/Ex. 1–3, p. 265). Studies of mice have also shown no mutagenic effects, and a study of chickens demonstrated no delayed neurotoxic efforts (WHO 1975 and Loeser and Kimmerle 1971, both as cited in ACGIH 1986/Ex. 1–3, p. 265). Fenamiphos is metabolized rapidly to sulfoxide and sulfone derivatives and is excreted primarily in the urine, as demonstrated in absorption tests of the skin and the digestive and respiratory tracts of rats and cows (Waggoner and Khasawinah 1974/Ex. 1–579).

There are no reports of human poisonings caused by exposure to fenamiphos, and no quantitative data are available relating adverse health effects to measurable airborne concentrations of fenamiphos. NIOSH submitted the only comment on fenamiphos.

In the final rule, OSHA is establishing a PEL for this substance of 0.1 mg/m³ TWA to protect against the significant risk of anticholinesterase effects presented by exposure to this substance at the levels formerly permitted by the absence of an OSHA limit. A skin notation is also established based on the evidence of systemic toxicity via percutaneous absorption of fenamiphos. The Agency concludes that these limits will substantially reduce these risks; OSHA finds that cholinesterase inhibition constitutes a material impairment of health.

FENSULFOTHION (DASANIT)

CAS: 115–90–2; Chemical Formula: $C_{11}H_{17}O_4PS_2$
H.S. No. 1174

Previously, OSHA had no limit for fensulfothion. The ACGIH has a TLV–TWA of 0.1 mg/m³. The proposed PEL was 0.1 mg/m³ as an 8-hour TWA; NIOSH (Ex. 8–47, Table N1) concurs, and this limit is established by the final rule. Fensulfothion is a brown liquid at room temperature.

Fensulfothion has an acute oral LD_{50} of 4 mg/kg in male rats and 1.8 mg/kg in female rats. Aerosol inhalation studies in rats have shown LC_{50}s of 113 mg/m³ for a one-hour exposure and 29.5 mg/m³ for a four-hour exposure (Loeser and Kimmerle 1971, as cited in ACGIH 1986/Ex. 1–3, p. 266). This insecticide has been shown to have effects similar to those of the other thiophosphates, which cause cholinesterase inhibition. Dermal toxicity is high, with LD_{50} values ranging between 14 and 30 mg/kg for male rats and between 3.5 and 3.0 mg/kg for females (NIOSH 1974d, as cited in ACGIH 1986/Ex. 1–3, p. 266). Tests of mice and rabbits have shown no embryotoxic, reproductive, or mutagenic effects. The no-effect dietary level in subchronic feeding studies is reported to be 1 ppm in rats and 2 ppm in dogs. The no-effect level for cholinesterase inhibition is reported as 1 ppm in the diet for both dogs and cats (ACGIH 1986/Ex. 1–3, p. 266).

In humans, dermal studies have shown irritation without cholinesterase effects from two-hour, twice-daily applications of a 5-percent granular formulation to the forearms of three subjects. Systemic absorption through the lungs has been demonstrated after inhalation of fensulfothion aerosols (ACGIH 1986/Ex. 1–3, p. 266). No comments, other than NIOSH's, were received on this substance.

In the final rule, OSHA is establishing a PEL of 0.1 mg/m³ TWA for this previously unregulated substance to reduce the significant risks of metabolic effects and skin irritation. The Agency concludes that this limit will substantially reduce these risks, and that skin irritation and cholinesterase inhibition are material impairments of health.

FENTHION

CAS: 55–38–9; Chemical Formula: $C_{10}H_{15}O_3PS_2$
H.S. No. 1175

Previously, OSHA had no limit for fenthion. The ACGIH has a TLV–TWA of 0.2 mg/m³, with a skin notation. The proposed PEL was 0.2 mg/m³, with a skin notation, and the final rule establishes this limit and a skin notation. Fenthion is an oily, yellow- to tan-colored liquid that smells slightly like garlic.

The primary health effect associated with exposure to fenthion is plasma cholinesterase inhibition. The oral LD_{50} values for the rat and rabbit are 215 and 150 mg/kg, respectively, and the dermal LD_{50} in rats is 330 mg/kg (*Farm Chemicals Handbook* 1976/Ex. 1–1147b; NIOSH 1977j, as cited in ACGIH 1986/Ex. 1–3, p. 268). Rats given single intramuscular injections of 5, 25, or 50 mg/kg of fenthion exhibited both enduring electroretinogram changes (ERG) and changes in cholinesterase activity; pseudocholinesterase activity in the plasma dropped to 50 percent of normal on the fourth day after injection. The retinal effects of fenthion persisted for as long as 50 days (Imai 1975/Ex. 1–910). Groups of Donryn rats fed 300 ppm fenthion daily showed symptoms of organophosphate intoxication, including nervousness, general spasms, diarrhea, salivation, and ophthalmologic effects (Kawai, Tojo, Miyazawa et al. 1976/Ex. 1–1157). The no-effect inhalation level for rats has been reported to be 1 mg/m³ for exposures to the aerosol of six hours/day, five days/week for three weeks; at a concentration of 3 mg/m³, cholinesterase inhibition was found (Thyssen 1979, as cited in ACGIH 1986/Ex. 1–3, p. 267). The four-hour inhalation LC_{50} in the rat is between 800 and 1200 mg/m³ (Thyssen 1978, as cited in ACGIH 1986/Ex. 1–3, p. 267).

No mutagenic, carcinogenic, or reproductive effects have been reported (Shirasu, Moriya, Kato et al. 1976/Ex. 1–1097; Hanna and Dyer 1975/Ex. 1–485; and WHO 1976, Food and Agriculture Organization (FAO/WHO) 1979, Oesch 1977, Simmon, Mitchell, and Jergenson 1977, and Herbold 1980, all as cited in ACGIH 1986/Ex. 1–3, p. 268). Single and repeated applications of the compound produced no delayed neurotoxic effects

in chickens (WHO 1972, as cited in ACGIH 1986/Ex. 1-3, p. 268). Two-year feeding studies of rhesus monkeys showed plasma cholinesterase inhibition at the highest oral dose given, i.e., 0.2 mg/kg daily (Rosenblum 1980, as cited in ACGIH 1986/Ex. 1-3, p. 268).

Griffin, Roseblum, and Coulston (1979, as cited in ACGIH 1986/Ex. 1-3, p. 268) reported cholinesterase depression in humans at oral doses of 0.07 mg/kg daily for four weeks, but no effect was observed at 0.02 mg/kg. The lowest lethal dose for humans is 50 mg/kg (*Farm Chemicals Handbook* 1976/Ex. 1-1147b; NIOSH 1977j, as cited in ACGIH 1986/Ex. 1-3, p. 100).

NIOSH (Ex. 8-47, Table N2) does not concur with OSHA's limit for fenthion because a significantly increased incidence of tumors was seen in male mice exposed to fenthion (NC1 1979e, as cited in ACGIH 1986/Ex. 1-3, p. 268) and fenthion is also a mutagen, embryotoxin, and teratogen (Chen, Sirianni, and Huang 1985 and Burdeau and Singh 1973, both as cited in NIOSH/Ex. 150. Comments on Fenthion). OSHA will monitor the toxicological literature on fenthion; however, the Agency believes that the new PEL will protect exposed workers from any of the adverse effects associated with exposure to this substance. No other comments on fenthion were received.

In the final rule, OSHA is establishing an 8-hour TWA limit of 0.2 mg/m^3, with a skin notation, for fenthion. The Agency concludes that these limits will protect workers against the significant risk of cholinergic effects associated with exposures to this substance at the levels formerly permitted by the absence of any OSHA limit. A skin notation is established because of evidence that fenthion is toxic when absorbed through the skin. OSHA finds that cholinesterase inhibition constitutes a material impairment of health.

METHOMYL

CAS: 16752-77-5; Chemical Formula: $C_5H_{10}N_2O_2S$

H.S. No. 1245

OSHA formerly had no limit for methomyl. The ACGIH has a TLV-TWA of 2.5 mg/m^3 for this white crystalline solid with a slightly sulfurous odor. The proposed PEL for methomyl was 2.5 mg/m^3 as an 8-hour TWA; NIOSH (Ex. 8-47, Table N1) concurs, and the final rule establishes this limit.

Methomyl is a cholinesterase-inhibiting insecticide. The oral LD_{50} in rats is reported to be between 25 and 40 mg/kg (Dashiell and Kennedy 1984/Ex. 1-548). Studies of dermal effects have reported no appreciable irritation or sensitization in guinea pigs. Instillation of a 10-percent solution of methomyl in propylene glycol or of the dry material into rabbit eyes caused mild conjunctivitis without corneal injury. However, marked pupillary constriction, a health effect produced commonly by cholinesterase inhibitors, was observed (E.I. du Pont de Nemours and Co., Inc., as cited in ACGIH 1986/Ex. 1-3, p. 363). The LC_{50} of unformulated methomyl as mist is 0.3 mg/L at four-hour exposures; the lethal concentration in rats exposed to a 90-percent water-soluble formulation with a particle size of less than 10 microns was approximately 0.45 mg/L.

Inhalation studies have reported no fatalities resulting from four-hour exposures to the saturated vapor. There is no clinical evidence of cumulative toxicity resulting from 10 doses of 5.1 mg/kg/day over a 14-day period (Harvey, Jelinek, and Sherman 1973/Ex. 1-486). Methomyl is rapidly metabolized and excreted in the urine, and cholinesterase inhibition is thus quickly reversed. In dogs, a dose of 20 mg/kg (one-half the lethal dose) produced symptoms of intoxication and cholinesterase inhibition that disappeared within two to four hours after cessation of exposure (E.I. du Pont de Nemours and Co., Inc. as cited in ACGIH 1986/Ex. 1-3, p. 363). No depression of cholinesterase activity could be detected in rats fed at levels of 0, 200, 400, or 800 ppm methomyl for 79 days. In dogs, 90-day and two-year feeding studies showed no effects at 0, 50, 100, or 400 ppm; however, animals fed at 1000 ppm did demonstrate toxicity. Similar studies of rats have shown kidney, liver, and spleen damage at higher feeding levels, but the no-effect level for both rats and dogs has been reported to be 100 ppm (Kaplan and Sherman 1977/Ex. 1-337). Only NIOSH submitted comments on methomyl.

In the final rule, OSHA is establishing a PEL of 2.5 mg/m^3 (8-hour TWA) for methomyl. The Agency concludes that this limit will protect exposed workers against the risk of cholinesterase inhibition to which they could formerly have been exposed in the absence of any OSHA limit. The Agency has determined that this limit will substantially reduce the significant risk of cholinergic effects, which constitute material impairments of health.

MONOMETHYLANILINE

CAS: 100-61-8; Chemical Formula: $C_6H_5NH(CH_3)$

H.S. No. 1280

OSHA's former PEL for monomethylaniline (N-methyl aniline) was 2 ppm, measured as an 8-hour TWA; this limit was accompanied by a skin notation, indicating that this chemical can readily penetrate the skin. The ACGIH has a limit of 0.5 ppm TWA for monomethylaniline, also with a skin notation. OSHA proposed to reduce the 8-hour TWA PEL to 0.5 ppm and to retain the skin notation; NIOSH (Ex. 8-47, Table N1) concurs, and these limits are established by the final rule. Monomethylaniline is a colorless liquid that turns reddish-brown after standing.

Treon, Deichmann, Sigmon, and associates (1949/Ex. 1-676) found that monomethylaniline applied to the skin of laboratory animals resulted in systemic poisoning, and that the oral LD_{50} in rabbits was 280 mg/kg. A later study by Treon and associates (1950/Ex. 1-533) showed that guinea pigs, rabbits, and rats died from 130 or fewer seven-hour exposures to 7.6 ppm monomethylaniline. In the same study, a monkey survived the same number and length of exposures at 2.4 ppm, and a dog survived 50 exposures at 86 ppm. Exposed animals later developed blood changes, including methemoglobinemia and Heinz bodies (Treon, Sigmon, Wright et al. 1950/Ex. 1-533). NIOSH was the only commenter to the record on monomethylaniline.

In the final rule, OSHA is establishing a 0.5-ppm TWA limit, with a skin notation, for this substance. The Agency concludes that these limits will protect workers from the significant risk of metabolic and blood effects, such as methemoglobinemia, potentially associated with exposure to monomethylaniline. The skin notation will protect workers from the risk of systemic poisoning posed by the skin absorption of this substance. OSHA finds that the methemoglobinemia and skin irritation associated with exposure to monomethylaniline exposure constitute material health impairments.

p-NITROCHLOROBENZENE

CAS: 100-00-5; Chemical Formula: $NO_2C_6H_4Cl$

H.S. No. 1288

OSHA formerly had an 8-hour TWA limit of 1 mg/m^3, with a skin notation, for p-nitrochlorobenzene (PNCB). The ACGIH's new TLV-TWA of 0.6 mg/m^3 (0.1 ppm), with a skin notation for this substance was recently reduced from a TLV-TWA of 3 mg/m^3 (0.5 ppm). The Agency proposed to retain its limit and the skin notation, and the final rule includes these limits. para-Nitrochlorobenzene takes the form of yellow crystals and has a sweet odor.

The primary hazards associated with exposure to PNCB include systemic toxicity to the liver, spleen, bone marrow, and kidneys, as well as

methemoglobinemia and DNA damage. The Monsanto Company (1977, as cited in ACGIH 1986/Ex. 1-3, p. 432.2) reported an oral LD_{50} in rats of 530 mg/kg and a dermal LD_{50} in rabbits of greater than 3040 mg/kg; PNCB was absorbed through rabbit skin to produce methemoglobinemia (Kubota 1960, as cited in ACGIH 1986/Ex. 1-3, p. 432.2), although application to the skin or eyes did not produce irritation (Monsanto Company 1977, as cited in ACGIH 1986/Ex. 1-3, p. 432.2). Rusakov, Korotkova, and Bikbulatov (1973/Ex. 1-660) described the development of sensitization in guinea pigs after dermal application of PNCB.

A four-hour inhalation exposure of rats (heads only) showed that the lethal concentration was approximately 16.1 mg/L (E.I. du Pont de Nemours & Co., Inc. 1981, as cited in ACGIH 1986/Ex. 1-3, p. 432.2). Head-only exposures at 0.05, 0.29, or 0.64 mg/L PNCB for six hours/day, five days/week for two weeks resulted in spleen-weight increases and blood effects in all groups. In addition, there were dose-related effects in blood methemoglobin levels (i.e., decreased hemoglobin, hematocrit, and red blood cell count values). Microscopic changes in the spleen, bone marrow, and kidneys were seen in the two higher-dose groups, and both pathological degeneration of the seminiferous tubules and abnormal epididymal sperm contents were also observed in these groups (E.I. du Pont de Nemours & Co., Inc. 1984, as cited in ACGIH 1986/Ex. 1-3, p. 432.2).

The Monsanto Company (1981, as cited in ACGIH 1986/Ex. 1-3, p. 432.2) reported that a 90-day gavage administration of PNCB at daily doses of 0.3, 10, or 30 mg/kg to male and female rats produced hemolytic effects and spleen changes at all levels, kidney and liver effects at mid- to high-level doses, and hyperplasia of bone marrow and testicular atrophy at the highest dose (30 mg/kg/day). In 1985, Monsanto reported the results of another gavage study in rats. After two years of PNCB feeding at 0.1, 0.7, or 5.0 mg/kg/day, animals in the mid- and high-dose groups exhibited hemolytic effects; in addition, mid- and high-dose groups showed microscopic spleen, kidney, and liver changes and, at the highest dose, bone marrow hyperplasia and testicular atrophy (Monsanto Company 1985, as cited in ACGIH 1986/Ex. 1-3, p. 432.2).

Rats fed PNCB at doses of 0, 0.1, 0.7, or 5 mg/kg/day for up to two years showed methemoglobinemia at the two highest levels, and animals in the 5-mg/kg/day group had indications of anemia and pigment accumulation in spleen cells. No treatment-related increase in tumors was observed (Monsanto Company 1985, as cited in ACGIH 1986/Ex. 1-3, p. 432.2). In a dietary cancer bioassay, rats and mice were given PNCB at unspecified levels for two years (Weisberger, Russfield, Homburger et al. 1978/Ex. 1-535). Only mice were affected, with mice of both sexes showing an increase in vascular tumors at the highest dose and male mice showing an increase in liver tumors at the lowest dose (Weisberger, Russfield, Homburger et al. 1978/Ex. 1-535).

Maternal toxicity was seen in rats given PNCB by gavage at doses of 15 and 45 mg/kg/day on days nine through 16 of gestation; at the 45-mg/kg level, fetotoxicity and teratogenicity were also observed (Nair, Johannsen, and Schroeder 1985/Ex. 1-752). At 15 mg/kg, maternal toxicity but no fetotoxicity or teratogenic effects occurred; at the lowest dose, the only effect was a small increase in maternal spleen weight. A two-generation reproductive study resulted in a reduced mating index in rats given 0.7 or 5.0 mg/kg/day (Monsanto Company 1984, as cited in ACGIH 1986/Ex. 1-3, p. 432.2). Positive responses were observed in a mutation assay of L5178Y TK mouse lymphoma cells (both with and without metabolic activation) and in a microbial assay of *Salmonella* strain TA 1535 (in the absence of metabolic activation); however, no evidence of mutagenicity was noted in assays of three other *Salmonella* strains or in assays of Chinese hamster ovary cells, rat hepatocyte primary culture/DNA repair, or rat bone marrow cell clastogenesis (Monsanto Company 1980-1984, as cited in ACGIH 1986/Ex. 1-3, p. 432.2). PNCB produced DNA damage in the liver, kidney, and brain cells of rats after a single intraperitoneal dose of 30 to 1000 mg/kg (Cesarone, Bolognesi, and Santi 1983/Ex. 1-542) and in cultured hepatocytes at 1.5 hours after a three-hour treatment (Cesarone, Fugassa, Galle et al. 1984/Ex. 1-541).

p-Nitrochlorobenzene may be absorbed through the lungs and skin in humans to produce methemoglobinemia. Reports of industrial exposures indicate that overexposure causes cyanosis, weakness, and headache (Saita and Moreo 1958/Ex. 1-930; Renshaw and Ashcroft 1926/Ex. 1-522). In a study of workmen exposed to average concentrations of PNCB at 55, 125, or 143 ppm and to a 23-ppm concentration of a PNCB-nitrophenol mixture, the authors concluded that the mixed exposure did not produce chronic intoxication, but did cause increased methemoglobin, the appearance of Heinz bodies, headache, vertigo, and occasional eczema; these effects could not be attributed definitely either to skin absorption or to the level of PNCB in the mixture (Pacséri, Magos, and Batskor 1958/Ex. 1-521). No data are reported for the p-nitrochlorobenzene exposures only (Pacséri, Magos, and Batskor 1958/Ex. 1-521).

Only NIOSH commented on p-nitrochlorobenzene. NIOSH (Ex. 8-47, Table N6B and Tr. III, pp. 97-98) notes that this substance is a potential occupational carcinogen and that the risk remaining at the PEL is substantial; NIOSH therefore regards p-nitrochlorobenzene as a candidate for a full section 6(b) rulemaking. OSHA is aware both of the recent toxicological data on this substance and of the ACGIH's recent lowering of the TLV to 0.6 mg/m^3. OSHA will carefully monitor the literature on PNCB and will revise the PEL in the future if such action is warranted.

In the final rule, OSHA is retaining its former 8-hour TWA limit of 1 mg/m^3 for p-nitrochlorobenzene, with a skin notation. The Agency concludes that these limits are necessary to protect workers from the significant risks of methemoglobinemia and changes in the spleen, liver, and kidney possible at higher exposure levels. OSHA is retaining the skin notation because dermal absorption of PNCB has been shown to cause systemic effects in humans and animals. The Agency finds that methemoglobinemia and spleen, kidney, and liver damage constitute material impairments of health.

PHORATE

CAS: 298-02-2; Chemical Formula: $C_7H_{17}O_2PS_3$

H.S. No. 1319

Previously, OSHA had no limit for phorate. The ACGIH has limits of 0.05 mg/m^3 as an 8-hour TWA and 0.2 mg/m^3 as a STEL for phorate, with a skin notation. The proposed PELs were 0.05 mg/m^3 as an 8-hour TWA PEL, with a STEL of 0.2 mg/m^3 and a skin notation; NIOSH concurs with these limits (Ex. 8-47, Table N1), which are established in the final rule. Phorate is an organophosphorus cholinesterase inhibitor that takes the form of a clear liquid and is used as an insecticide.

Phorate is a highly toxic compound in animals. Rats exposed to daily doses of phorate showed effects above 0.15 mg/kg/day but no effects below this level. The no-effect level in dogs is between 0.01 and 0.05 mg/kg/day (Gaines 1969/Ex. 1-320). The dermal LD_{50} in male rats

is 6.2 mg/kg and, for female rats, 25 mg/kg.

The final rule's limits of 0.05 mg/m³ as an 8-hour TWA, supplemented by a STEL of 0.2 mg/m³ and a skin notation, are based on calculations that the no-effect level in humans would lie in the range between 0.21 and 0.7 mg/day, and that use of an appropriate safety factor would suggest an 8-hour limit of 0.05 mg/m³, with a STEL of 0.2 mg/m³, to ensure against excursions greatly in excess of the TWA limit. OSHA received no comments on phorate except those from NIOSH.

OSHA finds that these limits will protect workers exposed to phorate against cholinesterase inhibition and its associated effects, which include respiratory symptoms, nausea, confusion, and vomiting. The Agency concludes that, in the absence of any OSHA limit, phorate-exposed employees were formerly at significant risk of experiencing such effects and that establishing a PEL, STEL, and skin notation will substantially reduce these risks. OSHA finds that cholinesterase inhibition and its symptoms clearly constitute material impairments of health.

PROPOXUR

CAS: 114-26-1; Chemical Formula: $C_{11}H_{15}NO_2$
H.S. No. 1337

OSHA had no former limit for propoxur. The ACGIH has established an 8-hour TLV-TWA of 0.5 mg/m³ for this white, odorless, crystalline compound. The proposed PEL was 0.5 mg/m³ as an 8-hour TWA; NIOSH (Ex. 8-47, Table N1) concurs with this limit, and the final rule establishes it.

The oral LD_{50}s in male and female rats are 83 and 86 mg/kg, respectively; for both sexes, the dermal LD_{50} is greater than 2400 mg/kg (Gaines 1969/Ex. 1-320). Dietary studies in rats at levels of 7.5 mg/kg/day for 28 days or at 800 ppm for three months produced no adverse effect (Association of American Pesticide Control Officials, Inc. 1966/Ex. 1-1011). Rats were exposed to propoxur concentrations of 5, 7, 18.7, or 31.7 mg/m³ six hours/day, five days/week for 12 weeks; animals in the high-dose group showed depressed red blood cell and brain cholinesterase levels, and plasma cholinesterase was depressed by as much as 20 to 30 percent (Association of American Pesticide Control Officials, Inc. 1966/Ex. 1-1011).

In humans, a few cases of mild propoxur poisoning have been reported among sprayers of this insecticide and among residents of propoxur-treated homes (Vandekar, Hedayat, Plestina, and Ahmady 1968/Ex. 1-679). In a study of human volunteers, a single oral dose of 1.5 mg/kg propoxur caused a depression in red blood cell cholinesterase and gastrointestinal symptoms that disappeared two hours after ingestion. Oral doses of 0.75 to 1.0 mg/kg produced no symptoms but did depress erythrocyte cholinesterase (Vandekar, Plestina, and Wilhelm 1971/Ex. 1-680). The only comment on this substance was submitted by NIOSH.

In the final rule, OSHA is establishing an 8-hour TWA of 0.5 mg/m³ for propoxur. The Agency concludes that this limit will protect workers against the significant risk of cholinesterase inhibition associated with exposure to this substance at the levels formerly permitted by the absence of any OSHA limit. OSHA finds that cholinesterase inhibition is a material health impairment.

RONNEL

CAS: 299-84-3; Chemical Formula: $(CH_3O)_2PSOC_6H_2Cl_3$
H.S. No. 1349

OSHA formerly had a limit of 15 mg/m³ TWA for ronnel. The ACGIH has a TLV-TWA of 10 mg/m³ for this white, noncumbustible powder. The proposed PEL was 10 mg/m³ as an 8-hour TWA; NIOSH (Ex. 8-47, Table N1) concurs with this limit, and it is established in the final rule.

Ronnel is an indirect cholinesterase inhibitor that affects the blood plasma rather than the red cell acetylcholinesterase (Plapp and Casida 1958a/Ex. 1-657). The acute oral LD_{50} for rats is reported as 1250 and 2630 mg/kg for males and females, respectively. The oral LD_{50} in dogs is greater than 500 mg/kg (McCollister, Oyen, and Rowe 1959/Ex. 1-594). Two-year dietary studies of rats fed up to 50 mg/kg/day showed no effect on growth rate, food consumption, survival, or hematopoesis (McCollister, Oyen, and Rowe 1959/Ex. 1-594). In a study by Gladenko and Stuk (1972, as cited in ACGIH 1986/Ex. 1-3, p. 513), albino rats developed clinical symptoms of motor irritation, tremor, increased auditory and tactile sensitivity, lacrimation, and salivation within two weeks of exposure at levels between 164 and 328 mg/kg; some animals died during the latter part of the study. At exposures below 16.4 mg/kg, no ill effects were observed (Gladenko and Stuk 1972, as cited in ACGIH 1986/Ex. 1-3, p. 513). A two-year feeding study in dogs exposed at 10 mg/kg showed no ill effects except cholinesterase depletion (Worden, Noel, and Mawdesley-Thomas 1972/Ex. 1-583).

Patch tests of 50 human subjects showed that ronnel has no skin-sensitizing potential (McCollister, Oyen, and Rowe 1959/Ex. 1-594). Only NIOSH submitted comments on this substance.

In the final rule, OSHA is establishing an 8-hour TWA limit of 10 mg/m³ for ronnel. The Agency concludes that this limit will protect workers against the significant risk of cholinergic effects associated with exposure to this substance. OSHA has determined that this limit will substantially reduce this significant risk, and that cholinesterase inhibition constitutes a material health impairment.

SULPROFOS

CAS: 35400-43-2; Chemical Formula: $C_{12}H_{19}O_2PS_3$
H.S. No. 1380

OSHA's Z tables formerly had no limit for sulprofos. The ACGIH has an exposure limit of 1 mg/m³ as an 8-hour TWA. The proposed PEL was 1 mg/m³ as an 8-hour TWA; NIOSH (Ex. 8-47, Table N1) concurred with this limit, and OSHA establishes this limit in the final rule. Sulprofos, also known as the insecticide Bolstar®, is a tan liquid.

Kimmerle (1982b, as cited in ACGIH 1986/Ex. 1-3, p. 547) conducted an extensive animal study on the effects of sulprofos. He reported that the acute toxicity of sulprofos is species-dependent; rats have an oral LD_{50} of 100 to 300 mg/kg and mice have an oral LD_{50} of 1600 to 1800 mg/kg. The reported dermal LD_{50}s are greater than 1000 ml/kg in rats and 800 to 1000 mg/kg in rabbits. In rabbits, sulprofos did not irritate the skin or eyes, and it had no dermal-sensitization effects in guinea pigs. Inhalation studies showed no fatalities in rats exposed to aerosol concentrations of up to 4130 mg/m³ of sulprofos over a period of four hours. In a three-week inhalation study in which rats were exposed to aerosol concentrations of 6, 14, or 74 mg/m³, the two highest concentrations produced cholinergic symptoms; no observable effects were seen at the lowest concentration. Two-year feeding studies by Kimmerle (1982b, as cited in ACGIH 1986/Ex. 1-3, p. 547) in dogs, rats, and mice showed that sulprofos concentrations of 150 ppm, 250 ppm, or 400 ppm were tolerated by all species, with no sulprofos-related tissue changes, signs of toxicity, or oncogenic effects. The overall NOELs were 10 ppm in dogs, 6 ppm in rats, and 2.5 ppm in mice. Kimmerle's ingestion studies in rats and rabbits dosed at levels of 3, 10, or 30 mg/kg/day of sulprofos showed no embryotoxic or teratogenic effects in these animals, and a three-generation diet study in rats also produced no adverse reproductive effects. Mutagenic studies reported by the same author in

mice were negative. Separate subacute inhalation studies also showed no effects on blood cholinesterase levels in rats exposed to 6 mg/m^3 (Zielhuis and van der Kreek 1979/Ex. 1–613). There are no reported cases of poisoning in humans (ACGIH 1986/Ex. 1–3, p. 547). NIOSH was the only commenter on sulprofos.

In the final rule, OSHA is establishing an 8-hour TWA limit of 1 mg/m^3 for sulprofos. The Agency concludes that this limit will protect workers from the significant risk of cholinesterase inhibition, the most sensitive indicator of exposure to this previously unregulated substance. The Agency has determined that this limit will substantially reduce this significant risk, and that cholinesterase inhibition constitutes a material impairment of health.

TERPHENYLS

CAS: 26140–60–3; Chemical Formula: $C_{18}H_{14}$
H.S. No. 1384

The former OSHA limit for the terphenyls was 1.0 ppm as a ceiling limit. The ACGIH has a 0.5-ppm ceiling limit for these substances. The proposed PEL for the terphenyls was 0.5 ppm as a ceiling; NIOSH (Ex. 8–47, Table N1) concurs with, and the final rule establishes, this limit. Terphenyls are colorless or light yellow solids and are used as coolants in nuclear reactors. Commercial preparations contain mixtures of ortho-, meta-, and para-terphenyls.

The terphenyls are primary irritants that cause eye, skin, and respiratory tract irritation. Haley, Detrick, Komesu et al. (1959/Ex. 1–326) reported that mixtures of terphenyls caused conjunctival irritation when instilled into the eyes of rabbits, and damaged guinea pig skin following intracutaneous injection. Cornish, Bahor, and Ryan (1962/Ex. 1–410) determined LD_{50} values of 1900, 2400, and greater than 10,000 mg/kg for the ortho-, meta-, and para-terphenyls, respectively. These authors also conducted 30-day feeding studies of rats involving doses of 250 or 500 mg/kg/day of the individual terphenyl isomers. Rats fed ortho-terphenyl showed elevated liver and kidney weight ratios; rats fed meta-terphenyl displayed elevated kidney weight ratios only; and rats fed para-terphenyl showed no elevation in liver or kidney weight ratios. Two studies by Petkau and Hoogstraaten (1965/Ex. 1–432) and Young, Petkau, and Hoogstraaten (1969/Ex. 1–459) have shown that the terphenyls have nephrotoxic effects and cause hepatic damage in rats fed 33 mg/kg/day. Adamson, Bowden, and Wyatt (1969/Ex. 1–293) published a study in which rats exposed to terphenyl aerosols for seven hours per day at a concentration of 50 mg/m^3 (approximately 5 ppm), for a period of eight days, developed morphological changes in their pulmonary cell mitochondria; the number of vacuolated mitochondria was directly related to duration of exposure.

Weeks (1971/Ex. 1–580) and Weeks and Lentle (1970/Ex. 1–682) conducted a clinical survey of 47 workers with ongoing exposure to terphenyl coolant in a nuclear facility. The study represented 122 man-years of occupational exposure, with durations of exposure ranging from six months to seven years. The airborne concentrations of terphenyl varied, measuring 0.094 mg/m^3 in general working areas and up to 0.89mg/m^3 in areas with organic piping equipment. The terphenyl coolant was determined to be a primary irritant, even in those workers wearing protective clothing, because skin moistness increased dermal sensitivity to the terphenyls (Weeks 1971/Ex. 1–580; Weeks and Lentle 1970/Ex. 1–682). Testa and Masi (1964/Ex. 1–578) reported that, at concentrations above 10 mg/m^3 (approximately 1 ppm, the former OSHA ceiling limit), workers reported both eye and respiratory irritation.

The Motor Vehicle Manufacturers Association (MVMA) recommended generally that OSHA delay rulemaking on a number of substances, including the terphenyls, on the grounds that the MVMA did not have sufficient time to review and evaluate the impacts of this rulemaking (Ex. 3–902). The MVMA did not provide any data or report any problems specific to the health effects or feasibility of the limit proposed for the terphenyls; instead, the MVMA merely listed these substances and many others in its submission. In response to the MVMA, OSHA notes that hundreds of commenters were able to provide detailed information to OSHA in the time allotted. In addition, no other comments were received on the subject of the terphenyls.

In the final rule, OSHA is establishing a ceiling limit of 0.5 ppm for the terphenyls. The Agency concludes that this limit will protect exposed workers against the significant risk of primary irritation of the eyes, skin, and upper respiratory tract and of mitochondrial changes potentially associated with exposure to very low airborne levels of the terphenyls. The Agency has determined that this limit will substantially reduce these significant risks and that primary irritation and metabolic effects constitute material health impairments.

m-TOLUIDINE

CAS: 108–44–1; Chemical Formula: C_7H_9N
H.S. No. 1401

m-Toluidine formerly had no OSHA permissible exposure limit. The ACGIH has a 2-ppm 8-hour TWA, with a skin notation. The proposed PEL was 2 ppm as an 8-hour TWA, with a skin notation, and the final rule establishes these limits. m-Toluidine is a light yellow liquid.

When m-toluidine was tested on the eyes and skin of rabbits, moderate to strong irritation effects resulted (NIOSH 1979b, as cited in ACGIH 1986/Ex. 1–3, p. 589). A mean maximal methemoglobinemia of 60.2 percent was reported to occur following the intravenous administration of 27 mg m-toluidine per kilogram of body weight in cats (McLean, Starmer, and Thomas 1969/Ex. 1–425). Rodent carcinogenicity studies cited by the ACGIH (1986/Ex. 1–3, p. 589) were either inconclusive or negative.

The effects in humans of exposure to m-toluidine, when it is either absorbed through the skin or delivered via inhalation, are hematuria and methemoglobinemia. Exposure to 40 ppm for 60 minutes causes severe poisoning (Goldblatt 1955/Ex. 1–417). There are no epidemiological studies of workers exposed only to m-toluidine (ACGIH 1986/Ex. 1–3, p. 589).

NIOSH does not concur with OSHA's limit (Ex. 8–47, Table N2; Tr. III, p. 86) and reports that, although the evidence for the carcinogenicity of m-toluidine is inconclusive (Weisberger, Russfield, Homburger et al. 1978/Ex. 1–535), it is important to remember that this substance is an aromatic amine, like o- and p-toluidine, both of which are carcinogenic. NIOSH commented that a lower PEL might be appropriate for this substance.

In the final rule, OSHA is establishing a 2-ppm 8-hour TWA and a skin notation for this previously unregulated chemical. The Agency concludes that this limit will protect workers from the significant risk of metabolic effects, such as hematuria and methemoglobinemia, associated with exposure to m-toluidine at the levels formerly permitted in the absence of any OSHA PEL. OSHA finds that hematuria, methemoglobinemia, and the other metabolic effects associated with exposure to m-toluidine constitute material impairments of health.

2,4,6–TRINITROTOLUENE

CAS: 118–96–7; Chemical Formula: $C_7H_5N_3O_6$
H.S. No. 1413

OSHA's former PEL for 2,4,6,-trinitrotoluene (TNT) was 1.5 mg/m ^{3} as an 8-hour TWA, with a skin notation. The ACGIH has set a TLV-TWA of 0.5 mg/m ^{3}, also with a skin notation, for this chemical. The proposed PEL was 0.5 mg/m ^{3} as an 8-hour TWA, and the final rule establishes this limit; the skin notation is retained. NIOSH (Ex. 8-47, Table N1) agrees that this limit is appropriate. TNT occurs as yellow, needle-like crystals and is used as an explosive.

The ACGIH's limit was selected on the basis of health surveys conducted among occupationally exposed workers. Fairhall (1957e, as cited in ACGIH 1986/Ex. 1-3, p. 610) describes dermatitis, cyanosis, gastritis, acute yellow atrophy of the liver, and aplastic anemia as possible effects of exposure to TNT. According to Sollman (1957/Ex. 1-991), blood destruction, leucocytosis or leucopenia, and varying degrees of central nervous system change (probably resulting from anoxia, peripheral neuritis and muscular pains, cardiac muscular and menstrual irregularities, and urinary and renal irritation) can also occur as a consequence of TNT exposure. TNT has irritant properties and may cause sneezing, sore throat, or skin irritation (von Oettingen 1941/Ex. 1-874).

A study by Goodwin (1972/Ex. 1-556) revealed 36 cases of liver damage in a munitions plant where workers were exposed to a mean air level of 2.38 mg/m ^{3} TNT over a period of 20 years. Another study (Morton, Ranadive, and Hathaway 1976/Ex. 1-566) found elevated levels of liver enzymes in 43 TNT shell-packers and loaders who worked where TNT exposures ranged from 0.3 to 0.8 mg/m ^{3} over a period of five months. In 1975, Djerassi and Vitany (Ex. 1-550) published a paper describing hemolytic episodes in three TNT workers with glucose-6-phosphate dehydrogenase deficiency; although these workers were from Iraq, where G-6-PDase deficiency has a high (25 percent) frequency of occurrence, the study is also of concern for other workers having a high frequency of G-6-PDase deficiency. NIOSH was the only commenter to the record on TNT.

In the final rule, OSHA is establishing an 8-hour TWA of 0.5 mg/m ^{3} for 2,4,6-trinitrotoluene; the skin notation is retained. The Agency concludes that this limit is necessary to protect workers against the significant risk of liver damage and hemolytic effects potentially associated with exposure to TNT. OSHA has determined that this limit will substantially reduce these significant risks and that liver damage and hemolysis constitute material health impairments.

Conclusions for the Group of Biochemical/Metabolic Toxins

For the class of toxic substances having biochemical/metabolic effects, OSHA concludes that occupational exposure presents significant risks. The effects associated with exposure to these substances (which inhibit cholinesterase activity, interfere with the blood's ability to carry oxygen, and produce Antabuse-like symptoms and signs) range from nausea, bronchoconstriction, cardiac irregularities, neurobehavioral effects, and unconsciousness to coma and death, depending on the severity of the exposure. OSHA finds that all of these symptoms and signs constitute material health impairments. Because many of these substances are relatively new on the industrial scene, OSHA previously had no limits for them. This situation meant that, in the past, occupational exposures to these substances could be essentially uncontrolled. The Agency finds that establishing or revising limits for this group of toxicants is necessary to reduce these significant occupational risks.

14. Substances for Which Limits Are Based on Avoidance of Sensitization Effects

Introduction

OSHA is establishing limits for eight substances on the basis of their ability to cause pulmonary or skin sensitization. Table C14-1 lists the former, proposed, and revised OSHA PELs and the CAS and HS numbers for these substances. For four of these substances, OSHA had no former permissible exposure limit. For two substances, OSHA has reduced its former 8-hour TWA PEL. In the case of picric acid, OSHA proposed to add a STEL to the former 8-hour TWA PEL for this substance but has determined in the final rule that no STEL is necessary. For toluene-2,4-diisocyanate, OSHA's ceiling limit has been revised to an 8-hour TWA and is supplemented with a STEL.

BILLING CODE 4510-26-M

Table C14-1. Substances for Which Limits Are Based on Avoidance of Sensitization

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1066 Captafol (Difolatan)	2425-06-1	--	0.1 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA
1100 Cobalt metal, fume, & dust	7440-48-4	0.1 mg/m^3 TWA	0.05 mg/m^3 TWA	0.05 mg/m^3 TWA
1222 Isophorone diisocyanate	4098-71-9	--	0.005 ppm TWA 0.02 ppm Ceiling (10 minutes), Skin	0.005 ppm TWA 0.02 ppm STEL, Skin
1313 Phenothiazine	92-84-2	--	5 mg/m^3 TWA, Skin	5 mg/m^3 TWA, Skin
1315 Phenyl glycidyl ether	122-60-1	10 ppm TWA	1 ppm TWA	1 ppm TWA
1329 Picric acid	88-89-1	0.1 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA 0.3 mg/m^3 STEL, Skin	0.1 mg/m^3 TWA, Skin
1373 Subtilisins (Proteolytic enzymes)	1395-21-7	--	0.06 ug/m^3 Ceiling	0.06 ug/m^3 Ceiling
1398 Toluene-2,4-diisocyanate	584-84-9	0.02 ppm Ceiling	0.005 ppm TWA 0.02 ppm STEL	0.005 ppm TWA 0.02 ppm STEL

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

BILLING CODE 4510-26-C

Description of the Health Effects

A sensitization reaction, also known as an allergic reaction, is defined as an adverse response to a chemical following a previous exposure to that substance or to a structurally similar one (Klaasen, Amdur, and Doull 1986/Ex. 1–99). A person who suffers an allergic reaction to a chemical is said to have become sensitized to that substance. Sensitization is the result of an immune reaction to a substance; although the initial exposure does not generate an immediate response, the immune system "remembers" the substance and reacts strongly at the next encounter. A related phenomenon is cross-sensitization. Cross-sensitization occurs when exposure to one substance elicits a sensitization reaction, not only upon subsequent exposure to the same substance, but also upon exposure to a different substance (usually one with a similar chemical structure).

The toxic manifestations of sensitization reactions vary in both location and severity. In humans, common target organs are the skin and the eyes; typical allergic conditions in these organ systems are allergic contact dermatitis and conjunctivitis, respectively. The respiratory system can also be sensitized; the resulting pathologies include bronchitis and asthma (Dean, Murray, and Ward 1986/Ex. 1–195). These allergic reactions are mediated by the two immunoglobulins IgD or IgE. The involvement of IgD results in delayed contact dermatitis. In contrast, IgE-mediated reactions cause very severe and potentially fatal effects, such as acute asthmatic attacks, urticaria, and anaphylactic shock. The unpredictability and potential seriousness of sensitization reactions demand that exposures to sensitizing substances be carefully controlled.

Sensitivity to a chemical frequently persists throughout the lifetime of an individual; in some cases, however, sensitization disappears over time. Sensitization symptoms are not observed after exposure to the sensitizing agent (or to a structurally similar chemical) has been discontinued. Although it is possible to treat some allergies, avoidance is considered the best way, and sometimes the only way, to regain good health.

An additional cause for concern about exposure to sensitizing chemicals is recent evidence that residual respiratory symptoms may continue even after exposure is discontinued. For example, in the case of toluene-2,4-diisocyanate (TDI), Weill, Butcher, Dharmarajan et al. (1981/Ex. 1–1188) and Innocenti, Franzinelli, Sartorelli et al. (1981/Ex. 1–180) found that sensitized workers may exhibit decreased pulmonary function or chronic bronchitis for as long as three and one-half years after cessation of exposure.

Dose-Response Relationships and Sensitization Effects

Like other toxic effects, allergic reactions are dose-related; that is, in response to larger doses of the substance, increasing numbers of subjects become sensitized and the subsequent reactions become more severe. The time course of sensitization for any one individual is unpredictable. Some individuals are sensitized after only one exposure; others remain resistant to sensitization after a lifetime of exposure. Different people are generally sensitive to different substances, although some substances are more universally reactive than others, such as the active agent in poison ivy. Various parameters influence the likelihood of sensitization by a particular chemical; these include such factors as "the nature of the chemical, concentration, type of exposure, genetic susceptibility and nongenetic idiosyncrasies" (Emmett 1986/Ex. 1–226). The sensitization reactions observed in occupational settings are often the result of dermal or inhalation exposure.

For most of the substances in this group, the revised limits have been set on the basis of health surveys and reports of occupationally exposed populations. These studies indicate that exposures below a certain no-effect level generally do not result in individuals becoming sensitized. Where human data were absent or sparse, OSHA relied on animal evidence to set the revised limit. However, since chemically induced immunological sensitization in laboratory animals involves the same mechanism as in humans (that is, immune reactions in animals can be mediated by either IgD or IgE immunoglobulins), sensitization reactions in animals are generally good predictors of immune reactions in humans.

The discussions below describe the record evidence and OSHA's finding for the substances in this group. These discussions illustrate the nature of the risk confronting exposed employees and the extent to which the risk of developing immune sensitization will be reduced among workers by the promulgation of these new or revised limits.

CAPTAFOL (DIFOLATAN)

CAS: 2425–06–1; Chemical Formula: $C_{10}H_9C1_4NO_2S$

H.S. No. 1066

OSHA formerly had no permissible exposure limit for captafol. The proposed limit for captafol was an 8-hour TWA of 0.1 mg/m^3, with a skin notation. The 0.1-mg/m^3 limit, which is consistent with that of the ACGIH, is the PEL included in this final rule; however, OSHA is not including in the final rule the skin notation proposed for this substance. NIOSH (Ex. 8–47, Table N6A) concurred with the Agency's selection of a PEL for captafol, which is a white, crystalline substance with a slight but characteristic odor.

In humans, skin irritation, skin sensitization, and respiratory sensitization have been reported in both American and Japanese studies of farmers applying captafol as a fungicide. Arimatsu (1970/Ex. 1–1010) reported that farmers using captafol have experienced acute contact dermatitis manifesting as erythematous dermatitis and phototoxic eruptions. Kahn (1975, as cited in ACGIH 1986/Ex. 1–3, p. 97) reported that workers cleaning up in an area where captafol was handled experienced skin and respiratory sensitization.

The ACGIH (1986/Ex. 1–3) reported that dermal LD_{50} for captafol is greater than 9 g/kg in rabbits, indicating that the substance is not readily absorbed through the skin. As discussed in Section VI.C.18, OSHA has determined that skin notations are appropriate only when there is evidence that indicates that dermal contact may lead to skin absorption and increase the potential for systemic poisoning. Since this is not the case for captafol, OSHA finds that a skin notation is not warranted.

A two-year study conducted by the World Health Organization (Reinhardt and Brittelli 1981/Ex. 1–1063) reported growth depression in rats at captafol dietary levels of 1500 and 5000 ppm, and histopathologic examination revealed changes in the livers and kidneys of the animals exposed at these levels. In male rats, an increase in liver-to-body-weight ratio was observed at levels of 250 ppm and higher after 12 months of captafol feeding (Reinhardt and Brittelli 1981/Ex. 1–1063). No tumors were observed in this study. However, NIOSH (Ex. 8–47, Table N6A) submitted comments on captafol showing that several newer studies demonstrated that captafol is a broad-spectrum carcinogen in mice and rats (Ito et al. 1984; EPA 1984, 1985, 1987). In 1987, the EPA cancelled the registration for captafol on the basis of

this substance's carcinogenicity in laboratory animals; the EPA considers captafol a Category C substance, i.e., a possible human carcinogen. OSHA is aware of these recent studies on captafol's carcinogenicity and finds that they lend urgency to the establishment of a PEL for this previously unregulated substance. NIOSH's was the only comment OSHA received on this substance.

In the final rule, the Agency is establishing a permissible exposure limit for captafol of 0.1 mg/m³ as an 8-hour TWA to protect workers against the significant risk of contact dermatitis and respiratory and skin irritation and sensitization, all material impairments of health, that are associated with exposure to captafol at the levels formerly permitted by the absence of an OSHA limit. The Agency concludes that this 8-hour TWA PEL will substantially reduce these significant risks.

COBALT METAL, DUST, AND FUME (as Co)

CAS: 7440-48-4; Chemical Formula: Co
H.S. No. 1100

OSHA formerly had an 8-hour TWA limit of 0.1 mg/m³ for cobalt metal, dust, and fume. The Agency proposed an 8-hour TWA of 0.05 mg/m³ for these substances, and NOISH (Ex. 8-47, Table N1) concurred with the proposed limit. The final rule establishes an 8-hour TWA PEL of 0.05 mg/m³ for cobalt metal, dust, and fume; this limit is consistent with that of the ACGIH. Cobalt is a gray, hard, magnetic, and somewhat malleable metal.

Animal studies indicate that high intratracheal doses (10, 25, or 50 mg) of cobalt metal dust can cause obliterative bronchiolitis adenomatosis in guinea pigs (Schepers 1955/Ex. 1-365). Additional studies in animals have shown that exposure to cobalt dust or fume causes hypersensitivity reactions. Increases in serum A-2 globulin and neuraminic acid occurred in dogs and rabbits exposed by inhalation to cobalt metal, metal fume, or carbide blend; injections of cobalt chloride produced similar reactions (Stokinger and Wagner 1958/Ex. 1-381). Studies conducted in miniswine have shown that inhalation of 0.1 mg/m³ cobalt metal dust (50 percent alpha and 50 percent beta variety, with a size range of from 0.4 um to 3.6 um) has caused early (onset within three months) pulmonary disease. Wheezing, which indicates hypersensitivity, occurred in these animals during the fourth week of exposure to 0.1 or 1.0 mg/m³ for six hours/day, five days/week, for three months following a one-week sensitizing dose (Kerfoot, Fredrick, and Domeier 1975/Ex. 1-145). NIOSH (Ex. 150, Comments on Cobalt) submitted comments pointing out that cobalt and cobalt compounds have caused local, injection-site tumors in experimental animals, and the AFL-CIO (Ex. 194, Appendix A) also noted that a "potential carcinogenic effect" has been identified for cobalt.

Pulmonary disease has been reported frequently in workers exposed to cobalt in the manufacture of cemented tungsten carbide (Miller, Davis, Goldman, and Wyatts 1953/Ex. 1-40; Lundgren and Ohman 1954, as cited in ACGIH 1986/Ex. 1-3, p. 144; Lundgren and Swenson 1953/Ex. 1-816). The adverse effect of exposure is generally chronic interstitial pneumonitis. Fatalities have been reported occasionally from exposures to cobalt at concentrations of 1 to 2 mg/m³ or less (Fairhall, Castberg, Carrozzo, and Brinton 1947/Ex. 1-954; Fairhall, Keenan, and Brinton 1949/Ex. 1-479). An increase in serum A-2 globulin fraction was reported in the case of a welder exposed to fumes containing cobalt; the welder had a history of exertional dyspnea and an abnormal chext X-ray (Siegesmund, Funahashi, and Pintar 1974/Ex. 1-372). Schwartz, Tulipan, and Birmingham (1957c, as cited in ACGIH 1986/Ex. 1-3, p. 144) reported that allergic dermatitis has been caused by contact with cobalt and its compounds. Dr. Michael Silverstein, representing the UAW, commented in testimony (Tr. pp. 7-44 to 7-46) that OSHA should develop ancillary provisions, such as those for medical surveillance and personal protective equipment, to protect exposed workers against skin contact with cobalt. However, as discussed earlier in this preamble, OSHA is currently developing generic standards to address these and other protective measures.

In studies undertaken by the Michigan Department of Health (1946-1964, as cited in ACGIH 1986/Ex. 1-3, p. 144), it was demonstrated that, in the period between 1946 and 1964, improved control measures had successfully reduced cobalt metal dust and fume levels from 14.42 mg/m³ to levels below 0.1 mg/m³; no new cases of systemic toxicity or dermatitis have since been associated with cobalt exposure in these facilities. The Pennsylvania Department of Health demonstrated that concentrations could be controlled easily to 0.07 mg/m³; without controls, concentrations were about 0.5 mg/m³ (ACGIH 1986/Ex. 1-3, p. 144).

In posthearing comments, NIOSH (Ex. 150, Comments on Cobalt) reported the findings of two recent epidemiological studies in cobalt-exposed workers. A cohort mortality study by Mur et al. (1987, as cited in Ex. 150) found a statistically significant increase in lung cancer mortality in cobalt and sodium workers in an electrochemical plant. NIOSH noted that this study had several limitations: The small number of lung cancer cases observed; ascertainment problems; no smoking data; and no exposure data. A recent study of hard metal workers in Great Britain (Kusaka et al. 1986, as cited in Ex. 150) found occupationally induced asthma in cobalt-exposed workers, some of whom had average exposure levels below 0.05 mg/m³. NIOSH (Ex. 150, Comments on Cobalt) also noted that this study had several limitations: the small number of workers with asthma; incomplete occupational histories for some cases; and failure to ascertain confounding exposures.

NIOSH also noted that the PEL of 0.05 mg/m³ may not protect all workers against the development of cobalt-induced asthma (Ex. 150, Comments on Cobalt). Both the UAW (Tr. 7-44/7-46) and Dr. James Melinus of the New York State Department of Public Heath (Tr. 11-108) commented that pulmonary disease and ischemic heart disease may be associated with exposures to cobalt at levels of 0.06 mg/m³ and, perhaps, to levels somewhat below. OSHA notes that the studies pointed to by these commenters involve confounding exposures to tungsten, cement, and other hazardous alloys and have other methodological limitations as well.

In the final rule, the Agency is revising its 8-hour TWA limit for cobalt metal, dust, and fumes from 0.1 mg/m³ to 0.05 mg/m³. The Agency concludes that this limit will reduce the significant risk of material impairment of health posed by respiratory disease and pulmonary sensitization, which have been demonstrated to occur at higher levels of exposure. OSHA notes that the very recent literature is suggestive of effects even below this level; the Agency intends to continue to monitor the literature on cobalt in the future.

ISOPHORONE DIISOCYANATE

CAS: 4098-71-9; Chemical Formula: $C_{12}H_{18}N_2O_2$
H.S. No. 1222

OSHA previously had no limit for isophorone diisocyanate (IPDI). The Agency proposed an 8-hour TWA of 0.005 ppm for this substance, with a 10-minute short-term limit of 0.02 ppm and a skin notation; these limits are consistent with NIOSH's recommended limits for all isocyanates, and on Table N1 of Exhibit 8-47, NIOSH indicated its concurrence with the selection of this PEL. The ACGIH has established an 8-

hour TWA of 0.01 ppm and a skin notation for IPDI. In the final rule, OSHA is establishing an 8-hour TWA of 0.005 ppm for isophorone diisocyanate, with a 15-minute STEL of 0.02 ppm and a skin notation.

To date, there is little direct information on the health effects associated with exposure to this particular isocyanate. However, diisocyanates, in general, cause irritation of the respiratory tract, decreases in pulmonary function, and sensitization. The ACGIH (1986/Ex. 1–3, p. 334) cited two reports in which workers exposed to isophorone diisocyanate suffered asthma or dyspnea; neither of these reports contained quantitative exposure data (Clarke and Aldons 1981/Ex. 1–475; Tyrer 1979/Ex. 1–396). The ACGIH (1986/Ex. 1–3, p. 334) recommended that the 0.01-ppm TLV–TWA established for 2,4-toluene diisocyanate (TDI) be applied to isophorone diisocyanate until information specific to IPDI becomes available; however, the ACGIH (1986/Ex. 1–3, p. 334) did not agree that the 0.02-ppm TLV–STEL established by the ACGIH for toluene diisocyanate should also apply to IPDI. In its criteria document on isocyanates, NIOSH (1978c/Ex. 1–259) used similar reasoning to reach the conclusion that, on a molar basis, all of the diisocyanates would react in a manner similar to that of TDI. NIOSH thus recommended that the limits established for TDI (0.005 ppm TWA and 0.02 ppm as a 10-minute short-term limit) be applied to all diisocyanates. In support of the recommended short-term exposure limit for all diisocyanates, NIOSH (1978c/Ex. 1–259) cited a study reporting that 12 workers in an automobile plant had developed severe respiratory symptoms after exposure to 0.03 to 0.07 ppm TDI for one week.

NIOSH (Ex. 150, Comments on Isophorone Diisocyanate) reported that IARC has recently (1986) published results of a positive carcinogenesis bioassay involving TDI that found TDI-induced tumors in both rats and mice. In response to IARC's determination that the evidence in animals is sufficient to classify TDI as a carcinogen in animals, NIOSH is developing a Current Intelligence Bulletin on TDI. OSHA received no comments suggesting that feasibility is a problem at the revised limits, although the proposal specifically requested additional feasibility information from the public. OSHA received several comments on IPDI (Exs. 116, 144, 194). The Workers Institute for Safety and Health (WISH) argued that OSHA should regulate all six of the isocyanates, rather than the three being regulated in this rulemaking, because employers would otherwise tend to substitute the unregulated members of the isocyanate family for those that are regulated, and the unregulated substances might in fact prove as hazardous as the regulated isocyanates (Ex. 116, p. 34). In response to WISH, OSHA notes that the scale and scope of the present rulemaking demanded that OSHA adopt certain methods of selecting substances to include in this rulemaking (see the discussion in the preamble section on "Boundaries to Regulation"); the Agency believes that the isocyanates included in the present rulemaking are those for which the health evidence is adequate to serve as a basis for limit-setting. The AFL–CIO (Ex. 194) commented along the same lines as WISH, but in addition was of the opinion that the proposed 8-hour TWA PEL was not necessary. OSHA does not agree, believing instead that the 8-hour TWA limit will provide additional protection and is appropriate in workplace exposure situations characterized by steady-state exposures. The New Jersey Department of Public Health (Ex. 144) recommended the use of EPA's IRIS data base to set a limit for IPDI; the appropriateness of the IRIS data for limit-setting is discussed in Section VI.A. of the preamble.

OSHA is establishing a 0.005-ppm 8-hour TWA, a 0.02-ppm 15-minute short-term limit, and a skin notation for IPDI. The short-term limit of 0.02 ppm is designed to prevent the severe irritation effects associated with exposure to the diisocyanates even in nonsensitized workers, and the skin notation will prevent dermal absorption of this substance. The Agency has established a 15-minute, rather than a 10-minute, short-term limit for isophorone diisocyanate because OSHA has decided, as a matter of policy, to conform all of its revised short-term limits (5, 10, 15, or 20 minutes) to a duration of 15 minutes. The Agency finds that the TWA and STEL limits will both protect nonsensitized workers against IPDI's sensitizing effects and minimize asthmatic reactions among sensitized workers. OSHA concludes that these revised limits will reduce the significant risk of material health impairment (i.e., immune-system-mediated pulmonary sensitization, which is associated with isocyanate exposure. In addition, the Agency also finds that these limits are feasible. OSHA will continue to monitor the toxicological literature on all of the isocyanates in the future.

PHENOTHIAZINE

CAS: 92–84–2; Chemical Formula: $S(C_6H_4)_2NH$
H.S. No. 1313

OSHA previously had no occupational exposure limit for phenothiazine. The Agency proposed an 8-hour TWA PEL of 5 mg/m^3 for this substance, with a skin notation; the final rule establishes this limit and a skin notation, which are consistent with the recommendations of the ACGIH. NIOSH (Ex. 8–47, Table N1) concurred with OSHA's proposed limit for phenothiazine.

OSHA is basing the PEL for phenothiazine primarily on the findings of a study by Mawhinney and Rakow (1968, as cited in ACGIH 1986/Ex. 1–3, p. 472) that showed that exposure to 15 to 48 mg/m^3 of phenothiazine was associated with skin sensitization but not with other acute systemic effects. Symptoms of sensitization in workers included burning and itching of the skin. Accompanying these sensitization reactions were pinkish-red-colored hair and brown fingernails. Phenothiazine has been reported to cause photosensitization of the skin, and intense irritation and itching of the skin have been associated with inhalation of phenothiazine spray (ACGIH 1986/Ex. 1–3, p. 472).

In the final rule, OSHA is establishing an 8-hour TWA PEL of 5 mg/m^3, with a skin notation; this limit is below the exposure range that has been shown to cause sensitization reactions in workers. OSHA concludes that the uncontrolled occupational exposures to phenothiazine that were possible in the absence of an OSHA limit pose a significant risk of sensitization, which is a material impairment of health. Accordingly, the Agency is establishing an exposure limit that will substantially reduce this significant risk.

PHENYL GLYCIDYL ETHER

CAS: 122–60–1; Chemical Formula $C_6H_5OCH_2CHOCH_2$
H.S. No. 1315

OSHA's former 8-hour TWA limit for phenyl glycidyl ether (PGE) was 10 ppm. The Agency proposed a TWA of 1 ppm for this substance, which is consistent with the ACGIH's limit for PGE. NIOSH recommends a 15-minute ceiling limit of 1 ppm for phenyl glycidyl ether, which is a colorless liquid. In the final rule, OSHA establishes an 8-hour TWA PEL of 1 ppm for phenyl glycidyl ether.

Exposure to PGE causes systemic effects and irritation. Studies by Hine, Kodama, Wellington, and colleagues (1956/Ex. 1–331) showed pulmonary inflammation and liver changes in some of the rats exposed to 100 ppm for seven hours daily for 50 days; respiratory

distress and minimal eye irritation were also observed in the exposed animals. Intragastric LD_{50} values of 1.40 g/kg for mice and 3.85 g/kg for rats were also reported. Animals displayed central nervous system (CNS) depression, and death was caused by respiratory paralysis; in the survivors, these CNS effects were transient. The percutaneous LD_{50} reported for rabbits was 2.99 g/kg. Other studies have reported a single-dose oral LD_{50} of 4.26 g/kg, although exposure for 8 hours to the near-saturated vapor was not lethal (Smyth, Carpenter, Weil, and Pozzani 1954/Ex. 1–440). Terrill and Lee (1977/Ex. 1–390) reported kidney, liver, spleen, thymus, and testicular changes in rats exposed to phenyl glycidyl ether at 29 ppm for four hours daily, five days/week for two weeks. At concentrations of 12 or 5 ppm, these authors observed no effects other than hair loss after exposures of six hours/day, five days/week for nine weeks; however, after 18 weeks, 10 percent of male and 25 percent of female rats exhibited alopecia (hair loss). These health effects were believed by the authors to reflect direct irritation of the skin rather than systemic absorption (Terrill and Lee 1977/Ex. 1–390).

Reports of workers using or handling phenyl glycidyl ether have described moderate skin irritation on prolonged or repeated contact. In addition, several cases of skin sensitization have been reported (ACGIH 1986/Ex. 1–3, p. 476).

NIOSH (1978d/Ex. 1–232) notes that glycidyl ethers are biologically reactive compounds because of the presence of the epoxide group; these compounds have also been shown to cause cytotoxic effects and to the mutagenic in short-term bioassays. Terrill and Lee (1977/Ex. 1–390) exposed rats repeatedly to 1 ppm PGE and observed no effects, although skin damage was observed at 5 ppm. Inconclusive evidence of testicular degeneration was reported in some of the rats exposed to levels as low as 1.75 ppm (Haskell Laboratory reports, as cited in NIOSH 1978d/Ex. 1–232, p. 114). At 10 ppm, five day/week exposures for 10 weeks caused respiratory tract irritation and early signs of liver necrosis in rats (Hine, Kodama, Wellington et al. 1956/Ex. 1–331).

OSHA received only one comment on this substance. NIOSH (Ex. 8–47, Table N6B) does not concur with OSHA's establishment of a PEL of 1 ppm for PGE; NIOSH recommends that this limit be expressed as a 15-minute short-term limit. The Agency concludes that the evidence indicates that repeated, prolonged exposures to PGE have been responsible for the adverse exposure effects observed and, therefore, that an 8-hour TWA PEL is more appropriate for this substance than a ceiling limit or STEL.

In the final rule, the Agency is reducing the 8-hour TWA PEL for phenyl glycidyl ether to 1 ppm. OSHA concludes that this limit will protect workers from the significant risk of skin sensitization, skin and respiratory tract irritation, testicular damage, and liver necrosis (all of which are material impairments of health) that are potentially associated with exposure to concentrations at the former PGE limit of 10 ppm. OSHA finds that the revised limit will substantially reduce these significant risks.

PICRIC ACID

CAS: 88–89–1; Chemical Formula: $HOC_6H_2(NO_2)_3$
H.S. No. 1329

OSHA's former limit for picric acid was 0.1 mg/m³ as an 8-hour TWA, with a skin notation. The Agency proposed to retain the 0.1-mg/m³ TWA limit and skin notation and to add a 15-minute STEL of 0.3 mg/m³ for this substance. NIOSH (Ex. 8–47, Table N1) concurred with the proposal. In the final rule, the Agency has retained the 8-hour TWA of 0.1 mg/m³ and a skin notation, but has determined that the evidence is insufficient to establish the 15-minute short-term exposure limit of 0.3 mg/m³ proposed by the Agency for this substance.

Picric acid occurs as colorless to pale yellow, odorless, intensely bitter crystals. Picric acid and its salts are toxic by ingestion, skin contact, or inhalation, and these substances also have skin-sensitization potential (Schwartz 1944/Ex. 1–367). Available reports concerning human exposures describe edema, papules, vesicles, and desquamations of the face, mouth, and nose (Sunderman, Weidman, and Batson 1945/Ex. 1–383). The symptoms of systemic poisoning following skin absorption include headache, vertigo, vomiting, nausea, diarrhea, and skin and conjunctival discoloration, as well as discoloration of urine and albuminuria; high-dose exposures caused destruction of erythrocytes and produced gastroenteritis, hemorrhagic nephritis, and acute hepatitis (Sunderman, Weidman, and Batson 1945/Ex. 1–383). Occupational exposure to ammonium picrate dust at concentrations of 0.0088 to 0.1947 mg/m³ caused dermatitis only in those workers who were least exposed; the ACGIH believes that this suggests that desensitization or adaptation occurs with repeated exposure (ACGIH 1986/Ex. 1–3, p. 490). Except for the concurrence of NIOSH (Ex. 8–47, Table N1), no comments related to picric acid were submitted to the record. Since the time of OSHA's proposal, the ACGIH has decided to delete its TLV–STEL for picric acid (*Threshold Limit Values and Biological Exposure Indices for 1988–1989,* ACGIH 1988b). OSHA has re-examined the evidence described above and has determined that the 0.1-mg/m³ TWA limit alone is sufficient to protect employees from the significant risk of contact dermatitis associated with exposure to picric acid (OSHA's general policies for establishing short-term limits are described in Section VI.C.17). Therefore, OSHA is not including a STEL for picric acid in the final rule.

In the final rule, OSHA is retaining an 8-hour TWA of 0.1 mg/m³ and a skin notation for picric acid. The Agency concludes that these limits will protect workers against the dermatitis and sensitization associated with occupational exposures to picric acid. OSHA finds that both dermatitis and sensitization are material impairments of health.

SUBTILISINS

CAS: 1395–21–7; Chemical Formula: None
H.S. No. 1373

OSHA did not formerly have an occupational exposure limit for the subtilisins; the ACGIH has established a ceiling limit of 0.06 ug/m³ for these substances. OSHA proposed a 0.06-ug/m³ ceiling for the subtilisins, and the final rule establishes this limit. NIOSH (Ex. 8–47, Table N1) agreed with the selection of this PEL. The subtilisins are proteolytic bacterial enzymes (produced by various *Bacillus* species) that are used primarily in laundry detergents but also in contact lens cleaners, film processing, and the food industry. They are considered a threat to occupational health because they cause immune-system-mediated bronchoconstriction and respiratory symptoms in addition to primary irritation of the skin and respiratory tract (ACGIH 1986/Ex. 1–3, p. 540; Pepys, Hargreave, Longbottom, and Faux 1969/Ex. 1–568).

A report by the California Department of Public Health (1969, as cited in ACGIH 1986/Ex. 1–3, p. 540) showed that several workers were hospitalized after exposure to subtilisins in a detergent formulation plant where the "safe limit" for subtilisins was set at 0.12 ug/m³. There is no information on whether this limit was exceeded in this episode or what other conditions prevailed. In addition to NIOSH's comment, OSHA received several other comments on the proposed limit for the subtilisins (Exs. 8–70, 3–684, 137, 164, and 98–13; Tr. p. 3–304; Tr. pp. 10–182 to

10–190). Most of these commenters were of the opinion that OSHA should not establish an exposure limit for the subtilisins because there is currently no method available to monitor workplace exposures to these substances. In addition, these commenters submitted information to the record on the subtilisins' health effects to supplement the data base relied on by OSHA in the proposed rule.

Typical of these comments was the submittal of Beth Concoby and Alice Caddow on behalf of Genencor, Inc., a manufacturer of subtilisins:

> Genencor concurs with OSHA that the PEL's should be reviewed on a periodic basis and updated as new valid scientific information becomes available. However, * * * ½i¾n the proposed standard OSHA is planning to adopt an exposure limit for subtilisins which does not currently have a validated sampling and analytical method for personal sampling * * *. There is also additional health data available that needs to be considered in promulgating an appropriate PEL for subtilisins (Ex. 3–684, pp. 1, 4).

The Enzyme Technical Association (Exs. 8–70, 164, 137, and 98–13; Tr. pp. 10–182 to 10–190) specifically objected to the Agency's use of an early study on the subtilisins; OSHA has responded to this comment by carefully reviewing the recent toxicological literature on the subtilisins, including several new health studies submitted by these participants. These studies document the respiratory toxicity and sensitization potential of the subtilisins. For example, a study by Juniper and Roberts (1984, as cited in Exs. 8–70 and 3–684) reports that 3.7 percent of exposed workers experienced dose-related sensitization symptoms (enzyme asthma, or EA) on exposure to enzyme detergent powders. Symptoms included sweating, headache, pain in the chest, influenza-like symptoms, cough, breathlessness, and wheezing "sufficient at times to incapacitate the patient completely" (Juniper and Roberts 1984, p. 128, as cited in Exs. 8–70 and 3–684). This study also reports that the "prime initiating cause of episodes of EA in otherwise asymptomatic subjects was undoubtedly dust level 'peaks' rather than a low but continuous exposure" (Juniper and Roberts 1984, p. 131, as cited in Exs. 8–70 and 3–684).

In response to the objections of commenters (Exs. 8–70 and 3–684) that no sampling and analytical method exists for the subtilisins, OSHA notes that several such methods have been published (Fulwiler 1971; Fulwiler, Abbot, and Darcy 1972; Bruce, Dunn, Brotherton et al. 1976). The American Industrial Hygiene Association (Tr. p. 3–304) was in favor of keeping the subtilisins in this rulemaking and submitted a sampling and analytical method for these substances. Thus, OSHA finds that there is no issue of monitoring feasibility for this group of enzymes.

Another study in monkeys (Coate, Busey, Schoenfisch, and Newmann 1978, as cited in Exs. 8–70 and 3–684) reports the effects of exposing animals 6 hours/day, 5 days/week for 6 months to atmospheres containing synthetic detergent dust at 1, 10, or 100 mg/m^3 together with enzyme dust at 0.001, 0.01, 0.1, or 1 mg/m^3. Exposures to 10 or 100 mg/m^3 detergent dust together with 0.01 or 1 mg/m^3 enzyme dust produced gross signs of respiratory distress, pulmonary histopathological effects, and pulmonary function impairment (Coate et al. 1978, as cited in Exs. 8–70 and 3–684).

These studies and others (McMurrain 1970; Medical Research Council 1976; Zachariae, Hoegh-Thomsen, Witmeur, and Wide 1981; Thorne Hillebrand, Magreni et al. 1986; Weill, Waggenspack, DeRouen, and Ziskind 1974, all as cited in Exs. 8–70 and 3–684) demonstrate convincingly the need for an exposure limit for the subtilisins. One study (Thorne, Hillebrand, Magreni et al. 1986, as cited in Exs. 8–70 and 3–684) reports that, in guinea pigs, the no-observed-effect level for pulmonary sensitization induced by exposure to the subtilisins for 15 minutes/day for 5 consecutive days was between 0.0083 and 0.041 mg/m^3. Animals exposed on the same regimen at higher levels developed enzyme asthma (Thorne, Hillebrand, Magreni et al. 1986, as cited in Exs. 8–70 and 3–684). Recent evidence also demonstrates that the manufacturers of these enzymes have been able to control the dust exposures of their employees to levels "considerably lower than the proposed * * * TLV * * * recommendation ½ of a ceiling of 0.06 ug/m^3 ¾" (McMurrain 1970, as cited in Ex. 3–684). These reductions in exposure have occurred as a result of a program of strict environmental controls and the adoption of a prilling process that encapsulates the enzymes to reduce enzyme-laden dust.

OSHA is establishing a ceiling limit of 0.06 ug/m ^{3} for the subtilisins; the evidence described above indicates that a ceiling limit of 0.06 ug/m^3 for the subtilisins is necessary to reduce the significant risks of respiratory sensitization, skin irritation, and respiratory effects among members of the exposed worker population; OSHA finds that all of these exposure-related health effects constitute material impairments of health. Recent studies (described above) show that this limit is being achieved at the present time. OSHA concludes that this limit will substantially reduce these significant risks.

TOLUENE-2, 4-DIISOCYANATE

CAS: 584–84–9; Chemical Formula: $CH_3C_6H_3(NCO)_2$
H.S. No. 1398

The former OSHA limit for toluene-2, 4-diisocyanate (TDI) was a ceiling of 0.02 ppm. OSHA's proposed and final rule limits for TDI are 0.005 ppm as an 8-hour TWA and 0.02 ppm as a 15-minute STEL. The ACGIH (1986/Ex. 1–3, p. 584) and NIOSH (Ex. 8–47, Table N1) both recommend a TWA of 0.005 ppm and a STEL of 0.02 ppm for TDI.

TDI is one of the most frequently encountered occupational sensitizers, and it is also a known cross-sensitizer. The revised limit is based on human data showing that workers can develop sensitization reactions at exposure levels below the 0.02-ppm level. Elkins and colleagues (1962/Ex. 1–138) reviewed the incidence of TDI intoxication in 14 plants in Massachusetts between 1957 and 1962. In eleven instances of TDI intoxication, the average concentration of TDI was 0.015 ppm, and in nine cases the average concentration was below 0.01 ppm. In all plants where the average levels were above 0.01 ppm, TDI had caused respiratory problems. TDI-related respiratory problems were not observed when the average concentration of TDI was maintained below 0.007 ppm (Elkins, McCarl, Brugsch, and Fahy 1962/Ex. 1–138).

Williamson conducted two TDI studies (1964 and 1965, as cited in ACGIH 1986/Ex. 1–3, p. 584) that revealed a 5-percent sensitization rate in 99 workers exposed for 18 months to average levels of TDI below 0.02 ppm. The author believed that accidental spills accounted for the high sensitization rate. Williamson also found that six sensitized workers out of 18 exposed to concentrations of TDI below 0.02 ppm for 14 months showed marked decreases in lung function (Williamson 1964 and 1965, as cited in ACGIH 1986/Ex. 1–3, p. 584).

A NOEL (no-observed-effect level) for TDI has been documented. In 1975, Roper and Cromer (Ex. 1–147) failed to observe any symptoms of respiratory illness or changes in pulmonary function in nine employees working in a plant where breathing zone samples showed TDI concentrations of 0.001 to 0.002 ppm.

Wegman and colleagues (1974/Ex. 1–112; 1977/Ex. 1–171; 1982/Ex. 1–133) observed a dose-response relationship between exposure and long-term decline

in lung function as documented by test results among TDI-exposed employees. Only for those workers exposed to less than 0.002 ppm TDI were the results of lung function tests normal (Wegman, Pagnotto, Fine, and Peters 1974/ Ex. 1–112; Wegman, Peters, Pagnotto, and Fine 1977/Ex. 1–171; Wegman, Musk, Main, and Pagnotto 1982/Ex. 1–133).

Several commenters submitted comments on TDI. NIOSH (Tr. 3–96, 97) and the United Auto Workers (Tr. 7–38 to 7–44) urged OSHA to designate TDI as a carcinogen, while the Dow Chemical Company (Ex. 106A) argued that TDI should not be so designated. As discussed in the preamble section entitled "Boundaries to Regulation," OSHA is not specifically designating substances as carcinogens; many other organizations, such as the International Agency for Research on Cancer, the ACGIH, NIOSH, etc. do so. The Workers Institute for Safety and Health (Ex. 106) urged OSHA to regulate all six of the isocyanates, rather than the three included in this rulemaking, on the grounds that employees would switch to the unregulated isocyanates, which might present as great a hazard as the regulated ones. In response to WISH, OSHA notes that the scale and scope of the present rulemaking required that OSHA make decisions on substances to be included to facilitate the process; the selection process is described in the preamble section entitled "Boundaries to Regualtion."

The Agency concludes that the evidence clearly demonstrates that workers are at significant risk of pulmonary sensitization reactions at the former PEL, as evidenced by declines in pulmonary function observed among workers exposed below this level. OSHA has determined that establishing a 0.005-ppm TWA with a 0.02-ppm STEL will substantially reduce this significant risk. The Agency notes that effects have been observed at levels somewhat below the final rule's PEL; OSHA will therefore continue to monitor the toxicological literature on this substance carefully in the future.

Conclusions for This Group of Sensitizing Toxicants

For the eight sensitizing agents included in this category of substances, OSHA concludes that there are significant occupational risks associated with exposure. The effects caused by such exposures are mediated by the immune system and include skin sensitization, substantial decrements in lung function, bronchoconstriction, asthma, and severe skin irritation, all of which constitute material impairments of health and functional capacity. Reducing or establishing exposure limits for these toxic substances will substantially reduce these significant workplace risks.

15. Substances for Which Limits Are Based on Avoidance of Cancer

Introduction

This group comprises 16 substances for which the ACGIH or NIOSH has recommended new or revised limits based on evidence that occupational exposure may be associated with an increased cancer risk. Table C15–1 lists the former OSHA permissible exposure levels (PELs), the proposed PELs, the PELs established in the final rule, and the CAS and HS numbers for these substances. OSHA is proposing to revise existing TWA and/or STEL limits for six substances, retain a PEL for four substances currently listed on Table Z–2, and add limits for four substances not currently listed on OSHA's Z tables. For one previously unregulated substance, chromyl chloride, OSHA has concluded that a separate 6(b) rulemaking is appropriate. For one substance OSHA is not establishing an exposure limit at this time.

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TABLE C15-1. Substances for Which Limits Are Based on Avoidance of Cancer

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1008 Acrylamide	79-06-1	0.3 mg/m^3 TWA, Skin	0.03 mg/m^3 TWA, Skin	0.03 mg/m^3 TWA, Skin
1020 Amitrole (3-Amino-1,2,4-triazole)	61-82-5	--	0.2 mg/m^3 TWA,	0.2 mg/m^3 TWA,
1028 Asphalt fumes	8052-42-4	--	5 mg/m^3 TWA	See text
1033 Beryllium & compounds	7440-41-7	0.002 mg/m^3 TWA 0.005 mg/m^3 STEL (30 min) 0.025 mg/m^3 Ceiling	0.002 mg/m^3 TWA 0.005 mg/m^3 STEL (30 min) 0.025 mg/m^3 Ceiling	0.002 mg/m^3 TWA 0.005 mg/m^3 STEL (30 min) 0.025 mg/m^3 Ceiling
1073 Carbon tetrachloride	56-23-5	10 ppm TWA 25 ppm STEL (5 min/4 hr) 200 ppm Ceiling	2 ppm STEL (60 min)	2 ppm TWA
1086 Chloroform	67-66-3	50 ppm Ceiling	2 ppm STEL (60 min)	2 ppm TWA

TABLE C15-1. Substances for Which Limits Are Based on Avoidance of Cancer (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL*	Proposed PEL	Final Rule PEL
1092 Chromic acid & chromates	Varies with Compound	0.1 mg/m^3 (as CrO_3) Ceiling	0.1 mg/m^3 (as CrO_3) Ceiling	0.1 mg/m^3 (as CrO_3) Ceiling
1094 Chromyl chloride	14977-61-8	--	--	--
1142 Dimethyl sulfate	77-78-1	1 ppm TWA, Skin	0.1 ppm TWA, Skin	0.1 ppm TWA, Skin
1291 2-Nitropropane	79-46-9	25 ppm TWA	10 ppm TWA	10 ppm TWA
1308 Perchloroethylene	127-18-4	100 ppm TWA 200 ppm STEL (5 min/3 hr) 300 ppm Ceiling	50 ppm TWA 200 ppm STEL	25 ppm TWA
1399 o-Toluidine	95-53-4	5 ppm TWA, Skin	2 ppm TWA, Skin	5 ppm TWA, Skin
1400 p-Toluidine	106-49-0	--	2 ppm TWA, Skin	2 ppm TWA, Skin
1425 Vinyl bromide	593-60-2	-	5 ppm TWA	5 ppm TWA
1426 Vinyl cyclohexene dioxide	106-87-6	--	10 ppm TWA, Skin	10 ppm TWA, Skin

TABLE C15-1. Substances for Which Limits Are Based on Avoidance of Cancer (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL*	Proposed PEL	Final Rule PEL
1436 Zinc chromates $(CrVI)^{++}$	Varies with compound	0.1 mg/m^3 (as CrO_3) Ceiling	0.1 mg/m^3 TWA (as CrO_3) Ceiling	0.1 mg/m^3 (as CrO_3) Ceiling

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15-minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

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The following discussion addresses some general aspects of carcinogenicity, together with the methodology used by OSHA in previous rulemakings to assess carcinogenic hazards. Two representative substances are reviewed in terms of their effects, dose-response considerations, and quantitative risk assessments to evaluate the decrease in risk of developing cancer that is expected after revising or establishing PELs for these substances. In this section, quantitative risk models that are widely accepted by the scientific community are used as a means of estimating cancer risks. The multistage model, which is the model primarily used by OSHA, is preferred over other models because it is based on a more plausible biological mechanism of cancer than the other models.

Description of the Health Effects

Cancer is a life-threatening and particularly insidious disease that is brought about by the invasion of organ systems by abnormal tissue growth. The abnormal issue is comprised of cells that have been altered in such a way as to cause unrestricted cell growth. As this unrestricted growth progresses, the abnormal tissue begins to interfere with the vital functions of normal organ systems. In the absence of medical intervention, most forms of cancer are ultimately lethal. In some instances (e.g., colon cancer, breast cancer), life can be prolonged through chemotherapy, radiation treatment, surgery, or some combination of these; however, the quality of life of the victims of cancer is usually severely affected. In other instances, such as lung cancer, there is little hope of survival, even when aggressive treatment strategies are employed. In past rulemakings on occupational carcinogens (see, for example, Benzene, Ethylene Oxide, Asbestos, and Formaldehyde), OSHA has held that malignant disease constitutes material impairment of health and functional capacity.

An increased risk of developing cancer has been associated with occupational or environmental exposure to a number of chemical substances. The development of chemically induced cancer in humans and animals is a complex and multistep process that is not completely understood. It is currently believed that the mechanism by which cancer develops requires at least two stages: initiation and promotion. Initiation occurs when chemicals interact either directly or indirectly with DNA to cause a heritable mutation. Alterations in DNA structure may cause an incorrect reading of the DNA sequence during replication and result in more altered cells, which may eventually be expressed as a tumor. There is a correlation between substances that are mutagenic in *in vitro* test systems and their ability to cause cancer. Although genotoxic assays are not capable of predicting carcinogenic potential with certainty, such assays are useful for the preliminary identification of substances that may have the potential to cause cancer.

The second stage in the carcinogenic process is promotion. Promotion is considered to be the likely mechanism of action when there is no evidence that a substance interacts with genetic material (e.g., when *in vitro* mutagenicity assays are negative). Peroxisome proliferation, immunosuppression, and hormonal alterations are examples of promotional events; these events facilitate the unrestricted multiplication of initiated cells, leading to the development of cancer. When a substance or its metabolite possesses both initiation and promotion capabilities, it is considered to be a complete carcinogen (i.e., exposure to the substance alone is sufficient to cause cancer). Examples of such substances that OSHA has recently regulated include asbestos, benzene, ethylene oxide, and formaldehyde.

In all of OSHA's past rulemakings for carcinogens, the Agency has used a weight-of-evidence approach to assess the carcinogenic potential of chemical substances. This approach involves examining all available human epidemiologic studies, clinical and case studies, animal studies, mutagenicity studies, and metabolic studies, combined with a quantitative assessment of cancer risk, to make determinations regarding the potential that occupational exposure to a substance increases the risk of cancer. OSHA relies most heavily on epidemiologic studies of worker populations and well-conducted animal bioassays to make these determinations. OSHA's overall approach to promulgating regulations for carcinogens has been upheld in a number of court decisions.

The following discussion summarizes how epidemiologic and animal studies are used to assess cancer risk.

Epidemiology studies. Epidemiological studies that include detailed exposure data provide the best evidence for describing a causal relationship between exposure to a substance and the onset of cancer in humans. Epidemiologic evidence has been relied on heavily in OSHA's decisions to promulgate standards for the carcinogens benzene, asbestos, and arsenic. At a minimum, positive epidemiologic studies provide qualitative proof of a causal relationship between exposure to a substance and the development of cancer. A general lack of quantitative exposure data and the long latencies between onset of exposure and appearance of disease may make it difficult to derive quantitative dose-response relationships from epidemiological studies. However, the ability of such studies to link exposures to carcinogens to cancer in humans outweighs these limitations.

Because of the long latency periods associated with chemically induced cancer in humans, these studies cannot be used to detect disease until after irreparable harm has been done. To protect workers or other human populations, therefore, it is necessary to assess the risk of such effects before they occur. The data used for this purpose derive from animal bioassays; these data are used to predict potential human responses and to infer a causal relationship between exposure to a substance and the onset of disease.

Animal data. Animal studies frequently provide the best dose-response data for chemically induced cancer. When relying on such studies, assumptions must be made in order to extrapolate from animal bioassay data to humans; the most important of these are that physiologic, pharmacokinetic, and biochemical parameters are similar between mammalian species. To the extent that adequate metabolic data are available, such data may be used to refine the extrapolation from animals to humans. Despite the need to make such assumptions, it is widely accepted that animals are acceptable surrogates for estimating potential cancer risks in humans. This confidence derives from the observation, after many years of conducting bioassay studies, that there appears to be a reasonable concordance between carcinogenic effects in animals and these effects in humans.

Dose-Response and Quantitative Assessment of Risk

Unlike other chemically induced toxic effects discussed in this preamble, a large body of scientific knowledge has accumulated regarding the mechanisms by which carcinogens act and the quantitative relationship between dose and biological response. As a result of these investigations, several mathematical approaches have been developed that permit estimates to be made of the cancer risk that is associated with exposure to low doses of carcinogenic substances.

Since the dominant view of the carcinogenic process holds that most cancer initiators cause irreversible damage to DNA, there is reason to assume that the dose-response of most carcinogens will follow a linear, nonthreshold relationship. The Office of Science and Technology Policy (OSTP 1985/Ex. 1–1128) recommends the use of models that incorporate low-dose linearity when the data are limited and uncertainty exists regarding the mechanisms of carcinogenic action. In conducting risk assessments for prior rulemakings, OSHA has generally relied on the linearized multistage model.

The multistage model used to assess cancer risks associated with exposure to substances in this group is GLOBAL83, a model developed by K.S. Crump and colleagues. If P(d) represents the lifetime risk of cancer at dose d, and A(d) is the extra risk over the background rate at dose d, then the multistage model has the following form:

$$A(d)=1-\exp[-(q_1d+q_2d^2+\ldots+q_kd^k)]$$

where:

$q_i>0$

$i=1,2,3,\ldots,k$

and $A(d)=[P(d-P(0)]/[1-P(0)]$

For a unique set of q_i, this function will adequately describe (or fit) the experimentally derived data. How well the model describes the data may be mathematically determined by what are termed goodness-of-fit tests. Once the model is fit to the data, the maximum likelihood estimate (MLE) and the 95-percent upper-confidence limit (UCL) of A(d) are calculated using the 95-percent upper-confidence limit on parameter q_1 (q_1^*). The MLE is the point estimate of A(d), and is therefore considered the best estimate of extra risk at dose d.

Dr. Nathan J. Karch, President of Karch Associates and an expert in risk assessment, testified on the appropriateness of using the linearized multistage model to estimate occupational cancer risk:

> The multistage model and the program upon which it is based [GLOBAL83] involves a number of assumptions that are considered unlikely to underestimate risk. At lower doses, the risk is assumed to be linear in dose, and no threshold is assumed to exist * * *. The risk was assumed to be independent of background rates of cancer * * *.
>
> I recognize with growing knowledge of the complexity of various possible mechanisms for cancer induction, that several aspects of the model have come under increasing investigation * * *. Despite what may appear to be conservative assumptions in the use of animal data with the multistage model * * * the multistage model is not likely to be overly conservative at most of the exposures contemplated by this rulemaking. Since the proposed PELs are similar to experimental doses in animals in many cases, the risk estimates from the multistage model tend to be less conservative unless [the PELs] are very high in relation to experimental doses. Moreover, at high doses the risk estimates produced by GLOBAL are similar to those generated by the other commonly used models (Tr. p. 13–50).

OSHA asked Dr. Karch to evaluate the scientific literature on the substances in this group to determine whether the data for each substance were suitable for estimating quantitative cancer risk using the multistage model. Dr. Karch found the data to be suitable for the following substances: acrylamide, amitrole, carbon tetrachloride, chloroform, styrene, o-toluidine, p-toluidine, and vinyl bromide (Ex. 85– Tr. pp. 13–50). (Dr. Karch also found the data adequate to perform quantitative assessments for nickel sulfide roasting and beryllium compounds; these assessments were not included in OSHA's NPRM since no revision of the PELs for these materials was proposed). For the remaining substances examined by Dr. Karch, the data were judged to be unsuitable for use with the multistage model, and, as such, it was impossible to estimate quantitative cancer risk associated with exposure to these substances.

> An analysis that we perform[ed on] extrapolations from annual data using the multistage model in previous OSHA rulemakings on benzene, ethylene oxide and formaldehyde disclosed that the best, meaning maximum likelihood estimates, from the multistage model were similar and not above the range of estimates from epidemiologic data available * * * [on] workers (Tr. pp. 13–5 to 13–53).

For those substances for which data were suitable for estimating quantitative cancer risks, OSHA relied on these estimates, in part, for making its significant risk findings. OSHA has discussed its approach for making significant risk determinations in a number of rulemakings dealing with carcinogens. This approach has been upheld by several courts of appeals. Although not discussed in detail here, the same methodology is being followed in this rulemaking. (See Arsenic, 48 *FR* 1816, 1901–1902 (Jan. 14, 1983), upheld *ASARCO* v. *OSHA*, 746 F.2d 483 (9th Cir., 1984); Benzene, 52 *FR* 34507 (September 11, 1987); Ethylene Oxide, 49 *FR* 25763 (June 22, 1984), *Public Citizen* v. *Tyson*, 796 F.2d 1479 (D.C. Cir., 1986); Asbestos, 51 *FR* 22646 (June 20, 1986), *Building and Construction Trades* v. *Brock*, 838 F.2d 1258 (D.C. Cir., 1988).)

The Supreme Court, in its *Benzene* decision, indicated when a reasonable person might consider a risk significant and take steps to decrease it. The Court stated:

> It is the Agency's responsibility to determine in the first instance what it considers to be a "significant" risk. Some risks are plainly acceptable and others are plainly unacceptable. If, for example, the odds are one in a billion that a person will die from cancer by taking a drink of chlorinated water, the risk clearly could not be considered significant. On the other hand, if the odds are one in a thousand that regular inhalation of gasoline vapors that are 2% benzene will be fatal a reasonable person might well consider the risk significant and take the appropriate steps to decrease or eliminate it. (*I.U.D.* v. *A.P.I.*, 448 U.S. at 655).

The Court stated that "while the Agency must support its findings that a certain level of risk exists with substantial evidence, we recognize that its determination that a particular level of risk is 'significant' will be based largely on policy considerations." The Court added that the significant risk determination required by the OSH Act is "not a mathematical straitjacket," and that "OSHA is not required to support its findings with anything approaching scientific certainty." The Court ruled that "a reviewing court [is] to give OSHA some leeway where its findings must be made on the frontiers of scientific knowledge [and that * * *] the Agency is free to use conservative assumptions in interpreting the data with respect to carcinogens, risking error on the side of overprotection rather than underprotection" (448 U.S. at 655, 656).

As part of the overall significant risk determination, OSHA considers a number of factors. These include the type of the risk presented, the quality of the underlying data, the reasonableness of the risk assessments, the statistical significance of the findings and the significance of risk (see, for example, Arsenic, 48 FR 1864, January 14, 1983).

OSHA pointed out that guidance for the Agency in evaluating significant risk is provided by an examination of occupational risk rates and legislative intent. For example, in the high-risk occupations of firefighting, and mining and quarrying, the average risk of death from all causes of occupational injury or an acute occupationally related illness from a lifetime of employment (45 years) is 27.45 and 20.16 per 1,000 employees respectively. Typical occupational risk of death in occupations of average risk are 2.7 per 1,000 for all service employment. Typical lifetime occupational risks of death in occupations of relatively low risk are 0.48 per 1,000 in electric equipment and 0.07 per 1,000 in retail clothing. These rates are derived from 1979 and 1980 Bureau of Labor Statistics data from employers with 11 or more employees adjusted to 45 years of employment for 46 weeks per year.

Congress passed the Occupational Safety and Health Act of 1970 because of a determination that occupational safety and health risks were too high. Based on this it is clear that Congress gave OSHA authority to reduce risks of average or above average magnitude when feasible. Further the Supreme Court stated that "if the odds are

one in a thousand that regular inhalation of gasoline vapors that are 2% benzene will be fatal a reasonable person might well take the appropriate steps to decrease or eliminate it" (448 U.S. at 656).

In this final rule, OSHA is using the general approach and guidance described above for making significant risk determinations for carcinogens.

The following sections discuss the carcinogenicity evidence for the chemicals listed in Table C15-1. A brief discussion of the data and a quantitative risk assessment (where appropriate) are included to demonstrate the reduction in cancer risk that could result from lowering the current OSHA PELs or establishing new limits for these potential carcinogens.

ACRYLAMIDE

CAS: 79-06-1; Chemical Formula: $CH_2=CHCONH_2$
H.S. No. 1008

The former OSHA 8-hour TWA permissible exposure limit for acrylamide was 0.3 mg/m^3, with a skin notation, and the Agency proposed a revised PEL of 0.03 mg/m^3, with a skin notation, for this substance, based on evidence of its carcinogenicity in animals. NIOSH (Ex. 8-47, Table N6A) concurs that these limits are appropriate for acrylamide. The ACGIH recommends a TLV of 0.03 mg/m^3 for this substance (ACGIH 1986/Ex. 1-3, p. 12). The final rule promulgates an 8-hour TWA PEL of 0.03 mg/m^3, with a skin notation, for acrylamide.

Acrylamide is a white solid and is widely used as a reactive monomer or intermediate in organic synthesis, and polyacrylamide is a polymer that is used in the manufacture of a host of products, including adhesives, mining chemicals, fibers, pharmaceuticals, animal feed, paper sizing, molded parts, textiles, and coagulant aids (American Cyanamid Company, Ex. 94: ACGIH 1986/Ex. 1-3, p. 12). Chronic exposure to acrylamide has been associated with neurotoxic effects in animals and humans; in cats, the no-effect dose level for neurotoxic effects ranges from 0.3 to 1.0 mg/kg/day (ACGIH 1986/Ex. 1-3, p. 12). Neuropathic effects caused by exposure to acrylamide are dose-related and have been seen in rats, cats, and monkeys. Observed effects in humans included muscular weakening, ataxia, incoordination, tremors, and hallucinations. Acrylamide can be absorbed through the skin in sufficient quantities to be systemically toxic; the dermal LDLo in rabbits is 1000 mg/kg (RTECS 1988).

Tests on the mutagenicity of acrylamide have produced conflicting results (ACGIH 1986/Ex. 1-3, p. 12). However, acrylamide is associated with reproductive effects; based on a drinking water study by Smith, Zenick, Preston et al. (1986/Ex. 1-1123), OSHA concluded that acrylamide causes dominant lethality in the male rat (53 FR 21191).

Two studies are available that demonstrate the carcinogenicity of acrylamide: Johnson, Gorzinsky, Bodner et al. (1986/Ex. 1-825) and Bull, Robinson, Laurie et al. (1984/Ex. 1-252). OSHA described both of these studies in the preamble to the proposed rule (53 FR 21191); they are briefly summarized here. In the Bull et al. (1984/Ex. 1-252) study, acrylamide was tested as a skin tumor initiator in female Sencar mice; 12-o-tetradecanoylphorbol-13-acetate (TPA) was used as a promoter. The authors administered six doses ranging from 0 to 50 mg/kg body weight over a two-week period. A dose-related increase in tumor incidence was observed for all routes of exposure tested, including topical, gastric intubation, and intraperitoneal injection. The same authors (Bull, Robinson, Laurie et al. 1986/Ex. 1-252) noted a dose-related increase in lung adenomas in A/J mice administered acrylamide either by gastric intubation or intraperitoneal injection.

The second study was performed by Johnson et al. (1986/Ex. 1-825) on male and female Fischer 344 rats given 0 to 2.0 mg/kg/day acrylamide in drinking water for a period of two years. During the last four months of this study, mortality from cancer was observed at a statistically significant rate in rats exposed at the highest dose level; in addition, tumor incidence increased in animals of both sexes in the highest dose group. In females, tumors of the mammary gland, central nervous system thyroid gland, oral tissues, uterus, and clitoral gland were seen, while males developed tumors of the central nervous system, thyroid, adrenal gland, and scrotum (Johnson, Gorzinsky, Bodner et al. 1986/Ex. 1-825). Peripheral nerve degeneration was also seen in female rats exposed at the 2-mg/kg/day level (ACGIH 1986/Ex. 1-3, p. 13).

OSHA received comments on the proposed limit for acrylamide from NIOSH and from one other rulemaking participant. Linda Dulak, Toxicology Program Manager for the American Cyanamid Company, submitted a detailed critique of OSHA's discussion (53 FR 21191) of acrylamide's carcinogenicity (Ex. 94). According to Dr. Dulak: (1) The Johnson et al. (1986/Ex. 1-825) study described above is "inconclusive" with regard to acrylamide's carcinogenicity; (2) the Bull et al. (1984/Ex. 1-252) study demonstrates only that acrylamide is not a "complete" carcinogen; (3) OSHA has not demonstrated that the risk of exposure to acrylamide at the former PEL of 0.3 mg/m^3 is significant; and (4) OSHA has not demonstrated that it is feasible, either technologically or economically, to achieve the proposed 0.03-mg/m^3 limit (Ex. 94). The paragraphs below discuss each of these points in turn.

Dr. Dulak believes that the results of the Johnson et al. (1986/Ex. 1-825) study should be regarded as "inconclusive" because (1) the presence of a vital infection in the animals of all dose groups "complicates the evaluation of the data"; (2) the highest dose administered was toxic to female rats; and (3) there were high background incidences of tumors among the controls (Ex. 94, p. 6). In addition, American Cyanamid states that the Bull et al. (1984/Ex. 1-252) study demonstrates only that acrylamide is not a complete carcinogen because animals administered acrylamide alone did not develop skin tumors (Ex. 94, p. 8). Dr. Dulak reported that American Cyanamid is currently conducting a second carcinogencity study designed to clarify the questions that arose during the Johnson et al. (1986/Ex. 1-825) study, and preliminary review suggests that these results will differ significantly from those of the early study (Ex. 94, pp. 6-8). Dr. Dulak notes that the ACGIH is planning to review the expanded toxicological data base for acrylamide in the fall of 1988 and that the Food and Drug Administration and the Science Advisory Board of EPA are preparing to review the second American Cyanamid study when it becomes available (Exs. 3-961 and 8-76; Ex. 94, pp. 2-3). OSHA's response to Dr. Dulak's comments follows.

First, as regards the Bull et al. (1984/Ex. 1-252) study, prudent public-health policy dictates that all carcinogens, rather than only complete carcinogens, be regulated to levels that will provide worker protection. Second, OSHA notes that the authors of the Bull et al. (1984/Ex. 1-252) study are of the opinion that the potency of acrylamide as a tumor initiator is equal to that of ethyl carbamate, a widely recognized tumorigen (Klaasen, Amdur, and Doull 1986/Ex. 1-99, p. 123); in addition, these authors demonstrated that mice of a different strain (ie., A/J mice) developed lung adenomas when given acrylamide by gastric intubation or intraperitoneal injection. Third, OSHA finds the Bull et al. (1984/Ex. 1-252) study, which showed a dose-related increase in skin tumors in one strain of mouse by three

different routes of exposure and the development of lung tumors in another strain of mouse by two routes of administration, convincing evidence of acrylamide's carcinogenicity. OSHA looks forward to reviewing both the results of American Cyanamid's second study and the ACGIH TLV Committee's comments on acrylamide when these become available. However, the risk demonstrated by OSHA's risk assessment for acrylamide indicates that delaying regulatory action until additional research has been done would be inappropriate; further, it is the Agency's experience that research results are often not published for several years and that the deliberations of the ACGIH Committee are often time-consuming. OSHA finds it inappropriate to delay action when the best available evidence at present indicates a significant risk at the former PEL. Further, OSHA notes that the ACGIH was sufficiently persuaded of acrylamide's carcinogenicity by the findings of the Bull et al. (1984/Ex. 1–252) and Johnson et al. (1986/Ex. 1–825) studies to assign this substance an A2 (suspected human carcinogen) designation. The International Agency for Research on Cancer (IARC) was also convinced by the evidence presented in these studies; IARC judged that the evidence for the carcinogenicity of acrylamide in animals was sufficient (IARC 1986). However, in light of the ongoing research being conducted by American Cyanamid, OSHA will consider new evidence as it becomes available and will revise its limit if this action appears to be warranted.

In response to Dr. Dulak's third point (that, in American Cyanamid's view, OSHA has not demonstrated that risk at the former PEL of 0.3 mg/m^3 is significant), OSHA points to the results of the Agency's quantitative risk assessment, which show that the maximum likelihood estimate of the risk at the former PEL of 0.3 mg/m^3 is 10 cancer deaths per 1,000 workers exposed at that level over their working lifetimes (Table C15–1).

TABLE C15-2. Multistage Model Estimates of Cancer Risk Associated with Working Lifetime Exposure to Acrylamide

	Excess cancer deaths per 1,000 workers	
Exposure level	MLE	UCL
0.3 mg/m^{3a}	10	45
0.03 mg/m^{3b}	1	5

[a] Former OSHA PEL.
[b] Final Rule PEL.
MLE = Maximum likelihood estimate of risk.
UCL = 95-percent upper-confidence limit on maximum likelihood estimate of risk.

American Cyanamid believes that both the recent epidemiological findings of Sobel, Bond, Parsons, and Brenner (1986, as cited in Ex. 94) in a cohort mortality study of Dow Chemical Company acrylamide-exposed workers and additional results from a more recent mortality study (Collins et al. 1987/Ex. 3–961) of American Cyanamid's workers show that "acrylamide is not carcinogenic to people" (Ex. 94, pp. 9–10).

Dr. Dulak discussed the Collins et al. study (1987/Ex. 3–961, Appendix V) at length in posthearing comment:

> It was determined that the study was large enough to detect the increased risk of cancer which OSHA has indicated would occur at present exposure limits. These findings, therefore, indicate that OSHA has overestimated the risk of cancer among acrylamide workers at the present PEL (Ex. 94, p. 9).

OSHA does not agree that its quantitative risk assessment is inconsistent with the findings of the Collins et al. (1987) study. These investigators reported that the average cumulative exposures (defined as mg/m^3-years, the product of airborne concentration and duration of exposure) for each of the four subcohort plants included in the study ranged from 0.07 to 1.54 mg/m^3-years, with an overall average of 1.0 mg/m^3-years. This cumulative exposure corresponds to a 45-year exposure to 0.02 mg/m^3; at this level of exposure, OSHA's risk assessment shows that the excess lifetime cancer risk is less than one death per 1,000 workers. Thus, at the levels and durations of exposure experienced by the cohort studied by Collins et al. (1987), OSHA's risk assessment suggests that only one or two exposure-related excess cancer deaths would be expected among the 2,293 exposed employees; clearly, such a small excess cancer death rate, which represents an increase of only 3 percent over background rates for all neoplasms,

would not have been detected by this study. OSHA finds that the results presented by Collins et al. (1987) are not inconsistent with the results of OSHA's quantitative risk assessment. OSHA therefore reaffirms in this final rule that it is appropriate to treat acrylamide as a potential occupational carcinogen.

In response to American Cyanamid's final point, which relates to the technological and economic feasibility of achieving the final rule's 0.03-mg/m^3 8-hour TWA level, OSHA notes the following. First, with very few exceptions, the Agency's final Regulatory Impact Assessment (Section VII) has determined that the controls necessary to achieve compliance with the limits proposed in this rulemaking are both technologically and economically feasible. This is clearly the case for firms, such as American Cyanamid, in the Chemical Manufacturing sector, SIC 28. Second, the EPA (1986b) study submitted by American Cyanamid (Ex. 94), entitled *Assessment of Airborne Exposure and Dermal Contact to Acrylamide During Chemical Grouting Operations,* showed that most worker exposures were consistently below the 0.03-mg/m^3 level at the present time. Third, a NIOSH study (Hills and Greife 1986, as cited in Ex. 94) of facilities engaged in acrylamide monomer manufacturing reported considerable variability in exposure levels between the four plants surveyed; the observed variability was due in part to differences in housekeeping practices, age and maintenance of equipment, and use of engineering controls and natural dilution ventilation. NIOSH recommended that both frequent washing of the production area and ventilation be used to reduce airborne exposures to acrylamide. OSHA believes that it is technologically feasible for affected facilities to achieve compliance with the level promulgated by this final rule (see the Technological Feasibility section of this preamble). The Agency is therefore setting a revised 8-hour TWA exposure limit of 0.03 mg/m^3 for acrylamide, with a skin notation, based on the significant risk of cancer posed to workers exposed to this substance in the workplace. OSHA concludes that this effect represents a material impairment of health and functional capacity, and the Agency concludes that the 0.03-mg/m^3 PEL will substantially reduce this significant occupational risk.

AMITROLE

CAS: 61-82-5; Chemical Formula: $C_2H_4N_4$
H.S. No. 1020

OSHA had no former PEL for amitrole; the Agency proposed a TWA limit of 0.2 mg/m^3, which is also the limit recommended by the ACGIH (1986/Ex. 1-3, p. 25) on the basis of positive carcinogenicity studies conducted in rats and mice. OSHA is establishing these limits in the final rule. NIOSH recommends a 10-hour TWA of 0.3 mg/m^3 for amitrole, which is a crystalline solid; however, NIOSH specifically concurred with the limit for this substance being established by the final rule (Ex. 8-47, Table N6A). Amitrole is used as a herbicide and plant growth regulator.

Amitrole is a potent antithyroid agent and has been shown to cause tumors, particularly of the thyroid and pituitary glands, in experimental animals (ACGIH 1986/Ex. 1-3, p. 25). Its tumor-producing activity is thought to be related to its goitrogenic effects, which cause an increase in thyroid-stimulating hormone (TSH). Other antithyroid agents that cause TSH stimulation, such as propylthiouracil, have also been shown to produce thyroid tumors (Guyton 1981/Ex. 1-1002). Amitrole has not been shown to be mutagenic in the Ames bacterial mutation assay, a dominant lethal test in male mice, or in assays that measure recessive sex-linked lethal mutations in *Drosophila melanogaster* (ACGIH 1986/Ex. 1-3, p. 25).

An excess incidence of tumors has been reported to occur among pesticide workers exposed to amitrole alone or in combination with phenoxy herbicides. Although these studies indicate the possible association of increased tumor incidence with exposure to amitrole, confounding factors, such as smoking and concurrent exposure to other pesticides, complicate the interpretation of these data.

The Swedish National Board of Occupational Safety and Health ordered an epidemiological evaluation to assess the incidence of cancer among railroad workers exposed to herbicides (Axelson and Sundell 1974/Ex. 1-812). Amitrole was among the pesticides utilized by these workers. Cohorts were separated into groups according to whether they were exposed to amitrole and combinations of other herbicides, phenoxy acids and combinations of other herbicides, or other herbicides alone. A statistically significant increase in the incidence of total tumors and lung tumors was found among workers exposed to amitrole and combinations of other herbicides. Smoking frequency among members of this group was reported to be similar to the frequency of smoking in the general Swedish population.

In a 1980 follow-up to the Axelson and Sundell (1974/Ex. 1-812) study, Axelson and co-workers (Ex. 1-242) combined data from the earlier study with data on workers exposed from 1972 to 1978. Cohorts were divided into the following exposure groups: amitrole alone, phenoxy acids alone, and amitrole and phenoxy acids combined. The reanalyzed data did not show a statistically significant increase in cancer incidence among the workers exposed to amitrole alone; however, the incidence of tumors among workers exposed to amitrole and phenoxy acids together was significantly increased (Axelson, Sundell, Andersson et al. 1980/Ex. 1-242).

Amitrole has been found to be carcinogenic in laboratory animals following dietary exposure to relatively high doses. Attempts to induce tumors by dermal application and subcutaneous injection have been unsuccessful. Studies investigating the carcinogenic potential of amitrole in laboratory animals are reviewed below.

The effects of lifetime exposure to amitrole were investigated in rats, mice, and hamsters fed diets containing 1, 10, or 100 ppm amitrole (Steinhoff, Weber, Mohr, and Boehme 1983/Ex. 1-208). There was a significant increase in the incidence of thyroid tumors in male and female rats and in the incidence of pituitary tumors in female rats exposed to 100 ppm. An excess incidence of tumors was not found in male or female rats exposed to 1 or 10 ppm. The results of this experiment are presented in Table C15-3. Tumor induction was not observed in male or female mice or hamsters. Another study reported negative results for rats fed diets containing 10, 50, or 100 ppm amitrole (Jukes and Schaffer 1960/Ex. 1-213).

Dermal applications of 0.1 or 10 mg of amitrole produced no increased incidence of tumors in mice (IARC 1982a/Ex. 1-1112).

In contrast to the negative results obtained in mice following lifetime dietary exposure to 1, 10, or 100 ppm amitrole (Steinhoff, Weber, Mohr, and Boehme 1983/Ex. 1-208), positive results were observed in male and female mice following dietary exposure to higher levels (2192 ppm) of amitrole for one year (Innes, Ulland, Valerio et al. 1969/Ex. 1-270). Carcinomas of the thyroid were observed in 89 percent (64/72) of the exposed animals (tumor incidence in controls was not reported).

TABLE C15-3. Incidence of Rat Thyroid and Pituitary Tumors Associated With Ingestion of Amitrole

Tumor Site	Concentration in diet (ppm)			
	0	1	10	100
Thyroid (Male)				
- Benign	5/75	9/75	4/75	45/75*
- Malignant	3/75	0/75	3/75	18/75*
Thyroid (Female)				
- Benign	7/75	12/75	8/75	44/75*
- Malignant	0/75	1/75	4/75	28/75*
Pituitary (Female)				
- Benign	14/75	20/75	15/75	36/75*
- Malignant	1/75	2/75	4/75	5/75

* p 0.001, Fisher Exact Test.

Positive results were also observed in mice exposed to 1 percent (10,000 ppm) amitrole in the diet in a lifetime study (exposure for four weeks followed by one week with no exposure) (Feinstein, Fry, and Staffeld 1978a/Ex. 1-281). Liver tumors developed in 100 percent of the exposed mice; however, the incidence of tumors in unexposed controls was not reported. A small number of thyroid tumors was also reported. The authors hypothesized that the reason more thyroid tumors were not seen was because the animals died of the high toxic doses before such tumors were expressed.

Chronic dietary administration of amitrole in dogs (10, 50, 100, or 500 ppm) and in rainbow trout (1200 or 4800 ppm) did not result in the development of tumors (IARC 1982a/Ex. 1-1112), but these experiments were not long enough to allow for evaluation of the carcinogenicity of the chemical.

Risk estimate for amitrole. The study by Steinhoff et al. (1983/Ex. 1-208) provides sufficient information to estimate quantitatively the excess cancer risk associated with exposure to amitrole in the workplace. The linearized multistage model was chosen to estimate risk. The incidence of malignant thyroid tumors in female rats was used because these tumors demonstrate a clear monotonic response. Female rats were assumed to weigh 250 g and to consume 25 g of food per day. Human risks were estimated at exposure levels corresponding to the proposed PEL of 0.2 mg/m^3, as well as for exposure levels of 0.4 mg/m^3 and 1.0 mg/m^3. OSHA has revised the risk estimates presented in the NPRM on amitrole to correct an overestimate in the calculation of lifetime dose (Ex. 110). The revised excess estimated cancer risk, in terms of excess deaths per 1,000 employees is shown in Table C15-4.

TABLE C15-4. Multistage Model Estimates of Cancer Risk Associated With Working Lifetime Exposure to Amitrole

Exposure Level	Excess Cancer Deaths per 1,000 Workers	
	MLE	UCL
0.2 mg/m^3[a]	2.7	3.5
0.4 mg/m^3	5.3	7.0
1.0 mg/m^3	13	17

[a] Final rule PEL.
MLE = Maximum likelihood estimate of risk.
UCL = 95-percent upper-confidence limit on maximum likelihood estimate of risk.

Exposure to 0.2 mg/m^3 of amitrole for an occupational lifetime (45 years) is associated with an estimated 3 excess cancer deaths per 1,000 employees (0.3 percent). This rate is based on the maximum likelihood estimate (MLE). The 95-percent upper-bound estimate of risk corresponding to this dose is about 4 excess cancer deaths per 1,000 workers. By comparison, the maximum likelihood estimates of risk for lifetime exposure to 0.4 mg/m^3 or 1.0 mg/m^3 are 5 or 13 excess deaths per 1,000 employees, respectively.

NIOSH (Ex. 8-47, Table N6A) concurred with OSHA's 0.2-mg/m^3 TWA limit for amitrole. Two other rulemaking participants commented on OSHA's assessment (Ex. 3-894; Tr. pp. 3-13 to 3-14). The American Industrial Health Council (AIHC) (Ex. 3-894) urged OSHA to use a different risk assessment procedure for amitrole that incorporates information on the mechanism by which amitrole induces thyroid tumors:

> Where, as in the case of amitrole, the data indicate that the tumors in the experimental animals are in endocrine sensitive tissue and the mechanism is a secondary hormonal action, a risk assessment procedure incorporating these mechanistic data should be used (Ex. 3-894, p. I-8).

The AIHC cites a draft EPA report on thyroid follicular cell carcinogenesis (EPA/625/3-88/014A, EPA 1988), which indicates that the steps leading to thyroid follicular cell tumors are expected to show a threshold effect. The AIHC also cites an FDA report (*General Principles for Evaluating the Safety of Compounds Used in Food-Producing Animals,* issued in conjunction with 52 FR 49572, FDA 1988), in which FDA concludes that, for the group of "endogenous sex steroids that have been adequately tested," the oncogenic response is related to overstimulation of the hormonal system and no cancer hazard is perceived to exist if the hormonal system is not overstimulated (Ex. 3-894, p. I-8).

Dr. Isadore Rosenthal, Corporate Director for Safety and Health at the Rohm and Haas Company, also testified on the mechanism of amitrole's carcinogenicity:

> There is much scientific evidence on threshold effects in regard to the generation of thyroid cancers by goiterogenic agents. In fact, the EPA has proposed using a new threshold risk assessment method for evaluating thyroid carcinogens (Tr. pp. 3-13 to 3-14).

OSHA recognizes the possibility that a threshold effect level may exist for the development of tumors induced by this special class of substances that act on endocrine-sensitive tissues. OSHA notes that EPA's preliminary findings and proposed threshold risk assessment model are still under review by the Science Advisory Board, and the Agency eagerly awaits EPA's final conclusions on this issue. OSHA points out, however, that amitrole has produced liver tumors in mice (Innes, Ulland, Valerio et al. 1969/Ex. 1-270; Feinstein, Fry, and Staffeld 1978a/Ex. 1-281) and, in one instance (Feinstein, Fry, and Staffeld 1978a/Ex. 1-281), the liver tumors appeared at an earlier age and at a higher incidence than did thyroid tumors. It is not clear from the present data that the mechanism for the development of these liver tumors is the same as that for thyroid tumors. OSHA also notes that the proposed 0.2-mg/m^3 PEL is, according to the ACGIH's calculations, only a factor of 10 lower than the demonstrated effect level for amitrole-induced effects of thyroid function; even assuming that amitrole-induced carcinogenesis follows a dose-threshold pattern, use of a tenfold safety margin when the risk involved is related to a disease as serious as cancer cannot be viewed as unreasonable.

Occupational exposure to amitrole has been shown to be associated with an increased incidence of thyroid and pituitary tumors in experimental animals. Although human studies have not demonstrated conclusively that amitrole is carcinogenic, the studies by Axelson and Sundell (1974/Ex. 1-812) and Axelson, Sundell, Andersson et al. (1980/Ex. 1-242) provide evidence that amitrole may increase the risk of cancer among exposed workers. The Agency concludes that the adverse effects resulting from exposure to amitrole constitute material impairment of health and functional capacity. OSHA's risk assessment, based on the animal data, shows that this significant excess cancer risk can be substantially reduced for employees who are currently exposed above the final rule's 0.2-mg/m^3 limit. Therefore, OSHA is establishing a 0.2-mg/m^3 TWA exposure limit for amitrole.

ASPHALT FUMES

CAS: 8052-42-4; Chemical formula: None
H.S. No. 1028

OSHA proposed an 8-hour TWA PEL of 5 mg/m^3 for asphalt fume. The ACGIH has a TLV-TWA of 5 mg/m^3, and NIOSH recommends a ceiling (15 minutes) of 5 mg/m^3 for asphalt fumes.

Extensive evidence was submitted to the record regarding the proposed PEL for asphalt fume. Because of the conflicting nature of some of the evidence and the complexity of the issues raised, OSHA has not yet been able to reach a final conclusion. Therefore, OSHA is temporarily delaying a final decision regarding the establishment of a separate PEL for asphalt fume; however, OSHA will make this final decision in a reasonable period of time.

BERYLLIUM AND COMPOUNDS

CAS No.: 7440-41-7
H.S. No. 1033

OSHA's current limits for beryllium are 0.002 mg/m^3 as an 8-hour TWA, 0.005 mg/m^3 as a 30-minute STEL, and 0.025 mg/m^3 as a ceiling. OSHA is retaining these limits in the final rule. The ACGIH has a TLV-TWA for beryllium of 0.002 mg/m^3. NIOSH (1977o; Baier 1977b/Ex. 1-831) recommends a ceiling limit of 0.5 ug/m^3 (0.0005 mg/m^3). Beryllium is a hard, brittle, gray-white metal.

The ACGIH recommendation is based on human evidence describing nonmalignant respiratory disease and berylliosis associated with exposure to beryllium. Because of the uncertainty regarding the concentrations of beryllium necessary to produce chronic respiratory disease, and because of the serious nature of the disease, the ACGIH set a TLV-TWA of 0.002 mg/m^3.

At the time of publication of NIOSH's criteria document on beryllium (NIOSH 1972a, as cited in ACGIH 1986/Ex. 1-3, p. 56), NIOSH judged the evidence on beryllium-related cancer to be equivocal. In testimony at OSHA's 1977 hearing on a standard for beryllium, however, NIOSH presented additional epidemiologic and animal evidence indicating that beryllium is carcinogenic. In particular, NIOSH (1977o; Baier 1977b/Ex. 1-831) cited the studies of Bayliss and Wagoner (1977) and Mancuso (1977), which showed significant increases in bronchogenic cancer among beryllium-exposed workers. NIOSH therefore recommended at the 1977 hearing that exposure to beryllium not exceed the reliable limit of detection of 0.5 ug/m^3 (NIOSH 1977o; Baier 1977b/Ex. 1-831).

George M. Talley and Michael C. Garcia, Industrial Hygienists for the Los Alamos National Laboratory (Ex. 3-1095), commented that beryllium should not be included in the group of potential carcinogens in this rulemaking because OSHA is not revising its limits for beryllium on the basis of carcinogenicity. As discussed in other sections of this preamble, OSHA's classification of substances in this preamble is not meant to have regulatory implications but to facilitate generic rulemaking. OSHA also notes, as do Mr. Talley and Mr. Garcia, that the ACGIH has designated beryllium as a potential (A2) human carcinogen.

Representatives of the International Chemical Workers Union (Tr. p. 9-217) and the AFL-CIO (Ex. 194) urged OSHA to issue a standard for beryllium based on the NIOSH REL. As explained in the proposal and in Section III of this preamble, however, OSHA has used its priority-setting authority to focus this rulemaking on substances selected for inclusion on the basis of certain decision rules. Beryllium is a substance with an extensive rulemaking history, in that OSHA has previously issued an NPRM and developed an extensive record for beryllium. The Agency determined that, because of this extensive prior history, beryllium presents issues that are too complex for a decision at this time.

The ACGIH TLV of 0.002 mg/m^3 (TWA) was judged in the NPRM to be less stringent than OSHA's existing PELs. NIOSH (Ex. 8-47, Table N6B) did not concur with OSHA's proposal to retain the existing PELs. As OSHA explained in the NPRM, the NIOSH REL is based on analytical and sampling limits of detection, an approach to limit-setting that does not necessarily satisfy OSHA's requirements regarding significant risk and feasibility. In the final rule, OSHA is retaining the Agency's PELs of 0.002 mg/m^3 TWA, 0.005 mg/m^3 as a 30-minute STEL, and 0.025 mg/m^3 as a ceiling for beryllium and compounds.

CARBON TETRACHLORIDE

CAS: 56-23-5; Chemical Formula: CCl_4
H.S. No. 1073

The current OSHA PELs for carbon tetrachloride are 10 ppm as an 8-hour TWA, 25 ppm as a STEL not to be exceeded for more than five minutes every four hours, and 200 ppm as a ceiling. OSHA proposed to revise these limits to a single limit of 2 ppm measured over 60 minutes, based on the NIOSH (1975a/Ex. 1-186) REL. The ACGIH has established a 5-ppm 8-hour TWA limit, with a skin notation, for this substance. Carbon tetrachloride is classified as a probable human carcinogen by EPA (Group B2) and IARC (Group 2B), and as a suspected human carcinogen by the ACGIH (Category A2), based on positive carcinogenicity studies in rats, mice, and hamsters. In the final rule, OSHA is establishing a 2-ppm 8-hour TWA limit for carbon tetrachloride. Carbon tetrachloride is a heavy, mobile liquid with a sweet odor.

In humans, there have been three case reports of liver tumors developing after carbon tetrachloride exposure (Tracy and Sherlock 1968/Ex. 1-152; Johnstone 1948/Ex. 1-817; Simler, Maurer, and Mandard 1964/Ex. 1-225). In each case, the patient has been acutely overexposed to carbon tetracholoride, leading to nausea, stomach pains, and signs of severe liver damage.

Blair, Decoufle, and Grauman (1979/Ex. 1-150) studied causes of death in 330 laundry and dry cleaning workers potentially exposed to carbon tetrachloride, as well as to trichloroethylene and tetrachloroethylene. Causes of death based on death certificates were compared to the age, sex, race, and cause-specific distribution of U.S. deaths from the same time period. The proportionate mortality ratio (PMR) for all malignant neoplasms was 128, which was statistically significant, indicating that the study group had a 28-percent higher proportion of total deaths due to cancer compared with the U.S. general population. The excess cancer deaths were due to liver, lung, and cervical cancer and leukemia. Although the excess lung and cervical cancer may reflect socioeconomic differences among these workers, the excess liver cancer seen in this study is consistent with findings in animal studies on carbon tetrachloride.

In animals, carbon tetrachloride has produced hepatocellular carcinomas in all species evaluated (rats, mice, and hamsters). Male rats were given 47 or 94 mg/kg carbon tetrachloride and females were given 80 or 159 mg/kg by gavage for 78 weeks (NCI 1976a/Ex. 1-119; NCI 1976b/Ex. 1-168; NCI 1977b/Ex. 1-169). The incidence of hepatocellular carcinomas was increased in animals exposed to carbon tetrachloride as compared with pooled colony controls but was statistically significant only for low-dose females. The lower incidence of carcinomas in female rats at the high dose (1/49) compared to the low dose (4/49) was attributed by the authors to the increased lethality that occurred among these rats before tumors could be expressed.

In this same study, mice of both sexes received 1250 or 2500 mg/kg carbon tetrachloride by gavage. Hepatocellular carcinomas were found in 49/49 low-dose and 47/48 high-dose males (compared with 5/77 in the control males) and in 40/40 low-dose and 43/45

high-dose females (compared with 1/80 in the control females) (NCI 1976a/Ex. 1-119; NCI 1976b/Ex. 1-168; NCI 1977b/Ex. 1-169).

Edwards, Heston, and Dalton (1942/Ex. 1-68) administered carbon tetrachloride by gavage (64 mg/mouse administered 46 times over four months) to a mouse strain known to have a low incidence of spontaneous hepatomas. The incidence of hepatomas was 52 percent (28/54) for males and 32 percent (6/19) for females. Previous hepatoma incidence data for untreated mice of this strain were 2/71 for males and 0/81 for females. Carbon tetrachloride administered by gavage has also been shown to produce neoplastic changes in the livers of four additional strains of mice (Andervont 1958/Ex. 1-81; Edwards 1941/ Ex. 1-86; Eschenbrenner and Miller 1943/Ex. 1-113).

Della Porta, Terracini, and Shubik (1961/Ex. 1-136) gave weekly gavage treatments of 10 to 20 ug to hamsters for 30 weeks, and the animals were observed for an additional 25 weeks. All 10 hamsters dying or killed between weeks 43 and 55 had liver cell carcinomas, in comparison with 0/254 in historical controls.

Risk estimate for carbon tetrachloride. Three data sets have sufficient dose-response information to allow quantitative risk estimation: the rat and mouse bioassay data (NCI 1976a/Ex. 1-119; NCI 1976b/Ex. 1-168; NCI 1977b/Ex. 1-169) and the Edwards, Heston, and Dalton (1942/Ex. 1-68) mouse data. To increase sample sizes, the data were pooled for male and female animals in each of the three studies. (In the NPRM, OSHA erroneously indicated that four data sets were pooled; see Ex. 110.) The estimated risk presented in Table C15-5 is the geometric mean of the risk calculated from each of the three data sets.

TABLE C15-5. Multistage Model Estimates of Cancer Risk Associated with Working Lifetime Exposure to Carbon Tetrachloride

Exposure Level	Excess Cancer Deaths per 1,000 Workers	
	MLE	UCL
2 ppm[a]	3.7	5.2
5 ppm[b]	9.2	13.0
10 ppm[c]	17.9	26.0

[a] Revised OSHA PEL.
[b] ACGIH TLV.
[c] Former OSHA TWA PEL.
MLE = Maximum likelihood estimate of risk.
UCL = 95-percent upper-confidence limit on the maximum likelihood estimate of risk.

Inhalation risk was calculated assuming an air intake of 20 m^3 per 24-hour day and a 40-percent absorption rate for humans (EPA 1984a/Ex. 1-1130). All four studies suggest that a common biological mechanism, cell death and regeneration, occurs and leads to the development of the same tumor type.

Table C15-5 presents the estimates of lifetime human risk from carbon tetrachloride exposure, calculated by the linearized multistage model (GLOBAL83), at the final rule's 2-ppm limit, the ACGIH limit of 5 ppm, and the former 10-ppm OSHA PEL. Both the maximum likelihood estimates (MLE) and the (95-percent upper-confidence limits of human risk are given, as well as the corresponding expected number of excess cancer deaths per 1,000 workers exposed over a working lifetime.

Based on this risk estimate, the MLE at the former OSHA limit of 10 ppm is 17.9 excess deaths per 1,000 exposed workers, clearly indicating that a significant cancer risk exists at the former PEL.

Risk at the current ACGIH limit of 5 ppm is estimated to be 9.2 excess deaths per 1,000 workers exposed over their working lifetimes. At the final rule's limit of 2 ppm, residual risk continues to be significant, according to the Supreme Court's guidance in the *Benzene* decision and the analysis presented in the introduction to this section; the risk predicted at 2 ppm is 3.7 excess deaths per 1,000 workers exposed over their working lifetimes. However, risk at the 2-ppm limit is substantially reduced compared with risk at the former OSHA PEL of 10 ppm. The estimate shows that approximately 14 cancer deaths per 1,000 workers would potentially be avoided over a lifetime by reducing the limit to 2 ppm.

Both NIOSH (Ex. 193) and the AFL-CIO (Ex. 194) supported OSHA's proposed 2-ppm 60-minute ceiling PEL and believed that carbon tetrachloride should be designated as a potential carcinogen. The Dow Chemical Company (Ex. 3-741), however, stated that, for a number of reasons, it believes that the cancer risk from exposure to carbon tetrachloride has been overestimated. First, Dow argues that carbon tetrachloride enhances the occurrence of naturally forming liver tumors by causing increased cell death and turnover. Because clear threshold effect levels have been demonstrated for liver toxicity, Dow believes that a threshold-type response would be expected for carcinogenic effects "since liver toxicity appears to be a precursor to carcinogenic activity" (Ex. 3-741, p. 34). The Halogenated Solvents Industry Alliance (Ex. 8-89) also expressed the opinion that OSHA overstated the potential cancer risk by using a linear, threshold model. Dow (Ex. 3-741) concludes that a level of 50 ppm (the threshold for liver toxicity observed in six-month inhalation studies in monkeys) represents the threshold concentration for human toxicity and carcinogenicity from exposure to carbon tetrachloride.

In support of its position, Dow included a review of toxicity data by J.M. Norris of Dow Chemical (Ex. 3-741, Appendix A). Mr. Norris cites studies that suggest that species sensitivity to liver toxicity is related to cytochrome P-450 content in liver and that rodents have greater unit P-450 activity and are more sensitive to carbon tetrachloride-induced liver toxicity than are Rhesus monkeys. Since the unit P-450 activity of Rhesus monkeys is comparable to that of humans, Mr. Norris concludes that "the monkey may be the appropriate animal for extrapolation to man" (Ex. 3-741, Appendix A, p. 10).

After reviewing the evidence presented by Mr. Norris, OSHA is unpersuaded that the 50-ppm no-effect level observed in monkeys should be used to establish a PEL to protect workers from the significant cancer risk associated with exposure to carbon tetrachloride. The monkey data cited by Dow and Mr. Norris are results from a study of only six months' duration, and only one or two monkeys were tested at dose levels near the no-observed-effect level. Mr. Norris acknowledged that these limitations warrant the use of a safety factor to derive an adequate exposure limit; applying an appropriate safety factor to the 50-ppm NOEL would yield a PEL no higher, and perhaps well below, the final rule's 2-ppm limit, given the seriousness of the toxicologic endpoint (carcinogenicity). OSHA concludes that the approach it has used to assess cancer risk (i.e., combining

data from several animal studies to estimate risk with a widely used dose-response model) provides better information on which to base a revised PEL than do the results of a single short-term, small-sample animal study.

Based on the evidence presented above and the quantitative estimates of carbon tetrachloride-related cancer risk, OSHA concludes that occupational exposure to carbon tetrachloride at the former 10-ppm PEL presents a significant risk of cancer to workers (13.9 cancer deaths per 1,000 workers). OSHA's risk assessment shows that reducing this limit to 2 ppm will substantially reduce this risk (3.7 deaths per 1,000 workers). The Agency concludes that cancer represents a material impairment of health and functional capacity. Accordingly, OSHA is revising its limits for carbon tetrachloride to a single limit of 2 ppm; however, in the final rule, OSHA is establishing this limit as an 8-hour TWA. OSHA has determined that a TWA limit is more appropriate for carbon tetrachloride since low-level exposure to carbon tetrachloride presents a chronic, rather than an acute, health hazard. OSHA also believes that establishing a TWA limit will simplify the development of compliance and exposure monitoring strategies for employers, since an 8-hour TWA limit is more conventional than a 60-minute limit. Therefore, in the final rule, OSHA is establishing a 2-ppm 8-hour TWA PEL for carbon tetrachloride.

CHLOROFORM

CAS: 67-66-3; Chemical Formula: $CHCl_3$
H.S. No. 1086

The former OSHA PEL for chloroform was 50 ppm as a ceiling limit. OSHA proposed to revise this limit to 2 ppm, measured over a 60-minute period. This limit was based on the NIOSH (1977p, as cited in ACGIH 1986/Ex. 1-3, p. 130) REL, and NIOSH (Ex. 8-47, Table N6A) has indicated its concurrence with the proposed limit. In the final rule, OSHA is establishing a 2-ppm limit for chloroform, but is expressing this PEL as an 8-hour TWA limit. The ACGIH has established a TLV-TWA of 10 ppm and assigned chloroform an A2 designation. Chloroform is a clear, colorless, nonflammable, volatile liquid with a pleasant odor.

Chloroform is considered by the ACGIH, the United States Environmental Protection Agency (EPA), and the International Agency for Research on Cancer (IARC) as a probable carcinogen in humans. Chloroform is given an overall weight-of-evidence classification of B2 by the EPA and a classification of 2B by IARC. These classifications are based on these organization's determination that there is sufficient evidence for the carcinogenicity of chloroform in animals and insufficient evidence in humans. The following discussion is based on information from the EPA *Health Assessment Document for Chloroform* (EPA 1984f/Ex.1-216)./

It is currently believed that the carcinogenicity of chloroform results from the formation of reactive metabolites, such as phosgene, that bind to cellular macromolecules. Although there is some evidence to suggest that chloroform is weakly mutagenic, the results of most mutagenicity tests are negative.

In humans, there are no epidemiological studies that evaluate populations exposed only to chloroform, although there are several studies that examine populations exposed to chloroform in chlorinated drinking water. However, because chloroform is not the only potential carcinogen present in chlorinated water, the epidemiological data are considered inadequate to use as the basis for a quantitative risk assessment. Thus, a causal relationship between cancer and chloroform exposure cannot be determined based on epidemiological studies alone, although these studies can be used to provide general support for findings in animal studies.

A case-controlled study indicated a significant association between colon cancer and exposure to chlorinated drinking water contaminated with organic material (Young, Kanarek, and Tsiatis 1981/Ex. 1-118). Significant positive associations were also found for chloroform levels in drinking water and the incidence of morality due to cancer of the bladder, rectum, and large intestine (Hogan, Chi, Hoel, and Mitchell 1979/Ex. 1-159). Similar results also have been found by others (Cantor, Hoover, Mason, and McCabe 1978/Ex.1-50; and Gottlieb, Carr, and Morris 1981/Ex. 1-72). However, although these studies suggest an association between exposure to chloroform and an increased risk of cancer, a definite causal relationship between the development of colon and bladder cancer and exposure to chloroform cannot be determined solely from these studies.

In animals, several long-term studies provide strong evidence for the carcinogenic activity of chloroform. Chloroform has been shown to produce statistically significant increases in renal epithelial tumors in male rats and hepatocelluluar carcinomas in several strains of mice. The carcinogenic activity of chloroform in these studies is specific to the kidney and liver.

The carinogenic activity of chloroform was investigated in rates exposed to chloroform by gavage for 78 weeks (NCI 1976a/Ex. 1-119). Male rats were administered doses of 90 or 180 mg/kg/day, and female rats were administered doses of 100 or 200 mg/kg/day. A statistically significant dose-related increase in renal epithelial tumors was observed in treated male rats compared with untreated, matched controls; these tumors were described as carcinomas and adenomas. No increase in the incidence of tumors was observed in chloroform-treated female rats.

In this same study, the carcinogenicity of chloroform was evaluated in mice exposed chronically to chloroform by gavage (NCI 1976a/Ex. 1-119). Male mice were exposed to doses of 138 or 277 mg/kg/day and females to 238 or 477 mg/kg/day for 78 weeks. There were significant dose-related increases in the incidence of hepatocellular carcinomas in chloroform-treated male and female mice. The increase of tumors in male mice for low and high doses was 36 percent and 98 percent, respectively. For female mice, the increases were 80 percent for the low dose and 95 percent for the high dose of chloroform.

The carcinogenic potential of chloroform in mice was further investigated in two additional studies (Roe, Palmer, and Worden 1979/Ex. 1-108; Jorgenson, Meierhenry, Rushbrook et al. 1985/Ex. 1-117). Doses of 17, 60, or 100 mg/kg/day were administered to four different strains of male and female mice (C57BL, CBA, CF/1, and ICI) by gavage for 80 weeks (Roe, Palmer, and Worden 1979/Ex.1-108). The incidence of kidney tumors, described as hypernephromas, was significantly elevated in the ICI strains. Moderate to severe renal changes were observed in the male mice of the other strains, but no significant increase in renal tumors was reported. Tumors were not observed in female mice.

The carcinogenicity of chloroform administered in drinking water was investigated in male rats and female mice (Jorgenson, Meierhenry, Rushbrook et al. 1985/Ex. 1-117). Animals were treated with drinking water containing chloroform concentrations of 200, 400, 900, or 1800 mg/L for 104 weeks. There was a marked increase in the number of kidney tumors (described as tubular cell adenomas and adenocarcinomas) in rats. However, the incidence of tumors in female mice was not signficantly increased.

Risk estimate for chloroform. The Jorgenson et al. (1985/Ex. 1-117) rat

study, which demonstrated a statistically significant increase in the incidence of renal tumors in male rats, was the data set used for the quantitative risk estimation. (In the NPRM, OSHA inadvertently identified the NCI (1976a/Ex. 1-119) study as forming the basis for risk assessment; see Ex. 110.) Although there are no data concerning the carcinogenicity of chloroform following inhalation exposure, the risk from inhaled chloroform is considered to be equivalent to the risk from ingested chloroform. The linearized multistage, one hit, and Weibull models were used. The maximum likelihood estimates of excess cancers over an occupational lifetime for a population of 1,000 and the 95-percent upper-bound estimates are summarized in Table C15/6. The Weibull model is similar to the logit and probit models. However, by using only one data set, the logit, probit, and multihit models failed to converge.

Table C15 6. Multistage Model Estimates of Cancer Risk Associated with Working Lifetime Exposure to Chloroform

Exposure Level	Excess Cancer Deaths Per 1,000 Workers	
	MLE	UCL
Multistage		
2 ppm[a]	0.27	1.80
10 ppm[b]	1.90	9.00
50 ppm[c]	22.40	46.10
One Hit		
2 ppm[a]	1.40	2.20
10 ppm[b]	7.00	11.10
50 ppm[c]	34.50	54.20
Weibull		
2 ppm[a]	0.11	0.60
10 ppm[b]	1.60	6.30
50 ppm[c]	24.50	51.30

[a] Final rule PEL.
[b] ACGIH TLV.
[c] Former OSHA PEL.
MLE = Maximum likelihood estimate of risk.
UCL = 95-percent upper-confidence limit on the maximum likelihood estimate of risk.

The results of the data analysis presented here are similar to the results of other models described by the EPA (1984f/Ex. 1-216) for chloroform. These three models clearly demonstrate, based on the MLE estimates, that a significant cancer risk exists at the former PEL of 50 ppm. The risks estimated to exist at the former PEL are of the same order of magnitude as the risks determined by OSHA to be associated with other carcinogens that OSHA has regulated (e.g., benzene, ethylene oxide). Some commenters (Exs. 3-685, 3-741, 3-958, 8-89, and L3-1262) stated that OSHA's risk assessment approach for chloroform overstated the risk by not accounting for certain aspects of the mechanism by which chloroform induces cancer. Dow Chemical (Ex. 3-741, p. 45), Hoffmann-LaRoche (Ex. L3-1262), and the American Paper Institute (Ex. 3-685) presented evidence that the mouse liver tumors resulting from chloroform exposure arise secondarily to organ toxicity, which is a threshold phenomenon. As such, they argue that the use of linearized, non-threshold model will overstate cancer risk. Theodore J. Berger, Assistant Vice President and Director of Corporate Environmental and Safety Affairs at Hoffmann-LaRoche, pointed out that the

ACIGH TLV of 10 ppm for chloroform was based on this consideration, and that the 10-ppm level was one-fifth the level at which organ injury has been observed (in rats).

On the issue of the carcinogenic mechanism of chloroform, rulemaking participants (Exs. 3–685, 3–341, and L3–1262) and the EPA (1984f/Ex. 1–216) cite evidence that suggest that increased cell death brought about by the formation of reactive metabolities may be one mechanism by which chloroform has caused cancer in animals, particularly in the liver. The EPA (1984f/Ex. 1–216) also cites evidence that chloroform metabolites may deplete glutahione, which results in less effective cellular detoxification. In addition, a genotoxic mechanism cannot be entirely ruled out, although the data are equivocal; chloroform has produced positive results in the micronucleus test and host-mediated mutagenicity assay in the mouse; mutations in yeast; abnormal sperm morphology in mice; and sister chromatid exchange in human lymphocytes and mouse marrow (EPA 1984f/Ex. 1–216).

Richard Bull commented on EPA's 1984 *Health Assessment Document on Chloroform* (1984f/Ex. 1–216) on behalf of the American Paper Institute (Ex. 3–685, Appedix B). Although Dr. Bull concluded that chloroform produces liver tumors in mice by causing organ toxicity and cell damage, he also states that this mechanism did not necessarily explain the kidney tumors observed in rats:

> [T]here is strong evidence that chloroform acts by producing cell damage in the already initiated liver of B6C3F1 mice. There is a similar case to the made in terms of renal tumors in ICI mice. A less convincing case can be made in the Osborne-Mendel rat, since there is no data to indicate a relationship between renal damage and carcinogenic response in this strain. In addition, there is no evidence that there are spontaneously initiated cells in the kidney of this strain of rat. The Jorgenson et al. (1985/Ex.1–117) study indicated that the spontaneous rate for renal tubular adenomas and adenocarcinomas was 4/301 animals or slightly more than 1 percent (Ex. 3–685, Appendix B, p. 8).

Dr. Bull also stated that an "an acceptable conservative approach [for assessing cancer risk for chloroform] would be to utilize the multistage model on the development of renal tumors in the rat because the case has yet to be made that nongenotoxic mechanisms may have been involved in the induction of these tumors * * *" (Ex. 3–685, Appendix B; Ex. 1–0000, p. 8). Thus, based on the evidence presented by EPA (1984f/Ex. 1–216) and the comments on EPA's document by Dr. Bull, OSHA concludes that its use of the Jorgenson et al. (1985/Ex. 1–117) rat kidney data and multistage model is a reasonable approach for estimating the risk of cancer associated with exposure to chloroform. Furthermore, OSHA concludes that, even if one were to accept both that chloroform increases cancer risk via a cell-death mechanism, and that a threshold does for this effect exists, the 10-ppm TLV recommended by the ACIGH provides an inadequate margin (fivefold) of protection against this life-threatening disease.

Dow Chemical Company (Ex. 3–741) and the American Paper Institute (Ex. 3–685) also commented that, because humans metabolize chloroform to a lesser degree than do rodents, quantitative risk assessments should consider such differences. Dow submitted a discussion (Ex. 3–958) of the preliminary results of an assessment based on the use of a physiologically based pharmacokinetic model (PB–PK) similar to that developed by Andersen et al. (1987) for methylene chloride. In this assessment, the researchers reported that the estimated cancer risk for chloroform was one to two orders of magnitude lower than the risks estimated using the multistage model. However, since this work is currently underway, details of the assessment are not available.

Dow Chemical also applied EPA's (1984f/Ex. 1–216) approach to the rat data from the Jorgenson et al. (1985/Ex. 1–117) study (Ex. 3–741, pp. 45–47). This approach uses metabolic data to express the active dose in units of average mg metabolite produced per day per liter of tissue; this method contrasts with OSHA's approach of using applied dose for the risk assessment. Dow's MLE estimate of lifetime occupational cancer risk associated with exposure to 2 ppm is 0.17 deaths per 1,000 workers (upper-confidence limit of 0.46/1,000), based on the amount of chloroform metabolized per unit volume of kidney tissue. The estimate based on chloroform metabolism in the liver is 0.27/1,000 (upper-confidence limit of 0.74/1,000). OSHA does not believe that these estimates, which account for interspecies differences in chloroform metabolism, are substantially different from OSHA's estimates, which are based on the use of applied dose; Dow's MLE estimate based on metabolism in the kidney is not quite half of OSHA's MLE estimate, and Dow's MLE estimate based on liver metabolism is the same as OSHA's. These findings give OSHA greater confidence in the estimates of chloroform-related cancer risk presented in Table C15–6 above.

The AFL–CIO (Ex. 194) supported OSHA's proposed PEL for chloroform. However, the New Jersey Department of Public Health (Ex. 144) urged OSHA to set a limit for chloroform based on EPA's IRIS data. The use of such an approach for setting exposure limits is discussed in Section VI.A of the preamble.

Based on the evidence presented above, OSHA concludes that a significant risk of cancer, which OSHA considers a material impairment of health and functional capacity, exists at the former PEL of 50 ppm, with estimated risks ranging from 22 to 34 excess deaths per 1,000 workers. The Supreme Court indicates that a reasonable person "might well consider a risk of 1.0 per 1,000 significant, and take steps to decrease or eliminate that risk" (*I.U.D.* v. *A.P.I.*, 448 U.S. 655) (see the discussion in Section VI.A of this preamble). Based on OSHA's risk assessment, significant risk of cancer remains at the ACGIH TLV of 10 ppm (1.6 deaths per 1,000 workers). OSHA also finds that revising the PEL to 2 ppm will substantially reduce this risk by from 96 to 99 percent. Therefore, OSHA is establishing a 2-ppm limit as the PEL for chloroform. However, in the final rule, OSHA is establishing this limit as an 8-hour TWA limit, rather than a 60-minute limit as proposed. OSHA has determined that a TWA limit is more appropriate for chloroform since low-level exposure to chloroform presents a chronic, rather than acute, health hazard. OSHA also believes that establishing a TWA limit will simplify the development of compliance and exposure-monitoring strategies for employers, since an 8-hour TWA limit is more conventional than a 60-minute limit. Therefore, in the final rule, OSHA is establishing a 2-ppm 8-hour TWA PEL for chloroform.

CHROMIC ACID, CHROMATES; ZINC CHROMATES

CAS: Varies with compound
H.S. No. 1092; 1436

The current OSHA limit for chromic acid and chromates is a ceiling limit of 0.1 mg/m^3 measured as CrO_3. The Agency did not propose to revise this limit. The ACGIH has established a TLV–TWA of 0.05 mg/m^3 as Cr(VI) for both the soluble and insoluble forms of chromate (except zinc chromate), and has designated insoluble chromates as confirmed human carcinogens (A1). (It should be noted that the 0.05-mg/m^3 limit, expressed as Cr(VI), approximates 0.01 mg/m^3 measured as CrO.) NIOSH (1975b/Ex. 1–258) has recommended that exposure to the noncarcinogenic

forms of chromium (VI) be limited to 0.025 mg Cr(VI)/m^3 as a 10-hour TWA and 0.05 mg Cr(VI)/m^3 as a 15-minute ceiling. For the carcinogenic (i.e., insoluble) forms of chromium (VI), NIOSH recommends a 10-hour TWA limit of 0.001 mg Cr(VI)/m^3.

The ACGIH recommendation for both soluble (noncarcinogenic) and insoluble (carcinogenic) forms of Cr(VI) is based largely on reports by Bloomfield and Blum (1928/Ex. 1–822) and by the Federal Security Agency of the U.S. Public Health Service (Federal Security Agency 1953, as cited in ACGIH 1986/Ex. 1–3, p. 140) that demonstrate nasal irritation and some evidence of liver enlargement and kidney dysfunction among chromate workers exposed to 0.06 to 0.07 mg Cr(VI)/m^3. The ACGIH also cites a report by Mancuso and Hueper (1951/Ex. 1–215) of excess lung cancer among chromate workers exposed to 0.01 to 0.15 mg/m^3 soluble chromate and 0.1 to 0.58 mg/m^3 insoluble chromate. Animal data cited by the ACGIH indicate that insoluble chromate salts were likely to have been responsible for the increased incidence of cancer seen in the Mancuso and Hueper (1951/Ex. 1–215) study. The ACGIH (1986/Ex. 1–3) concluded that the 0.05-mg/m^3 TLV–TWA would protect workers from chromium-induced nasal irritation and possible liver or kidney damage, and, in the case of the insoluble chromates, would provide an adequate margin of safety from respiratory cancer.

NIOSH (1975a/Ex. 1–185) cited several studies showing inflammation and ulceration of the nasal cavity at short-term exposure levels greater than 0.1 mg CrO_3/m^3. In its criteria document on chromic acid (NIOSH 1973e/Ex. 1–264), NIOSH recommended that the current OSHA ceiling limit (0.1 mg CrO_3/m^3) be supplemented with a 0.05-mg CrO_3/m^3 10-hour TWA limit. In its criteria document on chromium (VI), NIOSH (1975b/Ex. 1–258) reaffirmed these limits but extended their application to all forms of noncarcinogenic chromate. Thus, the 0.1-mg CrO_3/m^3 ceiling limit corresponds to a 0.05-mg Cr(VI)/m^3 ceiling limit, and the 0.05-mg CrO_3/m^3 TWA limit corresponds to a 0.025-mg Cr(VI)/m^3 TWA. For the carcinogenic (insoluble) forms of Cr(VI), NIOSH recommends the lowest detectable level, which is 0.001 mg Cr(VI)/m^3 as a 10-hour TWA.

Zinc chromate is an insoluble, carcinogenic form of chromate. Accordingly, the current OSHA limit for chromic acid and chromates applies, as does the NIOSH limit of 0.001 mg/m^3 for carcinogenic chromates. The ACGIH (1986/Ex. 1–3) reviewed several small epidemiologic studies of zinc chromate workers, all of which reported excesses of lung cancer. Because of the consistency of this evidence, the ACGIH (1986/Ex. 1–3) classified zinc chromate as a confirmed human carcinogen (A1) and reduced the TLV to 0.05 mg Cr(VI)m^3. xzx

Evaluation of the alternate recommendations is complicated by the different valence states of chromium compounds, the different methods of measurement (CrO_3 or Cr(VI)), and differences in defining these substances that present a cancer hazard (soluble vs. insoluble or valence state). The 0.05-ppm TWA–TLV is less restrictive than the current 0.05-ppm ceiling limit (as Cr(VI)), and would not be considered a revised PEL. In the NPRM, OSHA proposed that the existing PEL of 0.1 mg/m^3 (measured as CrO_3) be maintained. In the NPRM OSHA stated that it would consider whether to place these substances on its regulatory agenda for future consideration for section 6(b) rulemaking, rather than making any changes as part of this rulemaking. NIOSH (Ex. 8–47, Table N6B) concurred that the chromates should be part of a separate 6(b) rulemaking, and both the AFL–CIO (Ex. 194) and the UAW (Tr pp. 7–65 to 7–67) agreed that a comprehensive standard would be appropriate for chromates. No other comments were received on this issue. Therefore, because of the complexities of the scientific issues regarding the carcinogenicity of the various forms of chromates, OSHA is not at this time revising its current PEL for chromic acid or chromates, but will continue to evaluate the need for a separate 6(b) rulemaking for these substances.

CHROMYL CHLORIDE

CAS: 14977–61–8; Chemical Formula: CrO_2Cl_2 H.S. No. 1094

There is no existing OSHA PEL for chromyl chloride. The ACGIH recommended that a TWA of 0.025 ppm be established, based on this substance's carcinogenic potential (ACGIH 1986/Ex. 1–3, p. 141). The evidence in humans is considered sufficient for the carcinogenicity of chromium and chromium compounds, and these have been given a Group 1 classification by the International Agency for Research on Cancer (IARC). As discussed above in connection with chromic acid, chromates, and zinc chromates, the chromium compounds present several important issues that require detailed analysis and can most appropriately be handled in an individual section 6(b) rulemaking. NIOSH concurred with this approach (Ex. 8–47, Table N6B), and no other comments on chromyl chloride were received. OSHA intends to commence work on this rulemaking as priorities permit.

DIMETHYL SULFATE

CAS: 77–78–1; Chemical Formula: $(CH_3)_2SO_4$ H.S. No. 1142

OSHA's former limit for dimethyl sulfate was 1 ppm TWA, with a skin notation. The ACGIH considers this substance a suspected human carcinogen and has given it a classification of A2 (ACGIH 1986/Ex. 1–3, p. 212). The ACGIH's TLV–TWA for this substance is 0.1 ppm with a skin notation. OSHA proposed, and the final rule establishes, a 0.1-ppm TWA PEL, with a skin notation, for dimethyl sulfate, which is an oily, colorless liquid with a faint, onion-like odor. NIOSH (Ex. 8–47, Table N6A) concurs with the selection of this limit and considers dimethyl sulfate to be a potential human carcinogen.

Dimethyl sulfate is commonly used in the manufacture of many organic chemicals. It has been shown to be carcinogenic in rats by inhalation exposure, subcutaneous injection, and prenatal exposure. The rat is the only animal species in which the carcinogensis of dimethyl sulfate has been tested (IARC 1982c, as cited in ACGIH 1986/Ex. 1–3, Appendix A).

The carcinogenic activity of dimethyl sulfate was investigated in male rats chronically exposed to subcutaneous injections of 8 or 16 mg/kg body weight per week (Druckrey, Preussman, Nashed, and Ivanovic 1966/Ex. 1–245). Local sarcomas with metastases to the lung and regional lymph nodes were observed at both dose levels. A single subcutaneous injection of dimethyl sulfate (50 mg/kg) also produced local sarcomas with metastases to the lung (Druckrey, Kruse, Preussman et al. 1970/Ex. 1–246). However, tumors did not develop following chronic weekly intravenous injections of dimethyl sulfate (2 or 4 mg/kg) (Druckrey, Kruse, Preussman et al. 1970/Ex. 1–246). Control data were not reported for either of these studies.

The carcinogenic potential of dimethyl sulfate exposure by inhalation was also evaluated in male rats (Druckrey, Kruse, Preussman et al 1970/Ex. 1–246). Animals were exposed to approximately 3 or 10 ppm dimethyl sulfate for one hour per day, five times weekly, for 130 days. Malignant tumors developed in 15 percent (3/20) of the rats exposed at 3 ppm and in 18 percent (5/27) of the rats exposed at 10 ppm.

Pregnant rats were exposed to a single intravenous injection of dimethyl sulfate (20 mg/kg body weight) on day 15 of gestation and the incidence of malignant tumors in the offspring was investigated for one year. Tumors were reported in 7/59 of the offspring. However, the incidence of tumors in the control group was not indicated. The results of this study are complicated because several rats died (number of deaths not specified) from the acute toxic effects of dimethyl sulfate, and the incidence of tumors in the control group was not reported.

There is little information available regarding the carcinogenicity of dimethyl sulfate in humans. A case study of workers exposed to dimethyl sulfate reported that three workers developed bronchial cancer (Druckrey, Preussman, Nashed, and Ivanovic 1966/Ex. 1–245). However, an epidemiological study by the E.I. du Pont de Nemours Company (1975, as cited in ACGIH 1986/Ex. 1–3, p. 213) demonstrated no increase in the incidence of respiratory cancer among workers exposed to dimethyl sulfate.

OSHA considered the possibility of performing a quantitative risk assessment for dimethyl sulfate and concluded that the studies described above did not have sufficient dose-response data to provide an adequate basis for such a risk assessment (see Ex. 85). Dimethyl sulfate induces malignant tumors in animals both by inhalation and ingestion, and there is thus sufficient evidence in animals to predict that workers exposed to dimethyl sulfate are at significant risk of developing cancer, which OSHA considers to be a material impairment of health; exposures at levels only three times the former PEL (1 ppm) resulted in a significant number of tumors. No comments, other than those from NIOSH were received on dimethyl sulfate. OSHA concludes that reducing the former limit to 0.1 ppm as an 8-hour TWA with a skin notation will substantially reduce the significant risk of cancer mortality associated with exposure to dimethyl sulfate.

2-NITROPROPANE

CAS: 79–46–9; Chemical Formula: $CH_3CH(NO_2)CH_3$
H.S. No. 1291

OSHA's former limit for 2-nitropropane (2-NP) was 25 ppm; OSHA proposed a limit of 10 ppm as an 8-hour TWA, and the final rule establishes this limit. The ACGIH classifies 2-nitropropane as a suspected human carcinogen (A2). NIOSH recommends that 2-nitropropane exposure be reduced to the lowest feasible limit. 2-Nitropropane is used as a chemical intermediate, a solvent, and a component in paint, ink, and varnishes (Fiala, Czenniak, Castonguay et al. 1987/Ex. 1–248). 2-Nitropropane is a colorless liquid.

In rats and chimpanzees, 2-NP is metabolized by microsomal enzymes in the liver to acetone, low levels of isopropanol, and nitrite (Mueller, Coulston, and Korte 1983/Ex. 1–247). Methemoglobin formation is associated with the metabolism of nitropropane and has been reported in cats exposed to 280 ppm of 2-NP for seven hours. Sensitivity to the toxic effects of 2-NP in animals varies by species (Dequidt, Vasseur, and Potencier 1972/Ex. 1–813; ACGIH 1986/Ex. 1–3, p. 441).

The mechanisms of carcinogenicity of 2-NP are thought to involve the release of nitrite and the formation of a reactive azoxy intermediate that can react with cellular macromolecules (Williams and Weisburger 1986/Ex. 1–65).

In mutagenicity tests, 2-NP increased the frequency of mutations in all strains of *Salmonella typhimurium* with and without metabolic activation. Positive mutagenicity results were reported in *Salmonella typhimurium* strains TA100, TA1535, and TA98 by Lofroth, Nilsson, and Anderson (1981, as cited in ACGIH 1986/Ex. 1–3, p. 441) and Speck, Meyer, Zeiger, and Rosenkranz (1982/Ex. 1–290). 2-NP was not shown to be mutagenic in the mouse micronucleus test (Hite and Skaggs 1979/Ex. 1–280).

Acute exposures to 2-NP from occupational accidents have been reported to cause severe liver toxicity and subsequent death in humans (ACGIH 1986/Ex. 1–3, p. 441). However, the available epidemiology data on the chronic health effects of occupational exposure to 2-NP do not contain sufficient dose-response data to use as a basis for quantitative risk estimation. An unpublished retrospective mortality study of 1,481 potentially exposed workers from a nitropropane production plant found no increase in liver cancer or liver disease mortality. However, lack of exposure data, the small number of workers with long exposures (greater than 15 years), and a short latency period make interpretation of the results of this study difficult (Miller and Temple 1979, and Bolender 1983, both as cited in ACGIH 1986/Ex. 1–3, p. 441).

There are two studies that report high incidences of liver tumors in male rats exposed to 2-NP by gavage and inhalation. Fiala et al. (1987/Ex. 1–248) administered, by gavage, 1 mmol/kg body weight (approximately 27 mg per treatment per 300-gram rat) of 2-NP in a 10-percent aqueous Emulphor EL–620 vehicle to male Sprague-Dawley rats three times weekly for 16 weeks. Dosing was discontinued after 16 weeks because of excessive mortality in the treated rats. Seventy-seven weeks from the first treatment, the surviving rats were sacrificed and subjected to necropsy. All (100 percent) of the treated rats examined had developed hepatocarcinomas (Fiala, Czenniak, Castonguay et al. 1987/Ex. 1–248).

The results of the Fiala et al. (1987/Ex. 1–248) study support the earlier positive results reported by Lewis, Ulrich, and Busey (1979/Ex. 1–826). In the Lewis et al. (1979/Ex. 1–826) study, male Sprague-Dawley rats and male New Zealand White rabbits were exposed via inhalation to 27 ppm or 207 ppm of 2-NP for seven hours/day, five days/week for six months. At the end of six months, all 10 rats in the high-dose group exhibited hepatocellular carcinomas and neoplastic nodules. No exposure-related lesions were seen in the rats exposed to 27 ppm, and no exposure-related lesions were observed in any of the rabbits.

One high-dose and two low-dose studies reported negative results for rats exposed to 2-NP vapors. Griffin, Benitz, Coulston, and Rosenblum (1978/Ex. 1–243) reported no hepatic carcinomas in male and female rats exposed to 200 ppm of 2-NP by inhalation using a protocol similar to that described by Lewis et al. (1979/Ex. 1–826). Although no hepatic carcinomas were observed, the following effects (generally occurring more extensively in males) were seen: Increased liver weights (both sexes); hepatic nodules; hepatocellular necrosis; and peripheral compression.

Two low-dose studies (Griffin, Coulston, and Stein 1980/Ex. 1–268; Griffin, Stein, and Coulston 1981/Ex. 1–279) also produced negative results. Male and female Sprague-Dawley rats were exposed by inhalation to 25 ppm of 2-NP for seven hours/day, five days/week for 22 months. No pathological changes associated with exposure to 2-NP were seen.

Although the results of both the Lewis et al. (1979/Ex. 1–826) and the Fiala et al. (1987/Ex. 1–248) studies show statistically significant increases in liver carcinomas, neither study provides sufficient dose-response information to use as a basis to quantify the excess cancer risk to humans exposed to 2-NP. Both studies were terminated before the natural lifetime expectancy of the controls, so it is not possible to determine a background incidence of cancer risk. No historical information is provided on tumor incidence for these animals.

2-Nitropropane produced a high incidence of liver tumors in male rats by two routes of administration: inhalation and ingestion. Its ability to cause mutations in *Salmonella typhimurium* further supports the premise that 2-NP is a potential human carcinogen. OSHA considered whether to perform a quantitative risk assessment on 2-NP, and concludes that the studies described above do not contain sufficient dose-response data to use as the basis for quantitative risk estimation using standardized risk assessment models. However, two studies (Fiala, Czenniak, Castonguay et al. 1987/Ex. 1–248; Lewis, Ulrich, and Busey 1979/Ex. 1–826) demonstrate that exposure to 2-NP, either by gavage or inhalation, produced hepatocarcinomas in rats. In addition, this substance produced positive results in two mutagenic assays (Lofroth, Nilsson, and Andersson 1981, as cited in ACGIH 1986/Ex. 1–3, p. 441; Speck, Meyer, Zeiger, and Rosenkranz 1982/Ex. 1–290). NIOSH (Ex. 8–47, Table N6B) was of the opinion that this evidence warranted a separate 6(b) rulemaking.

OSHA is establishing an 8-hour PEL for 2-NP of 10 ppm. The Agency concludes that a reduction in the PEL is necessary to protect exposed workers from the significant risk of cancer potentially associated with exposure to 2-NP at the former PEL. The Agency has also concluded that the effects associated with exposure to 2-NP constitute material impairments of health.

PERCHLOROETHYLENE (TETRACHLOROETHYLENE)

CAS: 127–18–4; Chemical Formula: CCl_2–CCl_2 H.S. No. 1308

OSHA's former permissible exposure limits for perchloroethylene (tetrachloroethylene) were 100 ppm as an 8-hour TWA, 200 ppm as a STEL not to be exceeded for more than five minutes in any three-hour period, and 300 ppm as a ceiling. On the basis of the chemical's narcotic effects in humans, the Agency proposed a revised PEL of 50 ppm TWA and a 15-minute STEL of 200 ppm for perchloroethylene; these are the limits recommended by the ACGIH (ACGIH 1986/Ex. 1–3, p. 464). NIOSH (Ex. 8–47, Table N6B) did not concur with the proposed limits and recommended that exposures be maintained at the lowest feasible limit and that this chemical be classified as a potential occupational carcinogen. OSHA has evaluated the health evidence for this substance and has determined that a further reduction in the PEL to 25 ppm as a TWA is warranted, and the Agency is establishing this limit in the final rule.

Perchloroethylene is a clear, colorless, nonflammable liquid with an etheral odor.

Perchloroethylene is widely used as a solvent in the dry cleaning industry and in industrial degreasing operations. The narcotic effects associated with exposure to high levels of this chemical are well documented. A worker exposed to an estimated concentration of 1470 ppm perchloroethylene and Stoddard solvent for 3.5 hours lost consciousness (Stewart, Erley, Schaffer, and Gay 1961/Ex. 1–807). The most comprehensive studies of the effects of prolonged exposure to perchloroethylene vapors on human volunteers were conducted by Stewart and colleagues (Stewart, Hake, LeBrun et al. 1974/Ex. 1–970; Stewart, Hake, Wu et al. 1977/Ex. 1–971); these investigators concluded that prolonged exposure to 200 ppm results in early signs of CNS depression, while no response was elicited in men or women exposed repeatedly to 100 ppm for seven hours/day, except that performance on the Flanagan coordination test was significantly decreased in some exposed subjects (Stewart, Hake, Wu et al. 1977/Ex. 1–971, p. 28).

Based on these findings, the Agency concluded that its former PEL permitted workers to be exposed to a significant risk of CNS effects. In addition to examining the evidence for the chemical's narcotic effects, OSHA has reviewed a number of studies on the carcinogenicity of perchloroethylene. These investigations are summarized below.

In a 1977 gavage bioassay for carcinogenicity, perchloroethylene proved to be a liver carcinogen in mice but not in rats (NCI 1977c, as cited in ACGIH 1986/Ex. 1–3, p. 464). In 1986, the NTP conducted an inhalation bioassay of perchloroethylene (NTP 1986b/Ex. 8–31, Appendix 4), in which groups of 50 male and 50 female F344/N rats and B6C3F1 mice were exposed to perchloroethylene for six hours/day, five days/week, for two years. The exposure concentrations were 0, 200, or 400 ppm for rats and 0, 100, or 200 ppm for mice. Male and female rats exposed to either 200 or 400 ppm developed statistically significant increases in mononuclear cell leukemias. According to the NTP report (NTP 1986b/Ex. 8–31, Appendix 4), the increased incidences of leukemias were responsible for the early deaths observed in male and female rats exposed to perchloroethylene. At autopsy, most of the leukemias were determined to be in an advanced and probably fatal stage. Because of the effect of the leukemias on the early mortality of the exposed rats, a life-table analysis was used to test for the statistical significance of the findings; this analysis revealed that the increased incidence of leukemia was statistically significant in both low- and high-dose male rats and in low-dose female rats, and was marginally significant ($p = 0.053$) in high-dose female rats.

Male rats also developed a significant increase in renal tubular cell adenomas and carcinomas. Perchloroethylene induced a significantly increased incidence of hepatocellular carcinomas at both dose levels in mice of both sexes. The NTP Peer Review Panel concluded that there was "clear evidence of carcinogenicity of tetrachloroethylene" (perchloroethylene) in male rats and in male and female mice, and "some evidence" in female rats (Ex. 8–31, Appendix 4; Ex. 1–0000, p. 11).

In addition, a number of human studies were submitted to the rulemaking record that implicate perchloroethylene as a potential carcinogen (Ex. 8–31). Among these was a study by Brown and Kaplan (1987/Ex. 8–31, Appendix 6), which reported a statistically significant elevation in urinary tract cancer deaths among 1,690 dry cleaning workers exposed to perchloroethylene and other petroleum solvents. However, a subcohort of workers who used perchloroethylene as the primary solvent showed no increase in bladder cancer mortality. Brown and Kaplan concluded that "confounding exposure to petroleum solvents complicates any conclusions regarding the association between * * * [perchloroethylene] and cancer of the urinary tract" (Brown and Kaplan 1987/Ex. 8–31, Appendix 6, p. 540).

Katz and Jowett (1981/Ex. 8–31, Appendix 9) studied the mortality pattern of 671 female dry cleaning workers for the period 1963 through 1977. Elevated incidences of cancers of the kidney and genitals were reported, along with a smaller excess of bladder and skin cancers and lymphosarcomas. The authors concluded that, although results obtained with the methodology used (proportionate mortality ratios) require careful interpretation, "this study raises the possibility that exposure to dry cleaning fluids may increase the risk of certain cancers" (Katz and Jowett 1981/Ex. 8–31, Appendix 9, p. 510). The dry cleaning fluids used by members of the cohort included carbon tetrachloride, trichloroethylene, and perchloroethylene.

Steinhagen et al. (1983/Ex. 8–31, Appendix 8) reported a significant excess of liver cancer among male

workers in the laundry and dry cleaning industry in New Jersey. This study was a retrospective case-control study. The liver cancer cases were concentrated among individuals who processed clothes and were exposed to chemicals. The report did not identify the solvents in use (Steinhagen, Slade, Altman, and Bill 1983/Ex. 8–31, Appendix 8).

Duh and Asal (1984/Ex. 8–31, Appendix 7) examined the mortality experience of 440 dry cleaning workers in Oklahoma for the period 1975 through 1981. Elevated standardized mortality odds ratios (SMORs) were found for both lung cancer (SMOR=1.7) and kidney cancer (SMOR=3.8) (Duh and Asal 1984/Ex. 8–31, Appendix 7).

Eric Frumin of the Amalgamated Clothing and Textile Workers Union (ACTWU) submitted a quantitative risk assessment conducted by Dr. Dale Hattis of the Center for Technology Policy and Industrial Development at the Massachusetts Institute of Technology (Hattis 1986/Ex. 8–31, Appendix 11–A). This work was conducted in 1986 for the National Institute for Environmental Health Sciences. Dr. Hattis used a pharmacokinetic model that incorporated species-specific rates of formation for the metabolites of perchlorethylene. Using the rat leukemia and mouse liver tumor data from the NTP (1986b/Ex. 8–31, Appendix 4) bioassay, Dr. Hattis obtained a "best estimate" of the lifetime cancer risk (for workers exposed at the former 100-ppm OSHA limit for 45 years to perchloroethylene) of 45 deaths per 1,000 workers. The plausible upper limit at this level of exposure was 650 per 1,000 workers. The best-estimate lifetime risks associated with 45 years of exposure to 50 or 10 ppm of perchloroethylene were 25 and 6.4 deaths per 1,000 workers, respectively (the upper-confidence limits were 420 and 110 deaths per 1,000 workers, respectively). The ACTWU asserted that the studies reviewed above provide "overwhelming" evidence that perchloroethylene is a potential human carcinogen, and urged OSHA to establish a PEL lower than the proposed 50-ppm limit.

In its posthearing comments, the Halogenated Solvents Industry Alliance (HSIA) (Ex. 186) discussed several aspects of the data on perchlorethylene to support its contention that perchloroethylene should not be considered a probable human carcinogen. Specifically, the HSIA pointed out the following:

- Brown and Kaplan (1987/Ex. 8–31, Appendix 6) found no increased evidence of cancer among a subcohort of workers exposed only to perchloroethylene and not to other dry cleaning solvents.
- Both EPA and IARC have determined the human evidence on the carcinogenicity of perchloroethylene to be "inadequate."
- The National Research Council of the National Academy of Sciences concluded that the results of the NCI gavage study (NCI 1977c) should be interpreted with caution because of the large doses administered, early mortality of the treated animals, and observed nephrotoxicity.
- Regarding the NTP inhalation bioassay (NTP 1986b), the EPA Science Advisory Board (SAB) determined that the incidence of rat leukemia was not related to perchloroethylene exposure, and that the development of male rat kidney tumors was brought about by a mechanism unique to male rats.
- The EPA SAB stated that the mouse liver tumors observed in both the gavage (NCI 1977c) and inhalation (NTP 1986b) bioassays arose as a result of perchloroethylene-induced peroxisomal proliferation, a mechanism specific to rodents.

OSHA does not agree with the HSIA's interpretation of the meaning of the points raised by this group. First, the authors of the Brown and Kaplan (1987/Ex. 8–31, Appendix 6) study themselves pointed to the difficulty of establishing a definitive link between a particular solvent and an increased incidence of cancer in workers in the dry cleaning industry. For example, in the case of the group exposed to perchloroethylene only, the number of workers in the cohort was so small that even two or three exposure-related deaths in the perchloroethylene-only group would have caused a drastic swing in the SMR for bladder cancer in this subcohort. Thus, OSHA does not find that this study demonstrates the noncarcinogenicity of perchloroethylene.

As to the HSIA's second point, that neither the EPA nor IARC found the evidence for the carcinogenicity of perchloroethylene in humans adequate, OSHA notes that such evidence exists only for a handful of carcinogens (e.g., asbestos, benzene, vinyl chloride, arsenic), and that the overwhelming number of substances recognized as posing carcinogenic risks to workers have been determined to be carcinogenic on the basis of results in animals only. OSHA also believes that the regulation of many substances that have been designated as potential human carcinogens on the basis of clear evidence of their carcinogenicity in animals has undoubtedly contributed to the lack of evidence in humans by preventing overexposures to these substances in the workplace, and thus preventing cancer among these workers. Therefore, OSHA believes it appropriate and prudent to reduce workplace exposures to substances that have caused cancer in animals, especially when the animal studies are well-designed and carefully conducted bioassays.

The HSIA's third point, that the NCI gavage bioassay (NCI 1977c) has limitations, is irrelevant in the context of this discussion because OSHA is not relying on this bioassay to establish an appropriate limit for perchloroethylene.

The fourth point raised by the HSIA was that the Science Advisory Board of the EPA has questioned the relevance for human cancer risk of some of the tumors seen in the NTP (1986b/Ex. 8–31, Appendix 4) inhalation bioassay. OSHA believes that an explanation of the nature of the SAB's concern will demonstrate that an interpretation of the meaning of these data is a matter of professional judgment on which expert scientists themselves can differ. The SAB noted that there is some uncertainty regarding the significance of the leukemias observed in the perchloroethylene-exposed rats in the NTP (1986b/Ex. 8–31, Appendix 4) inhalation bioassay becasue the control rats in *another* NTP bioassay (NTP 1986c, the bioassay for methylene chloride) showed the same incidence of leukemias as the perchloroethylene-exposed rats (Ex. 186, pp. 6–7). However, OSHA points out that the independent peer review panel appointed by the NTP to evaluate the strength of the evidence for the carcinogenicity of perchloroethylene also considered the appropriateness of including the rat leukemia data when weighing the evidence for the carcinogenicity of perchloroethylene; the NTP panel concluded that the NTP (1986b/Ex. 8–31, Appendix 4) bioassay presented "clear evidence" of perchloroethylene's carcinogenicity in male rats (Ex. 8–31, Appendix 4, pp. 14–15). Thus, different scientists or groups of experts may interpret the same data differently; in this case, OSHA is not prepared to dismiss out-of-hand the leukemia data, given that leukemia contributed significantly to excess mortality in the perchloroethylene-exposed groups (NTP 1986b/Ex. 8–31, Appendix 4). The HSIA also questioned the relevance of the kidney tumors in male rats found in the NTP (1986b) bioassay. OSHA agrees with the SAB that these tumors may not be good predictors of human risk; however, the Hattis (1986/Ex. 8–31, Appendix 11–A) risk assessment did not

use the rat kidney tumor data, and, in addition, OSHA is not relying on these findings to set the final rule's limit for perchloroethylene.

On the HSIA's fifth point, the significance of rat liver tumors as predictors of human cancer risk, OSHA notes that the SAB did not believe it appropriate to disregard the findings in the recent NTP (1986b/Ex. 8–31, Appendix 4) bioassay of perchloroethylene-dose-related increases in the incidence of liver tumors in mice. In a letter dated to EPA Administrator Lee Thomas in March 1988 (Ex. 186D), the SAB concluded:

> The Board's consensus on the significance of mouse liver tumors is that mechanistic explanations are not sufficiently well developed and validated at this time to change EPA's present approach expressed in its risk assessment guidelines for carcinogenicity. It concludes that the generation of mouse liver tumors by chemicals is an important predictor of potential risks to humans (Ex. 186D, p. 2).

Based on the expert opinion of the NTP Peer Review panel and the EPA SAB, OSHA finds that the NCI (1986b/Ex. 8–31, Appendix 4) inhalation bioassay rat leukemia and mouse liver tumor data, which form the basis for the perchloroethylene quantitative risk assessment performed by Dr. Hattis (1986/Ex. 8–31, Appendix 11–A), should be regarded at this time as being relevant to the determination of potential human cancer risk from exposure to perchloroethylene in the workplace. The use of the rat leukemia data for the risk assessment may, however, add additional uncertainty to the risk estimates.

When EPA's Science Advisory Board considered perchloroethylene in January of 1987 (Ex./186C), it designated this substance as a Category C substance (i.e., a possible human carcinogen). However, in a letter to EPA Administrator Lee Thomas in March of 1988 (Ex. 186D), the SAB concluded that the overall weight of evidence for perchloroethylene "lies on the continuum between categories B2 [probable human carcinogen] and C." The SAB also stated that

> the distinction between the B2 and C categories can be an arbitrary distinction on a continuum of weight of evidence. The "black-white interpretation" * * * is indeed troubling * * *. A substance classified as [Category] C * * * for which human exposure is high may represent a much greater potential threat to human health [than substances classified as Category B2, B1, or A].
>
> EPA and other agencies * * * may, therefore, wish to take steps to reduce high exposures to substances in the C category whenever there appears to be a potentially significant threat to human health * * *. Indoor exposures to perchloroethylene, such as might be found in dry cleaning establishments not using the equivalent of good industrial hygiene practices, could merit action under this criteria. So might high levels of exposure to other solvents * * * that have been considered by the public as "safe" in the absence of sufficient evidence of carcinogenicity in animals. In many instances, this appearance of safety results from not yet having the results from well-designed bioassays such as those conducted by the National Toxicology Program.

OSHA agrees with the SAB that perchloroethylene is a substance that meets several of the criteria regarded by the SAB as meriting regulatory action. First, current exposures to perchloroethylene are high, often reaching the levels permitted by OSHA's existing PEL of 100 ppm. Second, several hundred thousand employees are regularly exposed to this widely used solvent. Third, the Hattis (1986/Ex. 8–31, Appendix 11–A) quantitative risk assessment suggests that a high cancer risk may be associated with exposure to perchloroethylene at OSHA's former or proposed PELs, indicating that exposures should be reduced to levels below the proposed 50-ppm level. Finally, the evidence for the carcinogenicity of perchloroethylene, which is briefly summarized below, is convincing.

The NTP (1986b/Ex. 8–31, Appendix 4) has concluded that perchloroethylene is carcinogenic by inhalation in both rats and mice. Based predominantly on the animal data, NIOSH has also concluded that perchoroethylene is a potential human carcinogen; NIOSH judged the evidence for perchloroethylene's carcinogenicity sufficient to warrant a separate 6(b) rulemaking (Ex. 8–47, Table N6B). In 1987, the International Agency for Research on Cancer (IARC) also classified perchloroethylene as a Category 2B carcinogen (i.e., a substance for which the evidence in animals is sufficient). The EPA's SAB has determined that perchloroethylene is a Category C carcinogen (i.e., a possible human carcinogen, and a carcinogen in animals). In addition, a number of human studies suggest elevated cancer risks, particularly of the kidney and bladder, among workers exposed to perchloroethylene and other solvents in dry cleaning facilities. Based on a review of all of the available evidence on perchloroethylene, including the testimony and briefs submitted by the parties, OSHA has determined that perchloroethylene is a potential human carcinogen that presents a significant risk of material health impairment to workers exposed to it in their places of work. This view was shared by several parties commenting in the record, including the Amalgamated Clothing and Textile Workers Union (Ex. 192), the AFL–CIO (Ex. 194), the American Public Health Association (Ex. 151), and NIOSH (Ex. 8–47).

The risk assessment conducted by Hattis (1986/Ex. 8–31, Appendix 11–A) estimates that there is an excess lifetime cancer mortality risk of 45 deaths per 1,000 workers exposed for 45 years to the current 100-ppm TWA PEL. Clearly, this high risk of mortality represents a significant risk. At the proposed level of 50 ppm, Dr. Hattis estimated the excess lifetime risk to be 27 deaths per 1,000 workers. OSHA concludes that this assessment and the underlying evidence clearly indicate that a further reduction in the PEL is necessary.

OSHA's analysis of the technological feasibility of reducing perchloroethylene exposures in affected industries, particularly in the dry cleaning industry, demonstrates that a PEL of 25 ppm is achievable using engineering and work practice controls; however, OSHA does not believe that information in the record at the present time demonstrates that it is feasible to reduce exposures to lower levels (see Section VII). In the dry cleaning industry, newer equipment, such as dry-to-dry dry cleaning machines, can achieve 25 ppm with engineering and work practice controls. This is true of smaller as well as larger operations.

The industry is gradually replacing older equipment with newer equipment, and a significant percentage of operations, including smaller operations, have installed such equipment. According to the industry, dry cleaning equipment is replaced at approximately 10-year intervals.

OSHA is providing a four-year phase-in period for the industry to come into compliance with the new levels through the use of engineering controls. Accordingly, OSHA believes that both smaller and larger dry cleaning operations can achieve the new 25–ppm TWA level in the ordinary course of the equipment replacement schedule. Consequently, the economic impact of the change to new equipment would not be great even for smaller operations.

In addition, use of older equipment in good condition results in employee exposure levels not much above the new 25-ppm PEL. Industry estimates indicate that levels of approximately 40 ppm can be attained. During the four-year interval noted in this regulation, reasonably priced retrofits for older

equipment may be developed that can be used to achieve the 25-ppm PEL.

OSHA is, of course, sympathetic to the circumstances of small businesses. If, after three years following publication of this regulation, it appears that there will be significant economic impacts for small dry cleaning operations attempting to convert to new equipment or retrofit within the four years permitted by the standard, OSHA will consider extending the period for smaller dry cleaning operations to achieve compliance using engineering and work practice controls. If that situation develops, OSHA believes that a trade association petition bringing the facts to OSHA's attention would be appropriate. OSHA would, at that time, evaluate the available information and make a decision based on all the information obtainable.

OSHA is establishing in the final rule a revised 8-hour TWA PEL of 25 ppm for perchloroethylene. OSHA concludes that the revised limit will substantially reduce the significant risk of material impairment of health presented by exposure to this substance at the Agency's former PEL of 100 ppm.

o-TOLUIDINE

CAS: 95-53-4; Chemical Formula $CH_3C_6H_4NH_2$

H.S. No. 1399

OSHA's former 8-hour TWA for o-toluidine was 5 ppm, with a skin notation. OSHA proposed to revise this limit to 2 ppm as an 8-hour TWA, with a skin notation. NIOSH (Ex. 8-47, Table N6A) concurs with the selection of this limit and notes that o-toluidine meets the OSHA definition of a potential human carcinogen. The ACGIH identifies o-toluidine as a suspected human carcinogen and has accordingly placed it in the A2 category (ACGIH 1986/Ex. 1-3, p. 586). The International Agency for Research on Cancer (IARC 1982b, as cited in ACGIH 1986/Ex. 1-3, p. 586) classifies o-toluidine as a probable carcinogen (category 2A) based on sufficient evidence of its carcinogenicity in rats and mice following oral administration (IARC 1982b, as cited in ACGIH 1986/Ex. 1-3, p. 586). IARC judged the evidence inadequate to establish o-toluidine's carcinogenicity in humans. In the final rule, OSHA has determined that it is appropriate to retain both the existing 5-ppm exposure limit and the skin notation for this substance. o-Toluidine is a light yellow liquid that rapidly darkens on exposure to air and light.

o-Toluidine is mutagenic in short-term tests, inducing sister chromatid exchanges and unscheduled DNA synthesis in mammalian cells in *in vitro* and chromosomal anomalies in yeast. o-Toluidine was negative in the micronucleus test in mice *in vivo*, but induced cell transformations in the BHK (baby hamster kidney) assay. IARC considers these data to be sufficient evidence of o-toluidine's activity in short-term tests (IARC 1982b, as cited in ACGIH 1986/Ex. 1-3, p. 586).

There are a number of studies that report an excess of bladder tumors in dyestuff workers exposed to o-toluidine and other chemicals; however, there are no studies that examine a population of workers exposed to o-toluidine alone. Workers exposed to toluene, o-nitrotoluene, o-toluidine, and 4,4-methylene bis (2-methylaniline) in manufacturing were observed to have an excess of bladder tumors. However, the concurrent exposures of these workers to these other potential carcinogens make these data inappropriate for use in the quantitative assessment of o-toluidine's carcinogenic risk in human populations. A few reports of bladder tumors in persons exposed primarily to o-toluidine have been reported, but insufficient follow-up time and incomplete data have prevented the establishment of a clear quantitative association between o-toluidine exposure and cancer in humans. For this reason, IARC considers the data from human studies inadequate to establish an association between exposure to o-toluidine and cancer (IARC 1982b, as cited in ACGIH 1986/Ex. 1-3, p. 586).

o-Toluidine has been determined to be carcinogenic in rats and mice following oral administration. In rats, statistically significant increases in subcutaneous fibromas, fibrosarcomas, and cancers of the urinary bladder have been reported. Studies in mice have resulted in statistically significant increases in hemangiosarcomas and hepatocellular carcinomas.

The National Cancer Institute (NCI 1979c, as cited in ACGIH 1986/Ex. 1-3, p. 586) conducted long-term carcinogenicity studies with o-toluidine in rats and mice. Both of these studies were positive for carcinogenicity. The mouse study used groups of 50 female and 50 male B6C3F1 mice fed o-toluidine hydrochloride in the diet at levels of 1000 ppm or 3000 ppm for 102 to 103 weeks. There was no excess mortality in the tested animals. At the 3000-ppm dose level, there was a statistically significant increase in hemangiosarcomas at all sites in males and a statistically significant increase in hepatocellular carcinomas and adenomas in females.

The National Cancer Institute also conducted a two-year feeding study with 50 male and 50 female Fischer 344 rats. There was a dose-related trend in mortality (which was not caused by cancer); all the males in the high-dose group died by 100 weeks. However, the females at both dose levels were observed to have significant increases in transitional-cell carcinomas or papillomas of the urinary bladder, and the high-dose females developed fibroadenomas of the mammary gland. The males at both dose levels showed significant increases in fibromas of the subcutaneous tissue and mesotheliomas in multiple organs (NCI 1979c, as cited in ACGIH 1986/Ex. 1-3, p. 586). The high mortality in the males complicates the interpretation of these latter findings.

Weisburger, Russfield, Homburger et al. (1978/Ex. 1-535) reported positive findings for o-toluidine in long-term feeding studies in rats and mice. The study in rats was conducted with two groups of 25 male CD rats fed o-toluidine in the diet via one of two regimens: 8000 ppm for three months and then 4000 ppm for an additional 15 months; or 16,000 ppm for three months and then 8000 ppm for an additional 15 months. Statistically significant increases in the incidence of subcutaneous fibromas and fibrosarcomas were observed in both dose groups. In addition, there was a nonstatistically significant increase in the incidence of transitional-cell carcinomas of the urinary bladder in these animals.

Weisburger, Russfield, Homburger et al. (1978/Ex. 1-535) also reported the results of a long-term study in mice. Groups of 25 males and 25 female CD-1 mice were fed diets containing o-toluidine at two dose levels: 16,000 ppm for three months and then 8000 ppm for an additional 15 months; or 32,000 ppm for three months and then 8000 ppm for an additional 15 months. There was a statistically significant, dose-related increase in the incidences of vascular tumors (hemangiosarcomas and hemangiomas of the abdominal viscera) in both sexes of treated mice, compared with results in control mice.

Risk estimate for o-toluidine. Four of these carcinogenicity studies of o-toluidine have yielded sufficient and adequate data for quantitative risk estimation: the two NCI studies (NCI 1979c, as cited in ACGIH 1986/Ex. 1-3, p. 586) and the two Weisburger et al. (1978/Ex. 1-535) studies. OSHA has used the NCI (1979c) study in rats as the basis for its quantitative risk assessment because it provides the most appropriate data. Table C15-7 presents the Maximum Likelihood Estimates (MLEs) of excess deaths per 1,000 employees

predicted to result from exposure to o toluidine at the current OSHA PEL of 5 ppm and at the proposed PEL of 2 ppm. These data were calculated using a multistage model, GLOBAL83.

TABLE C15-7. Multistage Model Estimates of Cancer Risk Associated with Working Lifetime Exposure to o-Toluidine

Workers	Excess Cancer Deaths per 1,000	
Exposure Level	MLE	UCL
5 ppm[a]	0.137	1.6
2 ppm[b]	0.055	0.64

[a] Former OSHA PEL.
[b] Final rule PEL.
MLE = Maximum likelihood estimate of risk.
UCL = Upper-bound (95-percent) confidence limit on maximum likelihood estimate of risk.

Table C15-7 shows an excess MLE estimate of risk of 1.4 per 10,000 workers exposed over their working lifetimes at the current PEL. This risk would be reduced to 0.5 per 10,000 exposed workers after promulgation of the final rule's limit of 2 ppm. This level of risk is lower than the levels OSHA has regulated for some carcinogens, such as ethylene oxide, arsenic, and benzene. George M. Talley and Michael C. Garcia, Industrial Hygienists for the Los Alamos National Laboratory, commented that OSHA's risk assessment does not support a reduction in the PEL for o-toluidine (Ex. 3-1095).

Lawrence Hecker of Abbott Laboratories (Tr. pp. 9-149) commented that OSHA's skin notation for o-toluidine is not supported by the available evidence. As described in Section VI.C.18 of the preamble regarding OSHA's general policy for establishing skin notations in this rulemaking, OSHA has determined that removal of an existing skin notation is not warranted unless human data are available that demonstrate the absence of a significant health risk from dermal contact with the hazardous substance in question. No such data exist for o-toluidine; therefore, OSHA is retaining the skin notation for o-toluidine in the final rule.

OSHA has concluded that further reduction in the exposure limit for o-toluidine would require a detailed analysis of the levels at which significant risk is eliminated. Both because of the scope of this rulemaking and because there were few comments on this issue, OSHA has not directed its limited resources to conduct a detailed analysis of this issue at this time. Accordingly, OSHA has concluded that it is appropriate to retain both the existing 5-ppm PEL and the skin notation for o-toluidine in this proceeding. OSHA is not making any final determination on either the general policy issue or what its conclusion might be in a single-substance rulemaking involving extensive public comment and detailed analysis.

p-TOLUIDINE

CAS: 106-49-0; Chemical Formula: $CH_3C_6H_4NH_2$

H.S. No. 1400

OSHA had no former PEL for p-toluidine. OSHA proposed establishing a 2-ppm PEL, with a skin notation, and these limits are established in the final rule. The ACGIH considers this substance a suspected human carcinogen and has given it a classification of A2 (ACGIH 1986/Ex. 1-3). p-Toluidine is a white solid.

One study investigates the carcinogenic potential of lifetime exposure to p-toluidine in experimental animals (Weisburger, Russfield, Homburger et al. 1978/Ex. 1-535). Male and female mice were exposed to p-toluidine in the diet for a total of 18 months. During the first six months of the experiment, mice were exposed to 1000 or 2000 mg p-toluidine/kg diet. As a result of the weight loss that occurred in mice exposed to the 2000 mg/kg diet dose, the concentrations of p-toluidine were reduced to 500 and 1000 mg/kg diet during the last 12 months of exposure. The rate of food consumption by the animals was not reported and was assumed to be 3 g/day. Thus, the average doses of p-toluidine received during the 18-month exposure were calculated to be 80 and 160 mg/kg body weight per day (Weisburger, Russfield, Homburger et al. 1978/Ex. 1-535).

For both the low and high dietary doses of p-toluidine, a significant increase in the incidence of hepatomas was observed. The incidence of tumors in the control, 80, and 160 mg/kg/day groups were 3/38, 10/38, and 12/35, respectively. The same study (Weisburger, Russfield, Homburger et al. 1978/Ex. 1–535) showed negative results in male rats exposed to two doses of p-toluidine in the diet for 18 months (1000- and 2000-mg/kg diet).

Risk estimate for p-toluidine. To assess the quantitative risk of p-toluidine's carcinogenicity. OSHA used the Weisburger et al. (1978/Ex. 1–535) data which, despite some limitations (e.g., changes in dose levels during the experiment and the absence of data concerning the amount of food animals consumed during the exposure period), were considered adequate for risk assessment purposes.

The maximum likelihood estimates (MLE) of excess cancers per 1,000 workers over an occupational lifetime and the 95-percent upper-bound estimates were obtained by using a linearized multistage model (GLOBAL83). These values are summarized in Table C15–8. This table shows the number of cancer deaths potentially associated with working lifetime exposure to 20, 5, or 2 ppm p-toluidine.

TABLE C15-8. Multistage Model Estimates of Cancer Risk Associated with Working Lifetime Exposure to p-Toluidine

Exposure Level	Excess Cancer Deaths per 1,000 Workers	
	MLE	UCL
2 ppm[a]	12	19
5 ppm	29	46
20 ppm	112	172

[a] Proposed OSHA PEL.
MLE = Maximum likelihood estimate of risk.
UCL = Upper-bound (95-percent) confidence limit on maximum likelihood estimate of risk.

OSHA concludes, as Table C15–8 shows, that workers exposed to p-toluidine, which was formerly not regulated by OSHA, are at significant risk of development hepatomas; an effect that the Agency considers a material impairment of health. For example, the MLE at 20 ppm is 112 excess cancer deaths per 1,000 workers exposed over a working lifetime. Promulgating a PEL of 2 ppm will substantially reduce this significant risk. According to this scenario, a 90-percent reduction in excess cancer deaths will be achieved by establishing the 2-ppm limit. The risks existing at the former uncontrolled level are clearly significant. NIOSH (Ex. 8–47, Table N6B) judged the evidence on p-toluidine sufficient to warrant a separate 6(b) rulemaking. OSHA is establishing an 8-hour TWA limit of 2 ppm for p-toluidine in the final rule; a skin notation is included to protect against percutaneous absorption of this substance.

VINYL BROMIDE

CAS: 593–60–2; Chemical Formula: C_2H_3Br
H.S. No. 1025

OSHA had no former PEL for vinyl bromide. Based on the ACGIH recommendation, OSHA proposed a 5-ppm TWA PEL; this limit is established in the final rule. NIOSH has no REL for vinyl bromide. The ACGIH places vinyl bromide on its A2 list of industrial substances suspected of having carcinogenic potential in humans. Vinyl bromide is a colorless gas with a characteristic odor and is used as an intermediate in organic synthesis and in the manufacture of polymers, copolymers, and flame retardants. Its principal use is as a flame retardant.

Henschler and Hoos (1982/Ex. 1–818) believe that vinyl bromide undergoes the same mechanism of biotransformation as its structural analog, vinyl chloride, a recognized human carcinogen that has been regulated by OSHA in a section 6(b) rulemaking. The microsomal oxidation of vinyl bromide leads to epoxide formation, which results, in turn, in the formation of a reactive intermediate. This intermediate has that potential to form covalent bonds with DNA to produce a mutagenic response. Vinyl bromide has been reported to be mutagenic in *Salmonella typhimurium*

and *tradescantia* (IARC 1979a/Ex. 1-1125; NIOSH/OSHA 1978/Ex. 1-1119).

No epidemiological studies have been conducted on populations exposed to vinyl bromide. Benya, Busey, Dorato, and Berteau (1982/Ex. 1-244) reported a positive carcinogenic response in an inhalation study of rats exposed to vinyl bromide vapor; this study is important because inhalation is a major mode of occupational exposure. The results of the Van Duuren (1977/Ex. 1-284) study were equivocal (described below), in that female Swiss albino mice were exposed dermally or by subcutaneous injection either to vinyl bromide in acetone or to polymerized vinyl bromide in an aqueous latex solution.

Benya et al. (1982/Ex. 1-244) exposed male and female Sprague-Dawley rats to 0, 9.7, 52, 247, or 1235 ppm vinyl bromide by inhalation for six hours daily, five days per week, for two years. The incidence of angiosarcomas, primarily of the liver, was found to be statistically significant in all dose groups tested except controls. It should be noted that a closely related chemical analog, vinyl chloride, also causes liver angiosarcoma in humans and animals. The combined incidences of hepatic angiosarcomas in the treated male and female rats were 1/288, 17/240, 86/240, 122/240, and 84/240 for their respective dose levels. One female rat in the control group developed an hepatic angiosarcoma. Table C15-9 summarizes the incidence of angiosarcoma in control and treated rats.

TABLE C15-9. Incidence of Angiosarcomas in Control and Vinyl-Bromide-Exposed Rats

		Males			Females		
Group	Exposure level (ppm)	No. of animals	No. with angio-sarcoma	p	No. of animals	No. with angio-sarcoma	p
1	Control	144	0	--	144	1	--
2	10	120	7	<0.025	120	10	<0.01
3	50	120	36	<0.001	120	50	<0.001
4	250	120	61	<0.001	120	61	<0.001
5	1250	120	43	<0.001	120	41	<0.001

Source: Benya, Busey, Dorato, and Berteau (1982/Ex. 1-244)

Van Duuren (1977/Ex. 1-284) injected a group of female ICR/Ha Swiss mice once weekly for 48 weeks with 0.05 ml of commerical polymerized vinyl bromide aqueous latex suspension; the animals were observed for 420 days. Nineteen of the 30 mice developed sarcomas at the site of injection. Animals in a positive control group that had been injected with b-propriolactone (0.3 mg/.05 ml trioctanoin) developed 18 sarcomas and three squamous cell carcinomas (in 30 mice). No tumors developed in untreated controls or in controls injected with trioctanoin, an organic solvent, alone (Van Duuren 1977/Ex. 1-284).

In another injection study by the same author, a group of female IRC/Ha Swiss mice were treated with 25 mg vinyl bromide per animal in 0.05 ml trioctanoin once weekly for 48 weeks. The mice were observed for 420 days. One control group was given a weekly injection of trioctanoin alone and the other control group was untreated. No local tumors were seen in any of the test groups, although pathological examination of the animals appears to have been incomplete (Van Duuren 1977/Ex. 1-284).

Application of vinyl bromide to the skin of female ICR/Ha Swiss mice at a dose of 15 mg per animal administered in 0.1 ml of acetone three times weekly for 420 days resulted in no tumors. When this solution was applied once and was followed by an application of phorbol myristyl acetate (PMA) three times weekly, one of 30 mice developed a skin papilloma at 412 days, one control treated with PMA developed a tumor after 44 days, and no untreated controls developed tumors (Van Duuren 1977/Ex. 1-284).

In another dermal study, a dose of 0.1 ml of polymerized vinyl bromide in an aqueous latex suspension was applied three times weekly to the skin of female ICR/Ha Swiss mice for 420 days. No skin tumors developed. When this solution was applied once, followed by an application of PMA three times weekly, one of 30 mice developed a skin tumor at 175 days. No untreated controls developed skin tumors (Van Duuren 1977/Ex. 1-284).

Risk estimate for vinyl bromide. The Benya et al. (1982/Ex. 1-244) study was a well-designed and -conducted study

that yielded sufficient information for quantitative risk estimation. The route of administration used in the study, inhalation, is directly applicable to occupational exposure, and the incidence of hepatic angiosarcoma was significant. Angiosarcoma is a rare and malignant neoplasm that has a very low background incidence in animals and humans. Therefore, its appearance in the exposed rats supports the premise that vinyl bromide is potentially carcinogenic in humans. Also, it is the same tumor that is associated with the exposure of workers and animals to vinyl chloride, a recognized human carcinogen and a compound whose structure is similar to that of vinyl bromide.

To estimate excess cancer risk over background incidence for a chemical, experimental data (experimental doses and corresponding responses) are used to define various parameters of an assumed response model. At low doses, the slope of this dose-response curve is referred to as q_1. The 95-percent upper-bound confidence limit for this slope is referred to as q_1^* or the chemical's potency. q_1 and q_1^* are then used to determine the respective maximum likelihood estimate (MLE) of risk and the 95-percent upper-bound confidence limit (UCL) on risk associated with a given lifetime occupational exposure. A nonthreshold, linearized multistage model (GLOBAL83) was chosen to estimate the risk potentially associated with exposure to vinyl bromide because the scientific rationale for this model is biologically the most plausible. Additionally, the choice of a nonthreshold model is consistent with current methodologies when positive mutagenicity data are available (*Guidelines for Carcinogenic Risk Assessment,* EPA 1984d).

Since both male and female rats responded equally to vinyl bromide treatment, data from the two groups were combined by calculating the geometric means of the risk estimates derived from the male and female response data (Anderson 1983/Ex. 1–1009). The high-dose data for each test group were dropped, since their inclusion makes the dose-response curve nonmonotonic and precludes proper fitting of the linearized multistage risk model (EPA 1984d).

Since cancer risk modeling assumes lifetime exposure, adjustments were made to fit the animal data to this criterion. The adjustments made for the data in the Benya et al. (1982/Ex. 1–244) study were: multiplying dose by 5/7 to adjust for days of exposure per week and by 6/24 to adjust for hours of exposure per day. These adjusted doses were then changed to human equivalent doses.

Three hypothetical occupational exposure limits, 5 ppm, 20 ppm, and 250 ppm, were used to calculate the maximum likelihood estimates of risk of developing angiosarcoma of the liver. Five ppm has been the ACGIH limit since 1978. Twenty ppm was chosen as an intermediate exposure level, and 250 ppm was the ACGIH TLV before the ACGIH reduced it in 1978. These occupational dose levels were also adjusted for lifetime exposure. The adjustments made were: multiplying dose by 5/7 to adjust for days worked per week, by 50/52 to adjust for vacation time, by 6/24 to adjust for hours of exposure per day, and by 45/70 to adjust for work years per lifetime.

Because inhalation is the primary route of exposure to vinyl bromide in occupational settings, the occupational dose was calculated assuming that air intake in humans is 20 m^3 per 24-hour day (Anderson 1983/Ex. 1–1009). The fraction of vinyl bromide absorbed was assumed to be 100 percent, because no absorption rate data were available for vinyl bromide. Because the log p (lipid solubility) value for vinyl bromide (1.52) is similar to that for vinyl chloride (1.38), OSHA assumed that the absorption rates of these two compounds would also be similar. The absorption rate for vinyl chloride used in risk estimations is assumed to be 100 percent (IRIS 1988).

The MLE shown in Table C15–10 for an occupational exposure to 250 ppm of vinyl bromide is 870 excess deaths per 1,000 workers. According to the linearized multistage risk model, 870 of 1,000 workers exposed over their working lifetimes to vinyl bromide at 250 ppm are at risk of developing angiosarcoma. The MLE for an occupational exposure to 5 ppm of vinyl bromide is 0.04; this indicates that, at the proposed PEL, 40 workers per 1,000 exposed to this substance over their occupational lifetimes are at risk of developing angiosarcoma.

TABLE C15-10. Multistage Model Estimates of Cancer Risk Associated with Lifetime Exposure to Vinyl Bromide

Exposure Level	Excess Cancer Deaths per 1,000 Workers	
	MLE[a]	UCL[a]
5 ppm[b]	40	48
20 ppm[c]	155	180
250 ppm[d]	870	930

[a] Geometric mean of male and female rats.
[b] Final rule PEL.
[c] Intermediate exposure level.
[d] ACGIH limit before 1978.
MLE = Maximum likelihood estimate of risk.
UCL = 95-percent upper-confidence limit on maximum likelihood estimate on risk.

NIOSH (Ex. 8-47, Table N6B) believes that the limit for vinyl bromide should be determined in a full section 6(b) rulemaking. The International Chemical Workers Union (Tr. p. 216) and the Workers Institute for Safety and Health (Ex. 116) were both of the opinion that the residual cancer risk remaining at the proposed 5-ppm PEL is excessive, and that a further reduction in the PEL is warranted. OSHA agrees with these commenters that significant risk has not been eliminated at the 5-ppm level. However, as explained in Section III of this preamble, the broad scope of this rulemaking prevented the Agency from making detailed analyses of risk and feasibility for alternative PELs. As priorities indicate and resources permit in the future, OSHA may consider the need for a further reduction in the PEL for vinyl bromide.

Table C15-10 shows that workers exposed to this substance, which was formerly not regulated by OSHA, are clearly at significant risk of developing hepatic angiosarcomas, the same rare type of tumor associated with exposure to vinyl chloride, a structurally similar substance. OSHA determined in its prior rulemaking on vinyl chloride that this disease constitutes a material impairment of health and functional capacity. Promulgating a PEL of 5 ppm will not eliminate this significant risk, because, as Table C15-10 shows, the MLE estimate of residual risk at 5 ppm is 40 excess deaths per 1,000 exposed workers. Thus, residual risk at 5 ppm is clearly significant. At the present time, OSHA concludes that establishing a PEL of 5 ppm TWA will substantially reduce the significant risk of cancer potentially associated with exposure at the uncontrolled levels formerly permitted in the absence of an OSHA limit for this substance.

VINYL CYCLOHEXENE DIOXIDE

CAS: 106-87-6; Chemical Formula: $C_8H_{12}O_2$
H.S. No. 1426

OSHA had no former PEL for vinyl cyclohexene dioxide (VCD). OSHA proposed establishing a 10-ppm TWA PEL, with a skin notation, for VCD, and this limit is established in the final rule. NIOSH (Ex. 8-47, Table N6A) agrees that this limit is appropriate and notes its determination that VCD is a potential human carcinogen. The ACGIH classifies VCD as a suspected human carcinogen (A2). Vinyl cyclohexene dioxide is a colorless liquid used as a chemical intermediate and as a monomer in the manufacture of polyglycols containing unreacted epoxy groups (Hine, Rowe, White, Darmer, and Youngblood 1981/Ex. 1-976). It is also used as a reactive diluent for other diepoxides and certain epoxy resins (IARC 1976).

Turchi, Bonatti, Citti et al. (1981/Ex. 1-282) assayed the mutagenicity of VCD and several other epoxides using the TA100 strain of *S. typhimurium* and V79 Chinese hamster cells; these authors also investigated the alkylating properties of these chemicals. VCD tested positive in both the *S. typhimurium* test (point mutation) and the V79 Chinese hamster cell test (both point mutation and chromosome aberration), and had an intermediate alkylating capacity relative to other epoxide compounds tested.

There are no data concerning the adverse health effects of VCD in humans. There are no reports as a result of industrial experience that reveal carcinogenic effects in workers caused by VCD exposure (ACGIH 1986/Ex. 1-3).

Four studies have reported the development of skin tumors in mice exposed dermally to VCD (Hendry, Homer, and Rose 1951/Ex. 1–250; Kotin and Falk 1963/Ex. 1–287; Weil, Condra, Haun, and Streigel 1963/Ex. 1–257; and Van Duuren, Nelson, Orris, Palmes, and Schmitt 1963/Ex. 1–288). The study of Van Duuren et al. (1963/Ex. 1–288) included controls and is thus particularly well suited for an evaluation of VCD's carcinogenic potential.

These authors painted 30 male Swiss ICR/Ha mice with 0.1 ml of a 10-percent solution of VCD in benzene three times per week (approximately 100 mg of solution per application). Two negative controls were used; one set of 150 mice was treated with benzene alone and another set of 207 mice was not treated with anything. Fourteen of the 30 VCD-treated mice developed skin tumors after an undefined length of time (mean survival time was 326 days). The incidences of skin tumors in the controls were 11/150 and 13/207 for the benzene-treated and untreated mice, respectively. The incidence of skin tumors in the VCD-treated mice was significantly greater than the incidence observed in either of the controls (Van Duuren, Nelson, Orris, Palmes, and Schmitt 1963/Ex. 1–288).

The study of Van Duuren et al. (1963/Ex. 1–288) demonstrates the carcinogenicity of VCD in experimental animals. OSHA considered the possibility of conducting a quantitative risk assessment for VCD, and the Agency concluded that the dose-response data in this study are unsuitable for quantitative risk assessment purposes because the VCD was administered in a solution of benzene, which is itself regulated as a carcinogen and classified as such by several authorities (IARC, NTP, NIOSH, and ACGIH). Even though the Van Duuren et al. (1963/Ex. 1–288) study included a control for the independent carcinogenic effects of benzene, the possibility of a synergistic or additive effect of benzene on VCD cannot be completely ruled out.

Vinyl cyclohexene dioxide has been shown to be carcinogenic by dermal application in mice, and four studies have confirmed these effects. Based on these animal studies showing VCD's carcinogenicity, OSHA concludes that exposed employees are at significant risk of cancer potentially associated with exposure to VCD at the uncontrolled levels formerly permitted by the absence of an OSHA limit. The Agency considers this effect a material impairment of health. No comments, other than NIOSH's, were received pertaining to VCD. The Agency concludes that promulgation of a 10-ppm 8-hour-TWA PEL, with a skin notation, will substantially reduce the significant occupational risk confronting VCD-exposed employees.

Conclusions for This Group of Substances

The Supreme Court in *I.U.D.* v. *A.P.I.* (*supra*, the *Benzene* decision) gave OSHA directions as to its decisional process; that case involved a carcinogen. OSHA is using the Supreme Court's guidance within the context of this present broader rulemaking. OSHA is also using the approach it has taken in the regulation of arsenic, benzene, EtO, asbestos, and formaldehyde; this approach has been upheld in the Courts of Appeals (see the introduction to this section). In the current rulemaking, OSHA has considered or performed quantitative risk assessments for each of the 17 chemicals discussed in this section; when less detailed dose-response data were available, OSHA performed qualitative appraisals of the significance of the risk. The risk assessments follow the approach OSHA has used in prior rulemakings for carcinogens, a process that has repeatedly been upheld by the courts. The risk assessment review process in this broader rulemaking has necessarily been more limited than is the case for single-substance rulemakings.

OSHA conducted its significant risk analyses using the principles suggested by the Supreme Court and adopted in its carcinogen rulemakings subsequent to *I.U.D.* v. *A.P.I.* OSHA has established new or revised exposure limits based on these analyses when they demonstrated that significant risk existed at the former PEL.

In some cases, it was not possible for OSHA to conduct quantitative estimates of cancer risk at the level of detail the Agency has formerly used. In these cases, OSHA believes that it has adequately justified the limits established in the final rule; without this latitude, the Agency would indeed be in the "mathematical straitjacket" alluded to by the Court in the *Benzene* decision.

In sum, where OSHA has concluded that there was sufficient evidence of potential carcinogenicity to meet the Agency's legal requirements, the Agency has established an exposure level based on the potential risk of occupational cancer. OSHA detemined in several prior rulemakings that this disease constitutes a material impairment of health and functional capacity. In the future, depending on priorities and resources, OSHA will further review the data to determine whether a second-stage rulemaking based on carcinogenicity is appropriate for some of the chemicals where a significant cancer risk appears to remain at the limits promulgated today.

Overall, OSHA believes its analyses of the new or revised limits for carcinogenic chemicals meet the Agency's legal requirements. Accordingly, OSHA concludes that these limits will lead to substantial reductions in the significant risk currently confronting workers exposed to these substances.

16. Substances for Which Current ACGIH TLVs Are Less Stringent Than Existing OSHA PELs

Introduction

As discussed in Section IV.D of this preamble, OSHA used either the ACGIH or NIOSH limits as a starting point in this rulemaking. There are 14 substances for which the ACGIH has increased its recommended TLVs since the time that OSHA adopted the 1968 TLVs under the authority of section 6(a) of the Act. These substances are listed in Table C16–1, along with their former, proposed, and final rule PELs, CAS numbers, and HS numbers. Evaluating the protectiveness and appropriateness of exposure limits that are less stringent than their former Z-table limits represents a special case in this rulemaking. OSHA has previously stated (see 50 FR 51120, December 13, 1985) the principles to be followed before the Agency raises an exposure limit. This issue is discussed below.

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TABLE C16-1. Substances for Which the ACGIH's Limits Were Higher Than the Former OSHA PELs

H.S. Number/ Chemical Name	CAS No.	Current PEL	ACGIH TLV**	Final Rule PEL*
1063 Camphor (synthetic)	76-22-2	2 mg/m^3 TWA	2 ppm TWA (12 mg/m^3 TWA) 3 ppm STEL (18 mg/m^3)	2 mg/m^3 TWA
1101 Copper fume (as Cu)	7440-50-8	0.1 mg/m^3 TWA	0.2 mg/m^3 TWA	0.1 mg/m^3 TWA
1126 1,1-Dichloroethane	75-34-3	100 ppm TWA	200 ppm TWA 250 ppm STEL	100 ppm TWA
1179 Fluorine	7782-41-4	0.1 ppm TWA	1 ppm TWA 2 ppm STEL	0.1 ppm TWA
1197 Hexachloroethan.	67-72-1	1 ppm TWA, Skin	10 ppm TWA	1 ppm TWA, Skin
1284 Nickel carbonyl (as Ni)	13463-39-3	0.001 ppm TWA	0.05 ppm TWA	0.001 ppm TWA
1347 Rhodium (as Rh), metal fume and insoluble salts	7440-16-6	0.1 mg/^{3} TWA	1 mg/m^3 TWA	0.1 mg/m^3 TWA
1348 Rhodium (as Rh), solub'e salts	7440-16-6	0.001 mg/m^3 TWA	0.01 mg/m^3 TWA	0.001 mg/m^3 TWA

TABLE C16-1. Substances for Which the ACGIH's Limits Were Higher Than the Former OSHA PELs

H.S. Number/ Chemical Name	CAS No.	Current PEL	ACGIH TLV**	Final Rule PEL*
1352 Silica, amorphous-diatomaceous earth (containing less than 1 percent crystalline silica)	68855-54-9	20 mppcf TWA (6 mg/m^3)	10 mg/m^3 TWA	6 mg/m^3 TWA
1353 Silica, amorphous-precipitate and gel	None	20 mppcf TWA (6 mg/m^3)	10 mg/m^3 TWA	6 mg/m^3 TWA
1362 Silver (as Ag), metal dust and fume	7440-22-4	0.01 mg/m^3 TWA	0.1 mg/m^3 TWA	0.01 mg/m^3 TWA
1386 Tetraethyl lead (as Pb)	78-00-2	0.075 mg/m^3 TWA, Skin	0.1 mg/m^3 TWA, Skin	0.075 mg/m^3 TWA, Skin
1388 Tetramethyl lead (as Pb)	75-74-1	0.075 mg/m^3 TWA, Skin	0.15 mg/m^3 TWA, Skin	0.075 mg/m^3 TWA, Skin
1419 Uranium (as U), soluble compounds	7440-61-1	0.05 mg/m^3 TWA	0.2 $mg.m^3$ TWA 0.6 mg/m^3 STEL	0.05 mg/m^3 TWA

* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

** The ACGIH TWA-TLV is for an 8-hour exposure; its STELs are 15-minute limits not to be exceeded more than 4 times per day with a minimum of 60 minutes between successive STEL exposures; and its ceilings are peaks not to be exceeded for any period of time.

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In 1978, OSHA issued a cotton dust standard; this standard did not go into effect in any of the nontextile industries. However, although the new standard's PEL for cotton dust did not apply in these segments, the cotton dust limit on Table Z–1 continued to apply to them. In 1983, OSHA determined that it would better effectuate the purposes of the Act to exclude the knitting and other nontextile industries from coverage by the Z-table limit for cotton dust. In revoking the Z-table limit, OSHA stated:

> When it [the Agency] proposes to eliminate a class [of operations or industry sectors] from either a 6(a) or 6(b) standard on health grounds, the evidence must affirmatively indicate that significant risk is unlikely to exist for that class at exposures likely to exist after the standard has been eliminated * * * OSHA must be able to support with substantial evidence any change it is propounding (50 FR 51120 *et seq.*, December 13, 1985).

Accordingly, the Agency must be able to show that exposed workers will not be placed at increased risk of the health effects at issue even *after* the limit in question has been raised or revoked. In conformance with this interpretation, OSHA has carefully examined the bases underlying the adoption of increased exposure limits by the ACGIH. After reviewing the available data for these substances, OSHA has made a determination that adequate evidence does not exist to increase the permissible exposure limits for any of these substances. For the 14 substances in this group, OSHA finds that the available toxicological data are insufficient to meet the increased burden of proof appropriate when the raising of an exposure limit is under consideration. For these substances, OSHA is therefore not revising its PELs at this time.

The following discussion summarizes OSHA's analyses and findings for each of the 14 substances in this group.

CAMPHOR (SYNTHETIC)

CAS: 76–22–2; Chemical Formula: $C_{10}H_{16}O$
H.S. No. 1063

In the NPRM (53 FR 21029), OSHA inadvertently indicated that its current limit for synthetic camphor is 2 ppm as an 8-hour TWA; however, the limit previously listed in 29 CFR 1910.1000, Table Z–1 (which is shown on Table Z–1–A of this final rule) was 2 mg/m³, or approximately 0.3 ppm. The ACGIH TLVs for camphor are a 2-ppm (12-mg/m³) TWA with a 3-ppm (18-mg/m³) STEL. This misrepresentation of the Agency's existing limit made the ACGIH limits appear more protective by comparison, and thus OSHA erroneously proposed to revise the PEL upward, an action that would constitute a relaxing of the current 2-mg/m³ TWA PEL. Consequently, OSHA has reconsidered its discussion of the evidence on synthetic camphor. In the final rule, OSHA is retaining its 2-mg/m³ (0.3-ppm) TWA PEL; NIOSH's comments (Ex. 8–47) on the proposal support this decision. Synthetic camphor is a colorless or white crystalline substance with an aromatic odor.

Synthetic camphor is known to cause severe injuries in animals exposed for prolonged periods by inhalation to a level of 6 mg/m³. Exposure may cause convulsions, congestion, changes in the gastrointestinal tract, and damage to the kidneys and brain (Flury and Zernik 1931b/Ex. 1–996). Animal bioassays showed that camphor was not carcinogenic in rats injected subcutaneously; however, when the cancer promoter, croton oil, was concurrently applied to the skin of mice, 2 of 110 treated mice developed carcinomas (Graffi, Vlamynck, Hoffman, and Schultz 1953/Ex. 1–903).

In humans, there are reports of industrial exposure to camphor that resulted in coma, dyspnea, and headache; one fatality from inhalation of the vapor has been noted (Flury and Zernik 1931b/Ex. 1–996).

The basis for ACGIH adopting the 2-ppm TLV–TWA and 3-ppm TLV–STEL is a report by Gronka, Bobkoski, Tomchick and Rakow (1969/Ex. 1–1043), which evaluated airborne exposures and the health status of six employees in a synthetic-camphor-processing plant. The authors reported that exposure for up to 10 months did not produce eye or nasal irritation if concentrations of camphor were maintained at or below 2 ppm. The investigators recommended that the former TLV of 2 mg/m³ be revised to 2 ppm (12 mg/m³).

The health status of the six employees was determined before the plant installed local ventilation and improved handling procedures; at that time, camphor concentrations ranged from 24 to 43 mg/m³. Four of the six employees examined showed inflammation of the nose and throat, and one reported having occasional numbness in the fingers. After process improvements were installed, only two of the employees were still working in the camphor-processing area; the remaining four had been away from direct contact with camphor.

OSHA concludes that the results of this study provide an inadequate basis for increasing the 2-mg/m³ PEL to 12 mg/m³ (2 ppm). The small number of employees examined by Gronka et al. (1969/Ex. 1–1043) and the lack of comprehensive medical examinations after exposures declined to 2 ppm provide no assurance that long-term exposure to 2 ppm is not associated with adverse health effects. In addition, the animal study conducted by Flury and Zernik (1931b/Ex. 1–996) demonstrated severe effects in animals exposed for prolonged periods to a level one-half that found in the plant studied by Gronka et al. (1969/Ex. 1–1043). Therefore, OSHA concludes that establishing the 2-ppm (12-mg/m³) limit is unwarranted, and the Agency is retaining its 2-mg/m³ (0.3-ppm) limit for synthetic camphor in the final rule. No comments, other than those made by NIOSH, were submitted to the record.

COPPER (FUME)

CAS: 7440–50–8; Chemical Formula: Cu
H.S. No. 1101

The current OSHA limit for copper fume is 0.1 mg/m³ as an 8-hour TWA. Since OSHA adopted this limit in 1971, the ACGIH has increased the recommended TLV to 0.2 mg/m³ as an 8-hour TWA. The ACGIH's previously recommended TLV of 0.1 mg/m³ was based on a personal communication (Whitman 1957 and 1962, as cited in ACGIH 1986/Ex. 1–3, p. 146) that reported that the taste perception of welders was altered when they were exposed to copper fume at levels ranging from 1 to 3 mg/m³ for short periods but that exposure to from 0.02 to 0.4 mg/m³ did not cause such complaints (ACGIH 1966/Ex. 1–13). At the time, the ACGIH judged the 0.1-mg/m³ TLV to be "sufficiently low to provide freedom from irritation from the fume by a reasonable margin" (ACGIH 1966/Ex. 1–13). NIOSH (Ex. 8–47, Table N1) concurs that OSHA's 0.1=mg/m³ limit is appropriate. Copper is a reddish-colored metal.

In 1972, the ACGIH received a personal communication from a member of the U.K. Industrial Hygiene Unit, Her Majesty's Factory Inspectorate (Luxon 1972, as cited in ACGIH 1986/Ex. 1–3, p. 146) reporting that employees exposed to copper fume at levels up to 0.4 mg/m³ during welding and copper metal refining operations experienced no ill effects from exposure. Based on this additional evidence, the ACGIH increased its TLV for copper fume to 0.2 mg/m³ in 1975.

Commenters to the docket urged OSHA to revise the PEL for copper fume to the ACGIH limit. BP America (Ex. 8–57; Tr. pp. 9–126 to 9–127) argued that the Agency should increase its PEL even though the only basis for doing so was a personal communication to the ACGIH TLV Committee. In response to these commenters, OSHA reiterates the

position described in the introduction to this section (i.e., that the Agency must demonstrate that exposed workers will not be placed at increased risk even *after* the limit has been raised). Because the personal communication on which the ACGIH has based its increased limit cannot be examined to determine information of this type, OSHA cannot consider raising the limit at this time.

OSHA concludes that the evidence cited by the ACGIH (1986/Ex. 1-3) in support of the increase in its TLV-TWA for copper fume is not sufficient to support an increase in OSHA's PEL for this substance. OSHA reasons that the ACGIH's action was based largely on a personal communication, which makes it impossible for the Agency to evaluate the evidence appropriately.

1,1-DICHLOROETHANE

CAS: 75-34-3; Chemical Formula: Ch_3CHCl_2
H.R. No. 1126

The current OSHA limit for 1,1-dichloroethane, which is a hepatotoxin, is 100 ppm TWA. The ACGIH TLV-TWA is 200 ppm, with a 250-ppm STEL: NIOSH has no REL for this substance. The previous ACGIH TLV of 100 ppm was based on the observation that 1,1-dichlorethane has an acute toxicity approximately half that of carbon tetrachloride and a chronic toxicity somewhat less than that of carbon tetrachloride (for which a TLV of 10 ppm had been set). In 1973, the ACGIH adopted the higher 200-ppm TLV based on unpublished data from the Dow Chemical Company (AIHA 1971, as cited in ACGIH 1986/Ex. 1-3, p. 184) showing that rats, rabbits, guinea pigs, and dogs exhibited no gross or microscopic organ pathology after exposure to 500 or 1000 ppm of 1,1-dichloroethane for six months. The ACGIH cited no human data in support of its increase in the TLV. NIOSH (Ex. 8-47, Table N1) concurs that OSHA's 100-ppm 8-hour TWA is appropriate. OSHA received no other comments on 1,1-dichloroethane.

Because no human toxicity data are available for 1,1-dichloroethane and because the Dow data are unpublished and thus not available for scrutiny, OSHA concludes that the evidence for this substance is insufficient to warrant increasing the PEL at this time.

FLUORINE

CAS: 7782-41-4; Chemical Formula: F
H.S. No. 1179

OSHA's current PEL for fluorine is 0.1 ppm; NIOSH has no REL for fluorine. In 1973, the ACGIH revised its Tl V to 1 ppm and, subsequent to that change, adopted a TLV-STEL of 2 ppm. OSHA proposed these ACGIH limits of 1 ppm TWA and 2 ppm STEL; however, the final rule retains the Agency's existing 0.1-ppm TWA limit. Fluorine is a pale yellow gas with a pungent irritating odor.

The ACGIH's previous 0.1-ppm TLV, which was adopted by OSHA in 1971, was based on a 30-day inhalation study in rats and dogs (Stokinger 1949b, as cited in ACGIH 1986/Ex. 1-3, p. 274) in which no consistent pulmonary, renal, or blood effects were observed following exposure to 0.5 ppm. The ACGIH believed that a TLV of 0.1 ppm would "provide a working environment of probable safety from the effects of F_2" (ACGIH 1966/Ex. 1-13). Subsequently, the ACGIH reviewed a seven-year study (Lyon 1962/Ex. 1-639) of 61 workers exposed to fluorine concentrations "far in excess of 0.1 ppm" (ACGIH 1986/Ex. 1-3, p. 2274),which reported a lack of significant medical findings. This evidence, along with more recent animal evidence (Keplinger and Suissa 1968/Ex. 1-342) suggesting that animals were not as sensitive to fluorine as was reported by Stokinger (1949b, as cited in ACGIH 1986/Ex. 1-3, p. 274), led the ACGIH to increase its TLV to 1 ppm. The STEL of 2 ppm was supported by a study (Ricca 1970/Ex. 1-357) in which human volunteers repeatedly exposed to 10 ppm reported only slight-irritation.

NIOSH (Ex. 8-47, Table N2) submitted extensive comments to the record criticizing the ACGIH's reasoning in raising the limit for fluorine. NIOSH concluded:

> [T]here is no data existing to support raising the limit. The Lyon (1962/Ex. 1-639) study is severely limited and a review of the actual paper indicates [that] it has far less value than reported in the ACGIH documentation. All the animal data is, in fact, consistent with the original exposure data on which the 0.1-ppm level was based (Ex. 8-47, p. 3).

OSHA agrees with NIOSH and has determined that it is not appropriate, as had originally been proposed by the Agency, to increase the limit for fluorine at this time. OSHA concludes that the human and animal evidence is inadequate to support an increase in the 8-hour TWA for this substance from 0.1 ppm to 1 ppm. OSHA is therefore retaining its PEL of 0.1 ppm as an 8-hour TWA.

HEXACHLOROETHANE

CAS: 67-72-1; Chemical Formula: CCl_3CCl_3
H.S. No. 1197

OSHA's current PEL for hexachloroethane is a 1-ppm TWA, with a skin notation, which was adopted from the 1968 ACGIH TLV. The NIOSH REL for this substance is the lowest feasible level, based on hexachloroethane's potential carcinogenicity. Hexachloroethane is a nonflammable white solid.

The basis for the 1-ppm TLV was to prevent the "serious injury potential to several organ systems" shown by animal studies (ACGIH 1986/Ex. 1-3, p. 301). Subsequently, the ACGIH revised its TLV upward to 10 ppm based, in part, on a study by Weeks, Angerhofer, Bishop et al. (1979/Ex. 1-400) that reported no adverse effects among several animal species exposed daily to 15- or 48-ppm concentrations of hexachloroethane. The ACGIH also cited an NCI study (NCI 1978b/Ex. 1-949), in which "extremely heavy dosages . . . administered continuously for a long period of time" resulted in the development of hepatocellular tumors in mice but not in rats. The 10-ppm TLV was further supported by a personal communication of a TLV Committee member who reported that no ill effects occurred among workers "who handled the material with few precautions" during World War II (ACGIH 1986/Ex. 1-3, p. 301). No exposure data were supplied to support this personal communication.

In 1978, NIOSH reviewed the results of an NCI (1978b/Ex. 1-949) bioassay in which hexachloroethane was administered by gavage to mice and rats. Both male and female mice exhibited an excess incidence of hepatocellular carcinoma, but rats did not. NCI concluded that early mortality may have obscured detection of a carcinogenic effect in rats (NCI 1978b/Ex. 1-949). Toxic kidney damage was also found in mice and rats treated with hexachloroethane. Based on this evidence, NIOSH (*Chloroethanes: Review of Toxicity*, Current Intelligence Bulletin 27, NIOSH 1978r) has recommended that exposure to hexachloroethane be maintained at the lowest detectable level.

Several participants (Exs. 3-678, 116, 144, and 194; Tr. pp. 9-149, 9-218) commented on hexachloroethane. The New Jersey Department of Health (Ex. 144) discussed the use of ERA's IRIS system to determine limits (OSHA's discussion of this approach is presented in Section V1.A of this preamble). The Workers Institute of Safety and Health (WISH) (Ex. 116; Tr. p. 9-218) and the AFL-CIO (Ex. 194) stated that the ACGIH's increase in the limit for hexachloroethane reflects an inappropriate use of safety factors; WISH was also of the opinion that OSHA should have performed a quantitative risk assessment for hexachloroethane. In response to WISH, OSHA notes: (1) That the Agency is no. following the ACGIH's move to a higher

limit for hexachloroethane; and (2) that OSHA performed risk assessments only for those substances classified in the carcinogen section of this preamble.

Lawrence Hecker, Corporate Director of Industrial Hygiene and Toxicology for Abbott Laboratories (Ex. 3-678; Tr. p. 9-1149) stated that the skin notation should not be retained for hexachloroethane because this material is not systematically toxic via dermal absorption. However, in accordance with the Agency's policy on skin notations (see Section VI.C.18 of this preamble), OSHA is retaining a skin notation for hexachloroethane in the final rule.

OSHA concludes that the evidence relied on by the ACGIH is not adequate to support raising the PEL at this time. The human evidence cited by the ACGIH is anecdotal and lacks the exposure data necessary to permit OSHA to assess whether significant risk is absent (and likely to remain so) at the 10-ppm exposure level. In addition, OSHA is concerned, as is NIOSH (Ex. 8-47, Table N6A), about the development of tumors in hexachloroethane-exposed mice demonstrated in the NCI (1978b/Ex. 1-949) study. OSHA therefore retains its PEL of 1 ppm TWA, with a skin notation, and concludes that increasing the PEL for hexachloroethane would increase the significant risk of cancer potentially associated with exposure to this substance.

NICKEL CARBONYL

CAS: 13463-39-3; Chemical Formula: $Ni(CO)_4$ H.S. No. 1284

The current OSHA PEL and the NIOSH recommended limit for nickel carbonyl is 0.001 ppm TWA, as Ni. Nickel carbonyl is a gaseous compound at ordinary pressure or a colorless, highly volatile liquid, with a musty odor. In 1976, the ACGIH increased its TLV for nickel carbonyl from 0.001 to 0.05 ppm. The ACGIH's former 0.001-ppm TLV was based primarily on the high incidence of nasal and lung cancer among workers exposed to nickel carbonyl during work in nickel refinery operations. In addition, the ACGIH cited evidence (Sunderman, West, and Kincaid 1959/Ex. 1-384) that rats exposed to nickel carbonyl developed lung tumors that metastasized to the kidneys. At the time, the ACGIH (1966/Ex. 1-13) noted that these tumors were not of a type generally associated with exposure to environmental agents.

In its 1976 documentation for the 0.05-ppm TLV for nickel carbonyl, the ACGIH cited the work of Doll, Morgan, and Speizer (1970/Ex. 1-821), who evaluated the exposures of nickel refinery workers in whom cancers had been found. Doll and associates (1970/Ex. 1-821) found that there had been no exposures to nickel carbonyl in the facility, and this finding led the ACGIH to conclude that nickel carbonyl was not the causative agent of the cancers reported among the refinery workers in the earlier studies it had relied on to set the 0.001-ppm TLV. A report that no excess nasal or lung tumors had occurred among workers exposed over a 50-year period in a nickel refinery in Wales (Renzoni, personal communication, 1975, as cited in *Documentation of the Threshold Limit Values for Substances in Workroom Air*, 3rd ed., ACGIH 1976) appeared to the ACHIH to corroborate Doll et al.'s (1970/Ex. 1-821) results. The ACGIH concluded that the TLV for nickel carbonyl should be raised based on the acute, systemic effects of this substance, and that carcinogenicity was not an appropriate basis for limit-setting (ACGIH 1976). In the 1986 *Documentation* for the 0.05-ppm TLV for nickel carbonyl, the ACGIH (Ex. 1-3) concluded that, "although the evidence that nickel carbonyl is carcinogenic to humans is inconclusive, this recommended TLV (i.e., one set at 0.05 ppm) is also adequate to minimize any potential carcinogenic effects" (ACGIH 1986/Ex. 1-3, p. 424).

OSHA received comments on nickel carbonyl from NIOSH (Ex. 8-47, Table N6A) and from Inco United States, Inc. and Inco Limited (Exs. 3-915 and 167). Inco urged OSHA to adjust the PEL for nickel carbonyl to 0.05 ppm and also stated that the limit for this substance should not be enforced until an adequate sampling and analytical method has been developed. On this issue of the health basis for an increase in the PEL, OSHA notes that Inco, like the ACGIH (1986/Ex. 1-3), believes that it is appropriate to increase this limit on the sole basis of results of negative epidemiological studies and a personal communication attesting to the absence of a "significant positive association with risk" in a Welsh refinery. However, as described in the introduction to this section, OSHA must meet a more stringent test before raising a limit. In addition, the interpretation of negative studies in humans is complicated by a host of factors (see Section V1.A of this preamble).

As to Inco's second point, OSHA notes that it has an in-house sampling and analytical method for nickel carbonyl that is available from the Agency on request. In addition, the limit for nickel carbonyl at issue is the limit that was assigned to this substance in 1971, at the time the Agency was established. OSHA is not required to perform feasibility analyses on its existing limits, and the Agency is unaware of any unusual compliance difficulties with this substance.

NIOSH (Ex. 8-47, Table N6A) concurs that retention of OSHA's limit is appropriate because NIOSH regards nickel carbonyl as a potential occupational carcinogen. Thus, OSHA finds the evidence discussed by the ACGIH insufficient to warrant an increase in the limit; some of this evidence is in the form of a personal communication. The Agency concludes that increasing the limit for this substance would increase the significant risk for exposed workers. In the final rule, OSHA is therefore retaining the existing PEL for nickel carbonyl of 0.001 ppm as an 8-hour TWA.

RHODIUM COMPOUNDS (METAL FUME; SOLUBLE AND INSOLUBLE SALTS)

CAS: 7440-16-6: Chemical Formula: Rh H.S. No. 1347; 1348

The current OSHA PEL for rhodium metal fume and insoluble salts is 0.1 mg/m^3 as Rh; the current PEL for soluble rhodium compounds is 0.001 mg/m^3 as Rh. Rhodium is a silvery white, hard, ductile, and malleable metal. The ACGIH recommends a 1-mg/m^3 TLV for rhodium metal and insoluble salts and a 0.01-mg/m^3 TLV for soluble rhodium salts. The current OSHA PELs for rhodium compounds (i.e., the 1968 ACGIH TLVs) were based on the then-existing TLVs for platinum because of concern that exposure to rhodium might be associated with respiratory sensitization effects. This concern was prevalent because rhodium belongs to the platinum family of metals and because the toxicologic data on rhodium that were formerly available were "meager" (ACGIH 1966/Ex. 1-13).

The ACGIH's decision to increase the TLVs for rhodium compounds was based primarily on a personal communication to the TLV Committee (Johnson, Matthey and Co., Ltd. 1981b, as cited in ACGIH 1986/Ex. 1-3, p. 512). This communication indicated that, in a major precious metals refinery, "procedures which were abandoned for the refining of platinum because of cases of sensitization have been carried out for a year with analogous rhodium compounds without any problems" (ACGIH 1986/Ex. 1-3, p. 512). In addition, the ACGIH noted that none of the substances in the platinum group was known to produce respiratory effects similar to those of platinum. The ACGIH reported that rhodium exhibited "slight" carcinogenic activity in mice (ACGIH 1986/Ex. 1-3). After considering all of this evidence, the ACGIH judged the previoius TLVs to be inappropriate and increased them tenfold.

NIOSH (Ex. 8-47, Table N1) concurs that OSHA should retain its PELs for

these substances. No other comments on rhodium were received. OSHA concludes that the evidence adduced by the ACGIH is not sufficient to meet the standard of proof the Agency must achieve before it can raise an exposure limit. This conclusion is based on that fact that the ACGIH relied heavily on a personal communication when making its decision, and no exposure or other data are available to support the ACGIH's action. Thus OSHA is unable to adequately evaluate the toxicologic evidence pertaining to the rhodium compounds and retains the existing PELs for rhodium metal fume and insoluble salts (0.1 mg/m³ TWA) and rhodium soluble salts (0.001 mg/m³ TWA).

SILICA, AMORPHOUS—DIATOMACEOUS EARTH

CAS: 68855-54-9; Chemical Formula: SiO_2
H.S. No. 1352

OSHA's current limit for amorphous silica is 20 mppcf, which is equivalent to 6 mg/m³ TWA (ACGIH 1984), measured as total dust. The ACGIH has established a limit for this dust (measured as total dust) of 10 mg/m³ (8-hour TLV-TWA). Amorphous silica (diatomaceous earth) is composed of the skeletons of prehistoric plants known as diatoms. These skeletons are largely noncrystalline, although diatomaceous earth can contain varying amounts of crystalline quartz, which has led, in the opinion of the ACGIH (1986/Ex. 1-3, p. 520), to conflicting results in studies of the pulmonary effects of exposure to this colorless to gray, odorless powder.

Cooper and Cralley (1958-Ex. 1-1145) reported "doubtful" linear-nodular changes in the lungs of workers exposed only to amorphous (noncrystalline) silica for five years or more. Other studies (Vigliani and Mottura 1948/Ex. 1-534; Gardner 1942, as cited in ACGIH 1986/Ex. 1-3, p. 520) either found mild silicosis only or no evidence of serious lung pathology in diatomite workers. Kovalevich (1957, as cited in ACGIH 1986/Ex. 1-3, p. 520) reported silicosis in diatomite workers, but intratracheal instillation of diatomaceous earth dust in animals caused evidence of fibrosis (Gardner 1942, as cited in ACGIH 1986/Ex. 1-3, p. 520) and silicosis (Kovalevich 1957, as cited in ACGIH 1986/Ex. 1-3, p. 520). Another study (Tebbens and Beard 1957/Ex. 1-531) exposed guinea pigs to this substance at an average concentration of 60 mg/m³ for 37 to 50 weeks and found both accumulations of dust-laden macrophages and alveolar epithelialization but no fibrosis.

In setting its limit for diatomaceous earth, the ACGIH (1986/Ex. 1-3, p. 520) assumed that this substance itself is either "weakly fibrogenic or nonfibrogenic," and thus that those studies discussed above that report adverse pulmonary effects actually involved exposure to diatomaceous earth having an unmeasured but significant crystalline quartz content. Based on this reasoning, the ACGIH considers amorphous silica (diatomaceous earth) to have low biological activity.

OSHA received few comments on its proposal to retain the 6-mg/m³ PEL for diatomaceous earth. The Synthetic Amorphous Silica and Silicates Industry Association (SASSI) (Ex. 1-630) requested that OSHA revise its entry for "silica, amorphous, diatomaceous earth" to "silica, crystalline, diatomaceous earth" to reflect the fact that diatomaceous earth frequently contains crystalline silica. OSHA intends the PEL for crystalline quartz of 0.1 mg/m³ to apply to diatomaceous earth containing more than 1 percent crystalline silica. For clarification, OSHA has added the designation "containing less than 1 percent crystalline silica" to the entry for diatomaceous earth on Table Z-1-A of the final rule, for which the 6-mg/m³ limit is applicable.

SASSI also suggested that the crystalline silica PEL apply to any silicates containing more than 0.1 percent, rather than 1 percent, crystalline silica because of recent concerns regarding the potential carcinogenicity of silica. As discussed in the section on crystalline silica (see Section VI.C.6), OSHA has not made a final determination on the carcinogenicity of silica; therefore, at this time, OSHA will apply the limits for silicates to those materials containing less than 1 percent silica.

NIOSH (Ex. 8-47) concurred with the 6-mg/m³ TWA PEL for diatomaceous earth, provided the silica content does not exceed 1 percent. Chevron Corporation (Ex. 3-896) also agreed with OSHA's proposal. Both Chevron (Ex. 3-896) and SASSI (Ex. 3-630) agreed that the former mppcf limit should be revised to a limit expressed as mg/m³, since the use of mppcf units is outdated.

OSHA is retaining an 8-hour TWA of 6 mg/m³ (equivalent to 20 mppcf) for this form of silica. OSHA finds that the health evidence for this substance is not sufficiently persuasive to permit an increase in the limit at the present time. The Agency is revising the units in which its permissible exposure limit for diatomaceous earth is expressed; this change is being made to facilitate the accurate monitoring of employee exposures and does not represent a change in the value of the limit.

SILICA, AMORPHOUS, PRECIPITATED AND GEL

CAS: None; Chemical Formula: SiO_2
H.S. No. 1353

OSHA currently has a limit of 20 mppcf (which is equivalent to a limit of 6 mg/m³) for amorphous silica. The ACGIH recommends a TLV-TWA of 10 mg/m³ measured as total dust containing less than 1 percent quartz. OSHA is retaining the current PEL in the final rule but is expressing this limit in milligrams per cubic meter; NIOSH (Ex. 8-47, Table N1) concurs with the Agency's decision. There are numerous methods of producing precipitated silica; those that apply heat to siliceous products produce airborne dusts that are less toxic than quartz dust because the particles are generally sheathed in a molecular layer of amorphous silica (ACGIH 1986/Ex. 1-3, p. 521).

Studies of laboratory animals have shown no fibrosis after intratracheal and intraperitoneal injection of precipitated silica or silica gel (Klosterkotter 1954/Ex. 1-1156; Klosterkotter 1958/Ex. 1-1039). Schepers and colleagues reported in 1957 that rats exposed for one year and guinea pigs and rabbits exposed for two years to a concentration of 126 mg/m³ of precipitated amorphous silica displayed no pulmonary fibrosis; the effects of exposure were limited to macrophage accumulations and mild proliferation of reticulin fibers (Schepers, Durkan, Delahant et al. 1957/Ex. 1-755).

In a study of human exposures to precipitated amorphous silica, Wilson and associates reported no ill effects in 165 workers exposed for an average of 8.6 years (Wilson, Stevens, Lovejoy et al. 1981/Ex. 1-1177).

The ACGIH considers the precipitated and gel forms of amorphous silica to have low biological activity, based on the evidence discussed above. PPG Industries (Ex. 3-1158) commented that an unpublished NIOSH study (Groth, Kommineni, Stettler et al. 1979, as cited by H.E. Stokinger in *Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., Vol. 2B, pp. 3011-3014) showed that rats, guinea pigs, and monkeys developed accumulations of macrophages in the lungs following exposure to precipitated silica. In addition, the presence of collagen was seen in "very few" monkeys; by comparison, collagen was not seen in any animal exposed to silica gel but was seen in significant amounts in monkeys exposed to fumed silica. PPG remarked that the findings in animals exposed to precipitated silica showed "no evidence for effects * * * which are inconsistent with the ACGIH

criteria for nuisance particulates" (Ex. 3–1158). PPG urged OSHA to adopt a 10-mg/m³ PEL for precipitated silica based on this observation. SASSI (Ex. 3–630) also requested that OSHA adopt either a 10-mg/m³ total dust limit or a 5-mg/m³ respirable dust limit for precipitated silica, based on the recommendation of ASTM's E34.16 Committee.

After reviewing these comments, OSHA concludes that the available evidence does not meet the criteria described earlier in this section for determining that an increase in the present PEL is warranted. OSHA notes that, in the study cited by PPG, there was collagen formation only in a few animals exposed to precipitated silica. Furthermore, the report by Wilson et al. (1981/Ex. 1–1177) involved only a relatively small number of employees who had been exposed for fewer than 10 years. Accordingly, OSHA is retaining its current PEL of 6 mg/m³ (equivalent to 20 mppcf) at the present time. However, to facilitate the accurate monitoring of employee exposures, the Agency is changing the units in which its permissible exposure limit for amorphous silica is expressed.

SILVER (METAL DUST AND FUME)

CAS: 7440–22–4; Chemical Formula: Ag
H.S. No. 1362

The current OSHA standard for silver metal and soluble compounds (including the metal dust and fume) is 0.01 mg/m³, as Ag. NIOSH has no REL for this substance, but the ACGIH has established a 0.1-mg/m³ TLV for silver metal dust and fume. NIOSH concurs with OSHA's decision not to increase the limit for silver (Ex. 8–47). Silver is a hard, brilliant, white, ductile, malleable metal.

The previous TLV of 0.01 mg/m³, which was established for all forms of silver, was designed to protect workers against developing argyria. This condition arises from the accumulation of silver in the body and results in an unsightly, widespread blue-grey discoloration of the skin that can persist for long periods of time. The skin of exposed workers may also become black and have a metallic luster. Argyria may manifest in the conjunctiva of the eye, which may be affected sufficiently to cause lens and visual disturbances.

In arriving at the previous TLV of 0.01 mg/m³ for silver, the ACGIH relied on a publication by Pillsbury and Hill (1939, as cited in ACGIH 1986/Ex. 1–3, p. 529), which stated that an accumulated intake of from 1 to 5 grams of silver would lead to generalized argyria. Assuming a 20-year exposure duration, a 10-m³/day respiratory volume, and a 50-percent body retention, the ACGIH estimated that exposure to 0.05 mg/m³ was sufficient to cause argyria. The former TLV of 0.01 mg/m³ thus appeared to incorporate a safety factor to account for the uncertainties involved in using this approach to develop a TLV. The ACGIH's current TLV of 0.1 mg/m³ for silver metal dust and fume was determined in a similar fashion, except that the ACGIH assumed a lower percent retention and apparently did not incorporate a safety margin (ACGIH 1986/Ex. 1–3).

OSHA received several comments on its proposal to retain the existing limit for silver (Ex. 8–47, 8–57, 3–876, 46, and 105; Tr. pp. 9–126 to 9–127). The American Mining Congress (Ex. 3–876) stated that argyria, the blue-grey discoloration of the skin caused by exposure to silver, is caused only by exposure to the soluble silver salts and not by metallic silver. BP America, a company that operates a silver smelting and refining operation in Utah, is also of the opinion that OSHA should increase its limit for silver (metal, dust, and fumes) because, although argyria "can be cosmetically unpleasant, it is not known to result in any adverse health consequences" (Ex. 8–57).

OSHA responds to these commenters as follows. First, OSHA does not agree that having one's skin discolored, on a semipermanent basis, is a "minor" effect. On the contrary, OSHA believes that argyria causes emotional stress, acute personal discomfort, and feelings of insecurity, all of which are symptoms of severe psychological distress.

In addition, although the American Mining Congress is certain that only the soluble forms cause argyria, OSHA notes that Wolf Wagner, Manager of Industrial Hygiene for BP America, expressed uncertainty on this point at the hearing; he reported that argyria is "most likely due to a soluble silver rather than an insoluble silver" (Ex. 8–57; Tr. pp. 9–126 to 9–127). OSHA agrees that considerable uncertainty surrounds the issues of the causative agents of argyria and the specific level at which this effect occurs. As the ACGIH (1986/Ex. 1–3, p. 529) reports:

> The concentration of silver in the air which will result in generalized argyria is not known with certainty.

Thus, OSHA concludes that the evidence needed to raise the limit for silver is lacking. OSHA is therefore retaining its former limit for silver (metal, dust, and fume) of 0.01 mg/m³ as an 8-hour TWA.

TETRAETHYL LEAD (TEL)

CAS: 78–00–2; Chemical Formula: $(C_2H_5)_4Pb$
H.S. No. 1386

OSHA's current 8-hour limit for tetraethyl lead is 0.075 mg/m³, measured as lead, with a skin notation; NIOSH has no REL for this substance. The ACGIH is now recommending that worker exposure to TEL not exceed 0.1 mg/m³ TWA; the ACGIH also recommends a skin notation. Tetraethyl lead is a colorless liquid, which may be dyed red, orange, or blue, and has a slightly musty odor.

The previous TLV of 0.075 mg/m³ was based almost exclusively on a personal communication from the Medical Department of the Ethyl Corporation, which stated that a level of 0.075 mg/m³ "is a good guideline for an allowable air concentration of TEL" (ACGIH 1966/Ex. 1–13). The ACGIH documentation for the 0.075-mg/m³ TLV also pointed out that the ability of tetraethyl lead to penetrate the skin "makes reliance on the airborne concentration impractical in many situations," and that urinary lead levels are a more reliable indicator of exposure than blood lead levels (ACGIH 1966/Ex. 1–13).

In its documentation for the 0.1-mg/m³ TLV, the ACGIH (1986/Ex. 1–3, p. 563) again cited the communication from the Ethyl Corporation. In addition, the organization cited a personal communication from Linch (1968, as cited in ACGIH 1986/Ex. 1–3, p. 563), who reported that an improved analytical procedure for measuring airborne concentrations of tetraethyl lead had been used to determine the relationship between airborne tetraethyl lead levels and urinary lead levels. He reported that urinary lead concentration was not significantly elevated "above a high normal" value (0.15 mg/L) when the airborne TEL level was 121 μg/m³ (ACGIH 1986/Ex. 1–3, p. 563). As a result of this communication, the ACGIH adopted a revised TLV of 0.1 mg/m³ in 1970.

NIOSH (Ex. 8–47, Table N1) concurs that OSHA should retain its existing limit for TEL; no other comments on this substance were submitted.

OSHA does not find the evidence presented by the ACGIH to be sufficiently comprehensive or detailed to permit significant risk to be ruled out at the 0.1-ppm level. The Agency is also reluctant to increase the PEL for TEL in light of this substance's ability to be absorbed percutaneously. OSHA is therefore retaining the existing PEL of 0.075 mg/m³, measured as Pb and with a skin notation, for tetraethyl lead.

TETRAMETHYL LEAD (TML)

CAS: 75–74–1; Chemical Formula: $(CH_3)_4Pb$
H.S. No. 1388

The current OSHA limit for tetramethyl lead (TML) is 0.075 mg/m³ TWA, with a skin notation, while the ACGIH has recommended a TLV of 0.15 mg/m³, measured as Pb and with a skin notation. There is no NIOSH REL for TML. Tetraethyl lead is a colorless liquid, which may be dyed blue, orange, or red; it has a slight musty odor.

In establishing the previous TLV of 0.15 mg/m³, the ACGIH cited the work of de Treville, Wheeler, and Sterling (1962/Ex. 1–310), who reported that tetramethyl lead is about three times more volatile than tetraethyl lead and thus results in airborne TML levels that are about three times higher than those for TEL. Despite the heavier TML exposure of employees, urinary lead levels were not significantly different from the urinary lead levels of employees exposed to TEL. The ACGIH concluded that a 0.075-mg/m³ TLV for TML, identical to the TLV recommended at the time for TEL, should furnish an adequate margin of safety. The revised TLV of 0.15 mg/m³ was based on a personal communication by Linch (1968, as cited in ACGIH 1986/Ex. 1–3, p. 563), who reported that exposure to 0.179 mg/m³ tetramethyl lead was not associated with a significant increase in urinary lead levels.

NIOSH concurs (Ex. 8–47, Table N1) that the retention of the Agency's 0.075-mg/m³ limit is appropriate, and no other comments on TML were received. Based on the same reasoning as that described above in connection with tetraethyl lead, OSHA is not increasing its existing TWA limit for TML; the skin notation for TML is also retained.

URANIUM (SOLUBLE COMPOUNDS)

CAS: 7440–61–1; Chemical Formula: U
H.S. No. 1419

The current OSHA limit for soluble uranium compounds is 0.05 mg/m³ TWA, measured as uranium. NIOSH has no REL for soluble uranium compounds. Since 1968, the ACGIH has increased its TLV for soluble uranium from 0.05 mg/m³ to 0.2 mg/m³, with a 0.6-mg/m³ STEL. The previous TLV of 0.05 mg/m³ was based on animal studies relating exposure level and duration to the resulting tissue concentration of uranium and on other chronic animal studies showing the kidney to be the most sensitive target organ. In 1968, the ACGIH's *List of Intended Changes* included a TLV of 0.2 mg/m³ for all forms of uranium, and this value was dropped by the ACGIH in 1969. The basis for adopting the 0.2-mg/m³ TLV for soluble uranium compounds was a study by Wing, Heatherton, and Quigley (1963, as cited in ACGIH 1986/Ex. p. 617) reporting no adverse effects from radiation exposure over a 25-year period. Although no data were discussed in the ACGIH (1986/Ex. 1–3) *Documentation* regarding typical exposure levels at the plants studied, the documentation does mention that seven accidental, brief exposures to soluble uranium compounds at levels two- to fivefold the former TLV of 0.05 mg/m³ did not result in physiologic changes or significant body burden.

Allied Signal, Inc. (Ex. 3–1084) is of the opinion that OSHA's limit for the soluble compounds of uranium is "unrealistically low based on NRC (Nuclear Regulatory Commission) and industry experience." This company states that the fact that soluble uranium "exits the body quite rapidly" means that it does not produce radiation-induced cancer. OSHA finds that this evidence is not sufficiently detailed to use as a basis for raising its limit for these compounds, and NIOSH (Ex. 8–47, Table N1) concurs.

OSHA does not find this evidence adequate to meet the Agency's more stringent standard of proof for relaxing an existing exposure limit. In addition, OSHA notes that the 25-year period of observation in the Wing et al. (1963, as cited in ACGIH 1986/Ex. 1–3, p. 617) study is not long enough to rule out the occurrence of some forms of radiation-induced cancer and, further, that the power of this study to detect health effects occurring in a small percentage of the population was very limited. OSHA is accordingly not raising its current PEL for the soluble uranium compounds.

17. Substances for Which OSHA is Establishing Short-Term Exposure Limits

Introduction

OSHA is establishing a short-term exposure limit (STEL) for a total of 116 substances; these substances are listed in Table C17–1.

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Table C17-1. Substances for Which OSHA is Establishing STELs to Supplement TWA Limits

H.S. Number/ Chemical Name	CAS No.	Final Rule STEL
1001 Acetaldehyde	75-07-0	150 ppm
1004 Acetone	67-64-1	1000 ppm
1005 Acetonitrile	75-05-8	60 ppm
1007 Acrolein	107-02-8	0.3 ppm
1010 Allyl alcohol	107-18-6	4 ppm
1011 Allyl chloride	107-05-1	2 ppm
1012 Allyl glycidyl ether (AGE)	106-92-3	10 ppm
1013 Allyl propyl disulfide	2179-59-1	3 ppm
1022 Ammonium chloride fume	12125-02-9	20 mg/m^3
1042 Bromine	7726-95-6	0.3 ppm
1045 2-Butanone (MEK)	78-93-3	300 ppm
1047 n-Butyl acetate	123-86-4	200 ppm
1050 tert-Butyl alcohol	75-65-0	150 ppm
1056 p-tert-Butyltoluene	98-51-1	20 ppm
1064 Caprolactam Dust	105-60-2	3 mg/m^3
1065 Caprolactam Vapor	105-60-2	40 mg/m^3
1069 Carbon dioxide	124-38-9	30,000 ppm
1070 Carbon disulfide	75-15-0	12 ppm
1072 Carbon tetrabromide	558-13-4	0.3 ppm
1074 Carbonyl fluoride	353-50-4	5 ppm
1078 Chlorinated camphene	8001-35-2	1 mg/m^3
1079 Chlorine	7782-50-5	1 ppm
1080 Chlorine dioxide	10049-04-4	0.3 ppm
1089 o-Chlorostyrene	2039-87-4	75 ppm
1114 Decaborane	17702-41-9	0.15 ppm
1116 Di-sec-octyl-phthalate	117-81-7	10 mg/m^3
1119 Dibutyl phosphate	107-66-4	2 ppm
1122 1,3-Dichloro-5,5-dimethylhydantoin	118-52-5	0.4 mg/m^3
1125 p-Dichlorobenzene	106-46-7	110 ppm
1127 Dichloroethyl ether	111-44-4	10 ppm
1137 Diethylamine	109-89-7	25 ppm
1143 Dimethylaniline	121-69-7	10 ppm
1149 Dipropylene glycol methyl ether	34590-94-8	150 ppm
1159 Ethanolamine	141-43-5	6 ppm
1161 Ethyl acrylate	140-88-5	25 ppm
1162 Ethyl benzene	100-41-4	125 ppm
1163 Ethyl bromide	74-96-4	250 ppm
1164 Ethyl ether	60-29-7	500 ppm

Table C17-1. Substances for Which OSHA is Establishing STELs To Supplement TWA Limits (continued)

H.S. Number/ Chemical Name	CAS No.	Final Rule STEL
1168 Ethylene dichloride	107-06-2	2 ppm
1177 Ferrovanadium dust	12604-58-9	3 mg/m^3
1182 Formamide	75-12-7	30 ppm
1184 Furfuryl alcohol	98-00-0	15 ppm
1185 Gasoline	8006-61-9	500 ppm
1194 n-Heptane	142-82-5	500 ppm
1201 Hexane isomers	Varies	1000 ppm
1203 Hexone (Methyl isobutyl ketone)	108-10-1	75 ppm
1208 Hydrogen fluoride	7664-39-3	6 ppm
1209 Hydrogen sulfide	7783-06-4	15 ppm
1216 Iron pentacarbonyl	13463-40-6	0.2 ppm
1218 Isoamyl alcohol	123-51-3	125 ppm
1222 Isophorone diisocyanate	4098-71-9	0.02 ppm
1224 Isopropyl acetate	108-21-4	310 ppm
1225 Isopropyl alcohol	67-63-0	500 ppm
1227 Isopropyl glycidyl ether	4016-14-2	75 ppm
1228 Isopropylamine	75-31-0	10 ppm
1231 Ketene	463-51-4	1.5 ppm
1236A Manganese fume	7439-96-5	3 mg/m^3
1242 Mercury (organo), alkyl compounds	7439-97-6	0.03 mg/m
1243 Mesityl oxide	141-79-7	25 ppm
1248 Methyl 2-cyanoacrylate	137-05-3	4 ppm
1249 Methyl acetate	79-20-9	250 ppm
1250 Methyl acetylene/ propadiene mixture	None	1250 ppm
1252 Methyl alcohol	67-56-1	250 ppm
1254 Methyl chloride	74-87-3	100 ppm
1255 Methyl chloroform (1,1,1-trichloroethane)	71-55-6	450 ppm
1258 Methyl formate	107-31-3	150 ppm
1261 Methyl isobutyl carbinol	108-11-2	40 ppm
1267 alpha-Methyl styrene	98-83-9	100 ppm
1270 o-Methylcyclohexanone	583-60-8	75 ppm
1281 Morpholine	110-91-8	30 ppm
1282 Naphthalene	91-20-3	15 ppm
1286 Nitric acid	7697-37-2	4 ppm
1295 Octachloronaphthalene	2234-13-1	0.3 mg/m^3
1296 Octane	111-65-9	375 ppm
1298 Osmium tetroxide	20816-12-0	0.006 mg/m^3
1299 Oxalic acid	144-62-7	2 mg/m^3

Table C17-1. Substances for Which OSHA is Establishing STELs To Supplement TWA Limits (continued)

H.S. Number/ Chemical Name	CAS No.	Final Rule STEL
1301 Ozone	10028-15-6	0.3 ppm
1304 Pentaborane	19624-22-7	0.015 ppm
1306 Pentane	109-66-0	750 ppm
1307 2-Pentanone (Methyl propyl ketone)	107-87-9	250 ppm
1309 Perchloryl fluoride	7616-94-6	6 ppm
1317 Phenylhydrazine	100-63-0	10 ppm
1319 Phorate (Thimet)	298-02-2	0.2 mg/m^3
1320 Phosdrin (Mevinphos)	7786-34-7	0.3 mg/m^3
1321 Phosphine	7803-51-2	1 ppm
1322 Phosphoric acid	7664-38-2	3 mg/m^3
1324 Phosphorus pentasulfide	1314-80-3	3 mg/m^3
1325 Phosphorus trichloride	7719-12-2	0.5 ppm
1338 n-Propyl acetate	109-60-4	250 ppm
1339 Propyl alcohol	71-23-8	250 ppm
1340 n-Propyl nitrate	627-13-4	40 ppm
1341 Propylene dichloride	78-87-5	110 ppm
1343 Propylene glycol monomethyl ether	107-98-2	150 ppm
1346 Resorcinol	108-46-3	20 ppm
1366 Sodium fluoroacetate	62-74-8	0.15 mg/m^3
1372 Styrene (Phenylethylene)	100-42-5	100 ppm
1375 Sulfur dioxide	7446-09-5	5 ppm
1379 Sulfuryl fluoride	2699-79-8	10 ppm
1387 Tetrahydrofuran	109-99-9	250 ppm
1397 Toluene	108-88-3	150 ppm
1398 Toluene-2,4-diisocyanate	584-84-9	0.02 ppm
1403 1,1,2-Trichloro-1,2,2-trifluoroethane	76-13-1	1250 ppm
1406 Trichloroethylene	79-01-6	200 ppm
1408 Triethylamine	121-44-8	15 ppm
1411 Trimethylamine	75-50-3	15 ppm
1416 Tungsten & compounds (insoluble)	7440-33-7	10 mg/m^3
1417 Tungsten & compounds (soluble)	7440-33-7	3 mg/m^3
1418 Uranium (insoluble compounds)	7440-61-1	0.6 mg/m^3
1424 Vinyl acetate	108-05-4	20 ppm
1429 VM&P Naphtha	8032-32-4	400 ppm

Table C17-1. Substances for Which OSHA is Establishing STELs To Supplement TWA Limits (continued)

H.S. Number/ Chemical Name	CAS No.	Final Rule STEL
1430a, Wood dust 1430b all soft and hard-woods except Western red cedar	None	10 mg/m^3
1431 Xylene (o,m,p-isomers)	1330-20-7	150 ppm
1435 Zinc chloride fume	7646-85-7	2 mg/m^3
1437 Zinc oxide fume	1314-13-2	10 mg/m^3
1435 Zirconium compounds	7440-67-7	10 mg/m^3

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When OSHA adopted the ACGIH TLVs in 1971, the ACGIH had not established the short-term TLV category; as a consequence, none of the substances on OSHA's Z-1 table have STELs. (Some of the substances on OSHA's current Z-2 tables, whose limits derive from standards established by the American National Standards Institute rather than the ACGIH, have "acceptable ceiling concentrations" that act, in effect, as short-term exposure limits.)

The ACGIH defines a STEL as

> a 15-minute time-weighted average exposure which should not be exceeded at any time during a work day even if the eight-hour time-weighted average is within the TLV. Exposures at the STEL should not be longer than 15 minutes and should not be repeated more than four times per day. There should be at least 60 minutes between successive exposures at the STEL. An averaging period other than 15 minutes may be recommended when this is warranted by observed biological effects (ACGIH 1987/Ex. 1-16).

Basis Under Which ACGIH Established STELs

The ACGIH establishes STELs for substances that cause a wide variety of acute effects; these effects include irritation, narcosis, lung damage, systemic effects, and organic poisoning. The ACGIH first considered adding STELs to the TLV-TWAs for some substances in 1971 when it appointed a subcommittee to study the appropriateness of adding such exposure limits to its TLV list.

In 1973, this subcommittee recommended that the ACGIH establish STELs as a third category (along with TLV-TWAs and TLV-ceilings) of exposure limits. The STEL was defined as the maximum concentration to which workers can be exposed continuously *for a period of up to 15 minutes* without suffering from

1. Intolerable irritation,
2. Chronic or irreversible tissue change, or
3. Narcosis of sufficient degree to increase accident proneness, impair self-rescue, or materially reduce work efficiency (*Supplemental Documentation to the Fourth Edition of the Documentation of the Threshold Limit Values,* ACGIH 1984).

The ACGIH stipulated that no more than four such excursions per day were permissible, with at least 60 minutes between exposure periods, and that the daily TLV-TWA could not be exceeded.

In 1974, the ACGIH agreed by consensus that 425 of the 520 compounds in its 1973 list should have STELs assigned to them, but these were not in fact published until 1976, when "Tentative Values" for STELs were listed in the organization's annual booklet. The 1987-1988 ACGIH TLV booklet states that the TLV-STEL is "the concentration to which workers can be exposed continuously for a short period of time without suffering from (1) irritation, (2) chronic or irreversible tissue damage, or (3) narcosis of sufficient degree to increase the likelihood of accidental injury, impair self-rescue or materially reduce work efficiency . . . provided that the daily TLV-TWA is not exceeded."

In 1982, the ACGIH qualified the conditions under which STELs are recommended to "only [those situations] where toxic effects have been reported from high short-term exposures in either humans or animals." Since that time, the ACGIH has re-examined the toxicological data and subsequently deleted the STELs for 297 substances because of insufficient evidence that adverse effects result from acute exposures. The most recent (1988-1989) edition of the *Threshold Limit Values and Biological Exposure Indices* (ACGIH 1988b) proposes deletion of the short-term limit for an additional 18 substances. The ACGIH has stressed that STELs are set on physiological grounds rather than in response to sampling and analytical limitations (ACGIH 1984).

Separate from the STEL category, the ACGIH in the 1970s established a fourth limit, a general "excursion factor" that should always be observed implicitly but is not specifically assigned to each chemical. The "excursion limit" recommended by the ACGIH is defined as follows:

> Short-term exposures should exceed three times the TLV-TWA for no more than a total of 30 minutes during a work day and under no circumstances should they exceed five times the TLV-TWA, provided that the TLV-TWA is not exceeded (ACGIH 1987).

The basis for this excursion limit is that any process having emissions that display a variability greater than would be permitted by this excursion factor is not under good industrial hygiene control, and the ACGIH believes that, in such cases, efforts should be made to restore control (ACGIH 1986x). Where a specific STEL exists for a substance, the specific STEL takes precedence over the general excursion limit (ACGIH 1987). Thus *all* ACGIH TLV-TWAs have implicit excursion limits, but only a few substances (i.e., those for which specific toxicological evidence indicates that a STEL is necessary) have explicit STELs.

Basis for Short-Term Limits Being Promulgated by OSHA

The STELs being promulgated by OSHA in this rulemaking, which parallel those STELs remaining in the ACGIH's most recent list (ACGIH 1987-1988) are thus limits for substances where there is toxicological evidence of recognized acute effects resulting from short-term exposure. The health effects associated with short-term exposures for some of these substances are shown in Table C17-2.

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TABLE C17-2. Health Effects Supporting Final Rule STELs

H.S. Number/ Chemical Name	Final Rule STEL	Health Effects
1001 Acetaldehyde	150 ppm	Eye irritation; narcosis; potential injury to respiratory tract
1004 Acetone	1000 ppm	Eye, nose, and throat irritation; narcosis
1005 Acetonitrile	60 ppm	Nausea; headache; convulsions
1007 Acrolein	0.3 ppm	Irritation; lung edema
1010 Allyl alcohol	4 ppm	Irritation
1011 Allyl chloride	2 ppm	Mucous membrane irritation
1012 Allyl glycidyl ether (AGE)	10 ppm	Irritation
1013 Allyl propyl disulfide	3 ppm	Irritation; lacrimation
1022 Ammonium chloride fume	20 mg/m^3	Irritation
1042 Bromine	0.3 ppm	Respiratory tract irritation
1045 2-Butanone (MEK)	300 ppm	Eye and nose irritation
1047 n-Butyl acetate	200 ppm	Throat irritation
1050 tert-Butyl alcohol	150 ppm	Narcosis
1056 p-tert-Butyltoluene	20 ppm	Eye, nose, and throat irritation
1064 Caprolactam dust	3 mg/m^3	Irritation

TABLE C17-2. Health Effects Supporting Final Rule STELs (continued)

H.S. Number/ Chemical Name	Final Rule STEL	Health Effects
1065 Caprolactam vapor	40 mg/m^3	Irritation
1069 Carbon dioxide	30,000 ppm	Central nervous system effects; asphyxiation
1072 Carbon tetrabromide	0.3 ppm	Upper respiratory tract irritation; injury to lungs, liver, and kidney
1074 Carbonyl fluoride	5 ppm	Respiratory irritation
1079 Chlorine	1 ppm	Eye, mucous membrane, skin, and pulmonary irritation
1080 Chlorine dioxide	0.3 ppm	Irritation
1089 o-Chlorostyrene	75 ppm	Dizziness; nausea; headache
1114 Decaborane	0.15 ppm	Hyperexcitability; narcosis
1119 Dibutyl phosphate	2 ppm	Irritation to respiratory tract; headaches
1122 1,3-Dichloro-5,-5-dimethylhydantoin	0.4 mg/m^3	Respiratory irritation
1125 p-Dichlorobenzene	110 ppm	Acute poisoning
1127 Dichloroethyl ether	10 ppm	Upper respiratory tract and eye irritation

TABLE C17-2. Health Effects Supporting Final Rule STELs (continued)

H.S. Number/ Chemical Name	Final Rule STEL	Health Effects
1137 Diethylamine	25 ppm	Acute toxicity characterized by strong local irritation
1143 Dimethylaniline	10 ppm	Methemoglobinemia; CNS depression
1149 Dipropylene glycol methyl ether	150 ppm	Eye, nose, and throat irritation; central nervous system impairment
1159 Ethanolamine	6 ppm	Pulmonary irritation
1161 Ethyl acrylate	25 ppm	Irritation
1162 Ethyl benzene	125 ppm	Skin and eye irritation
1163 Ethyl bromide	250 ppm	Narcosis
1164 Ethyl ether	500 ppm	Narcosis; nasal irritation
1168 Ethylene dichloride	2 ppm	Central nervous system effects
1177 Ferrovanadium dust	3 mg/m^3	Eye and respiratory irritation
1184 Furfuryl alcohol	15 ppm	Eye irritation
1185 Gasoline	500 ppm	Narcosis; irritation
1194 n-Heptane	500 ppm	Narcosis; respiratory irritation

TABLE C17-2. Health Effects Supporting Final Rule STELs (continued)

H.S. Number/ Chemical Name	Final Rule STEL	Health Effects
1201 Hexane isomers	1000 ppm	Narcotic symptoms; eye and throat irritation; slight nausea, headache
1203 Hexone (MIBK)	75 ppm	Irritant effects
1208 Hydrogen fluoride	6 ppm	Eye and respiratory irritation
1209 Hydrogen sulfide	15 ppm	Eye irritation
1216 Iron pentacarbonyl	0.2 ppm	Headaches; dizziness
1218 Isoamyl alcohol	125 ppm	Respiratory and eye irritation
1222 Isophorone diisocyanate	0.02 ppm	Respiratory effects and sensitization; pulmonary irritation
1224 Isopropyl acetate	310 ppm	Eye and respiratory irritation
1225 Isopropyl alcohol	500 ppm	Narcotic effects and irritation
1227 Isopropyl glycidyl ether	75 ppm	Respiratory tract and eye irritation
1228 Isopropylamine	10 ppm	Respiratory irritation

TABLE C17-2. Health Effects Supporting Final Rule STELs (continued)

H.S. Number/ Chemical Name	Final Rule STEL	Health Effects
1231 Ketene	1.5 ppm	Respiratory irritation
1236A Manganese fume	3 mg/m^3	Central nervous system effects
1242 Mercury, (organo) alkyl compounds	0.03 mg/m^3	Central nervous system effects; irritation
1243 Mesityl oxide	25 ppm	Eye and mucous membrane irrita- tion, breathing difficulty, head- ache and vertigo
1248 Methyl 2-cyanoacrylate	4 ppm	Nasal and eye irritation
1249 Methyl acetate	250 ppm	Ocular and nervous disturbances; eye, mucous membrane, upper and lower respiratory tract irritation
1252 Methyl alcohol	250 ppm	Recurrent headaches; diminution of vision
1254 Methyl chloride	100 ppm	Narcosis
1255 Methyl chloroform (1,1,1-trichloroethane)	450 ppm	Anesthesia
1258 Methyl formate	150 ppm	Visual disturbances (temporary blindness); narcotic symptoms, mucous membrane irritation; dyspnea
1261 Methyl isobutyl carbinol	40 ppm	Eye irritation

TABLE C17-2. Health Effects Supporting Final Rule STELs (continued)

H.S. Number/ Chemical Name	Final Rule STEL	Health Effects
1267 alpha-Methyl styrene	100 ppm	Eye irritation
1270 o-Methylcyclohexanone	75 ppm	Eye and respiratory irritation
1281 Morpholine	30 ppm	Irritation and harmful effects to eyes and vision
1282 Naphthalene	15 ppm	Ocular effects
1286 Nitric acid	4 ppm	Respiratory irritation
1296 Octane	375 ppm	Acute effects on nervous system
1298 Osmium tetroxide	0.006 mg/m^3	Irritation; conjunctivitis
1299 Oxalic acid	2 mg/m^3	Severe local burns to eyes, mucous membranes, and skin
1301 Ozone	0.3 ppm	Pulmonary congestion; eye, nose, and throat irritation
1304 Pentaborane	0.015 ppm	Central nervous system effects
1306 Pentane	750 ppm	Narcotic and irritative effects

TABLE C17-2. Health Effects Supporting Final Rule STELs (continued)

H.S. Number/ Chemical Name	Final Rule STEL	Health Effects
1307 2-Pentanone (MPK)	250 ppm	Narcotic effects; irritation
1309 Perchloryl fluoride	6 ppm	Respiratory irritation; fluorosis
1317 Phenylhydrazine	10 ppm	Sensitization effects
1319 Phorate (Thimet)	0.2 mg/m^3	Cholinesterase inhibition
1320 Phosdrin (Mevinphos)	0.3 mg/m^3	Cholinesterase inhibition
1321 Phosphine	1 ppm	Pulmonary irritation
1322 Phosphoric acid	3 mg/m^3	Respiratory irritation
1324 Phosphorus pentasulfide	3 mg/m^3	Respiratory irritation
1325 Phosphorus trichloride	0.5 ppm	Respiratory irritation
1338 n-Propyl acetate	250 ppm	Irritation; narcosis
1339 Propyl alcohol	250 ppm	Possible deep narcosis
1340 n-Propyl nitrate	40 ppm	Irritation; headache, nausea
1341 Propylene dichloride	110 ppm	Eye irritation; central nervous system effects

TABLE C17-2. Health Effects Supporting Final Rule STELs (continued)

H.S. Number/ Chemical Name	Final Rule STEL	Health Effects
1343 Propylene glycol monomethyl ether	150 ppm	Eye irritation
1346 Resorcinol	20 ppm	Eye and skin irritation
1366 Sodium Fluoroacetate	0.15 mg/m	Metabolic inhibition
1372 Styrene, monomer	100 ppm	Tremors with subsequent severe convulsions; pulmonary edema may follow severe single exposure
1375 Sulfur dioxide	5 ppm	Respiratory effects
1379 Sulfuryl fluoride	10 ppm	Central nervous system effects; pulmonary irritation
1387 Tetrahydrofuran	250 ppm	Narcotic and irritative effects
1397 Toluene	150 ppm	Impairment of coordination, momentary memory loss, anorexia
1398 Toluene-2,4-diisocyanate	0.02 ppm	Sensitization effects
1403 1,1,2-Trichloro-1,2,2-tri-fluoroethane	1250 ppm	Impairment of psychomotor performance
1406 Trichloroethylene	200 ppm	Narcosis

TABLE C17-2. Health Effects Supporting Final Rule STELs (continued)

H.S. Number/ Chemical Name	Final Rule STEL	Health Effects
1408 Triethylamine	15 ppm	Acute irritation of eyes, mucous membranes, and lungs
1411 Trimethylamine	15 ppm	Irritation
1424 Vinyl acetate	20 ppm	Irritation
1428 Vinylidene chloride	20 ppm	Overt toxicity
1430a Wood dust 1430b All soft and hardwoods, except Western red cedar	10 mg/m^3	Respiratory effects
1431 Xylene (o,m,p-isomers)	150 ppm	Narcosis, irritant effects
1435 Zinc chloride (fume)	2 mg/m^3	Respiratory irritation
1437 Zinc oxide fume	10 mg/m^3	Metal fume fever

BILLING CODE 4510-26-C

OSHA received comments on the issue of STELs from many rulemaking participants (Exs. 3–217, 3–623, 3–678, 3–868, 3–891, 3–902, 3–904, 3–905, 3–1008, 3–1012, 3–1053, 3–1057, 3–1099, and 3–1246; Tr. p. 3–333; Tr. pp. 10–12 to 10–14; Tr. p. 11–231), who expressed the opinion that STELs and ceiling limits should only be established when a toxicologic basis exists that demonstrates the need for a short-term limit. Many of these rulemaking participants urged OSHA not to establish STELs for substances in this rulemaking, in view of the fact that the ACGIH is still evaluating the basis for its TLV–STELs and recently deleted the TLV–STELs for a number of substances.

NIOSH (Ex. 8–47) also discussed the basis under which short-term limits (called "ceilings" by NIOSH) are appropriate:

> Ceiling values are intended to minimize toxic effects related to the peak exposure. Ceiling values are necessary when there are immediate acute responses to an air contaminant independent of the total daily dose or when chronic effects are dose-rate response related. Ceiling values are also used to minimize the total daily dose when there is intermittent occupational exposure, e.g., ethylene oxide (Ex. 8–47).

The Workers' Institute for Safety and Health (WISH) had a similar view of the need for short-term limits, and expressed the belief that OSHA should adopt some guidelines for decisionmaking in this area (Ex. 116).

In the final rule, OSHA finds that the STELs and ceilings being established reflect the concerns of rulemaking participants that short-term limits be promulgated when a toxicologic basis exists for the short-term limit. In general, OSHA is establishing STELs or ceiling limits when the toxicologic evidence for a particular substance indicates that the 8-hour TWA PEL alone would be insufficient to protect employees from experiencing adverse effects related to short-term exposure to elevated concentrations of that substance. In making these determinations, OSHA has considered the record evidence on specific short-term limits that were proposed (see discussions for individual substances).

In addition, for substances for which the ACGIH has recently proposed deleting STELs (ACGIH 1988b), OSHA has reevaluated the toxicologic basis for the STELs proposed in the NPRM. Both as a result of this analysis and in response to the record evidence on specific substances, OSHA is not establishing short-term limits, as originally proposed, for the following substances:

Acetic acid
sec-Butyl alcohol
Camphor (synthetic)
2-Chloro-6-trichloromethyl pyridine
Chlorodifluoromethane
o-Chlorotoluene
Chlorpyrifos
Clopidol
Crufomate
Cyclonite
Fluorine
Oil mist
Perchloroethylene
Phosphorus oxychloride
Picloram
Picric acid
Propionic acid
Tantalum
Vinylidene chloride
Zinc stearate

For the remaining substances for which STELs or ceiling limits were proposed, OSHA has determined that the toxicologic evidence demonstrates that a short-term limit is necessary to provide employee protection that would not otherwise be provided by an 8-hour TWA limit alone.

WISH (Ex. 116, p. 20) suggested that OSHA adopt a "generic STEL" applicable to all substances regulated by OSHA, similar to the general excursion limit recommended by the ACGIH. Another commenter, the Chemical Manufacturers Association (CMA) (Tr. pp. 10–12 to 10–14), was not in favor of such a provision because in CMA's view, a general excursion limit relates to statistical variability in sampling data rather than to toxicological factors.

In the final rule, OSHA has not established a general excursion limit that applies to all regulated substances. However, there are workplace situations where OSHA believes that worker protection requires the implementation of a STEL. For example, OSHA believes that the severity of the health effect caused by exposure and the pattern of exposure prevalent in operations involving a given substance are both factors that should be considered when determining whether a short-term limit is appropriate. OSHA concludes that, in these instances, promulgating a STEL is a necessary and appropriate measure for ensuring that workplace conditions will be maintained under a sufficient degree of control to ensure that workers are protected from experiencing serious exposure-related health effects.

18. Substances for Which OSHA Is Adding Skin Designations

For 49 substances included in this rulemaking, OSHA is adding skin designations in recognition of the capacity of these substances to be absorbed through the skin in sufficient quantities to cause systemic toxicity. Table C18–1 shows all of the substances for which the Agency is establishing skin notations.

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TABLE C18-1. List of Substances for Which OSHA Is Adding a Skin Designation

H.S. Number/ Chemical Name	CAS No.
1009 Acrylic acid	79-10-7
1051 n-Butyl alcohol	71-36-3
1055 o-sec-Butylphenol	89-72-5
1070 Carbon disulfide	75-15-0
1075 Catechol	120-80-9
1084 o-Chlorobenzylidene malononitrile	2698-41-1
1091 Chlorpyrifos	2921-88-2
1107 Cyclohexanol	108-93-0
1108 Cyclohexanone	108-94-1
1110 Cyclonite	121-82-4
1118 Diazinon	333-41-5
1129 1,3-Dichloropropene	542-75-6
1131 Dicrotophos (Bidrin)	141-66-2
1141 Dimethyl 1,2-dibromo-2,2-dichloroethyl phosphate	300-76-5
1146 Dioxathion (Delnav)	78-34-2
1152 Disulfoton	298-04-4
1156 Endosulfan	115-29-7
1160 Ethion (Nialate)	563-12-2
1173 Fenamiphos	22224-92-6
1175 Fenthion	55-38-9
1181 Fonofos	944-22-9
1184 Furfuryl alcohol	98-00-0

TABLE C18-1. List of Substances for Which OSHA Is Adding a Skin Designation (continued)

H.S. Number/ Chemical Name	CAS No.
1198 Hexafluoroacetone	684-16-2
1211 2-Hydroxypropyl acrylate	999-61-1
1220 Isooctyl alcohol	26952-21-6
1222 Isophorone diisocyanate	4098-71-9
1229 N-Isopropylaniline	768-52-5
1237 Manganese cyclopentadienyl tricarbonyl	12079-65-1
1240 Mercury (aryl and inorganic compounds)	7439-97-6
1241 Mercury (vapor)	7439-97-6
1242 Mercury (organic), alkyl compounds	7439-97-6
1244 Methacrylic acid	79-41-4
1251 Methyl acrylonitrile	126-98-7
1252 Methyl alcohol	67-56-1
1256 Methyl demeton	8022-00-2
1265 Methyl parathion	298-00-0
1271 Methylcyclopentadienyl manganese tricarbonyl	12108-13-3
1273 4,4'-Methylene bis(2-chloroaniline)	101-14-4
1313 Phenothiazine	92-84-2
1319 Phorate (Thimet)	298-02-2
1335 Propargyl alcohol	107-19-7
1364 Sodium azide	26628-22-8
1392 Thioglycolic acid	68-11-1
1394 Tin (organic compounds)	7440-31-5
1400 p-Toluidine	106-49-0

TABLE C18-1. List of Substances for Which OSHA Is Adding a Skin Designation (continued)

H.S. Number/ Chemical Name	CAS No.
1401 m-Toluidine	108-44-1
1414 Triorthocresyl phosphate	78-30-8
1426 Vinyl cyclohexene dioxide	106-87-6
1432 m-Xylene-alpha,alpha'-diamine	1477-55-0

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The ACGIH began to include skin designations for the chemicals in its list for the first time in 1961 (Stokinger 1962/Ex. 1-998). At that time, the organization stated that:

> This notation is to be interpreted simply as an indicator that skin absorption may contribute to the overall intake from exposure in addition to that from inhalation. It refers mainly to absorption from liquid contamination (Stokinger 1962/Ex. 1-998).

The ACGIH has expanded on its reasoning since the 1960s, and the preface to the most recent *Threshold Limit Values and Biological Exposure Indices for 1987-1988* (ACGIH 1987/Ex. 1-16) explains that the skin designation is designed to call attention to the need for "appropriate measures for the prevention of cutaneous absorption so that the threshold limit is not invalidated" (ACGIH 1987/Ex. 1-16, p. 7). Thus, a skin notation warns that exposure via the cutaneous route, including absorption through the eyes or mucous membranes by either inhalation or direct contact, may contribute substantially to an employee's overall exposure and cause systemic toxicity.

The ACGIH has a policy of using a dermal LD_{50} of 2 g/kg as a general cutoff for determining when to classify a substance as sufficiently absorbable to present a hazard via the percutaneous route; that is, substances having a single-dose dermal LD_{50} of less than 2 g/kg receive a skin notation, while those with dermal LD_{50}s above this cutoff do not (ACGIH 1986/Ex. 1-3, p. 332). The *Documentation* (ACGIH 1986/Ex. 1-3) contains no cutoff value for chronic dermal exposures (i.e., for toxicity resulting from repeated applications of substances to the skin).

OSHA proposed to include as paragraph 3[2] of the standard the following language regarding the use of skin notations:

> [2]. An employee's skin exposure to materials listed in the Table Z-4 with an "S" Notation shall be limited through the use of gloves, coveralls, goggles, or other appropriate personal protection equipment or method necessary to prevent possible skin absorption.

The NPRM explained further that a skin designation is employed "where the substance may be absorbed through the skin" or "where skin contact could damage or irritate the skin." In addition, the NPRM mentioned that both engineering controls and work practices may be used to limit skin exposure, but that the hierarchy of controls, i.e., a preference for engineering controls, would not be enforced in the case of skin notations.

A number or rulemaking participants (Exs. 8-44, 8-64, 3-661, 3-678, 3-683, 3-877, 3-891, 3-1008, and 3-1053; Tr. 8/1/88, pp. 304-305, 337; Tr. 8/9/88, pp. 136, 148) objected to the skin notation language contained in the Summary and Explanation section (Section VII) of the NPRM and urged OSHA to enunciate a clear and consistent policy on the use of the skin designations. According to Dr. Lawrence Hecker of Abbott Laboratories:

> In industrial hygiene practice, the use of the skin notation, as recommended by the ACGIH, long ago became a widely used indicator of chemicals for which skin absorption represents a significant route of entry for systemic effects. In general, the term has not been used to denote irritants or other materials that have their primary effects on the skin itself. Abbott Laboratories agrees with and advocates this philosophy.
>
> We recommend that the use of the skin notation be restricted to chemicals for which the skin is a significant route of entry by at least one of the following two criteria: 1. the material is highly toxic with a dermal LD_{50} value based on animal tests of 200 milligrams per kilogram or less. . . . 2. The material has exhibited clear systemic effects in people as the result of skin contact (Ex. 3-678, pp. 2-3).

The American Industrial Hygiene Association (Tr. 8/9/88, p. 148) believes that OSHA's regulatory language should stipulate that protection against skin absorption should be achieved by the "use of engineering controls and work practices, where practicable, and shall be supplemented, where necessary, by the use of suitable gloves, coveralls, goggles, or other appropriate personal protective equipment." The Eastman Kodak Company (Ex. 3-661, p. 3) states that:

> The proposed wording contains the phrase "necessary to prevent possible skin absorption," which may be interpreted as permitting no skin contact or absorption. This is more limiting than equivalent provisions in Part 1910 for control of airborne exposure and may be infeasible or impractical in many cases. OSHA should change the phrase to "necessary to minimize skin contact" or "necessary to minimize skin absorption."

In response to these comments, the Agency has developed new language for paragraph (a)(3)(ii) of § 1910.1000, which reads as follows:

> An employee's skin exposure to substances listed in Table Z-1-A with the designation "Skin" following the substance name shall be *prevented or reduced to the extent possible* through the use of gloves, coveralls, goggles, or other appropriate personal protective equipment, *engineering controls or work practices* (emphasis added).

OSHA is not requiring that engineering controls be used preferentially to protect against skin absorption; the Agency notes that this decision is consistent with 29 CFR 1910.132 and 1910.134, which require the use of engineering controls and work practices in preference to personal protective equipment only when inhalation is the route of entry.

OSHA agrees with Dr. Hecker and several other commenters that dermal irritation alone should not warrant a skin designation; instead, OSHA believes that skin designations should be used only in instances where a substance can be percutaneously absorbed in quantities sufficient to cause systemic poisoning. However, the Agency has determined that more protective policy than that advocated by Dr. Hecker should be used to decide when a skin designation is appropriate. In this rulemaking, OSHA's decision logic for establishing skin notations derives from the Agency's Hazard Communication Standard (29 CFR 1910.1200). Appendix A of that regulation defines, in measurable terms, the possible health effects that may occur in the workplace as a result of chemical exposures. These definitions set forth quantitative guidelines for determining if chemicals are "highly toxic" or merely "toxic" by the dermal route of exposure. A chemical is considered *highly toxic* via skin absorption if

> . . . [it] has a median lethal dose (LD_{50}) of 200 milligrams or less per kilogram of body weight when administered by continuous contact for 24 hours (or less if death occurs within 24 hours) with the bare skin of albino rabbits weighing between two and three kilograms each.

It is considered *toxic* via skin absorption if

> . . . [it] has a median lethal dose (LD_{50}) of more than 200 milligrams per kilogram but not more than 1,000 milligrams per kilogram of body weight when administered by continuous contact for 24 hours (or less if death occurs within 24 hours) with the bare skin of albino rabbits weighing between two and three kilograms each.

Accordingly, OSHA has determined that a skin notation is necessary for substances that have median lethal dose (LD_{50}) values in rabbits on single-dose applications of less than 1000 mg/kg. In addition, in very rare cases where available data (for any species) indicate that dermal contact results in a systemic dose that is equivalent to or greater than the dose that would be permitted by the PEL via inhalation, OSHA believes that a skin designation is warranted. In addition to this animal evidence, OSHA believes that the availability of human data demonstrating that systemic injury has occurred as a result of skin

absorption is sufficient evidence that a skin notation is warranted.

OSHA has followed these guidelines in establishing skin designations in this rulemaking. As a consequence, the skin designations proposed in the NPRM for eight substances have been deleted in the final rule because the Agency found in its subsequent review of the record that the substances failed to meet the requisite animal and/or human criteria, thus demonstrating an absence of significant risk as a result of percutaneous absorption:

Allyl glycidyl ether;
Captafol;
2-N-Dibutylaminoethanol;
Diethylene triamine;
Hexachlorobutadiene;
Propyl alcohol;
1,2-Propylene glycol dinitrate; and
1,2,3-Trichloropropane.

The evidence that OSHA finds acceptable in humans is, of course, impossible to quantify in terms of laboratory measurements. For example, much of the information on the dermal toxicity of substances in humans is anecdotal and derives from accidental poisonings. Nevertheless, OSHA believes that such evidence should constitute a sufficient basis for the establishment of a skin designation.

In addition, OSHA has added skin designations to a number of substances where none were originally proposed if commenters submitted evidence to the record that supported these additions. These substances are:

Acrylic acid;
Carbon disulfide;
Catechol;
Disulfoton;
Isophorone diisocyanate;
Mercury (aryl and inorganic);
Methacrylic acid; and
Sodium azide.

Substances for which the ACGIH Has Deleted the Skin Notation

For four substances, the ACGIH has deleted the skin notations that appeared in the 1968 edition of the *Documentation* and that were subsequently adopted by OSHA under the Section 6(a) mechanism in 1971. Table C18-2 shows these chemicals

TABLE C18-2. List of Substances for Which the ACGIH Has Deleted the Skin Notation

H.S. Number/ Chemical Name	CAS No.
1113 DDT	50-29-3
1149 Dipropylene glycol methyl ether	34590-94-8
1197 Hexachloroethane	67-72-1
1303 Paraquat, respirable dust	4685-14-7

OSHA is retaining the skin notations for these four substances (DDT, dipropylene glycol methyl ether, hexachloroethane, and paraquat). The Agency believes that deletion of these designations would consititute an increase in the level of exposure permitted and would thus decrease the extent of worker protection provided by OSHA. In accordance with principles established by OSHA (see the preamble for the final revisions to the cotton dust standard, 50 *FR* 51120 *et seq.*), the Agency must demonstrate, on the basis of human data, that deleting these skin designations, which were established under the 6(a) mechanism, will not pose a significant risk to exposed workers. The discussion below describes the ACGIH's reasons for recommending deletion of these notations.

The evidence on which the ACGIH based its decision to delete skin notations for the four chemicals in question is primarily animal evidence. For DDT and hexachloroethane, the ACGIH deleted the skin designation based on the relatively low dermal toxicity demonstrated by these substances in animal studies. OSHA concludes, however, that the absence of significant risk to humans via dermal absorption has not been sufficiently shown for either DDT or hexachloroethane. In the case of the latter substance, there are no human data with which to demonstrate an absence of risk, and to decrease the

amount of protection provided by the existing limit would not be appropriate.

For the two remaining substances in this group, paraquat and dipropylene glycol methyl ether (DPGME), the skin notation was deleted because the ACGIH believes that the substance does not, in the case of paraquat, "penetrate the unbroken or uninjured skin" or believes, as in the case of DPGME, that the substance is "practically nontoxic . . . by the dermal route for rabbits" (ACGIH 1986/Ex. 1–3). However, OSHA notes that at high doses paraquat does "injure and break down dermal barriers" and gain entry to the body. The *Documentation* records the case of a 44-year-old man who died of respiratory insufficiency after he was poisoned by the percutaneous absorption of an acutely toxic quantity of undiluted paraquat (ACGIH 1986/Ex. 1–3). Therefore OSHA is retaining the skin notation for paraquat.

In addition, the Agency notes that DPGME, applied essentially according to the method prescribed by Draize et al., was absorbed in sufficient quantities through rabbit skin to result in transient narcosis (*Patty's Industrial Hygiene and Toxicology,* Vol. 2C, p. 3990, Clayton and Clayton 1982). However, topical administration of only 10 mg/kg DPGME five times per week for 13 weeks to shaved rabbit skin caused six deaths among seven animals (*Chemical Hazards of the Workplace,* 2nd ed., p. 221, Proctor, Hughes, and Fischman 1988). In light of this evidence, OSHA believes that deleting the skin notation for DPGME would be inappropriate, since the absence of significant risk in humans cannot be clearly demonstrated. Consequently, OSHA is retaining the skin notation for this substance.

In accordance with the principles stated in the cotton dust preamble (50 *FR* 51120), OSHA does not find the evidence adduced by the ACGIH sufficient to provide a basis for the deletion of the skin notations for this group of four substances. The Agency concludes that deleting the skin notation from the limits for DDT, dipropylene glycol methyl ether, hexachloroethane, and paraquat will not ensure that workers are protected against the significant risk potentially posed by percutaneous absorption of these substances.

D. References

OSHA will publish in the Federal Register in the near future a reference list that includes all of the additional references cited in this preamble. This list will supplement the reference list that appeared in the June 7 notice of proposed rulemaking (NPRM).

VII. FEASIBILITY AND REGULATORY ANALYSES

A. Detailed Table of Contents

Note.—Supplements 2 through 4 are available in the OSHA Docket Office, Room N2634, U.S. Department of Labor, 200 Constitution Avenue NW., Washington, DC 20210, (202) 523–7894

B. Introduction and Executive Summary

Introduction

The Occupational Safety and Health Administration (OSHA) is amending its existing air contaminant standards at 29 CFR 1910.1000, Tables Z–1, Z–2, and Z–3. The amendments provide more protective permissible exposure limits (PELS) for about 230 substances, and set new exposure limits for 165 substances currently not regulated by OSHA. The PELs include time-weighted average limits, short term exposure limits, ceiling limits, and, in some cases, skin designations. No changes are being made to the PELs for 30 substances.

Background. Congress enacted the Occupational Safety and Health Act of 1970 (the Act) to achieve several goals, one of which was to protect workers from occupational health hazards. Congress acknowledged the role of occupational exposure in the development of diseases, and addressed in the Act the need to quickly establish minimum health standards to control exposure to hazardous substances. To accomplish Congress' intent, OSHA adopted initial exposure limits for approximately 430 chemicals. Four hundred of these exposure limits were based on the recommendations of the American Conference of Governmnental Industrial Hygienists (ACGIH), and 21 were from the American Standards Association (now called the American National Standards Institute). The list of exposure limits was to be updated, improved, and expanded as new

knowledge and techniques were developed. To date OSHA has promulgated extensive health standards for only 24 individual chemicals. The rulemaking under consideration here would set exposure limits for about 430 chemicals based on the 1987–88 Threshold Limit Values of the ACGIH, and recommendations of the National Institute for Occupational Safety and Health (NIOSH) of the U.S. Department of Health and Human Services.

The OSH Act requires the Agency to consider the feasibility of proposed and final standards. Executive Order 12291 (46 FR 13197) requires that a regulatory analysis be conducted for any rule having major economic consequences on the national economy, individual industries, geographical regions, or levels of government. The Regulatory Flexibility Act (5 U.S.C. 601 et seq.) similarly requires OSHA to consider the impact of the proposed and final regulations on small entities. This analysis covers these requirements.

Approach. Because this rulemaking involves about 430 chemicals, OSHA has prepared the regulatory impact analysis in two phases. Phase I involved the use of a number of secondary data bases to collect information on the chemicals to be regulated and the industries in which they are used. These data bases provided information on the toxicity and health effects of exposure to the chemicals, and current information on engineering controls in use and emergency response procedures. Two data bases provided information on employee exposures. The 1982 National Occupational Exposure Survey (NOES) was based on a sample of about 4,500 businesses. The data base developed from this survey contains an estimate of the number of persons occupationally exposed to hazardous substances by Standard Industrial Classification (SIC). The second data base was OSHA's Integrated Management Information System (IMIS). The IMIS contains the results of air samples taken since 1979 by OSHA industrial hygienists in the course of compliance inspections. Both the NOES and IMIS data bases provided valuable information on the nature and extent of employee exposures to the substances to be regulated; however, they did not provide complete information on all substances. Supplementary information was obtained from industrial hygienists and engineers. These experts identified exposure controls in use and the number and size of plants most likely to be affected by this rulemaking. These sources have provided OSHA with a substantial body of information on chemical use, exposures and controls.

Phase II of the data collection effort involved a sampling survey of about 5,700 firms in industries where chemical exposures were believed to pose potential problems. The survey, conducted during the first part of 1988, gathered data on chemicals, processes, exposures and controls currently in use. These additional data have permitted OSHA to refine the Phase I preliminary estimates of technical and economic feasibility. In addition, site visits to 90 firms were conducted to verify the data collected on chemicals, processes, controls, and employee exposures.

OSHA has used contractors to assist in these data collection efforts. Three contractors have supplied expert knowledge on the industries affected and the engineering controls needed to reach the proposed exposure levels. These contractors are Kearney/Centaur Division of A.T. Kearney, Meridian Research, and CONSAD. Fu Associates provided data base management support during all phases of the project. Washington Consulting Group designed the sample for the surveyed firms and KCA Research conducted the telephone interviews of these firms.

Employee Exposure and Benefits

Revising OSHA's Z-Table limits for hazardous substances is expected to result in reduced risk of chemically-related disease among exposed employees. Exposure to substances included in the rulemaking has been associated with a variety of adverse health effects, including impairment of organ system functions, mucous membrane irritation, neuropathy, narcosis, allergic sensitization, respiratory disease, cardiovascular disease, and cancer.

Using data from OSHA's IMIS system and information collected from the survey of about 5,700 establishments, OSHA estimates that over 21 million employees are potentially exposed to hazardous substances in the workplace. OSHA also estimates that over four and one-half million employees are currently exposed above the proposed exposure limits for these substances. Table B–1 summarizes OSHA's estimates of the number of workers currently at risk of adverse health effects. OSHA estimates that promulgation of the final rule's exposure limits will result in a potential reduction of over 55,000 work-related illness cases per year, over 23,000 lost-workday illness cases per year, and almost 520,000 lost workdays due to illness per year. OSHA's estimate is that industry compliance with the final rule's exposure limits will result in a reduction of an average of 683 fatalities annually that are caused by exposure to substances that cause cancer, respiratory disease, cardiovascular disease, or liver or kidney disease.

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TABLE B-1

Estimated Number of Workers Potentially at Risk of Experiencing Adverse Effects, by Type of Adverse Effect*

ADVERSE HEALTH EFFECT	NO. OF WORKERS POTENTIALLY EXPOSED TO SUBSTANCES ASSOCIATED WITH EFFECT, MINIMUM ESTIMATE	NO. OF WORKERS POTENTIALLY EXPOSED TO SUBSTANCES ASSOCIATED WITH EFFECT, MAXIMUM ESTIMATE	NO. OF WORKERS EXPOSED ABOVE FINAL LIMITS FOR SUBSTANCES, MINIMUM ESTIMATE	NO. OF WORKERS EXPOSED ABOVE FINAL LIMITS FOR SUBSTANCES, MAXIMUM ESTIMATE
PHYSICAL IRRITANT EFFECTS	3,375,472	3,889,261	222,191	222,191
ODOR EFFECTS	519,318	521,938	3,597	3,597
SYSTEMIC TOXICITY	4,305,578	5,038,573	457,104	490,282
MUCOUS MEMBRANE IRRITATION	10,730,691	14,906,090	789,461	1,141,133
METABOLIC INTERFERENCES	4,015,702	4,205,530	1,233,413	1,241,564
LIVER/KIDNEY DISEASE	3,292,993	3,806,226	536,945	546,429
OCULAR DISTURBANCES	2,482,449	2,569,950	83,272	110,560
RESPIRATORY DISEASE	4,231,235	4,782,280	1,405,501	1,568,579
CARDIOVASCULAR DISEASE	166,077	166,868	44,403	44,403
NEUROPATHY	2,212,358	2,463,583	379,974	401,576
NARCOSIS	6,966,024	10,520,982	941,472	1,073,717
CANCER	1,712,799	1,851,342	465,013	528,650
ALLERGIC SENSITIZATION	2,545,551	2,648,973	305,955	305,955

* Double counting of employees simultaneously exposed to more than one substance in different adverse health effects categories prevents the summation of workers exposed to all adverse health effects in this table.

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Nonregulatory Alternatives

OSHA believes that there are no nonregulatory alternatives that adequately protect most workers from the adverse health effects associated with exposure to the chemicals under consideration. OSHA believes that the tort liability laws and Workers' Compensation do not provide adequate worker protection due to market imperfections. Some employers have not complied with the standards recommended by professional organizations. The deleterious health effects resulting from continued high levels of exposure to hazardous substances require a regulatory solution, and the OSH Act requires the Agency to protect workers' health.

Technological Feasibility

Consistent with OSHA regulations and policy, engineering controls and work practices are preferred over personal protective equipment to control employee exposures to airborne contaminants.

Engineering controls involve the use of a local exhaust ventilation, general ventilation, isolation of the worker and enclosure of the source of emissions, process modifications, equipment modifications, and substitution of non-hazardous or less hazardous chemicals. These methods may be used alone or in combination, depending upon the industrial processes involved. These controls are widely used and will effectively control exposures either by themselves, or coupled with changes in work practices.

Perhaps the most widely used technique for controlling chemical exposure is the use of ventilation. General ventilation uses the movement of air within the general work space to displace or dilute the contaminant with fresh outside air. General ventilation may not be the preferred control method, however, due to the large volumes of air movement required. Local exhaust ventilation uses much smaller volume of air and controls emissions at the point or source from which contaminants are generated.

Isolation involves placing a physical barrier between the hazardous operation and the worker. Many modern, automated manufacturing processes are now fully enclosed in ventilated cabinets. The effectiveness of such a control technique depends on the frequency with which the workers have to enter the enclosure during normal operations. In other situations, the worker, rather than the process or machine, can be placed in an enclosure having a controlled atmosphere. Many processes which involve potential chemical exposures are operated remotely by operators from air-conditioned booths isolated from the hazardous materials.

Substitution refers to the replacement of a toxic chemical in a particular process or work area with another, less toxic or non-toxic product. Properly applied, substitution can be a very effective control technique. However, care must be taken to ensure that the proposed substitute performs in a similar manner to the product being replaced. In addition, it is essential that the substitute be carefully evaluated to ensure that in controlling one hazard, another different hazard is not inadvertently introduced. The substitute must also be compatible with existing manufacturing equipment and processes.

The success of these engineering control techniques will depend on the physical properties of the chemicals and emissions encountered (boiling point, vapor pressure, etc.) and the process operating conditions. In some cases, particularly with cleaning solvents, substitution may provide the quickest and most effective means of reducing exposure. In other situations, a major effort may be required to alter processes or install or expand local or general dilution ventilation.

OSHA has found that engineering controls and improved work practices are available to reduce exposure levels to the new levels in almost all circumstances. Standard controls have been adapted in numerous situations to solve situation-specific problems in all of the industry sectors affected. Detailed industry-specific illustrations of this point are presented in the Technological Feasibility Chapter of this Feasibility and Regulatory Analysis. OSHA does recognize, however, that in some circumstances, respiratory protection may be necessary to complement engineering controls and that respiratory protection may also be necessary to achieve compliance in some specific operations in some industries.

Costs of Compliance

Costs of compliance with the proposed rulemaking would result from industry actions to lower workers' chemical exposures to the levels promulgated in the final rule. The 1988 sample survey of almost 5,700 firms was drawn from a universe of over one million firms potentially affected by the rule. Table B–5 at the end of this section presents a list of industries included in the analysis.

Survey respondents verified the number of work stations and workers related to each process, the process location and configuration, the controls already in place, and potential chemical exposures above new proposed levels. Process controls in place were compared to a list of control designs needed to limit exposures to the new, lower levels. Where the required controls were not reported to be in place, a compliance cost per work station was assigned. Process control costs were summed per establishment and certain maintenance workers were assigned a respirator cost. Costs for the surveyed establishments were then weighted (by SIC and size) to represent compliance costs for the universe of affected plants.

The survey found that over 500,000 establishments (of the 1,101,600 establishments covered by the survey) reported using the chemicals being regulated. Of this number, 131,005 would incur some costs to comply with the new limits. The total estimated annualized capital plus annual operating costs are $787.98 million. Table B–2 presents the annual cost by industry sector and the average per plant annual cost for large and small (fewer than 20 employees) plants.

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TABLE B-2

AVERAGE PER PLANT ANNUAL COSTS AND NUMBERS OF AFFECTED PLANTS (a)

SIC (b)	SIC DESCRIPTION	ANNUAL COST	TOTAL # OF PLANTS	# OF AFFECTED PLANTS	% AFFECTED	AVERAGE COST PER AFFTED PLANT	AVERAGE COST PER LARGE AFFTED PLANT	AVERAGE COST PER SMALL AFFTED PLANT
20	FOOD PROD. (c)	$33,493,100	29,000	4,932	16.98%	$6,800	$13,000	$3,600
21	TOBACCO (c)	$19,700	200	3	1.39%	$6,600	$6,600	$0
22	TEXT. MILL (c)	$29,478,400	11,000	2,765	25.08%	$10,700	$21,400	$3,700
23	APPAREL PROD. (c)	$31,744,200	30,000	6,179	20.57%	$5,100	$11,500	$2,000
24	LUMBER & WOOD	$56,720,800	27,100	18,427	68.00%	$3,100	$4,200	$2,700
25	FURNITURE	$21,075,800	12,700	5,062	40.00%	$4,200	$12,400	$1,800
26	PAPER PROD.	$30,998,700	7,000	3,518	50.00%	$8,800	$15,200	$800
27	PRINTING & PUB.	$33,754,500	60,300	3,597	6.88%	$9,400	$6,200	$10,600
28	CHEMICAL PROD.	$35,454,700	16,400	3,007	18.31%	$11,800	$16,200	$5,400
29	PETRO. REFINING	$23,686,000	2,300	306	13.25%	$77,400	$109,600	$700
30	RUBBER & PLASTICS	$111,093,400	15,100	3,562	26.22%	$31,200	$27,000	$35,100
31	LEATHER PROD.	$2,414,700	2,300	300	13.46%	$8,000	$10,400	$6,400
32	STONE & CLAY	$22,457,800	15,900	3,267	22.80%	$6,900	$12,200	$3,400
33	PRIM. METAL	$70,957,600	8,000	2,411	30.03%	$29,400	$41,900	$6,200
34	FAB. METALS	$39,419,700	37,300	4,397	14.50%	$8,600	$15,800	$3,800
35	MACHINERY	$45,206,600	64,400	6,801	10.56%	$7,800	$14,600	$3,000
36	ELEC. MACH.	$20,667,500	21,600	2,359	10.92%	$7,800	$14,500	$3,000
37	TRANS. EQUIP.	$49,792,400	13,600	4,979	36.56%	$10,000	$11,800	$8,800
38	INSTRUMENTS	$9,633,500	12,000	1,289	10.74%	$7,800	$14,500	$3,000
39	MISC. MANUF.	$15,842,600	25,300	2,649	10.47%	$7,800	$14,600	$3,000
40	R.R. TRANS.	$1,083,400	400	93	20.86%	$11,700	$11,700	$0
45	AIR TRANS.	$3,740,500	5,500	320	5.79%	$11,700	$11,700	$0
47	TRANS. SERV.	$3,789,400	26,200	324	1.24%	$11,700	$11,700	$0
49	ELEC. GAS. SAN.	$38,009,300	15,800	3,485	22.24%	$10,900	$17,000	$3,600
50	WHOLESALE TRADE	$2,995,300	5,800	801	13.78%	$3,400	$6,200	$2,900
51	WHOLESALE, NON-DUR	$14,215,800	33,600	4,436	13.22%	$3,400	$6,200	$2,900
55	AUTO DEALERS	$13,550,500	165,800	24,847	14.99%	$360	$2,000	$300
72	PERSONAL SRV.	$10,872,100	95,500	5,217	5.47%	$2,200	$6,000	$1,000
73	BUSINESS SRV.	$2,422,100	12,100	800	6.61%	$2,200	$8,300	$1,500
75	AUTO REPAIR	$6,143,500	91,500	8,351	9.13%	$600	$3,500	$300
76	MISC. REPAIR SRV.	$2,809,900	15,100	1,163	11.56%	$2,400	$12,400	$2,100
80	HEALTH SERV. (c)	$4,439,400	222,800	1,158	0.52%	$3,800	$12,500	$2,100
TOTAL		$787,982,900	1,101,600	131,005	11.89%	$6,000	$13,000	$3,100

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

(a) Costs were calculated by annualizing the capital cost over the projected life of the equipment (10 years) using a 10 percent cost of capital and adding an annual operating and maintenance cost estimated at 10 percent of the capital cost.

(b) Industry sectors not identified in this table include industries with no major cost impact expected, the construction industry, which will be the subject of a separate regulatory analysis, and industries such as mining, over which OSHA has no jurisdiction.

(c) Costs in these sectors were based on expert judgement and secondary data collection.

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Among all industry sectors the average annual cost per affected establishment will be $6,000.

Economic Impact

OSHA prepared two estimates of the economic effects of this regulation on potentially affected firms. The two estimates were based upon No Cost-Passthrough ("worst case") and Total Cost-Passthrough ("best case") scenarios.

In the first scenario it was assumed that all compliance costs would be absorbed by firms in the form of reduced profits. Table B–3 contains a summary of this "worst case" analysis. Under this scenario, the estimated average percent reduction in profits for all affected firms was less than one percent. The estimated reduction in profit of 2.3 percent for SIC 30 Rubber and Plastics was the highest among all industries.

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TABLE B-3

ECONOMIC EFFECTS: NO-COST PASSTHROUGH SCENARIO[1]

SIC	Industry	Annual Costs[2] ($ millions)	Total Sales[3] ($ millions)	R.o.R. on Sales (%)[4]	Pre-Reg Profits ($ m)	Post-Reg Profits ($ m)	% Change in Profits
20	FOOD PROD.	33.49	353,780.38	1.9	8,008.04	7,986.29	- 0.2715
21	TOBACCO	0.02	74,030.13	5.3	3,923.60	3,923.59	- 0.0003
22	TEXT. MILL	29.48	60,735.22	2.7	1,765.42	1,747.59	- 1.0100
23	APPAREL PROD.	31.74	74,474.65	2.8	1,813.22	1,793.56	- 1.0845
24	LUMBER & WOOD	56.72	57,994.48	3.9	1,974.51	1,931.92	- 2.1574
25	FURNITURE	21.08	37,648.27	3.5	1,411.02	1,398.82	- 0.8645
26	PAPER PROD.	31.00	103,694.14	3.7	3,778.20	3,761.12	- 0.4519
27	PRINTING & PUB.	33.75	134,830.21	4.8	6,471.85	6,444.77	- 0.4185
28	CHEMICAL PROD.	35.45	272,759.67	3.7	11,738.80	11,717.79	- 0.1790
29	PETRO. REFINING	23.69	196,400.57	2.7	4,964.85	4,952.04	- 0.2579
30	RUBBER & PLASTICS	111.09	86,538.58	4.3	3,423.75	3,343.76	- 2.3361
31	LEATHER PROD.	2.41	15,449.56	2.6	401.69	400.03	- 0.4127
32	STONE & CLAY	22.46	46,094.04	4.1	1,954.99	1,940.97	- 0.7170
33	PRIMARY METALS	70.96	112,564.26	3.3	3,714.62	3,674.83	- 1.0712
34	FAB. METALS	39.42	150,146.41	4.0	6,005.86	5,981.33	- 0.4084
35	MACHINERY	45.21	345,144.89	5.1	17,602.39	17,573.57	- 0.1637
36	ELEC. MACH.	20.67	245,982.70	5.0	12,299.14	12,286.86	- 0.0998
37	TRANS. EQUIP.	49.79	365,427.20	3.9	14,520.25	14,485.24	- 0.2411
38	INSTRUMENTS	9.63	83,359.57	4.9	3,373.26	3,367.32	- 0.1763
39	MISC. MANUF.	15.84	41,870.30	4.4	1,788.56	1,778.14	- 0.5825
40	R.R. TRANS.	1.08	43,869.14	10.0	3,969.62	3,969.04	- 0.0147
45	AIR TRANS.	3.74	109,538.08	3.6	3,251.40	3,249.38	- 0.0621
47	TRANS. SERVICES	3.79	12,254.96	2.7	582.18	580.13	- 0.3515
49	ELEC., GAS & SAN.	38.01	300,254.83	7.0	21,017.84	20,994.71	- 0.1100
50	WHOLESALE TRADE[5]	3.00	13,853.52	2.0	277.07	274.56	- 0.9048
51	WHOLESALE, NON-DUR	14.22	113,848.20	1.5	1,726.26	1,718.59	- 0.4447
55	AUTO DEALERS	13.55	341,574.50	1.9	6,489.92	6,480.69	- 0.1422
72	PERSONAL SERV.	10.87	24,270.74	7.3	1,771.76	1,763.60	- 0.4606
73	BUSINESS SERV.	2.42	22,165.94	6.6	1,462.95	1,460.94	- 0.1375
75	AUTO REPAIR	6.14	45,750.92	5.1	2,492.19	2,488.29	- 0.1563
76	MISC. REPAIR SERV.	2.81	2,665.52	5.5	146.60	144.36	- 1.5298
80	HEALTH SERVICES	4.44	170,234.25	4.5	7,807.72	7,804.54	- 0.0406

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

Notes:
1. All values in 1985 dollars.
2. Reproduced from Table G-1.
3. Dun and Bradstreet, Dun's Marketing Identifiers (DMI) Database.
4. Rate of Return on Sales, Dun and Bradstreet, Industry Norms Database.
5. Consists of SIC 5093 (scrap and waste materials) only.

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In the second scenario it was assumed that all compliance costs would be passed on to the consumer in the form of higher prices. The potential price increase for an industry sector at the two-digit SIC level was estimated by dividing the sector's compliance cost by its total sales. In this scenario, there would be little impact on market prices; none of the estimated price increases exceeded one-half of one percent (see Table B–4).

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Table B-4

ECONOMIC EFFECTS: TOTAL-COST PASSTHROUGH

SIC	Industry	Annual Costs ($ millions)	Total Sales ($ millions)	Costs as a Percent of Sales
20	FOOD PROD.	33.49	353,780.38	0.0095
21	TOBACCO	0.02	74,030.13	0.0000
22	TEXT. MILL	29.48	60,735.22	0.0485
23	APPAREL PROD.	31.74	74,474.65	0.0426
24	LUMBER & WOOD	56.63	57,994.48	0.0978
25	FURNITURE	26.28	37,648.28	0.0560
26	PAPER PROD.	33.00	103,694.14	0.0299
27	PRINTING & PUB	34.39	134,830.21	0.0250
28	CHEMICAL PROD.	38.87	272,759.67	0.0130
29	PETRO. REFINING	23.91	196,400.57	0.0121
30	RUBBER & PLASTICS	121.93	86,538.58	0.1284
31	LEATHER PRODUCTS	2.66	15,449.56	0.0156
32	STONE & CLAY	25.83	46,094.04	0.0487
33	PRIM. METALS	78.24	112,564.26	0.0630
34	FAB. METALS	53.51	150,146.41	0.0263
35	MACHINERY	50.00	345,144.89	0.0131
36	ELEC. MACH.	23.30	245,982.70	0.0084
37	TRANS. EQUIP.	49.79	365,427.20	0.0136
38	INSTRUMENTS	10.75	83,359.57	0.0116
39	MISC. MANUF.	17.29	41,870.30	0.0378
40	R.R. TRANS.	1.09	43,869.14	0.0025
45	AIR TRANS.	3.76	109,538.08	0.0034
47	TRANS. SERVICES	3.81	12,254.96	0.0309
49	ELEC., GAS & SAN.	37.83	300,254.83	0.0127
50	WHOLESALE TRADE[1]	3.13	13,853.52	0.0216
51	WHOLESALE, NON-DUR.	14.80	113,848.20	0.0125
55	AUTO DEALERS	22.72	341,574.50	0.0040
72	PERSONAL SERVICES	10.87	24,270.74	0.0448
73	BUSINESS SERVICES	2.42	22,165.94	0.0109
75	AUTO REPAIRS	10.25	45,750.92	0.0134
76	MISC. REPAIR SERV.	4.86	2,665.52	0.1054
80	HEALTH SERVICES	4.44	170,234.25	0.0026

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

Notes: 1. Consists of SIC 5093 (scrap and waste materials) only.

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Based on this analysis, OSHA concludes that the final standard is economically feasible for each sector. The impact on prices is slight and, even in the worst cases, the reductions in profitability are small.

Regulatory Flexibility Analysis

In accordance with the Regulatory Flexibility Act (Pub. L. 96–353, 94 Stat. 1664) [5 U.S.C. 601 et seq.], OSHA has made a preliminary assessment of how this rulemaking will affect large and small establishments. The results of this preliminary assessment indicate that some small establishments may experience some adverse impact. The smaller profit margins of some small establishments may make it difficult for them to absorb increases in compliance costs. An important ameliorating factor for each affected firm will be its ability to pass through additional costs to the consumer. The ability of individual firms to do this will be dependent upon product demand elasticities. It is expected that all impacted firms will be able to pass through some portion of their increased costs.

Environmental Impact

The standard has been reviewed in accordance with the requirements of the National Environmental Policy Act of 1969 (NEPA), the Council on Environmental Quality NEPA regulations, and the Department of Labor's NEPA compliance procedures and is not anticipated to have significant impact on the external environment.

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TABLE B-5

SIC GROUPS COVERED IN THE OSHA ANALYSIS

Division D. Manufacturing	
	Major Group 20. Food and kindred products
	Major Group 21. Tobacco manufactures
	Major Group 22. Textile mill products
	Major Group 23. Apparel and other finished products, made from fabrics and similar materials
	Major Group 24. Lumber and wood products, except furniture
	Major Group 25. Furniture
	Major Group 26. Paper and allied products
	Major Group 27. Printing, publishing, and allied industries
	Major Group 28. Chemicals and allied products
	Major Group 29. Petroleum refining and related industries
	Major Group 30. Rubber and miscellaneous plastics products
	Major Group 31. Leather and leather products
	Major Group 32. Stone, clay, glass, and concrete products
	Major Group 33. Primary metal industries
	Major Group 34. Fabricated metal products, except machinery and transportation equipment
	Major Group 35. Machinery, except electrical
	Major Group 36. Electrical and electronic machinery, equipment, and supplies
	Major Group 37. Transportation equipment
	Major Group 38. Measuring, analyzing, and controlling instruments; photographic, medical and optical goods; watches and clocks
	Major Group 39. Miscellaneous manufacturing industries
Division E. Transportation, Communications, Electric, Gas, and Sanitary Services	
	Major Group 40. Railroad transportation
	Major Group 45. Transportation by air
	Major Group 47. Transportation services
	Major Group 49. Electric, gas, and sanitary services

TABLE B-5

SIC GROUPS COVERED IN THE OSHA ANALYSIS
(CONTINUED)

Division F.	Wholesale Trade
	Major Group 50. Wholesale trade - durable goods
	Major Group 51. Wholesale trade - nondurable goods
Division G.	Retail Trade
	Major Group 55. Automotive dealers and gasoline service stations
Division I.	Services
	Major Group 72. Personal services
	Major Group 73. Business services
	Major Group 75. Automotive repair, services, and garages
	Major Group 76. Miscellaneous repair services
	Major Group 80. Health services

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis, as derived from Standard Industrial Classification Manual 1972, Executive Office of the President -- Office of Management and Budget.

The listing excludes the construction industry (SICs 15, 16, and 17) which will be the subject of a separate regulatory analysis.

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C. Survey of Affected Industries

Chemicals and other hazardous substances are present to some degree in all industries. However, some industry sectors use chemicals more extensively than others and have controls in place which do not always reduce workers' exposures below permissible exposure levels. This chapter presents an overview of those industries which OSHA believes may experience costs and benefits as a result of this rulemaking. In order to estimate and quantify the potential impact of the rule, a sample survey of about 5,700 establishments was conducted during the first part of 1988. The results of the survey provided the basis for the cost and benefit estimates presented in this Feasibility and Regulatory Analyses.

Table C–1 at the end of this chapter shows establishment and employment data for the industries where OSHA expects costs and benefits. In order to determine which industries to include in the sample survey, OSHA relied primarily on two data sources: 1) the NIOSH National Occupational Exposure Survey (NOES) of 1982 and supplementary information from the NIOSH 1972 survey; and 2) data in the OSHA Integrated Management Information System (IMIS). The 1982 NOES data base contains a sample of the number of persons exposed by substance and industry from almost 4,500 businesses in 98 different geographic areas in the United States. OSHA's IMIS contains the results of exposure samples taken since 1979 by industrial hygienists during the course of compliance inspections. Using these two data bases, industries which are likely to use the substances in this rulemaking at levels which might exceed the proposed exposure limits were identified.

As a check on this list of industries, OSHA contracted with about one dozen industrial hygienists and chemical engineers to review the list. Based on their professional knowledge, these experts verified the industries with potential exposure problems. The final list of industries selected for the sample survey included over 30 two-, three-, and four-digit SICs where it is believed that chemical exposures potentially exceed the new or revised levels.

Industry sectors not included in the survey are those where OSHA believes there is little potential chemical exposure or where existing exposures are well controlled. Industries which were not surveyed for these reasons included finance, real estate, insurance and most service and retail trade sectors. The construction industry was also excluded and will be the subject of a separate rulemaking action. Industries such as mining and certain transportation sectors were not included since other agencies have safety and health enforcement jurisdiction. Certain industry sectors including textile, apparel, food and tobacco products are expected to incur some costs as a result of this rulemaking, but these were not included in the sample survey. The reasons for not including these sectors in the survey were restraints on the sample size, relatively low hazardous substance exposure levels, and the availability of adequate information on the engineering controls currently in use in these industries.

Industrial hygienists and engineers under contract to OSHA also identified the processes used in the industries surveyed, and the chemicals used in those processes. Expected levels of exposure and the number of employees potentially exposed were estimated. The list of processes and chemicals determined to be in common use in each industry sector was subsequently verified in the sample survey.

Establishments to be surveyed were selected based on a statistical sample of all establishments in the surveyed industry sectors. For each SIC, establishments were selected from four size categories.

(a) 0–19 employees
(b) 20–99 employees
(c) 100–249 employees
(d) 250 or more employees

This permitted analysis of the effects of the rulemaking by establishment employment size.

About 5,000 completed responses were required to obtain statistically valid results. The field survey was conducted by KCA Research using Computer-Assisted Telephone Interviewing (CATI). Trained interviewers requested data from each establishment regarding production employment, chemical usage, and exposure guidelines in use. Respondents were asked to verify the presence or absence of chemicals and processes believed to be found in establishments in their industry, and were asked to volunteer information on other chemicals not included on the interviewers' "prompt" list of chemicals in use. For each chemical present, the respondent was asked about amounts used, employee exposure levels, and processes where used. For each process, the respondent was asked questions concerning its configuration, frequency of use, and the types of controls and personal protective equipment in use. This information was used to develop the estimates of costs and benefits presented in this RIA.

Supplement 1 contains a technical summary of the survey and Supplement 4 contains tabulations of the survey results. (Survey results include some responses from SIC 44—Water Transportation and SIC 46—Pipelines. These were included prior to a determination that the SICs included industries not within the scope of this rulemaking, or where other agencies have jurisdiction.) The results generally corroborated the preliminary assessments of potential industry exposures and overexposure to chemicals and provided a general picture of workers' exposure in these industries. In the sample of about 5,700 firms, over one-half reported chemicals being used in the workplace. Most of the firms which reported no chemical usage were small administrative or distribution units of multi-plant companies. Among the firms surveyed which use chemicals, almost one-third use specific exposure standards as targets for maintaining workers' exposure. The OSHA PELs are used by 59 percent of firms with specific exposure standards, ACGIH TLVs are used by 22 percent and the NIOSH RELs by one percent. Table C–2 shows the distribution of adopted exposure standards by surveyed industry groups. Over one-third of all firms reported that they have a hazard communication training program; however, less than one-half of the firms using chemicals reported having a hazard communication program (see Table C–3).

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NUMBER OF FIRMS WITH CHEMICALS USING SPECIFIC EXPOSURE STANDARDS

CELL	OSHA PEL'S	NIOSH REL'S	ACGIH TLV'S	OTHER	NONE	DON'T KNOW REFUSED	TOTAL
CELL 1 SIC'S 243	1,133	215	307	1,109	6,238	397	10,137
CELL 2 SIC'S 245	282	16	16	35	437	20	756
CELL 3 SIC'S 249	932	7	32	326	1,752	169	3,582
CELL 4 SIC'S 25	2,266	9	465	1,477	3,648	0	8,361
CELL 5 SIC'S 26	1,381	11	176	51	1,273	256	3,498
CELL 6 SIC'S 27	4,980	362	2,297	7,148	24,008	3,438	45,757
CELL 7 SIC'S 281	705	32	245	35	268	2	1,298
CELL 8 SIC'S 282	393	39	198	53	123	0	830
CELL 9 SIC'S 283	297	24	149	86	282	20	913
CELL 10 SIC'S 284	653	13	272	23	89	0	1,285
CELL 11 SIC'S 285	720	36	240	135	112	24	1,377
CELL 12 SIC'S 286	318	10	210	13	128	3	742
CELL 13 SIC'S 287	105	8	100	143	568	0	929
CELL 14 SIC'S 289	580	147	525	2	680	2	1,970
CELL 15 SIC'S 291	220	2	43	19	43	0	347
CELL 16 SIC'S 295	153	12	27	11	163	26	417
CELL 17 SIC'S 299	107	8	65	1	97	6	285
CELL 18 SIC'S 307	2,155	0	1,593	291	2,117	108	6,280
CELL 19 SIC'S 301-306	281	16	1,575	27	202	3	2,128
CELL 20 SIC'S 3111	32	2	8	0	52	0	94
CELL 21 SIC'S 313-319	253	0	25	25	599	186	1,306
CELL 22 SIC'S 32	4,028	0	714	11	5,363	92	11,308
CELL 23 SIC'S 33	2,999	108	641	151	1,152	133	5,297
CELL 24 SIC'S 34	8,072	31	2,162	242	6,104	1,575	19,386
CELL 25 SIC'S 35,36,38,39	18,560	588	4,560	2,878	25,724	2,637	57,492
CELL 26 SIC'S 40,44,45,47	293	0	287	0	213	72	815
CELL 27 SIC'S 46	170	0	28	0	19	1	226
CELL 28 SIC'S 49	2,513	19	718	138	1,609	458	5,885
CELL 30 SIC'S 5093,5153,5161,5191,5198	4,098	60	2,199	769	4,377	338	12,024
CELL 31 SIC'S 55,75	39,349	0	29,674	82	58,039	28,443	169,312
CELL 32 SIC'S 72,73	18,615	0	5,368	5,251	13,208	8,713	52,189
CELL 33 SIC'S 7641,7692	2,082	0	708	242	2,872	2,564	8,963
CELL 34 SIC'S 80	40,858	117	4,768	518	34,671	8,077	94,282
CELL 99 SIC'S 37	6,924	14	411	0	3,908	0	11,263
TOTAL	166,425	1,907	68,720	28,476	208,131	57,762	540,285

Table C-3

FIRMS WITH CHEMICALS REPORTING HAZARD COMMUNICATIONS TRAINING (Q61) (WEIGHTED)

CELL	TRAINING PROGRAM	NO TRAINING PROGRAM	DON'T KNOW REFUSED	TOTAL
CELL 1 SIC'S 243	4,478	5,310	349	10,137
CELL 2 SIC'S 245	651	76	28	754
CELL 3 SIC'S 249	1,202	2,080	20	3,302
CELL 4 SIC'S 25	3,110	5,246	5	8,361
CELL 5 SIC'S 26	2,906	374	210	3,490
CELL 6 SIC'S 27	19,543	26,214	0	45,757
CELL 7 SIC'S 281	1,193	81	24	1,298
CELL 8 SIC'S 282	673	156	0	830
CELL 9 SIC'S 283	752	155	5	913
CELL 10 SIC'S 284	1,044	232	8	1,285
CELL 11 SIC'S 285	1,175	177	24	1,377
CELL 12 SIC'S 286	595	136	11	742
CELL 13 SIC'S 287	925	3	2	929
CELL 14 SIC'S 289	1,802	166	2	1,970
CELL 15 SIC'S 291	326	16	4	347
CELL 16 SIC'S 295	317	100	0	417
CELL 17 SIC'S 299	205	74	6	285
CELL 18 SIC'S 307	4,578	1,701	0	6,280
CELL 19 SIC'S 301-306	2,069	55	3	2,128
CELL 20 SIC'S 3111	68	26	0	94
CELL 21 SIC'S 313-319	418	703	186	1,306
CELL 22 SIC'S 32	6,322	4,894	92	11,308
CELL 23 SIC'S 33	4,364	923	10	5,297
CELL 24 SIC'S 34	12,136	7,226	24	19,386
CELL 25 SIC'S 35,36,38,39	29,929	25,527	2,037	57,492
CELL 26 SIC'S 40,44,45,47	793	22	0	815
CELL 27 SIC'S 46	226	0	0	226
CELL 28 SIC'S 49	4,193	1,612	0	5,805
CELL 30 SIC'S 5093,5153,5161,5191,5198	8,231	3,789	4	12,024
CELL 31 SIC'S 55,75	57,277	112,035	0	169,312
CELL 32 SIC'S 72,73	25,332	26,857	0	52,189
CELL 33 SIC'S 7641,7692	2,791	5,955	217	8,963
CELL 34 SIC'S 80	23,583	66,579	4,040	94,202
CELL 99 SIC'S 37	7,631	3,633	0	11,263
TOTAL	230,839	302,135	7,311	540,285

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Some commenters objected to the use of the telephone survey method in lieu of written responses [see, for example, Exs. 3–750, 3–877 and 3–747]. They stated that the questions were too diverse and complex to be answered by a single person, and that the use of the CATI techniques necessitated simplified responses to questions.

As preparation for the survey, OSHA sent a letter to each potential respondent approximately two weeks in advance of the initial phone contact. The letter described the nature of the project, the topics to be covered by the survey and a response card to be returned to the survey contractor listing the name of the person best able to answer the questions. When requested, a copy of the survey questionnaire was provided. With this advance preparation, OSHA believes that respondents were able to accurately and completely answer the questions. While all firms were encouraged to complete the survey over the telephone so that the responses could be entered on the computer during the interview, some firms refused to do so and also failed to return the written survey forms. Overall, the survey achieved a 60 percent completion ratio (the ratio of completed questionnaires to total sample cases drawn, both in and out of scope). OSHA believes that the use of the CATI technique greatly improved the response rate to the survey. Previous OSHA surveys have had completion ratios as low as 30 percent.

To reduce the burden on respondents, process and chemical lists were used to prompt respondents. Two commenters [Ex. 3–625, Ex. 3–750] stated that the lists were incomplete and thereby biased the final data. However, one of these [Ex. 3–750) correctly stated that ". . . responders were also asked to volunteer additional processes or chemicals present in their plants." Since respondents did indeed volunteer "Other" chemicals, OSHA believes that the use of the prompt "Other" improved the final data instead of biasing it. The Inter-Industry Wood Dust Coordinating Committee commented that the survey did not include wood dust and processes specifically related to wood dust exposure, as prompts [Ex. 3–750]. However, respondents replied, in many instances, that there was exposure to "nuisance particulates". OSHA used these responses as surrogates for responses on wood dust. However, the Agency concluded that the costs in the Preliminary Regulatory Impact Analysis understated "the extent of new controls that would be needed in order to comply with the proposed wood dust standard" [Ex. 38A]. In SICs 24 and 25, the number of work stations where wood dust is found, the percent of work stations which would be out of compliance with the proposed levels, and unit costs for controlling exposure were revised to supplement the survey results, and the recalculated costs of compliance were provided to interested parties and entered into the docket [Ex. 38A].

Commenters also objected to the inclusion of non-production facilities in the survey [Ex. 3–1196, Ex. 3–877, Tr. 8/15/88, p. 105]. The sample survey was designed to represent the universe of facilities in each SIC. There are always a certain number of facilities in each SIC which are headquarters, distribution centers, or sales offices. Where workers at these facilities have no exposure to chemicals, there is no cost to control exposure and no benefits to accrue from lowered exposure levels. Inclusion of these facilities is statistically correct in order to represent that portion of the facilities in an SIC which would incur no cost.

The survey sample was statistically designed to include a higher proportion of larger establishments (20 or more employees) because of the wider variation in costs expected for large firms to comply. The American Mining Congress [Ex. 3–976] expressed concern about "the underrepresentation of small companies" in SICs 32 and 33, while the American Iron and Steel Institute [Ex. 3–1123] commented that average costs for large firms are not representative of costs for large steel facilities in SIC 33. OSHA believes that the generic nature of this rulemaking allows a greater latitude in grouping industries in order to estimate "average" costs, and that the higher proportion of large firms surveyed has provided a more valid estimate of the average costs. Small firms were not underrepresented. Rather, firms in the large size classes were "oversampled" using accepted statistical techniques.

Based on the survey, OSHA estimates that over 60 percent of production workers in most of the industries surveyed are potentially exposed to chemicals and about 10–15 percent of these would be overexposed at the levels proposed in this rulemaking. Chapter D presents OSHA's estimates of the benefits occurring from a reduction in the number of employees exposed to these chemicals.

The industry profiles that follow present economic information on industry sectors expected to be affected by the rulemaking. Most but not all of these industries were included in the samply survey. Table C–1, presented at the end of the chapter, contains employment and establishment data for each industry profiled. The number of establishments in that table was produced from 1985 Dun and Bradstreet data, to be consistent with the employment and economic impact data used in this chapter and in Chapter H.

Table C–4 shows the number of establishments estimated from the 1988 sample survey as compared to the number in the 1986–87 Dun and Bradstreet (D & B) file from which the sample was selected. In general, the estimated number of establishments from the survey is lower than the number in the original D & B file. Survey telephone contracts found that some sampled firms were either out of business, out of the scope of the survey (wrong SIC), or listed more than once on the file.

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Table C-4

ESTABLISHMENT COUNTS FROM SAMPLE ESTIMATES AND ORIGINAL DUN & BRADSTREET

ESTIMATION CELLS	SAMPLE UNITS	ORIGINAL NUMBER ESTAB.'S D&B FILE	NUMBER OF ESTAB.'S FROM CNTY BUS. PTRNS	NUMBER OF ESTAB.'S FROM BLS 202	SURVEY ESTIMATED NUMBER OF ESTAB.	PERCENT CHANGE FROM ORIG. D&B TO SAMPLE	PERCENT CHANGE FROM CNTY BUS. PTRNS TO SAMPLE	PERCENT CHANGE FROM BLS 202 TO SAMPLE
CELL 1 SIC'S 243	160	13486	6510	8333	12150	-10%	87%	46%
CELL 2 SIC'S 245	74	1512	1101	1114	1099	-27%	-0%	-1%
CELL 3 SIC'S 249	115	5364	3682	3638	4868	-9%	32%	34%
CELL 4 SIC'S 25	154	16129	10812	10041	12804	-21%	18%	28%
CELL 5 SIC'S 26	309	8228	6324	6731	7022	-15%	11%	4%
CELL 6 SIC'S 27	234	78345	56137	57928	60282	-23%	7%	4%
CELL 7 SIC'S 281	155	2797	1342	1445	2013	-28%	50%	39%
CELL 8 SIC'S 282	123	1562	648	845	1380	-12%	113%	63%
CELL 9 SIC'S 283	176	2288	1277	1407	1740	-24%	36%	24%
CELL 10 SIC'S 284	137	4202	2375	2359	3656	-13%	54%	55%
CELL 11 SIC'S 285	104	1789	1433	1419	1693	-5%	18%	19%
CELL 12 SIC'S 286	124	1399	918	1028	1224	-13%	33%	19%
CELL 13 SIC'S 287	71	1752	1028	1377	1421	-19%	38%	3%
CELL 14 SIC'S 289	110	3733	2724	2367	3299	-12%	21%	39%
CELL 15 SIC'S 291	122	1055	442	796	722	-32%	63%	-9%
CELL 16 SIC'S 295	88	1160	1294	917	960	-17%	-26%	5%
CELL 17 SIC'S 299	73	735	581	461	628	-15%	8%	36%
CELL 18 SIC'S 307	80	13876	12112	11762	12316	-11%	2%	5%
CELL 19 SIC'S 301-306	158	3187	1857	1884	2778	-13%	50%	47%
CELL 20 SIC'S 3111	22	454	379	346	338	-26%	-11%	-2%
CELL 21 SIC'S 313-319	59	3262	2063	2081	1987	-39%	-4%	-5%
CELL 22 SIC'S 32	127	20103	16159	15704	15920	-21%	-1%	1%
CELL 23 SIC'S 33	360	9527	6921	7152	8028	-16%	16%	12%
CELL 24 SIC'S 34	325	44328	35380	34209	37315	-16%	5%	9%
CELL 25 SIC'S 35,36,38,39	395	160950	92313	92707	123365	-23%	34%	33%
CELL 26 SIC'S 40,44,45,47	82	52277	-- --	-- --	42025	-20%	-- --	-- --
CELL 27 SIC'S 46	63	624	605	953	577	-8%	-5%	-39%
CELL 28 SIC'S 49	368	18430	17725	16019	15812	-14%	-11%	-1%
CELL 30 SIC'S 5093,5153,5161,5191,5198	418	49144	47142	51437	39371	-20%	-16%	-23%
CELL 31 SIC'S 55,75	92	307117	328578	300397	257267	-16%	-22%	-14%
CELL 32 SIC'S 72,73	203	146035	-- --	-- --	107685	-26%	-- --	-- --
CELL 33 SIC'S 7641,7692	107	18399	10162	13514	15095	-18%	49%	12%
CELL 34 SIC'S 80	402	261380	390223	377887	222843	-15%	-43%	-41%
CELL 99 SIC'S 37	102	14958	9498	11438	13617	-9%	43%	19%

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It is possible to compare these survey establishment counts by comparing them to two alternative governmental sources: 1985 County Business Patterns from the U.S. Department of Commerce and 1987 ES–202 data from the Bureau of Labor Statistics (BLS), U.S. Department of Labor. Table C–4 also provides these governmental establishment counts.

In general, these two databases showed fewer establishments than either the survey or the D & B file. Some of these differences are due to the way an "establishment" is defined. D & B may split one establishment at a particular address into several establishments based on the various activities performed there; ES–202 and County Business Patterns may categorize the same establishment as one unit. Also, many state and federally-run establishments are included in the D & B file in the SIC related to their primary activity, rather than as governmental units, which would be the ES–202 and County Business Pattern classification.

There is no concensus among experts as to which source provides the most accurate establishment counts. Based on this comparison and other quality checks, OSHA believes that the survey has provided a sound basis for estimating the economic impact of this rulemaking.

SIC 20—Food and Kindred Products

This major industry group includes establishments that manufacture or process food and beverages for human consumption as well as certain related products such as ice, chewing gum, vegetable and animal fats and oils, and prepared animal feeds [1, pp. 59 to 69]. This industry group was not included in the sample survey. Rather, industry data, costs and economic impact were estimated by experts familiar with this industry sector.

Employment and establishment data are shown in Table C–1. The total 1985 value of SIC 20 shipments ($301.6 billion) was 13 percent of the value of all manufacturing industry shipments; this represented the largest share of any two-digit manufacturing industry. The most important industry within SIC 20 is meat products, accounting for 22 percent of the value of shipments, followed by beverages and dairy products, accounting for 14 percent each [2, Vol 1:8].

In 1985, 1.6 million workers in over 29,000 establishments were employed in SIC 20. About 70 percent of these are production workers [Table C–1]. Employment has declined since 1979. The largest employer is the meat products industry, with 23 percent of the workforce in 1986, followed by preserved fruits and vegetables (15 percent) and beverages (13 percent). Meat and miscellaneous food products both experienced 1986 employment levels slightly above the 1979 peak [5]. The largest number of food products establishments are in the manufacturing or processing of miscellaneous foods and meat products (17 percent and 16 percent, respectively).

Establishments in SIC 20 are similar in size to those in the manufacturing industry as a whole, although there is a smaller concentration of very large establishments. Mean establishment size is 55 workers.

Most recent growth by large food processors has been through business acquisitions rather than internal expansion. The food and beverage sector is becoming more concentrated and efficient. In most food industries for which data are available, concentration is moderate, with the largest four firms having a 30 percent share of sales. Exceptions can be found in cereal breakfast foods, where the four-firm concentration ratio is just over 75 percent, and in soft drinks, where it is 88 percent [3, pp. 33–1 to 39–39].

In the next few years, most food and beverage producers will benefit from increases in disposable income, favorable trends in consumer purchasing patterns, and continued low commodity prices. Decreased operating costs and expenses have resulted in a 6 percent increase in (revenue) income in 1985–86 for large food and beverage processors, despite sales gains of only a little more than 1 percent [3, p. 39–1].

In 1985, the median rate of return on assets in the food and kindred products industry was 5.1 percent; this was the third lowest for the 20 two-digit manufacturing industry group. The highest rates of return were registered by the cookie and cracker industry and the blended and prepared flour industry (11.8 percent and 11.1 percent, respectively), followed by the flavoring extracts industry (9.2 percent). At the other extreme, the wine and brandy industry registered a −0.9 percent rate of return on assets in 1985, with an average rate of under 0.1 percent for the 1984–86 period. The cheese and rice milling industries also have relatively low rates or return on assets (2.2 percent) [6].

OSHA received docket comments pertaining to several four-digit SICs (2011, 2013, 2016, 2017, and 2074) falling within the Food and Kindred Products industrial classification. Most comments addressed the use of three substances included in this rulemaking—carbon disulfide, ammonia, and chlorine—in the meat products sector (SIC 2013).

Commenters noted that firms in SIC 2013 produce hot dogs, luncheon meats, and boneless hams; production of these processed meats uses 3.6 billion pounds of meat and 0.9 billion pounds of poultry annually. Ten percent of all meat production goes into the production of processed meats; for some meats, the share is larger: 83 percent of all ham is processed into boneless hams [Exs. 3–421, 3–898]. Most of the meat used in processed meat is trimmings, which are not suitable for use in other meats. An estimated 65 percent of all processed meats are dependent on cellulosic materials for their manufacture [Ex. 3–421]. The production of casings of this type involves the use of carbon disulfide, and, according to commenters, achieving the proposed limit of 1 ppm for this substance would have created issues of technological and economic feasibility (discussed in greater detail in the Technological Feasibility chapter, below). In the final rule, the limits for carbon disulfide are 4 ppm as an 8-hour TWA and 12 ppm as a STEL; these limits should ameliorate any feasibility problems. In addition, the final rule has increased PEL and STEL limits for both ammonia (35 ppm STEL only) and chlorine (0.5 ppm TWA, 1.0 ppm STEL) which should further reduce the economic impact on this industry sector.

OSHA received many comments addressing the proposed 4-mg/m^3 TWA limit for grain dust (wheat, oats, and barley) in facilities classified in SIC 204, Grain Mill Products [Exs. 3–63, 3–110, 3–237, 3–299, 3–405, 3–752, and 3–755]. Comments were received from the owners of flour mills, rice mills, and feed mills. The National Feed and Grain Association (NFGA) [Ex. 3–752] estimated that the number of feed mills that use wheat, oats, or barley to produce feed is 1,260 facilities, or about 14 percent of all feed mills. The NFGA arrived at this estimate by assuming that feed mills use oats, wheat, and barley in proportion to the total U.S. usage of these grains as compared with the usage of other feed grains [Ex. 3–752]. (Estimates of the number of feed mills is difficult because feed mills are often classified in other industrial classifications if the major portion of their sales is generated by non-feed-mill activities, such as grain elevator operations or sales of farm supplies.) The NFGA characterized its estimate of 1,260 potentially affected feed mills as "conservative" [Ex. 3–752]. Commenters to the record from the Grain Mill Products segment of SIC 20 stated unanimously that achievement of the

proposed 4 mg/m³ limit for grain dust was not feasible in affected grain mills [Exs. 3–63, 3–110, 3–237, 3–299, 3–405, 3–752, and 3–755]. This issue is discussed further in the Technological Feasibility chapter. Because OSHA has revised the PEL for grain dust to 10 mg/m³ in the final rule, most of the problems with technological feasibility raised by these commenters are likely to have been mitigated.

A comment to the record [Ex. 3–1080] from the National Cotton Council of America (NCCA) stated that cottonseed oil mills (SIC 2074) will be adversely impacted by the proposed rule. These mills process cottonseed and its various components for use in animal feed, edible oil, and cellulose products; their concerns are with the proposed limits for n-hexane and hexane insomers, vegetable oil mist, and grain dust. According to the NCCA, there are 50 operating cottonseed mills in the United States, and most of these are small, rural businesses without in-house industrial hygiene capabilitiy. The NCCA anticipates that its members will have difficulty measuring the proposed levels for these substances [Ex. 3–1080, p. 1]. The NCCA's comments are discussed in greater detail in Chapter F, Technological Feasibility.

SIC 21—Tobacco Manufactures

Establishments in the tobacco manufactures industry produce cigarettes (SIC 211), cigars (SIC 212), chewing and smoking tobacco, and snuff (SIC 213), or they engage in tobacco stemming and redrying (SIC 214) [1, p. 70]. The major worker exposures in these industries are to particulates not otherwise regulated during the initial handling of tobacco and to chemicals that have been used to treat the tobacco. This industry was not included in the sample survey.

Data on employment and establishments for SIC 21 are shown in Table C–1. In 1985, the value of tobacco manufacturing shipments was $18.5 billion, slightly more than 6 percent of the value of shipments for all manufacturing [2, Vol. 1:8]. SIC 21 has less than 0.3 percent of the total employment or establishments in manufacturing [7, pp. 10, 15]. Three-quarters of the employees in this industry are production workers. The cigarette industry is the most important component of SIC 21, accounting for more than 80 percent of the value of shipments [2, Vol. 1:8] and 70 pecent of employment for this sector, but only 9 percent of establishments [7, p. 15].

Establishments in SIC 21 are large, with a mean size of 296 employees, compared to 55 for all manufacturing. More than half of the establishments in this two-digit SIC have fewer than 20 employees [Table C–1]; and more than 17 percent have 250 or more employees. The cigarette industry is especially highly concentrated, with a mean establishment size of 2,430 employees. Eleven establishments in the cigarette industry employ 1,000 or more workers, and 99.8 percent of all cigarette manufacturing employees work in these large establishments. Mean establishment sizes in other tobacco industries range from 80 to 135 employees [7, pp. 10, 15]. Employment in the tobacco products industry has declined every year since 1976 (except in 1981), with a total decline in employment of more than 23 percent over the last decade [5].

Most tobacco firms remain profitable because input costs have been relatively stable and prices have increased faster than consumption has declined. The major tobacco companies are continuing to reduce their vulnerability through mergers and diversification [3, pp. 40–1 to 40–7]. Thus, profitability in the tobacco manufactures industry is good. The 1985 median rate of return on assets (7.7 percent) was the fifth highest median rate of return on assets among firms in the 20 manufacturing industry groups [6].

OSHA received no comments or testimony on the tobacco manufacturing sector in the course of this rulemaking.

SIC 22—Textile Mill Products

SIC 22 includes those establishments that perform any of the following six operations: (1) Preparation of fiber and subsequent manufacturing of yarn, thread, braids, twine and cordage; (2) manufacturing broadwoven fabrics, narrow woven fabrics, knit fabrics, and carpets and rugs from yarn; (3) dyeing and finishing fiber, yarn, fabrics and knit apparel; (4) coating, waterproofing, or otherwise treating fabrics; (5) the integrated manufacturing of knit apparel and other finished articles from yarn; and (6) the manufacture of felt goods, lace goods, nonwoven fabrics, and miscellaneous textiles [1, p. 85].

According to the Department of Commerce, in 1986, shipments for the textile industry increased 4 percent. The value of shipments in 1985 ($53.3 billion) has increased 6 percent since 1981. Employment, however, remained on a long-term downward trend, although the 1986 drop was marginal. An upward trend in output and relatively high operating rates helped to keep the drop in employment to a minimum. Also, average hours worked, which increased in the second half of 1985, continued to rise in 1986 [3, p. 41–1].

Table C–1 presents data on the number of establishments and employment in SIC 22. Similar to other manufacturing industries, the mean establishment size in SIC 22 was 64 employees. Between 1981 and 1985, SIC 22 experienced a 15 percent decrease in employment. In 1985, almost 86 percent of the total number of employees were production workers [5]. The median rate of return on assets in the textile mill products industry was 5.6 percent in 1985 [6].

No commenters provided additional information on this industry, and it was not included in the 1988 sample survey.

SIC 23—Apparel and Other Products

SIC 23 is referred to as the "cutting-up and needle trades," and includes establishments producing clothing and fabricating products by cutting and sewing purchased woven or knot textile fabrics and related materials. These materials may include leather, rubberized fabrics, plastics, and furs. In addition, establishments that manufacture clothing by cutting and joining materials are included [1, p. 97].

SIC 23 includes three types of apparel establishments: (1) The regular or inside factories, which perform the usual manufacturing functions within their own plant; (2) contract factories, which manufacture apparel from materials owned by others; and (3) apparel jobbers, which buy raw materials, design and prepare samples, arrange for the manufacture of clothing from their materials, and sell the finished product [1, p. 97]. According to U.S. Department of Commerce estimates, the 1987 value of shipments for SIC 23 experienced a growth rate of 5 percent over 1986 values [4, p. 45–1].

Between 1980 and 1985, SIC 23 was among the top ten SICs to experience the greatest employment decline. Due to large inventories at both retail and wholesale levels, and low consumer demand, there were decreases in both shipments and employment in 1985. In several geographic areas, plants were forced to close. The drop in employment has been attributed to the recent rise of imports into the U.S. market and to improvements in industry efficiency through streamlined operations and increased productivity [3, p. 42–2].

The apparel industry is a major employer of women and minorities, employing more than 6 percent of the manufacturing workforce in plants. Due to intense competition in the industry, profits and wages are lower in this industry than in most other manufacturing industries. The price of labor is the single most important cost

component in the industry, which accounts for the sensitivity that employment levels have to industry growth levels. Production workers make up 85 percent of the apparel work force. Typically, as inventory levels grow, production slows down and employment drops [3, p. 42–2].

In 1986, current-dollar shipments in the apparel industry expanded in value by 3 percent. An increase in consumer demand was the major factor contributing to the upturn. Output levels began to regain former levels of output, and the falling rate of employment of about 1 percent was well below the 3.1 percent annual rate of decline during the 1980–1986 period [3. p. 42–2].

Table C–1 presents employment and establishment data for SIC 23 for 1985. During the period of 1981 through 1985, SIC 23 experienced a 10 percent decrease in employment. Almost 84 percent of the total number of employees were production workers [5]. In 1985, the median rate of return on assets in this SIC was 6.3 percent [6]. This sector was not included in the sample survey. Beginning with SIC 24—Lumber and Wood Products, all the remaining major manufacturing SIC groups were included in the 1988 survey unless otherwise noted in the text.

SIC 24—Lumber and Wood Products

This industry produces logs, pickets and fences, mining timbers, railroad ties, poles and pulpwood. SIC 24 includes establishments that cut timber and pulpwood, merchant sawmills, lath mills, shingle mills, cooperage stock mills, planing mills, and plywood mills and veneer mills engaged in producing lumber and wood basic materials; and establishments that manufacture finished articles made entirely or mainly of wood or related materials [1, p. 107]. According to U.S. Department of Commerce estimates, the logging industry's timber harvest in 1987 was an estimated $9.1 billion, compared with $8.8 billion in 1986. [4, p. 5–2].

The Department of Commerce reports that a strong expansion of the market for wood products took place in 1985 due to gains in housing and nonresidential construction activities. Although domestic demand for softwood lumber was strong, Canadian imports displaced American products and contributed to an oversupply, depressing prices. These lower prices lowered U.S. lumber producer profit margins and induced industrywide efforts to restrict imports of lower priced Canadian softwood lumber. In addition to the oversupply, accelerated harvesting to avoid pest damage forced inventories to go up and prices to fall further [3, p. 4–1].

In 1986, similar trends continued in the domestic market for wood products. This was due to a 6 percent rise in housing starts, continued growth in home remodeling and renovation, and strong demand from furniture markets and other end users. However, lower-priced softwood lumber imports from Canada continued to squeeze profits in 1986 [3, p. 4–1].

The Canadian softwood lumber prices brought about a trade agreement on December 30, 1986 between the United States and Canada, in which Canada agreed to set a 15 percent export tax on its softwood lumber. Canadian softwood lumber prices in the United States have risen 3 to 4 percent and imports have decreased about 3 to 4 percent. Since the agreement, Canada's market share has dropped from 33 percent to 28 percent. U.S. company earnings have increased despite a drop in housing starts. It is expected that the trade agreement will keep Canadian softwood prices up and continue to aid the domestic softwood lumber market [8].

Table C–1 presents employment and establishment data for SIC 24 for 1985, as well as for the three individual three-digit industry groups which were surveyed. In 1985, the mean establishment size in SIC 24 was 19 employees, significantly smaller than the average size in other manufacturing sectors. The median rate of return on assets in the SIC was 7.3 percent [6].

The National Kitchen Cabinet Association stated that the Dun and Bradstreet sampling frame used for the survey seriously underestimated the number of establishments [Ex. 80L]. Dun and Bradstreet estimated the number of establishments in SIC 24 as 36,710 [6]. The Department of Commerce in the 1985 County Business Patterns estimated the number to be 32,205 [7]. In making cost estimates for the Inter-Industry Wood Dust Coordinating Committee, National Economic Research Associates (NERA) assumed that there were only 26,485 establishments (using the 1982 Census of Manufacturers) [Ex. 3–748]. Thus, OSHA believes that the Dun and Bradstreet data used for the survey do not underestimate the total number of establishments.

Additionally, OSHA does not differ significantly from NERA on the total number of employees in SIC 24. OSHA used the Labstat Database of the U.S. Department of Labor to conclude that 697,000 persons were employed in 1985 [5]. NERA estimated that the industry employed 691,656 workers in 1986. Thus it appears that NERA accepts OSHA's estimate relating to the number of employees.

SIC 242—Sawmills and Planing Mills

This SIC includes sawmills and planing mills, hardwood dimension and flooring mills, and special product sawmills. The U.S. Department of Commerce reported that in 1985, SIC 242 employed 26 percent of all employees and represented 21 percent of all establishments in SIC 24 [7]. The value of shipments from 1981 to 1985 rose 10 percent in SIC 2421 and 45 percent in SIC 2426 [4]. The Department of Commerce also reported that employment declined 14 percent in sawmills and rose 3 percent in hardwood dimension and flooring over the same time period. Production workers represent 87 percent of all employees in this industry. Special product sawmills (SIC 249) include facilities that produce shakes and shingles; approximately 290 firms use Western red cedar [9].

Firms in this sector were not included in the 1988 sample survey. OSHA relied on Dun and Bradstreet data to estimate the number of establishments in SIC 242. The National Dimension Manufacturers Association quoted the 1982 Census of Manufactures by stating that 789 establishments were in SIC 2426, of which 306 had 20 or more employees [Ex. 3–1160]. The Department of Commerce stated that there were 714 establishments in 1984, of which 320 had 20 or more employees [7]. Similarly the Census of Manufactures estimated the employment at 29,100 workers in 1987, while County Business Patterns estimated 26,841 in 1984. The difference in these estimates appears to be minor and largely associated with the difference in time and methods of data collection.

SIC 243—Millwork, Veneer and Plywood

This SIC includes establishments that manufacture fabricated wood millwork, covered with materials such as metal and plastics. According to the U.S. Department of Commerce, the value of shipments for SIC 243 was $16.7 billion in 1985, which represents 31 percent of the value of shipments for SIC 24 [3]. The value of shipments in SIC 243 increased 27 percent since 1981. In 1985, the number of employees in SIC 243 was about 37 percent of SIC 24. The number of employees in SIC 243 increased by 18 percent from 1981 to 1985 [5]. Average hourly earnings dropped about 17 percent during that same time period. The number of establishments in SIC 243 in 1985 was about 38 percent of all establishments in SIC 24 [5].

In SIC 243, the 1988 survey identified more than twice as many small firms (fewer than 20 production workers) as large firms. In the small firms, maintenance work is performed for the most part by production workers. By contrast, in large firms, maintenance work is predominantly performed by dedicated maintenance workers.

The manufacturers classified in SIC 243 usually have one to three basic processes, with potential exposure to one to three substances. Thirty-nine percent of these processes involve exposure to chemicals or substances on an intermittent short-term basis (up to 30 minutes) with large firms tending to have more long-term exposures. Twenty-nine percent of the firms in this SIC reported the adoption of internal exposure standards. Of those small firms with internal exposure standards, most have adopted OSHA PELs. Nearly 71 percent of the large firms with standards reported using the OSHA PELS; the balance indicated that they rely on ACGIH TLVs or other standards. Employee monitoring had been performed at 17 percent of the processes.

The survey found that about 29 percent of the processes in SIC 243 are totally enclosed and 8 percent are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed. Nearly 72 percent of the firms with chemical exposures have respirators for employee use, with a higher percentage of small firms reporting the presence of respirators than large firms. The combined data on exposure levels and methods of exposure control indicate that many plants which are estimated to incur some cost of compliance have overexposures in all processes at that plant.

Survey respondents in SIC 243 identified the presence of 35 different substances in SIC 243. Particulates not otherwise regulated were estimated to occur most frequently at a total of 8,956 processes. Particulates not otherwise regulated were identified in bleaching, coating/spraying/finishing/layup, cutting/sawing/planing, drying/baking, gluing/hot pressing, sanding/polishing/grinding, and metal working (rolling, milling, shaping). The final rule does not change the existing limit on these particulates. Wood dust exposures occur in cutting/sawing/planing and sanding/polishing/grinding.

SIC 244—Wood Containers

This SIC represents manufacturers of wood containers, including wood pallets and skids. The pallet industry is the third largest consumer of lumber in the United States, after the construction and furniture industries [Ex. 3–1125]. According to the U.S. Department of Commerce, more than 70 percent of the establishments in SIC 244 employ 20 or fewer people [7]. SIC 244 accounts for 6 percent of the establishments and employment in SIC 24. The total value of shipments in pallets and skids in 1987 was $1.5 billion, thus continuing the industry's third year of economic expansion [4, p. 5–9]. The number of establishments producing pallets and skids rose more than 67 percent from 1982 to 1986 [3, p. 4–10]. The National Wooden Pallet and Container Association (NWPCA) quoted the U.S. Forest Service's 1985 estimate of 2,340 firms in SIC 244 [Ex. 3–899]. The 1984 County Business Patterns estimated 2,103 establishments [7]. The number of employees quoted by the NWPCA was 44,600, somewhat higher than Labstat's estimate of 40,500. However, County Business Patterns estimated 38,478. Labstat estimated 40,500 employees in this industry in 1986 [5]. Hourly earnings of employees in pallets and skids rose 3 percent in 1986 to $6.32. This SIC was not included in the 1987 survey.

SIC 245—Wood Buildings and Mobile Homes

This SIC includes manufacturers of wood buildings and mobile homes. The 1985 value of shipments for SIC 245 ($6.0 billion) represented 11 percent of the total value of shipments for SIC 24 [3]. The value of shipments in SIC 245 increased 6 percent from 1981 to 1985. In 1985, the number of employees in SIC 245 was 10.5 percent of SIC 24. Almost 77 percent of these employees are production workers. The number of establishments in SIC 245 in 1985 was 4.4 percent of all establishments in SIC 24 [5].

In SIC 245, the survey identified about as many small firms (fewer than 20 production workers) as large firms. In the small firms, maintenance work is generally performed by production workers. In large firms maintenance work is mostly performed by dedicated maintenance workers.

The manufacturers classified in SIC 245 usually have one to three basic processes, with potential exposure to one to three substances. Thirty percent of these processes involve exposure to these chemicals or substances on an intermittent short-term basis (up to 30 minutes) with large firms tending to have more long-term exposures. Almost thirty-seven percent of the firms in this SIC reported the adoption of internal exposure standards. Among small firms with internal exposure standards, most have adopted OSHA PELs. Nearly 67 percent of the large firms with standards reported using the OSHA PELs; the balance indicated that they rely on ACGIH TLVs. Employee monitoring had been performed at 40 percent of the processes.

The survey found that about 16 percent of the processes are totally enclosed and 16 percent are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed. Nearly 78 percent of the firms with chemical exposures have respirators for employee use, with a higher percentage of large firms reporting the presence of respirators than small firms. The combined data on exposure levels and methods of exposure control indicate that many plants which are estimated to incur some cost of compliance have overexposures in all processes at that plant.

Survey respondents in SIC 245 identified the presence of 10 different substances. Particulates not otherwise regulated were estimated to occur the most frequently at a total of 703 processes. The final rule retains the existing limit for these particulars. Toluene, the second most frequently used chemical, was identified in coating/spraying/finishing/layup, gluing/hot pressing, and sanding/polishing/grinding. Wood dust occurs in cutting/sawing/planing and sanding/polishing/grinding operations.

SIC 249—Miscellaneous Wood Products

This SIC covers miscellaneous wood products, and includes four four-digit SICs. SIC 249 represented 12 percent of the value of shipments for SIC 24 in 1985 [4]. The value of shipments in SIC 249 ($6.6 billion) increased almost 24 percent over 1981. In 1985, the number of employees in SIC 249 was about 15 percent of SIC 24. From 1981 to 1985, the number of employees in SIC 249 decreased by 4 percent. The number of establishments in SIC 249 in 1985 was about 14 percent of all establishments in SIC 24 [5].

SIC 2491 includes establishments that treat wood, sawed or planed in other establishments, with creosote or other preservatives to prevent decay and to protect against fire and insects. This industry also includes facilities that cut, treat, and sell poles, posts, and pilings. The Department of Commerce reports that during 1985 there was increased use of treated wood for home improvement projects, such as new decks and all-weather wood foundations. The market for railroad ties in 1985 was strong, as

railroads replaced worn out ties. In 1986, however, sales of railroad ties declined. About 30 percent of total treated wood shipments are lumber and plywood [3, p. 4–14]. The Department of Commerce estimated that in 1986, the value of shipments in this industry increased by 7 percent [4, p. 5–15]. SIC 2491 represents 23 percent of the value of shipments for SIC 249. Employment rose in 1986 by 2.7 percent [3, p. 4–14]. The number of employees in SIC 2491 was almost 16 percent of SIC 249 and SIC 2491 represents almost 11 percent of all establishments in SIC 249 [6].

In SIC 249, the survey identified three times as many small firms (fewer than 20 production workers) as large firms. In the small firms, maintenance work is performed for the most part by production workers or a dedicated maintenance staff. Large firms primarily use dedicated maintenance workers to perform maintenance duties.

The manufacturers classified in SIC 249 usually have one to four basic processes, with potential exposure to one of four substances. Thirty-four percent of the processes in this SIC involve exposure to these chemicals or substances on an intermittent short-term basis (up to 30 minutes), with large firms tending to have more long-term exposures. Forty-three percent of the firms in this SIC reported the adoption of internal exposure standards. Of these, most small firms have adopted OSHA PELs. Nearly 83 percent of the large firms with standards reported using the OSHA PELs; the balance indicated that they rely on ACGIH TLVs. Employee monitoring had been performed at 11 percent of the processes.

The survey found that about 22 percent of the processes are totally enclosed and 11 percent are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed. Roughly 48 percent of the firms with chemical exposures have respirators for employee use, with a higher percentage of large firms reporting the presence of respirators than small firms. The combined data on exposure levels and methods of exposure control indicate that many plants which are estimated to incur some cost of compliance have overexposures in all processes at that plant.

Survey respondents identified the presence of 25 different substances in SIC 249. Particulates not otherwise regulated were estimated to occur most frequently at a total of 1,678 processes. Particulates were identified in coating/spraying/finishing/layup, cutting/sawing/planing, drying/baking, gluing/hot pressing, sanding/polishing/grinding, stamping/shaping/molding/pressing, and assembly. Wood dust exposures occur in cutting/sawing/planing and sanding/polishing/grinding.

SIC 25—Furniture and Fixtures

Manufacturers of household, office, public building, and restaurant furniture and office and store fixtures are included in SIC 25 [1, p. 114]. The U.S. Department of Commerce states that producers of furniture and fixtures recently have benefited from lower real interest rates, a reduction in the value of the dollar versus other major currencies, and changes in the tax laws [3]. In addition, the U.S. furniture industry is undergoing consolidation; big firms are becoming larger and accounting for a greater share of the market. The remaining smaller firms are finding it more difficult to compete, given the rapid increase in low-priced imports. Moreover, new manufacturing technologies require large capital investments and large volume, neither of which are readily available to small firms [3, p. 44–2].

For the industry, the value of shipments in 1985 increased by 31 percent over the level in 1981. In household furniture, the value of shipments for 1987 increased an estimated 7 percent following a growth of 5.4 percent in 1986 [4, p. 47–2]. Although furniture manufacturers anticipate stronger demand in the future, these manufacturers remain uncertain as to the duration and extent of increased demand. Therefore, rather than hiring additional workers, producers have increased the average number of hours worked by current employees. This trend was evident in the wood and metal furniture plants, where average overtime hours increased 16 percent and 24 percent, respectively, in the first half of 1986 [3, p. 44–21].

Table C–1 presents employment and establishment data for SIC 25 for 1985. Almost 80 percent of the total number of employees working in SIC 25 were production workers and the median rate of return on assets in the furniture industry was 7.3 percent in 1985.

In SIC 25, the survey detected twice as many small firms (fewer than 20 production workers) as large firms. In small firms, maintenance work is performed for the most part by production workers, whereas large firms primarily use dedicated maintenance workers.

The manufacturers classified in SIC 25 usually have one to four basic processes, with potential exposure to as many as six substances. Twenty-four percent of these processes involve exposure to these chemicals or substances on an intermittent short term basis (up to 30 minutes), with large firms tending to have more long-term exposures. Fifty-four percent of the firms in this SIC reported the adoption of internal exposure standards. Among small firms with internal exposure standards, most use the OSHA PELs. About 66 percent of the large firms with standards reported using the OSHA PELs; the remainder indicated that they rely on ACGIH TLVs. Employee monitoring had been performed at 19 percent of the processes.

The survey found that about 25 percent of the processes are totally enclosed and 3 percent are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed. Nearly 78 percent of the firms with exposures provide respirators for employee use, with a higher percentage of small firms reporting the presence of respirators than large firms. The combined data on exposure levels and methods of exposure control indicate that many plants which are estimated to incur some cost of compliance do not have overexposures in all processes at that plant.

Survey respondents identified the presence of 36 different substances in SIC 25. Particulates not otherwise regulated were estimated to occur most frequently at a total of 3,433 processes. Toluene, the second most frequently used chemical, was identified at processes in coating/spraying/finishing/layup, drying/baking, gluing/hot pressing, drilling/boring and sanding/polishing/grinding. Wood dust exposures occur in cutting/sawing/planing, drilling/boring, and sanding/polishing/grinding.

SIC 26—Paper and Allied Products

Establishments in this industry process fiber from trees, wastepaper, and other fibrous materials, into end products that are used by both consumers and industry [1, p. 100]. Based on U.S. Department of Commerce estimates, the paper and allied products industry experienced an increase of 16 percent in the value of shipments from 1981 to 1985, and over 10 percent between 1985 and 1986 [3]. Net profits for 27 paper industry firms were reported to have averaged nearly 60 percent higher in the first six months of 1987 than in the first half of 1986 [4, p. 6–1].

The industry's overall demand patterns are closely linked to rates of change in GNP. In 1985, for example, real growth for the industry was judged to be flat, trailing that of the GNP. The

largest fluctuations in the industry's shipments have occurred in products geared specifically for commercial-industrial use, which are tied to the annual rate of business activity [3, p. 5–1].

Table C–1 presents employment and establishment data for SIC 26 for 1985. From 1981 to 1985, employment declined by approximately 2 percent. Almost 76 percent of the total number of employees were production workers [5]. In 1985, the median rate of return on assets was 7.4 percent [6].

Within SIC 26, there are six, three-digit SIC groups. SIC 261 includes manufacturers of pulp from wood or other materials. The Department of Commerce reports that U.S. market pulp prices dropped nearly 10 percent in the first six months of 1985. By the end of 1985, however, producers' pulp mill inventories had dropped, helping to stabilize pulp prices. About one-fourth of all market pulp companies either shut down some of their mills in 1985 or curtailed production to reduce the oversupply in the market. In 1986, the industry experienced increased productivity, higher prices and improved worldwide demand. For SIC 261, the value of shipments in 1987 increased by 2.7 percent over 1986. SIC 261 represents 3.5 percent of the value of shipments for SIC 26 [3, p. 5–2].

SIC 262 includes manufacturers of paper from wood pulp and other fiber pulp, and manufacturers of converted paper products. SIC 263 includes manufacturers of paperboard. SIC 262 represents 11 percent of the value of shipments for SIC 26. The value of shipments decreased by 3.6 percent. The number of employees in SIC 263 was less than 1 percent of SIC 26 [5].

SIC 264 includes manufacturers of coated or laminated flexible materials used for packaging purposes. In this sector, the value of shipments, which represents 36 percent of the value of shipments for SIC 26, increased by 17 percent during the same period. The number of employees in SIC 264 was 34 percent of SIC 26 [5]. SIC 265 includes manufacturers of setup paperboard boxes from purchased paperboard. Corrugated boxes have taken the place of wooden shipping containers, pallets, and metal drums in the U.S. packaging market in recent years [3, p. 5–6]. Similarly, consumption of folding boxes continued steadily in 1985. This pattern continued in 1986 with shipments of corrugated boxes increasing 5.5 percent and 3 percent for folding boxes. Several important nondurable end users of folding cartons, such as producers of beverages, dry food, textiles, sporting goods and toys, hardware, candy, and cosmetics, showed significant declines in real growth in 1985, while the market for boxed paper goods either grew slightly or remained fairly level [3, p. 5–9].

Manufacturers of sanitary food containers, such as paperboard milk cartons and paper serving and eating utensils, are also included in SIC 265. This industry has been strongly influenced by the shift to plastic containers. Having experienced two successive years of decline, the industry increased the value of shipments by 2 percent in 1986. Since 1983, the most rapid growth area within the sanitary food container industry has been aseptic packaging. This is specially treated paperboard combined with plastic film and aluminum foil.

The value of shipments for SIC 265 increased by 16 percent from 1981 to 1985. This three-digit SIC represents 24 percent of the value of shipments for all of SIC 26. In 1985, the number of employees in SIC 265 was 29 percent of SIC 26 [5]. SIC 266 includes manufacturers of building paper and building board from wood pulp and other fibrous materials. Trends in employment and value of shipments have followed overall trends in SIC 26.

In SIC 26, the survey identified half as many small firms (fewer than 20 production workers) as large firms. In small firms, maintenance work is performed largely by production workers, although some firms use outside contractors. Large firms generally use a separate maintenance staff to perform maintenance duties.

The manufacturers classified in SIC 26 usually have one to six basic processes, with potential exposure to as many as seven chemicals or substances. Twenty-nine percent of these processes involve exposure to these chemicals or substances on an intermittent short-term basis (up to 30 minutes), with large firms tending to have more long-term exposures. Firms in this SIC are equally divided between those adopting no internal exposure standards and those adopting OSHA PELs. Among small firms with internal exposure standards, all have adopted OSHA PELs. Nearly 81 percent of the large firms with standards reported adopting the OSHA PELs; the balance indicated that they rely on ACGIH TLVs. Employee monitoring had been performed at 36 percent of the processes.

The survey found that about 25 percent of the processes are totally enclosed and 4 percent are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed. Approximately 42 percent of the firms with chemical exposures have respirators for employee use, with a higher percentage of small firms reporting the presence of respirators than large firms. The combined data on exposure levels and methods of exposure control indicate that many plants which are estimated to incur some cost of compliance have overexposures in all processes at that plant.

Survey respondents indentified the presence of 46 different chemicals or substances in SIC 26. Particulate not otherwise classified occurred the most frequently at a total of 1,753 processs. Cellulose was identified at 664 processes. The final rule retains the existing limits for both particulates not otherwise regulated and cellulose. Wood dust exposures occurred in cutting/sawing/planing.

SIC 27—Printing, Publishing, and Allied Industries

This industry is divided into a publishing sector, a printing sector and a sector of related services. The publishing sector includes newspaper publishing (SIC 271), periodical publishing (SIC 272), book publishing (SIC 2731), miscellaneous publishing (SIC 274) and greeting card publishing (SIC 277). The printing sector includes commercial printing (SIC 275), book printing (SIC 2732), and printing trade services (SIC 279). The related services sector includes manifold business forms (SIC 276) and blankbooks and bookbinding (SIC 278) [1, pp. 106–110].

There were approximately 84,279 establishments in the printing and publishing business in 1985. The majority of these firms (84.1 percent) had fewer than 20 employees, and the mean establishment size was 17 employees. The firms in SIC 27 had 1.4 million employees and 789,000 production workers [Table C–1]. According to the U.S. Department of Commerce, the value of shipments for all printing and publishing establishments in 1986 ($118.6 billion) was 5.2 percent of the value of shipments for all manufacturing industries. Most of the value of shipments in SIC 27 is from the commercial printing sector (32.1 percent) [4]. In 1985, the median rate of return on assets was 8.2 percent for the printing and publishing industry [6].

Foreign trade has not been a major concern for this industry in the past, but imports are beginning to increase at a steady rate. The respective values of imports and exports were very close in 1987, with $1.6 billion in imports and $1.5 billion in exports [3, p. 29–2].

The newspaper industry has improved its performance after several years of slow growth. The value of shipments for SIC 271 was $29.2 billion in 1986. Total employment rose an estimated 2.2 percent in 1986 to 420,000 employees, but production employment remained virtually unchanged at 151,900 employees. Sales revenues increased by 8.9 percent, from $14.8 billion in 1986 to $16.2 billion in 1987. Advertising revenues rose slightly, but most of this gain was due to rate increases and growth in classified ad volume. Total net worth increased by 14.2 percent from 1986 to 1987 [10].

The periodical industry has experienced moderate growth in both advertising receipts and circulation. Advertising revenue increased about 4 percent in 1987, while circulation revenues increased slightly due to the increase in subscriptions for consumer magazines. There was another large increase in the number of new publications entering the market; over 250 new periodicals were published in 1987 [3, p. 29–6]. The value of shipments of the periodical industry was $15.7 billion in 1986, an increase of 3.1 percent over the 1985 figure of $15.2 billion. The total number of employees in the periodical industry increased in 1986 (98,100 employees), while the number of production workers decreased (14,200 employees). The periodical industry has the lowest ratio of production workers to total employees (14.5 percent) within SIC 27.

The commercial printing industry (SIC 275) has been very profitable over the last decade. The 1987 value of shipments ($40.9 billion) increased 7.5 percent over the 1986 value of shipments ($38.0 billion). Between 1980 and 1985, the value of shipments increased by 11.5 percent compounded annually. Total employment and production employment have also been increasing substantially from 1986 to 1987 (3.7 percent and 2.7 percent, respectively). The outlook for this industry is steady growth [3, pp. 29–12 to 29–14].

Both book publishing and printing showed strong gains over the last several years. Value of shipments and total employment increased by 5.5 percent and 0.7 percent, respectively, from 1985 to 1986. Spurred by the increase in school enrollment, sales of textbooks were projected to reach 29 percent of total industry sales in 1988. Book printing usually follows the path of book publishing, increasing substantially when book publishing has a strong year [3, pp. 29–9 to 29–13].

Miscellaneous publishing and printing consists of newsletters, catalogs, directories, greeting cards, and business forms. This industry has seen steady gains due in part to the success of mail-order catalogs, telephone directories, and newsletters [3, pp. 29–13 to 29–19].

In this SIC, the survey identified six times as many small firms (fewer than 20 production workers) as large firms. In the small firms, approximately two-thirds of maintenance work is performed by production workers. Outside contractors do approximately one-fourth of maintenance work, and maintenance staff and other sources make up the remainder. Large firms divide maintenance work about equally between a dedicated maintenance staff, production workers, and outside contractors.

The manufacturers classified in this SIC usually have one to three basic processes, with potential exposure to as many as six chemicals. Employees are exposed to these chemicals on an intermittent short term basis (up to 30 minutes) or continuously (up to 8 hours per day) with large firms tending to have more long-term exposures. Small firms generally have no internal exposure standards; when they do, the OSHA PELs are followed about fifteen percent of the time. Over one-half of large firms reported using the OSHA PELs; the balance indicated that they have no standards or they rely on ACGIH TLVs or NIOSH RELs. Air monitoring data were provided for about one-tenth of the processes found in all plants, and for about one-third of the processes found in large firms.

The survey found that about two-thirds of the processes are totally enclosed and less than one percent are located outdoors. Local exhaust ventilation and general dilution are used most frequently to control exposures at processes not enclosed or outdoors. In less than five percent of the firms with chemical exposures, production workers use respirators with a higher percentage of large firms using respirators than small firms.

Survey respondents identified isopropyl alcohol, stoddard solvent, and methyl alcohol among the chemicals most prevalent in this SIC. These are used in lithographic printing and platemaking and letterpress printing which were the processes most frequently listed by respondents. Toluene, xylene, and trichloroethylene were also identified in the survey. A large commercial printer, R.R. Donnelly and Sons, confirmed the presence of toluene in press operations and expressed concern over the ability to meet the proposed levels, especially during cleaning [Ex. 3–916].

SIC 28—Chemicals and Allied Products

SIC 28 includes establishments that produce basic chemicals, and establishments that manufacture products using chemical processes. There are three general classes of products: (1) basic chemicals, such as acids, alkalies, salts, and organic chemicals; (2) chemical products to be used in further manufacturing, such as synthetic fibers, plastics materials, dry colors, and pigments; and (3) finished chemical products to be used for consumption, such as drugs, cosmetics, and soaps; or to be used as materials or supplies in other industries, such as paints, fertilizers, and explosives [1, p. 132].

The chemical and allied products industries have experienced small but steady growth over the recent past. Total shipments by the chemical industry increased approximately 3.1 percent in 1987, following a 3.5 percent gain in 1986 [3]. Chemical prices have been stable since 1982, due to steady or declining energy costs. Like many other U.S. industries, various sectors within the chemical industry are undergoing structural changes, such as mergers, plant closings, sale of plants, and other adjustments. This industry employs approximately 5 percent of all industry workers, but more than 10 percent of all U.S. scientists and engineers. SIC 28 experienced a 6 percent decline in employment between 1981 and 1985. In 1985, 55.4 percent of the total number of employees in SIC 28 were production workers. The value of shipments increased 8.9 percent during the 1981 to 1985 time period. The median rate of return on assets in the chemical industry was 6.3 percent [6].

Within SIC 28, there are eight, three-digit SICs, which are described below.

SIC 281—Industrial Inorganic Chemicals

This SIC includes establishments that manufacture basic industrial inorganic chemicals. SIC 281 represented 10.3 percent of the value of shipments of SIC 28 in 1985 [2]. The value of shipments increased 12.9 percent since 1981, and employment declined by 12 percent. Production workers equaled almost 51 percent of all workers. The number of establishments in SIC 281 was 14.5 percent of all establishments in SIC 28 [Table C–1].

SIC 281 is subdivided into four groups. Examples of the products of each four-digit SIC are given below.

SIC 2812 Products—Chlorine, soda ash, caustic potash, caustic soda, washing soda, and sodium bicarbonate

SIC 2813 Products—Oxygen, acetylene, argon, carbon dioxide, and hydrogen.
SIC 2816 Products—Color pigments, iron colors, iron oxide, lead oxide pigments, mineral colors, titanium pigments, and zinc oxide pigments.
SIC 2819 Products—Sulfuric, hydrochloric, and hydrofluoric acids.

In SIC 281, the survey identified three times as many small firms (fewer than 20 production workers) as large firms. In the small firms, maintenance work is performed for the most part by production workers, although some firms employ dedicated maintenance workers. Large firms predominantly employ workers specifically for maintenance duties.

The manufacturers classified in this SIC usually have one to two basic processes, with potential exposure to as many as six substances. Fifty-two percent of these processes involve exposure to chemicals or substances on an intermittent short-term basis (up to 30 minutes), with large firms tending to have longer-term exposures. Most firms in SIC 281 reported the adoption of OSHA PELs as their internal standards. Employee monitoring had been performed at 67 percent of the processes. The survey found that about 32 percent of the processes are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed. About 24 percent of the firms with chemical exposures have respirators for employee use, with a higher percentage of small firms reporting the presence of respirators than large firms. The combined data on exposure levels and methods of exposure control indicate that most plants which are estimated to incur some cost of compliance do not have overexposures in all processes at that plant.

Survey respondents identified the presence of 58 different substances in SIC 281. Carbon dioxide was estimated to occur the most frequently at a total of 292 processes. Carbon dioxide was identified in recovery/reprocessing, packaging/bagging, loading/offloading/receiving/handling, process inspection, reaction/fermentation, and separation. Another common substance, sodium hydroxide, was identified in boilers.

SIC 282—Plastics Materials and Synthetics

This SIC includes manufacturers of plastics materials and synthetic resins, synthetic rubbers, and cellulosic and other manmade fibers. Plastics make up a variety of products which are used in diverse markets. Packaging and construction account for over 50 percent of consumption, with the remainder going into the transportation, electronics, and medical industries [3]. SIC 282 represents almost 17 percent of the value of shipments of SIC 28. The value of shipments in SIC 282 increased 9.2 percent over the period 1981 to 1985 [2]. Industry shipments of plastics in 1986 gained 6.3 percent as volume rose in response to slightly increased demand for materials. However, declining prices of plastic materials held shipments to a 2 percent increase [3, p. 14–1].

Table C–1 gives employment and establishment data for this segment. The number of employees in SIC 282 in 1985 was almost 16 percent of SIC 28 and the number of establishments was 8 percent of all establishments in that SIC. In 1985, employment in SIC 282 declined by 12 percent, and production workers equaled 66.5 percent of all workers [5].

SIC 282 is subdivided into four groups. Examples of the products from each of these four-digit SICs are given below.

SIC 2821 Products—Cellulose plastics materials, phenolic and other tar acid resins, acrylic resins, polyethylene resins, coumarone-indene and petroleum polymer resins, and casein plastics.
SIC 2822 Products—Copolymers of butadiene and styrene, or butadiene and acrylonitrile, and polybutadienes.
SIC 2823 Products—Cellulose, rayon, and triacetate fibers.
SIC 2824 Products—Fibers of acrylic, acrylonitrile, polyvinyl ester, and nylon.

In SIC 282, the survey identified twice as many small firms (fewer than 20 production workers) as large firms. In small firms, maintenance work is either performed by production workers or dedicated maintenance workers. Large firms primarily employ workers specifically for maintenance duties.

The manufacturers classified in SIC 282 usually have one to six basic processes, with numerous firms having exposures to as many as six different substances. Forty-five percent of these processes involve exposure to these chemicals or substances on an intermittant short-term basis (up to 30 minutes), with large firms tending to have more short-term exposures. Most firms in this SIC have adopted OSHA PELs as their internal standards. Of the small firms with internal exposure standards, most have adopted OSHA PELs or ACGIH TLVs. About 49 percent of large firms reported using OSHA PELs, with 36 percent reporting the adoption of ACGIH TLVs.

The survey found that about 33 percent of the processes are totally enclosed and 24 percent are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed. More than 28 percent of the firms with chemical exposures have respirators, with an equal percentage of small and large firms reporting the availability of respirators for employee use. The combined data on exposure levels and methods of exposure control indicate that many plants which are estimated to have some cost of compliance have overexposures in some, but not all, processes in the plant.

Survey respondents identified the presence of 53 different substances in SIC 282. Styrene was estimated to occur the most frequently at a total of 209 processes. Styrene was identified in recovery/reprocessing/reclamation, drying/baking, separation, blending/mixing/formulating, packaging/bagging, extrusion, crushing/grinding/calcining, loading/offloading/receiving/handling, and reaction/fermentation. Another common substance in SIC 282 was isopropyl alcohol, which occurred in eight different processes.

SIC 283—Drugs

This group includes establishments that manufacture, fabricate, or process medicinal chemicals and pharmaceutical products. The value of shipments in SIC 283 has increased 40 percent from 1981 to 1985 [2]. SIC 283 represents 16 percent of the value of shipments of SIC 28 and almost 20 percent of the number of employees. The U.S. Department of Commerce estimated that the pharmaceutical industry experienced a 6.3 percent increase in the value of shipments in 1986. However, after adjusting for price changes, this growth rate was closer to 1.8 percent. Productivity also increased in 1986, growing by approximately 2.6 percent [3, p. 17–1].

As seen in Table C–1, the number of establishments in SIC 283 was almost 12 percent of all establishments in SIC 28. Employment increased by 3 percent since 1981, and production workers equaled approximately 46 percent of all workers in SIC 283. Agar, vitamins, antibiotics, vaccines, and viruses are examples of the products of this SIC.

In SIC 283, the survey identified three times as many small firms (fewer than 20 production workers) as large firms. In the small firms, maintenance work is generally performed by either dedicated maintenance workers or by general production workers. In large firms, most maintenance work is performed by workers specifically employed for maintenance duties.

The manufacturers classified in SIC 283 usually have one to five basic processes, with potential exposure to one to three substances. Fifty percent of all employees are exposed to these chemicals or substances on an intermittent basis (up to 30 minutes), with small firms tending to have more long-term exposures. Among small firms with exposure standards, most have adopted OSHA PELs. Among large firms, a significant percentage have adopted ACGIH TLVs, although most still rely on OSHA PELs. Employee monitoring had been performed at 34 percent of the processes.

The survey found that about 53 percent of the processes are totally enclosed and 4 percent are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed. In 21 percent of the firms with chemical exposures, respirators were available for employee use, with a higher percentage of large firms reporting the presence of respirators than small firms. The combined data on exposure levels and methods of exposure control indicate that very few plants which are estimated to incur some cost of compliance have overexposures at all processes in that plant.

Survey respondents identified the presence of 40 different substances in SIC 283. Isopropyl alcohol was estimated to occur the most frequently at a total of 577 processes. Isopropyl alcohol was identified in boilers, coating/spraying/finishing/layup, drying/baking, blending/mixing/formulating, packaging/bagging, loading/offloading/receiving/handling, reaction/fermentation, and separation.

SIC 284—Soaps, Cleaners, and Toilet Goods

This SIC includes manufacturers of detergents, emulsifiers, cosmetics, and producers of glycerin. SIC 284 represents 15 percent of the value of shipments of SIC 28 [3]. The value of shipments in SIC 284 increased 20 percent from 1981 to 1985 [2]. In 1986, the value of shipments was estimated at $31 billion, which represents about a 4 percent increase over 1985 values [3, p. 16–1].

The number of employees in this SIC was almost 15 percent of SIC 28 and the number of establishments was almost 22 percent. In 1985, employment in SIC 284 had increased by 1 percent since 1981, and production workers equaled approximately 63 percent of all workers in SIC 284 [5].

There are four subgroups within SIC 284. Examples of the products produced by each four-digit SIC are given below.

SIC 2841 Products—Soap, synthetic organic detergents, inorganic alkaline detergents, and crude and refined glycerin from vegetable and animal fats and oils.

SIC 2842 Products—Household, institutional, and industrial plant disinfectants, nonpersonal deodorants, dry cleaning preparations, household bleaches, and other sanitation products.

SIC 2843 Products—Textile and leather finishing agents, soluble oil and greases.

SIC 2844 Products—Perfumes, cosmetics, home permanent kits, shampoos, shaving products, and talcum powder.

In SIC 284, the survey identified twice as many small firms (fewer than 20 production workers) as large firms. Maintenance work in small firms is basically performed by production workers, while dedicated maintenance workers and in some firms production workers, handle this task in large firms.

The manufacturers classified in SIC 284 usually have one to three basic processes, with potential exposure to one to eight substances. Fifty percent of all employees are exposed to these chemicals or substances on an intermittent basis (up to 30 minutes), with large firms tending to have more long-term exposures. Most firms in this SIC have adopted OSHA PELs. Employee monitoring had been performed at 26 percent of the processes.

The survey found that roughly 38 percent of the processes are totally enclosed and 9 percent are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed. Nearly 44 percent of the firms with chemical exposures have respirators for employee use, with a higher percentage of large firms reporting the presence of respirators than small firms. The combined data on exposure levels and methods of control indicate that very few plants which are estimated to incur some cost of compliance have overexposures at all processes at that plant.

Survey respondents identified the presence of 52 different substances in SIC 284. Sodium hydroxide was estimated to occur most frequently at a total of 452 processes. Sodium hydroxide was identified in drying/baking, blending/mixing/formulating, packaging/bagging, loading/offloading/receiving/handling, reaction/fermentation, and separation.

SIC 285—Paints and Allied Products

This SIC includes manufacturers of paints and allied paint products such as varnishes, shellacs, and paint removers. The paint industry grew by about 5.3 percent in 1986, compared to 1985's decline of 2.9 percent [3, p. 15–1]. Estimated shipments for 1986 were $11.1 billion, of which architectural coatings accounted for about 41 percent, followed by product coatings (35 percent) and specialty products (24 percent) [3, p. 15–2].

SIC 285 represents about 6 percent of the value of shipments of SIC 28. The value of shipments increased almost 26 percent from 1981 to 1985 [2]. The number of employees in SIC 285 was 6 percent of SIC 28 and the number of establishments was 9 percent.

In SIC 285, the survey identified three-fourths as many small firms (fewer than 20 production workers) as large firms. In small firms, maintenance work is either performed by production workers or dedicated maintenance workers. Large firms predominantly use workers dedicated to maintenance duties.

The manufacturers classified in SIC 285 usually have one to five basic processes, with numerous firms having potential exposures to as many as seven different substances. About 40 percent of the employees are exposed to these chemicals or substances on an intermittent short-term basis (up to 30 minutes), with large firms tending to have longer-term exposures. Most firms in this SIC have adopted OSHA PELs or ACGIH TLVs as their internal standard; about 58 percent of the firms reported using OSHA PELs and 19 percent reported the adoption of ACGIH TLVs.

The survey found that about 34 percent of the processes are totally enclosed and 11 percent are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed. About 17 percent of the firms with chemical exposures have respirators for employee use, with a higher percentage of large firms reporting the presence of respirators. The combined data on exposure levels and methods of exposure control indicate that most plants which are estimated to incur some cost of compliance have overexposures in some, but not all, processes at that plant.

Survey respondents identified the presence of 39 different substances in SIC 285. Stoddard solvent was estimated to occur most frequently at a total of 941 processes. Stoddard solvent was identified in recovery/reprocessing/reclamation, coating/spraying/

finishing/layup, drying/baking, blending/mixing/formulating, packaging/bagging, crushing/grinding/calcining, loading/offloading/receiving/handling, reaction/fermentation, and separation. Another common substance in this SIC was ethylene glycol, which occurred in seven processes.

SIC 286—Industrial Organic Chemicals

This SIC includes manufacturers of a variety of industrial organic chemicals. Industry shipments of organic chemicals increased approximately 2 percent over 1985, which was the same level of growth experienced in the previous year [3]. In 1985, the value of shipments for SIC 286 was $41.8 million, representing 21 percent of the value of shipments of SIC 28 [2]. The number of employees in SIC 286 was almost 11 percent of SIC 28 and the number of establishments was approximately 7 percent. Employment in SIC 286 increased by 10 percent, and production workers equaled 51 percent of all workers [Table C-1].

There are three subgroups in SIC 286. Examples of products for each four-digit SIC are given below.

SIC 2861 Products—Hardwood and softwood distillation products, wood and gum naval stores, charcoal, natural dyestuffs and natural tanning materials.

SIC 2865 Products—Toluene, benzene, synthetic organic dyes and pigments.

SIC 2869 Products—Alcohols, caprolactam, and ethylene glycol.

In SIC 286, the survey identified three-fourths as many small firms (fewer than 20 production workers) as large firms. Small firms primarily use production workers to perform maintenance tasks. Large firms, on the other hand, primarily use dedicated maintenance workers to perform maintenance duties. Some small and large firms use outside contractors.

The manufacturers classified in this SIC usually have two to four basic processes, with potential exposure to as many as six substances. Fifty-six percent of the employees are exposed to these chemicals or substances on an intermittent short-term basis (up to 30 minutes), with large firms tending to have longer-term exposure. Most firms in SIC 286 have adopted OSHA PELs or ACGIH TLVs as their internal standards. Employee monitoring had been performed at 78 percent of the processes.

The survey found that about 34 percent of the processes are totally enclosed and that nearly 38 percent of the processes are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed. Roughly 34 percent of the firms with chemical exposures have respirators for employee use, with a higher percentage of large firms reporting the presence of respirators than small firms. The combined data on exposure levels and methods of exposure control indicate that very few plants which are estimated to incur some cost of compliance have overexposures in all processes at that plant.

Survey respondents identified the presence of 57 different substances in SIC 286. Particulates not otherwise regulated and ethylene glycol were estimated to occur most frequently at a total of 222 and 184 processes, respectively. OSHA has retained the existing limit for particulates not otherwise regulated.

SIC 287—Agricultural Chemicals

This SIC includes establishments that manufacture agricultural chemicals and pesticides. According to the U.S. Department of Commerce, the 1985 value of shipments of SIC 287 ($14.8 billion) represents 7.5 percent of the value of shipments of SIC 28 [2]. The value of shipments in SIC 287 decreased 9.6 percent from 1981 to 1985. Employment in SIC 287 represented 5 percent of SIC 28, but has declined by 16 percent since 1981. The number of establishments in SIC 287 was approximately 9 percent of all establishments in SIC 28 and production workers account for approximately 62 percent of total employment [Table C-1].

SIC 2873 includes manufacturers of nitrogenous and mixed fertilizers. The value of shipments of nitrogenous fertilizers in 1986 was $2.82 billion, a decrease over 1985 shipments [3, p. 13-1].

SIC 2874 includes manufacturers of phosphatic fertilizers, such as phosphoric acid, made from phosphate rock. The value of shipments of phosphatic fertilizers in 1986 was $3.71 billion, which represents a decrease over 1985 shipments [3, p. 13-3]. Ammonia and phosphoric acid are two substances with potential exposure problems that are produced and/or used in SIC 2874.

SIC 2875 includes establishments that mix fertilizers from purchased fertilizer materials. SIC 2879 includes formulators and preparers of ready-to-use agricultural and household pest control chemicals, such as fungicides, insecticides, and herbicides.

In SIC 287, the survey detected more than twice as many small firms (fewer than 20 production workers) as large firms. In small firms, maintenance work is mostly performed by production workers or dedicated maintenance workers. Large firms primarily employ workers specifically for maintenance duties, although some large firms use outside contractors.

The manufacturers classified in SIC 287 usually have two to four basic processes, with numerous firms having potential exposures to as many as five different substances. Thirty-three percent of the processes involve exposure to these chemicals or substances on an intermittent short-term basis (up to 30 minutes), with large firms tending to have longer-term exposures. Thirty-nine percent of the firms in this SIC reported the adoption of an internal exposure standard. Twenty-three percent of the small firms reported the adoption of an internal exposure standard. Nearly 45 percent of the large firms with standards reported using the OSHA PELs; the remainder indicated that they use ACGIH TLVs. The survey found that about 43 percent of the processes are totally enclosed and about 37 percent are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed. About 42 percent of the firms with chemical exposures have respirators for employee use, with a higher percentage of small firms than large firms reporting the presence of respirators. The combined data on exposure levels and methods of exposure control indicate that most plants which are estimated to incur some cost of compliance do not have overexposures at all processes in that plant.

Survey respondents identified the presence of 32 different substances. Ammonia and particulates not otherwise regulated were estimated to occur the most frequently in a total of 344 and 334 processes, respectively. Ammonia was identified in drying/baking, blending/mixing/formulating, packaging/bagging, crushing/grinding/calcining, loading/offloading/receiving/handling, reaction/fermentation, and separation.

SIC 289—Miscellaneous Chemical Products

This group includes manufacturers of miscellaneous chemical products. For 1985, SIC 289 represented 7 percent ($14.6 billion) of the value of shipments of SIC 28 [2]. From 1981 to 1985, the value of shipments in SIC 289 increased 18.3 percent. The number of employees in SIC 289 was almost 10 percent of SIC 28 and has remained unchanged since 1981. The number of establishments in SIC 289 was approximately 19 percent of all establishments in SIC 28. Production

workers equalled approximately 62 percent of all workers [Table C–1].

SIC 2891 includes manufacturers of industrial and household adhesives and sealants. Industry shipments for adhesives and sealants in 1986 amounted to $4.2 billion, of which about 60 percent were by synthetic resins and rubber-based adhesives; 20 percent by sealant and caulking compounds; and the remaining 20 percent by natural-based adhesives and miscellaneous compounds [3, p. 15–3].

SIC 2892 includes manufacturers of explosives, such as TNT (trinitrotoluene). Ethylene glycol dinitrate is one of the products of this SIC which may have potential exposure problems.

SIC 2893 includes manufacturers of printing ink and SIC 2895 includes manufacturers of carbon black. SIC 2899 includes manufacturers of miscellaneous chemical products, not elsewhere classified. Among these three SICs, ethylene glycol, nitrotoluene, hexylene glycol, trimellitic anhydride, and coal dust are all substances with suspected exposure problems that are either produced or used in these sectors.

In SIC 289, the survey identified less than half as many small firms (fewer than 20 production workers) as large firms. In the small firms, maintenance work is performed largely by production workers, whereas large firms primarily rely on a separate maintenance staff.

The manufacturers classified in SIC 289 usually have one to four basic processes, with potential exposure to one to five substances. Forty-seven percent of all employees are exposed to these chemicals or substances on an intermittent basis (up to 30 minutes), with large firms tending to have more long-term exposures. Sixty-five percent of the firms in this SIC reported the adoption of internal exposure standards. Roughly 48 percent of the small firms and 36 percent of the large firms with standards have adopted OSHA PELs. Employee monitoring had been performed at 67 percent of the processes.

The survey found that nearly 29 percent of the processes are totally enclosed and 12 percent are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed. Almost 28 percent of the firms with chemical exposures have respirators for employee use, with a higher percentage of large firms reporting the presence of respirators than small firms. The combined data on exposure levels and methods of exposure control indicate that very few plants which are estimated to incur some cost of compliance have overexposures at all processes in that plant.

Survey respondents identified the presence of 50 different substances in SIC 289. Toluene was estimated to occur the most frequently at a total of 661 processes. Toluene was identified in packaging/bagging, blending/mixing/formulating, crushing/grinding/calcining, loading/offloading/receiving/handling, and reaction/fermentation.

SIC 29—Petroleum and Related Industries

This industry is divided into petroleum refiners and producers of other related products. Petroleum refineries (SIC 2911) produce fuels (such as gasoline, kerosene, and distillate and residual fuel oils) as well as lubricants and chemical feedstocks. These products are produced through straight distillation of crude oil, redistillation of unfinished petroleum derivatives, cracking, or other processes. Other producers in this sector manufacture asphalt and tar products for paving and roofing (SIC 295) and other lubricating oils, greases, and petroleum and coal products (SIC 299) [1, pp. 127–128].

The 1985 value of shipments for SIC 29 ($179.1 billion) was 7.9 percent of the value of shipments for all manufacturing industries. Petroleum refining dominates SIC 29, accounting for 94 percent of this sector's value of shipments [3, pp. 10–8 to 10–14].

The number and size distribution of establishments in SIC 29 are shown in Table C–1, as is total employment. Relative to value of output, SIC 29 has few establishments and low employment, accounting for less than 1 percent of all manufacturing establishments and employment [7, pp.10, 30].

About 40 percent of the establishments in SIC 29 are petroleum refineries [10], which are large and extremely capital intensive. Production is highly automated; enclosed processes are used throughout. Mean employment size is 105 employees. By contrast, plants in the other industries within SIC 29 are relatively small and less capital intensive, and processes are generally not automated. Mean establishment size in the rest of SIC 29 is 19 employees.

The real value of petroleum product shipments, consumption of petroleum products, petroleum refining capacity, and employment in SIC 29 all peaked between 1977 and 1981. There has been an upturn since 1985, resulting principally from a sharp decline in crude oil prices in the first half of 1986, which stimulated demand for refinery products [3, pp. 10–1 and 10–2]. Demand for petroleum products is expected to grow only slightly in the short run. In the past, trends have been strongly influenced by sharp fluctuations in the price of crude oil [3, pp. 10–3 and 10–4]. In general, low prices for crude oil translate into increased activity for domestic refineries.

The profitability of firms in SIC 29 is low. The median 1985 rate of return on assets (4.4 percent) is the second lowest median return on assets among all 20 two-digit manufacturing industries [6].

Docket comments pertaining to this industry were concerned exclusively with one regulated substance, asphalt. Asphalt is manufactured in petroleum refineries (SIC 2911) and is used to make paving materials (SIC 2951) and roofing materials (SIC 2952). Many commenters [see, for example, Exs. 3–162; 3–420B; 3–895; 3–240; 3–658; 8–5, 581, 3–493B; 3–294; 3–64; 3–22; 3–74; 3–354; 3–966; Tr. 8/9/88, pp. 9–63, 9–65, 9–66, 9–79] provided information on asphalt paving manufacturing, employee exposures, potential costs, and possible impacts; other asphalt applications were not commented on in docket submissions.

Information submitted by firms and trade groups concerned with the manufacture and application of hot-mix asphalt indicated that the manufacture of asphalt paving material falls within SIC 2951, while the activity of paving falls within SIC 1611, Street and Highway Construction. Because the scope of this rulemaking is restricted exclusively to general industry, OSHA has determined that it is most appropriate at this time to defer regulation of asphalt fumes until the Agency has had sufficient time to address the complex health issues associated with this substance and to analyze the impact on the construction industry of establishing a PEL for this substance.

In SIC 29, three out of four firms identified by the survey were small firms (firms with fewer than 20 production workers). In about half of the small firms, maintenance work is performed by production workers; the remainder of small firms employ maintenance workers more often than they use outside contractors for maintenance. Large firms most commonly have a dedicated maintenance staff.

Most employee exposures are intermittent and short-term (up to 30 minutes); of the remaining employee exposures, most are for durations of from 4 to 8 hours (for large firms), or of 1 to 8 hours (for small firms). A slight majority of small firms use some internal exposure standards; most of those that do use internal exposure

standards report using OSHA PELs or ACGIH TLVs. Almost 95 percent of larger firms report using internal exposure standards; of these, most report using OSHA PELs, and about one-quarter reported using ACGIH TLVs. Air monitoring data were collected for over half of the processes in large plants, but for less than one-fourth of the processes in small plants.

The survey found that about 30 percent of the processes are totally enclosed and almost two-thirds of plant processes are located outdoors. Production workers use respirators in over 25 percent of processes for firms reporting chemical exposures; however, small firms report a lower percentage of respirator use than do large firms.

Survey respondents identified the presence of 68 different substances in SIC 29. Toluene was estimated to occur the most frequently at a total of 175 processes; trichlororethylene was estimated to occur at a total of 162 processes. Toluene was identified in batch process/coke production and removal, blending/mixing/formulating, and process inspection. Trichloroethylene was identified in blending/mixing/formulating, drying/baking, loading/offloading and measurement.

SIC 30—Rubber and Miscellaneous Plastics Products Industry

This industry sector consists of establishments that manufacture a variety of products from plastic resins and from natural, synthetic, and reclaimed rubber. Although plastic products account for the largest share of the value of shipments of this industry group, the industry also manufactures a variety of rubber products, including tires, inner tubes, footwear, and belting [1, pp. 129–132]. The value of shipments for 1985 was $71.3 billion. This industry is dominated by the miscellaneous plastic products sector (SIC 307 until 1987 and now SIC 308), which accounts for 81 percent of the establishments, 66 percent of the value of shipments, and 70 percent of the employment for the entire industry group [10]. The tire and inner tube (SIC 301) sector and the miscellaneous rubber products (SIC 306) sector are the other major components of this industry.

Similar processes are used in manufacturing plastic and rubber products, with the nature and form of the final product determining the process more than the product's components. A product's components, however, determine the types of chemical exposures employees experience. Examples of particularly serious types of exposures are those to the foaming agents that are used in the production of foam rubber or plastic foams and to the styrene used to produce polystyrene or in lamination processes.

As shown in Table C–1, this industry sector is characterized by relatively small establishments; 61 percent of establishments have fewer than 20 employees, with an average of 43 employees per establishment. Employment in this industry grew by 7 percent between 1981 and 1985, with growth in the tire and inner tube and miscellaneous plastics product sectors outpacing declines in other sectors [4]. Firms in this industry have above-average profits for manufacturing industries, with a 7.7 percent median rate of return on assets compared with a 7.0 percent median for all manufacturing firms [6].

The only comments received by OSHA that were related to SIC 30, Rubber and Miscellaneous Plastics, concerned the Agency's proposed 50-ppm TWA and 100-ppm STEL limits for styrene [See, for example, Ex. 3–742; Tr. 8/8/88, pp. 95, 177, 178, 180]. Styrene is used in this sector to make a variety of rubber and plastic products, including polyester resins, polystyrene, and a widely used form of artificial rubber. Commenters stated that a small number of the facilities in this sector, i.e., those using styrene resins in open-mold processes, would encounter technological problems in attempting to comply with the proposed styrene limits [Ex. 3–742, pp. 34–36; Tr. 8/3/88, p. 5–95]. This issue is addressed in Chapter F—Technological Feasibility.

Open-mold processes were described by these commenters as operations in which the styrene resin is applied directly to the surface of a mold (generally by means of a spray gun) and is then rolled by hand to build up successive layers of reinforced plastic. When the objects being molded are large, as is the case with boats or underground storage tanks, commenters explained that it is more difficult to position and use local exhaust ventilation effectively [Ex. 3–742, p. 48]. Although most open-mold processes in this sector are involved in the manufacture of plastic bathroom fixtures (showers, tubs, hot tubs, and spas), makers of underground storage tanks and cultured marble products also rely on the open-molding process.

The Styrene Information and Research Council estimates that 265 facilities in this sector use this process to produce bathroom fixtures [Ex. 3–742, p. 105], and the Cultured Marble Institute estimates that a total of 1062 facilities, employing 17,000 workers, manufacture cultured marble products [Tr. 8/3/88, pp. 5–77, 5–177, 5–180]. These firms, like other styrene-using firms in this SIC code, are generally small, privately held firms. The Cultured Marble Institute characterized the typical open-mold-process firm in this sector as a company that employs 17 persons and has annual sales of less than $1 million. The issues of technological feasibility that pertain to users of this process in SIC 30 are discussed in detail in the Technological Feasibility chapter, below.

In this SIC, over 60 percent of the firms identified by the survey were small firms (firms having fewer than 20 production workers.) In the small firms, maintenance work is most commonly performed by production workers, although about one-quarter of small firms use outside contractors for maintenance work, and one in seven has a dedicated maintenance staff. Over two-thirds of large firms have dedicated maintenance staff; the remaining large firms use production workers for maintenance more often than they use outside contractors.

Most firms reported using from one to four processes. In SIC 307 (miscellaneous plastics manufactuing), most firms reported using from one to three chemicals, with styrene the most prevalent; however, in rubber manufacturing (SICs 301 to 306), almost half of the firms reported using 6 to 10 chemicals. Most employee exposures in small firms are intermittent and short-term (up to 30 minutes), and there are very few exposures for 4 hours or more. In large firms, by contrast, the majority of chemical exposures are for 4 to 8 hours a day. In this SIC, most small firms have internal exposure standards; the majority of these reported using ACGIH TLVs. Large firms most commonly use OSHA PELs, but many use ACGIH TLVs or have no internal exposure standards. Air monitoring data were provided for about 40 percent of large firms and for approximately 13 percent of small firms.

The survey found that about 37 percent of the processes are totally enclosed, and that very few processes are located outdoors. In one-third of the firms with chemical exposures, production workers use respirators.

Survey respondents identified the presence of 75 different substances in SIC 30. Ethylene glycol was estimated to occur the most frequently at a total of 1,889 processes, including assembly, blending/mixing/formulating, calendaring/winding and coating/spraying. Methyl chloroform was estimated to occur in 1,852 processes

including blending/mixing/formulating, coating/spraying, and cutting/sawing/planing.

SIC 31—Leather and Leather Products

The leather and leather products industry (SIC 31) consists of several sectors such as leather tanning (SIC 311), boot and shoe cut stock (SIC 313), non-rubber footwear (SIC 314), and luggage and leather goods (SICs 315–319). [1, pp. 133–135]. Shipments of leather products increased in 1987, while employment in the leather industry has been declining steadily over the past several years [3, p. 46–1].

Data on the number of establishments and employment for 1985 are shown in Table C–1. In 1985, there were approximately 3,940 establishments engaged in the production of leather and leather products. Over 64 percent of these establishments employed fewer than 20 workers. The largest employer is the non-rubber footwear industry, with 58 percent of the workforce in 1986. Production workers make up 84 percent of the total workforce in SIC 31.

According to the U.S. Department of Commerce, the 1986 value of shipments for leather and leather products ($7.8 billion) was down 8.8 percent from 1985. The total represents 0.4 percent of the value of shipments for all manufacturing industries. Non-rubber footwear (SIC 314) makes up most of the value of shipments in this industry, with 51 percent of the total value [2]. The median return on assets in 1985 for the leather and leather product industry was 6.3 percent [6].

The number of establishments in the leather tanning and finishing industry (SIC 311) has decreased by over 248 establishments, from 384 establishments in 1982 to 136 establishments in 1987. Employment has also decreased significantly while shipments increased to $2.0 billion in 1987 from $1.7 billion in 1986. Since the leather tanning industry is highly dependent on the demand from the non-rubber footwear industry, it is not likely that the situation will improve in the near future [3, pp. 46–1 and 46–2].

The non-rubber footwear industry (SIC 314) had a small increase in the value of shipments in 1987 ($4.1 billion), while total employment and the number of production workers declined 3.0 percent and 2.9 percent, respectively. This industry has suffered substantially since 1981 when an import restraint agreement with South Korea and Taiwan expired. Since then, import's share of the domestic market has increased to over 81 percent in 1987, to an estimated 226 million pairs [3, pp. 46–5 to 46–10].

The miscellaneous luggage and leather goods industry (SICs 315–319) saw improvements in production, employment, and shipments in 1987, reversing a past trend. Shipments were expected to increase 3.9 percent in 1987 to $1.9 billion. The estimated number of production workers also increased in 1987, to 27,200 employees from 27,000 employees in 1986. Imports reached over 52 percent of the domestic market in 1986 [3, pp. 46–10 to 46–14].

In this SIC, the survey identified almost twice as many small firms (fewer than 20 production workers) as large firms. In the small firms, a large share of maintenance work is performed by production workers, although one-fifth of the firms use outside contractors and one-fifth of the firms employ maintenance staffs. Large firms have a dedicated maintenance staff that performs most of the maintenance work, while production workers and outside contractors do the rest of the maintenance work.

The manufacturers classified in this SIC usually have one to three basic processes, with potential exposure to seven to eight chemicals. Employees are exposed to these chemicals on an intermitten short term basis (up to 30 minutes) or continuously (up to 8 hours per day) with large firms tending to have more long-term exposures. Small firms generally have no internal exposure standards. Around one-half of large firms reported using the OSHA PELs; the balance indicated that they have no standards. Air monitoring data was being done at about one-half of the processes found in large plants.

The survey found that over forty percent of the processes are totally enclosed and less than one percent are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed or outdoors. In one-tenth of the firms with chemical exposures, production workers use respirators, with a higher percentage of large firms using respirators than small firms. The combined data on exposure levels and methods of exposure control indicate that overexposure is not occurring at many processes in this SIC.

Survey respondents identified the presence of 42 different substances in SIC 31. N-hexane was estimated to occur the most frequently at a total of 426 processes, primarily gluing/hot pressing. Toluene was estimated to occur at a total of 319 processes including cleaning, coating/spraying, gluing/hot pressing, and stamping/shaping.

SIC 32—Stone, Clay, and Glass Products

This industry is made up of products such as cement (SIC 324), concrete (SIC 327), pottery (SIC 326), stone (SIC 328), glass (SICs 321–323), and structural clay products (SIC 325). Since these products are primarily used as construction materials, the industry is heavily dependent on the amount of new construction activity in a given year.

There were 21,054 establishments in the stone, clay and glass industry (SIC 32) in 1985. Most of these firms (73.7 percent) employed fewer than 20 workers in 1985. The mean establishment size was 28 employees. Total employment was 514,000 in 1986, a decrease of 1.1 percent over the 1985 total employment figure of 520,000 [7]. Production employment also declined from 1985 to 1986.

In 1986, the value of shipments in SIC 32 ($57.3 billion) increased 3.9 percent over the 1985 figure. The total value was 2.5 percent of the value of shipments for all manufacturing industries. The value of shipments is evenly distributed over the entire industry, except for the concrete sector (SIC 327) with 36.4 percent of shipment [2]. The median rate of return on assets for SIC 32 was 6.5 percent in 1985 [6].

The concrete industry (SIC 327) experienced a small decline in shipments in 1987 after considerable improvement in production, employment, and demand over the past years. The demand for concrete has increased substantially since 1982, when shipments were 23 percent below their current figure. Future demand for concrete depends mainly on non-residential building construction activity [3, pp. 2–7 to 2–8].

The cement industry (SIC 324) experienced a decline in the value of shipments, from $4.1 billion in 1986 to $3.9 billion in 1987, a decrease of 3.3 percent. Consumption of cement also declined in 1987 by 1 percent, the first annual decline since 1982. However, industry shipments were more than 26 percent higher than the 1982 low point of 65 million tons. Total employment was 19,500 in 1987. Production employment (14,500 employees in 1987) represented approximately 74 percent of the workforce [3, pp. 2–4 to 2–6].

The glass industry (SICs 321–323) has experienced steady growth over the past two years, mainly in production and shipments. The value of shipments for the glass industry increased from $13.9 billion in 1985 to $14.6 billion in 1986. New product introductions have allowed the glass industry to make substantial gains in winning market

share. Total employment and production employment declined for the glass industry (SICs 321–323) in 1986, but SIC 323 (products of purchased glass) did have increased in both total employment and production employment. The outlook for continued growth for the glass industry is good [3, pp. 2–9 to 2–12].

Shipments of structural clay products and pottery (SICs 325–326) have increased substantially over the past few years, from 5.1 billion bricks in 1982 to 7.4 billion bricks in 1986. The 1986 value of shipments for SICs 325–326 was $4.9 billion, an increase of 4.3 percent from 1985. The outlook for the industry is for slow growth in the near future [3, pp. 2–12 to 2–13].

The stone industry (SIC 328) had an increase of 1.3 percent in the value of shipments in 1986. Total employment and production employment stayed virtually the same [2].

In SIC 32, the survey identified over three times as many small firms (fewer than 20 production workers) as large firms. In the small firms, over three-fourths of maintenance work is performed by production workers, although some firms to employ a maintenance staff. Large firms use a dedicated maintenance staff for approximately two-thirds of the maintenance work, while one-fourth use production workers. The remainder of firms use outside contractors.

The manufacturers classified in this SIC usually have one to three basic processes, with potential exposures to as many as eight chemicals. Employees are exposed to these chemicals on an intermittent short term basis (up to 30 minutes) or continuously (up to 8 hours per day), with large firms tending to have more long-term exposures. Small firms generally have no internal exposure standards; when they do, the OSHA PELs are followed about seventy percent of the time. Approximately one-half of large firms reported using the OSHA PELs; the balance indicated that they have no standards or they rely on ACGIH TLVs. Air monitoring data were provided for over one-half of the processes found in large plants.

The survey found that about one-third of the processes are totally enclosed and around one-fifth are located outdoors. Local exhaust ventilation and respirators are used most frequently to control exposures at processes not enclosed or outdoors. In almost one-half of the firms with chemical exposures, production workers use respirators, with a higher percentage of large firms using respirators than small firms. The combined data on exposure level and methods of exposure control indicate that overexposure is not occurring at many processes in this industry.

Survey respondents in this SIC identified blending/mixing/formulating, chipping/grinding, drilling/cutting/flame-jet lancing, polishing (surface) grinding, cutting/sawing/planing, casting, batch making, and bonding as the processes used most often. Chemicals that were present in these processes included: acetone, ammonia, calcium oxide, furfuryl alcohol, graphite, magnesium oxide fume, and silica. The National Lime Association commented on the presence of calcium hydroxide and calcium oxide in this industry [Ex. 3–890].

SIC 33–Primary Metal Industries

The primary metal industry (SIC 33) is divided into two different sectors: nonferrous metals and foundries (SICs 333–336) and ferrous metals and foundries (SICs 331–332) [1, pp. 145–152]. This includes the basic iron and steel industry, and the metals industry. Both sectors have been hurt in the recent past by a decline in domestic consumption and the growing number of imports into the United States. The future for these industries, however, looks brighter due to an increase in orders, slowing imports, and a decrease in capacity [10]. These industries have had increases in prices, shipments, and profits in 1987 and 1988, helped by the fall in the value of the dollar.

As seen in Table C–1, the number of establishments in SIC 33 in 1985 totaled 10,101. The majority of these firms (55.3 percent) had fewer than 20 employees in 1985. Total employment (808,000 employees in 1985) and production employment (612,000 in 1985) have declined over the last several years, while the average hourly wage of production workers ($12.76 in 1986) has increased by 1.5 percent from 1985 to 1986 [7]. The mean establishment size was 80 employees in 1985. However, according to the American Iron and Steel Institute (AISI), integrated steel mills are typically much larger, averaging 825 workers [Ex. 3–1123, p. 14].

Production in the steel mill products industry has declined over the past few years, from 92.5 million tons in 1984 to 83.0 million tons in 1987, a decline of 10.3 percent [3, p. 20–1]. The 1986 value of shipments ($105.6 billion) in SIC 33 was 4.7 percent of the value of shipments for all manufacturing industries [2]. The median rate of return on assets in 1985 was 5.5 percent for the primary metal industry [6].

In 1987, the industry had its first profitable year since 1982. The industry has cut costs of production while prices have remained steady. In 1988, the industry experienced additional improvement; production was up 15 percent and shipments up 12 percent. Prices and profits rose considerably during the year, and the outlook for 1989 is good [11].

The import situation has also improved for the domestic steel industry, due in part to the falling value of the dollar against major competitors such as Japan and Europe. Imports have been declining since their peak of 26.2 million tons in 1984. Imports as a percent of domestic consumption fell to 22 percent in 1987, down from a peak of 26.4 percent in 1984. Exports reached 1.1 million tons in 1987 [3, pp. 20–1 to 20–9]. Exports during 1988 rose about 50 percent over the previous year.

The ferrous castings industry (SIC 332) has shown a poor performance over the past few years, but is starting to improve. The value of shipments for SIC 332 has increased, from $10.3 billion in 1986 to $10.8 billion in 1987. The value of shipments for SIC 332 is forecast to increase 5.2 percent in 1988, although this trend is not likely to continue in the future. Total employment and the number of production workers have also begun to increase, by 2.3 percent and 1.9 percent, respectively, from 1986 to 1987 [3, pp. 20–6 to 20–7].

Primary nonferrous metals can be classified in four categories: aluminum, zinc, lead, and copper. Aluminum industry shipments have increased steadily in the past few years, with an 11.3 percent increase in 1987. Shipments are projected to continue rising through 1992 [3, pp. 21–8 to 21–11]. Prices during the last several years have continued to increase, from 53¢/pound in the last quarter of 1986 to 83¢/pound at the end of 1987 and $1.12/pound by the end of 1988 [12, 13].

The zinc industry should have steady growth over the next few years, due mainly to an increase in consumption. The price of zinc has risen from 38¢/pound in 1986 to 42.5¢/pound in 1987. Domestic consumption increased to 1.014 million metric tons in 1987. The value of shipments increased by 1.4 percent in 1987, and is expected to increase by another 1.6 percent in 1988. Total employment and the number of production workers has remained steady for the past several years [3, pp. 21–14 to 21–16].

Consumption of primary lead products increased slightly over 3 percent in 1987–88 owing to increases in the replacement battery market. Automotive products account for about 70 percent of all demand for lead. Changes in recycling patterns due to EPA RCRA

regulations may increase demand for primary lead in the near future. The market in general has been growing at a steady 1 percent per year [3, pp. 21–6 to 21–8 and 14]. Prices have risen in recent months to 42¢/pound [12] from 36.9¢ in 1987. ASARCO, one of two primary lead producers in the U.S., is considering adopting London Metal Exchange prices in lieu of its own pricing [4].

The copper industry has been undergoing restructuring to remain competitive in the world market. Currently, there are seven operating copper smelters, compared to fourteen in 1970. This restructuring has forced the industry to decrease capacity and reduce employment [3, pp. 21–11 to 21–14]. The price of copper has increased from 66.1¢/pound in 1986 to 75.0¢/pound in 1987 due to a decline in inventories [3, pp. 21–11 to 21–14]. Current 1988 cash prices for copper have risen to $1.64/pound [12]. The Peruvian copper fields are estimated to need an additional 30 days to return to full production following the recent 54 day strike by miners [15] This should allow the industry to turn a profit for the first time in several years. The copper smelting industry is likely to be impacted by the proposed revision to the PEL for sulfur dioxide.

In SIC 33, the survey identified slightly more small firms (fewer than 20 production workers) than large firms. Maintenance work in the small firm is done primarily by production workers although some firms use a dedicated maintenance staff. Large firms generally have maintenance work performed by the maintenance staff, with the remainder of firms using production workers and outside contractors.

The manufacturers classified in this SIC who reported chemical or process use usually have one to four basic processes, with potential exposure to one to four chemicals. Employees are exposed to these chemicals on an intermittent short term basis (up to 30 minutes) or continuously (up to 8 hours per day), with large firms tending to have more long-term exposures. Small firms generally have some internal exposure standards; when they do, the OSHA PELs are followed about three-fourths of the time. Over one-half of large firms reported using the OSHA PELs; the balance indicated that they have no standards or they rely on ACGIH TLVs. Air monitoring data were provided for approximately two-thirds of the processes found in large plants.

The survey found that about one-fourth of the processes are totally enclosed and less than 3 percent are located outdoors. Local exhaust ventilation and respirators are used most frequently to control exposures at processes not enclosed or outdoors. In almost one-half of the firms with chemical exposures, production workers use respirators, with a higher percentage of large firms using respirators than small firms. The combined data on exposure levels and methods of exposure control indicate that overexposure may occur at less than one-tenth of processes in small firms and at about one-fifth of the process in large firms.

Survey respondents in SIC 33 identified metal melting/pouring/casting as the process most frequently used, with exposure to aluminum metals, carbon monoxide, and copper fume reported most frequently. The American Cast Metals Association confirmed the presence of most of the chemicals surveyed [Exs. 3–673 and 3–675]. The American Iron and Steel Institute also commented on several of the chemicals identified in the survey [See, for example, Ex. 3–1123].

SIC 34—Fabricated Metal Products

The fabricated metal products industry (SIC 34) can be broken down into nine categories: metal cans and shipping containers (SIC 341); cutlery and hand tools (SIC 342); heating equipment (SIC 343); fabricated structural metal products (SIC 344); screw machine products, bolts, and washers (SIC 345); forgings and stampings (SIC 346); plating and coating (SIC 347); small arms and ordnance (SIC 348); and miscellaneous wire and fabricated products (SIC 349). SIC 34 excludes machinery and transportation equipment [1, pp. 153–166].

The total number of establishments in the fabricated metal products industry in 1985 was 46,322. The majority of these firms (67.0 percent) have fewer than 20 employees, a change of 0.2 percent since 1984. Total employment in this industry has reached 1.5 million employees, an increase of 0.1 percent since 1984 [7].

The 1986 value of shipments for SIC 34 ($138.0 billion) represents a 1.1 percent decrease over 1985. This was 6.1 percent of the value of shipments for all manufacturing industries [2]. The median return on assets for the fabricated metal products industry in 1985 was 7.1 percent [2].

Metal can (SIC 3411) shipments have been increasing steadily in the past few years, from 104.7 billion units in 1986 to 109.3 billion units in 1987, an increase of over 4.4 percent. This was due mainly to the increase in soft drink and beer cans being shipped. The value of shipments has also increased, with a compound annual increase of 2.9 percent from 1980 to 1985. Total employment in the metal cans industry has remained steady, with a slight increase expected in 1987. The number of production workers has increased slightly, with an increase of 0.3 percent from 1986 to 1987. Exports of metal cans have decreased substantially since 1984 when they reached an all-time high of $56.5 million. Since that time they have decreased to $36.2 million in 1987 [3, pp. 7–1 to 7.4].

The fabricated structural metal industry (SIC 3441) produces structural metal components used primarily in the construction industry. Shipments of fabricated structural metal decreased slightly, from $9.0 billion in 1986 to $8.9 billion in 1987. Total employment decreased slightly in 1987 [3, pp. 2–3 to 2–5].

The value of shipments in the screw machine products, bolts, and washers industry (SIC 345) decreased slightly from 1986 to 1987, from $7.8 billion to $7.9 billion. Total employment increased from 94,000 in 1986 to 94,400 in 1987. Since the automotive industry is the major customer for this industry, stable automotive sales are the key to economic health for this industry sector [3, pp. 26–1 to 26–6].

In SIC 34, the survey identified over twice as many small firms (fewer than 20 production workers) as large firms. In the small firms, about one-half of the firms have maintenance work performed by production workers, the remaining firms using maintenance workers or outside contractors. Large firms generally employ a maintenance staff to do the majority of maintenance work, although some firms use production workers and outside contractors.

The manufacturers classified in this SIC usually have one to three basic processes, with potential exposure to one to four chemicals. Employees are exposed to these chemicals on an intermittent short term basis (up to 30 minutes) or continuously (up to 8 hours per day) with large firms tending to have more long-term exposures. Small firms generally have no internal exposure standards; when they do, the OSHA PELs are followed about one-half of the time. Over one-half of large firms reported using the OSHA PELs; the balance indicated that they have no standards or they rely on ACGIH TLVs. Air monitoring data were provided for about one-half of the processes found in large plants.

The survey found that about one-fourth of the processes are totally enclosed and around one-fifth are located outdoors. Local exhaust ventilation and respirators are used most frequently to control exposures at processes not enclosed or outdoors. In

over one-half of the firms with chemical exposures, production workers use respirators, with a higher percentage of large firms using respirators than small firms. The combined data on exposure levels and methods of exposure control indicate that overexposure is not occurring at any processes in small firms and at less than one-tenth of the processes in large firms.

Survey respondents in this SIC identified casting/painting, welding/soldering, polishing (surface)/grinding, and degreasing, as the processes most frequently used. Welding fumes, iron oxide, and isopropyl alcohol were the chemicals identified most often in the survey. OSHA has retained the existing limit for iron oxide. No comments were received relative to processes and chemicals in this SIC.

SIC 35—Non-Electrical Machinery

The non-electrical machinery industry (SIC 35) is made up of several different sectors: engines and turbines (SIC 351); farm and garden machinery (SIC 352); construction and related machinery (SIC 353); metal working machinery (SIC 354); special industry machinery (SIC 355); general industrial machinery (SIC 356); computer and office equipment (SIC 357); refrigeration and service industry machinery (SIC 358); and miscellaneous machinery and equipment (SIC 359) [1, pp. 167–183].

As seen in Table C–1, the number of establishments in 1985 totaled 77,748. The majority of these (77.1 percent) had fewer than 20 employees in 1985. Total employment and production employment have decreased over the last several years. The 1986 value of shipment ($208.5 billion) in SIC 35 was 9.2 percent of the value of shipments for all manufacturing industries [2]. In 1985, the median rate of return on assets for SIC 35 was 7.5 percent [6].

The 1986 value of shipments for engines and turbines (SIC 351) was $14.1 billion, a decrease of 5.5 percent of the 1985 value of shipments ($14.9 billion). Both total employment and production employment decreased from 1985 to 1986, by 8.8 percent and 9.3 percent, respectively. Major expansions of electrical power generation capacity and hence, turbine manufacture have been curtailed in recent years as cogeneration facilities are now providing additional power. Smaller units for these same cogeneration facilties have provided some additional sales [6]. The largest sector of SIC 351 is internal combustion engines, n.e.c., with 77 percent of the value of shipments in 1986.

The farm and garden machinery industry has experienced some improvement in 1987. While the value of shipments for lawn and garden equipment increased in 1987, the value of shipments for farm machinery and equipment ($7.0 billion) declined to their lowest level since 1973. Total employment, which exceeded 125,000 in 1981, dropped to around 67,000 in 1987. Production employment, which makes up approximately two-thirds of the work force, has also been declining since 1979. The prospects for lawn and garden equipment appear much better, with steady increases in the value of shipments since 1981. The 1987 value of shipments for lawn and garden equipment ($3.7 billion) was 4.2 percent greater than the 1986 value of shipments ($3.5 billion) [2]. This industry had a compound annual increase of 8.9 percent from 1980 to 1985 in value of shipments. Total employment and production employment have remained fairly steady, with compound annual increases of 1.9 percent and 3.8 percent, respectively [3, pp. 25–1 to 25–3].

The construction and related machinery industry (SIC 353) has experienced a decline in recent years. The value of shipments for SIC 353 declined by 6.2 percent, from $27.7 billion in 1985 to $25.9 billion in 1986. Both total employment and production employment fell from 1985 to 1986, by 8.7 percent and 11.7 percent, respectively [2]. The decline of the dollar value of shipments must be viewed against a background of reorganization and price cutting by American manufacturers resulting in leaner, more efficient organizations that can make a profit at lower levels of sales. Significant market share has been regained [17]. Construction machinery makes up the largest share of this industry, with approximately half of the total value of shipments.

The machine tool industry has had a major improvement in orders, and profits during 1988. "Orders for all of 1988 climbed to about $3.5 billion, up 66% from the $2.1 billion range for both 1986 and 1987" [18]. The 1986 value of shipments for metal working machinery (SIC 354) was $20.5 billion, an increase of 3.2 percent over 1985. Although shipments increased in this industry in 1986, both total employment and production employment fell during the same time period [2]. This is a reflection of the downsizing and modernizing that has been undertaken in this industry. In the future, moderate sales improvements showed a positive impact on earnings [19]. The largest sector within the metal-working industry is special dies, tools, jigs, and fixtures, with 38 percent of the value of shipments and 43 percent of the total workforce.

Special industry machinery (SIC 355) has experienced stable growth in the past, and this trend is likely to continue into the future. Industry shipments increased approximately one percent, from $14.8 billion in 1985 to $14.9 billion in 1986. Special industry machinery, n.e.c. (SIC 3559) is the largest sector within this industry, with 41.9 percent of the total value of shipments in 1986. Both total employment and production employment have been falling, by 5.0 percent and 7.5 percent, respectively, from 1985 to 1986. Production employees make up approximately 57.0 percent of the total workforce.

The 1986 value of shipments for general industrial machiney, SIC 356, ($24.8 billion) fell from the 1985 value of shipments ($25.3 billion) by an estimated 2.4 percent. Employment and industry shipments are divided fairly evenly over the entire industry, with pumps and pumping equipment (SIC 3561) and general industrial machinery, n.e.c. (SIC 3569) being the largest sectors. Total employment declined by 4.1 percent, while the number of production workers fell by 5.8 percent [4].

The computer industry (SIC 357) has had stable demand for its products in the U.S. market during 1986 and 1987. The value of shipments of office and computing machines (SIC 357) decreased from $62.2 billion in 1985 to $58.8 billion in 1986, a decline of 5.5 percent reflecting strong price competition. Electronic computing equipment (SIC 3573) is the largest segment, with 89 percent of the value of shipments. Total employment and the number of production workers have declined since 1985 by 10.8 percent and 12.7 percent, respectively. Imported computer equipment has made significant inroads into the domestic market, due mainly to the standardization of products and the fall in the price of computer equipment [3, pp. 30–1 to 30–11]. The dollar value of 1988 shipments is ahead of 1987 shipments [20].

The refrigeration and service machinery industry (SIC 358) had an annual rate of growth of 0.9 percent from 1985 to 1986, attributable to the increase in new residential construction. While total employment and the number of production workers have increased, imports have also been steadily increasing [3, pp. 22–9 to 22–11].

It appears that the general industrial machinery industry (SIC 35) could be affected by several of the proposed revisions. The following substances are used or generated by this industry: carbon dioxide, chlorine, chromium metal, fibrous glass dust, furfuryl

alcohol, iron oxide, manganese fumes, nitrogen dioxide, oil mist, sulfur dioxide, 1,1,2-trichloro-1,2,2-trifluorethane, triethylamine, tungsten, welding fumes, wood dust and asphalt fumes. The majority of comments from the general industrial machinery industry deal with the appropriateness of the PELs rather than technical or economic feasibility.

The Association of Reproduction Materials Manufacturers (ARMM) commented on their opposition to the proposed revision for ammonia based on health effects and the inappropriateness of adopting ACGIH standards. ARMM is a trade group with 47 company members who supply materials and equipment to over 5,000 commerical blueprinters [Ex. 8–29].

The International Institute of Ammonia Refrigeration opposed the proposed standard for ammonia based on health effects and economic feasibility. In the final rule, only a STEL of 35 ppm has been set for this substance.

In this SIC, the survey identified over four times as many small firms (fewer than 20 production workers) as large firms. In the small firms, maintenance work is performed in large part by production workers, although some firms employ dedicated maintenance staffs or use outside contractors. Large firms have the majority of maintenance work performed by a dedicated maintenance staff, with some use of production workers or outside contractors.

The manufacturers classified in this SIC usually have from one to four basic processes, with potential exposure to one to four chemicals. Employees in SIC 35 are exposed to these chemicals for varying amounts of time from intermittent short term periods (up to 30 minutes) to continuously (up to 8 hours per day), with small firms having more intermittent short term exposures and large firms tending to have more long-term exposures. Small firms generally have no internal exposure standards; when they do, the OSHA PELs are followed about one-third of the time. Over one-half of large firms reported using the OSHA PELs; the balance indicated that they have no standards or they rely on ACGIH TLVs. Air monitoring data were provided for about one-half of the processes found in large plants.

The survey found that about one-fifth of the processes are totally enclosed and 5 percent are located outdoors. Local exhaust ventilation, general dilution ventilation and respirators are used most frequently to control exposures at processes not enclosed or outdoors. In over one-half of the firms with chemical exposures, production workers use respirators, with large firms and small firms using respirators at about the same rate.

Survey respondents in this SIC identified polishing (surface)/grinding, coating/painting, and soldering as the processes which occur most frequently. Chemicals that were present most often were welding fumes, oil mist, and stoddard solvent. Comments from Caterpillar Incorporated and John Deere and Co. confirmed the presence of several of the survey chemicals in SIC 35 such as chromium metal, iron oxide, oil mist, welding fumes, and 1,1,2-trichloro-1,2,2-trifluorethane [Ex. 3–349]. In the final rule, OSHA has not revised the existing limits for chromium metal, iron oxide and oil mist.

SIC 36—Electric and Electronic Equipment

This industry is made up of several distinct sectors: electric distributing equipment (SIC 361); electrical industrial apparatus (SIC 362; household appliances (SIC 363); electrical lighting and wiring equipment (SIC 364); radio and TV receiving equipment and communication equipment (SIC 365–366); electronic components and accessories (SIC 367); and miscellaneous electronic equipment (SIC 369) [1, pp. 194–195].

In 1985, the electric and electronic equipment industry employed about 2.2 million workers. The majority of the firms had fewer than 20 employees. The value of shipments for all electric and electronic equipment establishments in 1986 was $196.2 billion. This was 8.7 percent of the value of shipments for all manufacturing industries. Most of the value of shipments in SIC 36 is from the communication equipment sector ($67.4 billion or 34.4 percent) [2]. The median return on assets for the electric and electronic equipment industry was 7.9 percent in 1985 [6].

The electric distributing equipment industry (SIC 361) had mixed performance during 1987. While the value of shipments increased for switchgear by 0.5 percent, the value of shipments for transformers decreased by 6.6 percent from 1986 to 1987. Total employment and the number of production workers has remained fairly steady since the early 1980's [3, p. 28–4].

Motors and industrial controls (SIC 362) have had stable sales during the last several years. Future growth is dependent upon the economy in general and construction growth for any sizable increases in sales. Motors have significant import pressure; several domestic manufacturers have manufacturing facilities in Mexico. Industrial controls are expected to grow by 2.5 percent [3, pp. 28–1 to 28–3].

The household appliance industry (SIC 363) has had a steady increase in shipments since the early 1980's, from $16.8 billion in 1986 to $17.7 billion in 1987, an increase of 5.7 percent. The industry is optimistic about its future, due mainly to increased residential construction and an anticipated increase in disposable income. Imports have not been a substantial factor in this industry (exports have not increased either). Total employment and the number of production workers declined from 1980 to 1985 by 4.4 percent and 4.3 percent, respectively. This decline in employment is due to the recent number of acquisitions within the industry and the need to cut costs of production. In 1987, total employment and production employment increased slightly [3, pp. 47–8 to 47–11].

The value of shipments for the electrical lighting and wiring industry (SIC 364) has been increasing steadily over the last decade, from 11,321 in 1980 to 15,806 in 1985, an increase of 39.6 percent. However, total employment and the number of production workers have decreased slightly. Performance in this industry is related, in part, to activity in the construction industry. Since the electrical lighting and wiring industry depends on both residential and non-residential construction, it is able to withstand a slowdown in one sector as long as the other sector is still active [3, pp. 4–1 to 4–4].

The consumer electronics and communication equipment industry (SICs 365–366) has had mixed performance in the past. The communication equipment industry has performed well, while the consumer electronics industry has not performed as well, due to import competition. Overall, the value of industry shipments has remained fairly stable, with shipments increasing in the communication equipment industry and shipments decreasing in the consumer electronic industry. Total employment and the number of production workers also follow this pattern, decreasing for consumer electronics and increasing for communication [3, pp. 31–1 to 31–8 and 32–1 to 32–6 and 47–7].

The electronic components and accessories industry (SIC 367) is expected to show record growth over the next few years. Industry shipments were up 8.3 percent, from $43.9 billion in 1986 to $47.5 billion in 1987. This was due, in part, to the strong performance of the defense electronics industry. The number of production workers and total employment have remained fairly

steady in 1986 and 1987. Imports are still increasing, but may be slowed due to the fall in the value of the dollar [2, pp. 32–1 to 32–4].

In SIC 36, the survey estimated that almost 70 percent of the firms are small firms (fewer than 20 production workers). Maintenance work is usually performed by production workers in the small firms and a dedicated maintenance staff for the large firms.

The manufacturers classified in this SIC usually have one to three processes, with potential exposure to one to six chemicals. Employees are exposed to these chemicals on an intermittent short term basis (up to 30 minutes) or continuously (up to 8 hours per day), with large firms tending to have more long-term exposures. Small firms generally have no internal exposure standards; when they do, the OSHA PELs are followed three-fourths of the time. Almost two-thirds of large firms reported using the OSHA PELs; the balance indicated that they use the ACGIH TLVs most frequently. Air monitoring data were provided for almost one-half of the processes found in large plants.

The survey found that less than one-fifth of the processes were totally enclosed and less than one percent located outdoors. General dilution and local exhaust ventilation are used about equally to control exposures at processes not enclosed or outdoors. In about one-quarter of the firms with chemical exposures, production workers use respirators, with a higher percentage of large firms using respirators than small firms. The combined data on exposure levels and methods of exposure indicate that overexposures may occur only in some processes in this industry.

Survey respondents in this SIC identified coating/painting, polishing (surface)/grinding, processing, and degreasing as the processes which occur most frequently, and tin, stoddard solvent, and zinc oxide as the chemicals most frequently used. No comments addressed the processes or chemicals in this SIC.

SIC 37—Transportation Equipment

This industry sector includes establishments engaged in manufacturing equipment for land, sea, air, or space transportation and includes manufacturers of parts and accessories as well as complete vehicles.

The major subdivisions within this sector are motor vehicles and motor vehicle equipment (SIC 371), aircraft and parts (SIC 372), ship and boat building and repair (SIC 373), railroad equipment (SIC 374), motorcycles, bicycles and parts (SIC 375), guided missiles, space vehicles and parts (SIC 376), and miscellaneous transportation equipment (SIC 379). Establishments in the miscellaneous subdivision manufacture a broad range of products (e.g., from tanks to wheelbarrows) [1, pp. 196–201]. Because the manufacture of transportation equipment involves a wide range of industrial processes, establishments in this sector often include or involve foundries, electroplating operators, various types of hot metal work, welding, laminating, plastic molding, and painting and coating. Workers may be exposed to many chemicals used in these processes.

Although the transportation equipment industry includes both very small and very large establishments, it has an unusual number of very large establishments employing thousands of employees. These very large establishments are most likely to be found in plants that produce final equipment on a mass-production basis (e.g., automobile plants, aircraft plants, tank assembly lines). However, as shown in Table C–1, 68 percent of all establishments have fewer than 20 employees.

The prosperity of the industry fluctuates with business cycles and with the value of the dollar. Employment in this industry declined between 1981 and 1982 but had recovered to the 1981 level by 1984 and had increased another 4 percent by 1985 [2].

The record contains comments from businesses which use styrene in open-molding processes to produce reinforced plastics products such as fiberglass boats, fiberglass car and truck bodies, and transportation equipment parts [Ex. 3–742, pp. 34–36; Tr. 8/3/88, pp. 5–95, 5–119]. Commenters noted that controlling employee exposures during the open molding of large components (e.g., boat hulls and decks, recreational vehicles) is made costly and difficult by the large sizes and bulky configurations of these products [Ex. 3–742, p. 48].

The open-mold process in this sector is similar to that in other reinforced plastics industries in that it involves the use of a styrene resin to make a mold, followed by the application of a fiberglass-styrene-catalyst mixture with a spray or "chopper" gun, followed by manual rolling of the recently applied surface. Workers bend over the mold to perform the layup operation, which requires rolling with a short- or long-handled roller. The roller, spray gun, and other tools used in this process all require repeated cleaning with acetone in order to operate smoothly, and the workers themselves use acetone at frequent intervals to clean the styrene resin from their skin.

The Styrene Information and Research Council (SIRC) estimates that there are 625 reinforced plastic boatmakers in this sector that produce boats under 30 feet in length, and 125 facilities that manufacture larger boats [Ex. 3–742, p. 105]. These boatmakers are estimated to employ about 32,000 production workers. However, SIRC estimates that no more than 20 percent of these employees engage in open molding or work in portions of these facilities where such molding in being done [Tr. 8/3/88, p. 5–100].

Most boat builders are small firms, and many are family-owned enterprises with only one facility. Because the purchase of a recreational boat is a discretionary expense, the industry is relatively price-sensitive. For example, Jeff Napler, president of the National Marine Manufacturers Association, stated that the price elasticity of boat sales was approximately 2, i.e., every 1-percent increase in price results in a 2-percent decline in sales [Tr. 8/3/88, pp. 5–168, 5–169]. The boat building industry is currently undergoing expansion and is enjoying relatively high profits [Tr. 8/10/88, pp. 10–144, 10–145]. Boat building is a labor-intensive industry, and firms in this sector argue that automation is not an option, since many recreational boats are custom designed [Tr. 8/10/88, pp. 10–144, 10–145].

In this SIC, the survey identified twice as many small firms (fewer than 20 production workers) as large firms. In the small firms, maintenance work is performed for the most part by production workers, although some firms use outside contractors or have a dedicated maintenance staff. Large firms divide maintenance work about equally between a dedicated maintenance staff and production workers.

The manufacturers classified in this SIC usually have two to four basic processes, with potential exposure to one to six chemicals being most common, though some firms report using up to ten chemicals. Employees are exposed to these chemicals on an intermittent short-term basis (up to 30 minutes) or continuously (up to 8 hours per day) with large firms tending to have more long-term exposures. The majority of small firms use the OSHA PELs. Over 60 percent of large firms reported using the OSHA PELs; the balance had no standards. Air monitoring data was being done for about one-half of the processes found in large plants.

The survey found that about 40 percent of the processes are totally enclosed and between 5 and 10 percent

are located outdoors. Local exhaust ventilation and respirators are used most frequently to control exposures at processes not enclosed or outdoors. In over 70 percent of the firms with chemical exposures, production workers use respirators, with a higher percentage of small firms using respirators than large firms. The combined data on exposure levels and methods of exposure control indicate that overexposures may occur at all processes in small firms and at about one-half of the processes in large firms.

Survey respondents identified the presence of 68 different substances in SIC 37. Welding fumes was estimated to occur the most frequently at a total of 3,508 processes, toluene at 3,191, and styrene at 2,541. Welding fumes were identified in machining/grinding, welding/brazing, coating/spraying, and materials manufacture/fabrication. Toluene was identified in adhesive binding, assembly, coating/spraying and cutting/sawing. Styrene was identified in injection molding, coating/spraying, sanding and assembly.

SIC 38—Measuring, Analyzing and Controlling Instruments

SIC 38 includes manufacturers of instruments used to measure, test, analyze and control. It also includes optical instruments and lenses; surveying and drafting instruments; hydrological, hydrographic, meteorological, and geophysical equipment; search, detection, navigation, and guidance systems and equipment; surgical, medical, and dental instruments, equipment, and supplies; and watches and clocks [1, p. 243].

The industries in this SIC rely heavily on research and development activities (R&D) of other industries for sales of their products. According to the U.S. Department of Commerce, increases in research and development expenditures by industry and government in 1986 caused increases in sales of scientific and industrial instruments [2]. High tech firms, which represent a large portion of SIC 38's product market, are the largest investors in research and development, where R&D expenditures are measured as a percentage of gross sales. Firms producing semiconductors, computers and related equipment, office equipment, and software, among others, were major sources of R&D funds in 1986. The pharmaceutical and chemical industries also have relied on R&D to a large extent. In addition, the decline in the price of oil, which raises profits by lowering production costs, is expected to further stimulate R&D expenditures by the chemical industry [3, p. 33–1].

Similarly, government outlays for R&D increased in 1986 by more than 9 percent in current dollars. Most of the R&D expenditures, however, were for defense-related research. In addition, the National Aeronautics and Space Administration (NASA) is expected to invest in new instrumentation for the redesign of the space shuttle and other rocket systems [3, p. 33–4].

According to the U.S. Department of Commerce, the value of shipments in 1985 ($61 billion) increased almost 26 percent since 1981 [2]. Between 1981 and 1985, SIC 38 experienced a 1 percent loss in employment [3]. Of all employees working in SIC 38, 54.4 percent were production workers [5]. In 1985, the median rate of return on assets in this SIC was 7.3 percent [6].

From 1981 to 1985, the value of shipments for SICs 383 and 384 experienced growth, rising 60 and 54.3 percent, respectively. SIC 383 comprises 8 percent of the total value of shipments in SIC 38, while SIC 384 represents 23 percent. In contrast, SIC 387 experienced a drop of 36 percent in the value of shipments, representing only 1.5 percent of the total value of shipments in SIC 38 [6].

In SIC 38, the survey identified nearly twice as many small firms (fewer than 20 production workers) as large firms. In both small and large firms, maintenance work is performed predominantly by workers specifically employed to handle maintenance duties.

The manufacturers classified in this SIC usually have two to six basic processes, with potential exposures to as many as six substances. Fourteen percent of the processes involve exposure to these chemicals or substances on a short-term basis (up to 30 minutes), with small firms tending to have shorter-term exposures. Fifty percent of the firms in SIC 38 have reported the adoption of internal monitoring standards. Of those firms with standards, the most frequently reported were OSHA PELs. Employee monitoring had been performed at 32 percent of the processes. The survey found that about 49 percent of the processes are totally enclosed and 10 percent of the processes are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed. Nearly 45 percent of the firms with chemical exposures have respirators for employee use, with a higher percentage of small firms reporting the presence of respirators than large firms. The combined data on exposure levels and methods of exposure control indicate that many plants which are estimated to incur some cost of compliance have overexposures in all processes at that plant.

Survey respondents identified the presence of 30 different substances in SIC 38. Isopropyl alcohol was estimated to occur the most frequently at a total of 2,294 processes. Isopropyl alcohol was identified in blending/mixing/formulating, adhesive binding, recovery/reprocessing/reclamation, drying/baking, packaging/bagging, extrusion, loading/offloading/receiving/handling, reaction/fermentation, boilers, coating/spraying/finishing/layup, separation, and crushing/grinding/calcining.

SIC 39—Miscellaneous Manufacturing Industries

Miscellaneous industries included in SIC 39 reflect a diverse group of producers. Most of the industries in SIC 39 produce discretionary durable consumer goods, some of which are luxury goods. Establishments that cannot be grouped together at the three-digit level are included in SIC 399. At the three-digit level, miscellaneous manufacturing industries include producers of jewelry, silverware, and plated ware (SIC 391); musical instruments (SIC 393); toys and sporting goods (SIC 394); pens, pencils, office and art supplies (SIC 394); and costume jewelry and notions (SIC 396). A sixth category, miscellaneous manufactures (SIC 399), includes producers of brooms and brushes, signs and advertising displays, burial caskets, hard surface floor coverings, and manufacturing industries "not elsewhere classified" [1, pp. 211–218].

The number of establishments and employment in SIC 39 are shown in Table C–1. Nearly three-quarters (72 percent) of these employees are production workers.

Establishments in SIC 39 are generally smaller than those in manufacturing as a whole, with higher proportions of employees concentrated in small establishments. The mean size of establishments is 11 employees, with 85 percent of establishments having fewer than 20 employees, compared with less than 65 percent for manufacturing establishments as a whole. Relatively few establishments in SIC 39 have 100 or more employees [7].

Miscellaneous manufactures (SIC 399) has the largest share (more than one-third) of the value of shipments for SIC 39 ($26.5 billion in 1985) [2, Vol. 1: 8, 22, 24]. Substantial import competition, however, poses a threat to various subsectors. Imports account for nearly 60 percent of the new supply of sporting and athletic goods and between one-

quarter and three-eighths of new supply in many other industries. The recent decline of the dollar has tended to halt or reverse import penetration [3, pp. 45–2 to 45–11; 46–10 to 46–13]; however, domestic production in SIC 39 will be affected by the trend among doll and toy manufactures to move offshore [3, pp. 45–2 to 45–11; 46–10 to 46–12].

In terms of profitability, the majority of industries in SIC 39 are more profitable than most manufacturing industries. The median 1985 rate of return on assets (8.0 percent) is the second highest median return on assets of all two-digit manufacturing industries. Median rates of return for four-digit industries within this sector range from 3.4 percent to 9.5 percent [6].

In this SIC, the survey found that small firms (fewer than 20 production workers) comprise 85 percent of the total number of firms. In the small firms, maintenance work is performed for the most part by production workers, where large firms have a dedicated maintenance staff.

The manufacturers classified in this SIC usually have two or three basic processes, with potential exposure to two chemicals. Employees in small firms are exposed to these chemicals about equally either on a short-term intermittent basis (up to 30 minutes) or continuously (up to 8 hours per day) with somewhat fewer employees exposed for periods between these two extremes. Large firms have more long-term exposures. Small firms generally either have no internal exposure standards, or use the OSHA PELs, with some using the ACGIH TLVs. About one-half of the large firms use the OSHA PELs; most of the balance indicated that they have no standards. Air monitoring data were provided for one-fifth of the processes found in small firms, and over one-third in large firms.

The survey found that about one-quarter of the processes are totally enclosed and 20 percent are located outdoors. Local exhaust ventilation is used most frequently to control exposures at processes not enclosed or outdoors. In almost one-half of the firms with chemical exposures, production workers use respirators, with a higher percentage of large firms using respirators than small firms. The combined data on exposure levels and methods of exposure control indicate that most firms will have no processes where overexposures may occur.

Survey respondents in this SIC identified gluing/hot pressing, coating/spraying/finishing/layup, and cutting/sawing/planing as the processes most frequently used. Stoddard solvent, toluene, particulates not otherwise regulated and styrene are the substances encountered most often.

The Casket Manufacturers Association of America (CMAA) [Ex. 8–78], representing firms in SIC 3995, Burial Caskets, submitted information to the docket describing the manufacturing processes and material used by facilities in this four-digit sector. According to the CMAA, the primary materials of construction of caskets are hardwood, metal, and cloth-covered board. Firms in the hardwood segment of this industry expressed concern that the proposed limit for hardwood of 1 mg/m^3 would require the installation of controls and the imposition of compliance costs. (OSHA notes that the final rule's PEL for hardwood dust is 5 mg/m^3.) The CMAA reports that there are about 20 companies that assemble hardwood caskets; two of these firms account for more than half of the total unit volume of production [Ex. 8–78]. Most firms in this segment have less than $5 million in annual sales, although larger firms have $15 million in sales annually [Ex. 8–78]. According to Robert Morris Associates' financial data for SIC 3995, after-tax profits in this sector are $7,900,000.

No comments representing firms in SIC codes other than 3995 submitted industry profile information to the docket; however, the sporting goods manufacturers (SIC 3949) submitted cost and feasibility data to OSHA, and these data are discussed in the Technological Feasibility and Costs of Compliance chapters, below.

SIC 40—Railroad Transportation

SIC 40 includes establishments that provide line-haul railroad transportation, and switching and terminal establishments. General authority for the working conditions at railroad operations is vested in the Federal Railroad Administration. For the most part, OSHA's standards apply only to off-track operations such as shops and servicing areas. The U.S. Department of Commerce estimates that in 1987, there were 23 individual Class I railroads (those with operating revenues of $88.5 million or more in 1986 dollars), which accounted for over 90 percent of the freight tonnage handled by the railroad industry [4]. The industry also includes about 480 smaller carriers, including shortlines and switching and terminal companies. The 1987 operating revenue for the railroad industry was estimated at $26.5 billion, representing a gain of 1.1 percent over 1986. Revenue ton-miles were estimated as 930 billion, which represents more than a 7 percent rate of growth [3, pp. 55–8]. Between 1980 and 1985, the industries in SIC 40 experienced a serious economic decline, as indicated by the fact that SIC 40 was the second slowest growing SIC (behind SIC 10, metal mining), and third highest in terms of employment losses behind SIC 33, primary metals and SIC 35, heavy machinery). During this period, employment declined by approximately 27 percent [3, pp. 13–14]. The median rate of return on assets in 1985 was 4.4 percent [6].

In SIC 40, establishments generally employ dedicated maintenance workers or hire an outside firm to perform maintenance functions. The establishments in this SIC generally have one or two basic processes with potential exposure to two or three substances. Over 80 percent of the processes involve exposure to these chemicals or substances on an intermittent short-term basis (up to 30 minutes). Fifty percent of the establishments in SIC 40 have reported the adoption of an internal exposure standards. Among those establishments with internal standards, most use the ACGIH TLVs.

The survey found that none of the interviewed establishments had totally enclosed processes, but 85 percent were located outdoors. Of those establishments with ventilation systems, all were locally exhausted. None of the respondents reported having respirators available in processes. The combined data on exposure levels and methods of exposure control indicate that many establishments which are estimated to incur some cost of compliance have overexposures in all processes at that establishment.

Survey respondents identified the presence of 10 different substances in SIC 40. Methyl alcohol was estimated to occur the most frequently at a total of 36 processes. Methyl alcohol was identified in maintenance activities. In addition, welding fumes occurred frequently in maintenance and welding activities.

SIC 42—Motor Freight Transportation and Warehousing

Grain elevators are classified in three different two-digit SIC codes: SIC 20, Food and Kindred Products; SIC 42, Motor Freight Transportation and Warehousing; and SIC 51, Wholesale Trade. Elevators falling within SIC 42 are those whose primary income derives from the storage of grain. Rulemaking participants who commented on the feasibility of achieving OSHA's proposed limit for grain dust in grain elevators did not designate SIC codes in their comments. The issue of grain dust exposure in grain elevators is discussed in connection with SIC 51, below. This SIC was not included in the 1988 survey.

SIC 45—Air Transportation

This SIC includes establishments that provide domestic and foreign transportation by aid and also those that operate airports and flying fields and provide terminal services. The Federal Aviation Administration (FAA), U.S. Department of Transportation, enforces rules and regulations governing the safety and health of flight and cabin crew of aircraft in flight. In general, the FAA also has jurisdiction over airline maintenance and ground/support personnel. According to the U.S. Department of Commerce, the U.S. airline industry consists of approximately 200 individual commercial air carriers operating over 4,400 aircraft and employing over 435,000 people [3]. In 1986, the industry served 418 million passengers and operated 7.4 billion freight and express cargo ton-miles. Nine major carriers account for 90 percent of all revenue passenger miles. (The U.S. Department of Commerce defines a major carrier as having annual revenues exceeding $1 billion, in 1982 dollars.) The remaining passenger revenue is shared by 16 carriers classified as nationals (each with annual revenues between $75 million and $1 billion in 1982 dollars), which account for about 12 percent, and by the regionals/commuters, which account for 4 percent. The U.S. Department of Commerce estimated the 1987 operating revenue for the airline industry as $55.6 billion, representing an annual growth rate of about 10 percent [3]. Revenue passenger miles were estimated as 435 billion, which represents a 3 perecent rate of growth [4, pp. 59–1]. In 1985, the median rate of return on assets in this sector was 4.3 percent [6].

In SIC 45, establishments generally employ dedicated maintenance workers to perform maintenance functions. The establishments in this SIC have up to five processes and as many as eight substances. Almost 60 percent of the processes involve potential exposure to these chemcials or substances on an intermittent short-term basis (up to 30 minutes). Fifty percent of the establishments in SIC 45 have reported the adoption of internal exposure standards. Among those establishments with internal standards, most use OSHA PELs.

The survey found that one third of the interviewed establishments had totally enclosed processes, but more than 65 percent were located outdoors. Of those establishments with ventilation systems, all were locally exhausted. More than 15 percent of the respondents reported having respirators for employee use. The combined data on exposure levels and methods of exposure control indicate that many establishments which are estimated to incur some cost of compliance have overexposures in all processes in the plant.

Survey respondents in SIC 45 identified 10 different substances. Ethylene glycol was estimated to occur the most frequently at 22 processes.

SIC 47—Transportation Services

SIC 47 includes establishments that furnish services related to transportation. Activities classified in SIC 47 include freight forwarding, arranging transportation for passengers and freight, renting railroad cars, inspection and weighing services; and freight car loading [1, pp. 280–281]. According to the U.S. Department of Commerce, between 1980 and 1985, SIC 47 was the third-fastest growing industry group, behind SIC 62 (Securities) and SIC 73 (Business Services) [3, pp. 13–14]. Between 1981 and 1985, SIC 47 experienced a 31 percent increase in employment. Table C–1 presents employment and establishment data for SIC 47. The median return on assets in this SIC was 7.1 percent [6].

In SIC 47, the survey identified more than eight times as many small firms (fewer than 20 production workers) as large firms. In the small firms maintenance work is predominantly performed by outside contractors. However in large firms a high percentage of maintenance work is performed by production workers.

The establishments classified in SIC 47 usually have one to four basic processes, with potential exposure to one to four substances. Ninety-four percent of the processes involve exposure to these chemicals or substances on an intermittent short-term basis (up to 30 minutes). Fifty percent of the firms in this SIC reported the adoption of an internal exposure standard. Of those large firms with exposure standards, most rely on OSHA PELs. Among small firms with exposure standards, most have adopted ACGIH TLVs. Internal monitoring had been performed at 50 percent of the processes.

The survey found that local exhaust ventilation is used most frequently to control exposures at processes not enclosed. Nearly 50 percent of the processes with chemical exposures have respirators for employee use, with a higher percentage of large firms reporting the presence of respirators than small firms. The combined data on exposure levels and methods of exposure control indicate that many establishments which are estimated to incur some cost of compliance have overexposures at all processes at that establishment.

Survey respondents identified the presence of 5 different substances in SIC 47. Exposure to gasoline was estimated to occur most frequently at a total of 206 processes. Gasoline was identified in loading/offloading/receiving/handling.

SIC 49—Electric, Gas and Sanitary Services

SIC 49 includes establishments that generate, transmit, and/or distribute electricity, gas, or steam. These establishments may be combinations of any of these services, but also may include other types of services, such as transportation, communications, refrigeration and pipelines for natural gas. Water and irrigation systems, and sanitary systems that collect and dispose of garbage, sewage, and other wastes, also are included in this SIC [1, p. 284].

In recent years the utilities covered in SIC 49 have been affected by ongoing changes in regulations regarding utility rates and competition. Some industrial customers have begun producing their own energy and utilities are now competing for customers outside their service areas. This competition has forced structural change and diversification. Utilities have been forced to upgrade their overall efficiency. With declining interest rates, regulators have been decreasing the allowed rate of return for utilities. This, too, has led to intensified pressures on competition [21, p. 56]. The Federal Energy Regulatory Commission is currently considering whether to allow utilities to open their power lines to other competing utilities. Users would be given the choice of suppliers. With the decreasing rate of return and the increasing competition, utilities have stepped up efficiency in order to offset the impending drop in their profit margins [22, p. 48].

Many of the industries in SICs 4911, 4931, 4932, and 4939 are represented by the national trade association, Edison Electric Institute (EEI). Ninety-seven percent of all customers serviced by the investor-owned segment of the industry purchase electricity from EEI members. Members generate 76 percent of the country's electricity [Ex. 3–831].

Table C–1 presents employment and establishment data for SIC 49 for 1985. Between 1981 and 1985, SIC 49 experienced a 6 percent growth in employment. In 1985, almost 80 percent of all employees were production

workers [2]. The median return on assets was 4.0 percent [6].

Within this SIC, there are seven three-digit SICs, including establishments that generate, transmit, or distribute electrical energy for sale and that operate crude petroleum and natural gas field properties; establishments that transmit and/or store natural gas for sale; establishments that provide electric or gas services in combination with other services, only if one service does not constitute 95 percent or more of revenues; establishments that distribute water for sale for domestic, commercial, and industrial use; establishments that collect and dispose of wastes conducted through a sewer system, including such treatment processes as may be provided; establishments that produce and/or distribute steam and heated or cooled air for sale; and establishments that operate water supply systems for the purpose of irrigation [1, pp. 284–286].

In SIC 49, the survey identified nearly twice as many small firms (fewer than 20 production workers) as large firms. In small firms, maintenance work is predominantly performed by production workers, although some firms also use dedicated maintenance workers or outside contractors. Large firms mainly employ workers specifically for maintenance duties.

The manufacturers classified in SIC 49 usually have one to four basic processes, with numerous firms having potential exposures to as many as six different substances. Fifty-five percent of the processes involve exposure to these chemicals or substances on an intermittent short-term basis (up to 30 minutes), with small firms tending to have more long-term exposures. Most firms in this SIC have adopted OSHA PELs as their internal standard. Of the large firms with internal exposure standards, most have adopted OSHA PELs or ACGIH TLVs. However, 52 percent of small firms reported having an internal exposure standard. Employee monitoring had been performed at 16 percent of the processes.

The survey found that about 25 percent of the processes are totally enclosed and 70 percent are located outdoors. Local exhaust and general ventilation are used frequently to control chemical exposures at processes. Nearly 45 percent of the firms with chemical exposures have respirators for employee use, with a higher percentage of large firms than small firms reporting the presence of respirators. The combined data on exposure levels and methods of exposure control indicate that many plants which are estimated to incur some cost of compliance do not have overexposures in all processes at that plant.

Survey respondents identified the presence of 47 different substances in SIC 49. Chlorine was estimated to occur most frequently at a total of 2,196 processes. Chlorine was identified in boilers, water treatment, handling spills/leaks, incineration, maintenance activities, use of chemical additives, use of disinfectants and solvents, and water purification.

SIC 50 and SIC 51—Wholesale Trade

The wholesale trade sector includes establishments engaged in the wholesale selling of merchandise to retailers; industrial, commercial, institutional, farm, or business users, or to other wholesalers or firms that act as agents or brokers in the wholesale buying or selling of merchandise. Wholesale trade is divided into trade in durable goods (SIC 50) and in nondurable goods (SIC 51). This analysis focuses only on a few of the wholesale trade industries (e.g., dealers in scrap and waste materials, SIC 5093; grain, SIC 5191; and paints, varnishes, and supplies, SIC 5198 [1, pp. 24, 250, 255–257].

Wholesale trade sales ($1,375 billion in 1985) were fairly equally divided between durable goods and nondurable goods—46 percent and 54 percent, respectively [3, p. 56–7]. Of the approximately 425,000 establishments in wholesale trade, about five-eighths were in durable goods, and three-eighths were in nondurable goods. The specific four-digit industries studied for this analysis include about 11 percent of all wholesale trade establishments [2, pp. 59, 62, 64–65].

Table C–1 shows employment data at the four-digit level. Somewhat less than 60 percent of total employment in wholesale trade is in durable goods, while a little more than 40 percent is in nondurable goods. The specific four-digit industries being analyzed here account for less than 9 percent of all employment in wholesale trade [7].

OSHA received no comments relating to SIC 50, Wholesale Trade, Durable Goods. However, in SIC 51, Wholesale Trade, Non-Durable Goods, OSHA received several comments.

A large number of SIC 51 commenters submitted data and testimony directed at OSHA's proposed 4-mg/m^3 TWA exposure limit for grain dust [see, for example, Exs. 3–47, 3–58, 3–59, 3–65, 3–110, 3–281, 3–347, 3–387, 3–496, 3–667, and 3–752]. Several of these commenters provided estimates of the number of grain elevators potentially affected by the proposed limit, which pertains only to wheat, oat, and barley dusts. (Elevators used exclusively for storage are classified in SIC 4221; those used principally for grain cleaning and preparation fall within SIC 0723; and elevators that are used primarily in wholesale marketing operations are classified in SIC 5153, Wholesale Trade, Grain and Field Beans.)

The National Grain and Feed Association (NGFA) estimates that approximately 87 percent of U.S. grain elevators handle oats, wheat, or barley, based on USDA records for 1985 that show that this percentage of government-grain-storing elevators reported handling and storing grains of these types [Ex. 3–752]; this yields an estimated total of 12,158 grain elevators that are potentially affected by the proposed grain dust limit. The NGFA estimates that approximately 49,063 full-time equivalent employees work in these elevators [Ex. 3–752].

According to the NGFA, many of the smaller grain elevators have relatively low profits:

> A January 1987 survey and analysis by [the] U.S. Department of Agriculture on cooperative grain elevators (Financial Profile of Cooperatives Handling Grain: First Handlers, $1 Million to $4.9 Million in Sales. USDA, ACS Report No. 58, January 1987) indicates the average annual profits for small elevator facilities is only $38,272. This report indicated that 24.8 percent of these facilities currently have negative profits and another 23.1 percent have profits of only $25,000 or less [Ex. 3–752, pp. 19–20].

In these SICs 19 out of 20 firms identified by the survey were small firms (fewer than 20 production workers). In the small firms, maintenance work is usually performed by production workers, but over 40 percent of all small firms use either outside contractors or have a dedicated maintenance staff. Large firms normally have a dedicated maintenance staff, but over one-quarter use production workers for maintenance and some use outside contractors.

Of those firms providing chemical or process information, the majority used from one to three chemicals in one or two processes. However, some firms reported using up to 10 chemicals. Over half of all exposures are short-term (up to 30 minutes), with the remaining exposures varying in length from 1 to 8 hours. The majority of small firms have internal exposure standards; most of these use OSHA PELs, but some use ACGIH TLVs. Almost 90 percent of large firms reported using internal exposure standards, and were about equally divided between those using OSHA PELs and those using ACGIH

TLVs. Air monitoring was being done for approximately one-fifth of the processes found in large plants.

The survey found that less than 20 percent of the processes are totally enclosed and that over 50 percent of the processes are located outdoors. In almost 40 percent of the firms with chemical exposures, production workers use respirators, with large firms reporting a higher percentage of use than small firms.

Survey respondents identified the presence of 80 chemicals in SICs 50 and 51. Grain dust was estimated to occur the most frequently at a total of 3,426 processes, including drying, packaging/repackaging, receiving and shipping, sorting and grinding.

SIC 55—Automotive Dealers and Service Stations

This industry sector includes retailers of transportation equipment for personal use (new and used automobiles) as well as recreational vehicles (boats, motor homes, and dune buggies); sellers of automobile parts and accessories; and gasoline stations. Although this industry does not include establishments whose primary business is automotive repair, it does include repair operations that are part of automobile dealerships or service stations. Only those retail outlets that earn more than 50 percent of their revenues from gasoline or lubrication oil sales are included. Many car washes and convenience stores that sell gasoline are excluded, as are traditional full-service gas stations that earn more than 50 percent of their revenues from such activities as repairs, towing, or the sale of auto accessories [1, pp. 265–266]. According to one estimate, this sector includes only 55 percent of all retail motor fuel outlets [23, pp. 6–13]. Although many employees are involved in selling, some are exposed to chemicals during painting or stripping or as a result of the indoor operation of engines or the use of solvents.

As shown in Table C–1, most establishments are relatively small (80 percent have fewer than 20 employees). Only in one sector, new and used automobile dealerships, do more than half of the establishments have more than 19 employees [10]. Even in this sector, however, 90 percent of the establishments have fewer than 100 employees [5]. Although the typical operation is relatively small, total employment is substantial because of the large number of establishments. New and used automobile dealerships account for 48 percent of total employment, gasoline service stations for 31 percent, and automobile and home supply stores for 16 percent.

Although many firms own only a single establishment, large firms own a significant portion of all establishments, which are operated as chains under leasing or franchising agreements.

The profitability of firms in SEC 55 is below the national average, with a median return on assets of 5.9 percent in 1986; however, this rate of return improved in 1986 as gasoline prices declined and new car sales increased [6].

In the survey, the analysis of SIC 55 was combined with SIC 75, automobile sales and service. The results for the two sectors combined will be reported here. In these two SICs, 19 out of 20 firms identified by the survey were small firms (fewer than 20 production workers). About two-thirds of the small firms use production workers to perform maintenance work. Over 70 percent of large firms have a dedicated maintenance staff; the remaining large firms are about equally divided between the use of outside contractors and the use of production employees for maintenance.

Of the firms reporting chemical or process use, most use from one to five chemicals in one to four processes, but 10 percent report using up to 10 chemicals. Employees are most commonly subject only to short-term exposures (up to 30 minutes) with less than one-fourth of firms reporting exposures of from 4 to 8 hours' duration. In these sectors, a majority of small firms have internal exposure standards. Those that use internal exposure standards are evenly divided between the use of OSHA PELs and the use of ACGIH TLVs. Almost 90 percent of large firms reported using internal exposure standards, usually OSHA PELs. Air monitoring data was being done for less than 10 percent of the processes found in large plants.

The survey found that approximately 10 percent of processes are totally enclosed and that less than 20 percent are located outdoors. In over 40 percent of firms with chemical exposures, production workers use respirators, with large firms having a higher percentage of use than small firms.

Survey respondents in SICs 55 and 75 identified the presence of 40 different substances. Carbon monoxide was estimated to occur the most frequently at a total of 109,093 processes, including cleaning, confined space exposure and maintenance activities. Gasoline was estimated to occur at 24,548 processes and toluene at 23,629.

SIC 72—Personal Services and SIC 73—Business Services

The personal services industry consists primarily of consumer services. SIC 721, laundry, cleaning and garment services has the highest potential for overexposure to chemicals. Other segments of SIC 72 include photographic studios (SIC 722); beauty shops, barber shops and shoe repair (SIC 723–725); and funeral service and crematories (SIC 726) [1, pp. 298–300].

As seen in Table C–1, the number of establishments in 1985 totaled 161,004. Almost all of these (96.9 percent) had fewer than 20 employees in 1985. The mean establishment size was 7 employees. The largest single segment of this industry is SIC 7231, beauty shops, which totaled 53,165 firms in 1986 [7]. Total employment (1,056,000 employees in 1985) has increased over the last several years. In 1986, the value of sales was $39.4 billion in the personal services industry, a 6.6 percent increase over 1985 [7]. The median rate of return on assets for the personal services industry was 10.5 percent in 1985 [6].

The dry cleaning industry is likely to be affected by the final rule's PEL for perchloroethylene. According to the 1982 Census of Service Industries, there were 13,049 dry cleaning plants in the U.S. that used perchloroethylene, with total employment of 89,896 workers.

The Amalgamated Clothing and Textile Workers Union (ACTWU) commented on the health risks associated with exposure to perchloroethylene. They also commented on the feasibility of reducing the exposure of perchloroethylene to substantially less than the 50 ppm proposed standard. (OSHA notes that, in the final rule, the PEL for perchloroethylene has been reduced to 25 ppm.) The International Fabricare Institute (IFI) supported the proposed revision of the PEL for perchloroethylene to 50 ppm. They stated that approximately 64 percent of the dry cleaning industry uses dry-to-dry equipment, and that over the past four years about 95 percent of all new equipment sold has been dry-to-dry equipment. The exposure to perchloroethylene is substantially greater when using transfer equipment versus dry-to-dry equipment [Ex. 8–31]. In 1982 there were 6,738 dry-to-dry machines compared to 12,929 dry-to-dry machines in 1988 [Ex. 3–671].

The business services industry (SIC 73) consists of several different sectors. Among the sectors included are mailing, reproduction, and commercial art and photography (SIC 733); building cleaning

and maintenance services (SIC 734); and miscellaneous business services (SIC 739), such as photofinishing laboratories and commercial testing laboratories [1, pp. 301–308].

The number of establishments in the business services industry in 1985 totaled 382,626. Almost all of these (90.5 percent) had fewer than 20 employees in 1985. The mean establishment size was 12 workers. Total employment (4,457,000 employees in 1985) has increased over the last several years (4,057,000 employees in 1984) [4]. In 1986, the value of sales was $198.7 billion in the business services industry, a 9.2 percent increase over 1985 [7]. The median rate of return on assets in SIC 73 was 11.1 percent in 1985 [6].

In these SICs, the survey identified over 20 times as many small firms (fewer than 20 production workers) as large firms. In the small firms, maintenance work is performed for the most part by production workers, although some firms use outside contractors and some firms employ maintenance staffs. Large firms divide maintenance work about equally between a dedicated maintenance staff, production workers, and outside contractors.

Employees are exposed to chemicals on an intermittent short term basis (up to 30 minutes) or continuously (up to 8 hours per day), with small firms tending to have more short-term and long-term exposures. Small firms generally have no internal exposure standards; when they do, the OSHA PELs are followed about one-half of the time. Over three-fourths of large firms reported using the OSHA PELs; the balance indicated that they have no standards or they rely on ACGIH TLVs. Air monitoring was being conducted at about one-tenth of the process found in large plants.

The survey found that about one-fourth of the processes are totally enclosed and less than 1 percent are located outdoors. Local exhaust ventilation and general dilution are used most frequently to control exposures at processes not enclosed or outdoors. In one-tenth of the firms with chemical exposures, production workers use respirators, with a higher percentage of large firms using respirators than small firms. The combined data on exposure levels and methods of exposure control indicate that overexposure is not occurring at any processes in small firms and at less than one percent of the processes in large firms.

Survey respondents in this SIC identified eight major processes: permanents, dry cleaning, manicure/pedicure, coloring/dyeing, embalming, washing, exterminating, and photofinishing. Chemicals that were present in these processes included: acetic acid, acetone, calcium hydroxide, chlorine, chlorpyrifos, diazinon, ethylene glycol, glutaraldehyde, iron oxide, isopropyl alcohol, methyl alcohol, methyl chloroform, perchloroethylene, naphtha, sodium hydroxide, toluene, trichloroethylene, xylene, and titanium dioxide.

SIC 75—Automotive Repair, Services, and Garages

This sector includes establishments that provide automotive repair, rental, leasing, and parking services to the general public, but excludes gasoline stations (SIC 55) and repair shops that are part of automobile dealerships or that service commercial fleets [1, p. 309]. Employees may be exposed to engine emissions in parking garages or repair shops, to a variety of chemical solvents (particularly in painting and stripping), and to dust from body work.

Eighty-five percent of the establishments are automotive repair shops, which is the sector most likely to have significant chemical exposure, and they employ 61 percent of all industry workers [10]. As shown in Table C–1, SIC 75 is dominated by businesses employing fewer than 20 workers (97 percent), with a median return on assets of 9.2 percent in 1985. The profitability of automotive repair and service firms is high, although it varies by size and industry sector. Small firms (under $100,000 in assets) had returns of 18.3 percent in 1985, while large businesses (over $1,000,000) had returns of 3.9 percent. Paint shops (SIC 7535) were the most profitable type of operation, while parking lots (SIC 7523) and parking structures (SIC 7525) registered significantly lower rates of return [6].

No rulemaking participants submitted comments on this sector, and the results of the survey are reported under SIC 55.

SIC 76—Miscellaneous Repair

This industry group includes a wide variety of repair services, differentiated by object repaired and processes used. Industries of particular concern include reupholstery and furniture repair (SIC 7641) and welding (SIC 7692) [1, pp. 312–314]. Reupholstery and furniture repair workers may be exposed to wood dust during wood working, and to solvents; welders may be exposed to fumes.

Nineteen percent of the 56,000 industry establishments in SIC 76 are in SIC 7641 and SIC 7692. These two industries account for approximately 14 percent of all SIC 76 employment [7, pp. 81–82].

The industry is made up almost entirely of very small firms, and the sector has extremely low concentration. Mean business size is 5.5 employees: more than 95 percent of all establishments have fewer than 20 employees, and 65 percent of all workers are employed by establishments of this size. Only 0.2 percent of all miscellaneous repair establishments (with about 6 percent of total employment) have 100 or more employees, and only 17 establishments have 250 or more. The four-digit industries of concern are even more completely dominated by small establishments, with a mean size of 4.8 employees in SIC 7641 and 3.4 employees in SIC 7692 [7, pp. 81–82].

Despite a slight decline in 1981 and 1982, employment in SIC 76 has grown fairly steadily, increasing by 23 percent between 1979 and 1984 and by 7 percent between 1984 and 1986.

Miscellaneous repair firms have high profit rates. The median 1985 rate of return on assets in SIC 76 was 10.0 percent. This rate of return was higher than that of any two-digit manufacturing industry. The median rates of return on assets in SIC 7641 and SIC 7692 are over 11 percent [6].

In this SIC, over 99 of 100 firms identified by the survey were small firms (fewer than 20 production workers). In the small firms, maintenance work is normally performed by production workers, with some use of dedicated maintenance workers or use of outside contractors. In the large firms, maintenance work is performed by either dedicated maintenance staff, production employees, or outside contractors, with a dedicated maintenance staff being the most common.

Most firms reporting process or chemical use report using one to four chemicals in one to three processes. For small firms, short-term exposures (up to 30 minutes) are the most common, with the remaining exposures ranging from 1 to 8 hours in length. In large firms, most exposures are for longer than one hour. Over three-quarters of large firms in this sector report using internal exposure standards; over 55 percent reported using OSHA PELs. A majority of small firms reported having internal exposure standards; more use OSHA PELs than use ACGIH TLVs. Air monitoring data had been collected for 26 percent of the processes found in large plants.

The survey found that just over one-quarter of the processes are totally enclosed and that one-quarter are located outdoors. In over 50 percent of the firms with chemical exposures, production workers use respirators;

large firms use respirators more commonly than do small firms.

Welding fumes were identified most frequently by respondents in SIC 76 in some 4,040 welding and brazing processes.

No rulemaking participants submitted comments on this sector.

SIC 80—Health Services

The health services industry encompasses a broad range of medical, surgical, and other health services, both public and commercially owned. These services are provided by a variety of practitioners (e.g., physicians, dentists, osteopathic physicians, chiropractors, optometrists) at a variety of facilities (e.g., hospitals, nursing facilities, outpatient care facilities, medical laboratories) [1, pp. 321–323].

Total expenditures on health care and medical services ($425 billion in 1985) are very large, with 40 percent of this amount going to hospital care and 20 percent to physicians' services. Expenditures on nursing home care, drugs and medical sundries, and dentists' services each accounted for 6 to 8 percent of all health and medical services expenditures [3, p. 54–1].

Data on health care establishments are shown in Table C–1. Although the number of health service establishments (313,000) is very large, 85 percent of these are offices of licensed practitioners. No other three-digit sector within the health services industry accounts for more than 4 percent of health service establishments.

Total health services employment is very large (6.3 million), with hospitals accounting for almost half (i.e., 48 percent) of this workforce. Because of their large numbers, practitioners' offices are next in percentage of workforce employed (24 percent), followed by nursing and personal care homes (18 percent). Mean establishment sizes range from six or fewer employees in practitioners' offices to 250 or more employees in hospitals. The overall mean size of establishments in this industry is 20 employees, with more than 91 percent of these establishments having fewer than 20 employees, and approximately 22 percent of all SIC 80 employees working in establishments of this size. SIC 80 facilities with more than 250 employees employ more than 50 percent of the workforce in this sector [7; 5].

The health and medical services industry has been expanding rapidly for more than a decade. A variety of factors have caused this increase, including the expansion of the elderly population and improved treatment for many illnesses. In addition, between 1985 and 1986, the price for most medical services rose between 6 and 9 percent, compared with 1.5 percent increase in consumer prices. The implementation of Medicare's prospective payment system is also causing major changes in the health care industry [3, pp. 54–1, 2].

Hospital care costs have been a major target of cost-cutting measures, resulting in a decline in hospital admissions, a shortening of hospital stays, and substantial industry restructuring, including increased mergers and acquisitions by large chains, vertical integration, diversification of services offered, expanded professional peer review, and more businesslike operations. Major investor-owned nursing home chains also have experienced rapid expansion and acquisition [3, pp. 54–1, 2].

For SIC 80 as a whole, the growth rate in expenditures averaged 12.6 percent per year from 1979 to 1984 and more than 9 percent for the next 3 years [3, p. 54–1]. Employment grew by 31 percent between 1979 and 1986, rising by 2 to 5 percent in each year [5]. The growth picture is fairly consistent across three-digit industries, although expenditures on "other professional services" have shown the most rapid growth of any health service (16.3 percent annually from 1979 to 1984). Expansion has been especially rapid in health maintenance organizations and home health care, both of which have the potential for reducing health costs and substituting, to some degree, for hospital care [3, pp. 54–1 to 54–4].

The median rate of return on assets in health services (5.0 percent in 1985) is relatively low compared with that in manufacturing industries, and hospitals have somewhat lower median rates of return than is the case for health services as a whole. Several "Offices" industries, on the other hand, have median rates of return higher than 13 percent. Medical and dental laboratories have median rates of return that are above the median for two-digit manufacturing industries [6].

In this SIC, nine out of ten firms identified by the survey were small firms (fewer than 20 production workers). The majority of small firms employ outside contractors for maintenance work. The majority of large firms use dedicated maintenance workers.

Of those firms reporting chemical or process use, over half report using one or two chemicals in one or two processes. In this SIC, most employee exposures are for less than 30 minutes in length; one-fourth of large firms report employee exposures of from 4 to 8 hours in duration. Approximately half of all small firms have no internal exposure standards and approximately half report using OSHA PELs. Over 60 percent of large firms report using OSHA PELs, with only a limited number reporting use of ACGIH TLVs. Large firms provided air monitoring data for slightly over one-third of all processes found in their plants.

The survey found that almost 50 percent of all processes are totally enclosed and that almost none are outdoors. One-quarter of all firms report using respirators, with large firms having somewhat greater respirator use than small firms.

Survey respondent identified the presence of 73 different substances in SIC 80. Isopropyl alcohol was estimated to occur the most frequently at a total of 38,575 processes, including administration of anesthesia, laboratory procedures, making of dental appliances and sterilization. Aryl and inorganic compounds of mercury were estimated to occur in a total of 25,197 processes, including preparation of dental amalgams and X-ray film processing.

No testimony or comments submitted to the docket pertained to the health industry.

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TABLE C - 1

Industries With Potential Hazardous Exposures, Number of Establishments and Employment (1985)

SIC	Description	Establishments[a] Total Number	Establishments[a] Percent Large[b]	Establishments[a] Percent Small[c]	Employment[d] Total (1,000)	Employment[d] Production Workers (1,000)
20	FOOD AND KINDRED PRODUCTS	29,043	37.14	62.86	1,603	1,118
21	TOBACCO MANUFACTURES	216	46.76	53.24	64	48
22	TEXTILE MILL PRODUCTS	11,023	39.40	60.60	702	607
23	APPAREL PRODUCTS	30,032	33.33	66.67	1,121	945
24	LUMBER & WOOD PRODUCTS, EXCEPT FURNITURE	36,710	19.73	80.27	697	584
242	SAWMILLS, PLANING MILLS	6,390	68.95	31.05	195	172
243	MILLWORK, VENEER & PLYWOOD	13,921	17.87	82.13	288	190
244	WOOD CONTAINERS	2,701	78.08	21.92	41	35
245	BUILDING & MOBILE HOMES	1,618	40.05	59.95	72	56
249	MISCELLANEOUS WOOD PRODUCTS	5,666	18.73	81.27	77	64
25	FURNITURE AND FIXTURES	16,791	27.16	72.84	494	394
26	PAPER AND ALLIED PRODUCTS	8,750	53.86	46.14	678	512
27	PRINTING, PUBLISHING & ALLIED INDUSTRIES	84,279	15.87	84.13	1,428	789
28	CHEMICAL AND ALLIED PRODUCTS	20,823	32.59	67.41	1,044	578
281	INDUSTRIAL INORGANIC CHEMICALS	3,024	35.42	64.58	142	72
282	PLASTICS & SYNTHETICS	1,666	51.50	48.50	172	114
283	DRUGS	2,454	37.82	62.18	206	95
284	SOAP, CLEANERS, & COSMETICS	4,498	24.59	75.41	148	94
285	PAINTS, VARNISHES, LACQUERS	1,880	36.54	63.46	64	31
286	INDUSTRIAL ORGANIC CHEMICALS	1,528	34.88	65.12	160	82
287	AGRICULTURAL CHEMICALS	1,843	23.77	76.23	59	37
289	MISCELLANEOUS CHEMICAL PRODUCTS	3,930	29.64	70.36	94	54
29	PETROLEUM REFINING & RELATED INDUSTRIES	3,334	28.40	71.60	179	109
291	PETROLEUM REFINING	1,332	33.18	66.82	141	82
295	PAVING & ROOFING MATERIALS	1,222	23.81	76.19	26	20
299	MISCELLANEOUS PETROLEUM & COAL PRODUCTS	780	27.44	72.56	-	-
30	RUBBER & PLASTICS PRODUCTS	18,002	38.85	61.15	786	607
307	MISCELLANEOUS PLASTIC PRODUCTS	14,638	39.62	60.38	550	435

[a] Dun and Bradstreet

[b] 20 or more employees

[c] Fewer than 20 employees

[d] Labstat, U.S. Department of Labor (Database)

TABLE C - 1

Industries With Potential Hazardous Exposures,
Number of Establishments and Employment
(1985)
(continued)

SIC	Description	Establishments[a] Total Number	Establishments[a] Percent Large[b]	Establishments[a] Percent Small[c]	Employment[d] Total (1,000)	Employment[d] Production Workers (1,000)
31	LEATHER AND LEATHER PRODUCTS	3,940	29.85	70.15	165	137
311	LEATHER TANNING & FINISHING	480	35.42	64.58	15	12
32	STONE, CLAY, GLASS, & CONCRETE PRODUCTS	21,054	26.26	73.74	588	451
33	PRIMARY METAL INDUSTRIES	10,101	44.75	55.25	808	612
34	FABRICATED METAL PRODUCTS	46,322	32.96	67.04	1,465	1,084
35	MACHINERY, EXCEPT ELECTRICAL	77,748	22.90	77.10	2,174	1,307
36	ELECTRICAL & ELECTRONIC MACHINERY, EQUIPMENT & SUPPLIES	28,478	37.64	62.36	2,197	1,300
37	TRANSPORTATION EQUIPMENT	16,132	31.58	68.42	1,980	1,257
38	INSTRUMENTS	16,814	29.42	70.58	720	391
39	MISCELLANEOUS MANUFACTURING INDUSTRIES	32,212	15.82	84.18	367	264
40	RAILROAD TRANSPORTATION	2,645	27.30	72.70	359	-
45	TRANSPORTATION BY AIR	11,832	19.46	80.54	522	-
47	TRANSPORTATION SERVICES	35,626	7.56	92.44	276	-
49	ELECTRICAL GAS, & SANITARY SERVICES	21,115	25.71	74.29	915	729
5093	SCRAP & WASTE MATERIALS	7,556	12.61	87.39	92	-
5153	GRAIN	7,523	5.84	94.16	-	-
5161	CHEMICALS & ALLIED PRODUCTS	13,045	8.51	91.49	-	-
5191	FARM SUPPLIES	20,392	4.55	95.45	151	-
5198	PAINTS, VARNISHES, & SUPPLIES	4,033	6.89	93.11	-	-
55	AUTO DEALERS & SERVICE STATIONS	189,214	9.77	90.23	1,890	1,886
72	PERSONAL SERVICES	161,004	3.13	96.87	1,056	-
73	BUSINESS SERVICES	382,626	9.46	90.54	4,457	3,863
75	AUTO REPAIR, SERVICES, & GARAGES	149,260	2.64	97.36	731	614
7641	REUPHOLSTERY & FURNITURE REPAIR	10,655	0.92	99.08	-	-
7692	WELDING REPAIR	9,413	2.21	97.79	-	-
80	HEALTH SERVICES	313,076	8.71	91.29	6,299	5,607

Source: U. S. Department of Labor Occupational Safety and Health Administration, Office of Regulatory Analysis.

[a] Dun and Bradstreet

[b] Labstat, U.S. Department of Labor (Database)

[c] 20 or more employees

[d] Fewer than 20 employees

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D. Employee Exposures and Benefits

Employee exposures to the substances included in the scope of this rulemaking are associated with a wide variety of acute and chronic conditions and illnesses. These include sensory irritation, narcosis, organ system dysfunctionn, chronic respiratory disease, neurological impairment, allergic sensitization, and cancer. Since OSHA's adoption of existing Federal and consensus standard limits in 1971, toxicologic evidence has become available that shows that adverse health effects can occur as a consequence of exposure to many of the substances listed in OSHA's Z tables, and that such health effects occur even when exposures are maintained at the current Z-table limits. In addition, many substances that have come into widespread use or been introduced since 1971 have been shown to be potentially hazardous in the workplace environment. OSHA thus believes that reducing worker exposures to such substances by lowering existing exposure limits or by adding limits for previously unregulated substances will result in a significantly reduced risk of illness to workers.

This chapter describes both the methodology used to identify workers potentially exposed to the hazardous substances included in this rulmaking and the expected benefits to those workers resulting from lowering permissible exposure levels. An important existing data base for identifying employees potentially exposed to hazardous substances was OSHA's Integrated Management Information System (IMIS). The IMIS data were used to project expected benefits resulting from lowering permissible exposure levels of the substances being regulated.

The IMIS data base, however, was incomplete, and its information on some hazardous chemicals may be out of date. For example, IMIS contained research information on about 160 substances among the approximately 430 substances covered by the final rule. While the IMIS data base contains the results for over 100,000 samples of substances currently regulated by OSHA , no plant-specific information was available for about 200 of the substances included in this rulemaking but currently not being regulated by OSHA. To both correct this data gap and obtain additional information on employee exposures, a nationwide survey was begun in January 1988, which was designed to collect worker exposure data from about 5,700 establishments nationwide in industries that are believed to be affected by this rulemaking. The survey results include industry-sector-specific data on the extent of employee exposures to hazardous materials and, unlike the IMIS data, provide specific information on the industrial processes in which these substances are used. While the sample survey confirmed potential exposures for many of the 160 chemicals in the IMIS data base, it identified potential exposure problems for about 62 additional substances subject to this rulemaking. Thus, the benefit estimates in this section are based upon employee exposures to 212 of the 428 substances being regulated.

To assess the benefits of revising OSHA's Z tables, OSHA relied on both the survey and IMIS data. The IMIS data were combined with raw survey data to estimate the extent to which employees are currently exposed to substances included in this rulemaking. From this analysis, OSHA estimated the reduction in illness cases and disease-related fatalities associated with reducing exposure limits for these substances.

Description of Data Sources Used

To assess the quantitative benefits associated with this rulemaking, the following data were used:

- The exposure limits for substances included in the rulemaking;
- Employee exposure data for these substances;
- Employment data by four-digit SIC code for the base year 1985;
- Annual illness and lost workday rates for the base year 1985; and
- Health effects information on the substances included in the rulemaking.

Employee exposure data for about 160 substances were obtained from OSHA's Integrated Management Information System (IMIS). This data base contains exposure measurements obtained by OSHA compliance officers during the conduct of thousands of health inspections. For each facility inspected, the IMIS file includes information on the number of employees at the facility, results of employee air monitoring for specific substances, and the number of employees potentially exposed to each substance monitored. To perform the benefits assessment, a summary IMIS file was created that contained the following information:

- A list of substances for which personal 8-hour TWA samples were taken, by four-digit SIC and facility inspected;
- The number of workers potentially exposed to each substance monitored, by four-digit SIC and facility;
- The number of employees at each facility inspected;
- The total number of personal 8-hour TWA samples obtained for each substance, by four-digit SIC and facility; and

• The number of samples taken at each facility that showed concentrations exceeding OSHA's limits.

Only those substances for which OSHA is reducing an existing 8-hour TWA limit or adding a new 8-hour TWA limit were included in the analysis. A total of approximately 37,500 personal air sample results for about 160 substances were appropriate for use in this analysis. This analysis does not estimate the benefits associated with reducing current ceiling limits or adding new short-term exposure limits (STELs), either because the data obtained from the IMIS did not include information on sample duration for ceiling or peak measurements, or because OSHA was not able to relate the IMIS data on ceiling or peak measurements to the final short-term or ceiling limits.

In addition to relying on the IMIS exposure data, OSHA completed a major telephone interview survey of about 5,700 workplaces that are potentially affected by the revision of OSHA's Z tables. Data from this survey provide information on substances that are used in a variety of industrial processes at the facilities surveyed, on the number of workers involved in those processes, and on whether personal exposure measurements taken at the processes exceeded OSHA, ACGIH, or NIOSH limits.

Employment data by four-digit SIC code were obtained from three data sources. For each four-digit SIC represented in the IMIS file, OSHA first relied on 1985 data from the BLS LABSTAT data base [1]. Where data were unavailable from this source at the four-digit SIC level, OSHA relied on Dun & Bradstreet's *Market Identifiers* file for 1985 [2]. Data from 1985 *County Business Patterns* [3] were used to obtain employment data for four-digit SIC groups not represented in either the LABSTAT or the Dun & Bradstreet file.

Data on illness and lost workday rates were obtained from the 1985 LABSTAT file for all industries (at the three- and four-digit level) represented in the IMIS file. These data included rates per 100 employees for total illness cases, lost-workday illness cases, and total number of lost workdays.

Estimates of the Number of Potentially Exposed Employees

Estimates of the number of employees potentially exposed to the substances included in this analysis were derived from the IMIS data, OSHA's survey data, and employment data bases. To conduct the analysis, OSHA used the IMIS and survey data separately to derive independent estimates of the number of workers potentially exposed and the number of workers exposed above the limits for each substance. The estimates derived from these two data sources were then combined to yield an overall assessment of the extent of employee exposure, by four digit SIC, to substances included in this rulemaking. The following sections described how each of the data bases was used to develop estimates of employee exposures, and how these estimates were then combined.

Estimates Derived From OSHA's IMIS Data Base. For each facility inspected, the IMIS contained information on the number of employees at the facility and the number of employees observed to be potentially exposed to each substance for which personal air samples were collected. For each substance sampled within an industry (at the four-digit level), the estimated number of employees potentially exposed to that substance in the industry was determined by the following formula:

$$\frac{\sum P_f}{\sum E_f} * W = P$$

where

P_f = number of employees observed to be potentially exposed to the substance at a facility;

E_f = total number of employees at the facility;

W = number of production workers in the industry in 1985; and

P = estimated number of employees potentially exposed to the substance in the industry.

The estimated number of workers currently exposed above the limits for each substance was calculated using the following formula:

$$\frac{\sum S_f}{\sum T_f} * P = Z$$

where

S_f = number of samples that exceeded the limit for the substance at all facilities in an industry sector;

T_f = total number of personal samples taken for the substance at all facilities in the industry sector;

P = estimated number of employees potentially exposed to the substance in the industry sector; and

Z = estimated number of workers in the industry sector currently exposed above the limits for the substance.

Estimates Derived From OSHA's Survey Data. Facilities participating in OSHA's telephone survey provided the following information that was useful for estimating the extent of employee exposures to chemical substances:

• The facility's four-digit SIC code;

• The total number of production employees at the facility;

• The number of employees involved in each process used at the facility;

• The substances used or present in each process;

• The exposure limits used as internal targets or goals at the facility (i.e., OSHA's current limits, ACGIH limits, NIOSH limits, or "Other" limits such as those from material safety data sheets or insurance carriers); and

• Whether employee exposures exceeded the targeted limits for each process/chemical combination present at the facility.

To estimate the number of employees potentially exposed to a given substance in a four-digit SIC industry group, OSHA assumed that all employees who are involved with processes in which the substance is used or present are potentially exposed. Thus, the formula for estimating the number of employees who are potentially exposed to a substance in a given industry sector is

$$\frac{\sum X_f}{\sum T_f} * W = P$$

where

X_f = number of employees at the facility who are involved in processes using a given substance;

T_f = total production workforce at the facility;

W = the number of production workers in the industry sector in 1985; and

P = estimated number of employees potentially exposed to the substance in the industry sector.

To estimate the number of employees currently exposed above the final limits, OSHA relied on survey responses that indicated whether exposure measurements associated with a process exceeded the facility's internal exposure limits. OSHA interpreted the survey responses as follows.

• OSHA assumed that none of the potentially exposed employees are currently exposed above the limit for any substance associated with a process if

(1) The revised limit is an ACGIH TLV and respondents indicated that

exposure measurements did not exceed ACGIH, NIOSH, or some "other" set of limits, or

(2) The revised limit is a NIOSH REL and respondents indicated that exposure measurements did not exceed NIOSH limits.

• OSHA assumed that all of the potentially exposed employees are currently exposed above the limits for all substances associated with a process if

(1) The revised limit is an ACGIH TLV and respondents indicated that exposure measurements did exceed OSHA, ACGIH, or some "other" set of limits, or

(2) The revised limit is a NIOSH REL and respondents indicated that exposure measurements did exceed OSHA, ACGIH, NIOSH, or some "other" set of limits.

The number of overexposed workers were then summed for each substance across all facilities that responded to the survey in the four-digit SIC industry group. In instances where the survey data yielded no information on whether employees are or are not exposed above the final rule limit for a substance, and no exposure data were available from IMIS on that substance, the number of workers exposed above the final rule limit for that substance is unknown (this is indicated in Supplement 2 by a blank space). Since it is likely that in some of these cases there are employees exposed above the final rule limits, OSHA believes that it has not necessarily accounted for all employees who are exposed above the final rule limits for the 212 substances included in this analysis. Thus, OSHA believes that the number of overexposed employees may be understated.

Since the publication of the PRIA, OSHA has identified the chemical composition of several generic and trade-name substances noted as being in use by survey respondents. The estimates of potential benefits presented in this final RIA include employees exposures to substances contained in these generic and trade-name products.

Approach for Combining Estimates Derived from the IMIS Data and Survey Data. To obtain an overall estimate of the extent of employee exposures to substances used in each four-digit SIC industry group, OSHA combined the estimates derived separately from the IMIS and survey data. Table D–1 illustrates how these estimates were combined to yield an overall estimate of the extent of employe exposures in SIC 2851. Where estimates for a given substance could be derived from one data set but not the other, the combined assessment uses the available estimates without adjustment. Where estimates could be derived from both data sets for the same substance, the combined assessment is based on the average of the available estimates; this approach has the effect of giving equal weight to estimates derived from either the IMIS or survey data.

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Table D-1. Analysis of Employee Exposures in SIC 2851 Derived From IMIS Data, Survey Data, and Both IMIS and Survey Data Combined

SIC 2851 Paints and Allied Products	Assessment from IMIS		Assessment from Survey		Combined Assessment	
Name	Workers Potentially Exposed	Workers Above Limits	Workers Potentially Exposed	Workers Above Limits	Workers Potentially Exposed	Workers Above Limits
ACETONE	2,875	0	63,553	0	33,214	0
ALPHA-ALUMINA	1,286	0			1,286	0
AMMONIA			14,041	0	14,041	0
BUTOXYETHANOL	2,121	0			2,121	0
N-BUTYL ACETATE			70,204	0	70,204	0
BUTYL ACRYLATE	13,302	0	61,336	0	37,319	0
N-BUTYL ALCOHOL			44,339	14,189	44,339	14,189
N-BUTYL GLYCIDYL ETHER	28,030	0			28,030	0
CARBON MONOXIDE	672	672			672	672
CARBON TETRACHLORIDE	5,912	0			5,912	0
COBALT AS CO	4,604	0			4,604	0
CYCLOHEXANONE	6,821	0			6,821	0
DIISOBUTYL KETONE	4,434	0			4,434	0
ETHYL ACRYLATE			73,899	0	73,899	0
ETHYLENE GLYCOL			46,556	17,691	46,556	17,691
ETHYLENE GLYCOL DINITRATE			70,204	0	70,204	0
FURFURAL	2,956	0			2,956	0
HEPTANE	5,454	0			5,454	0
HEXAFLUOROACETONE	41,383	41,383			41,383	41,383
HEXANE	4,678	0			4,678	0
2-HEXANONE	6,547	127	52,468	28,858	29,508	14,792
HEXONE	7,131	319			7,131	319
ISOBUTYL ALCOHOL	10,996	0			10,996	0
ISOPHORONE			42,122	16,007	42,122	16,007
ISOPROPYL ALCOHOL			28,082	0	28,082	0
KAOLIN, TOTAL DUST			69,465	0	69,465	0
MAGNESIUM OXIDE FUME, AS MG	10,560	0			10,560	0
METHYL CHLOROFORM (1,1,1-TRICHLOROETHANE)			73,899	0	73,899	0
METHYL ETHYL KETONE PEROXIDE			25,126	0	25,126	0
MOLYBDENUM, INSOLUBLE COMPOUNDS AS MO	11,853	0			11,853	0
PERCHLOROETHYLENE	1,973	0			1,973	0
PETROLEUM DISTILLATES, RUBBER SOLVENT	7,885	0	68,726	0	38,306	
PHTHALIC ANHYDRIDE	4,427	0			4,427	0

Table D-1. Analysis of Employee Exposures in SIC 2851 Derived From IMIS Data, Survey Data, and Both IMIS and Survey Data Combined (continued)

SIC 2851 Paints and Allied Products	Assessment from IMIS		Assessment from Survey		Combined Assessment	
Name	Workers Potentially Exposed	Workers Above Limits	Workers Potentially Exposed	Workers Above Limits	Workers Potentially Exposed	Workers Above Limits
PROPYL ALCOHOL			26,604	0	26,604	0
SODIUM HYDROXIDE			29,560	0	29,560	0
STODDARD SOLVENT	6,961	194	42,861	18,430	24,911	9,312
STYRENE	1,508	0	31,038	13,967	16,273	6,983
TALC (NON-ASBESTIFORM)			5,912	0	5,912	0
TIN METAL AND OXIDE	1,286	0			1,286	0
TITANIUM DIOXIDE	2,668	0	29,560	29,560	16,114	14,780
TOLUENE	7,538	187	25,865	0	16,701	94
TOLUENE-2,4-DIISOCYANATE			17,736	0	17,736	0
TRIBUTYL PHOSPHATE	244	0			244	0
TRICHLOROETHYLENE	3,695	0			3,695	0
TRIETHYLAMINE	244	0			244	0
TRIMELLITIC ANHYDRIDE	1,626	813	28,082	0	14,854	407
TRIMETHYL BENZENE	8,099	0			8,099	0
VINYL ACETATE	13,302	0	30,299	0	21,800	0
VM & P NAPHTHA	25,909	0			25,909	0
WOOD DUST	88,679	73,896			88,679	73,896
XYLENE (O,M,P-ISOMERS)			20,692	0	20,692	0
ZINC OXIDE (FUME)			57,641	0	57,641	0

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Estimates of both the number of employees potentially exposed and the number exposed above the limits are presented, by substance and four-digit SIC code, in Supplement 2. This supplement also identifies, by four-digit SIC code, substances that are judged to present potential exposure problems but for which no IMIS or survey data were available.

Aggregate estimates of the number of employees potentially exposed or exposed above the final limits to any substance considered in the analysis are presented by two-digit SIC code in Table D–2. Because an employee may be exposed to more than one substance in a given industry, aggregate estimates of the size of the exposed population are presented as minimum and maximum estimates. Maximum estimates of the size of the exposed population assume that no employee is exposed to more than one substance; minimum estimates assume the greatest possible extent of multiple-chemical exposure. For example, if 200 employees are estimated to be exposed to acetone and 300 employees are estimated to be exposed to toluene in a given industry, a minimum of 300 employees is estimated to be exposed to both substances in the industry, and maximum of 500 employees is estimated to be exposed to either substance in the industry.

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Table D-2

TABLE D-2. Number of Workers Exposed and Number of Workers for Whom Risk is Reduced, by 2-Digit SIC

SIC Code	Number of Production Workers	No. Workers Potentially Exposed, Minimum Estimate	No. Workers Potentially Exposed, Maximum Estimate	No Workers Exposed Above Limits, Minimum Estimate	No. Workers Exposed Above Limits, Maximum Estimate	No. Workers With Reduced Risk, (80% Risk Reduction)	No. Workers With Reduced Risk, (90% Risk Reduction)	No. Workers With Reduced Risk, (95% Risk Reduction)
20	978,126	130,431	163,211	85,097	91,363	70,584	79,407	83,819
22	461,701	147,121	178,071	12,501	12,684	10,074	11,333	11,963
23	581,733	83,337	148,661	12,364	13,860	10,490	11,801	12,456
24	571,195	325,616	410,618	102,189	133,484	94,269	106,053	111,945
25	497,030	189,875	435,912	68,961	88,742	63,081	70,966	74,909
26	667,085	337,138	529,422	64,957	82,604	59,024	66,402	70,091
27	1,255,639	1,088,136	1,194,491	117,056	129,403	98,584	110,907	117,068
28	867,659	820,699	862,707	336,379	457,433	317,525	357,215	377,061
29	178,202	177,071	178,202	135,036	135,036	108,029	121,532	128,284
30	817,652	608,461	810,617	152,668	528,822	272,596	306,671	323,708
31	165,086	47,401	96,642	7,182	8,967	6,460	7,267	7,671
32	581,740	279,657	430,016	32,404	45,923	31,331	35,247	37,205
33	754,180	382,962	688,325	161,573	280,691	176,906	199,019	210,075
34	1,197,681	445,143	1,005,075	125,015	191,870	126,754	142,598	150,520
35	1,513,419	705,075	1,213,624	122,931	159,542	112,989	127,113	134,175
36	1,421,373	764,578	1,027,410	206,952	251,456	183,363	206,284	217,744
37	1,270,731	703,707	968,832	78,976	161,816	96,317	108,356	114,376
38	635,519	387,077	477,259	25,701	25,943	20,658	23,240	24,531
39	396,813	224,118	292,568	54,683	95,354	60,015	67,517	71,268

Table D-2 continued

TABLE D-2. Number of Workers Exposed and Number of Workers for Whom Risk is Reduced, by 2-Digit SIC (Continued)

SIC Code	Number of Production Workers	No. Workers Potentially Exposed, Minimum Estimate	No. Workers Potentially Exposed, Maximum Estimate	No Workers Exposed Above Limits, Minimum Estimate	No. Workers Exposed Above Limits, Maximum Estimate	No. Workers With Reduced Risk, (80% Risk Reduction)	No. Workers With Reduced Risk, (90% Risk Reduction)	No. Workers With Reduced Risk, (95% Risk Reduction)
40	191,556	174,308	177,545	1,953	1,953	1,562	1,758	1,855
45	400,571	127,357	148,199	0	0	0	0	0
47	94,421	27,723	27,926	326	326	261	293	310
49	788,744	764,824	774,685	394,767	394,767	315,814	355,290	375,029
50	2,687,033	466,280	838,602	147,896	157,612	122,203	137,479	145,116
51	998,179	580,637	693,822	236,986	245,720	192,882	216,993	229,048
52	1,501,969	1,024,565	1,501,969	624,493	818,599	577,237	649,391	685,469
72	741,954	587,265	646,222	72,941	77,299	60,096	67,608	71,364
73	3,872,633	1,869,333	2,164,155	349,261	511,836	344,439	387,494	409,021
75	621,563	457,031	592,454	53,966	55,348	43,726	49,191	51,924
76	331,435	230,403	246,725	71,846	121,570	77,366	87,037	91,873
80	6,193,016	3,552,114	6,008,369	28,465	43,369	28,734	32,325	34,121
	33,235,635	17,709,443	24,932,336	3,885,525	5,322,892	3,683,369	4,143,787	4,373,999

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Employees exposed above the final limits are considered to be "at risk" of adverse health effects. It should be noted that this presentation shows risk reduction in employee-equivalent terms; while all (100 percent) of the workers currently exposed above the new limits would benefit from reduced risk, the new lower limits would not eliminate all chemical exposure risk. An estimated five, ten, or twenty percent residual risk equivalent would remain at the new lower limits. Although not quantified, all employees currently exposed to hazardous substances at or below the recommended new levels would experience this residual risk. To obtain an approximation of risk reduction at the revised exposure levels, OSHA estimated that 95, 90, or 80 percent of the workers currently exposed above the limits (i.e., the midpoint between the minimum and maximum estimates) will benefits from reduced risk after their exposures are lowered to or below the final limits. The results of this analysis are also presented by two-digit SIC codes in Table D–2.

The American Iron and Steel Institute (AISI) [Ex. 72] objected to the approach used by OSHA to obtain the combined assessment. The AISI illustrated this point with the following example:

> [T]he approach used by OSHA in obtaining [exposure estimates] . . . is not logical. If exposures to a substance are indicated in both the IMIS and survey results, the two numbers are averaged. If only one set reports exposures, then that set is used independently. Under this approach, the combined assessment for steelworkers exposed above the proposed standard for titanium dioxide is listed as 54,510 (the figure derived from OSHA's IMIS data alone) because no data was identified in this category in OSHA's telephone survey. It is not clear why the survey does not report data on titanium dioxide because according to OSHA the chemicals in the survey were "selected on the basis of . . . known exposure problems. . . ." If the survey had determined there were zero workers overexposed, then the combined assessment would have been reduced to 27,255—that is, 54,510 divided by 2 [Ex. 72, p. 11].

OSHA believes that it has designed an approach that makes optimum use of all of the exposure data available to the Agency. By using the averaging method described above, neither the survey data nor the IMIS data are given greater weight. OSHA realizes, as AISI points out, that the estimate of the number of employees exposed to a given chemical in a given industry sector is sensitive because OSHA's methodology uses an averaging approach. However, OSHA believes that the alternative, i.e., reliance on one data set as opposed to the other, is more disadvantageous because a vast amount of exposure information would have been ignored in the analysis. In addition, OSHA believes that making full use of both the IMIS and survey data minimizes any biases that may be inherent in the information contained in either data set alone. Furthermore, OSHA believes that, by determining maximum and minimum estimates of numbers of exposed employees, uncertainties in the analysis are appropriately recognized.

Some commenters [Exs. 3–890, 8–10, 8–31, and 8–32; Tr. 4–257] provided alternative estimates of the number of employees exposed and overexposed to specific chemicals in specific industries. For example, Dr. Boyd of the Styrene Information Research Council (SIRC) reports that, in the reinforced plastics industry segment, 30,000 workers are potentially exposed to styrene [Ex. 8–32, p.1], a figure smaller than that estimated by OSHA. Eric Frumin, Director of Occupational Safety and Health for the Amalgamated Clothing and Textile Workers Union (ACTWU), reports that OSHA's estimates severely understate the number of workers potentially exposed to perchloroethylene in the dry cleaning industry [Ex. 8–31, pp. 21–23]. OSHA evaluated each of these commenter's estimates to determine whether OSHA's aggregate benefits estimates of exposed workers needed to be revised to reflect this new record evidence. Because OSHA received comparatively few data on the number of employees exposed, when compared with the number of industry sectors and substances covered in this analysis, OSHA has determined that incorporating the estimates provided by commenters would not substantially alter OSHA's aggregate estimate of the benefits associated with revising the air contaminant limits.

Estimates of the Reduction in Illness Cases and Lost Workdays

The BLS LABSTAT data base contains illness and lost-workday rates by SIC code. These rates are expressed as the annual number of illness cases or number of lost workdays per 100 full-time-equivalent employees. Reducing employee exposures to hazardous substances to a level below that associated with adverse health effects will result in a decrease in the number of illness cases and lost workdays.

To assess the impact on illness and lost-workday rates of reducing employee exposures, OSHA first examined the relationship between the percentage of workers estimated to be exposed above the final exposure limits for the 160 substances represented in the IMIS data base and current illness and lost-workday rates. This analysis was conducted at the three-digit SIC code level because of the lack of illness-rate data for some of the four-digit SIC code groups. The results of this analysis are presented graphically in Figure D–1. Among three-digit industries for which OSHA has found that no employees are currently exposed above the final limits, total illness case rates reported by the BLS for the same industry group are usually less than 0.2 cases per 100 employees per year, and frequently are reported to be zero. In contrast, where OSHA has found that an industry group has more than 10 percent of its workforce exposed above the final limits, total illness case rates above 0.2 case per 100 employees are frequently reported. In few instances does an industry group having 10 percent or more of its workforce exposed above the final limits report a total illness case rate of zero. Among three-digit SIC code industry groups for which OSHA has not found employee exposures above the final limits, 38 percent of the groups reported an illness rate of zero, 43 percent reported an illness rate of 0.1 to 0.2 case per 100 employees, and only 19 percent of the industry groups reported an illness rate greater than 0.2 case per 100 employees (but none above 0.5 case per 100 employees). Given this distribution of illness rates across these particular industry groups, it is concluded that industry groups in which employee exposures have been controlled to or below the final limits will have an illness rate approximating 0.1 case per 100 employees. It is believed that total illness cases at the three-digit level will be reduced to no more than 0.1 case per 100 employees after exposures are reduced to or below the final limits.

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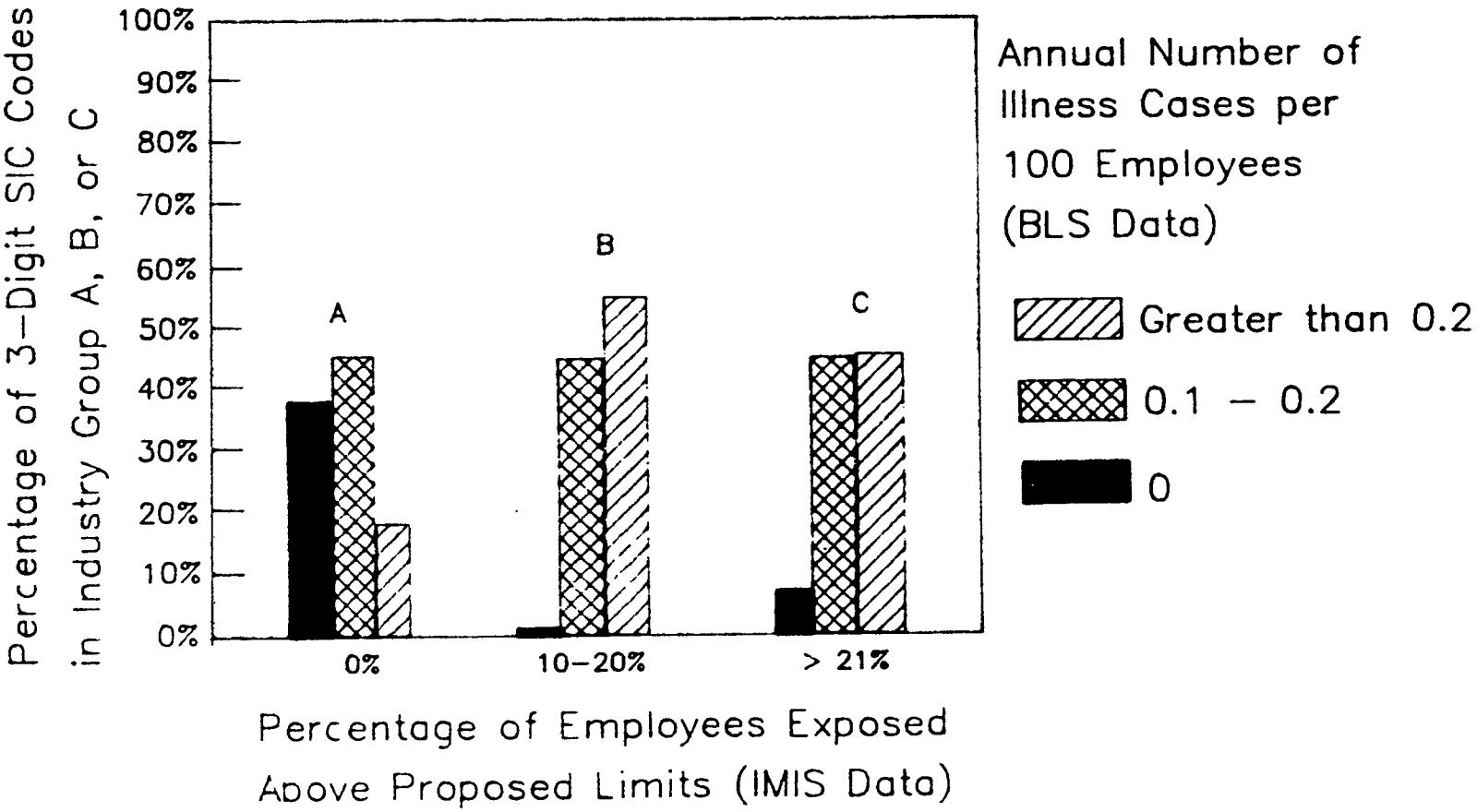
Figure D-1. Relationship Between Employee Exposures to Hazardous Substances and Industry Illness Rates



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OSHA performed a similar analysis that also indicates that the rate of lost-workday illness cases will decline to a base rate of 0.05 cases per 100 employees and the annual rate of lost workdays will decline to 1 case per 100 employees after employee exposures are reduced to or below the final limits.

OSHA estimated the number of illness cases and lost workdays potentially avoided annually by applying current rates to the estimated number of production workers per three-digit SIC group; this yielded an estimate of the annual number of illness cases and lost workdays reported by each three-digit SIC code industry group. It was assumed that these industries would experience illness rates of 0.1 cases per 100 employees, 0.05 lost-workday illness cases per 100 employees, and 1 lost workday per 100 employees per year. Using this approach, OSHA estimated that promulgation of the final limits will potentially avoid 55.365 illness cases per year, 23,346 lost-workday illness cases per year, and 519,421 lost workdays caused by illnesses per year.

The movement to a 0.1 illness rate is presented as a best estimate supported by OSHA's interpretation of the relationship between chemical exposure levels and current industry illness rates. It may be argued, however, that if a 0.1 illness rate were achieved, the reduction in illnesses could not be credited exclusively to OSHA's rulemaking initiative, since some portion of the current BLS illness rate is made up of illnesses associated with exposures to hazardous agents or physical stress (e.g., radiation, noise, ergonomic stress).

While no claim is made that this rulemaking action will reduce illnesses related to these causes, OSHA believes that the benefit estimates related to this final rule of over 55,000 illnesses, over 23,300 lost-workday illnesses, and over 519,000 lost workdays avoided each year are reasonable. This is based on the finding that company records, upon which the BLS data are based, rarely show chronic illnesses caused by exposures to toxic substances [4, 6]. The potential level of underreported illnesses in the BLS series is illustrated in a recent report by Landrigan and Markowitz [8]. Using California physicians' reports of occupational illnesses, these author's estimated an occupational illness rate among New York State employees that was more than twice the BLS illness rate [8].

Mr. Frank T. Ryan, Vice President of the Rubber Manufacturers Association (RMA), addressed OSHA's use of the illness rate data, commenting that

> there may be confounding factors, such as exposure to other substances, work practices, and especially non-occupational considerations, which affect the illness and lost workday rates. . . . Because this fundamental issue is not addressed in the calculations, the RIA does not derive meaningful illness or lost workday data [Ex. 3-877, p. 22].

Similarly, Peter Hernandez, Vice President for Employee Relations of the American Iron and Steel Institute (AISI), commented that the PRIA provided no basis for the assumption that including non-chemical-related illnesses in the calculation was offset by the underreporting of illnesses in the BLS statistic [Ex. 72].

Landrigan and Markowitz [8] provided a breakout of illness causes in the 1984 BLS statistics. They reported that about 30 percent of all illnesses were caused by trauma or exposure to physical agents. As described above, they also reported that illness cases may be underreported by as much as a factor of 2. This was also reported in a recent article by Suruda and Emmett [9]. Therefore, the extent to which illness cases are underreported far outweighs the proportion of illness cases not attributed to chemical exposures. As such, OSHA believes that inclusion of non-chemically related illnesses in the assessment does not result in an overestimate of the annual number of illness cases avoided by the final rule.

Mr. Hernandez of the AISI also suggested that OSHA's estimates for the illness rates are overestimated because they include dermatitis cases. He noted that:

> Because dermatitis is among the most common reported illnesses and is not associated with the employee airborne exposures, we believe OSHA's reliance on Bureau of Labor Statistic data overstates the benefits [Ex. 72, p. 13].

Although it is true that many of the reported cases of occupational illnesses are skin disorders, OSHA believes that reducing employee airborne exposures will contribute to a reduction in the number of cases of dermatitis. As a general rule, workplaces that have many cases of dermatitis are also more likely to use poor work practices and to be lacking in engineering controls; such facilities will have higher airborne exposures. On the other hand, a well-engineered facility with low airborne exposures generally also controls its employees' dermal exposures, and therefore has few, if any, cases of dermatitis. Therefore, OSHA believes that promulgation of these exposure limits for air contaminants will encourage the use of improved work practices, which will, in turn, reduce the incidence of dermatitis.

Estimates of the Number of Employees Potentially at Risk by Type of Hazard

In addition to estimating the number of employees exposed to the substances included in this analysis, OSHA also estimated the number of employees who are at risk of experiencing particular types of adverse health effects. To conduct this analysis, each substance included in the rulemaking was assigned to a health hazard category; these assignments were based on the primary health effects that provided the impetus for reducing a previous limit or establishing a new limit for a particular substance. (The assignment of substances to health effect categories is described in detail in Section VI-C of the preamble.) It should be noted that, in some instances, substances included in this rulemaking were grouped together in the preamble according to some basis other than a particular health effect; for example, several substances were grouped together because the ACGIH-recommended limits were derived based on the structural analogy of the grouped substances with that of other substances. For the benefits analysis described here, these substances were reclassified according to the primary health effect associated with exposure to the analogous chemical.

The number of employees estimated to be exposed to substances causing a particular health effect in an industry group was calculated by summing the number of employees exposed to all substances causing the same effect. Aggregate estimates across all affected industry sectors are presented in Table D-3. This table provides estimates of employees potentially exposed to substances in each health group, as well as estimates of employees exposed above the final limits for substances in each health group. Employees are frequently at risk from a variety of adverse health effects as a result of concurrent exposure to more than one toxic substance. Thus, the total number of employees considered to be at risk from any type of illness (as estimated in Table D-3) cannot be summed because the sum would result in doublecounting.

TABLE D-3. Estimated Number of Workers Potentially at Risk of Experiencing Adverse Effects, by Type of Adverse Effect*

ADVERSE HEALTH EFFECT	NO. OF WORKERS POTENTIALLY EXPOSED TO SUBSTANCES ASSOCIATED WITH EFFECT, MINIMUM ESTIMATE	NO. OF WORKERS POTENTIALLY EXPOSED TO SUBSTANCES ASSOCIATED WITH EFFECT, MAXIMUM ESTIMATE	NO. OF WORKERS EXPOSED ABOVE FINAL LIMITS FOR SUBSTANCES, MINIMUM ESTIMATE	NO. OF WORKERS EXPOSED ABOVE FINAL LIMITS FOR SUBSTANCES, MAXIMUM ESTIMATE
PHYSICAL IRRITANT EFFECTS	3,375,472	3,889,261	222,191	222,191
ODOR EFFECTS	519,318	521,938	3,597	3,597
SYSTEMIC TOXICITY	4,305,578	5,038,573	457,104	490,282
MUCOUS MEMBRANE IRRITATION	10,730,691	14,906,090	789,461	1,141,133
METABOLIC INTERFERENCES	4,015,702	4,205,530	1,233,413	1,241,564
LIVER/KIDNEY DISEASE	3,292,993	3,806,226	536,945	546,429
OCULAR DISTURBANCES	2,482,449	2,569,950	83,272	110,560
RESPIRATORY DISEASE	4,231,235	4,782,280	1,405,501	1,568,579
CARDIOVASCULAR DISEASE	166,077	166,868	44,403	44,403
NEUROPATHY	2,212,358	2,463,583	379,974	401,576
NARCOSIS	6,966,024	10,520,982	941,472	1,073,717
CANCER	1,712,799	1,851,342	465,013	528,650
ALLERGIC SENSITIZATION	2,545,551	2,648,973	305,955	305,955

* Double counting of employees simultaneously exposed to more than one substance in different adverse health effects categories prevents the summation of workers exposed to all adverse health effects in this table.

Estimates of the Number of Illness-Related Fatalities Avoided

As discussed in the preceding section, OSHA has estimated the number of employees currently at risk of experiencing a variety of adverse health effects brought about by overexposures to the substance included in this rulemaking. Many of these adverse effects, in particular, cancer, cardiovascular effects, chronic respiratory disease, and chronic liver and kidney damage, result in lethal outcomes. OSHA also believes that employees who are excessively exposed to substances causing systemic organ damage, neurological impairment, or metabolic effects (i.e., cardiovascular disease through excessive formation of methemoglobin or carboxyhemoglobin, and neurological impairment through cholinesterase inhibition) are at excess risk of incurring a fatal condition.

To estimate the number of fatalities associated with excessive exposure to the 212 substances included in this analysis, OSHA relied on standard U.S. mortality rates and on published estimates of the proportion of fatalities that are believed to be associated with occupational illnesses. These data allowed OSHA to calculate cause-specific mortality rates that are attributable to occupational illnesses (i.e., mortality rates that represent the excess risk of mortality from occupational disease). OSHA then applied these occupationally related mortality rates to its estimates of the number of employees exposed to the substances of concern at levels above the final limits. OSHA's methodology and estimates are presented in Table D-4, and are described in detail below.

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TABLE D-4. Estimated Annual Number of Fatalities Caused By Occupational Illness Among Workers Currently Exposed Above Final Limits, Using Alternative Assumptions

Cause of Death	U.S. Annual Death Rate Per 100,000 Residents (1985), by Cause[a]	Total Number of Deaths Per Year in U.S., by Cause[b]	Number of Deaths Attributed to Occupational Illnesses, by Cause	Annual Death Rate Per 100,000 Attributed to Occupational Illnesses	Number of Workers Exposed Above Final Limits	Annual Number of Fatalities Among This Group of Workers
Cancer	193.3	461,484	23,074[c]	27.7	496,832[f]	138
Chronic Pulmonary Disease	31.3	74,726	2,242[d] 747[e]	2.7 0.9	1,487,040[g]	40 13
Chronic Liver Disease	11.2	26,739	802[d] 267[e]	1.0 0.3	541,687[h] 541,687[h]	5 2
Cardiovascular, Neurological, and Renal	418.5	999,127	29,974[d] 9,991[e]	35.9 12.0	2,146,360[i]	771 258
TOTAL, All Causes						411-954

[a] Source: National Center for Health Statistics [5, Table 11].
[b] Based on a total residential population in 1985 of 238,740,000. [1, p. 18].
[c] Assumes 5 percent of all cancer deaths are of occupational origin (Landrigan and Markowitz, 1987).
[d] Assumes 3 percent of all deaths are of occupational origin (Landrigan and Markowitz, 1987).
[e] Assumes 1 percent of all deaths are of occupational origin (Landrigan and Markowitz, 1987).
[f] From Table D-3, midpoint estimate of number of workers exposed above final limits for potential carcinogens.
[g] From Table D-3, midpoint estimate of number of workers exposed above final limits for respiratory toxins.
[h] From Table D-3, midpoint estimate of number of workers exposed above final limits for liver toxins.
[i] From Table D-3, midpoint estimate of number of workers exposed above final limits for systemic toxins, metabolic toxins, cardiovascular toxins, and neuropathic agents.

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Estimate of the Number of Cancer Fatalities. The U.S. National Center for Health Statistics has published cause-specific U.S. mortality rates for 1985 (the most recent data available) [5]. This source reported that the annual U.S. cancer death rate in 1985 was 193.3 per 100,000 residents. Based on a total resident U.S. population of 238,740,000 in 1985 [7, p. 18], the number of cancer deaths that occurred in 1985 was 461,484. Landrigan and Markowitz [8] reviewed several published estimates of the percentage of cancer deaths that are attributable to occupational related disease; these estimates range from less than 5 percent to 33 percent of all cancer deaths. Landrigan and Markowitz believe that, as a best estimate, 10 percent of all cancer deaths have an occupational origin.

Several commenters (Exs. 3–527 and 3–877) expressed the opinion that OSHA had overestimated the number of occupationally induced cancers that would be prevented by promulgation of the final rule's limits. Specifically, they criticized the study by Landrigan and Markowitz, who estimated that 5 to 33 percent of cancer deaths and 1 to 3 percent of all deaths have occupational origins. To support this argument, Mr. Ryan, of the RMA, cited the Doll-Peto study, which stated:

> On present knowledge, it is impossible to make any precise estimate of the proportion of the cancers today that are attributable to hazards at work . . . and none of the estimates that have been made are claimed to be anything more than informed guesses. . . .
>
> . . . Until objective, nationally representative studies are undertaken, a more realistic assessment of the role of occupational hazards can probably be obtained by considering each type of cancer separately and estimating for each type the possible contribution of occupation.
>
> . . . The proportion of cancer deaths that we have tentatively attributed to occupational causes is, therefore, about 17,000 out of 400,000; i.e., about 4 percent of all U.S. cancer deaths (Ex. 3–877, pp. 23–24).

At the informal hearing, Dr. Landrigan testified that his estimate that 10 percent of all cancers are occupationally induced is reasonable:

> [T]he Doll-Peto estimate is low, for several reasons. First of all, they did not include in their estimate cancers which occurred in people over the age of 65.
>
> Many occupational cancers don't develop in people until 20, 30, or even 40 years after exposure has occurred. Therefore, to cut off attribution of cancer to occupational exposure at age 65 almost certainly reduces the proportion of all cancers which can be attributed to occupation.
>
> Another factor . . . which . . . diminishes the accuracy of the Doll-Peto estimate is that they excluded from consideration certain categories of cancer.
>
> I think that 10 percent is a reasonable middle-of-the-road estimate. If you like arithmetic manipulation, then that figure is the geometric mean . . . between the Doll-Peto estimate of 4 percent and the old Califano estimate of 38 percent (Tr. p. 3–285).

OSHA believes that Dr. Landrigan's assessment of the Doll and Peto study is reasonable. Given the wide range in published estimates of the proportion of cancers that are occupationally related (4 to 33 percent), OSHA used alternative estimates of five and ten percent in the PRIA; the assessment for this rule is based on the five percent estimate of all cancer deaths being occupationally related. Use of the five percent estimate is consistent with OSHA's recent benefits analysis for the Hazard Communication standard.

Using an occupational cancer death estimate of 5 percent and applying it to the estimated number of cancer deaths in 1985, OSHA estimates that 23,074 occupationally related cancer deaths occurred in the United States in that year (Table D–4).

As the next step, OSHA estimated the overall cancer death rate, both among the population that is occupationally exposed to chemicals and among the remainder of the population. In 1985, there were an average of 108,856,000 persons employed [10, p. 8]. However, 25,469,200 of these were employed in industries or occupations in which there is a low risk of exposure to toxic substances, such as finance, insurance, real estate, and private households [10, pp. 30, 84–88]. The remaining 83,386,800 persons are considered to be occupationally exposed to chemicals in varying degrees. Many would have only intermittent exposures at very low levels. Assuming that 5 percent of all cancer deaths are of occupational origin, OSHA calculated the annual cancer death rate attributed to occupational exposure by dividing the number of cancer deaths attributable to occupational illness by the population exposed, and multiplying that figure by 100,000. OSHA estimates that the annual cancer mortality rate attributable to occupational exposure to toxic substances is 27.7 per 100,000. OSHA then estimated that there are 496,832 workers currently exposed above the final limits to the potential carcinogens included in this rulemaking for which data were available. Applying the work-related cancer death rates to this population, OSHA estimates that 138 cancer fatalities occur each year among these workers, and that these fatalities will be prevented by the final rule.

In arriving at this estimate, two important offsetting arguments were considered. Because some of these workers may also be exposed to occupational carcinogens that are not covered in this rulemaking (such as asbestos or benzene), the number of occupational cancer deaths attributed to the substances included in this rulemaking may be overestimated. Offsetting this potential overestimate is the fact that the excess mortality rate of 27.7 per 100,000 workers was developed on the basis of occupational exposures among all workers. However, the excess mortality rate experienced among workers with high average exposures to hazardous chemicals typically runs at least two to three times higher than the national average rate. In consideration of this, OSHA believes that any overestimate of cancer fatalities avoided attributed to regulated chemicals not covered under this rulemaking is offset by the use of a mortality rate that understates the true excess mortality rate among workers with very high exposures to toxic chemicals. (Additional comments on excess mortality rate estimates are included in the final section of this chapter.)

An alternative analysis of the reduction in cancer mortality was conducted using OSHA's quantitative risk assessments for the potential human carcinogens included in this rulemaking (the results of OSHA's risk assessments are presented in the preamble to the final rule). This analysis is presented in Table D–5. Using available data from the combined IMIS and 1988 survey, OSHA found that employees are currently exposed above the final limits to four of the 17 potential carcinogens listed (acrylamide, carbon tetrachloride, chloroform, and perchloroethylene). Applying quantitative risk estimates to the estimated number of workers currently overexposed to these four substances only, OSHA estimates that compliance with the final limits will avoid 11,519 cancer fatalities over the working lifetime of the population (i.e., 45 years). The average annual reduction in the number of cancer fatalities avoided over 45 years is estimated to be 256.

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TABLE D-5. Estimates of Cancer Deaths Potentially Avoided, Based on Quantitative Risk Assessments, Over 45 Years

Substance	Number of Workers Above Final Limit	Estimated Number of Cancer Deaths	Estimated Number of Cancer Deaths Avoided
Acrylamide	7,896	79	71
Carbon Tetrachloride	97,134	1,739	1,380
Chloroform	123,950	2,776	2,743
Perchloro-ethylene	267,821	12,052	7,325
TOTAL, 45 years		16,646	11,519
ANNUAL		370	256

[a] Risk assessments are presented in Section IV-C-15 of the preamble. The assessment for perchloroethylene is based on risk estimates developed by Dr. Dale Hattis (Ex. 8-31, App. II-A).

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As noted, although OSHA has evaluated the cancer risk for 17 potential carcinogens, there were IMIS data, survey data, and quantitative risk assessments (all of which are necessary for benefits analysis) for only four of these. Lack of IMIS or survey data means that the substance has not been sampled by an OSHA compliance officer or that none of the survey participants indicated that the substance was used at their facilities. This does not mean that no workers are currently exposed to these substances. Lacking a basis for estimating the extent of employee exposure, OSHA could not estimate the extent of reduction in cancer deaths attributable to the reduction in exposure limits for these substances. To the extent that employee exposure to these carcinogens is reduced, further reductions in the number of cancer deaths will occur.

Estimated Reduction in Occupational Deaths from Causes Other than Cancer. As shown in Table D–4, OSHA also estimated the number of occupationally related fatalities that are expected to occur annually among employees exposed to substances associated with adverse health effects other than cancer. To perform this analysis, OSHA relied on an estimate made by Landrigan and Markowitz [7] that between 1 and 3 percent of all nonmalignant disease is of occupational origin. Using the 1- and 3-percent figures as alternative assumptions and using the same methodology as that described above for cancer deaths, Table D–4 shows the following:

—Between 13 and 40 deaths caused by respiratory disease are estimated to occur each year among workers exposed to respiratory toxins covered in this rulemaking;

—Between 2 and 5 deaths are estimated to occur each year among workers exposed to liver toxins covered in this rulemaking; and

—Between 258 and 771 deaths are estimated to occur each year among workers exposed to systemic toxins, cardiovascular toxins, metabolic toxins, and neurological toxins covered in this rulemaking.

Summing these estimates, OSHA believes that between 411 and 954 non-cancer-related occupational fatalities occur each year. The same offsetting considerations discussed in the analysis of the cancer fatalities avoided under this rule also apply here. While some substances are being controlled by activity outside of this rulemaking, any overestimation effect is balanced by an underestimate of the real excess mortality rate for workers with high exposure levels to the chemicals under consideration.

The Chemical Manufacturers Association (CMA) (Ex. 3–527) stated that OSHA made the assumption that reducing exposures will eliminate *all* cancer fatalities that are estimated to occur from exposure to carcinogens included in the rulemaking. They argue that this is inconsistent with OSHA's statement in the preamble that

> there is reason to assume that the dose-response of most carcinogens will follow a linear, non-threshold relationship (Preamble 21190. Col. 3) [Ex. 3–527, p. 42].

OSHA's approach, based on estimating excess death rates (Table D–4), did assume that all cancers caused by exposure to the four substances would be avoided; however, changing this assumption would not have a major impact on the estimated total number of fatalities avoided. For example, if it is assumed that only half of the estimated number of cancer fatalities would be avoided (a conservative assumption, given that most PELs are being reduced by more than a factor of 5), then the estimated annual number of cancer fatalities avoided would be 69 rather than 138. The estimated total annual number of avoidable deaths from all causes would range from 324 to 885. This estimate is only about 7 to 17 percent less than the estimate of 411 to 954 avoidable deaths reported in the Table D–4. Since cancer fatalities avoided represent only a part of the benefits to be achieved through this rulemaking, changing the assumption on cancers avoided will not result in a substantial change to the total number of avoidable fatalities attributable to revising the PELs. Furthermore, OSHA's alternative approach, which relies on the quantitative estimates of risk, identified a larger number of cancers avoided each year (256). This latter method takes into account the presence of residual risk at the revised PELs.

AISI (Ex. 188, p. 43) also argued that OSHA overstated benefits estimates presented in the PRIA because the effect of the Hazard Communication standard was not considered. In that rulemaking, OSHA determined that the hazard communication standard could reduce occupation-related cancers by 20 percent. If the beneficial effects of the Hazard Communication standard are considered in assessing the benefits associated with revising the PELS on Table Z, the maximum effect would be to reduce OSHA's estimate of cancer fatalities avoided by 20 percent (i.e., from 138 to 115).

In sum, the combined estimate for the number of cancer and noncancer deaths potentially avoided each year by compliance with the new limits is between 411 and 954 or an average of 683 fatalities avoided each year. OSHA considers these to be reasonable estimates of the benefits associated with revising the PELs on Table Z.

Additional Comments and an Alternative Method for Estimating Excess Mortality Rates. The analysis described above to estimate the number of fatalities that are potentially preventable relies on published estimates of the proportion of all U.S. fatalities that are believed to result from occupational illnesses. These estimates were used with U.S. cause-specific mortality rate figures to estimate the excess mortality rate among all U.S. workers, by cause of death (shown in Table D–4).

In making these excess mortality rate estimates, OSHA applied the excess number of fatalities across the entire U.S. working population. Implicit in this approach is an assumption that all workers are at some risk of fatality from all causes of death. In fact, only a portion of the workforce is at risk of fatality from each type of occupational illness. Deaths will occur only among workers who are potentially exposed to carcinogens; no excess deaths will occur among workers who are not so exposed. Similarly, not all workers are at risk of dying from occupationally related cardiovascular illnesses; only some portion of the workforce are at excess risk, and all fatalities resulting from occupationally related cardiovascular disease will occur among this subset of workers. Because OSHA's excess mortality rate estimates presented earlier were derived by applying the estimated number of work-related fatalities across the entire U.S. workforce, excess mortality rate figures are likely to be substantially understated.

To assess the magnitude of this bias, OSHA conducted an alternative analysis to estimate the number of work-related fatalities that are expected to occur among workers exposed above the final limits. This alternative assessment relied on judgments regarding the general increase in mortality rates that are frequently observed in epidemiologic studies that demonstrate a causal relationship between exposure to toxic substances and excess disease mortality. The alternative assessment is presented in Table D–6.

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TABLE D-6

Alternative Assessment of Number of Fatalities Expected to Occur Among Workers Currently Exposed Above Final Limits

Cause of Death	U.S. Cause-Specific Mortality Rate, Per 100,000 Residents[a] (1985)	Estimated Excess Mortality Rate Per 100,000 Workers at Risk From Hazard	Number of Workers Exposed Above Final Limits	Annual Number of Fatalities Among This Group of Workers
Cancer	193.3	193.3[b]	496,832[c]	960
Chronic Pulmonary Disease	31.3	9.4[d]	1,487,040[c]	140
Chronic Liver Disease	11.2	3.3[d]	541,687[c]	18
Cardiovascular,	418.5	125.6[d]	2,146,360[c]	2,696
TOTAL				3,814

[a] Source: National Center for Health Statistics [5, Table 11].

[b] Assumes that overall cancer mortality rate among workers at risk is twice the U.S. rate (i.e., a 100-percent excess rate).

[c] From Table D-4.

[d] Assumes that overall disease mortality rate among workers at risk is 1.3 times the U S. rate (i.e., a 30-percent excess risk).

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The overall U.S. cancer mortality rate for 1985 is 193.3 deaths per 100,000 residents (Table D–6). Typically, when causal relationships between exposure and excess lung cancer mortality are found in epidemiologic investigations, the exposed cohort frequently shows a cancer mortality rate of 1.1 to 10 times higher than the general population. For cancers that are more rare than lung cancer, mortality rates among working populations may be 50 times higher than for the general population. An alternative estimate of the number of cancer fatalities expected to occur among the estimated 499,716 workers exposed above the final limits for the potential carcinogens could be developed based on the assumption that the overall cancer fatality rate among these workers is twice that of the U.S. population (i.e., 386.6 per 100,000 workers versus 193.3 per 100,000 residents). The *excess* cancer mortality rate among these workers is therefore assumed to be 193.3 per 100,000 workers (386.6 minus 193.3). Applying this estimated excess cancer mortality rate to the 496,832 workers exposed above the final limits yields an estimated 960 cancer deaths occurring annually that are attributable to occupational exposure. For example, Dùh and Asal [Ex. 8–31, App. 7] reported excess lung cancer death rates for drycleaning workers of 1.7 times expected death rates, as well as kidney cancer death rates of 3.8 times expected rates. Similarly, EPA [Ex. 1–1132] reported standard mortality ratios for lung cancer of generally between one and ten for nickel refinery workers. This same approach could be used for estimating non-cancer-related fatalities assuming that the overall fatality rate among workers at risk from these illnesses is 1.3 times the corresponding U.S. mortality rate (mortality rates of 1.1 to 1.5 are frequently observed in epidemiologic studies demonstrating causal relationships between exposure and excess fatalities). This amounts to an *excess* mortality rate of 30 percent above the overall U.S. rate. Applying these excess mortality rate figures to the estimated worker populations exposed above the final limits, OSHA estimates that, among these workers, 140 deaths occur annually due to chronic pulmonary disease, 18 deaths occur annually due to liver disease, and 2,696 deaths occur annually due to cardiovascular, neurological, and renal diseases. In total, including cancer, OSHA estimates that 3,814 work-related fatalities (including those from cancer) may be occurring each year among employees who are exposed above the limits to the hazardous substances included in this rulemaking.

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E. Assessment of Nonregulatory Alternatives

Introduction

The declared purpose of the Occupational Safety and Health (OSH) Act of 1970 is ". . . to assure so far as possible every working man and woman in the Nation safe and healthful working conditions and to preserve our human resources. . . ." Thus, the Act requires the Secretary of Labor, when promulgating occupational safety and health standards for toxic materials or harmful physical agents, to set the standard ". . . that most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity. . . ." It is on the basis of this congressional directive that OSHA has initiated regulatory actions to reduce the adverse health effects associated with occupational exposure to hazardous substances.

Market Failure

Economic theory suggests that the need for government regulation is greatly reduced where private markets work efficiently and effectively to allocate health and safety resources. The theory typically assumes perfectly competitive labor markets where workers, having perfect knowledge of job risks and being perfectly mobile among jobs, command wage premiums that fully compensate for any risk of future harm. Thus, theoretically, the costs of occupational injury and illness are borne initially by the firms responsible for the hazardous workplace conditions and, ultimately, by the consumers who pay higher prices for the final goods and services produced by these firms. With all costs internalized, private employers have an incentive to reduce hazards wherever the cost of hazard abatement is less than the cost of the expected injury or illness. The resultant level of safety and health is considered "efficient" in the sense that it minimizes the sum of the costs of hazard prevention and of injury or illness. Perfectly competitive labor markets, however, do not exist for many industrial markets. OSHA, therefore, believes that it must take appropriate actions to provide greater health protection for workers exposed to toxic substances.

Evidence indicates that market forces have not been effective in reducing excessive occupational exposure to hazardous substances, thereby contributing to the consequent development of occupational diseases. In spite of the danger associated with the inhalation or other exposure to hazardous substances, the social costs of production have not been internalized, in part, because of market imperfections and the existence of externalities. Consequently, the amount of protection that the private market will offer to workers differs from the socially desired level.

First, evidence on occupational health hazards in general suggests that in the absence of immediate or clear-cut danger, employees and employers have little incentive to seek or provide information on the potential long-term effects of exposure. Employers faced with potentially high compensatory payments may, in fact, have a disincentive to provide information to employees. When relevant information is provided, however, employers and employees might still find informed decisionmaking a difficult task, especially where long latency periods precede the development of chronic disabling disease. Moreover, if signs and symptoms are nonspecific—that is, if an illness could be job-related or could have other causes—employees and employers may not link disease with such occupational exposure.

Second, even if workers were fully informed of the health risks associated

with exposure to hazardous substances, many face limited employment options. Nontransferability of occupational skills and high national unemployment rates sharply reduce a worker's expectation of obtaining alternative employment quickly or easily. A worker employed in a foundry, for example, could find it difficult to apply occupational skills to a new job in searching for a safer workplace.

In many regions of the country, the practical choice for workers is not between a safe job and a better paying but more hazardous position, but simply between employment and unemployment at the prevailing rates of pay and risk. In addition to the fear of substantial income loss from prolonged periods of unemployment, the high costs of relocation, the reluctance to break family and community ties, and the growth of institutional factors such as pension plans and seniority rights serve to elevate the cost of job transfer. Thus, especially where wages are more responsive to the demands of more mobile workers who tend to be younger and perhaps less aware of job risks, hazard premiums for the average worker will not fully compensate. Where this is the case, labor market negotiations are unlikely to reflect accurately the value that workers place on health.

In addition to the market imperfections, externalities occur if employers and employees settle for an inefficiency low level of protection from hazardous substances. For the competitive market to function efficiently, only workers and their employers should be affected by the level of safety and health provided in market transactions. In the case of occupational safety and health, however, society shares part of the financial burden of occupationally induced diseases, including the costs of premature death, chronic illness, and disability. Those individuals who suffer from occupationally related illness are cared for and compensated by society through taxpayer support of social programs, including welfare, Social Security, and Medicare. These combined factors of labor market imperfections and the existence of externalities contribute to the failure of the market to supply healthful working conditions in industries where hazardous substances exist.

Tort Liability

The use of liability under tort law is one nonregulatory alternative that has been increasingly used in litigation concerning occupationally related illnesses. Prosser [1] describes a tort, in part, as a "civil wrong, other than a breach of contract, for which the court will provide a remedy in the form of an action for damages," although he says that "a really satisfactory definition has yet to be found."

If the tort system applies, it would allow a worker whose health has been adversely affected by occupational exposure to a hazardous substance to sue and recover damages from the employer. Thus, if the tort system is effectively applied, it might shift the liability of direct costs of occupational disease from the worker to the firm under certain specific circumstances.

With very limited exceptions, however, the tort system is not a viable alternative in dealings between employees and their employers. All states have legislation providing that Workers' Compensation is either the exclusive or principal remedy available to employees against their employers. Thus, under tort law, workers with an occupational disease caused by exposure to a hazardous substance can only file a product liability suit against a third party manufacturer (e.g., Johns Manville), processor, distributor, sales firm, installer, agency, or contractor. It is often difficult, however, to demonstrate a direct link between an exposure to a hazardous substance and the illness.

In order to pursue litigation successfully, there must be specific knowledge of the magnitude and duration of a worker's exposure to a hazardous substance, as well as the causal link between the disease and the occupational exposure. Usually, it is extremely difficult to isolate the role of occupational exposures in causing the disease, especially if workers are exposed to many toxic substances. This difficulty is further compounded by the long latency periods that are frequently involved. In addition, the liable party must be identifiable, but workers may have several employers over a working lifetime. The burden of proof that an occupational exposure to a hazardous substance occurred, that a specific employer is the liable party, and that the exposure level was significant may prohibit the individual from initiating the suit.

The costs associated with producing information and with litigation itself may be quite substantial. First, information is a public good, which means that once produced it can be transmitted inexpensively to any number of individuals without diminishing the quality or quantity of the information. It is therefore, difficult to control distribution once the information is produced. A producer of information may find that information produced at great expense can be acquired freely by potential customers, and that consequently, the market for the information has virtually disappeared. As a result, public goods are typically underproduced relative to what is considered economically efficient. This general undersupply of information adversely affects workers' awareness of the cause of their illness and thus reduces the likelihood that they will pursue tort liability suits.

Second, legal proceedings impose costs on both plaintiffs and defendants. In deciding whether to sue, the tort victim must be sure that the size of the claim will be large enough to cover legal expenses. In effect, the plaintiff is likely to face substantial transaction costs in the form of a contingency fee, commonly 33 percent, plus additional legal expenses. The accused firm must also pay for its defense.

The majority of occupational disease tort activity has involved workers exposed to asbestos. To date, approximately 100,000 individual plaintiffs have filed asbestos lawsuits in the country. These employees avoided the exclusive remedy of Worker's Compensation by suing suppliers of asbestos instead of employers. A report prepared by the Research Triangle Institute entitled. *Tort Liability and Worker Health: An Examination of the Economic, Legal, and Scientific Issues Surrounding the Occupational Disease Protection Afforded by Tort Law* [2], contains some data pertaining to legal costs and the size of awards. One investigator, for example, found that an average ratio of legal costs to proceeds was 37 percent for a sample of cases. The data, however, do not separate legal fees paid by the defendants and plaintiffs.

Insurance and liability costs are not borne in full by the specific employer responsible for the risk involved. For firms that are insured, the premium determination process is such that premiums only partially reflect changes in risk associated with changes in exposure to hazardous substances. This lack of complete adjustment is the so-called "moral hazard problem," which is the risk that arises from the possible dishonesty or imprudence of the insured. As the insured firm has paid an insurance company to assume some of the risks, that firm has less reason to exercise the diligence necessary to avoid losses. Transfer of risk is a fundamental source of imperfection in markets.*

* For a general discussion of moral hazard as a source of market failure, see Arrow [4] and Spence

Continued

For firms that self-insure or carry liability insurance with a large deductible, the costs of a single claim may be fully borne by the firm. Very small firms, and large firms with a large number of claims, however, may fail to meet the full costs by declaring bankruptcy. For example, the Johns Manville Corporation** declared bankruptcy to avoid massive claims associated with asbestos-related disease. Although the firm experienced a sharp decline in the value of its stock, it is still in business, while its obligation to pay asbestos-related claims is in considerable doubt. Other asbestos producers, including U.N.R. Industries, Inc. and Amatex Corporation, have followed the example of the Manville Corporation by filing for bankruptcy [9], further reducing the chances that their workers or others who contract asbestos-related diseases will collect Worker's Compensation or tort liability awards

Workers' Compensation

The Workers' Compensation system is a result of the perceived inadequacies in liability or insurance systems to compel employers to prevent occupational disease or compensate workers fully for their losses. The system was designed to internalize some of the social costs of production, but in reality, it has fallen short of compensating workers adequately for occupationally related disease. Thus, society shares the burden of occupationally related adverse health effects, premature mortality, excess morbidity, and disability through taxpayer support of social programs such as welfare, Social Security disability payments, and Medicare.

Compensation tends to be inadequate, especially in permanent disability cases, in view of the expiration of benefit entitlements and the failure to adjust benefits for changes in a worker's expected earnings over time. As of January 1987, 8 states still restricted permanent disability benefits either by specifying a maximum number of weeks for which benefits could be paid or by imposing a ceiling on dollar payments [10].

At present, time and dollar restrictions on benefit payments are even more prevalent in the area of survivor benefits. The duration of survivor benefits is often restricted to 10 years, and dollar maximums on survivor payments range from $7,000 to $60,000. In addition, it should be noted that if the employee dies quickly from the occupational illness and has no dependents, the employer need pay only nominal damages under Workers' Compensation (i.e., a $1,000 death benefit).

Finally, in spite of current statutory protection, disability from occupational diseases represents a continuing, complex problem for Workers' Compensation programs. Occupational diseases may take years to develop, and more than one causal agent may be involved in their onset. Consequently, disabilities resulting from occupationally induced illness often are less clearly defined than those from occupationally induced injury. As a result, Workers' Compensation is often a weak remedy in the case of occupational disease. For example, as recently as April 1983, the U.S. Supreme Court refused to hear an occupational disease case (*Richard D. Bunker* v. *National Gypsum Co.*) involving a worker who was diagnosed as having asbestosis 23 years after the expiration of the 3-year time limit allowed by Indiana law for filing a compensation claim [11]. Indeed, there is some evidence indicating that the great majority of occupationally induced illnesses are never reported or compensated [12].

The insurance premiums paid by a firm under the Workers' Compensation system are generally not experience rated—that is, they do not reflect the individual firm's job safety and health record. About 80 percent of all firms are ineligible for experience rating because of their small size. Such firms are class rated, and rate reductions are granted only if the experience of the entire class improves. Even when firms have an experience rating, the premiums paid may not accurately reflect the true economic losses. Segregation of loss experience into classes is somewhat arbitrary, and an individual firm may be classified with other firms that have substantially different normal accident rates. An experience rating is generally based on the benefits paid to workers, not on the firm's safety record. Thus, employers may have a greater incentive to reduce premiums by contesting claims than by initiating safety measures.

In summary, the Workers' Compensation system suffers from several defects that seriously reduce its effectiveness in providing incentives for firms to create safe and healthful workplaces. The scheduled benefits are significantly less than the actual losses to the injured workers, and recovery is often very difficult in the case of occupational diseases. Thus, the existence of a Workers' Compensation system limits an employer's liability significantly below the actual costs of the injury. In addition, premiums for individual firms are unlikely to be specifically related to that firms's risk environment. The firm, therefore, does not receive the proper "signals" and consequently fails to invest sufficient resources in reducing workplace injuries and illnesses. The economic costs not borne by the employer are borne by the employee or, as is often the case, by society through public insurance and welfare programs.

Standards of Other Organizations

Traditionally, representatives of professional organizations have collectively developed voluntary guidelines to assist members in maintaining safe and healthful working conditions for their employees. These guidelines are widely disseminated among members of the organizations and, at times, have been adopted as guidelines by organizations beyond the initiating one as well as by industry groups. In some cases they have become the *de facto* industry standard. Three professional organizations have developed voluntary guidelines in the form of exposure limits for chemical substances: The American National Standards Institute (ANSI); the American Industrial Hygiene Association (AIHA); and the American Conference of Governmental Industrial Hygienists (ACGIH). ANSI has withdrawn its earlier hazardous substance standards and has stated it does not intend to publish any others. The AIHA has a rather limited list of recommended limits. However, the ACGIH has published an extensive list of threshold limit values (TLVs) for many years. The ACGIH is recognized throughout the world for its members' expertise and contribution to industrial hygiene.

In May 1971, OSHA adopted as Federal health standards the exposure limits recommended by ANSI and ACGIH for 425 chemicals. Since that time, advances in scientific knowledge have demonstrated that those limits are not always adequate to protect employee health. Consequently, the ACGIH, the professional organization which continues to develop TLVs, has

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and Zechhauser [5]. For applications of this concept to employee health and safety, see Chelius [6], Rea [7], and Consad and General Research Corporation [8, Section 5.1].

** Johns Manville Corporation, formerly the world's largest asbestos manufacturer, filed for Chapter XI protection under the Federal Bankruptcy Law in August 1982. The company was financially solvent when it filed for bankruptcy but estimated that it would ultimately face a cost of more than $2 billion to settle 52,000 asbestos-related claims. In the meantime, the company's assets have been frozen and successful plaintiffs cannot collect awards [9].

changed its recommendations yearly to reflect later information. However, adherence to the TLVs developed after 1971 is purely voluntary. Except for imminent hazards, there is no sanction for failure to comply with the limits and many employers have not adopted practices which would control employee exposure to these new levels.

In addition to professional organizations, international bodies such as the European Economic Community, the International Labor Organization, and the World Health Organization have recommended exposure limits for some hazardous substances. While these limits may not be as widely known in the United States as those of U.S. professional organizations, they are made available to the industrial hygiene community through professional journals and meetings. Within the U.S., the National Institute for Occupational Safety and Health (NIOSH) of the Department of Health and Human Services has published recommended exposure limits (RELs) for a number of chemicals. These are publicized through NIOSH Current Intelligence Bulletins and other publications which are widely disseminated.

Although the ACGIH TLVs and the NIOSH RELs are widely recognized by health professionals and employers alike, OSHA has found that some employers are not complying voluntarily with the newer TLVs, the RELs, or the standards of other bodies. Chapter D discussed OSHA's estimates of the extent of exposures in excess of the TLVs, and the adverse health effects resulting from such exposure. OSHA believes that significant numbers of employees are exposed to chemicals at levels exceeding those recommended by other organizations, and that OSHA cannot rely on employers to comply voluntarily with the recommendations. Therefore, OSHA concluded that this nonregulatory alternative is not generating the optimal level of occupational health.

Conclusion

OSHA believes that there are no nonregulatory alternatives that adequately protect workers from the adverse health effects associated with exposure to the chemicals regulated in this rulemaking. OSHA believes that tort liability laws and Workers' Compensation do not provide adequate worker protection due to market imperfections. Some employers have not complied with the standards recommended by professional organizations. The deleterious health effects resulting from continued high levels of exposure to hazardous substances require a regulatory solution.

The National Grain and Feed Association (NGFA) has disagreed with OSHA's conclusion that this rule is necessitated by a situation of market failure [Ex. 180A]. NGFA claims that OSHA "ultimately rejects each of the alternatives because of what it characterizes as imperfections in the ability of each of the alternatives to meet fully that alternative's theoretical objectives." OSHA is not implying that nonregulatory alternatives are complete failures, but that they are not total successes. That they are partial failures is precisely the situation that creates the need for OSHA. Because OSHA cannot write and enforce a unique set of regulations for each facility, the regulations must be written and enforced on an industry-wide basis. This does not imply that all firms have failed to adopt guidelines, but that some have and that workers at these firms are potentially at risk.

The rules will not impact on those entities which have already adopted the voluntary guidelines of ACGIH and NIOSH. What the rule will do is compel those firms that have done little voluntarily, to act. Furthermore, firms which comply voluntarily can be at a competitive disadvantage in the short run. When some firms don't comply with voluntary standards, the pressure not to comply increases on all firms. When all firms must comply with a regulation, none should be at a competitive advantage or disadvantage as a direct result of the regulation.

NGFA also states that although none of the nonregulatory alternatives are perfect, imperfection is not justification for "dismissing that alternative as a failure," and that "the relevant question is whether or not these alternatives—on the whole—promote workplace safety, and to what extent they do so." OSHA fully agrees with these statements. Certainly the alternatives, when followed, do promote workplace safety, but the extent to which they do so may not be sufficient. The 1988 sample survey showed that, among firms where there are chemical exposures, 20 percent would not be in compliance with the new standards and less than 15 percent of firms making or using hazardous substances, did exposure monitoring. OSHA believes these facts reflect the market's failure to better control exposures to hazardous substances.

The situation of imperfect information is also questioned by NGFA. They cite the availability of information from various sources (news media, labor unions, local public interest organizations, plaintiffs' attorneys), stating "all of whom—for their own reasons—aggressively spread the word about substances that may present occupational health risks." The comments and testimony received for this rulemaking present an ideal example of the problems of information. First, the information is often presented in a selective manner precisely because the presenters are working "for their own reasons." The NGFA maintained in the hearing that there are no substantive hazardous exposure problems at grain elevators and has sued OSHA to try to prevent the hazard communication standard from informing their workers of any risk. Yet grain dust has been known to cause disease since at least 1713 and most, if not all, impartial expert organizations have concluded it does cause disease. Second, the sheer volume of information in some cases is overwhelming. Third, the highly technical nature of much information makes analysis extremely difficult, except for the specialist. For example, the industrial structure of grain handling is segmented among several major industry categories (SICs). Information from one subcategory can be inadvertently misrepresented to apply to all grain handling facilities. The combination of these factors can make analysis of information, even when it is available, very difficult or inconclusive.

NGFA asserts that the tort system provides better recourse for employees than OSHA admits. As just discussed, the tort system provides only an imperfect remedy. The employees' damages are restricted under Workers' Compensation and it is difficult to prove causation. The only possible defendant is the supplier, not the employer. This does not encourage employers to take precautions. The greatest problem with the tort system is that torts are a retroactive remedy, after illness or death, whereas OSHA has a responsibility to assure, to the extent feasible, that no employee will suffer material impairment of health or functional capacity prospectively. Although the threat of a tort may help to prevent health damage to employees, it remains more a form of compensation for injuries suffered than a preventive measure.

NGFA contends that, "A profit-maximizing employer certainly will incorporate those additional costs (insurance, hiring, training, goodwill) in its consideration of necessary safety measures." OSHA agrees that, in the ideal, employers and manufacturers would provide a high level of safety and health protection to their employees.

This is not, however, reflected everywhere in reality. Long-term implications of safety and health problems are often ignored or underestimated in the pursuit of short-term profits.

OSHA, therefore, does no agree with the NGFA's arguments and continues to believe that there are no nonregulatory alternatives that adequately protect workers from the adverse health effects associated with exposure to the chemicals regulated in this final standard.

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F. Technological Feasibility

Feasibility Determination

This chapter presents a technological feasibility analysis of industry's ability to meet OSHA's proposed permissible exposure limits (PELs) for a wide range of occupational health hazards. These PELs would include limits on airborne concentrations of substances, and in some instances, direct contact of the skin with the substance.

The control of workplace exposures to toxic chemicals involves combining a variety of standard techniques to solve a situation-specific problem. OSHA believes that existing engineering controls are available to reduce exposure levels to the new levels.

In reviewing the comments and hearing testimony on the technological feasibility of achieving the PELs and other limits, OSHA has found that for the overwhelming majority of situations where air contaminants are encountered by workers, compliance can be achieved by applying known engineering control methods and work practice improvements. It is recognized, however, that in some circumstances, respiratory protection may be necessary.

Types of Controls

In general, three basic types of controls may be employed to reduce employee exposures:

- Engineering controls
- Work practices and administrative reforms
- Personal protective equipment

Consistent with OSHA regulations and policy, this chapter examines the feasibility of engineering controls and work practices to control employee exposure, in preference to personal protective equipment.

Engineering Controls. Engineering controls involve the use of local exhaust ventilation, general ventilation isolation of the worker and enclosure of the source of emissions process modifications equipment modifications and substitution of non-hazardous chemicals. These methods may be used alone or in combination of any two or more controls depending upon the needs of a specific situation. Variations in situations usually result from the type of process being used and the number of chemicals in the air. However, these controls are considered standard techniques which will effectively control these variables either by themselves, or coupled with changes in work practices.

Ventilation. Perhaps the most widely used technique for controlling chemical exposures is the use of ventilation. General ventilation uses the movement of air within the general work space to displace or dilute the contaminant with fresh outside air. General ventilation is not typically the preferred control method in most operations due to the large volumes of air movement required. Local exhaust ventilation uses much smaller volumes of air, exhausted from the point at which contaminants are generated to remove the contaminant at the source.

Isolation. Isolation involves placing a physical barrier between the hazardous operation and the worker. Many modern, automated manufacturing processes are now fully enclosed in ventilated cabinets. The effectiveness of such a control technique depends on the frequency with which the workers have to enter the enclosure during normal operations. In other situations, rather than placing the process or machine in an enclosure, the worker may be put into a controlled atmosphere enclosure. Many processes which involve potential chemical exposures are operated remotely by operators in air conditioned booths.

Substitution. Substitution refers to the replacement of a toxic chemical in a particular process or work area with another, less toxic product. Properly applied, substitution can be a very effective control technique. However, care must be taken to ensure that the proposed substitute performs in a similar manner to the product being replaced. In addition, it is essential that the substitute be carefully evaluated to ensure that in controlling one hazard, another different hazard is not inadvertently introduced. The substitute must also be compatible with existing manufacturing equipment and processes.

The success of these techniques will depend on the physical properties of the chemicals and emissions encountered (boiling point, vapor pressure, etc.) and the process operating conditions (temperature, pressure, etc.). In some cases, particularly with cleaning solvents, substitution may provide the quickest and most effective means of reducing exposure. In other situations where particular physical or chemical properties are required, major effort may be required to alter processes or install or expand local or general dilution ventilation. The extent to which engineering controls may be effectively used will vary from industry to industry, as well as plant to plant within an industry.

Work Practices and Administrative Reforms. Work practice controls include housekeeping procedures, material handling or transfer procedures, leak

detection programs, training and personal hygiene. In many cases, it is possible to bring about substantial reductions in employee exposures by applying work practice controls.

Personal Protective Equipment. Where it is impractical to apply engineering or work practice controls, or where their application will not consistently reduce employee exposures below the proposed PELs, personal protective equipment such as respirators, may be used to prevent and reduce exposures.

Industry Engineering Controls

To determine whether engineering controls and work practices can reduce employee exposures to the proposed PELs, OSHA, through its contractors, examined typical work processes found in a cross section of industries. Using this list, industry experts identified which major processes had potential hazardous exposures and may require additional engineering controls or different work practices in order to achieve the proposed PELs. To assess whether these would be feasible for the processes within the industry group, records maintained by OSHA and NIOSH were searched to identify examples of the successful application of controls to these processes. Based upon the judgments of the industry experts, a determination was made as to the probable feasibility of achieving the proposed PELs. A list of the processes and control measures is set out in Table V–4 at the end of this chapter.

This chapter presents examples of feasible methods of controlling exposure to hazardous substances encountered in processes used in the SICs for which costs and benefits have been identified. Unit costs for these or similar controls were used as the basis for the cost projections in Chapter V. In addition, this chapter summarizes the docket entries regarding technological feasibility.

Information from commenters to the docket or statements at the hearings indicate that for the vast majority of firms, the proposed PELs can be met using engineering controls alone. In the few isolated cases it is recognized that respiratory protection must be added to engineering controls to assure worker safety.

SIC 20—Food and Kindred Products

A major air contaminant in the food processing industry is carbon dioxide (CO_2). A milk products plant (SIC 2023) controlled carbon dioxide exposures by using a hood which fully enclosed the chiller-conveyor line and exhausted air from the system to an exterior baghouse. Carbon dioxide levels resulting from the use of dry ice were controlled at a meat packing plant (SICs 2011 and 2013) by a stainless steel exhaust hood. Similarly, a poultry dressing plant controlled carbon dioxide emissions by using a slotted hood exhaust ventilation system. A food processing plant (SIC 202) controlled carbon dioxide exposure by increasing the number of air changes in the packaging room.

OSHA is adopting a limit of 10,000 ppm as an 8-hour TWA for CO_2 and is supplementing this limit with a 15-minute STEL of 30,000 ppm. The Beer Institute [Ex. 49, 142, Tr. 8/9/88, p. 9–26] and the Brewing Industry Safety Advisory Committee submitted comments to OSHA on carbon dioxide. The industry argued at the public hearing and in docket submittals that the 8-hour TWA limit of 5,000 ppm for CO_2 was "unnecessarily low and restrictive" [Ex. 49, Tr. 8/9/88, p. 9–27]. According to the Beer Institute, the brewing industry "is unique relative to carbon dioxide exposure and control . . . no other industry faces the same engineering difficulties for controlling ambient carbon dioxide as the brewing industry" [Tr. 8/9/88]. No details explaining these difficulties were provided by these commenters.

Monitoring data taken on employees in one brewery, together with a description of the operations that cause the most exposures, are contained in a study of cellar workers [Riley and Bromberger—Barnes, 1979]. The data include samples taken over 14 eight-hour shifts. Eight-hour TWA exposures ranged from 0.5 percent (5,000 ppm) to 1.41 percent (14,100 ppm), with a mean of 1.08 percent (10,800 ppm). Data on "maximum acute exposure" were also provided. The period of maximum acute exposure ranged from 2 minutes to 240 minutes. Of the 14 samples, three exceeded a 3-percent (30,000 ppm) 15-minute STEL.

Exposures result from a build-up of CO_2 in large fermentation tanks during the beer fermentation process. These tanks are sealed systems; the CO_2 is normally piped away. Two circumstances were identified by commenters as causing CO_2 exposures. First, if excessive pressure builds up, an escape valve blows. The concentration of CO_2 in the vicinity of such a blow-out was measured at 60 percent (600,000 ppm), although the level in the area fell to 12 percent (12,000 ppm) within a few minutes. Such blow-outs are reportedly rare. The second, and routine source of CO_2 exposure is the opening of tank doors and the entry of workers into the tank to flush out sludge that remains after the tank has been drained. After opening the doors which are near the floor and open onto the central corridor, the cellar worker leaves the area until most of the CO_2 has been ventilated. The principal exposures to CO_2 in the beer industry thus involve either upset conditions (a blow-out) or maintenance activities (entry into the tank to clean it). For both of these circumstances, OSHA routinely permits the use of respiratory protection. Exposures in the corridor (resulting from the opening of tank doors) could be further controlled by the work practice of cracking the door and waiting longer before reentering the area or by adding local exhaust ventilation to capture the CO_2 escaping from the doors.

OSHA notes that commenters from the brewing industry supported the Agency's proposed STEL for CO_2 of 30,000 ppm [Tr. 8/9/88, p. 9–31], and advocated an 8-hour TWA of 10,000 ppm. In adopting 10,000 ppm as the 8-hour TWA and adding a 15-minute STEL of 30,000 ppm, the Agency believes that feasibility problems in this industry sector will be alleviated.

Grain dust exposures in this sector occur during grain handling operations in facilities that mill grain either for human use, e.g., flour mills and rice mills or, more commonly, for animal use, e.g., feed mills [Ex. 3–752, p. 10]. There is general agreement that the highest exposures in all types of grain-handling facilities occur during grain receiving operations [Ex. 3–752; Tr. 8/10/88, p. 10–46]; the grain receiving process is the same, regardless of the type of facility in which it occurs. OSHA proposed a level of 4 mg/m^3 for grain dust; because of feasibility considerations, the final standard is 10 mg/m^3.

Many commenters stated that a PEL of 4 mg/m^3 was not achievable, particularly in older mills [Exs. 3–63, 3–110, 3–237, 3–299, 3–405, 3–752, 3–755; Tr. 8/10/88, pp. 10–45/10–48; 10–50/10–54; 10–55/10–60; 10–61/10–70]. Industry representatives stated that current employee exposures to grain dust in mills often exceeded the proposed limit [Tr. 8/10/88, pp. 10–63, 10–46; Ex. 180]. For example, David Bossman, representing the American Feed Industry Association (AFIA), reported that "just over half [of 69 samples taken in 10 mills by the AFIA] exceed the proposed PEL. The average exposure was 10.9 milligrams per cubic meter" [Tr. 8/10/88, p. 10–46]. Mr. Bossman also stated that exposures in the bulk receiving areas of all 10 mills sampled exceeded 4 mg/m^3 and averaged 12.9 mg/m^3 [Tr. 8/10/88, p. 10–46].

According to a 1984 study by the T.E. Stivers Organization, 15 of 20

representative mills visited "had no dust control systems at all, and [the remaining] five had some dust control systems, but [these were] not comprehensive in scope" [Tr. 8/10/88, p. 10–63]. According to Gary Winsett, President of Winsett Engineering, Inc., an independent engineering firm that specializes in the feed, grain, and related agribusiness industries: "three separate control systems would be required in the main work areas of each mill" to bring 13 of the 20 mills included in this 20-mill survey down to the 4 mg/m^3 level of control, and six of these 13 mills would require "relatively extensive dust control systems in the receiving areas" to achieve the 4 mg/m^3 limit [Tr. 8/10/88, p. 10–63]. In cases where such controls are in place, however, Mr. Winsett reported that exposures had been reduced considerably [Tr. 8/10/88, p. 10–62]. In older mills, retrofitting has been successful in reducing grain dust exposure levels. For example, John Wolgemuth, Corporate Safety and Loss Control Manager for Agway, a farm supply and food marketing cooperative owned by 102,000 farmer-members, described the results achieved in one mill in which additional exhaust hoses had been installed. According to Mr. Wolgemuth, levels were reduced from above 15 mg/m^3 to below the 10 mg/m^3 level by retrofitting [Tr. 8/10/88, pp. 10–50/10–51].

In an effort to obtain information on conditions in small, rural mills, OSHA reviewed the docket developed in connection with the Agency's recent grain-handling standard [Docket H–0117]. A study performed by Dr. Buchan of Colorado State University reported that, in eight small grain elevators and feed mills in his state, 10 percent of exposure samples were above the 4 mg/m^3 proposed limit (Attachment 1, Ex. 3–751, Docket H–0117).

There are a variety of dust controls in use in grain mills at the present time. Dust collection systems, including pneumatic dust controls, are the most widely available and useful methods of controlling grain dust in mills in which dust is a problem [Ex. 3–752, p. 17; Tr. 8/10/88, p. 10–62]. A dust collector typically consists of a motor-driven fan, which creates the air flow necessary to capture dust particles and carry them through duct work to a dust collector. These aspiration systems are an "effective method of controlling dust emissions. Aspiration of the leg consists of a flow of air across the entire boot, which entrains the liberated dust and carries it up the up-leg to take-off points" (52 *FR* 49592, December 31, 1987). Depending on baseline levels of exposure, several collectors may be needed in a mill.

A second method of controlling dust that is becoming widely used is the application of oil mist to the grain to minimize dust generation. This oil mist, which consists of mineral oil, vegetable oil or some combination of the two, is normally applied when the grain is received at the mill. Ralph Mourer, testifying for the AFIA, stated that oil suppression of dust is a promising control that he has just installed in his feed mills. Although he has not yet had much experience with the system, he noted "people I've talked [to] and discussed the system with are very pleased" [Tr. 8/10/88, p. 10–78]. In an earlier study of grain handling facilities for OSHA, however, Arthur D. Little Inc. noted that there are some limitations to the use of this method:

Mineral oil is not approved for use as an additive on food grades of grain by the U.S. Food and Drug Administration. Vegetable oil may be an allowed additive, but its use can cause the grain to adhere into masses in cold climates. Further, there is concern that the oil will become rancid or create a commercially objectionable odor (Docket H–0117, ADL, p. VI–34).

Scott Bjornsom from Hunter Grain in North Dakota also reported that oil suppression cannot be used for malting barley "because of the absorption with the water in the malt process" [Tr. 8/10/88, p. 10–85]. Despite some limitations on its use, oil suppression appears to be an effective control.

A third control method that can be used in facilities with high dust exposures is the use of vacuum systems in place of manual sweeping or compressed air blowing during clean-up operations. A Canadian study [Farant and Moore, "Dust Exposures in the Canadian Grain Industry," *American Industrial Hygiene Journal*, March 1978] of grain elevators found that many very high exposures to grain dust were a result of dust raised during housekeeping operations that involved brooms or blowers [Page 193, Attachment to Ex. 3–751, Docket H–0117]; this study concluded that "the use of in-plant vacuum systems would reduce these exposures." Representatives of the AIFA reported that mills have switched to vacuum systems to control their employees' exposures during clean-up [Tr. 8/10/88, p. 10–78].

Industry representatives also described the filtration systems that are being installed on aspiration systems in feed mills; these systems are being installed in many areas of mills, expecially in the loading and unloading areas, where the highest exposures to grain dust occur [Tr. 8/10/88, p. 10–73]. In the past, unfiltered cyclones were in widespread use in feed mills [Tr. 8/10/88, p. 10–84].

To deal with the problem of grain dust in older mills, many owners are replacing the old-fashioned wooden legs with "good, tight, enclosed steel legs . . . [and in] old facilities . . . [that had] open grain drag conveyors . . . the conveying systems that used to be open have lids on them . . . to keep the dust where it belongs" [Tr. 8/10/88, p. 10–80]. Enclosure of this type is a standard and recommended industrial hygiene practice in all dusty environments.

OSHA notes that much of the control and exposure data relied on by AFIA representatives at the hearing, such as the 1984 Stivers study of the representative group of 20 feed mills, predates the promulgation of OHHA's grain handling facility standard; the Agency believes that many facilities in this sector are in the process of replacing outdated equipment, retrofitting existing equipment, and "tightening up all connections" throughout the mill [Tr. 8/10/88, pp. 10–82/10–83]. This is confirmed by industry representatives, who reported that these efforts are being undertaken in response to the OSHA standard, their insurance companies' suggestions, and industry concerns about dust levels [Tr. 8/10/88, p. 10–73]; according to industry representatives, these controls have reduced fire risks [Tr. 8/10/88, p. 10–74], improved productivity and quality, and led to better working conditions [Tr. 8/10/88, p. 10–81].

OSHA's review of all of the evidence in the record indicates that 10 mg/m^3 is a feasible limit in the grain and feed mill sector. The final rule includes this PEL as an 8-hour TWA; the Agency finds that the health evidence (see Section XIC of the preamble to the final rule) demonstrates a significant risk of material health impairment above this level. OSHA finds that feed mill employers will be able to achieve the 10 mg/m^3 limit in cases where exposures remain above 10 mg/m^3 [see [Tr. 8/10/88, p. 10–46] or where older mills are involved [see Tr. 8/10/88, p. 10–63] using any of a variety of controls; oil mist suppression in feed mills, aspiration systems (with or without filtration), enclosure of open conveyors and other grain-handling equipment, and the use of vacuuming in lieu of blowing or sweeping during cleanup. For some mills that are close to this limit at the present time, OSHA believes that the general "tightening up" described by Mr.

Wohlgemuth [Tr. 8/10/88, pp. 10–82 to 10–83] will be sufficient.

The International Institute of Ammonia Refrigeration (IIAR) argued that the proposed levels for ammonia (25 ppm TWA, 35 ppm STEL) would be viewed as a nuisance because most people cannot detect the odor of ammonia at 35 ppm. As such, employees would neglect proper control measures [Ex. 113]. David G. Kramer of Kahn's and Company [Ex. 113] stated that "No one in our plants is exposed to continuous exposure to 35 ppm ammonia concentrations".

One control approach for ammonia gas encountered in poultry processing (SICs 2016 and 2017) required the appropriate placement of cut-off valves to freezer coils and the use of an alarm detection system to monitor ambient air conditions.

Ammonia-based refrigeration systems are commonly used in the meat products industry. Commenters expressed concern that "ammonia based refrigeration systems . . . are subject to occasional leaks which may result in short-term high level exposures" [Exs. 3–897, 3–750]. The situation referred to by these commenters is an intermittent maintenance or upset condition, for which OSHA permits the use of respirators. In addition, a representative of the Food and Allied Services Trade Department of the AFL–CIO stated that two companies, Wilson Foods and Morrell, evacuate the workplaces if ammonia levels reach 25 ppm as a ceiling [Tr. 8/4/88, p. 311]. In addition, a representative of the Food and Allied Services Trade Department of the AFL–CIO stated that two companies, Wilson Foods and Morrell, evacuate the workplace if ammonia levels reach 25 ppm as a ceiling [Tr. 8/4/88, p. 311]. OSHA concludes that there is no issue of technical feasibility in regard to the proposed STEL of 35 ppm for ammonia and the 35 ppm STEL is retained in the final rule.

Chlorine is used extensively as an antibacterial agent in meat products plants to comply with USDA sanitation and microbiological contamination requirements. Commenters did not raise the issue of technical feasibility in regard to the proposed chlorine standard itself. Commenters did, however, express concern that a 0.5 ppm STEL for chlorine may be too stringent to allow compliance with USDA regulations [Exs. 3–756, 3–897], although no data to support this concern were provided. Responding to these concerns, OSHA has established a 0.5 ppm PEL and 1 ppm STEL for chlorine in the final rule.

Carbon disulfide itself is not used in the meat products industry, although it is a key solvent used in the manufacture of cellulosic food casings, which are used in the manufacture of processed meats. Suppliers of cellulosic food casings stated that a carbon disulfide standard of 1 ppm cannot be met in the production of such casings [Exs. 3–421, 3–633, and 3–896]; if this were the case, according to these commenters, domestic supplies of cellulosic casings would cease. Foreign supplies would gradually penetrate and supply the market for cellulosic food casings [Tr. 8/2/88, pp. 4–209, and 4–261]. OSHA concludes that there is no apparent issue of technical feasibility of the proposed carbon disulfide standard in SIC 20. However, the TWA for this substance has been increased to 4 ppm, in part, in consideration of the potential industrial displacement effect.

The National Cotton Council of America (NCCA) submitted a comment to the effect that the approximately 50 cotton mills in SIC 2074 would be adversely affected by the proposed limit for n-hexane and other hexane isomers, vegetable oil mist, and grain dust [Ex. 3–1080]. NCCA stated that its members would have difficulty measuring airborne concentrations of these substances because cottonseed mills are small, rural business without in-house industrial hygiene capability. OSHA notes, however, that methods are readily available to measure these airborne contaminants; an appendix to the final rule contains information on appropriate sampling methods for these substances. The Agency has responded to industry concerns by dropping its proposed 10 mg/m^3 STEL for oil mist but retaining the 5 mg/mg^3 TWA.

Sulfur dioxide (SO_2) exposures in the wet corn milling industry as a result from soaking of cleaned corn kernels in large vats (known as steep vats) for 30 to 50 hours. The purpose of the steeping process is to soften the corn in preparation for further grinding, screening, and centrifugal operations. This steeping process takes place in a dilute sulfur dioxide solution (sulfurous acid) [Tr. 8/15/88, pp. 9–10]. Worker exposures occur when sulfur dioxide is released from solution in the steeping tanks. The principal controls available to reduce exposures to sulfur dioxide are local exhaust ventilation, the use of isolated control rooms, process enclosure achieved by the use of closed stainless steel tanks, enclosed screening systems, and general automation [Tr. 8/15/88, pp. 8–77 to 8–78].

Exposure data for this segment of SIC 20 are sparse, except for data from a study conducted by the CRA in five of its member plants in 1977 in connection with the Agency's earlier SO_2 rulemaking. Eight-hour TWA samples were taken on 213 workers exposed to SO_2 in wet corn milling and on a group of 344 non-SO_2-exposed workers from other parts of the plant [Ex. 65, Tab 9]. (The "background" SO_2 level even for the controls, however, was determined to be 0.33 ppm (8-hour TWA).) The median exposure in the SO_2 group was 2 ppm; 15 percent of all workers were exposed above 5 ppm. No STEL measurements were taken. Exposures (8-hour TWAs) ranged from 0.1 to 10.8 ppm. According to industry sources, these results "represent worst-case" exposures because they were obtained during the winter months, when the plants' windows and doors were closed [Ex. 65, Tab 9, p. 7]. More recent exposure data, described at the hearing as "non-systematic" and variable in "sampling efforts, methods and results," were summarized as follows:

> While many plants report 1987–88 personal sampling results in the range of 2 ppm, even plants in that category are not below 2 ppm consistently, and a large number of employees are still exposed in the range of 4 to 5 ppm.

Industry representatives at the hearing indicated that opening the doors and windows even when there was only a 5-mph breeze outside increased the effectiveness of in-plant ventilation by a factor of five [Ex. 65, Attach. D, pp. 2392–2397]. In addition, testimony indicates that the higher 8-hour TWA readings and those above 10 ppm were caused by "emergencies," "pipes breaking, or the process getting out of control, the tank . . . [overflowing] as a pump seal breaks, or something of that sort" [Ex. 65, Attach. D, pp. 2314, 2315, 2319]. Testimony also indicates that many of the sampling results reported above were taken on maintenance workers, who are personnel dedicated to maintenance functions [Tr. 8/8/88, p. 8–85].

Industry representatives reported that major improvements in SO_2 exposures could be achieved:

> Plants vary widely in age, the degree of natural ventilation available, the degree to which their process is entirely closed, the location and source of the sulfur dioxide they use in steeping, the amount of local exhaust equipment already in place, the extent to which control rooms isolate the operator from the process, and various other factors [Ex. 65, Attach. F, pp. 35, 37].

Some equipment, such as the "steeps" or soaking tanks, are more than 40 years old; some of these tanks are still the wooden staved steeps of years ago [Ex.

65, Attach. D, p. 2324]. One company has milling plants that range from 30 to 97 years in age [Ex. 65, Attach. D, p. 2324]. Spokesmen reported that the industry's efforts to modernize plants has not resulted in appreciably lower employee SO_2 exposures because improvements in engineering controls, i.e., ventilation, have not kept up with increased production [Ex. 65, Tab 13, p. 7]. An OSHA-sponsored study performed by JRB Associates for the previous SO_2 rulemaking found that plants in this sector could achieve the 2-ppm TWA and 5-ppm STEL with the expenditure of a relatively small amount of money [Ex. 65, Attach. D, pp. 2322–2324].

There are no sampling data in the record relating to the 5-ppm STEL for SO_2, because the CRA-sponsored exposure survey undertaken in 1977 contained no STEL sampling results. The recent record [Ex. 65, Tab 13, p. 7] states simply that:

> Short term exposures, especially for maintenance job functions, can be considerably higher than 4 to 5 ppm [Ex. 65, Tab 13, p. 7].

OSHA notes, however that the wet corn milling process is a steady-state process:

> The process . . . is rather level as far as the [SO_2] concentration is concerned with the exception of emergencies, pipes breaking, or the process getting out of control . . . (Ex. 65, Attach. D, p. 2314–2315) . . . [except] for maintenance emergency problems, the exposure to sulfur dioxide in the process is fairly constant.

OSHA finds that the 2-ppm 8-hour TWA and the 5 ppm 15-minute STEL for sulfur dioxide are technologically feasible in the wet corn-milling process.

(1) In 1977, 50 percent of all SO_2-exposed employees had exposures at or below 2 ppm; because the sampling results for dedicated maintenance employees are contained in these numbers, the actual percentage is greater than 50 percent for non-maintenance workers;

(2) Most of the sampling results from the high end of the 0.8 to 10.8 ppm range of exposures cited by the CRA occurred during process upset or maintenance operations;

(3) The 1977 CRA sampling results were "worst case," so the number of overexposed employees is overstated; respirators are permitted in these operations;

(4) Because most exposures are already below 2 ppm, little in the way of additional control will be needed (note that opening the windows increased the efficiency of ventilation by a factor of 5, indicating that additional make-up air would do the same);

(5) STEL exposures are not a problem because the wet milling process, except when it is not being adequately controlled, is characterized, according to industry representatives, by relatively constant, non-fluctuating ambient concentrations of SO_2. Because most exposures are already below 2 ppm and the overwhelming majority are now at or below 5 ppm, the STEL has essentially been achieved in this sector. That is, in a steady-state exposure environment where 8-hour TWA exposures are below 5 ppm, 5 ppm STEL exposures are not a problem. In wet corn milling, short-term exposures are a problem only in maintenance and emergency operations; in both cases, respirators are both permitted and encouraged by OSHA.

(6) Where exposures are above 2 ppm, they are only slightly above 2 ppm. Minor upgrades in ventilation and some modernization of the oldest equipment will reduce exposures below 2 ppm.

SIC 21—Tobacco Products

Tobacco dust and residual pesticide dusts created during cutting and shredding operations have been reduced through the use of local exhaust ventilation. This has also been used to control emissions of ethyl alcohol-based chemical flavorings during blending operations.

There were no comments submitted to the docket for this sector.

SIC 22—Textile Mill Products

Textiles are dyed at various stages in their manufacture, including unspun fibers, unwoven yarn, and finished fabric. Workers who prepare fabrics from unspun fibers are of particular concern, since they could be potentially exposed to dyes contained on dusts generated during manufacture. In addition, some dyes possess much poorer fastness to wet treatment than do others; persons who launder such clothing are potentially exposed to the dyes. Stringent control measures and work practices can prevent such exposure.

Several generally acceptable practices for the control of hazardous materials can be used wherever there is the potential for exposure. For example, pressure failure alarms for closed systems and exhaust ventilation can rapidly indicate a system failure that might result in the release of substantial quantities of dyes. Continuous flow indicators, such as water or oil manometers properly mounted at the juncture of a fume hood and duct throat and marked to indicate acceptable airflow, will give a readily observable indication of decreased efficiency in the ventilation system for the hood. Wet methods, vacuum cleaning, or other methods that do not lead to redispersion of settled dust should be used for plant maintenance and sanitation. Dry sweeping or blowing with compressed air should be prohibited.

Evidence presented in the docket suggest that controls necessary to meet the proposed standards have already been installed at many facilities. The American Textile Manufacturers Institute, Inc. (ATMI), representing 85 percent of the industry's manufacturing capacity, reported that "member companies generally try to meet the ACGIH TLVs for both those chemicals which are regulated by PELs and those which are not. Because the ACGIH TLVs are annually reviewed and revised, ATMI's member companies believe compliance with these voluntary standards has led to safer and healthier workplaces for their employees" [Ex. 3–434].

The National Cotton Council of America [Ex. 3–1080] reported that "Textile manufacturers generally try to use the existing ACGIH TLVs as guidelines for good practice to provide a safer and healthier workplace for their employees." Their comments state that some of the levels are difficult to attain, but are said to be feasible.

SIC 23—Apparel

Chemical exposures in the apparel industry occur principally as a result of three exposure sources: spot cleaning, dry cleaning and contact with treated fabrics.

Spot cleaning and dry cleaning operations exposures to perchloroethylene can be controlled with the use of local exhaust ventilation and general ventilation. Work practice improvements help reduce solvent exposure during transfer operations. Routine scheduled maintenance can be used to detect and control leaks from door gaskets and seals. Contact dermatitis is reduced through the use of disposable gloves and adherence to a personal hygiene program.

A detailed discussion of perchloroethylene is presented in SIC 72.

SIC 24—Lumber and Wood

The primary worker exposure in the lumber and wood industry is wood dust. For the operation of large equipment (e.g. in debarking and sawmill activities), the operator can be placed in an enclosed control booth, or in the case of moving equipment (e.g. cherry pickers, loaders and cranes), the operator can be located in an enclosed cab. In both cases, air would be filtered and conditioned. In the case of felting or matting process lines, or such equipment as belt sanders, the equipment can be enclosed or hooded and vented to a baghouse. For smaller equipment, such as variety saws, tenoners, and dovetailers, hoods or various types of negative pressure (or combinations of positive and negative pressure) local ventilation devices can be used to

control wood dust. In the case of hand-held sanders, a vacuum system can sometimes be applied to the process. Some other wood dust generating equipment can also be enclosed (e.g. planers), but this is generally done for noise control.

The technical feasibility of a 5 mg/m^3 PEL for wood dust was challenged, indirectly, by only one commenter to the record. The American Furniture Manufacturers Association [Ex. 3-917], after speaking of the general technical feasibility of the proposed standard and the difficulty of controlling wood dust around some machines, stated: "Other machines are so complicated (such as multiple spindle boring machines and multiple spindle carvers) that no effective collection system has yet been defined." OSHA disagrees and concludes that exposures on these machines can be controlled. Included in the documentation of the site visits conducted for this rulemaking [Ex. 11] is at least one case of a multiple head boring machine which was equipped with local exahust ventilation and a multiple spindle "trim, bore and dowel" machine also equipped with local exhaust ventilation. TWA exposures to wood dust for the operators of these machines were 1.0 and 0.4 mg/m^3, respectively.

Vast numbers of commenters expressed their support for a 5 mg/m^3 PEL for wood dust without any question of technological feasibility. A few examples follow. Appalachian Hardwood Manufacturers, Inc. [Ex. 3-626] stated that, although they felt it would be expensive, "To bring all our mills into compliance with a five milligram per cubic meter standard would be technically feasible." Monadnock Forest Products, Inc. [Ex. 127, Attachment C and Tr. 8/12/88, p. 216] states that "5 mg/m^3 is technically feasible but due to cost it should be phased in over a number of years." The National Dimension Manufacturers Association [Ex. 3-1160] commented: "Achievement of a 5 mg/m^3 permissible exposure limit [for wood dust] is believed to be technically feasible. . . ." Others at the hearings supporting the adoption of the 5 mg/m^3 level included David Smith of Willamette Industries [Tr. 8/12/88, p. 369] and Charles Carey of Ross Associates [Tr. 8/12/88, p. 411]. Whirlpool Corporation [Ex. 3-824] provided exposure data for a sanding work station, before and after the installation of control equipment. Exposures before the ventilation equipment was installed ranged from 13.0 to 29.6 mg/m^3. With the equipment in place, exposures ranged from 0.88 to 3.16 mg/m^3.

Two surveys cited by Mr. Scott Schneider of the Workers' Institute for Safety and Heath [Tr. 8/15/88, p. 6 and Ex. 115, Attachment A] also support the feasibility of the 5 mg/m^3 PEL. A 1986 OSHA Health Response Team survey showed that "two thirds of the personal samples were below two milligrams per cubic meter and over 40 percent were below one." Twelve of the 23 plants in a 1985 survey by Haliday Associates in Ontario, Canada, had no exposures over five milligrams per cubic meter and two of the plants, one of which was a furniture plant, had no exposures above one milligram per cubic meter.

Exposure data from the Clayton Environmental Consultants' study for the Inter-Industry Wood Dust Coordinating Committee was cited by Mr. Michael Coffman at the informal public hearings [Tr. 8/12/88, p. 99]. Mr. Coffman stated: "Within SIC code 24, we collected a total of 676 dust measurements. Eight percent of these were found to exceed five milligrams per cubic meter; 30 percent exceeded one milligram per cubic meter. Within SIC Code 25, 107 total dust measurements were collected. Fifteen percent of these exceeded five milligrams per cubic meter, 40 percent exceeding one milligram per cubic meter. Within SIC Code 26, a total of 19 measurements were collected, five percent exceeding both one and five milligrams per cubic meter."

Exposure data presented by machine type in the Clayton study [Ex. 127A], and shown below in Table F-1, provide clear evidence that the 5 mg/m^3 level can be attained for many machines by using local exhaust ventilation. These data demonstrate that exposures of operators at these machines can be uniformly controlled. Table F-2 presents data showing the effectiveness of ventilation and air-conditioned booths for other machines.

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Table F-1

EXPOSURES TO WOOD DUST FOR MACHINES
EQUIPPED WITH LOCAL EXHAUST VENTILATION

	Number of Exposure Measurements	
Machine	Total	Above 5 mg/m^3
Bandsaw-finish	7	1
Cut-off saw	33	1
Gang rip saw	15	2
Rip saw	22	0
Variety saw	15	1
Belt sander	41	3
Hand-held sanders	8	
Molder	17	0
Planer	16	2
Router	4	0
Shaper	9	1
Tenoner	22	5

Table F-2

EXPOSURES TO WOOD DUST FOR MACHINES
WITHOUT CONTROLS OR WHERE OPERATOR IS ISOLATED
IN AIR-CONDITIONED BOOTH

	Number of Exposure Measurements		
Machine	Total	Above 5 mg/m^3	Controls
Drilling and Boring	7	1	No controls
Sorter/Stacker	12	1	No controls
Chipper	12	1	No controls
Dryer	7	0	No controls
Veneer Clipper	4	0	No controls
Hot Press	5	0	A/C booth
End Loader	6	0	A/C booth
Felter	3	0	A/C booth
	5	0	Ventilation
	4	1	No controls
Bandsaw-Sawmill	8	0	A/C booth
	4	1	Ventilators
	10	2	No controls

BILLING CODE 4510-26-C

A number of commenters also estimated what their costs of compliance would be if a 5 mg/m³ standard were adopted (specific examples are addressed in Chapter G). Estimating the costs implies the technical feasibility of meeting the standard. All of the foregoing is evidence that control of wood dust at or below 5 mg/m³ is technologically feasible.

Washington State has adopted exposure limits of 2.5 mg/m³ for western red cedar and 5 mg/m³ for other woods. According to Mr. Stephen Cant of the Occupational Health Program for the State of Washington, ". . . in our process of adopting this specific wood dust standard the industry presented absolutely no comment in terms of concerns regarding the limits" [Tr. 7/29/88, p. 102]. Mr. Cant also states: ". . . Industry has been comfortable with [these] limits in the northwest. We find that they can, in fact, in most cases comply with those limits, ..."

A study conducted by the University of Washington Department of Environmental Health [Ex. 127H] provides recommendations on achieving the 2.5 mg per cubic meter level, as well as some exposure data. The report notes that average exposures for shake saw operators in the mills surveyed were 1.63 mg/m³. The range of exposures for other mill workers (splittermen, deck hands and packers) ranged from 0.22 to 2.7 mg/m³. Shingle saw operators were the only workers routinely exposed at levels above the 2.5 mg/m³ limit, with average exposures of 3.84 mg/m³. From this study: "The Washington exposure levels can be compared to levels in Canadian sawmills after ten years of a Canadian regulatory limit of 2.5 mg/m³. Cedar dust levels were studied in Canadian sawmills in 1985 by a researcher named Vedal. Total dust exposure in observed Canadian mills ranged from undetectable to 6.0 mg/m³ averaged over an 8-hour work day, with an average dust exposure of 0.21 mg/m³. Only 10 percent of workers were exposed to more than 1.0 mg/m³, and of these only 3.9 percent had exposures greater than 2.0 mg/m³ TWA." Included in the University of Washington report are a number of specific designs for local ventilation, baffles for shake and shingle saws and general recommendations on housecleaning.

SIC 25—Furniture

The feasibility of the standard for wood dust in this SIC is discussed in conjunction with SIC 24, above.

A review of processes in the metal office furniture manufacturing sector (SIC 2522), shows that air contaminants from the coating process have been controlled. Prefabricated sheet components for file cabinets are prewashed and coated with polyester or acrylic on a high speed conveyor line. The application process includes manual spraying of cabinets with airless atomizing sprayers and electrostatic spray guns on reciprocators. Manual spraying operations are performed in downdraft booths. Filtered fresh air is supplied through the open top of the booths and removed at the bottom through a water curtain by exhaust fans mounted on the roof of the booth. Spray headers in exhaust plenums clear paint mist from the air stream. Automatic spray booths contain electrostatic spray guns and side draft ventilation. Furthermore, organic solvent vapors in the paint mixing and storage room are controlled by equipping each drum with a heavy barrel cover, an integral agitator, sealed pipe openings, and a closeable access line.

Masco Corporation [Ex. 3-682]: "Methodologies for control of solvents from finishes in the woodworking industry are limited. . . . OSHA therefore has presented no feasible methodology for the woodworking component of the furniture industry to control solvent or potential gaseous air toxics." OSHA concludes that control is feasible. The control for such potential exposures is, in almost all applications of lacquers, varnishes, sealers and stains, a form of local exhaust ventilation control commonly referred to as a spray booth.

Spray booths are in wide use in the furniture industry and were seen in use at several of the plants on the site visits conducted for this rulemaking analysis [Ex. 11]. In each case the exposures were far below the PELs, and in several cases the solvent levels were not detectable.

A NIOSH study [TA 79-047-825] recommends that the exhaust opening in a painting room should be located as close to the painting operation as possible to take advantage of spot ventilation. The exhaust opening location should be such that overspray is directed into the opening. The exhaust outlet should be placed so that all the air used for ventilation passes through the zone of contamination. The air flow should direct all contaminants away from the painter's breathing zone and into the exhaust outlet.

Most welding in furniture manufacturing occurs in a fixed location where exposures to the various components of welding fumes can best be controlled with adequate local ventilation. Numerous manufacturers have available local exhaust systems and source collection/filtration systems that will control welding fumes. These systems typically consist of a fan and either a fixed hood or a jointed, flexible arm, up to fifteen feet in length, at the end of which is a small hood. The flexible arm allows the exhaust system to cover a large work area. Such systems operate at air volumes of 400 to 1000 cfm and can be exhausted to an existing centralized ventilation system, to the vicinity of general shop ventilation (e.g., a roof fan) or directly to the outside. A wide variety of off-the-shelf local exhaust systems and collection filtration systems are available, including portable models. Custom ventilation systems can also be installed.

SIC 26—Paper and Allied Products

Pulp mills occur primarily in SIC 2611, but can also be present as part of the operations in SICs 2621 and 2631. High control costs could potentially be incurred because of the larger quantities of chemicals used in breaking down pulp to form cellulose and the reactions that occur in the digesting process between these chemicals and the substances contained in the wood fiber. Large quantities of chemicals such as chlorine and sodium hydroxide are also used in the bleaching operations. The digesting and bleaching operations are also very extensive. Large quantities of pulp are generally produced from wood in these mills either for captive use or for shipment to paper and paperboard mills. The type of controls that would be used include ventilation, enclosure and/or process change, but less likely the latter. Various engineering controls have been used by the paper mill industry to prevent the mixing of toxic chemicals in sewer lines. Tanks containing the hazardous chemicals have been isolated and surrounded by dikes. Discharge lines have been re-routed to prevent accidental mixing. OSHA believes that feasible controls are available.

Wood dust can be generated in some processes in pulp mills in SICs 2611, 2621, and 2631. Workers may be exposed to wood dust from debarking and chipping operations, as well as in the wood yard. Exposures to equipment operators in the wood yard can be controlled by installing enclosed, air-conditioned cabs on the equipment. Debarker operators are frequently protected by isolution in air-conditioned booths. Exposure data for debarkers included in the Clayton study of the Inter-Industry Wood Dust Coordinating Committee show only 2 of 21 measurements above the 5 mg/m³ level. Data for chippers, also from the Clayton

study, suggest that controls are rarely needed for chippers: Only one of the 13 exposure measurements for chippers that had no controls in place was in excess of 5 mg/m³. Based on all of the above, OSHA concludes that controls are feasible for this industry.

SIC 27—Printing and Publishing

The technological feasibility for the proposed standards for toluene was challenged by R.R. Donnelley and Sons (Ex. 3–916). "Donnelley believes that compliance with the proposed 150 ppm ceiling will be infeasible during certain press operations and especially during cleaning." Donnelley further state that "if the 100 ppm standard is achieved at great cost through general ventilation improvements, compliance with the 150 ppm 15-minute ceiling would be impossible without the use of local ventilation or respirators." The main concern of this discussion becomes apparent by the following remarks. "If, as proposed by the regulation, respirators cannot be used six months after the effective date of the regulation, local ventilation is the only option. Given the number of presses in service at Donnelley's plants . . . the proposed 150 ppm ceiling will require, at a minimum, a maze of ventilation equipment which can be expected to cost millions of dollars." These statements reflect the costliness of achieving the proposed standards for toluene in this firm but acknowledge that engineering controls are feasible. The commenter apparently misunderstood the discussion (**Federal Register**, Vol. 3, 6/7/88, pp. 21241, 2), on the proposed length of time for firms to achieve compliance under the hierarchy of controls. OSHA also believes that there are substitute solvents available such as ethanol, ethyl acetate or nitroethane that could be used in cleaning operations or water-based inks not requiring major solvent use that could enable printing firms to achieve compliance with the final PELs. In addition, OSHA believes that engineering controls in the form of local exhaust ventilation are technologically feasible for press applications in the printing industry.

Site visits and monitoring were conducted to two printing establishments (Ex. 11—Firms 14, 45). One was a letterpress and lithographic operation and the other was a "quick-print" shop. In neither case were there overexposures to any of the following chemicals in use: Antimony and compounds, dipropylene glycol methyl ether, ethylene glycol vapor, hexane isomers (other than n-hexane), n-hexane, naphtha, potassium hydroxide, propylene glycol monomethyl ether, sodium hydroxide, stoddard solvent (mineral spirits), tetrachloroethylene, and inorganic tin compounds (except oxides).

SIC 28—Chemicals and Allied Products

In its research on technological feasibility, OSHA found the following examples of controls currently in use:

- The plastic materials and resins manufacturing sector (SIC 2821) used a tank with a hinged cover and fixed ductwork as an exhaust when dumping dye and additives into hot methanol.
- Dust exposure during the bag opening operation in paint manufacturing (SIC 2851) was controlled by modifying the hood to increase dust capture. Likewise, a new dust collection system (collection hoods) with increased capture velocity was installed for use in the bagging and packaging of pesticides (SIC 287).
- Pharmaceutical manufacturers (SIC 2824) addressed the problem of nuisance dust particles by fitting vacuum crescents and elephant trunks on point sources, by fitting chutes with covers, and by placing vacuum attachments on receiving drum covers. Additionally, monitoring was performed from an outside room.
- In order to reduce employee exposure to sulfur dioxide while producing sulfur dioxide gas (SIC 2819), sample collection units were enclosed and attached to a fume collection system. Sample waste was recycled to prevent open exposure in process areas. Electronic spent acid interface detectors were installed to eliminate the need for employee visual inspection of intermittently pulled samples.
- To control TDI exposure in urethane foam manufacturing (SIC 2822), the bun conveyor was enclosed and exhausted. Employee exposure was limited to the startup and finish procedures when installing and removing bun support. A mechanism was designed to support the bun, which eliminated the need for it to be done manually.

In addition, OSHA looked at controls used in paint manufacturing processes. The production of paints (SIC 2851) is a batch procedure which involves the following steps: prebatching, mixing, dispersing, tinting and shading, filling, and storage or shipping. When prebatching or mixing, an employee will slit a bag of dry pigment with a knife and either scoop out the contents for weighing or dump the pigment into the mixer. In some cases, pigments are received in a slurry form and are piped directly into the mixer. Solvents and other raw materials are added into the mixer. Once combined, the mixture is in a paste or slurry form. This mixture is then thoroughly dispersed in a roller, ball, or sand mill or a high-speed disperser all of which are generally closed processes. The paste is transferred to a storage tank where thinning or other agents are added. The paint is later drawn off, filtered and packaged in cans or drums. Airborne dust exposures to components in dry pigments occur during the prebatching and mixing operations when the bags of pigments are opened and dumped. Exposure to chemicals in dry pigments can also occur from pigment spillage and empty bag flattening and disposal. Once the batch is in solution in the mixer, there are no further dust emission points. Exposure to solvents can occur during addition of these ingredients to the mixing tanks, during any leaks or spills, and during packaging.

Local exhaust ventilation would be used to control exposures to dusts and fumes in the paint production processes. Pigment dust exposures at the dumping station can be controlled with the use of a vented enclosure kept under negative pressure by a ventilation system. Empty bags would be manually ejected through a side opening into a large plastic disposal bag to mimimize dust generation during bag flattening and disposal.

Exposures to solvents would be minimized with the use of portable hoods attached to flexible ductwork. These ventilation hoods could be placed over the liquid dumping process and also the packaging operation if the percentage and volatility of the solvents would result in exposures.

Observations and judgments proffered by various chemical industry representatives and associations indicate general compliance with the PELs. Such statements indicating widespread compliance demonstrate the existence of available and practicable control methods for a number of chemicals and processes.

Technological feasibility in SIC 28 for most of the proposed PELs is not challenged in the record. Comments received from ARCO [Ex. 3–740] state: "In general, the petrochemical industry has been using the ACGIH TLVs as the primary workplace exposure guideline for years." This statement implies that most of the PELs are not only feasible but are currently being met.

Dr. Isadore Rosenthal has stated on behalf of Rohm and Haas Company that "experience and data tell us that it is feasible for our company to achieve the ACGIH TLV workplace exposures. This takes time to accomplish, however, and

therefore a phased in approach to controls is necessary. The period of time in which a firm has to achieve exposure controls should begin only after OSHA has certified a feasible analytical method" for determination of exposure [Tr. 8/1/88, pp. 15–16].

The Polyurethane Manufacturers Association (PMA), in expressing support for the proposed PEL for 4,4'-methylene bis(2-chloroaniline), also discussed the feasibility of achieving the proposed level of control [Ex. 3–683]. PMA stated that ". . . the various control technologies and personal protective equipment for these various situations [where exposures occur] is recognized in the industry. . ." . Representatives of the PMA also testified that they believe the industry can comply [Tr. 8/9/88, pp. 83 and 91].

Feasibility of controlling exposures to talc dust was indicated by the remarks of the R.T. Vanderbilt Company [Ex. 3–108]: "We would agree with the ACGIH that dust control has all but eliminated the excess death rates in the talc industry. We also support the 2 mg/m^3 respirable talc dust standard." Vanderbilt apparently foresees no difficulty in controlling talc dust at the new PEL.

The feasibility of the proposed 50 ppm PEL for styrene was asserted by the Dow Chemical Company in its comments to the record [Ex. 3–741]: "Dow manufactures styrene and uses styrene in several processes including the manufacture of styrene polymers and polyester resins. These operations can be controlled to reduce exposures below the proposed PEL of 50 ppm and, in fact, most Dow operations already operate at less than 50 ppm."

The Halogenated Solvents Industry Alliance (HSIA) expressed some concern about the feasibility of the proposed 2 ppm 60-minute STEL for carbon tetrachloride [Exs. 3–873, 8–86, 186], but did not identify specific areas where compliance might be infeasible. HSIA also stated "Since carbon tetrachloride is generally used as a process solvent or raw material, workplace exposures are quite low, generally, we believe, below the ACGIH TLV of 5 ppm." Dow Chemical identified specific tasks and operations such as sampling, loading and unloading, and maintenance where they felt that compliance might be difficult or impossible [Ex. 3–741].

Similar concerns were raised about the feasibility of the proposed 2 ppm STEL for chloroform. HSIA anticipated "that non-chemical industry users. . . would find a 2-ppm limit infeasible in some cases" [Ex. 3–873, 8–86, 186]. The only tasks or processes specifically mentioned in the record as potential problem areas for carbon tetrachloride and chloroform are sampling, loading and unloading, and maintenance [Dow Chemical Company, Ex. 3–741]. These three tasks were named by Dow as problem areas for ethylene dichloride, as well.

Hoffman-LaRoche Corporation stated that ". . . one of the most significant problems associated with the use of chloroform is its high vapor pressure which makes it extremely difficult to contain during processing. Although compliance with the ACGIH TLV of 10 ppm would be difficult to achieve, the proposed NIOSH REL of 2 ppm would, in our opinion, be technologically infeasible" [Ex. 3–749].

Chloroform is manufactured in a gas phase reaction at temperatures ranging from 350 to 750 °C. The major use of chloroform, production of chlorofluoromethanes, also involves reactions carried out at elevated temperatures and pressures. These reactions must be performed in a completely closed system, so routine exposures should be minimal. Given the nature of the production process and the primary use of chloroform, together with the absence of comments from other manufacturers regarding exposures during processing, OSHA concludes that the 2 ppm PEL is technologically feasible.

OSHA recognizes that brief exposure levels of over 2 ppm can be encountered during loading and unloading operations of carbon tetrachloride and chloroform. However, OSHA concludes that the 8-hour TWA PELs of 2 ppm for chloroform and carbon tetrachloride are feasible. Reduction of emissions from tank car and tank wagon openings can be achieved with the use of engineering controls such as vapor recovery systems. Workers should be exposed only for extremely brief periods when attaching or disconnecting lines during loading or unloading operations. Training in and use of proper work practices, in conjunction with proper maintenance or replacement of valves and couplings can reduce both levels and duration of exposures.

The laboratory analysis of samples should be performed under a hood. Overexposures during the collection of samples can be prevented by the installation of sampling boxes if adjustments in work practices are insufficient. Because OSHA allows the use of respirators to prevent overexposures during maintenance activities, feasibility of engineering controls is not a problem for these intermittent activities.

The Vinyl Institute [Ex. 3–624] asserted that modifications would be required to the tank farm vent controls in "a typical EDC/VCM [ethylene dichloride/vinyl chloride monomer] plant . . . to comply with the proposed regulation for EDC." OSHA concludes that significant exposures will not occur under ordinary conditions in a tank farm because workers are not normally stationed there. The Vinyl Institute also asserted that increased down time of plants would be necessary to clean process equipment of EDC before maintenance work could be performed on that equipment. Because OSHA allows the use of personal protective equipment for maintenance activities, no additional down time or problem of feasibility from this standpoint should be encountered.

The Dow Chemical Company [Ex. 3–741] and the Chemical Manufacturers Association [Ex. 3–874] asserted that the 1 ppm TWA for ethylene dichloride may not be feasible in maintenance, sampling, and loading. OSHA believes that there are engineering controls which can control exposures at these specific operations. However, if engineering and work practice controls cannot reduce exposures to the new PEL, respirators would be allowed.

The feasibility of controlling exposures to carbon disulfide in rayon manufacturing was questioned by the North American Rayon Corporation [Ex. 3–415] and the Inter-Industry Committee on Carbon Disulfide [Ex. 3–174]. The overexposures are said to occur only when "the windows and hoods are raised to allow access to the machine." The three tasks for which opening the hoods are necessary are for changing spinerettes, for removing filament bundles and to meet product line changes [BASF Corporation, Fibers Division, Exs. 3–674, 125]. BASF claims that in these areas, ambient workroom air cannot be reduced to 1 ppm as a TWA or 10 ppm as a STEL [Ex. 125]. Rayburn H. Dean, BASF Group Vice President, stated that at other times and in all other areas of the plant, exposures are below 1 ppm, although he refused to provide monitoring data on the grounds that it is proprietary [Tr. 8/2/88, pp. 157–159]. Mr. Dean also explaind that, referring to the cutting area, "We have some TV monitoring there so that fewer people are in that area. When they are, we have installed this special air conditioned room that you made reference to earlier, down in the spinning room" [Tr. 8/2/88, p. 151]. Manufacturers already have "NIOSH approved respirators that must be used" any time there is a short-term excursion

above 20 ppm. [Tr. 8/2/88, p. 136]. "The respirator is the only control available in these three routine operations, to prevent consistent and repeated exposure of the workers to carbon disulfide" [Ex. 125]. OSHA realizes the complexity of this process situation and concludes that the use of respirators during the three aforementioned situations will permit the highest level of protection to workers.

Dow Chemical [Ex. 3–741] and the Halogenated Solvents Industry Alliance [Ex. 3–873] questioned the feasibility of the proposed trichloroethylene (TCE) PEL of 25 ppm in degreasing operations. OSHA concludes that exposures in degreasing can be controlled at the final level of 50 ppm and that exposure data support this position. Both commenters provided information which indicated that, in 1974, 37 percent of open-top degreasers using TCE could maintain 25 ppm and a European estimate from 1980 stated that 50 percent of open-top degreasers and 60–65 percent of closed degreasers could meet a 25 ppm standard. OSHA believes that a considerable amount of overexposure in degreasing is due to inadequate engineering controls or insufficient attention paid to the problem of solvent "carryout." The addition of controls such as chillers, lip exhaust, drying tunnels and covers will reduce personal and ambient exposure levels.

Control of chlorine exposures to the proposed 0.5 ppm ceiling was expected by Dow Chemical Company [Ex. 3–741] to require increased use of respirators by employees engaged in some tasks. Referring to the current PEL of a 1 ppm ceiling, Dow stated: "We have been able to achieve this degree of control in our Cl_2 plants and in the majority of our normal work situations so that respiratory protection is needed only in a limited number of short-term situations." Only one area where these situations occur was mentioned: magnesium production. A NIOSH Health Hazard Evaluation [No. 75–113–249] found a median exposure level of 0.16 ppm for 19 operators's breathing zone samples in a magnesium extraction and chlorination operation. A second NIOSH report [No. 79–40–1381] found a median of 0.2 ppm for 54 samples taken at a chlorine production facility. Only 17 percent of these samples exceeded 0.5 ppm. OSHA concludes that these studies, and the fact that technological infeasibility was not claimed for any specific operations, indicate technological feasibility of a 0.5 ppm TWA PEL and 1.0 ppm STEL for chlorine and that few, if any, additional measures will be necessary to meet these limits.

The manufacturers of cellulose acetate, Tennessee Eastman and Hoechst-Celanese, asserted that the proposed PEL of 250 ppm for acetone is not technologically feasible by means of engineering controls [Exs. 128, 149, 8–54]. Four employee categories were specifically identified in testimony as situations where compliance by engineering controls would be infeasible: filtration workers, dope operators, spinning machine operators and doffers, and parts washing.

While the manufacturers stressed the importance of using acetone as the process solvent, substitute solvents, such as ethyl acetate, could be found for parts washing. In activities such as parts washing, the solvent cannot affect fiber quality, but need only dissolve the cellulose acetate.

Filtration workers at a Kingsport, Tennessee plant dress four or five presses each twelve hour work day [Tr. 8/4/88, p. 142]. According to Mr. Joseph Morton of Tennessee Eastman, "Each press dressing requires about 45 minutes to remove dirty filter media and 45 minutes to apply clean filter media." Assuming that exposures are significant only during the removal of dirty filter media, this would amount to three to four hours of exposure per 12-hour shift. Mr. Morton also observes that "exposure levels of filtration workers frequently are in the range of 500 to 700 parts per million" [Tr. 8/4/88, p. 142]. Exposure monitoring data submitted by the Chemical Manufacturers Association Ketones Program Panel showed filtration workers exposed across a wide range of levels: Four of the 25 samples were in the range 500–750 ppm, 12 of the 25 were 250–500 ppm, and the remaining 9 were below 250 ppm. These data clearly suggest that exposure of filtration workers can be controlled at levels below 750 ppm. Because these samples were taken for the same job title at the same facility, they suggest that the wide range of exposures are due to work practices rather than differences in controls or tasks.

According to the testimony of Mr. Morton, the dope operators are exposed to acetone for about four hours per shift. The primary responsibility of dope operators is to wash the filter cloths in acetone. This exposure generally results in 8-hour TWA levels of 250 to 500 ppm [Tr. 8/4/88, p. 145]. The possibility of using ethyl acetate in place of acetone should be considered for this function, also. Monitoring results provided by the CMA show that most dope operators at the facility where the samples were taken are exposed at levels below 250 ppm. Eleven of the 17 samples were less than 250 ppm and 4 were between 250 and 500 ppm. Again, these samples were taken for the same job at the same facility, suggesting that differences in work practices provide a primary reason for the different levels of exposures. OSHA concludes that improvement to the existing engineering controls and careful attention to work practices would be sufficient to protect the dope operators from overexposure to acetone.

Spinning machine workers can be looked at in three groups: Doffers, acetate yarn spinning machine operators, and tow spinning machine operators. Five 8-hour TWA exposure measurements for doffers were provided by the CMA. Four were in the range 250–500 ppm and one was over 500 ppm. Only one of the 20 samples for yarn spinning machine operators was under 250 ppm. The remaining 19 were between 250 and 750 ppm. Exposure measurements for tow spinning machine operators were split evenly below 250 ppm (26 of 50 samples) and in excess of 250 ppm. Both Tennessee Eastman and Hoechst-Celanese stated that additional engineering controls are not feasible to further protect these operators. Additional local ventilation would cause the fibers to become entangled and complete enclosure would prevent necessary access to the equipment, as well as allowing the possibility of unsafe levels of acetone to build up in the enclosed areas. ". . .The proposed PEL of 250 ppm [for acetone] is neither technologically nor economically feasible" according to the Chemical Manufacturers Association Ketones Programs Panel (the "Panel") [Ex. 98–15].

Based on the evidence submitted OSHA concludes that a PEL of 750 ppm for acetone is not only technically feasible, but is currently being met.

The technological feasibility of the proposed 0.1 mg/m^3 ceiling limit for exposures to nitroglycerin (NG) and ethylene glycol dinitrate (EGDN) is disputed by the Institute of Makers of Explosives (IME) [Ex. 3–749 and Ex. 190]. In this document, the Institute stated: "Reducing workplace levels of NG and EGDN to the proposed ceiling . . . through the application of administrative controls, engineering controls and/or personal protective equipment *is not feasible*" (IME's emphasis).

A number of arguments are presented to support this position. First, "Administrative controls (limiting the duration of a worker's exposure) are

applicable for reducing . . . time-weighted averages, but not to the exposure levels based on short-term exposure limits or ceiling limits." Engineering controls are considered infeasible primarily because of safety concerns, such as the collection of explosive materials in local exhaust ventilation ductwork and the dangers of enclosing equipment. IME stated that general ductwork dilution ventilation has been effective in meeting the current 0.2 ppm ceiling limits for NG and EGDN. An attempt has been made to estimate the cost and feasibility of engineering controls, but it was concluded that ". . . the system had a less than 50% probability of successfully attaining a level of 0.01 ppm (0.1 mg/m^3)."

On the feasibility of using personal protective equipment to comply with the proposed standards, IME contended that "Air purifying respirators are not generally suitable for use in NG/EGDN-containing atmospheres, and at least one manufacturer, Mine Safety Appliances, specifically warns against their use in such atmospheres." [Ex. 3-749]. NIOSH does not approve the use of canister or cartridge respirators for NG/EGDN because the odor threshold is above the PEL. This means that a worker could be overexposed while wearing a respirator and not be aware of it.

A self-contained breathing apparatus is not considered usable for long-term use because of its weight. Thus, air-line respirators are the single remaining alternative means of achieving compliance. However, the IME contended: "Air-line respirators are not feasible because the air lines restrict employee movement, thereby compromising several areas of operations safety as well as the ease of evacuation in the event of emergencies. In addition, lines trailing behind workers would hinder compliance with the long-standing industry standards for reducing to a minimum level all foreign items which might be accidentally introduced to the production equipment and product." [Ex. 3-749] No studies could be found concerning safety aspects of air line respirators. The lack of studies, compliants, or incidents involving safety problems with air lines despite very common and widespread use, leads to a conclusion that there are no significant problems.

The IME concluded that ". . . airborne concentrations of NG/EGDN have already been reduced to the practical minimum. Industry hygienists have concluded that reducing airborne concentrations would not decrease worker exposure and any further reductions must be accomplished through the implementation of improved personal hygiene and other workplace practices. The . . . industry cannot undergo further reductions without dramatically altering the manufacturing process. . . ."

OSHA recognizes the unique difficulties which arise from attempts to control exposures in the explosives industry, but *does* believe that the final limits can be met through a combination of equipment improvements and respiratory protection. The Institute of Makers of Explosives leaves open the possibility that exposures might be further reduced by process and/or equipment improvements. If compliance cannot be achieved via engineering controls or process improvements, then air-supplied respirators could be employed. Quick-release couplings on the air lines would eliminate problems relating to ease of evacuation in emergencies.

American Cyanamid Company suggested that the proposed standard of 0.03 mg/m^3 for acrylamide is not technologically feasible. To support this position, Cyanamid reports that "NIOSH surveyed the acrylamide monomer manufacturing facilities recently and found that exposure levels were above the 0.03 mg/m^3 level in all facilities" [Ex. 3-961 and Tr. 8/11/88. p. 57]. While exposures above the proposed level may have been found at all of Cyanamid's facilities, all of the personal exposure readings at one of the four facilities surveyed were less than 0.012 mg/m^3. All of the area samples at two of the four facilities were less than 0.15 mg/m^3. The conclusions of the NIOSH Hazard Study are that exposure levels were most dependent on the facility or location where the employee works rather than his job duties and that the primary difference in exposure levels between facilities was due to the background acrylamide air level (see *Applied Industrial Hygiene*, Vol. 1, No. 3, September 1986. "Evaluation of Occupational Acrylamide Exposures," Bruce Hills and A.L. Griefe, ACGIH, Cincinnati, pp. 148-152). The plant at which the highest exposure levels were measured, the only facility which manufactured dry acrylamide, has since closed. The range of personal exposure measurements at the remaining three facilities was 0.001-0.132 milligrams per cubic meter. Based on the data and conclusions of the NIOSH field studies described in the Hazard Evaluation, OSHA concludes that the PEL for acrylamide of 0.03 mg/m^3 is technologically feasible.

U.S. Borax submitted information regarding expenditures they have made on environmental controls to reduce exposures to borates. Since 1970, they have spent $7.5 million at the Boron, California, facility. Although some of these expenses have been related to the mining operation under the jurisdiction of the Mine Safety and Health Administration (MSHA), the remainder are related to operations which are of concern to OSHA. The range of dust levels in the fusing department has been reduced to 0.63-50.54 mg/m^3 and in the shipping department to 0.25-15.56 mg/m^3 [Ex. 3-744]. It is not clear whether further reductions could be achieved, and U.S. Borax does not address this issue. Part of U.S. Borax' efforts to control dust related to paving some areas to reduce background dust levels. This effort has apparently been at least partially successful, as evidenced by the minimum values in the range of exposures presented above. It appears that the 10 mg/m^3 PEL level for borates should/might be achievable under most circumstances. Further reduction in borate dust levels might be achieved through the installation of additional engineering controls such as dust collection systems for bagging and packaging, additional dust collection systems at critical release points and further reduction of background dust levels. If additional reductions do not achieve the required levels, the use of respirators will be necessary to protect workers.

SIC 29—Petroleum Refining

In order to assure the quality of petroleum products and determine quality of waste streams, petroleum refiners must sample their process streams periodically. As with maintenance, workers that sample process streams are at risk of being in close contact with a variety of chemicals. Controls for this operation involve sampling boxes that vent gases and vapors away from the operator and/or shield the operator from accidentally splashed or spilled material.

Process stream samples are taken to the laboratory to determine if their qualities lie within acceptable limits. As laboratory workers perform analyses, they can be exposed to various organic and inorganic chemicals if appropriate engineering controls are not in place or if proper procedures are not used. Exposure controls include exhaust fans and laboratory ventilation hoods. In general, this industry has extensive control technology in place for the primary processing equipment. Closed

processes with few exposed workers are predominant due to the requirements of process operation at elevated temperatures and pressures.

SIC 30—Miscellaneous Plastic Products

The Styrene Information and Research Council (SIRC) identified open-molding processes (i.e., processes in which styrene, frequently in combination with fibrous glass, is sprayed or rolled into a mold manually) as the type of process most likely to have difficulty meeting the proposed PEL for styrene [Ex. 3–742, p. 3 and Tr. 8/3/88, p. 5–94]. In SIC 3079, open-molding processes were identified as being used in the production of underground storage tanks, lavatory castings, tubs and spas, and cultured marble products [Ex. 3–742, p. 105 and Tr. 8/3/88, p. 5–181]. Other products that are made using open-molding processes include bridges for military vessels, [Tr. 8/3/88, p. 5–188], planters [Ex. 3–742, Attachment 2, p. 15], benches [Tr. 8/3/88, p. 5–195], and chimney stacks [Tr. 8/3/88, p. 5–188].

Worker exposures to styrene occur principally in two process steps in the open-molding process: gel-coating and lamination [Ex. 3–742, Attachment 2, pp. 17–19 and Tr. 8/3/88, pp. 5–131 to 5–133]. Gel-coat is a pigmented resin of polyester resin-based paint. The application of gel-goat is similar to the application of paint and is normally done using an air atomizer or airless spraygun. Lamination may be applied using either hand layup or hand sprayup. In hand layup, workers place a layer of fiberglass matting directly onto the mold and secure the fiberglass with a layer of resin, which is normally applied with rollers or brushes. In the sprayup process, a chopper gun chops fiberglass roving into pieces and sprays resin at the same time, so the two converge and are sprayed onto the mold simultaneously.

The most extensive data source on exposures to styrene in this industry sector is a study conducted by the State of California's Division of Occupational Safety and Health (DOSH) [Ex. 3–742, Attachment 2]. This study reported the results of an in-depth industrial hygiene survey of styrene and other hazardous workplace exposures in the fiberglass/reinforced plastics industry. A total of 141 workplaces were inspected, and 379 of the 2600 workers employed in these workplaces were sampled over a full workshift [Ex. 3–742, Attachment 2]. The report also recommends the best control measures to minimize hazardous exposures; the focus of the study was on large open-mold sprayup/layup operations, because earlier research had shown that these open-molding operations had the highest exposures of all operations in these workplaces [Ex. 3–742, Attachment 2]. Styrene exposures (8-hour TWAs) at these processes ranged from 0.2 to 288 ppm; the 8-hour TWA arithmetic mean and the median for these sample results were 43.0 ppm and 34.0 ppm, respectively [Ex. 3–742, Attachment 2].

In a comparison of worker exposure levels by industry, the California OSHA study showed that the geometric mean exposure levels were highest in tub/shower manufacturing facilities (53.6 ppm), followed by camper manufacturing facilities (41.0 ppm), spa manufacturing facilities (25.8 ppm), miscellaneous manufacturing facilities (22.0 ppm), and tank manufacturing facilities (12.7 ppm). Operations ranked according to percentage of styrene exposures above 100 ppm as an 8-hour TWA (the former OSHA limit) were: tub/shower manufacturing (19 percent); spa manufacturing (11 percent); camper manufacturing (6 percent); miscellaneous plastics manufacturing (4 percent); and boat and tank manufacturing (none).

The industrial hygienists who conducted this study initially believed that working on large molds, such as those involved in making boats (see discussion for SIC 37) or tanks, would result in the highest styrene exposure levels, because the mold almost surrounds the worker, making a kind of confined space. Workers engaged in boat and tank manufacturing, however, had the lowest overall exposure levels, while workers in the tub/shower and spa manufacturing sectors had more workers exposed above 100 ppm. A partial explanation for these differences in styrene exposure levels in various industry sectors is caused by differences in work production rates according to California OSHA. In boat manufacturing, for example, sprayup operations are performed at a slow and intermittent rate while tub/shower manufacturing is conducted at an assembly-line pace [Ex. 3–742, Attachment 2]. Industry representatives also believe that production volume plays a large role in determining styrene exposure levels. Jack Winnick, general manager of Gold Shield Fiberglass in Fontana, California, testified that plants in Western Europe can achieve much lower PELs than can plants in the United States because "[t]he volume of resin throughput and products produced is a mere fraction of the throughput in U.S. facilities . . ." [Tr. 8/3/88, p. 5–114].

California OSHA found, however, that the factor determining whether or not 50 ppm TWA is currently being reached in facilities producing reinforced plastics products is the degree to which effective controls have been implemented. The California OSHA researchers and Diane Factor of the AFL–CIO both reported that, in all cases where companies had implemented effective control measures, employee styrene exposure levels were below 50 ppm [Ex. 3–742, Attachment 2, and Tr. 8/4/88, p. 6–64].

There is some question regarding the representativeness of the California studies of conditions found elsewhere in the nation. First, SIRC notes that winter climates in the northern-tier states may present additional problems in achieving the proposed PELs [Ex. 181A, p. 38]. Furthermore, the findings of the California study were qualified by its authors as follows:

> This study was conducted in a CAL/OSHA compliance mode: This represents two problems: (1) industrial hygienists do not have the luxury of making frequent visits to any one site, and (2) employees have an understandable desire to minimize actual exposures by various means . . . in order to avoid CAL/OSHA citations and fines [Ex. 3–742, Attachment 2, p. 31].

OSHA appreciates the critiques of the CAL/OSHA study. OSHA found that employers would need to employ a flexible compliance strategy during manual layup/sprayup operations to achieve the proposed limits in boat-building facilities (see Technological Feasibility discussion for SIC 37). The California study indicates that employee exposures to styrene during manual layup/sprayup operations in facilities in SIC 30 are even higher than those for boat-building facilities. Thus, there is uncertainty about the technical feasibility of achieving the 50-ppm TWA and 100-ppm STEL limits exclusively by implementing engineering and work practice controls during manual layup/sprayup operations in SIC 30. Respirators as well as engineering and work practice controls may be necessary to achieve these limits in some operations. OSHA concludes, however, that for most operations in SIC 30 where styrene is used, the revised TWA and STEL limits are technologicaly feasible.

Daniel Boyd, representing the SIRC, also commented that the mixtures formula described in 1910.1000(d)(2)(i) would necessitate reducing employee exposures to well below 50 ppm. He stated that:

> Since the reinforced plastics environment consists of a number of chemical constituents, the allowed exposure to these various chemicals must be calculated through the mixture formula. . . . OSHA's application

of the mixture formula will require limits well below 50 ppm for styrene and [the proposed PEL of] 250 ppm for acetone [Tr. 8/3/88, p. 5–97].

The SIRC thus argued that, with the mixtures formula, "OSHA has proposed a rule [for styrene] that would impose an exposure level lower than it has accounted for in its feasibility analysis" [Ex. 181A, p. 44].

OSHA does not agree with the SIRC that the mixtures formula requirement will have a substantial impact on the ability of employers to comply with the 50 ppm PEL for styrene. Traditionally, OSHA does not apply the mixtures formula in most cases where multiple exposures occur. According to OSHA's *Field Operations Manual (FOM):*

> The use of . . . [the mixtures formula] requires that the exposures have an additive effect on the same body organ or system. Caution must be used in applying the additive formula, and prior consultation with the Regional Administrator is required (OSHA FOM, Chapter IV, Section 6(e)(i)).

Thus, in the case of styrene and acetone, which are both used in the reinforced plastics industry, OSHA does not believe that the mixture formula rule specified in paragraph (d)(2)(i) of the final rule would necessarily apply, because styrene is principally a narcotic agent that acts on the central nervous system and acetone is primarily an irritant that acts on the eyes and respiratory passages at concentrations at or below the final PEL. These substances therefore cannot be considered as having an additive effect. Consequently, OSHA has based its feasibility assessment for styrene and acetone in the reinforced plastics industry on the availability of the engineering and work practice controls necessary to achieve the PELs for these substances individually.

Carbon disulfide is a solvent used in the production of cellulosic food casings. It is reacted with cellulose to make xanthate and is slowly released during subsequent steps of production. The process currently used is the only known process for producing cellulosic food casing, and carbon disulfide is the only known solvent for this process [Ex. 8–45].

The feasibility of controlling exposures to carbon disulfide in the manufacturing of cellulosic food casings was questioned by representatives of the producers of these products, Viskase Corporation and Teepak, Incorporated [Exs. 33, 162, 3–753, 8–19, 8–45, Tr. 8/2/88, pp. 4–201–217]. These commenters noted that, in three specific operations, it is necessary to open the machinery to perform manual operations, (unloading the baratte, aligning strands in the cabinet, and manual puncturing of the casings). When unloading the baratte, manual raking is required because of the light and sticky characteristics of xanthate, the parent compound. Operator access is required to keep the strands of product properly aligned within the extrusion apparatus (or "cabinet"). Manual puncturing of the casings is required downstream of the extrusion nozzles [Ex. 8–45]. Personnel performing all three of these operations must open the process machinery while performing these tasks; currently, personnel wear air-supplied hoods to protect against the carbon disulfide excursions above 20 ppm associated with the operations [Tr. 8/2/88, p. 4–228]. Operator access is essential to assure casing quality [Ex. 162]. An engineering study conducted in one of Viskase's plants concluded that "it is highly unlikely that the 1 ppm [level] could be obtained at [these] three * * * routine operator tasks," even if "the most extreme measure that can be visualized as an effort to reduce the concentration" was employed [Ex. 8–45]. Teepak stated that "no one * * * has developed a system or knows of any engineering controls that . . . [are] capable of reducing CS_2 levels in the casing industry to the 1 ppm level proposed by OSHA," adding that Teepak had recently redesigned and rebuilt much of its plant using the best available technology [Ex. 162]. Commenters repeatedly stressed [Exs. 33, 162, Tr. 8/2/88, pp. 4–201 to 4–217] that feasibility was a problem only for these manual operations; thus, OSHA concludes that the use of respirators as well as engineering controls and work practices, may be necessary for unloading xanthate from the baratte, aligning strands in the extrusion cabinet, and manual puncturing of casings at the extrusion nozzles unless OSHA can demonstrate that engineering controls and work practices alone can achieve the PEL at the 8-hour TWA limit for carbon disulfide.

The Polyurethane Manufacturers Association (PMA) stated that the 0.02 ppm PEL for 4,4'-methylene bis (2-chloroaniline) (MBOCA) is technologically feasible and is already being achieved in many facilities [Ex. 3–683]. MBOCA is used as a fixative in producing castable polyurethane. The chemical is no longer produced in this country, but is still widely used to produce castable polyurethane products. PMA [Ex. 3–683] also stated that no substitute for MBOCA has been found that matches its physical properties and processing characteristics at a competitive price.

According to PMA, worker exposures to MBOCA occur chiefly during transfer operations. PMA stated that "once the melted MBOCA is mixed with prepolymer, there is no risk of employee exposure to MBOCA" [Ex. 3–683]. The industry has developed a number of methods to control employee exposure to MBOCA during transfer operations, including the use of isolated rooms, laboratory hoods or glove boxes, and vacuum transfer systems that carry MBOCA from drums to the melters in closed, automated systems. PMA also stated that "based upon considerable workplace monitoring [conducted] since the 1970s, it is apparent that an employer who observes the recognized industry practices for the use of MBOCA and who monitors the results . . . will feasibly comply with the proposed TWA level [of 0.02 ppm]" [Ex. 3–683]. Therefore, OSHA concludes, based on the proven effectiveness of currently available technology that a PEL of 0.02 ppm for MBOCA is technologically feasible.

SIC 31.—Leather and Leather Products

During a site visit performed by OSHA to a shoe production facility [Ex. 11—Firm 7], an overexposure to 2-butanone at the outer sole cementing operation was found. A small exhaust system used at the operation had inadequate air movement to reduce exposure. The exposure exceeded the current PEL of 200 ppm, as well as the proposed STEL of 300 ppm. The length of the exposure was 3.5 hours. The total cost for local exhaust ventilation to the five affected work stations, as estimated by Clayton Environmental Consultants, would be $20,000. This cost figure is based upon a flanged 4 foot by 12 foot exhaust hood with a capture velocity of 100 feet per minute (fpm). The flow rate is estimated to be 775 cubic feet per minute (cfm) per work station. By employing these control measures, worker exposures to 2-butanone, as well as to solvents in general, will be reduced.

The site visit firms did not have toluene overexposure. In general, toluene exposures can be decreased by revising standard work practices to reduce the contact time between leather and toluene.

SIC 32—Stone, Clay and Glass Products

In batch mixing of raw materials for glass production (SICs 321 and 322), OSHA found that drysweeping and/or the use of compressed air for cleaning may contribute substantially to the employees overall exposures. By

substituting vacuum cleaning systems, worker exposure can be reduced.

The Brick Institute of America (BIA) stated that limited success has been achieved by member companies in controlling clay and shale dust exposures. Despite the companies' efforts, employees are still required to wear respirators in some areas of the plants. Although local exhaust ventilation has been installed at work station, the nature of the job requires workers to leave the area of their work station. Furthermore, the moisture content of the raw materials "creates substantial maintenance problems for the control equipment." The BIA concludes ". . . it simply is not possible to reduce dust levels any further using known technology" [Ex. 130].

OSHA conducted site visits to manufacturers of both cement blocks [Ex. 11—Firms 2, 4] and a manufacturer of unglazed floor tiles [Ex. 11—Firm 12]. These firms have processes analogous to brick manufacture. No overexposures were found. In addition to good housekeeping measures, one of the principle means of controlling dust exposure was the use of wet materials. Dry material hoppers were located outside the building or in locations above the work floor. Mixers were generally fully automated. Workers who were required to work inside the mixer (after proper lockout procedures) using pneumatic hammers to remove hardened materials, used local exhaust tubes, and wore respirators. OSHA concludes it is technologically feasible to control dust exposure in this industry.

SIC 33—Primary Metals Industries

The American Iron and Steel Institute [Exs. 3–1123, 72, 129, 188] stated that the proposed PELs were not technologically feasible if retrofitting were required, and that most operations produce intermittent exposures where respiratory protection equipment is more appropriate. The AISI provided no support for their statement that retrofitting is not technologically feasible, merely stating that some controls can only be installed when a plant is built or modernized. However, they provided several examples of the cost of such retrofits, stating that the costs rendered the retrofits infeasible. This, however, does not support a finding that the technology does not exist to control the exposures.

During site visits, OSHA observed a wide variety of controls in place in this industry. How effective these controls are cannot be known for certain. When AISI agreed to assist OSHA in arranging site visits in this industry, they stipulated that the OSHA contractors could collect no air samples during the site visits. AISI submitted exposure data for different operations, but provided only the lowest and highest values for each chemical in each operation [Ex. 129]. When ranges are used, it is not possible to discern where most samples fall to assess potential feasibility. Where the highest value for a chemical is at or below the new PEL, the new PEL is clearly feasible. This is true in a number of cases, and demonstrates that controls are available which will maintain exposures below the new PEL for those chemicals. The exposure data does indicate that the STEL for sulfur dioxide cannot be regularly achieved with engineering and work practice controls in blast furnace operations and at sulfur plants. In addition, there is some evidence that the ceiling limit for carbon monoxide cannot be regularly achieved with engineering and work practice controls at blast furnace operations, vessel blowing, basic oxygen furnaces, and sinter plants. There is no evidence to the contrary in the record for these two substances. OSHA, therefore, will permit flexibility in the use of respirators for these operations. The burden of proof will not be on the employer to demonstrate that compliance with engineering and work practice controls are infeasible in a compliance action for exposure to the STEL for sulfur dioxide and the ceiling for carbon monoxide at these operations.

AISI also provided a list of occupations and the related duties where exposures are intermittent. Many of these would be considered cleaning and maintenance [Ex. 72]. Where exposures are brief and intermittent, or where they are related to cleaning and maintenance, respiratory protection may be the appropriate control technology in accord with OSHA's traditional policies.

SIC 331—Basic Steel Products

OSHA, through its contractors, has conducted site visits of various operations associated with steel manufacturing. During these visits, OSHA observed that engineering controls were in use. Due to a pre-visit stipulation of the American Iron and Steel Institute, OSHA was not able to monitor exposures at any of these operations.

In a site visit to a sintering plant, OSHA observed the application of a hood and local exhaust at the end of the sintering conveyor, a transfer point for sintered material. Also, at the same plant, local exhaust piping on the pug mills and the sinter air cooler was in place. The emissions from these sources were directed to a centralized baghouse [Ex. 120—Firm 28].

At hot strip production facilities [Ex. 129—Firms 29, 31], workers controlling the rolling process were positioned in air conditioned control stations or pulpits. Workers engaged in the coiling and marking area were provided with dilution ventilation.

At Basic Oxygen Furnace (BOF) facilities [Ex. 129—Firms 37, 38] emissions generated during desulfurization, deslagging and oxygen injection were vented to an electrostatic precipitator and/or baghouse. BOFs also emit carbon monoxide as a byproduct. Some BOF processes use this as a means of controlling the metallurgical reaction. This is controlled through the exhaust system. Continuous CO monitors are used to alert workers to peak or emergency conditions.

During a visit to an electric arc furnace operation [Ex. 129—Firm 39], emission control equipment was an integral part of the furnace's rotating roof. Contaminant generation during this operation was vented to an electrostatic precipitator. The building in which this process was conducted had been modified to incorporate roof level hoods and ducts that carried escaping contaminants to a centralized baghouse.

At a coke oven gas processing facility [Ex. 129—Firm 32], operations were carried out in enclosed vessels or process equipment (similar to those found in chemical processing facilities) that provide protection from continuous direct exposure. OSHA believes that compliance with the proposed PELs can be achieved since exposures are primarily the result of fugitive emissions and operational upsets.

At a blast furnace operation [Ex. 129—Firm 27] which was visited, the firm indicated its concern over the proposed PELs for carbon monoxide, sulfur dioxide, and calcium oxide. [Iron oxides were also generated in these operations but it was not clear that exposures to iron oxide were problematic. The final rule retains the existing limit for iron oxide). Blast furnaces operate under positive pressure and extremely rigorous conditions. These conditions do have a severe effect on the refractory lining of the furnace wall. Over time contaminant release, particularly carbon monoxide, will occur. Thus blast furnaces of older design or furnaces reaching the end of their life cycle will tend to have greater emissions of air contaminants. During the tapping of the furnace, workers are exposed to iron oxides, carbon monoxide, sulfur dioxide, and calcium oxide as hot metal pours into the

transfer car via runners on the floor. In one site, these runners were covered to reduce emissions.

Workers typically move between areas of high exposure in the manufacturing area, and areas of low exposure in air-conditioned or heated control rooms or "shanties." Exposures are intermittent. Processes at which they work also have episodic periods of air contaminant emission. This dual variability suggests that respiratory protection may be needed to control worker exposure to such metallurgical air contaminants as carbon monoxide, sulfur dioxide, iron oxide, and calcium oxide.

SIC 332—Iron and Steel Foundries

OSHA conducted a site visit to a gray iron foundry [Ex. 11—Firm 13] engaged in the manufacturing of gray and ductile iron castings. Exposure samples were taken in the grinding process. The result of the sampling disclosed an exposure level of 39.0 mg/m^3 TWA for iron oxide which is above both the current and final PEL of 10 mg/m^3 TWA.

Clayton Environmental Consultants recommended a number of actions that can be taken to reduce iron oxide dust levels. A mechanical shakeout and automatic sand handling system complete with dust collection can be implemented to substantially reduce dust levels. The mechanical shakeout would consist of a 16 square foot enclosure in which the molds and castings can be manipulated and then brought back out for further processing. The existing muller hood should be maintained or upgraded to produce 900 CFM of local exhaust ventilation. It was also recommended that dust collection and make-up air systems would be needed to replace exhausted air. For this purpose, make-up air should be ducted for release near the work station and workers should be provided with movable diffusers or a means of controlling airflow at the work station. The purchase of a rider-type sweeper was also recommended as a means to control dust levels.

Clayton also recommended controls to reduce grinding exposures. Local exhaust ventilation can be installed on pedestal grinders. Grinders of this type should be exhausted at 1,000 CFM assuming 16 inch wheel diameters. For hand-held grinders, a 2 foot by 3 foot ventilated table/bench is the recommended control. A 200 CFM/foot^2 of grinding bench area exhaust rate is the estimated requirement for this application.

Additional controls applicable to foundries were found during OSHA's review of this industry. These controls either individually or in combination should generally control exposures to the final PELs.

- The arc air process in steel foundries (SICs 3324, 3325) was used during the processing of steel castings to control fumes. In order to ventilate the arc air booths, fumes were exhausted through the back of the booth and fresh air was supplied from above and behind the operator.
- Steel foundries (SICs 3324, 3325) used an overhead canopy hood during the induction melting of steel to control fumes. The hood consisted of sheet metal barriers extending down from the roof to the top of the hot metal ladle monorail. Thermal drafts carried the fumes upward into the hood where they were exhausted by ventilators. Mancooler fans behind the workers pushed some fumes under the hood.
- Emissions during the oxy-acetylene torch cutoff of risers from steel castings was encountered in iron and steel foundries (SIC 332). Castings were cut in a specially designed booth with a rear exhaust flow and a frontal air supply flow. Air pressure from the cutting nozzle of the torch was directed toward the rear exhaust port for effective dust and fume control.
- Fume control of a sandwich-type innoculation in iron foundries (SICs 3321, 3322) was achieved through the use of a commercially available canopy hood. The fume-laden air was exhausted through mobile duct work and cleaned by a fabric collector before being discharged into the surrounding environment. The hood tilted with the furnace so that it always was directly over the ladle for fume capture.
- Fume, dust, and gas control from the melting of iron (SICs 3321, 3322) in an arc furnace was achieved by the installation of a hood. The exhausts collected by the hood were filtered by cloth filters before being released into the external environment.
- Control of dust and gas emissions from phenolic urethane cold box core-making in iron foundries (SIC 3321, 3322) included local exhaust ventilation which provided negative pressure at the core box. Parting line gaskets, blow seals, and stripper pin o-rings were regularly maintained for emission control. Exhaust outlets captured excessive dust.
- In an iron foundry (SICs 3321, 3322), hot combustion gases were exhausted and flowed through an after burner, cooled, and then passed through a dust collector. Tapping emissions were captured by a canopy hood. General ventilation was provided by mancooler fans.

The UAW [Ex. 197] listed additional feasible control measures in foundry and other metallurgical operations. These include:

(a) adequate make-up air;

(b) supplied air islands for operator stations (laminar flow, down draft make up air supply units);

(c) tempered (cooled) make up air to reduce the need for high velocity air for heat stress relief;

(d) process arrangement to remove loose sand from castings after shakeout before they are finished;

(e) maintenance of enclosures and exhaust volume for sand handling equipment to prevent emission of dust;

(f) reduction of silica burn-in on castings to reduce exposures at cleaning and finishing operations;

(g) use of wet methods.

SIC 333/334—Primary and Secondary Non-Ferrous Metals

In its review of technological feasibility in the non-ferrous metals industry, OSHA identified the following examples of engineering control and work practice measures:

- Control of emissions from aluminum ore handling and storage (SIC 3334) was addressed with an unloader which uses movable vacuum nozzles to remove alumina and coke from barges. The ore was moved on an enclosed conveyor which was equipped with air exhaust hoods at loading and transfer points. The operator can be situated in an air conditioned cab.
- Reduction of alumina dust emissions during ship unloading (SIC 3334) was achieved by automating and controlling operations from an enclosed control booth. Furthermore, mixing operations were hooded and exhausted.
- During anode rodding in prebake plants during primary aluminum production (SIC 3334), spent butt remover, butt crushers, cast iron remover, and shot cleaner were exhausted to a bag filter dust collector. Use of induction furnaces and exhaust hooding reduced metal fume exposure during melting. Hoods and slotted hoods were also used. The operator can maintain controls from an enclosed console.
- Control of air emissions during potline operations of aluminum smelters (SIC 3334) was achieved through the use of potroom ventilation and automated processes such as the use of hooding which consists of curved and ribbed shields, the employment of a dual draft system, and an exhaust system which leads to a dry scrubber. Other control methods included hooding with rigid air-operated doors which exhausted the emissions through air takeoffs to an expanding duct exhaust manifold which,

in turn, was exhausted by a fan. Furthermore, computer-controlled systems existed which could automatically perform production functions without requiring workers to open pots or hood shields above pots.

• The mercury cell process may be used in aluminum smeltering (SIC 3334) to produce chlorine gas from brine water. To reduce chlorine gas exposure as a result of this production, the diameter of the brine header was increased to accommodate the gas phase above the liquid phase; the number of cells in the system was increased; the pH of the brine was adjusted; the compressor controls were modified to accommodate surges in pressure; inlet box covers were replaced with better covers; and the brine feed nozzle flange was modified.

• Several engineering controls have been recommended for copper smelting locations (SICs 3331, 3341). A preventive maintenance program can be developed and implemented to insure that ventilation and conveyor systems are operating properly. Dead beds can be installed in chutes to break the fall of material and reduce the level of dust generated. Pneumatic aerators can be installed to eliminate the need for manual air lancing in bins and chutes. Industrial vacuum systems can be used.

• Collection hoods can be installed at each conveyor transfer point at copper smelter sites (SICs 3331, 3341) to control copper particulate. Primary copper smelting conveyor skirting can be properly adjusted, and fingers installed at discharge points. Inspection doors should not be left open, and the lunchroom/breakroom should be located outside of the reclaim building. General measures throughout copper smelting plants (SICs 3331, 3341) to control copper dust emissions included: using local exhaust ventilation for localized sources, and general exhaust ventilation for areas with unidentifiable sources; enclosing conveyor belts and transfer points; enclosing the air conveying system for the transfer of flue dusts; enclosing workers' operating vehicles; installing secondary hoods on converters; prohibiting the blowing out of converters while on stack; performing preventive maintenance on balloon flues; not allowing converters to remain rolled out for extended periods of time; and providing cleaning rooms with filtered, tempered, positive pressure air. When hauling slag from the metal smelting operation, slag can be granulated after skimming with high velocity water; a chemical dust suppression system can be used when crushing any cooled slag; and the slag crew can ride downwind from fumes. Further engineering controls include constructing pulpits for operators; close-coupling the ventilation system to the Larry car; using dead beds in calcine loading; enclosing a portion of the building to block wind; and vacuuming the superstructure of the Larry car and any spills.

• Controls used to decrease exposures to arsenic, dust and sulfur dioxide at primary copper and lead smelters (SICs 3331, 3332) included upgrading the present ventilation systems; operating electric furnaces at negative pressure; eliminating air lancing as a method of removing concentrates from receiving hoppers; using pneumatic aerators or belt wipes; using wet techniques in storage; reclaiming concentrates; and improving general housekeeping.

• Exposures to lead, cadmium, and arsenic at lead and copper smelters (SICs 3331, 3332) were reduced by the replacement of old sintering machines with ones equipped with dust and fume controls and by placing a cover over the charge hole when slag was not being charged into the reverberatory furnace.

• Use of a multipurpose crane with an enclosed cab reduced operator exposures to air emissions at carbon bake plants (SIC 3334). The cab was supplied with filtered conditioned air. The crane was equipped with a vacuum system which could aspirate cake from ovens and separate fines.

• Controls for exposure to soluble platinum salts in precious metal refining (SIC 3339) included local exhaust ventilation used in jaw crusher and recovery sampling, maintenance of a closed system in refinery through use of glove box filters, the use of borohydrate solution to wash down spills and reduce salts to insoluble platinum metal, and mandatory showers and daily clothing changes.

• Controls for the primary non-ferrous metals industry (SIC 333) included local exhaust ventilation systems; general dilution ventilation; covers, hoods and exhaust systems for belts, material handling and transfer systems; enclosure and exhaust of sinter machine area; local exhaust and dilution ventilation for the reverberatory and refinery areas.

• The reduction of exposures to inert cadmium and silver dust during a ball mill operation was accomplished by building and equipment process changes such as local exhaust ventilation, hood enclosure of process or worker, and air cleaning equipment.

• In the secondary smelting and refining of non-ferrous metals (SIC 334), particulate emissions from a dross mill were reduced by making modifications to the dust collection system and to air volumes drawn through the baghouse. Engineering controls used include increasing fan efficiency through the use of sheaves and belts, installing water sprays on crusher infeeds, running new pipe to localized dust areas, installing additional cleanout ports, and replacing the top of the baghouse.

• Employee exposure to nuisance dust from zinc smelters (SIC 3333) was controlled by replacing the dross handling operation with a dross mill. The crusher was replaced with a rotating mixer, thus eliminating fugitive dust from this part of the process.

Asarco, Inc., questioned the technological feasibility of achieving the proposed PELs for sulfur dioxide. In its written statement to the docket [Tr. 8/15/88, pp. 120–124], Asarco provided several examples of the nature of engineering controls that have been installed at its plants. "Asarco's copper smelter in Hayden, Arizona, has been modernized with the installation of an Inco flash smelting furnace, as well as the installation of control devices, such as secondary converter hoods. Additional controls, including secondary converter hoods with an air current design have also been installed in Asarco's copper smelter at El Paso, Texas." Asarco maintains "despite these controls, however, SO_2 concentrations for a number of job classifications at Asarco's copper smelters exceed or may exceed the proposed TWA of 2 ppm. Morever, it appears that most smelter jobs in molten areas would frequently exceed the proposed STEL of 5 ppm, because of the episodic nature of smelting operations. Asarco is not aware of any combination of engineering and work practice controls that can feasibly reduce exposures to the levels required by the standard."

Magma Copper Company, in written testimony [Tr. 8/12/88] has also expressed concern on [Ex. 3–91, pp. 92–105] the achievement of the proposed standards for SO_2. Magma is in the process of installing new emission controls at its smelter at a cost of $132,000,000. The elements of this retrofit include "a new state of the art Outokumpu flash furnace obviating three existing reverberatory furnaces. The retrofit has also an improved converter gas handling system as well as a new secondary gas collection system. The smelter has local ventilation to all areas that have historically been sources of SO_2 emissions, and as such, the smelter

should have limited problems with fugitive emissions. . . ." "Additionally, the converters have two separate local ventilation systems. The primary system collects the highest concentration of SO_2 and supplies an acid plant. The secondary local ventilation system collects gases during some phases of the converter roll out operation." Magma has stated that the flash furnace "was placed on line in July of this year" and "start-up is estimated to be completed with normal operations in place by November 1, 1988. Therefore, a comprehensive survey of our new engineering control system is not feasible at present." However, Magma has estimated from past data and "for various configurations" that the 5 ppm STEL "is not likely to be met."

Asarco has also submitted as an attachment to its written statement a portion of its 1977 post-hearing brief that discusses the problems of SO_2 control. In it, Asarco refers to the report "Environmental Conditions in U.S. Copper Smelters" by Willian L. Wagner of NIOSH as evidence of the need to use respirators. Quoting from the report, Asarco cites, "In most smelters the use of respirators is essential on charge floors of reverberating furnaces, in the green feed galleries, tripper decks above these furnaces or any areas above these furnaces. In these areas, the SO_2 concentrations varied from non-detectable levels to many hundreds of parts per million. . . ." In addition, Asarco also relies on the testimony of Mr. Wagner at the 1977 hearings in which he addressed the then-proposed "ceiling limit" of 10 ppm, "for most parts of the smelter." "There are a number of areas where you could get concentrations of sulfur dioxide for periods of time greater than 10 parts per million." According to Mr. Wagner, concentrations exceeding 10 ppm could easily last 15 to 20 min.

As this discussion indicates, there are many engineering and work practice controls available to reduce exposure to SO_2 and the other contaminants present. They will frequently be able to control exposure to 2 ppm. However, for some operations, feasible engineering controls may not be available. OSHA will accept the use of respirators in conjunction with engineering controls unless OSHA can demonstrate that engineering controls and work practices alone can achieve the 2 ppm PEL.

Brief peak exposures will occur over 5 ppm in several areas of a lead or copper smelter. Good work practices will curtail many of these. However, respirators may be appropriate in smelters along with other controls to control peak exposures unless OSHA can demonstrate that engineering controls and work practices alone can achieve a 5 ppm STEL.

SIS 336—Non-Ferrous Foundries

Fumes were controlled during the casting of bronze in foundries (SIC 3362) through the use of enclosing hoods. A mobile hood exhausted the ladle at all hot metal transfer points. Flexible ducting connected the hood to a traveling exhaust carriage.

SIC 339—Miscellaneous Primary Metals Products

Manufacturers improved dust control using closed screw conveyors in the transport and manufacture of iron powder (SIC 3399). Open conveyor belts were changed to a closed screw conveyor system. Duct work was totally replaced. Local exhaust was provided for the rotary screens. New baghouses and electrostatic precipitators were also installed. OSHA visited a manufacturer of metal alloy powders [Ex. 145, Attachment A]. Although overexposures to the current PELs were not found, a reduction to the new PELs would result in overexposures. To reduce cobalt dust exposures below the 0.05 mg/m^3 level, additional monitoring should be conducted to verify the need for engineering controls. The following measures were recommended and determined to be necessary: (1) Use of an exhausted booth for developmental screening, (2) routing air discharged from the dust collector associated with the vortex classifer to the outdoors or into the plant's main dust collection system, (3) providing exhaust ducts to be connected to atomizer drums during cleanout periods, and (4) discontinue the practice of dry-sweeping the floors and either acquire a vacuum sweeper truck or use the central vacuum system more extensively.

SIC 34—Fabricated Metal Products

Control of copper dust at a cookware manufacturing plant (SIC 3469) was addressed by unclogging the ventilation system, repositioning cooling fans, and instituting weekly ventilation system inspection and maintenance programs.

A plating shop (SIC 3471) uses extensive local exhaust ventilation to control worker exposure. Each part to be plated undergoes some surface pretreatment. This can consist of shot-peening, abrasive blasting, degreasing, wax or tape masking and other treatments. Parts are manually placed into the tank using an overhead hoist for large parts.

The tanks are set on top of concrete ducts. The floors of the shop and the aisles between the tanks are reinforced concrete, however the area around the perimeter of the tank is open to the basement and covered by steel grating. The ducts are connected to a fan on the roof of the building.

The largest of the hard chrome tanks, holding over 1000 gallons of plating solution, has a two sided lateral exhaust ventilation system. The slot on each side consists of a series of seven slots. The slots are set back from the edge of the tank but an overhanging hood extends to the edge of the tank. A second tank has both a two sided slot ventilation system and a cover. This two piece cover is hinged to a ventilation manifold and extends beyond the front and rear edges of the tank.

Arc welding is performed in many SICs as an auxiliary process and in several industries such as fabricated structural metals (SIC 3441), as the principal process requiring engineering control. During the welding process, temperatures are sufficiently high to vaporize some of the base material of the electrode and produce large quantities of fumes containing the elements in the electrode and the base metal. Thus welders and other workers in the vicinity are exposed to mixtures of fume-sized particulates and both irritant and toxic gases which in combination may have additive or synergistic physiological effects.

Differences in worker exposure are attributable to a variety of factors including type of welding helmet worn, position of the welding operator, the work environment, arc time, and the availability and performance of ventilation equipment. Arc time varies greatly due to differences in work schedules, set up times, and the sizes, shapes and types of tasks. Tasks can vary from short-term repairs conducted irregularly to full time production welding.

During arc time the fume is generated within or close to the worker's breathing zone. Background fume concentrations could also be significant if a large number of welders are working or the work is being performed in a relatively confined space.

Because of the numerous factors that can influence exposure levels during welding, three different types of controls can be used for various welding situations. The controls include: (1) Local exhaust ventilation for welding in shops; (2) ambient air cleaning devices to minimize background fume concentrations; and (3) a portable blower for use in confined areas.

Local exhaust ventilation configurations include: a welding bench

with a backdraft hood for small to medium work pieces; a fixed close-capture hood placed at the back of a work rest table; a portable close-capture system including electrostatic precipitator; or an exhaust hose incorporated into the structure of the welding gun.

Ambient air cleaning devices are designed to lower background welding fumes which escape collection by the local exhaust system. The ambient air cleaner is expected to surpass general dilution ventilation systems in terms of both fume removal and cost.

A portable blower system works by exhausting fumes from a confined space through a large flexible tube.

No commenter questioned technological feasibility for this industry.

SIC 35—Machinery

In addition to techniques for weld fume control mentioned above, in the manufacture of pumps, employee exposure to welding fumes was controlled (SIC 3561) through the use of an air lux fume eliminator.

In the milling of tungsten carbide tools (SIC 354), the placement of local exhaust ventilation controlled cobalt exposures during the transfer of carbide.

Oil mist is used in the SIC during the manufacturing of parts on screw machines or other machine parts. There is a wide assortment of engineering and work practice controls to reduce exposure to oil mist [Tr. 8/5/88, p. 7-53]. Since OSHA is retaining the existing PEL of oil mist, OSHA concludes that the PEL is technologically feasible.

In farm equipment manufacturing and repair (SIC 3523), paint mist was controlled through sophisticated application techniques as applied to downdraft spray booths. The use of heated paint in the painting of hay stack wagons allowed the airless atomization to take place at relatively low paint pressures. This resulted in low droplet velocity with little rebound.

In the manufacture of machinery, degreasing operations using refined petroleum solvents are prevalent. The AFL-CIO [Ex. 194] and UAW [Ex 197] noted additional feasible measures for control of exposure to refined petroleum solvents (RPS) such as VMP naptha:

(1) Spray application of liquids containing RPS should be permitted only in exhaust ventilated enclosures such as spray booths.

(2) Articles coated with liquids containing RPS should be kept in containers equipped with local exhaust ventilation to prevent evaporation of RPS into work room air.

(3) Equipment for bulk transfer of RPS should be equipped with vapor capture systems.

(4) Exhaust ventilation should not recirculate RPS vapors into workroom air.

(5) Cleaning of floors with RPS should not be permitted.

(6) Where spray booths are cleaned with RPS, ventilation should be maintained during cleaning.

(7) Quantities of RPS used and surface area coated with RPS containing liquids should be kept to a minimum.

(8) Splashing of RPS containing liquids or creating of puddles on floor or other surfaces should not be permitted.

(9) Open surface tanks containing RPS should be equipped with covers and local exhaust ventilation. Covers should be closed when not in use. Special attention should be paid to preventing forced expulsion of vapors during addition of materials and entrainment of vapors when articles are added to or removed from open surface tanks.

(10) Open buckets of RPS should not be permitted. Containers for RPS should be equipped with self closing covers. Rags or other material soaking in RPS should be kept in closed containers.

(11) Procedures for response to spills and leaks, including criteria for evacuation of personnel not essential to safe cleanup, should be devised.

(12) Skin contact should be prevented by redesign of operations to eliminate dipping of hand into RPS containing liquids, minimizing splashing or mist contact and wetting of skin and clothing. Gloves and impervious clothing should be supplied where wetting of skin and clothing can't be prevented.

No commenters challenged the technological feasibility of meeting the proposed PELs in this industry.

SIC 36—Electric and Electronic Equipment

Electric lamp manufacturers (SIC 3641) have reduced mercury vapor in lighting plants. Glass pellets used as starters for fluorescent lamps were flame sealed after mercury had been injected into them. Overhead suction velocity of the exhaust system was increased to reduce mercury overexposure. Also, as a special vacuum cleaner was employed to clean the turntable.

The use of styrene for open mold fiberglass operations in the manufacture of household refrigeration equipment (SIC 3632) is similar to the use in SIC 37—Transportation Equipment. Thus, respirators may be required to augment engineering controls during manual layup/sprayup operations, as discussed in SIC 37, below.

Technological feasibility was not addressed by commenters to the docket in this sector.

SIC 37—Transportation Equipment

The Styrene Information Research Council (SIRC) identified manual layup and sprayup processes as operations in this sector that would not be able to meet either the 50-ppm PEL or the 100-ppm ceiling that the Agency has proposed as limits for styrene [Exs. 187, 3-742; Tr. 3/8/80, p. 5-94]. The open-molding process is primarily used in this sector to make fiberglass boats and fiberglass car and truck bodies (especially bodies for recreational vehicles). The feasibility issue that is raised relates to production operations that involve the spraying of large volumes of resin containing styrene on large surfaces where volatilization occurs [Ex. 198, Tr. 8/3/88, p. 5-95].

The single most extensive data source on exposures to styrene is a study conducted by the State of California's Division of Occupational Safety and Health (DOSH) [Ex. 3-742, Attachment 2]. This study reported the results of an in-depth industrial hygiene survey of styrene and other hazardous exposures in the fiberglass/reinforced plastics industry. A total of 141 workplaces were inspected, and 379 of the 2,600 workers employed in these workplaces were sampled over a full workshift [Ex. 3-742, Attachment 2]. The report also recommended the best control measures to minimize hazardous exposures; the focus of the study was on large open-mold sprayup/layup operations because earlier research had shown that these open-molding operations had the highest exposures of all operations in these workplaces [Ex. 3-742, Attachment 2]. Styrene exposures (8-hour TWAs) at these processes ranged from 0.2 to 288 ppm. The overall arithmetic and geometric means for these sample results were 43.0 ppm and 34.0 ppm, respectively [Ex. 3-742, Attachment 2], but exposures in some industries and for some processes were substantially higher.

The range of exposures in boat-building facilities was found to be 3.4 to 90.8 ppm (92 workers sampled); for workers in the recreational vehicle (camper) segment, this range was 7.3 to 130.3 ppm (48 workers sampled). Because few firms in the recreational vehicle and boat-building segments of this industry had adequate and effective ventilation controls, the California study concluded "that feasible engineering controls exist to reduce exposures to levels recommended by ACGIH and NIOSH of [a] 50-ppm TWA and [a] 100-

ppm excursion limit for styrene" [Ex. 3–742, Attachment 2, p. 36].

During the course of this rulemaking, OSHA conducted site visits to two boat-making facilities that use the open-mold process to build fiberglass boats [Exs. M–20, M–21]. These two sites were characterized by the industry as a facility that used traditional ventilation to control chemical exposures, and a facility that represented the "best available technology." In both facilities, both the full-shift and the excursion exposures of the gel-coat operators were below the proposed levels. However, the layup and sprayup processes in the traditional facility were conducted in an open area that was ventilated only with general dilution ventilation. In this plant, lamination employees' styrene exposures ranged from 61.9 to 341.5 ppm as 8-hour TWAs and from 98.7 to 311.0 ppm as 15-minute STELs [Ex. M–21]. In the "best available technology" facility, three-sided booths were used for the lamination operations. Here the lamination employees' exposures ranged from 36.7 to 93.8 ppm as 8-hour TWAs, with only one in three exposures below 60 ppm, and from 64.0 to 199.0 ppm as 15-minute STELs [Ex. M–20]. The additional control measures that are available, such as increasing the face velocity of exhaust equipment, may not enable this facility to reduce the exposures of its lamination workers from their current levels (ranging between 36.7 to 93.8 ppm) to levels within the proposed limits without interfering substantially with the correct consistency of the resin.

The extensive exposure and control data reported in the California study indicate that current styrene exposures are within, or can be controlled to, the Agency's proposed limits in some industries and occupations. These data, together with OSHA's on-site observations, are considerably less certain when it comes to the feasibility of the proposed limits for the large-volume open-mold processes necessary to produce boats and campers (as well as other large molded products in other industries).

The California data (Ex. 3–742, Attach. 2] and the OSHA data [Exs. M–20, M–21] showed somewhat different patterns of exposure. Whereas all industry and occupational subgroups of the California data had at least a substantial minority of exposures below 50 ppm, only one exposure observation for lamination in either facility visited by OSHA was below 60 ppm. The maximum 8-hour TWA for boat building observed in the California study was 90.8 ppm, and the large-mold boat manufacturers were described as having low average exposures [Ex. 3–742, Attachment 2]. At the facilities visited by OSHA, on the other hand, the maximum exposure was 341.5 ppm.

The large-scale open-mold processes were described in the California study as "intermittent," and the authors attributed the lower-than-expected styrene exposures to this characteristic [Ex. 3–742, Attachment 2]. SIRC notes that additional feasibility problems may arise in extreme environments, and that northern-tier states, where many boat builders are located, have winter climates that are quite different from that of California [Ex. 181A, p. 38]. A report submitted to the docket by the Wisconsin Department of Industry (Ex. HSP) concludes that many of the existing boat-building plants in Wisconsin will be physically unable to accommodate the complex controls needed to reduce employee exposures to styrene to below the 50-ppm TWA and the 100-ppm STEL (Ex. HSP). Since the plants visited by OSHA were in the Midwest, regional differences may help to explain the discrepant findings.

The authors of the Cal/OSHA study qualified their findings as follows:

> This study was conducted in a CAL/OSHA compliance mode: This represents two problems: (1) industrial hygienists do not have the luxury of making frequent visits to any one site, and (2) employees have an understandable desire to minimize actual exposures by various means . . . in order to avoid CAL/OSHA citations and fines [Ex. 3–742, Attachment 2, p. 31].

The California study notes that these factors may well have contributed to the relatively low mean exposures found in the study. However, OSHA notes that industrial hygienists were present in the plants long enough to demonstrate that high exposures can be controlled.

OSHA concludes that, in many operations within SIC 37, the proposed limits for styrene are feasible with the use of engineering and work practice controls. However, the record evidence demonstrates considerable uncertainty about the technical feasibility of achieving the 50 ppm TWA and 100 ppm STEL exclusively by means of engineering controls and work practices during manual sprayup/layup operations in this sector. Accordingly, OSHA concludes that the use of respirators as well as engineering and work practice controls may be necessary to achieve these limits in these manual operations, unless OSHA can demonstrate that engineering and work practice controls alone can achieve the PEL.

OSHA also received some comments regarding the feasibility of achieving the proposed 0.2 ppm PEL for MEKP in boat manufacturing facilities. Robert Schumacker, a certified industrial hygienist representing a group of six manufacturing companies (including the U.S. Marine Corporation), stated that information is lacking as to what concentrations of MEKP currently exist in the workplace, how to measure MEKP in the occupational environment, and the feasibility of engineering controls for reducing exposure to MEKP [Ex. 3–1172, Attachment; Exs. 8–86, 151]. The National Marine Manufacturers Association [Ex. 181] expressed similar opinions.

OSHA believes that the record contains substantial information demonstrating that the final rule's PEL of 0.7 ppm for MEKP is technologically feasible in boat manufacturing facilities. The record contains several NIOSH health hazard evaluations and technical assistance surveys conducted in workplaces where MEKP was used as a reaction catalyst in operations similar to those in boat building, including manual layup and sprayup operations (NIOSH: HE–79–132–673; TA–76–66; and HE–78–3–555). At three sites surveyed, all personal and area samples were below the proposed 0.7 ppm level. The NMMA [Ex. 181] reviewed these and other NIOSH reports (NIOSH: HE–79–092–629; HE–79–012–809) and noted that NIOSH recommended a number of engineering methods to reduce employee exposures to MEKP. These methods, which were supported by NMMA, included:

—Preventive maintenance on a scheduled basis for cleaning and changing filters on spray booths;
—Improvements in general dilution ventilation;
—Release of residual pressure from an MEKP container through the spray gun under local ventilation rather than through the pop-off valve.

OSHA also conducted two site visits to boat-building facilities in which MEKP was used [Exs. 136B]. One plant was a high-volume facility that produced 24 boats per day, while the other plant produced two to three boats per day. At both of these facilities, MEKP samples taken on gel coat and lamination workers were below the final rule's 0.7 ppm limit.

In regard to sampling and analytical methods for MEKP, OSHA notes that NIOSH has published a method (PECA or 3508) for this substance, and OSHA has developed an in-house method that is available from the Agency on request; the OSHA method was used successfully on the two site visits to MEKP-using facilities conducted for this

rulemaking. Therefore, based on the information contained in the record and summarized above, OSHA finds that the 0.7 ppm PEL for MEKP is technologically feasible in transportation facilities.

SIC 38—Instruments

Many fluxing agents are used in soldering and brazing operations during instrument manufacture. In most cases, these fluxes give off acid or alkali fumes when heated that can irritate the skin. Conducting soldering and brazing operations in well-ventilated areas and use of protective clothing and gloves is recommended.

For many soldering and brazing operations, general dilution ventilation will control fumes and vapors; that is, enough fresh air is added to the contaminated air that hazardous concentrations do not develop.

Local exhaust ventilation is the most effective means of control for airborne contaminants produced by the soldering or brazing process. Local exhaust ventilation can be provided by several types of equipment: freely movable hoods, fixed enclosures (booths), and down-draft benches.

A freely movable hood consists of a movable hood attached to a fan. The fan draws air from the work space and exhausts it outdoors, either directly or through a dust collection system. The hoods are normally constructed so that they can be moved into place by the solderer. The air handling system should move air at least 100 feet per minute across the soldering site at even the most remote point from the exhaust opening. It is important that the exhaust hood be placed as near as possible to the work being done. As such, the proper functioning of a freely movable hood is dependent upon good work practices of the solderer.

In some instances soldering or brazing operations carried out in a fixed location can be provided with a fixed enclosure. This is a structure built around the soldering or brazing operation which has a top and at least two sides. A means for drawing air through the work area is provided so that the work space is flushed continuously with fresh air.

Within such an enclosure, work should be arranged and conducted in such a way that the fresh air enters the enclosure through the worker's breathing zone and then through the work space in which the contaminants are produced. For most fixed enclosure, the air should move at least 100 feet per minute across the entrance to the enclosure.

A third type of level exhaust ventilation system is the down-draft bench or table. The soldering or brazing is performed on a bench or table which has an open grid as the work surface. Air is drawn downward through the grid, into the duct work, and then exhausted.

The Health Industry Manufacturers Association (HIMA) expressed support for "the phased-in period of compliance, which would allow the use of engineering controls, work practices, and respirators for a period of four years while employers evaluate and reduce/eliminate potential exposures to these substances in the workplace" [Ex. 3-910]. They raised no technological feasibility issue.

SIC 39—Miscellaneous Manufacturing

In the manufacture of hard surface floor coverings (SIC 3996), processes include pre-weighing and blending raw materials, followed by mixing and gelling of the composition in internal batch mixers of the Banbury type or by continuous mixing operations carried out in mixers of the extruder type.

Potential worker exposures may result from dusts of the raw materials as they are handled (automatically or manually) prior to and during charging of the mixer. Fumes and dusts can emanate from leaks on the mixer and from hot, freshly mixed material as it is discharged.

The types of exposures depend on the substances used. Applicable exposure controls include local exhaust ventilation at the mixer doors and over conveyor transfer points. The use of good working practices is extremely effective in controlling exposures during the opening of the mixers and the pouring of materials.

OSHA received one comment related to the issue of technological feasibility in the Sporting and Athletic Goods sector (SIC 3949). Robert Sigler, president of S.R. Smith, Inc., a manufacturer of diving boards, stands, and other reinforced plastics accessories for swimming pools, commented that his plant "will face severe economic hardship and possible closure" if the proposed 50 ppm limit for styrene is retained. Mr. Sigler believes this would be the case because "the entire layout of our manufacturing area and the entire ventilation system would have to be completely stripped and replaced" [Ex. 3-380].

OSHA has evaluated the technological feasibility of achieving compliance with a 50 ppm limit for styrene in reinforced plastics operations in several sectors (recreational boat manufacture, cultured marble tubs and showers, and underground storage tanks). Manufacture of fiberglass burial vaults (SIC 3995) is similar to these also. There is a considerable similarity among these various reinforced plastics operations: all involve the use of a styrene resin that is reinforced with fiberglass and applied by "chopper gun" and all involve manual layup and rolling. Thus, although the size and shape of the piece being built may vary, the exposure problems encountered by operators in these facilities are similar in nature. OSHA has determined that employers whose employees perform manual layup and rollup in reinforced plastics operations may need to use a combination of engineering controls, work practices and personal protective equipment to achieve the proposed styrene limit. OSHA's reasoning on this issue can be found in the previous discussion, under SIC 37, Transportation Equipment, and SIC 30, Rubber and Plastic Products.

The Casket Manufacturers Association of America (CMAA) submitted information to the record on the technological feasibility of achieving compliance with the Agency's proposed hardwood dust standard of 1 mg/m^3 [Ex. 8-78]. The CMAA reported that finishing operations, particularly machine and hand sanding of "white" wood caskets, often are associated with dust "levels 3-5 times higher than those in the furniture industry. . ." [Ex. 8-78]. In support of this position, the CMAA submitted two sets of exposure results: 9 results from samples taken specifically for this rulemaking, and 24 sample results described as "historical" and drawn from a variety of sources [Ex. 8-78].

Results from the CMAA's recent analysis ranged from 0.8 to 72 mg/m^3; however sampling times ranged from 90 minutes to 390 minutes. Seven of these nine recent samples showed results below 5 mg/m^3. Results from the historical set of samples ranged from 0.42 to 29 mg/m^3; sampling periods were even shorter than those for the recent set, ranging from 55 to 133 minutes [Ex. 8-78].

The data provided by the CMAA are not adequate to draw firm conclusions about the feasibility of achieving compliance with a 5 mg/m^3 standard for hardwood dust in the hardwood casket manufacturing segment. For example, these data cannot be used to evaluate employees' full-shift exposures because they do not represent 8-hour sampling periods. In addition, no job descriptions or task analyses are presented, and thus no deductions can be drawn about exposures for the unsampled portion of the day. In addition, no details are provided about the specific type of

wood involved in casket making, beyond stating that it is a hardwood.

However, OSHA believes that the final rule's 5 mg/m³ PEL for wood dust is already being achieved in hardwood casket making. The Agency's reasoning is as follows. First, OSHA believes that the results gathered by the CMAA for this rulemaking are more representative than the historical sampling data because they are more recent and generally involved longer sampling periods. Second, OSHA believes that these results reflect wood dust levels during hand or machine finishing operations because it is these operations that the CMAA is concerned about from the technological feasibility perspective. Third, an analysis of these recent results shows that, even using the worst-case assumption that employee exposures continue at the reported levels for the entire work shift (a highly unlikely exposure scenario), 7 out of 9 results would be below 5 mg/m³ as 8-hour TWAs. For example, the median 8-hour TWA exposure level for this group of samples under this worst-case scenario would be 2.34 mg/m³.

For these reasons, OSHA finds that hardwood casket manufacturers are already achieving the final rule's PEL of 5 mg/m³ in almost all cases, even in their dustiest operations (hand and machine finishing). Because Western red cedar is not used to make caskets, the Agency concludes that no casket makers will be affected by the final rule's 2.5 mg/m³ PEL for this allergenic wood dust. Thus OSHA finds no technological reasons for casket manufacturers to have difficulty complying with the final PEL for wood dust.

SIC 42—Motor Freight Transportation and Warehousing

Grain elevators whose primary income derives from the storage of grain are classified in SIC 42. Employees working in these elevators, have the same kinds of exposures as workers in other types of elevators, which are classified in SIC 51. OSHA's reasoning on the technological feasibility of achieving the proposed grain dust limit in all grain elevators is discussed fully in the technological feasibility section for SIC 51, below.

SICs 40, 45, 47—Transportation

Cleaning and coating operations are conducted in rail (SIC 40), and air transport industries (SIC 45), as well as in transportation services (SIC 47). These operations require the application of cleaning agents and/or the sandblasting of particles prior to the application of paints or coating. Spraying processes are required for the application of both the cleaning agents and the paints and coatings.

Rail car applications, for example, are generally performed within a large facility, part of which is established as a spray room. The cars are rolled into an enclosed spray area. In manual spray painting rooms, the operator is required to enter and move about the enclosure during spraying. Automatic spray rooms (or booths) are similar but the pressurized spray guns are automatically operated.

Three major spray techniques are used to apply cleaning agents, coatings or paints. These are: compressed air spraying (low-pressure spraying); airless spraying (high-pressure spraying); and electrostatic spraying. The compressed air spray gun atomizes a stream of liquid by impaction with a jet of air. Atomization may take place inside or external to the gun. The air stream and paint droplets intersect the prepared surface. The airless spray gun atomizes the liquid by forcing it through a small orifice under high pressure. The resulting particulate cloud is impelled by the pressure-created momentum toward the surface. Electrostatic spray equipment is based upon the attractive force between two oppositely charged objects. The liquid is atomized by compressed air, airless, or electrostatic techniques. The particles are given either positive or negative charge and the conductive surface to be sprayed is grounded. In general, electrostatic spray techniques result in the lowest exposure levels, followed by airless and then compressed air spraying.

In enclosed spray rooms, particulates enter the operator's breathing zone due to backspray. Exhaust ventilation to control exposure can be designed using down draft or a multiple sidedraft system. Worker positioning in relation to the spray plume is also critical in minimizing exposures. These include minimized line pressure, changing and cleaning of filter banks, enclosure integrity and ventilation maintenance. Personal protective equipment is also generally worn to insure the worker protection.

The industry representatives did not challenge the technological feasibility of the proposed PEL's. However, the Air Transport Association did object to the six month compliance period established by OSHA because of the unique character of the industry and the time required to establish proper controls [Ex. 3-1122]. OSHA recognizes this difficulty but believes that the six month/five year phase in period for complying with this rule, addresses this objection.

SIC 49—Electric, Gas, and Sanitary Services

Coal-fed power plants present the potential for exposure to coal dust as well as a number of other substances. Coal dust exposures potentially occur in the area where coal is fed into the furnaces. The coal is generally fed into large hoppers off conveyors. Conveyors are filled by front-end loaders from the coal storage area. The operators of the front-end loaders are protected from coal dust exposure with the use of closed, air-conditioned cabs which provide purified breathing air.

Evidence was presented by the Edison Electric Institute regarding technological feasibility during intermittent exposures. Dr. Louis Hosek, representing EEI, argued that "Many intermittent exposures occur in situations where engineering controls are likely to be substantially less feasible, both technologically and economically, than respirators, personal protective equipment, and work practices" [Tr. 8/11/88, pp. 228, 229].

This would clearly be the case in the tasks of cleaning the boilers and precipitators at electric power plants. Installing engineering controls to reduce exposures inside boilers would not be feasible. A power plant visited for this rulemaking had installed deluge systems for the precipitators to wash down as much fly ash as possible before workers could enter the area for cleaning or maintenance. Workers had to wear protective clothing and respirators when they cleaned the precipitator areas even after the wash system was employed [Ex. 11]. These tasks are occasional, performed maybe four times each year as the opportunity arises when the boilers are shut down. The crews used to perform the cleaning are as large as is practical so that the duration of the operation will be minimized. This is a maintenance situation where engineering controls would not feasibly achieve the exposure level and supplementary respiratory use is appropriate under OSHA's traditional policies.

The Edison Electric Institute questioned whether OSHA had found that compliance with the PEL for ozone was technologically feasible in the electric utility industry [Ex. 133, Tr. 8/11/88, pp. 232-233]. However, the Gulf Power Company, an electric utility, stated that, "Normal operating procedures would prevent exposure exceeding 0.3 ppm, since most operations occur in well ventilated

environments" [Exs. 3–938, 3–1144]. Therefore, OSHA concludes that the PEL for ozone is feasible.

SICs 50 and 51—Wholesale Trade

Some firms in this classification receive liquid chemicals in bulk quantities from a tank truck, store them and then redistribute them in smaller containers. Solvents, for example, emit considerable vapor when poured from one container to another or when a container is being filled, displacing the air in it. Pouring and filling operations are often enclosed to minimize vapor losses (this helps to reduce product loss as well as prevent exposures). In addition, secondary vapor recovery is often incorporated, whereby vapors emitted at the transfer points are captured and returned through a separate circuit to the storage tank from which the volatile liquid is being removed.

Grain dust exposures in this sector occur during grain handling operations in wholesale grain elevators. The majority of commenters on the technological feasibility of the proposed 4 mg/m³ PEL for grain dust (oats, wheat, and barley) maintained that this limit is not being met currently and cannot be met with available engineering controls [Exs. 8–55, 3–77, 3–201, 3–343, 3–347, 3–496, 3–1119, and 3–1196]. Typical of these comments is one from the Union Equity Coop Exchange, which stated that "over $9 million has been spent to install dust collection equipment in the facilities Union Equity currently operates. Thousands of dollars are spent monthly to maintain and operate this equipment. Many of these systems are state-of-the-art design for functional operation. None of these systems would allow any of our facilities to meet the proposed 4 mg/m³ exposure level" [Ex. 3–343, p. 2]. Edward X. Junia, Esq., representing the Andersons Management Corporation, was more specific: "there are certain operations in every grain-handling facility where there are no technically feasible engineering controls to reach such a level. The regular unloading and cleaning of storage bins/buildings and the housekeeping activities required under other OSHA standards are two areas where compliance through engineering methods is virtually impossible" [Ex. 3–77, p. 2].

OSHA does not agree that no controls are available to handle employee grain exposures during these operations. For example, in-plant vacuum systems (Farant and Moore, "Dust Exposures in the Canadian Grain Industry," *AIHAJ* 1978, pp. 177–193) would reduce exposures during housekeeping and maintenance; this control method should be used in lieu of manual sweeping or compressed air cleaning, two housekeeping methods that are still widely used in grain elevators (Ex. 3–751, Attachment, Docket H–0117).

Employers owning elevators that are operated in connection with feed mills (SIC 20) have found that the use of aspirators with filtration systems is highly effective in controlling grain dust during loading and unloading operations in receiving areas (Tr. 8/10/88, p. 10–73). To deal with the problem of grain dust in older mills, owners are replacing old-fashioned wooden legs with "good, tight, enclosed steel legs . . . old facilities . . . that had open grain drag conveyors . . . have been replaced in many cases with enclosed-type conveyors . . . the conveying systems that used to be open have lids on them . . . to keep the dust where it belongs" (Tr. 8/10/88, p. 10–80). Such enclosure is recommended by industrial hygienists whenever workers must work in dusty environments.

For some facilities, oil suppression of dust may be a useful control measure. An oil mist, which consists of mineral oil, vegetable oil, or some combination of the two, is normally applied when the grain is received at the mill. Ralph Mourer, testifying for the American Feed Industry Association, stated that oil suppression of dust is a promising control that he has just installed in his feed mills. Although he has not yet had much experience with the system, he noted: "[P]eople I've talked [to] and discussed the system with are very pleased" [Tr. 8/10/88, p. 10–78]. In an earlier study of grain-handling facilities for OSHA, however, Arthur D. Little, Inc., noted that there are some limitations to this process:

> Mineral oil is not approved for use as an additive on food grades of grain by the U.S. Food and Drug Administration. Vegetable oil may be an allowed additive, but its use can cause the grain to adhere into masses in cold climates. Further, there is concern that the oil will become rancid or create a commercially objectionable odor" (Docket H–0117, ADL, p. VI–34).

Scott Bjornsom from Hunter Grain in North Dakota also reported that oil suppression cannot be used for malting barley "because of the absorption with the water in the malt process" (Tr. 8/10/88, p. 10–85). Despite some limitations on its use in elevators, oil suppression appears to be an effective control for many elevators.

OSHA notes that the grain dust exposures of employees working in grain elevators classified in SIC 51 are sometimes below the 10 mg/m³ (the grain dust limit in the final rule) at the present time. A recent NIOSH study reports that only five percent of samples in the mills surveyed exceeded 10 mg/m³ (Rankin et al. 1986), and a NIOSH Health Hazard Evaluation from a Cargill elevator showed many sampling results that were below 10 mg/m³ or only slightly above this level (NIOSH HHE 76–13–316). These exposure levels are being achieved despite the fact that most grain elevators do not now have pneumatic dust control systems (RIA for the Grain Handling standard).

After considering the comments received on the proposed level, the controls available to reduce exposures and the impact on certain segments of the industry, OSHA has set the PEL for grain dust at 10 mg/m³.

OSHA believes that the controls described above, which are being installed in many elevators at the present time in response to the recent promulgation of OSHA's grain-handling standard, the recommendations of insurers, and the industry's concern for worker safety and health [Tr. 8/10/88, p. 10–73], are capable of achieving the 10 mg/m³ limit in those facilities and operations that are not now achieving this level. Industry representatives have reported that these systems have several additional benefits for employers; they improve productivity, have a positive effect on the quality of the grain, and create a better working environment [Tr. 8/10/88, pp. 10–80 and 10–81]. Thus, OSHA concludes that a variety of control strategies are available to employers operating grain elevators and these controls are installed in many elevators at the present time. The Agency finds that implementation of these controls will achieve the final rule's grain dust limit of 10 mg/m³ in those elevators and areas that have not already achieved this level.

SIC 72—Personal Services

To control dry cleaning emissions (SICs 7216, 7217), louvered wall fans and grilled ducts were installed to provide ventilation. Ceiling exhaust fans provided general ventilation. Natural ventilation was provided by through doors in the production area and by louvered panels along walls in the plant. Forced ventilation was provided by ceiling mounted exhaust fans and evaporative coolers. A local exhaust system with a standard single floor pickup exhausted air through a carbon absorption unit to the outside. Gaskets in machinery doors and ductwork needed routine maintenance to prevent deterioration. Various cleaning machines, pressure filter extractors and dryers were used. Dryers and drying

cabinets were provided with local exhaust ventilation.

In addition to the controls mentioned above, information has been reported by the Amalgamated Clothing and Textile Workers Union (ACTWU) which indicates that exposure to perchloroethylene is reduced when using the unitary dry-to-dry equipment (10.7 ppm for operator) as opposed to transfer-type dual washer/dryer equipment (58.4 ppm for operator) [Tr. 8/5/88, pp. 159–186]. ACTWU estimated that over 100,000 workers are exposed to perchloroethylene on a routine basis in the apparel cleaning industry. According to the 1982 Census of Service Industries, there were 13,049 dry cleaning plants in the U.S. that used perchloroethylene, with total employment of 89,896 workers. ACTWU calculated that approximately two-thirds of these workers are exposed in plants using transfer equipment, and one-third of these workers are exposed to perchloroethylene in plants using dry-to-dry equipment [Ex. 8–31]. The ACTWU also commented on the feasibility of reducing the exposure of perchloroethylene to substantially less than the 50 ppm proposed standard. Mitchell Brathwaite, an industrial hygienist representing the ACTWU, stated that OSHA "could reasonably propose a much lower PEL. . . . For instance, NIOSH reported that [the] mean exposure for 80 percent of [the] plant[s] study [studied] were below 50 ppm. In fact, machine operators in these plants had mean exposures of 22 ppm" [Tr. 8/5/88, p. 190].

NIOSH determined that "the 'combination washer/dryer' machines significantly reduce worker exposure to perchloroethylene when compared to exposures for separate or 'scanter' equipment" [Ex. 150]. Mr. Brathwaite further cited that based on NIOSH data (the Ludwig study) and Mount Sinai Hospital's Division of Occupational Safety and Health Data, "exposures could be reduced below ten ppm" in the dry cleaning industry with the utilization of the dry-to-dry equipment [Tr. 8/5/88, pp. 203–204].

The International Fabricare Institute (IFI) supported the proposed revision of the PEL for perchloroethylene at 50 ppm. They stated that approximately 64 percent of the dry cleaning industry uses dry-to-dry equipment, and that over the past four years about 95 percent of all new equipment sold has been dry-to-dry equipment [Ex. 3–671]. This indicates a continuing increase from 1982 when 35 percent of the firms had dry transfer machines. Based on this trend and the belief that all machines purchased in the future will be of the dry transfer type, all equipment in the dry cleaning industry will be dry-to-dry equipment. This will be accomplished through the normal replacement cycle. The ACTWU [Exs. 153G, 192] reports that the machinery census in Michigan for the period 1983–1988 indicates "there was a 34% increase in dry-to-dry machines, and an 11% decrease in transfer machines during this period. These data demonstrate vividly that replacement of transfer equipment with dry-to-dry equipment is not only technically feasible, but is indeed the economic choice of employers."

According to the testimony of Mr. William Fisher, vice president of IFI, the ambient perchloroethylene concentration in the cleaning area of a dry cleaning shop is approximately 20 to 30 ppm. The concentration decreases to approximately 10 to 15 ppm in the finishing area (at a range of approximately 15 to 20 feet from the cleaning area) to 1 to 3 ppm at the counter. "There can be some variations in those numbers. However, Ludwig's study from NIOSH indicated the same numbers as did the Westinghouse Behavioral Research Center study * * *" [Tr. 8/5/88, p. 281]. The actual concentration to which a person would be exposed is dependent upon the ambient environmental conditions and the ventilation characteristics.

Ludwig addressed the issue of engineering controls in dry cleaning facilities:

> The dryer is a closed system while in operation and the PCE-laden air leaving the dryer is passed over a water-cooled condenser for solvent recovery before the air is reheated and recirculated through the tumbler. . . . The processing equipment, whether a combination unit or separate washers and dryers, has interlock systems which insure that there is exhaust ventilation pulling air into the machines and out through ducting whenever the doors are opened. The recommended air velocity in through the loading door is an average of 100 feet per minute across the entire door opening.
>
> An activated charcoal adsorber is often added to the control scheme to remove PCE from the air exhausted from the washer during loading and transfer, and the dryer tumbler when the textiles are being aerated (deodorized), from the air intakes in the processing area, and from the vents of the muck cookers or stills. . . . The collection efficiency of the activated charcoal is extremely high (greater than 99%) up until breakthrough; however, if the adsorber becomes saturated, all PCE collected by the ventilation system will pass directly through the charcoal. It is for this reason that the adsorber should be vented to the outside of the building.
>
> Local exhaust ventilation in the processing area is also ducted to the adsorber. Ideally, the air intakes are between the level of the equipment doors and the worker's breathing zone. However, due to the mistaken notion that since PCE vapors are heavier than air they collect on the floor, most local exhaust intakes are at floor level; PCE vapors are likely to be found in high concentrations near the floor only if there has been a spill. Along with reducing PCE levels in the processing area, the use of local exhaust when ducted to the adsorber tends to cool the charcoal bed, thereby increasing its adsorption efficiency. Another type of ventilation utilized in some facilities is a low velocity fan 7' to 9' above the floor directed toward the center of the processing area. This concept, when combined with general room ventilation in which the fan is located on the wall or ceiling behind the dry cleaning area, results in reduced employee exposures not only to PCE but also to heat and humidity. A complete room air change every 5 minutes is recommended. Engineering controls such as exhaust ventilation of process equipment vented to a charcoal adsorber, local exhaust in the dry cleaning area, fans, and general room ventilation all contribute to lower ambient PCE concentrations. Also important is an active maintenance program. Typical sources of PCE vapor leaks are the button trap and the doors of the washer, dryer, cooker, or dryer lint trap. Most of these localized leaks are avoided by replacing door gaskets and adjusting the springs and hinges on the doors. Improperly seated air-inlet dampers on the dryer (used during aeration) and ducting are other potential sources of PCE emissions [Ex. 8–31, Appendix 13].

Data compiled by NIOSH, the dry cleaning industry, the ACTWU, and independent investigators demonstrates that virtually all employers can achieve exposure limits lower than the 50 ppm originally proposed by OSHA by using existing, readily available control technology. OSHA concludes, therefore, that a 25 ppm standard for perchloroethylene is feasible.

SIC 73—Business Services

Blueprinting and photocopying firms (SIC 7332) control ammonia fumes from blueprint duplication machines through use of local exhaust ventilation. The exhaust system is often built into large, high volume machines. Improvements in work practices control exposures during transfer.

The blueprint reproduction process uses ammonia to develop the image on the finished reproduction. Some machines are designed to contain the ammonia and its vapors; others are vented to the outside atmosphere. However, the odor of ammonia is present around the machines, especially where copies exit the machine and are trimmed [Tr. 8/5/88, p. 216, p. 228, p. 236]. Mr. Lucas Seeman, representing the Association of Reproduction Materials Manufacturers, discussed a survey

conducted in 1975–1976 of 75 blueprinting materials installations to determine levels of ammonia exposure found during blueprint reproduction. This survey found that, "the dominant part of this group was well under 25 ppm." Mr. Seeman stated that higher levels might occur "at the export end of some of the machines where the paper is coming out" [Tr. 8/5/88, pp. 228, 229]. OSHA concludes that local exhaust would be sufficient to bring these "export end" work stations into compliance with a 35 ppm STEL.

SIC 55,75—Automotive Repair Shops, Dealers

Exposure to carbon monoxide presents the major hazard in these industries. To control this in automobile engine reconditioning lines (SIC 7538), exhaust fans and flexible ducts which extend directly over the engines have been installed. OSHA received no comments on the proposed rule from this sector.

SIC 76—Miscellaneous Repair Services

Many repair services involve welding. In addition to techniques suggested in the discussion on welding and brazing in SICs 34 and 38, another control technique for welding fumes in SIC 7692 uses a "smoke exhaust" welding gun which captures and removes fumes. These guns have some limitations and are applicable to continuous or semicontinuous flux core or metal inert gas welding operations. Crossdraft airflow has also been suggested. The use of a portable fan is not recommended. OSHA concludes that it is feasible to control exposures to the final levels.

There were no docket comments on any aspect of this rulemaking for this sector.

SIC 80—Health Services

Many medical and dental practitioners perform surgery in outpatient clinics and private offices outfitted for the procedure. Air contamination in an operating room may consist of waste anesthetic, the propellants of different sprays, scrubbing agents, cleansing agents, ethylmethacrylate (released from surgical cement) and the possible decomposition products of the volatile or gaseous agents. The magnitude of gas flow, type of flow circuit and scavenging of waste gases significantly influence the levels of waste gases in the room air. Exposures are usually controlled by general dilution ventilation. Some clinics and offices, which are specifically designed for surgical use, may have local exhaust systems installed.

Glutaraldehyde is used in a limited number of applications, rather than as a general disinfectant. Specific applications include use as a disinfecting agent for respiratory therapy equipment, bronchoscopes, physical therapy whirlpool tubs, surgical instruments, anesthesia equipment parts, x-ray table-tops, dialyzers and dialysis treatment equipment. Presently there are no safer disinfectants which are as effective as glutaraldehyde.

Based on NIOSH Health Hazard Evaluations [1], OSHA concludes that the proposed ceiling limit for glutaraldehyde of 0.8 mg/m^3 (0.2 ppm) is technologically feasible. NIOSH states that those facilities and areas where exposures are below 0.2 ppm achieve these levels through the use of ventilation. NIOSH has found that through a combination of work practice improvements and engineering controls, levels below 0.2 ppm can be achieved. Specific recommendations include using increased dilution ventilation in whirlpool rooms and x-ray rooms, along with the careful application of the glutaraldehyde with a long-handled brush, rather than a spray applicator. NIOSH also recommends the construction of a workstation (similar to a lab hood) for cleaning surgical instruments and equipment parts.

OSHA received no comments on the impact of the proposed rule on facilities in this sector.

Personal Protective Equipment

In the operations and processes included in Table F–4 reductions in exposure limits can be achieved through engineering controls and work practice modifications. However, certain generic work activities are more problematical and may require the use of personal protection equipment. OSHA recognizes in 29 CFR 1910.1000(e), that respiratory protection can be an important adjunct to engineering controls. Because of specific task and process considerations, it may sometimes be necessary to augment engineering controls with the use of respiratory protection.

BILLING CODE 4510-26-M

Table F-3

INDUSTRIES AND PROCESSES WHERE SKIN PROTECTION HAS BEEN ADDED

SIC #	Chemical Name	Process Name
20	CAPTAFOL (DIFOLATAN)	Food Storage and Preservation
25	N-BUTYL ALCOHOL	Coating
26	METHYL ALCOHOL	Chemical Recovery
27	CYCLOHEXANONE	Plate cleaning
	FURFURYL ALCOHOL	Plate cleaning
	HYDROGEN CYANIDE	Plate making/Engraving
	METHYL ALCOHOL	Plate cleaning
28	N-BUTYL ALCOHOL	Blending, Packaging
	DIAZINON	Blending, Packaging
	METHYL ALCOHOL	Blending, Packaging
	METHYL PARATHION	Blending, Packaging
30	N-BUTYL ALCOHOL	Finishing, Trimming,Painting
31	DDT	Defestation/Disinfestion
	HYDROGEN CYANIDE	Beamhouse
	METHYL ALCOHOL	Finishing/Degreasing
	TRIORTHOCRESYL PHOSPHATE	Finishing/Degreasing
32	METHYL ALCOHOL	Batch preparation
	THALLIUM (SOLUBLE)	Batch preparation
	TIN	Float Process
33	HYDROGEN CYANIDE	Coremaking
34	N-BUTYL ALCOHOL	Coating/Painting
35	N-BUTYL ALCOHOL	Coating/Painting
	HYDROGEN CYANIDE	Soldering/Brazing
36	N-BUTYL ALCOHOL	Coating/Painting
	HYDROGEN CYANIDE	Soldering/Brazing
	MERCURY	Soldering/Brazing
	METHYL ALCOHOL	Cleaning
38	MERCURY	Handling of meaurement liquids Preparation of Special Tubes Assembling
	METHYL ALCOHOL	Blending/Packaging
39	N-BUTYL ALCOHOL	Painting, Coating

Table F-3

INDUSTRIES AND PROCESSES WHERE SKIN PROTECTION HAS BEEN ADDED

45	N-BUTYL ALCOHOL	Cleaning/Spraying
	METHYL ALCOHOL	Cleaning/Spraying
55	N-BUTYL ALCOHOL	Painting/ Coating
72	MERCURY	Embalming
	METHYL ALCOHOL	Embalming
73	DIAZINON	Exterminating
	DIOXATHION	Exterminating
	PHENOTHIAZINE	Exterminating
75	N-BUTYL ALCOHOL	Painting/ Coating
80	N-BUTYL ALCOHOL	Disinfectant and solvent use
	MERCURY	Preparation of amalgams
	SODIUM AZIDE	Laboratory analysis

Table F-4

PROCESSES TO BE CONTROLLED

SIC 20 -- Food Products	
Refrigeration/charging	Local ventilation: a hood exhausted to a baghouse, appropriate placement of cutoff values to freezer coils, an alarm detection system
Dry ice manufacture and use	Local ventilation: slotted hood exhaust system, adjustment of the number of air changes
Food storage and preservation	Local ventilation: slotted hood exhaust system, adjustment of the number of air changes
Grain elevators	Local ventilation, enclosure of the Boerner divider
SIC 21 -- Tobacco	Local ventilation
Cutting and shredding	
Flavor additive blending	
SIC 22 -- Textile Mills (except 2294)	Local ventilation, pressure failure alarms for closed systems, continuous flow indicators to indicate acceptable airflow
Wet methods, vacuum cleaning	
Weaving (SICs 2251,2295,2299 only)	
Dying/curing	
Coating/finishing	
Cutting	
Printing	
Spinning (SICs 228, 2299)	
Bonding (SIC 2295)	
SIC 2294 -- Processed Waste	Local ventilation, pressure failure alarms for closed systems, continuous flow indicators to indicate acceptable airflow

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

Wet methods, vacuum cleaning	
Processing of textile mill waste and fiber	
Fiber recovery from clippings and rags	
SIC 23 -- Apparel Products	
Bonding	Local ventilation, general ventilation
Dying	
Cleaning	
SIC 24 -- Lumber and Wood Products	Local ventilation: hoods or various types of negative pressure (or combinations of positive and negative pressure) devices; enclosed or hooded equipment vented to a baghouse, industrial vacuum system; enclosures: booth or cab supplied with filtered conditioned air
Drying/baking	
Coating/spraying/finishing	
Sanding/grinding/polishing	
Spraying/coating preservatives (SIC 2491 only)	
Cutting/sawing/planning	
Adhesive binding	
Gluing/hot pressing	
SIC 25 -- Furniture and Fixtures	
Gluing/hot pressing	Local ventilation
Coating/spraying/finishing	Local ventilation: downdraft spray booths, side draft ventilation; airless atomizing sprayers, electrostatic spray

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

Spraying/coating preservatives	Local ventilation: downdraft spray booths, side draft ventilation; airless atomizing sprayers, electrostatic spray guns on reciprocators; drums equipped with heavy barrel covers, an internal agitator, closeable access lines
Grinding	Local ventilation
Sanding/polishing	Local ventilation
Deburring	Local ventilation
Cutting/sawing/planing	Local ventilation
Layup/sprayup/coating	Local ventilation
Baking/drying	Local ventilation
Drilling/boring	Local ventilation

SIC 26 -- Paper and Allied Products

SIC 261 -- Pulp Mills

Digester	Enclosure, local ventilation
Pulp screening/washing	Ventilation and air purification in control rooms
Chemical recovery	Local ventilation
Bleaching	Ventilation and air purification in control rooms
Boilers	Local ventilation
Water treatment	Local ventilation, enclosure: storage of chemicals isolated and surrounded by dikes, rerouting of discharge lines

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

Recovery/reprocess/reclamation	Local ventilation, enclosure: storage of chemicals isolated and surrounded by dikes, rerouting of discharge lines
SICs 262, 263 -- Paper and Paperboard Mills	
Wet end Press section Drying	Local ventilation, enclosure: air-conditioned cabs or booths
Size press and coaters Calendars and winders	
SIC 264, 265, 266 -- Paperboard Products and Building Paper	
Mixing/blending (SIC 2641 only)	Local ventilation
Coating/finishing	Local ventilation
Gluing	Local ventilation
Drying	Local ventilation
Cutting/sawing/planing	Local ventilation
Packaging	Local ventilation with partial enclosure
Shredding/waste processing	Enclosure, local ventilation
Stamping/shaping/molding/pressing	Ventilation and air purification in control rooms
SIC 27 -- Printing and Publishing	Local ventilation
Printing process and plate cleaning Platemaking Photoengraving Gravure Lithographic (Offset)	

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

Screen stencil	
Letterpress	
Flexographic	
Intaglio	
Adhesive binding	
Mono or linotype setting	
Film processing	
SIC 28 -- Chemicals and Allied Products	
Reaction/fermentation	Local ventilation: enclosing and exhausting equipment, fitting vacuum crescents and elephant trunks on paint sources, fitting chutes with covers, placing vacuum attachments on receiving drum covers, using fixed ductwork as an exhaust, installing electronic-spent acid interface detectors; equipping vessels with hinged covers
Separation (many types)	Local ventilation
Crushing/grinding	Local ventilation
Loading/offloading	Local ventilation with partial enclosure: vapor recovery systems
Drying/baking	Local ventilation
Packaging/bagging	Local ventilation with partial enclosure: dust collection hoods
Reaction/fermentation	Local ventilation
Coatings/spraying	Local ventilation: portable hoods attached to flexible ductwork; enclosure: vented enclosures kept under negative pressure by a ventilation system

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

Blending/mixing/formulating	Local ventilation: enclosing and exhausting equipment, modification of hoods, fitting vacuum crescents and elephant trunks on paint sources, fitting chutes with covers, placing vacuum attachments on receiving drum covers, using fixed ductwork as an exhaust, installing electronic-spent acid interface detectors; equipping vessels with hinged covers
Impregnation	Local ventilation
Extrusion	Local ventilation
Recovery/reprocess/reclamation	Enclosure, local ventilation
SIC 29 -- Petroleum Refining	
Coke production	Worker enclosure, scrubber, computer control instrumentation, hardware modifications
Blending/mixing	Local ventilation
SIC 2911 -- Petroleum Refining	
Loading and unloading	Local ventilation with partial enclosure
Sampling	Local ventilation with partial enclosure: sampling boxes that vent gases and vapors away from operator and/or shield the operator from accidently splashed or spilled material

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

Process inspection and supervision	Local ventilation with partial enclosure
Quality control analysis	Local ventilation with partial enclosure: exhaust fans and ventilation hoods
Waste water treatment	Enclosure, local ventilation
Batch process coke production and removal	Worker enclosure, scrubber, computer control instrumentation, hardware modifications
SIC 2951 -- Paving Mixtures	
Materials receiving and handling	Local ventilation with partial enclosure
Measurement	Local ventilation
Drying/baking	Local ventilation
Mixing (Continuous/Batch)	Local ventilation
SIC 299 -- Miscellaneous Petroleum and Coal Products	
Materials receiving and handling	Local ventilation with partial enclosure
Blending/mixing	Local ventilation
Reprocessing or reclamation	Enclosure, local ventilation
Packing and loading	Local ventilation with partial enclosure
Adhesive binding	Local ventilation

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

SIC 30 -- Rubber and Miscellaneous Plastics Products	Local ventilation: hoods, automated batching systems, use of rubber bins rather than screw conveyors, substitution of chemicals
SIC 301 -- Tires and Inner Tubes	
Materials receiving and initial handling	Local ventilation with partial enclosure
Compounding and mixing	Local ventilation: hoods; automated batching systems, use of rubber bins rather than screw conveyors, substitution of chemicals
Vulcanization or curing	Local ventilation
Calendering and milling	Local ventilation
Solvent mixing and distribution	Local ventilation
Tire building	Local ventilation
Reblending/remixing	Local ventilation
Coating/spraying	Local ventilation
Stamping/shaping/molding/pressing	Local ventilation

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

SIC 306 -- Fabricated Rubber Products	
Materials receiving and initial handling	Local ventilation with partial enclosure
Blending, compounding, and mixing	Local ventilation: hoods, automated batching systems, use of rubber bins rather than screw conveyors, substitution of chemicals
Extrusion	Local ventilation
Coating/spraying	Local ventilation
Calendering and milling	Local ventilation
Vulcanization or curing	Local ventilation
Finishing, trimming, and painting	Local ventilation
SIC 307 -- Misceallaneous Plastic Products	
Material handling	Local ventilation enclosure
Blending, mixing and compounding	Local ventilation: hoods, automated batching systems, use of rubber bins rather than screw conveyors, substitution of chemicals
Calendering	Local ventilation
Molding and mold cleaning	Local ventilation
Assembly (including lamination, gluing, etc.)	Local ventilation
Foam processing	Local ventilation
Finishing, trimming, and painting	Local ventilation
Coating/spraying	Local ventilation
SIC 31 -- Leather and Leather Products (except SIC 311)	
Gluing and cementing	Local ventilation Work practice changes

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

SIC 311 -- Leather Tanning and Finishing

Preservation	General ventilation
Defestation and disinfection	General ventilation
Beamhouse	Local ventilation
Tanning	Local ventilation
Splitting and shaving	General ventilation
Neutralizing	Local ventilation
Retanning	Local ventilation
Coloring or dyeing	General ventilation
Fat liquoring	General ventilation
Drying	Local ventilation
Finishing (includes degreasing)	Local ventilation

SIC 32 -- Stone, Clay, and Glass Products

SICs 321, 322, 323 -- Glass

Batching	Local ventilation: industrial vacuums system/work practice changes
Melting	Local ventilation
Plate process (SIC 3211 only)	Enclosure
Sheet process (SIC 3211 only)	Enclosure
Float process (Sic 3211 only)	Local ventilation
Molding and blowing	Enclosure
Annealing	Local ventilation
Coating and etching	Local ventilation

SICs 324, 327 -- Cement, Concrete, Gypsum, Plaster

Gypsum, Plaster	Local ventilatin: local exhaust tubes, use of wet materials, industrial vacuum sytems
Crusher, grinder and sizing	
Blending	
Calcining kiln	

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

SIC 325, 326 -- Clay, Pottery	
Crushing, grinding, calcining	Local ventilation: local
Slip house (blending)	exhaust tubes, use of wet
Forming and shaping	materials, industrial vacuum
Biscuit firing	systems
Glaze application	
Gloss firing	
Decoration	
SIC 328 -- Stone	
Drilling, cutting, flame-jet lancing	Local ventilation
Chipping and grindig	
Surface polishing	
SIC 329 -- Abrasives	
Crusher, grinding, sizing	Local ventilation
Calcining (abrasives)	Local ventilation
Bonding	Local ventilation
Melting (Cupola furnace) and raw material handling	Local ventilation
Fiber forming (steam jet process, Powell process, Downey process, dry spinning)	Enclosure
Blowing/molding	Enclosure

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

SIC 33 -- Primary Metal Industries

SIC 331 -- Basic Steel Products

Coke Manufacture	Worker enclosure, scrubber, computer control instrumentation, hardware modification, enclosed vessels and process equipment
Ore Handling	Local ventilation
Blast furnace operation (including furnance charging BOF, ladle repair)	Local ventilation; vented to an electrostatic precipitator and/or baghouse, covering of runners; replacing older blast furnances; filtered air for work enclosure
Melting, pouring (electric arc or induction) (including electrode production/baking)	Local ventilation: roof-level hoods and ducts, ventilation to an electrostatic precipitator or baghouse
Hot shaping of metal (including rolling mill, metal extrusion, wire drawing, forging press)	Local ventilation
Annealing, quench and temper	Local ventilation
Pickling	Local ventilation
Hot dip galvanizing	Local ventilation
Cold rolling mill	Local ventilation
Abrasive blasting	Local ventilation
Grinding/polishing	Local ventilation
Degreasing	Local ventilation
Sintering	Local ventilation: hoods and sinter air cooler directed to a baghouse
Hot strip	General dilution ventilation enclosure: air-conditioned control stations

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

SIC 332 -- Iron and Steel Foundries	
Melting (electric arc or cupola (including electrode production and baking, charging and ladle repair)	Local ventilation: overhead canopy hoods, ventilation of arc air booths, mancooler fans enclosed crane operator cabs
Metal (sand) casting (or pouring)	Local ventilation
Investment casting	Local ventilation
Annealing, quench and temper	Local ventilation
Abrasive blasting	Local ventilation
Finishing (including: torch cutting grinding/polishing)	Local ventilation
Degreasing	Local ventilation
Shakeout	Local ventilation
Coremaking	Local ventilation: to provide negative pressure at the core box
Moldmaking	Local ventilation
Sand reclamation	Local ventilation, mechanical shakeout and automatic sand handling complete with dust collection, make-up air systems
SIC 333 -- Primary Nonferrous Metals	
Ore handling	Local ventilation: covers hoods, and exhaust systems for belts, material handling, and transfer systems, enclosing and exhausting equipment; enclosed air-conditioned control booth; computer controlled systems; movable nozzles; wet techniques in storage; general dilution ventilation; replacing the dross handling operation with a dross mill
Melting (electric arc or induction) including electrode production/baking, charging ladle repair, demagging (for Alum.plants only)	Local ventilation: slotted hoods, secondary converter system; induction furnaces; enclosed consoles; operating electric furnaces of negative pressure

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

Metal pouring	Local ventilation: hooding with air-operated doors, a dual draft system, an exhaust system vented to a dry scrubber;
Hot shaping (including rolling mill, forging, wire drawing)	Local ventilation
Annealing, quench and temper	Local ventilation
Degreasing	Local ventilation
SIC 334 -- Secondary Nonferrous Metals	
Melting (electric are or induction) including electrode production/baking, charging ladle repair, demagging (for Alum. plants only)]	Local ventilation: slotted hoods, secondary converter hoods, converter gas handling system, induction furnaces; enclosed consoles; operating electric furnaces at negative pressure
Metal casting or pouring	Local ventilation
Forging press (SIC 334, only)	Local ventilation
Torch cutting	Local ventilation
Raw materials preparation (SIC 334, only) [including metal preheat borings dryer) scrap shredder, slag recovery]	Local ventilation: industrial vacuum systems, pneumatic aerators, use of deadbeds, eliminate air lancing.
Degreasing	Local ventilation
SIC 335 -- Nonferrous Rolling and Drawing	
Hot shaping (including rolling mill, wire drawing, metal extrusion)	Local ventilation
SIC 336 -- Nonferrous Foundries	
Melting (electric arc or induction) (including electrode production/baking, charging, ladle repair)	Local ventilation

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

Metal (sand) casting (or pouring)	Local ventilation: hooded enclosures, flexible ducting connecting a mobile hood to a traveling exhaust carriage
Investment casting	Local ventilation
Annealing, quench and tempter	Local ventilation
Pickling	Local ventilation
Abrasive blasting	Local ventilation
Grinding/polishing	Local ventilation
Degreasing	Local ventilation
Shakeout	Local ventilation
Coremaking	Local ventilation
Moldmaking	Local ventilation
Sand reclamation	Local ventilation

SIC 339 -- Miscellaneous Primary Metal Products

Sintering	Enclosure; local ventilation: use of closed screw conveyors, baghouse, and electrostatic precipitators
Strip annealing	Local ventilation: use of closed screw conveyors, baghouses, and electrostatic precipitators
Bimetal production	Local ventilation: use of closed screw conveyors, baghouse, and electrostatic precipitators

SIC 34 -- Fabricated Metal Products

Electroplating	Local ventilation

SIC 341, 342, 343, 348 -- Cans, Cutlery, Hand Tools
Heating Equipment Ordnance

Pressing	Local ventilation
Acid washing	Local ventilation
Degreasing	Local ventilation

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

Painting and coating	Local ventilation
Electroplating	Local ventilation
Welding	Local ventilation: a welding bench with a backdraft hood, a fixed close-capture hood placed at the back of the work table, a portable close-capture system including an electrostatic precipitator, an, exhaust hose incorporated into the structure of the welding gun; ambient air cleaning devices; a portable blower for use in confined areas
Grinding/polshing	Local ventilation
Abrasive blasting	Local ventilation
Hot shaping (including: rolling mill, wire drawing, metal extrusion)	Local ventilation
SIC 344 -- Structural Products	
Painting and Coating	Local ventilation
Welding	Local ventilation; welding bench with a backdraft hood, a fixed close-capture hood placed at the back of the work table, a portable close-capture system including an electrostatic precipitator, an, exhaust hose

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

	incorporated into the structure of the welding gun; ambient air cleaning devices; a portable blower for use in confined areas
Grinding/polishing	Local ventilation
Abrasive blasting	Local ventilation
Acid washing	Local ventilation
SIC 345, 347 -- Screw Machine Products	
Coating and engraving	Local ventilation
Coating (enamels, lacquers, varnishes) (SIC 347 only)	Local ventilation
Hot dip galvanizing (SIC 347 only)	Local ventilation: two-sided lateral exhaust system, two-sided slot ventilation system, a cover which is hinged to a ventilation manifold
Engraving and etching (SIC 347 only)	Local ventilation
Degreasing	Local ventilation
Grinding/polishing	Local ventilation
Hot shaping (SIC 345 only)	Local ventilation
Acid washing	Local ventilation
SIC 346 -- Iron and Steel Forgings	Local ventilation
Hot shaping	
Acid washing	
Pressing	
SIC 35 -- Machinery	Local ventilation
Pressing	Local ventilation
Acid washing	Local ventilation
Degreasing	Local ventilation

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

Painting and coating	Local ventilation: downdraft spray booths
Electroplating	Local ventilation
Grinding/polishing	Local ventilation
Abrasive blasting	Local ventilation
Welding	Local ventilation; welding bench with a backdraft hood, a fixed close-capture hood placed at the back of the work table, a portable close-capture system including an electrostatic precipitator, an, exhaust hose incorporated into the structure of the welding gun; ambient air cleaning devices; a portable blower for use in confined areas; an air lux fume eliminator
Hot shaping (including: rolling mill, wire drawing, metal extrusion	Local ventilation
Soldering (SIC 357, only)	Local ventilation
Refrigerant charging (SIC 358, only)	Local ventilation
SIC 36 -- Electric and Electronic Equipment	Local ventilation
Cleaning	
SICs 361, 362, 363 -- Transmission Distribution; Industrial Household	Local ventilation
Pressing	
Acid washing	
Degreasing	
Painting and coating	

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

Regrigerant cooling	
Electroplating	
Grinding/polishing	
Abrasive blasting	
Welding	
Soldering	
Epoxy coating	
SIC 364 -- Lighting and Wiring	Local ventilation
Wire drawing	Local ventilation
Patenting	Local ventilation
Descaling	Local ventilation
Coating	Local ventilation
Extrusion	Local ventilation
Cleaning	Local ventilation
Coil production	Local ventilation
Coating and drawing	Local ventilation
Gas filling	Local ventilation: increase overhead suction velocity, industrial vaccum systems
Glass blowing	Local ventilation
Soldering	Local ventilation
Glassmaking	Local ventilation
SIC 365, 367, -- Radio, TV: Communications; Electronics	Local ventilation
Semiconductor-photoresist stripping	
Semiconductor-chemical etchants	
Semiconductor-diffusion and ion implant	
PC-boards-etching	
PC-boards-soldering	
Mixing of cermanic powders	

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

SIC 369 -- Miscellaneous Electrical	Local ventilation
Ingredients grinding	
Mixing	
Casting	
Assembly	
SIC 37 -- Transportation Equipment	Local ventilation
Metal melting	Local ventilation
Metal pouring	Local ventilation
Hot metal working (rolling, shaping or drawing)	Local ventilation
Metal machining or grinding	Local ventilation: hoods, exhaust fans, "upblast" roof ventilator fans to change airflow
Welding or brazing	Local ventilation; a four sided enclosure with electrostatic precipitator ventilation
Solvent or vapor degreasing	Local ventilation
Painting or coating	Local ventilation: enclosed booths, three-sided booths for lamination
Degreasing/cleaning	Local ventilation
Electroplating or electrical discharge manchinery	Local ventilation
SIC 38 -- Instruments	Local ventilation
Forming/fabricating of metal	Local ventilation
Welding	Local ventilation
Injection molding	Local ventilation
Handling of measurment and testing liquids, gases, materials	Local ventilation
Quality control testing	Local ventilation
Foaming, packaging	Local ventilation
Coating, painting	Local ventilation
Sterilization	Local ventilation
Film and pring papermaking and coating (SIC 3861 only)	Local ventilation

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

SIC 39 -- Miscellaneous Manufacturing Industries

Roughing milling or sawing	Local ventilation
Sanding	Local ventilation
Gluing	Local ventilation
Finishing or staining	Local ventilation
Welding, casting, brazing	Local ventilation
Hot metal work	Local ventilation
Mono or linotype setting	Local ventilation
Abrasive blasting	Local ventilation
Degreasing	Local ventilation
Electroplating	Local ventilation
Machining	Local ventilation
Blending, mixing or compounding	Local ventilation
Molding or mold cleaning	Ventilation and air purification in control rooms
Foam processing	Local ventilation
Painting/cooling	Local ventilation
Metal melting and pouring	Local ventilation
Pressing	Local ventilation
Stamping/shaping/molding/pressing	Ventilation and air purification in control rooms
Cutting/sawing/planning	Local ventilation
Lacquering/enameling	Local ventilation
Bristle/fiber cleaning	General ventilation
Metal plating	Local ventilation
Engraving/etching	Local ventilation
Acid washing	Local ventilation
Hot dip galvanizing	Local ventilation
welding, degreasing, metal working sand blasting	
Engine fueling	General ventilation
Handling spills, leaks	Local ventilation

SIC 45 -- Transportation by Air

Loading/offloading	Local ventilation
Maintenance-related activities: Cleaning/coating/spraying welding, degreasing, metal working, sand blasting	Local ventilation: spray room

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

Engine fueling	General ventilation
Handling spills, leaks	Local ventilation
Fuel preparation	Local ventilation with partial enclosure
Deicing	Local ventilation
Refueling	General ventilation
Painting/coating	Local ventilation
SIC 47 -- Transportation Services	
Loading/offloading (SIC 4742 only)	Local ventilation with partial enclosure
Maintenance related activities	Local ventilation: spray room
Engine fueling and fumes	General ventilation
Handling spills, leaks	Local ventilation
Special care of lading service	General ventilation
SIC 49 -- Electric, Gas, and Sanitary Services	
Maintenance related activities	Local ventilation
Boiler furnace feed	Enclosure, local ventilation
Stripping of chemicals	Local ventilation
Collection/transport	Respirators
Engine fueling	General ventilation
Odorant addition	Local ventilation
Condensate collection	Local ventilation
Incineration (SIC 4953, only)	Local ventilation
Detoxification (SIC 4953, only)	Local ventilation
Recycling, reclamation (SIC 4953 only)	Enclosure, local ventilation
Chemical preparation/application	Local ventilation
Sampling of pipelines	Local ventilation with partial enclosure
Water purification	Enclosure, local ventilation
Water treatment	Enclosure, local ventilation

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

SIC 50 and 51 (except 5093) -- Wholesale Trade	
Materal handling, shipping and receiving	Local ventilation with partial enclosure, secondary vapor recovery
Material packing or repacking	Local ventilation
Grain elevators	Local ventilation
SIC 5093 -- Scrap and Waste Materials	
Assembling and collecting scrap and waste materials	Respirators
Breaking up waste materials	Enclosure, local ventilation
Sorting scrap and waste materials	Enclosure, local ventilation
Baling or compacting	Enclosure, local ventilation
SIC 72 -- Personal Services	
Washing (SIC 721 only)	General ventilation
Dry cleaning (SIC 721 only)	Local ventilation: equipment change; exhaust ventilation of process equipment vented to charcoal adsorber, louvered wall fans and grilled ducts, louvered wall panels, evaporative coolers; general ventilation: ceiling exhaust fans
Manicure/pedicures (SICs 723 and 724 only	General ventilation
Permanents (SICs 723 and 724 only)	General ventilation
Coloring (SICs 723 and 724 only)	General ventilation
Embalming (SIC 726 only)	General ventilation
SIC 73 -- Business Services	Local ventilation
Blueprint copying (SIC 733 only)	
Exterminating (SIC 734 only)	
Photofinishing (SIC 739 only)	

Table F-4

PROCESSES TO BE CONTROLLED
(continued)

SIC 55 75 -- Automotive Repair Shops, Dealers	
Confined space - exhaust fume	General ventilation: exhaust fnas and flexible ducts
Welding	Local ventilation
Paint stripping	Local ventilation
Cleaning with solvents	Local ventilation
Painting/coating	Local ventilation
Other solvent use	Local ventilation
SIC 76 -- Miscellaneous Repair Services	Local ventilation
Welding or brazing	Local ventilation: "smoke exhaust" welding gun, crossdraft airflow
Paint stripping	Local ventilation
Sanding or grinding	
Gluing	Local ventilation
Painting, coating or lacquering	Local ventilation
Other solvent use	Local ventilation
SIC 80 -- Health Services	Local ventilation, general dilution ventilation
Administration of anesthesia	Local ventilation
Preparation of dental amalgams and alloys	Local ventilation
Laboratory procedures such as tissue staining	Local ventilation
X-ray film processing	Local ventilation
Use of disinfectants, solvents	Local ventilation, respirators
Making of dental appliances	Local ventilation

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Exterminating: Exposure of pesticide applicators cannot be controlled through engineering controls because their work does not take place in a fixed place of employment, but rather at a customer's facility. Personal protective equipment and/or work practice controls would therefore be required. EPA has jurisdiction in most situations.

Welding: In certain situations, such as in confined spaces, or where the welder must be positioned directly above the fume plume, welders cannot be sufficiently protected by local exhaust ventilation. Personal protective equipment would be required.

In addition to these general industry operations, certain industry specific situations have been identified where the use of respirators is recognized as an important complement to other control measures. These situations include the following:

- During exposure to carbon disulfide in the cellulosic food casings industry.
- During exposure to carbon disulfide while changing spinerettes, removing filament bundles and making product line changes in the manufacture of rayon fibers.
- During episodic emissions of sulfur dioxide in the smelting of copper.
- During manual layup/sprayup operations using styrene.
- During episodic emissions or intermittent worker exposures of such fumes as carbon monoxide or sulfur dioxide from blast furnaces or BOF.

In addition to the above examples, a number of the substances included in this rulemaking carry the designation "Skin." This refers to potential exposure though the skin. Table F–3 presents a list of chemicals for which skin protection would be required. Employees exposed to substances with the "Skin" notation would be required to wear protective equipment, including gloves, long sleeved shirts and coveralls.

Products are commercially available to adequately protect workers from dermal exposure. In some cases the permeability of currently used materials may be inadequate and firms will have to change the specific product now used to one offering greater protection.

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Examples of these are:

Maintenance Activities in all SIC codes. In certain cases, it may be more difficult to control exposures of plant maintenance personnel by using engineering controls. These maintenance employees may work in areas not normally covered by engineering controls or in situations where engineering controls must be shutdown. Respiratory protection is therefore sometimes the appropriate control technology.

Painting and Coating Activities in all SIC codes. Although production spray painting operations are performed in exhausted paint booths, the painting of many larger non-production items, such as construction equipment and heavy machinery, requires that the operator enter the booth. The booth is then primarily a control to prevent migration of the paint spray into other areas of the plant. In these circumstances it is usually necessary to provide respiratory protection to the workers painting.

G. Costs of Compliance

Costs of compliance result from the purchase, installation, operation and maintenance of equipment to maintain workers' exposures at or below the levels specified in the final standard. Costs are related to the engineering controls and personal protective equipment needed for specific processes which involve the use of hazardous substances. Given the large number of substances being regulated, the cost assessment was required to examine a large number of processes over many industry segments. The approach needed to be generic in scope and specific in detail. OSHA has reviewed this approach and the resulting cost analysis and incorporated extensive public testimony and voluminous docket submissions. The Agency concludes that the costs presented in this chapter accurately reflect industries' requirements for compliance.

Existing data sources and expert judgment were initially used to sort the approximately 430 substances being regulated, by industry and by process within industry segments. Given the large number of substances being regulated, a process of orientation rather than a chemical-specific focus was recommended, since prescribed engineering controls can address worker exposure problems to several chemicals, involving the same general process, simultaneously. The approach has proven to be efficient analytically and reduces the problem of double counting the costs of similar or the same engineering controls for separate chemicals involved in the same process or operation.

OSHA had a large amount of exposure data in its Integrated Management Information System (IMIS) and from NIOSH and other sources. But to improve the available information on the use of substances, OSHA decided to engage in a nationwide field survey of affected establishments. This survey, involving about 5,700 establishments in both manufacturing and non-manufacturing sectors, has provided valuable information on chemical usage by industry process and potential worker exposures to these chemicals. Supplement 1 contains a description of the sample survey design and a statistical evaluation of the data collected.

In order to maximize the efficiency of this nationwide sample survey and limit the number of required sample observations per SIC category, a considerable effort was made to verify chemical by industry usage from existing data sources and to make best estimates of where likely or potential worker exposure problems (and consequently engineering costs) existed. For the purposes of the statistical survey being conducted, the larger the suspected potential exposure/cost problem in a particular industry sector, the more important it was to insure a large enough sample of firms in that sector so as to reduce the standard error of the cost estimates.

The following sections of this chapter outline the methodology adopted to identify:

- Chemicals by their industrial usage and employee exposures
- Processes involving known or suspected chemical exposures and control costs
- Industry costs for the controls needed to reduce industry exposure levels.

Linking Hazardous Substances by Industry Use and Employee Exposure

Figure G–1 presents a flow chart of the methodology used for identifying chemicals by industry use and employee exposure. The first step in the methodology was an analysis of the chemicals for which OSHA proposes new exposure limits. The 1982 NIOSH National Occupational Exposure Survey (NOES) and the OSHA IMIS data files were searched to determine the potential for worker exposure to each of the chemicals on the proposed list. The objective of this analysis was to create a subset of chemicals which are known to be present in specific industries at exposure levels above the proposed limits. These chemicals would then be considered to generate potential compliance costs within a specific industry sector.

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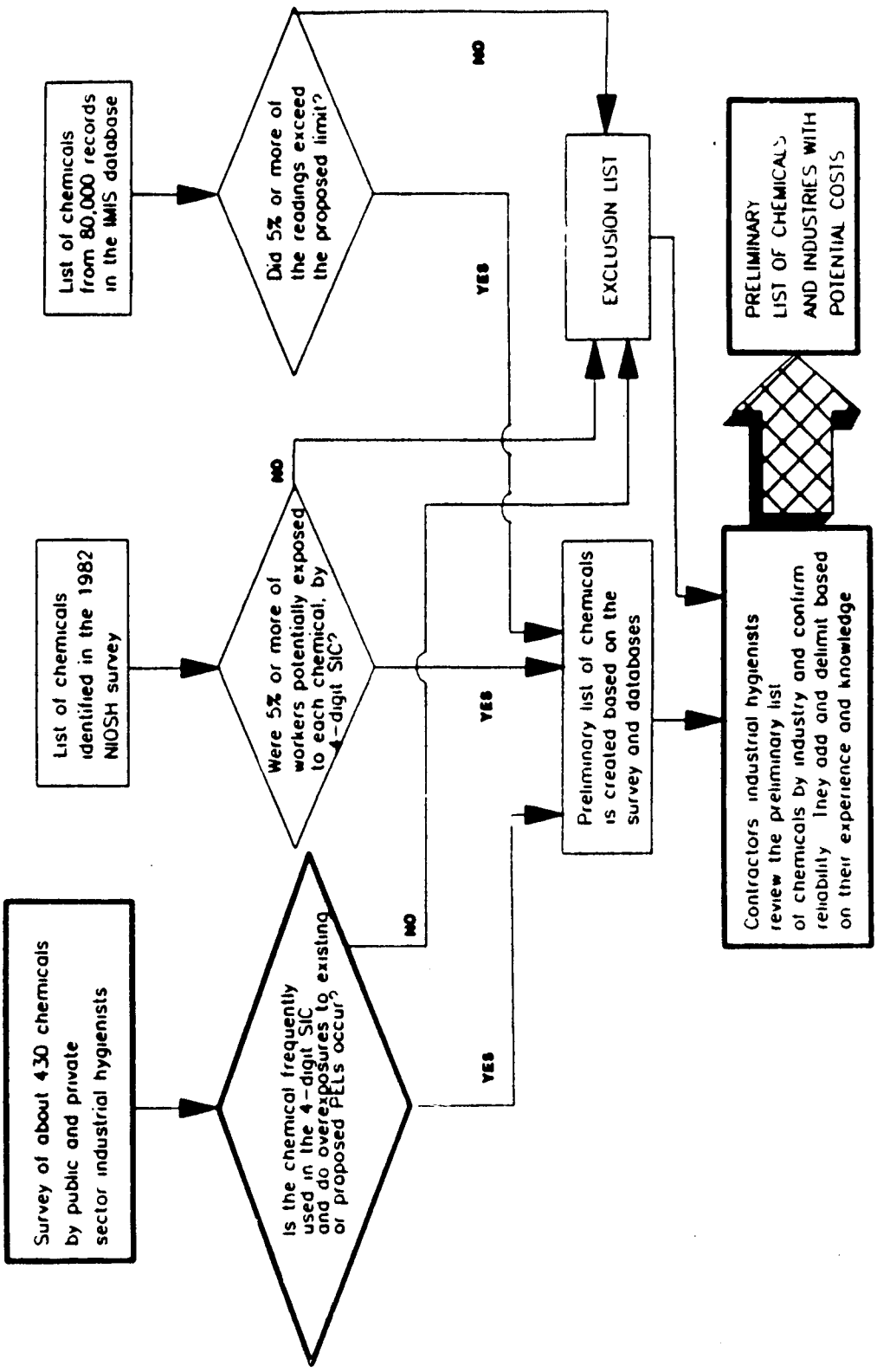
Figure G-1

METHODOLOGY FOR IDENTIFYING CHEMICALS WITH POTENTIAL COST IN EACH 4-DIGIT SIC



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The 1982 NIOSH National Occupational Exposure Survey (NOES) data (supplemented by results from NIOSH's 1972 survey) provided an estimate of the number of workers potentially exposed to a specific chemical in a four-digit SIC. OSHA divided this estimate by the total number of employees in an industry segment to get a percentage of workers potentially exposed to that chemical. If 5 percent or more of the workers were potentially exposed, that chemical was considered to present a potential cost within the four-digit SIC. For example, in SIC 3011, Tires and Innertubes Manufacturing, 1,532 persons were potentially at risk of exposure to n-hexane at the sample of plants included in the NOES database. This represented 21.9 percent of all workers sampled in the four-digit industry sector and this chemical would have a potential cost impact depending upon current exposure levels.

From the OSHA IMIS data, the severity of exposure within a four-digit SIC was estimated. OSHA compared the total number of monitored readings for each chemical with the number of readings which exceeded the proposed limits and calculated the percentage of all sample monitor readings which were above the proposed limits. If there were no readings which exceeded the proposed limits, the chemical was not considered to have a compliance cost within the four-digit SIC. If 5 percent or more of the readings exceeded the proposed limits, then the chemical was identified as having a potential compliance cost within the four-digit SIC. For example, in SIC 2641, Paper Coating and Glazing, 22 samples were taken for n-hexane. Thirteen of these, or 59 percent, were above the proposed standard for n-hexane. This chemical, therefore, was believed to have a potential cost impact and questions regarding its use were included in the field survey. Chemicals with non-compliance percentages between zero and 5 percent were evaluated individually by industrial hygienists to determine whether or not specific survey questions needed to be asked about their industrial usage.

In addition to the IMIS and NOES databases, a survey of about one dozen industrial hygienists was conducted. The purpose of this survey was to identify any additional hazardous substances or industry sectors not identified in the IMIS or NOES databases with potential exposure problems at new recommended levels. For example, in SIC 2891, Adhesives and Sealants Manufacturing, the surveyed industrial hygienists reported that n-hexane overexposures could exist under the proposed standard. (Overexposures in SIC 2891 were not previously identified in the IMIS or NOES databases.)

The information from all sources was combined to compile a preliminary list of substances with potential compliance costs by four-digit SIC classification. To further refine the list of chemicals, a second group of six industrial hygienists, using personal industry knowledge and the information gathered from the survey of the initial group of industrial hygienists, reviewed once again the chemicals which appeared in the NOES and IMIS datasets. They also made chemical by industry use linkages when particular chemicals were known to be present in certain SICs, but had not been identified in the NOES and IMIS databases.

Upon completion of the two-tier industrial hygienist review, a list of chemicals believed to be present at exposure levels above the proposed standard, within specific four-digit SIC industry sectors was finalized. This list identified those industry segments where potential compliance costs would be incurred to achieve the proposed standards. The presence of the identified chemicals was confirmed by survey respondents. The method used during the survey listed likely chemicals and asked for any other chemicals present.

Industrial Processes and Control Costs

The number of industrial processes, exposure levels, and exposure controls in place varies greatly within industry segments. In order to efficiently structure the statistical sample of surveyed firms, it was necessary to make a best estimate of which industry segments were likely to experience compliance costs. As noted above, the survey was designed to limit the standard error for potential high cost industry sectors. To concentrate the survey on the potential high cost sectors, a process orientation was adopted which supplemented and refined the chemical use information. The validity of this approach was confirmed in the review of docket materials. The vast majority of submissions that addressed industry costs linked process operations with compliance cost. Industry sectors having few processes and chemicals and low potential exposure levels (and consequently low potential compliance costs) were included in OSHA's secondary data collection and evaluated by experts, but not included in the sample survey.

A team of engineers and industrial hygienists analyzed each four-digit SIC to assess the process in which worker exposure to listed chemicals occur. Examples of industrial processes included grinding, mixing, spraying, degreasing, separation, bagging and loading. A list of potential cost chemicals and related processes was then developed to identify potentially high impact (cost) industries. The presence of the identified processes was confirmed by survey respondents. The method used during the survey listed likely processes and asked for other processes ongoing at the establishment. In general, an industry segment with a relatively large number of processes using chemicals with suspected high exposure levels was sampled at the three-digit industry level. Industries with fewer processes and low chemical exposures were sampled at the two-digit level. (See Supplement 1 for a more detailed explanation of the survey design.)

Approximately 5,700 respondents in the survey were asked to verify the chemicals used, manufactured or generated by process within their establishment. Thus, chemicals were linked to specific processes, process controls and workers exposed at the process in the surveyed industries. Control methods and costs were then assigned for each process where employee exposures would exceed the proposed PELs.

Controls were assigned to protect workers exposed to all chemicals in total at a process. The controls were designed and costed to lower exposure to the chemical(s) with the greatest change in the permissible exposure limit (PEL). It was the judgment of the experts involved that by assigning controls for the "major" chemicals, exposures for all other chemicals would be controlled. Chemicals and/or processes not included in the proposed standard (e.g., those covered by separate 6(b) rulemaking) were excluded from the survey. Examples of chemicals not included in the survey are asbestos, formaldehyde and benzene.

Survey information collected from each respondent included:

- Type of processes at the establishment;
- Type and amount of chemical used, manufactured, or generated in each process;
- Number of work stations and workers related to the process;
- Potential chemical exposure above the proposed standards (monitoring data, recorded overexposures) at the process;

- Process location (indoors/outdoors), and configuration (size, full enclosure, partial enclosure);
- Ventilation or other controls in place; and
- Economic and other characteristics of the plant.

A computer algorithm was developed to assess survey data to determine if potential worker overexposure and therefore compliance costs occur for each process at an establishment. Figure G–2 presents a general diagram of the computer logic adopted for use in the survey. The logic assesses potential overexposures on the basis of: actual reported monitoring data; statements that overexposures occur; and the particular process location, configuration, type and amount of chemical use and existing controls in place.

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FIGURE G-2

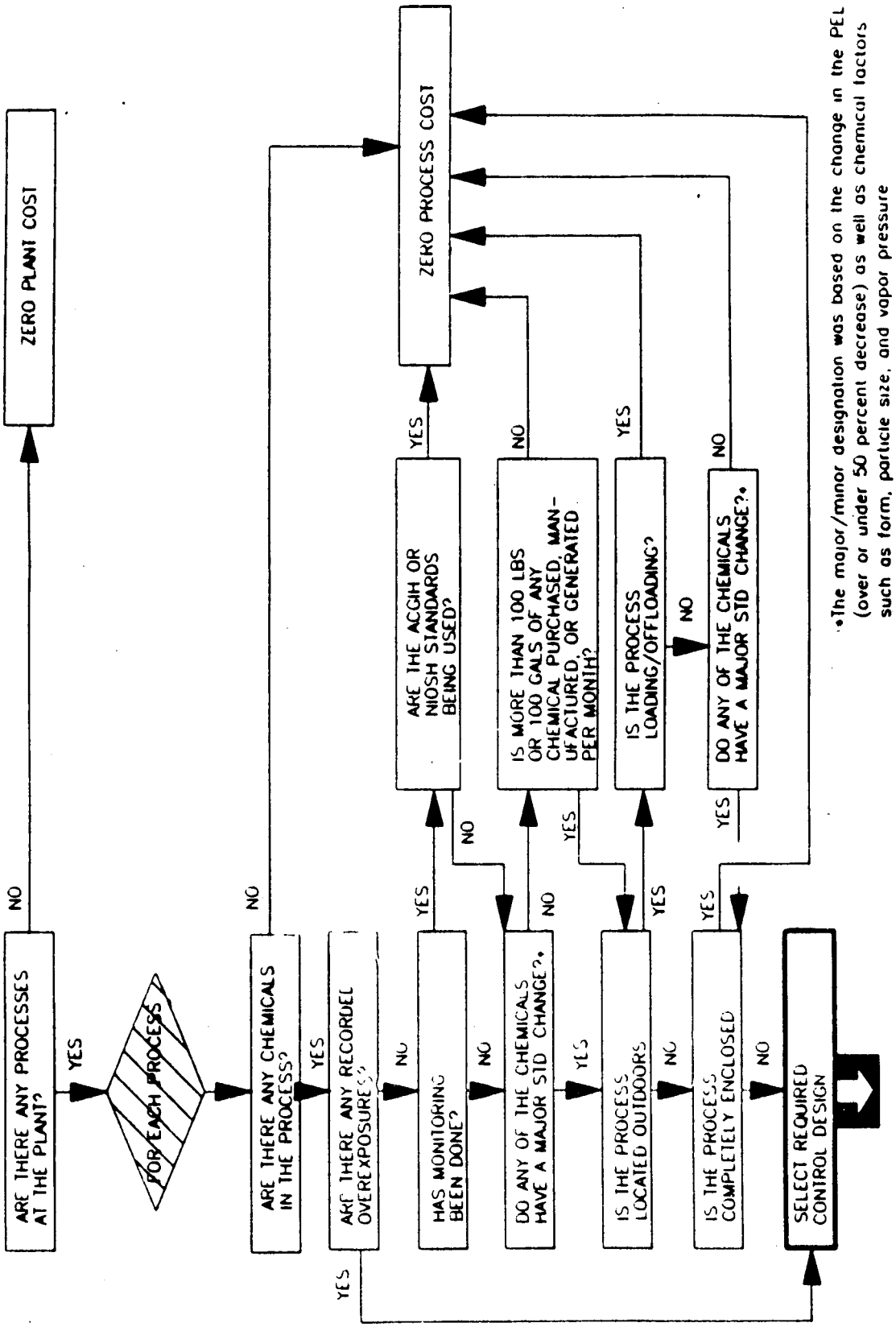
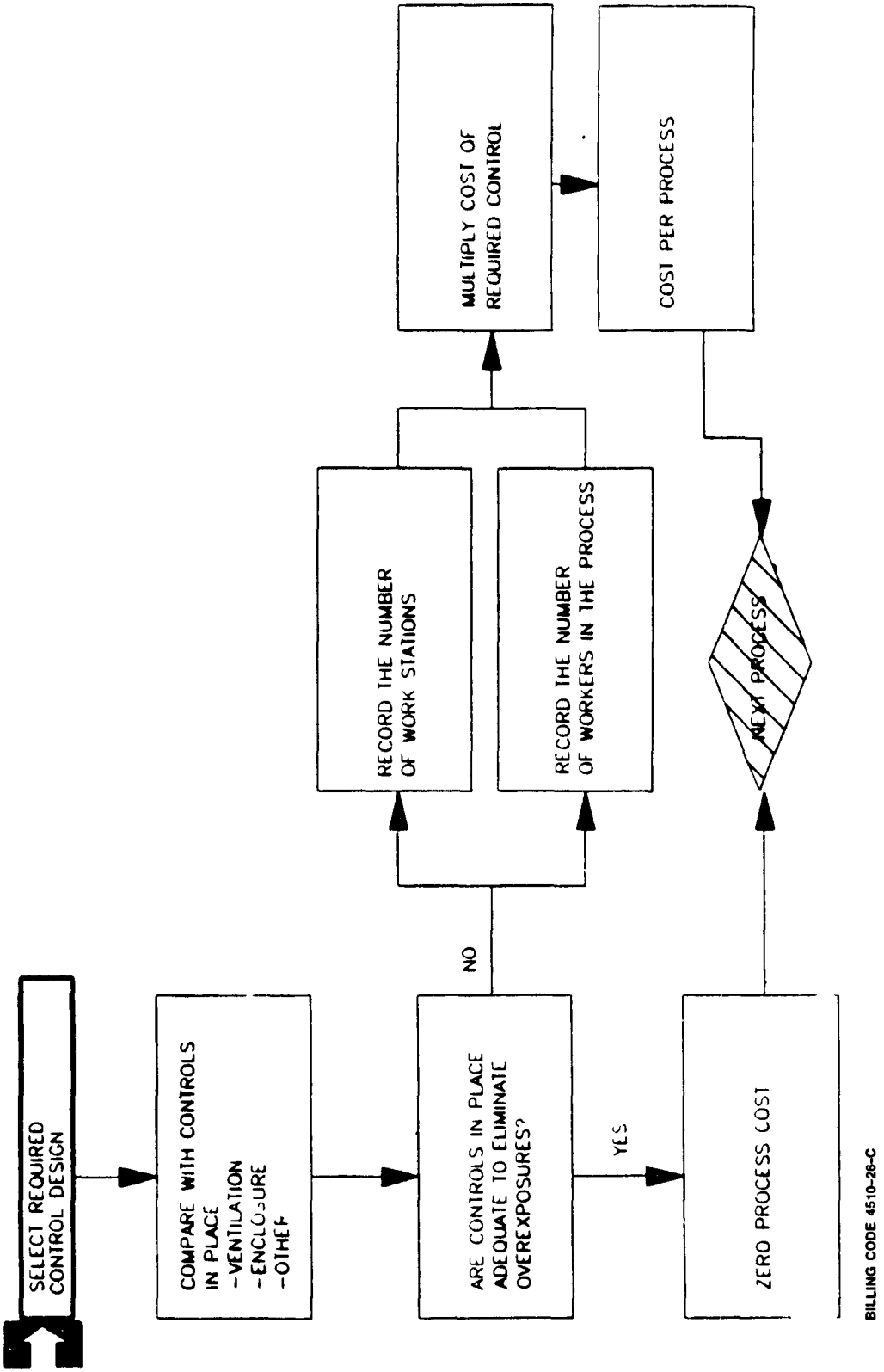

COMPUTER LOGIC FOR DERIVING INDUSTRY COST OF COMPLIANCE
ARE THERE ANY PROCESSES AT THE PLANT?
NO
ZERO PLANT COST
YES
FOR EACH PROCESS
ARE THERE ANY CHEMICALS IN THE PROCESS?
NO
YES
ARE THERE ANY RECORDED OVEREXPOSURES?
YES
NO
HAS MONITORING BEEN DONE?
YES
NO
ARE THE ACGIH OR NIOSH STANDARDS BEING USED?
YES
NO
DO ANY OF THE CHEMICALS HAVE A MAJOR STD CHANGE?*
YES
NO
IS MORE THAN 100 LBS OR 100 GALS OF ANY CHEMICAL PURCHASED, MANUFACTURED, OR GENERATED PER MONTH?
YES
NO
IS THE PROCESS LOCATED OUTDOORS
YES
NO
IS THE PROCESS LOADING/OFFLOADING?
YES
NO
DO ANY OF THE CHEMICALS HAVE A MAJOR STD CHANGE?*
YES
NO
IS THE PROCESS COMPLETELY ENCLOSED
YES
NO
SELECT REQUIRED CONTROL DESIGN
ZERO PROCESS COST
*The major/minor designation was based on the change in the PEL (over or under 50 percent decrease) as well as chemical factors such as form, particle size, and vapor pressure

FIGURE G-2
(continued)

COMPUTER LOGIC FOR DERIVING INDUSTRY COST OF COMPLIANCE



BILLING CODE 4510-26-C

When a respondent provided actual monitoring data for a process that indicated chemical exposures above the proposed standard, compliance costs were assigned to that process on the basis of prescribed controls for the given process. Where no monitoring data or reports of overexposure were available, the computer algorithm logic examined process and chemical characteristics to determine if workers at the process were potentially exposed to chemicals at levels over the proposed standard. The logic assessed the controls reported to be in place at the process and compared them with a list of controls thought necessary to control exposures in that process within the industry. When the required controls were reported to be in place, no compliance cost was assigned. When the required controls were not reported to be in place, a compliance cost per work station was assigned.

The computer algorithm determined that some processes within plants had no overexposures and consequently no compliance costs. Zero compliance costs resulted where no processes and/or chemicals were reported to occur at the establishment. Zero compliance costs also resulted when the respondent had monitored a process using ACGIH or NIOSH standards and found no overexposures. When only very small quantities of chemicals were present in a process, none of which had a "major" proposed exposure limit changes, no overexposure was determined and zero compliance costs were assigned. The major/minor designation was based on the proposed change in the PEL (over or under a 50 percent decrease) as well as chemical characteristics such as form, particle size, and vapor pressure.

Process configurations and location also were indications of compliance. Processes which were reported as completely enclosed with no worker entry were assumed to be in compliance with the proposed standard (have no compliance cost). Outdoor loading/offloading processes or other outdoor/processes with no chemicals with "major" proposed exposure limit changes were assumed not to require control equipment and costs. Zero compliance costs were also assessed where processes which required control equipment reported that the prescribed equipment was currently in place.

An example of a process which was assigned a cost of compliance to install engineering controls is a coating and spraying process in SIC 2511, Wood Household Furniture. The survey respondent reported that toluene, n-butyl alcohol and xylene were used in this operation. The proposed standard for toluene reduces the existing PEL by 50 percent. This reduction is considered to require concerted exposure control and is considered a "major" proposed exposure limit change. Because workers were involved in the process and the process was reported to be neither located outdoors nor fully enclosed, controls were assumed to be necessary to insure compliance with the proposed standard. The control required for controlling exposures at this process was determined to be local ventilation. The type of local ventilation prescribed in this case is a spray booth at an estimated cost of $3,070 annually per work station. Because the respondent reported no local ventilation, the cost was assigned for the eight work stations reported, resulting in a total estimated annual cost of $24,560 for this process at this site.

Expert engineering and industrial hygiene judgment was used to determine which of the various controls would be necessary to control for exposures by process in the affected industries. Engineering controls identified included exhaust ventilation (local and general), process enclosure, and process change. Some of all of these will be required by affected plants for compliance with the proposed exposure levels. In addition, personal protective equipment such as respirators will be needed for intermittent maintenance activities where engineering controls are not feasible.

The engineers and industrial hygienists classified the approximately 180 specific processes identified in the survey into about 30 process groups for the purpose of assessing required controls and estimating costs. These process groupings were based on similarities in the processes and levels and types of exposures resulting from the processes. Factors used to group processes include the chemicals generally involved in the process, type and usual configuration of the equipment, usual workstation design, level and route of exposure, industry group where the process exists and worker tasks in relation to the equipment and exposure route. The process similarities translated into likenesses in required controls such as type of ventilation hood, booth or enclosure, air flow rates, duct configuration and type and size of filters or scrubbers. Organizations presenting process data to the docket that varied from that derived by OSHA are referenced in the specific industry descriptions in this chapter. The compliance cost framework is presented in Table G–1. This table presents the process groups, the industries where the processes were identified, the general classification of controls specified and work station unit costs for the required controls assigned.

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TABLE G-1.
COMPLIANCE COST FRAMEWORK AND WORK STATION UNIT COSTS

PROCESS GROUP (1)	INDUSTRY GROUP (SICs)	REQUIRED CONTROL CONFIGURATION (2)	ANNUAL COST PER WORK STATION
Leather Processing, major	31	Local Ventilation	$ 2,510
Leather Processing, minor	31	General Ventilation	$ 720
Electrical & Electronics Manufacture	36	Local Ventilation	$ 2,520
Printing Processes, minor	27, 38, 73, 80	Local Ventilation	$ 1,240
Printing Processes, major	27, 39	Local Ventilation	$ 1,380
Glass Processing, major	32	Local Ventilation	$ 3,890
Glass Processing, minor	32, 36	Enclosure	$ 90
Resource Recovery & Water Treatment, major	28, 29, 33, 49	Enclosure & Local Ventilation	$ 21,900
Resource Recovery & Water Treatment, major	26	Enclosure & Local Ventilation	$ 14,000
Resource Recovery & Water Treatment, minor	26, 29, 49, 50	Enclosure & Local Ventilation	$ 14,000
Foundry Operations, major	33	Local Ventilation	$ 2,520
Foundry operations, minor	33, 39	Local Ventilation	$ 1,820
Grinding, Blasting, & Metalworking, major	25, 33, 36, 39	Local Ventilation	$ 7,200
Metalworking & Welding	All SICs	Local Ventilation	$ 1,140
Coke Ovens	29 (3)	Enclosures, Local Ventilation & Air Purifiers	$150,000

1 The "major" and "minor" designation of process groups refers to the level of the exposure change and consequently the extent of required control configuration costs within a given control and process configuration. For example, leather processing is the general process group and processes within that group are classified based on whether the employee exposure control requires major or minor control costs.

2 The specific required control configuration cost was estimated including all necessary components, such as ductwork, fans, hoods, baghouses, etc.

3 Coke ovens in SIC 33 are not included as they are covered by OSHA's Coke Oven Standard.

TABLE G-1 (Cont.)
COMPLIANCE COST FRAMEWORK AND WORK STATION UNIT COSTS

PROCESS GROUP	INDUSTRY GROUP (SICs)	REQUIRED CONTROL CONFIGURATION	ANNUAL COST PER WORK STATION
Paper Manufacturing, major	26, 30, 39	Ventilation & Air Purification in Control Rooms	$ 2,900
Paper Manufacturing, minor	All SICs	Local Ventilation	$ 180
High Temperature Drying	All SICs	Local Ventilation	$ 4,740
Layup	3632,3715,3732 3792,3995	Local Ventilation	$ 16,550
Coating, Spraying, & Adhesive Application	All SICs	Local Ventilation	$ 3,070
Chemical Handling & Formulation	All SICs	Local Ventilation	$ 1,760
Material Handling & Inspection, major	All SICs	Local Ventilation & Partial Enclosure	$ 1,120
Material Handling & Inspection, minor	All SICs	General Ventilation	$ 560
Cleaning & General Solvent Use, major	All SICs	Local Ventilation	$ 2,410
Cleaning & General Solvent Use, minor	All SICs	Local Ventilation	$ 710
Waste Collection & Transport	4953, 5093	Respirators (4)	$520 per worker
Painting, Maintenance	All SICs	Respirators (4)	$520 per worker
Welding, Maintenance	All SICs	Respirators (4)	$520 per worker

1. Use of respirators is considered the only feasible control for these processes due to their intermittent performance and because they are generally not performed at a fixed site.

TABLE G-1. (Cont.)
COMPLIANCE COST FRAMEWORK AND WORK STATION UNIT COSTS

PROCESS GROUP	INDUSTRY GROUP (SICs)	REQUIRED CONTROL CONFIGURATION	ANNUAL COST PER WORK STATION
Sanding & Drilling/Boring	24, 25	Local Ventilation	$ 2,200
Cutting, Sawing & Planing	24, 25	Local Ventilation	$ 1,900
Zero Cost Processes:			
Laundering	72		
Embalming	72		
Permanents	72		
Anesthesia	80		

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The development of unit costs for each control configuration required the development of "model" control designs. Model control configurations were selected to provide exposure control at "typical" process/work stations within the specified process group. This costing approach (model" configurations for "typical" work stations) required the differentiation of some process groupings as major or minor. The major/minor differentiation addresses the expected level of control required.

The control designs were developed by engineers based on their experience in industry and extensive secondary research on operations and exposure situations in each industry sector. This research included an examination of industry and industrial hygiene journals, engineering process reports, and texts. Included in the detailed cost calculations for the control configurations were costs for enclosure construction, baffles, fans, ductwork, filters, scrubbers, baghouses, and all other equipment required for exposure control. All of the costs were developed on a per work station basis so that an average size did not need to be estimated for the process. Investment costs were assigned to each control design on the basis of engineering handbooks and supplier catalogs. Investment costs were annualized over the projected life of equipment (10 years) using a 10 percent cost of capital and adding annual operating and maintenance costs estimated at 10 percent of the capital cost. Respirator costs for use by maintenance workers for intermittent activities were considered annual costs and include the respirator purchase as well as an estimated year's worth of cartridges and canisters.

Process control costs were summed per establishment and any maintenance worker respirator costs were included. A total annualized capital cost and annual operating cost was developed for each establishment. Costs for the survey establishment were then weighted (by SIC and size) to represent compliance costs for the universe of affected plants.

The United Auto Workers (UAW) International Union contended that the OSHA analysis "establishes a far outer bound" for the costs of compliance for several reasons [Ex. 197]. Two reasons claimed by the UAW are that the survey failed to account for the current state of control of process units and that not all process units would require the full application of the control schemes specified in the OSHA analysis. These potential problems were explicitly considered in developing the estimation method and the method was designed to minimize the effects of these factors. Questions in the survey *did* ask about the controls in place for every process. But the mere presence of controls does not assure the ability to achieve proposed levels. OSHA believes that the assignment of full control costs to uncontrolled processes, although not always necessary, is approximately offset by not costing, in all situations, the upgrading of insufficient control systems already in place.

The UAW also contended that "OSHA has refused to collect readily available exposure data which would have supported the feasibility of much lower PELs." On the contrary, OSHA solicited exposure data in two ways, as well as searching for data from public agencies. In addition to asking for data in the public hearings and for submission to the docket, every survey respondent was asked to provide exposure data. In addition, data in OSHA's IMIS database, in NIOSH reports and in journal articles were used.

The fourth point made by the UAW to support its position that costs were overestimated is that "The ongoing replacement of plant and equipment has not been accounted for." OSHA did consider this factor where information was available which allowed a quantitative assessment of the effect it would have on compliance, such as in dry cleaning, although it could not be considered in all areas of industry.

Finally, the UAW disapproves of the method by which unit costs were estimated, claiming that OSHA's approach "degrades the value of the analysis by obstructing generalization. In addition, past cost estimates have been sensitive to a per cfm cost of ventilation. The present evaluation fails to present such a cost." Estimation of costs on a per cfm basis was avoided because OSHA felt that better estimates could be made by estimating more detailed unit costs. Rather than having only one per cfm cost, the OSHA analysis uses 30 control scheme costs which are able to take into account different characteristics of both the process equipment and the chemicals being controlled. OSHA believes that this method creates the ability to estimate costs much more accurately than a per cfm estimate would allow across the broad spectrum of industries and processes which this rule affects. The per cfm basis of cost estimation was not used because it would require much *broader* assumptions about average characteristics of control systems such as ductwork, baghouses, etc. OSHA views its method as an improvement on the previous methods because this method requires fewer generalizations and assumptions and allows the inclusion of more information in estimating costs.

Compliance Costs by Industry Sector

Following the methodology described in the preceding section of this chapter, annual compliance costs were estimated by industry sector. The costs presented for the surveyed industries are based on the data collected from the about 5,700 respondents. (For industries not included in the survey, expert judgment and secondary sources were used for estimating costs.) Table F-4 (shown at the end of the chapter) presents the detailed breakdown of compliance costs for each industry sector included in the survey. The table illustrates the processes reported in the survey, the number of work stations by process, and the number of work stations determined to require the addition of compliance controls. The process and work station frequencies are weighted to reflect the total universe of affected plants.

A small percentage of respondents (less than 5 percent) actually provided monitoring data during the survey. However, based on information from the survey, it was determined that about 86 percent of all establishments in the surveyed industries (74 percent of those with hazardous substances) have no exposures in excess of the final standard and will not incur any costs to comply with the standard. This conclusion was derived by comparing controls in place with controls deemed necessary to reduce exposures to the regulated limits. Thus a cost was assigned if the existing ventilation system was estimated to be insufficient to control these chemicals at the new levels. About 22 percent of establishments with hazardous substances will incur costs to provide engineering controls for processes within the plant. About 4 percent of the establishments with hazardous substances will be required to provide personal protective equipment only for maintenance workers whose intermittent operations cannot be controlled with engineering controls.

Table G-2 presents the total annualized capital and annual operating cost for compliance with the standard by industry. As shown, annual compliance costs are estimated to total $788 million. Upon review and incorporation of all docket materials, OSHA believes that these costs are fully representative of costs of compliance with the standard. These costs represent

an estimate of compliance costs for large and small plants affected by the exposure limit changes. Industries with some anticipated cost impact are identified below. Included in the industry description are data provided to OSHA during testimony at the public hearing and in the docket submissions.

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TABLE G-2

ANNUAL OPERATING AND ANNUALIZED CAPITAL COST OF COMPLIANCE BY INDUSTRIAL SECTOR (a)

SIC (b)	SIC DESCRIPTION	LARGE PLANTS	SMALL PLANTS	ANNUAL COST
20	FOOD PROD. (c)	$21,704,100	$11,789,000	$33,493,100
21	TOBACCO (c)	$19,700	$0	$19,700
22	TEXT. MILL (c)	$23,308,400	$6,170,000	$29,478,400
23	APPAREL PROD. (c)	$23,604,300	$8,139,900	$31,744,200
24	LUMBER & WOOD	$18,112,100	$38,608,700	$56,720,800
25	FURNITURE	$18,440,900	$2,634,900	$21,075,800
26	PAPER PROD.	$29,807,900	$1,190,800	$30,998,700
27	PRINTING & PUB.	$5,186,400	$28,568,100	$33,754,500
28	CHEMICAL PROD.	$28,793,500	$6,661,200	$35,454,700
29	PETRO. REFINING	$23,635,000	$51,000	$23,686,000
30	RUBBER & PLASTICS	$46,605,200	$64,488,200	$111,093,400
31	LEATHER PROD.	$1,272,700	$1,142,000	$2,414,700
32	STONE & CLAY	$15,704,300	$6,753,500	$22,457,800
33	PRIM. METAL	$65,691,400	$5,266,200	$70,957,600
34	FAB. METALS	$28,964,500	$10,455,200	$39,419,700
35	MACHINERY	$30,994,600	$14,212,000	$45,206,600
36	ELEC. MACH.	$17,060,100	$3,607,400	$20,667,500
37	TRANS. EQUIP.	$23,577,900	$26,214,500 (c)	$49,792,400
38	INSTRUMENTS	$7,227,700	$2,405,800	$9,633,500
39	MISC. MANUF.	$9,829,300	$6,013,300	$15,842,600
40	R.R. TRANS.	$1,083,400	$0	$1,083,400
45	AIR TRANS.	$3,740,500	$0	$3,740,500
47	TRANS. SERV.	$3,789,400	$0	$3,789,400
49	ELEC. GAS. SAN.	$32,355,100	$5,654,200	$38,009,300
50	WHOLESALE TRADE	$1,306,700	$1,688,600	$2,995,300
51	WHOLESALE, NON-DUR	$2,854,900	$11,360,900	$14,215,800
55	AUTO DEALERS	$7,382,200	$6,168,300	$13,550,500
72	PERSONAL SRV.	$3,487,100	$7,385,000	$10,872,100
73	BUSINESS SRV.	$1,596,100	$826,000	$2,422,100
75	AUTO REPAIR	$4,280,100	$1,863,400	$6,143,500
76	MISC. REPAIR SRV.	$469,700	$2,340,200	$2,809,900
80	HEALTH SERV. (c)	$2,413,000	$2,026,400	$4,439,400
TOTAL		$504,298,200	$283,684,700	$787,982,900

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

(a) Costs were calculated by annualizing the capital cost over the projected life of the equipment (10 years) using a 10 percent cost of capital and adding an annual operating and maintenance cost estimated at 10 percent of the capital cost.

(b) Industry sectors not identified in this table include industries with no major cost impact expected, the construction industry, which will be the subject of a separate regulatory analysis, and industries such as mining, over which OSHA has no jurisdiction.

(c) Costs in these sectors were based on expert judgement and secondary data collection.

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In addition to review of submitted materials, OSHA also undertook a site visit survey of about 90 plants to examine and compare data collected by telephone and in the field. Various statistical tests were performed on the telephone survey and site visit data to detect biases in the cost algorithm [Supplement 1]. These analyses tested the hypothesis that the telephone survey did not systematically differ from the site visits in the number of estimated firms out of compliance and the actual cost assigned to those firms. Using a 95 percent confidence interval, these tests revealed no aggregate bias in the assignment of costs by the telephone survey as compared to the site visits.

Food and Kindred Products (SIC 20). Costs are projected for a large number of establishments in this sector. The prepared feeds and feed ingredients, not elsewhere classified (SIC 2048), are estimated to account for a large percentage of the $33.5 million annual costs in SIC 20. Controls may be necessary for dust exposures and chemical fumigants.

Two commenters provided detailed cost estimates for firms to achieve the Agency's proposed grain dust limit of 4 mg/m^3: the National Grain and Feed Association (NGFA) [Ex. 3–752] and the National Feed Industry Association (NFIA) [Tr. 8/10/88, pp. 10–61—10–69].

The National Grain and Feed Association provided alternative cost estimates for feed mills and for flour mills. These estimates were based on the following assumptions:

1. All affected facilities will need pneumatic dust control systems and do not now have them;

2. Only 13 percent of food mills handle wheat, oats, or barley, and thus only 13 percent will be affected by the grain dust limits; and

3. The costs of pneumatic dust control systems are the same as those estimated by Booz Allen in a 1984 study conducted for OSHA in connection with the Agency's grain handling standard (these costs were inflated by 15 percent to convert them from 1984 to 1988 dollars).

Using these assumptions, the NGFA concluded that the capital costs of compliance for all feed mills would be $213 million and for all flour mills would be $81 million [Ex. 3–752]. If these costs are annualized using OSHA's interest-rate and life-of-equipment assumptions, annualized costs for feed mills (using a 10-percent operating cost figure) would be $56 million per year, and annualized costs for flour mills would be $21 million per year. Average annualized costs per affected feed mill would be $44,000 per year and per affected flour mill, $225,000 per year.

The NFIA also provided estimates of the total costs of compliance for feed mills. The NFIA's costs were based on an evaluation of 20 existing feed mills; the NFIA, therefore, only attributed costs for pneumatic dust control systems to facilities that do not now have them. Thus, the NFIA study attempts to take into account baseline controls; it reported that 5 of these 20 mills had some level of dust control in place. The NFIA estimated that the capital costs of compliance for all feed mills would be $664.5 million [Tr. 8/10/88, pp. 10–61—10–69]. If OSHA's interest rate and life-of-equipment assumptions are used and operating costs are assumed to be 10 percent of capital costs, annualized costs for all feed mills would be $175 million per year. Average annualized costs per affected feed mill would be $22,000 per year.

OSHA's Preliminary Regulatory Impact Analysis (PRIA), published with the proposed rule, estimated costs for all facilities in SIC 20 to comply with the proposed PELs; the grain dust portion of this overall cost was based on the assumption that affected facilities would have to meet a 4-mg/m^3 PEL for grain dust. Based on health effects and economic feasibility considerations, the Agency has changed the grain dust levels to 10 mg/m^3 TWA. OSHA's analysis shows that the great majority of employee exposures are at or below 10 mg/m^3, and thus, few additional controls will be needed to achieve the final rule's limit of 10 mg/m^3. OSHA believes that the cost estimates for this sector are conservative and probably overstate the costs that affected employers will be required to expend to achieve the 10 mg/m^3 limit.

Several comments were received from employers in SIC 201, Meat Products, who were concerned that the proposed limit of 1 ppm for carbon disulfide would force the manufacturers of the cellulosic casings that are made in facilities classified in SIC 3089, Miscellaneous Plastics, to go out of business [Exs. 3–421, 3–659, 3–897; Tr. 8/2/88, pp. 4–209, 4–261]. In the opinion of these concerned meat packers and processors, the impact of the carbon disulfide limit on firms in SIC 3089 would be so great that all domestic supplies of the cellulosic casings needed by the meat packers and processors would disappear. These commenters were particularly concerned because there are no substitutes for cellulosic food casings except natural casings, which can only be used for cooked sausage and cannot be used with automatic machinery [Ex. 3–897].

The costs anticipated by the meat packers and processors were presented in a study conducted by Wharton Economic Forecasting Associates (WEFA) [Ex. 3–659]. WEFA forecast a loss of 12,000 to 20,000 jobs in meat processing and 12,000 to 16,000 jobs in meat packing, and also projected an 8 and 16 percent reduction in the price paid to farmers for cattle and hogs, respectively [Ex. 3–659].

WEFA based its forecast on the following assumption: that processed meats dependent on cellulosic casings would disappear from the marketplace altogether [Ex. 3–659]. OSHA finds this scenario unlikely, since the cost impacts in SIC 3089 are likely to be greatly reduced because the Agency has established a 4 ppm limit, rather than the proposed 1 ppm limit, for carbon disulfide in the final rule. Domestic production of casings should not cease or be disrupted in a major way.

OSHA concludes that the concerns of the meat packers and processors in SIC 201 have been addressed and their supplies of cellulosic food casings should not be disrupted.

The National Cotton Council of America [Ex. 3–1080] expressed the concerns of its members over the difficulty small, rural cottonseed mills would have in sampling their employees' exposures to hexane and grain dust. As discussed above in the section on Technological Feasibility for SIC 20, OSHA determined that sampling and analytical methods are available for these contaminants and that consultant industrial hygienists can be employed by mill owners on an as-needed basis. OSHA is aware that the services of competent and experienced industrial hygienists can be obtained for fees beginning at $300 per day and that laboratory fees for analysis range from $20 to $40 per sample, depending on the substance being analyzed. OSHA does not believe that costs of this magnitude will have a significant impact on cottonseed mills.

Although carbon dioxide exposures in the beer industry were described as "unique" [Tr. 8/9/88], the principal sources of exposure are blow-outs of safety valves, opening of tank doors, and entry into tanks for cleaning. For both blow-outs (an upset condition) and tank entry (a maintenance operation), OSHA permits the use of respiratory protection to meet the PEL. Exposures resulting from opening tank doors can be reduced by implementing the work practice of cracking the door and remaining out of the area for a few minutes to allow the CO_2 to dissipate. Since the final rule establishes an 8-hour TWA of 10,000 ppm, rather than the 5,000 ppm proposed, OSHA concludes

that the cost estimates presented in the PRIA for SIC 20 do not need to be revised and include all potential costs of compliance for breweries.

The Corn Refiners Association (CRA) estimates that the wet corn milling industry would incur $24,097,000 in capital costs, with annual operating costs of $6,244,000 million, to meet the proposed 2- and 5-ppm SO_2 standard. Of this, CRA estimates that $12,809,000 in capital costs and $3,266,000 in operating costs would be incurred to meet the 5-ppm STEL, and $11,288,000 in capital costs and $2,878,000 in operating costs would be incurred to meet the 2-ppm PEL [Ex. 65, Tab 13, pp. 7–8].

OSHA notes that 47 percent of CRA's estimated costs are attributed to meeting a STEL. However, the record indicates that short-term excursions do not typically occur during normal operations; instead, they occur during maintenance activities and in emergency conditions. In these situations, the standard practice in the industry is to use respiratory protection [Tr. 8/8/88, p. 8–90], as would be permitted by OSHA.

Furthermore, OSHA's technological feasibility assessment shows that the 2-ppm TWA and 5-ppm STEL can be achieved in all routine operations in this sector with the addition of a small amount of make-up air (or by opening the windows in warmer months). Employers in this sector also need to reduce the number of process upsets and maintenance problems in their plants by instituting manual leak detection programs, improving maintenance, replacing pump seals before they leak, and phasing out outdated process equipment. Evidence in the record reports that the volume of production and sales has risen so quickly that control equipment has been unable to keep pace [Ex. 65, Tab 13, p. 7]; this sector should therefore not have difficulty absorbing the negligible costs associated with the minimal control procedures needed for this sector to achieve compliance with the final rule's limits for SO_2.

CRA notes that its estimates of costs constitutes 18.3 percent of OSHA's total cost estimate for all chemicals in all parts of SIC 20. OSHA notes that, generally, within a 2-digit SIC industry group, most industry sectors are estimated to incur minimal costs to comply with the final rule, and a few industry sectors will incur higher costs. Thus, even assuming that CRA's estimated costs are accurate, OSHA's aggregate estimate for SIC 20 are not necessarily substantially understated. Thus, OSHA concludes that the cost estimates presented in the PRIA for SIC 20 do not need to be revised based on the record evidence pertaining to the potential costs of compliance for wet corn milling.

Tobacco Manufactures (SIC 21). The lowest cost of compliance in the manufacturing sector is expected to occur in SIC 21, Tobacco Manufacturers, ($20,000). It is estimated that very few plants will incur costs in the tobacco manufacturing industry.

Textile Mill Products (SIC 22) and Apparel and Other Finished Products (SIC 23). These sectors have a large number of establishments which may incur compliance costs. The apparel industry is estimated to incur about $31.7 million in annual compliance costs. Many of the affected establishments in SIC 23 may require controls for cleaning solvents such as perchloroethylene. The $29.5 million annual costs in the textile industry are estimated to result from control of exposures to solvents, dyes and other substances. No differing cost estimates in opposition to OSHA's cost calculations were presented in the docket or testimony.

Lumber and Wood Products (SIC 24). The annual costs of compliance in the lumber and wood products industry are estimated to total $56.7 million. The compliance costs for this sector primarily reflect the cost of controls required to lower exposures of wood dust to 5 mg/m^3 (2.5 mg/m^3 for Western red cedar wood). The survey indicated that sanding and other "dusty" processes would require controls to lower wood dust exposure. The large number of establishments that must engineer ventilation systems for wood dust control account for the substantial proportion of compliance costs to be incurred by small establishments in this sector.

OSHA's estimates in the preliminary analysis were based on a standard of 5 mg/m^3 for softwood and 1 mg/m^3 for hardwood, using survey responses for particulates not otherwise regulated as a surrogate for wood dust.

In determining the total cost of compliance for wood dust at the final PEL of 5 mg/m^3 (2.5 mg/m^3 for Western red cedar), OSHA carefully considered data presented in to the record by National Economic Research Associates (NERA), Clayton Environmental Consultants, the Workers' Institute for Safety and Health (WISH), the Holliday report, and numerous other government, union, and industry respondents [Exs. 3–748, 8–127, 8–196, Tr. 8/18/88, p. 13–5, etc.]. Researchers from Clayton Environmental Consultants and NERA, on behalf of the Inter-Industry Wood Dust Coordinating Committee, performed a study on the impacts of the proposed air contaminants rule on SICs 24 and 25 [Exs. 3–748, 8–127]. NERA concluded that it would cost firms in SICs 24 and 25 $266 million annually for a 5 mg/m^3 standard for all woods. Under a 1 mg/m^3 standard for all wood dust, NERA estimated that costs would exceed $1.9 billion annually and under the proposed standard of 5 mg/m^3 for softwood and 1 mg/m^3 for hardwood, annual compliance costs would be approximately $1.5 billion. NERA's estimate of $1.5 billion was more than four times higher than OSHA's August 1st estimate of $341 million annually for a 5 mg/m^3 softwood standard, 1 mg/m^3 hardwood standard [Ex. 38a]. Mark Berkman, repsesenting NERA, testified that the cost discrepancies between the OSHA study and their estimates were due to the differences in unit costs and in the number of work stations out of compliance in SICs 24 and 25 [Tr. 8–12–88, p. 107, 111].

OSHA determined that annual unit costs of compliance per work station of $1,900 for cutting/sawing/planing, $2,200 for sanding/polishing and grinding, and $2,200 for drilling/boring are the best estimates currently available to comply with the final standard. These unit costs are not significantly different from the unit costs presented in the Clayton study. (The unit cost presented by NERA in one case does not accurately reflect the findings of Clayton Environmental Consultants. Apparently the cost applied to the "belt sander" in the NERA study was derived for "sander, belt (widebelt)" in the Clayton study. However, the cost developed for "sander, edge" would have been more appropriate. "Edge sander" was never identified in the NERA survey. The cost for the widebelt sander is $50,800, while that for the edge sander is $12,900. The capital cost for control on a belt sander developed for OSHA was $8,000.)

In analyzing NERA's methodology, there were significant differences between OSHA's estimates of costs and work stations when compared to NERA's. NERA's methodology begins by surveying "industry experts" (via the Inter-Industry Wood Dust Coordinating Committee) to derive the number of machines in typical small and large establishments. In a number of industries, these experts estimated that there would be many more machines than total employees. For example in SIC 2426, NERA's survey respondents estimated that there would be 32 machines in a "typical" small plant (fewer than 20 total employees). NERA's next step was to multiply the number of machines in a typical plant by the percentage of machines out of

compliance from the Clayton study. This revealed an estimated number of machines out of compliance in a typical plant at the four-digit level for small and large firms. This estimate of machines out of compliance was then multiplied by the per machine unit cost to arrive at an average cost per typical plant. Finally, this number was multiplied by the number of plants reported in the 1982 Census of Manufactures for each four-digit SIC in order to arrive at an aggregate cost.

OSHA believes that its methodology for deriving total work stations is more accurate. OSHA used a telephone survey which requested information about work stations specifically at the plants being interviewed. However, NERA sent surveys to "industry experts" who were asked to describe a "typical plant". NERA never explains the number or identity of respondents in its survey. However, it is important to note that NERA received no responses in numerous four-digit SICs (large plants in SICs 2429, 2491, 2515, 2517, 2519, 2531, and 2541 and small plants in SICs 2436, 2451, 2452, 2491, 2512, 2515, 2517, 2531, and 2541). For industries with no response rate, a weighted average cost was used as a surrogate. In the case of SIC 25, 10 of the 16 size and industry categories were derived by using surrogates. Thus non-surveyed industries such as mattresses and bedsprings were estimated to have the same costs and work stations out of compliance as surveyed industries with high wood-dust-generating processes such as wood and upholstered furniture. The cost surrogate used for these 10 categories in SIC 25 is the third highest per plant cost, despite the fact that it was derived without specific exposure data by Clayton or estimates of machines used in a typical plant for these specific four-digit SICs. OSHA conculdes that such extrapolation is based on less comprehensive data than the 1988 telephone survey. This widespread use of surrogates partly explains why total work stations has been overestimated by NERA.

Additionally, OSHA concludes that NERA's survey estimates of total machines is high, and therefore the number of machines out of compliance is overestimated. Including those four-digit SICs where surrogates are used (and therefore total estimated number of machines is implied), NERA assumed a total of approximately 800,000 machines in SICs 24 and 25. This estimate is roughly equivalent to the number of employees in these two SICs. The statement by NERA that ". . . workers (or work stations, assuming one worker per work station)" [Ex. 3-748, p. 13] implies that NERA does not find it unreasonable that 800,000 wood-dust-generating machines are used continually by every worker in SICs 24 and 25. OSHA concludes that its estimate of 300,000 total work stations (200,000 wood-dust-generating work stations) as derived from the 1988 telephone survey, is a more accurate estimate. OSHA's site visits and survey indicate that there are far fewer work stations than workers in SICs 24 and 25. One cause for this difference is the amount of shift work performed, thus allowing one work station to be used by two or three workers in a single day. Another cause for this difference is the number of technical, clerical, managerial, and maintenance staff, many of whom do not work consistently around machines which generate substances regulated under this rulemaking. Thus OSHA has determined that its estimate of total work stations is an accurate assessment for firms in SICs 24 and 25.

Next, it was necessary for OSHA to derive the percentage of wood-dust-generating processes (sanding/polishing/grinding, cutting/sawing planing, and drilling/boring) out of compliance with the final standard in SICs 24, 25, and 26. OSHA combined its monitoring data from site visits with Clayton's samples to estimate that 16 percent of the wood-dust-generating work stations (including those involving Western red cedar) would be out of compliance with the final standard. This percentage seems to be reasonably close to the 13.5 percent figure for 5 mg/m^3 from the OSHA Health Response Team Survey referenced by Scott Schneider of the Workers' Institute for Safety Health (WISH) [Tr. 8/15/88, p. 13-5].

Since a 2.5 mg/m^3 standard was established for Western red cedar, OSHA performed a separate analysis on compliance cost for this substance. OSHA believes that there are approximately 290 firms involved in the production of shakes and shingles with Western red cedar in SIC 2429 [U.S. Department of Commerce, Office of the Census]. Studies on Western Red Cedar asthma [Ex. 82D, Captain James J. Edwards, Jr.] indicate that approximately 90 percent of these firms operate in Washington State, where the permissible exposure limit is currently 2.5 mg/m^3. Data presented by Stephen Cant of the Washington Department of Labor & Industry indicated that "they can, in fact, in most cases, comply with those limits, and that there are studies that support, certainly I think, the 2.5 limit as regards allergenic wood dust with respect to Western red cedar." [Tr. 7/29/88, p. 2-103]. However, studies performed by the University of Washington in 1987 indicate that "Labor and industries inspectors found a large number of mills out of compliance with the new regulatory standards." [Ex. 127.H] OSHA assumed that compliance with the wood dust standard relative to Western red cedar in the shakes and shingles industry would not be significantly different from compliance with the overall wood dust standard. OSHA concluded that 16 percent of the work stations would be out of compliance with the final standard in the shakes and shingles industry.

To derive the cost for wood dust in SIC 24, OSHA estimated that 142,000 of the 215,000 total work stations are wood-dust-generating, and that 1,500 involve Western red cedar. Sixteen percent, or 23,000, of the wood-dust-generating work stations were determined to exceed the final standard (240 for Western red cedar). Wood dust thus accounted for $45 million of the $56 million in SIC 24. For reasons explained above concerning the total number of work stations and work stations affected, OSHA concludes that NERA's estimate of $137.1 million for a 5 mg/m^3 standard is an overestimate.

In addition to wood dust, controls for exposures to solvents, wood preservatives, and other chemicals in coating processes are estimated to result in compliance costs in SIC 24. Overall, about 68 percent of all establishments in SIC 24 are estimated to incur compliance costs. OSHA thus concluded that the annual operating and annualized capital cost to comply with all standards would be $56.6 million in SIC 24.

Furniture and Fixtures (SIC 25). Annual costs of compliance in the furniture and fixtures industry are estimated to total $21.1 million. Costs to control wood dust exposures at 5 mg/m^3 wood (2.5 mg/m^3 for Western red cedar) during sanding, cutting, drilling, and other dusty processes are the major components of compliance costs in this sector. Establishments would also incur costs for control of exposures to coatings and solvents. The survey indicated that the furniture sectors which include metal working (SICs 2514, 2515, 2522, 2542, 2591 and 2599) would also require controls for welding fumes and various metal particulates resulting from grinding and other processes. OSHA believes that local exhaust ventilation will reduce exposures to permissible levels during welding operations.

OSHA again believes that NERA overestimated the costs and the number of work stations used in its cost estimations for SIC 25. An explanation of cost differences is provided in SIC 24. OSHA calculated 89,000 total work stations (57,000 at wood-dust-generating work stations) in the furniture industry, based on responses provided by the telephone survey. NERA's estimate of work stations, which relied heavily on surrogates, resulted in a significant overestimate of the number of total work stations and work stations out of compliance with the OSHA standard. This overestimation of the total number of work stations distorted NERA's cost estimates for SIC 25 ($128.9 million for a 5mg/m^3 standard).

To derive the cost to control wood dust exposures in SIC 25, OSHA estimated from the 1988 telephone survey that 57,000 of the 89,000 total work stations would be wood-dust-generating. Sixteen percent, or 9,000, of the wood-dust-generating work stations were expected to exceed the final standard. Wood dust thus accounted for $19 million of the compliance cost in SIC 25. OSHA believes that its total cost estimate of $21.1 million is an accurate estimate of the actual cost of compliance for this sector.

Paper and Allied Products (SIC 26). Annual costs in the paper and allied products industry are estimated to be $31.0 million. Much of the estimated costs in SIC 26 will be associated with the cost of controls in large pulp mills and associated operations. Pulp mills are operated separately (those listed in SIC 2611) or as part of paper or paperboard mills (SIC 2621 and SIC 2631 respectively). Some of the cost of compliance in these operations would result from controlling the large quantities of chemicals used in breaking down the pulp to form cellulose and the reactions that occur in the digesting process. The digesting and bleaching operations require ventilation or enclosure.

A portion of the costs associated with SIC 26 relate to controlling exposures to wood dust levels at 5 mg/m^3 for wood dust (2.5 mg/m^3 for Western red cedar). Data presented on wood dust exposures by Clayton were derived from only 2 site visits in SIC 26. NERA presented no cost estimates for this industry. Thus OSHA retained its estimate that sixteen percent of all wood-dust-generating work stations would be out of compliance with the final standard in SIC 26. Data from the 1988 sample survey indicated that the total cost for this SIC would amount to an annual operating and annualized capital cost of compliance of approximately $31.0 million.

Printing and Allied Industries (SIC 27). Compliance costs in the printing industry sector (an estimated $33.8 billion) would result from ventilation requirements to control exposures to cleaning solvents and ink spray generated within the printing process. A very large number of small establishments are involved in printing and over 3,100 of them would be affected by the revised standards. The survey indicated that a large number of small establishments currently lack exposure controls and provision of these controls accounts for the high control costs in this sector. However, OSHA's field visits in this sector [Ex. 8–11] indicated that the unit costs initially estimated for printing processes were somewhat high. The final cost estimate was adjusted to reflect the information collected during the field site visits.

Chemicals and Allied Products (SIC 28). Annual compliance costs in SIC 28 are estimated to total $35.5 million. Over 35 percent of the costs in SIC 28 are estimated to occur in Paints and Allied Products Manufacturing (SIC 2851). The survey indicated that a large proportion of plants will require additional controls for a number of processes found in paint and paint products manufacturing. There are many chemicals in this industry segment which present exposure problems in a variety of both wet and dry processes, including reaction, separation, crushing, mixing, drying and bagging.

According to U.S. Borax, the average annual operating costs for environmental controls at Borax in SIC 2819, Industrial Inorganic Chemicals (NEC), is considerably higher than OSHA had predicted for a large plant. As an example, OSHA estimated operating costs of $18,000 per year for large plants in SIC 28. U.S. Borax estimated an average operating cost of $37,600 per year. [Tr. 8/9/88, 9–113.] It is not clear from the testimony or from submissions to the docket [Ex. 3–744] which of the costs listed by Borax are associated with the mining and initial processing of the ore. These processes fall under the jurisdiction of the Mine Safety and Health Administration. OSHA believes a significant portion of these costs estimated by Borax are associated with the mining operation rather than downstream activities. After reviewing the rulemaking record, OSHA increased the TWA for all borates to 10 mg/m^3 and adjusted plant costs downward to reflect the change.

Industry group SIC 282, Plastics Materials, Synthetic Resins and Synthetic Rubber accounts for about 22 percent of compliance costs in this sector. Compliance costs are related to ventilation and other requirements to control exposures to carbon disulfide, acetone and other emissions in the manufacture of rayon, cellulose acetate fibers and other plastics materials and synthetic rubber. The Vinyl Institute contended that a number of the processes found in member companies would have to be modified at an estimated capital expense of $10–25 million. The additional annual expense to maintain the required level of compliance was estimated by the Institute to be $4–5 million. The industry-wide estimated initial capital expense was estimated to be $160–400 million and annual expenses $60–80 million [Ex. 3–624]. The cost estimates submitted by the Vinyl Institute included tank farm vent controls which OSHA, as explained in Chapter F (Technological Feasibility), concludes would not be necessary. The remaining areas identified by the Institute are loading/unloading operations and process sewer systems. While the costs are not presented in a disaggregated form, OSHA believes that the costs to bring these two areas into compliance would be only a fraction of the Institute's total cost estimate and OSHA's estimated costs are more accurate. OSHA also notes that the final limits for several chemicals of interest to the Vinyl Institute (acetone, carbon disulfide) are less stringent than those proposed, which should mitigate cost problems for affected firms.

In SIC 2823, Cellulosic Manmade Fibers, the Inter-Industry Committee on Carbon Disulfide asserts that "the cost of making even small improvements below the 20 ppm limit is significant—$16.6 million." These costs would be for preventing the escape of carbon disulfide into the work area (process enclosures) and for increased ventilation [Ex. 3–747, p. 82]. As explained in the discussion of technological feasibility, OSHA believes that the evidence indicates the problem to be much less severe than is suggested above, and that most exposures are of short duration. The industry can comply with the final carbon disulfide standard of 4 ppm by using respirators in a limited number of designated processes (see Chapter F, Technological Feasibility) and adjusting work practices to control exposures. Costs for this subsector are reflected in the total estimate for SIC 28. Also in SIC 2823, the manufacturers of cellulose acetate claim that compliance with the acetone standard is not economically feasible at

the three existing facilities. Tennessee Eastman estimated that costs of compliance with the proposed standard of 250 ppm for acetone to be $11.2 million annually in its facility [Ex. 3–745]. Mr. Vernon G. Knight of Hoechst Celanese, estimated that the costs of compliance for its two facilities would total $40.2 million in capital costs [Ex. 3–745]. OSHA does not believe that costs of this magnitude will be incurred. OSHA has revised its original proposal of 250 ppm for acetone. OSHA believes that a 750 ppm TWA and 1000 ppm STEL is economically and technologically feasible, and the costs for this sector have been reduced to reflect this change.

For SIC 2892, Explosives, the Institute of Makers of Explosives mentions "a study conducted in one nitroglycerin/ethylene glycol dinitrate (NG/EGDN) manufacturing facility in which the concept of reducing workplace concentrations to a 0.01 ppm (0.1 mg/m^3) level was examined." This study indicated that the costs of engineering controls at this facility would exceed $4 million (1979 dollars) in capital costs to achieve the proposed standard for NG/EGDN [Ex. 3–749, 190]. OSHA believes that the principal cost for SIC 2892 would be for air line respirators and this cost is included in the total cost estimated for SIC 28.

Petroleum Refining and Related Products (SIC 29). Although only 13 percent of all facilities in SIC 29 are expected to be affected, nearly 25 percent of the large refineries will incur costs. Of those firms with more than 100 employees, almost 59 percent incurred some cost. Approximately 90 percent of the $23.7 million annual costs in SIC 29 are expected to be incurred by facilities in SIC 2911, Petroleum Refining. Most of these costs will be related to water treatment processes and sampling/quality control tasks because of a lack of controls in place in these two areas. In general, however, this industry has extensive control technology in place for the primary processing equipment. Closed processes with few exposed workers are predominant due to the requirements of process operation at elevated temperatures and pressures.

Costs in SIC 2951, Paving and Roofing Materials, arise mainly from smaller blending and formulating operations which usually involve few employees. Packaging and loading/offloading processes account for the majority of costs in SIC 299, Miscellaneous Products of Petroleum and Coal. The remainder of costs in SIC 299 are attributable to the blending and formulating of lubricating oils and greases.

Rubber and Miscellaneous Plastics Products (SIC 30). Annual costs of compliance in this industry sector are estimated to total about $111.1 million. Controls were required for processes such as molding and vulcanizing. Worker exposure to chemical vapors require the addition of local ventilation to many processes. The miscellaneous plastic products industry (SIC 3079) accounts for over 20 percent of the annual costs in this sector. The costs in SIC 3079 result from the high proportion of small plants in this sector which will incur costs of compliance. Controls are required in SIC 3079 for many crushing and grinding operations used to prepare plastic material for hot processes.

The Styrene Information and Research Council (SIRC) presented estimates of the costs SIRC believes will be required to control styrene exposures to a TWA of 50 ppm in selected segments of the miscellaneous plastics industry [Exs. 3–742, 34A, Tr. 8/3/88, pp. 117–130]. These costs estimates were developed for SIRC by Arthur D. Little, Inc. (ADL) and represent a partial update of a large study done by ADL in 1980 on the costs and technical feasibility of styrene control. This updated study estimated costs for the tub/shower, lavatory, hot tub/spa, and resin-applied-at-press segments. All of these segments are classified in SIC 308, Miscellaneous Plastic Manufacturing. ADL estimated that total capital costs for these segments to comply with an 8-hour TWA of 50 ppm for styrene would be $1.169 billion and that operating costs would be $204.6 million per year [Ex. 34A, Table 2]. Using OSHA's interest rate and life-of-equipment assumptions, the annualized costs for this sector, using these capital and operating costs, would be $395 million per year, a value in excess of the PRIA's total estimated costs of $75 million per year for all of SIC 30. ADL estimated that 19,230 employees in these segments are exposed to styrene at levels above 50 ppm; according to ADL, there are a total of 48,885 employees in these segments at 1550 plants [Ex. 34A, Table 1].

OSHA finds several difficulties with the ADL study. First, ADL used the exposure data from its 1980 study for SIRC as the basis for estimating what controls (and therefore costs) would be involved in achieving compliance; these exposure data showed considerably higher exposure levels (with one exception) than more recent data, e.g., the Cal/OSHA study of styrene exposures in this industry. ADL did use the Cal/OSHA data in one case (for the tub/shower segment), the only instance in which the Cal/OSHA exposure data were actually *higher* than the 1980 ADL data. Thus, ADL relied on the highest exposure data as a cost baseline, even when more recent data were available, and only used recent data when they were higher than the outdated data. This factor would contribute significantly to an overestimate of costs, especially since representatives of SIRC reported at the hearing that conditions in the industry have improved considerably in the last 10 years [Tr. 8/4/88, p. 5–94].

Second, compared with other sources (SIRC's prehearing submittal, the Cal/OSHA study), ADL estimates that many more workers are overexposed to styrene in these reinforced plastics industry segments. For example, ADL [Table 10, Ex. 34A] estimates that 20 percent of the workforce in the reinforced plastics segments of concern is overexposed to styrene, while the Cal/OSHA stydy [Ex. 3–742, Attach. 2, p. 30] reports that only 22 percent of the gel coat/lamination workers (who constitute approximately 36 percent of this work force) are overexposed. Thus, ADL used an inflated estimate of the number of overexposed workers in these segments; this factor also contributes substantially to an overestimation of costs.

Third, ADL seriously underestimates the existing level of baseline control in these segments. For example, ADL assumes that facilities have no controls in place. However, as the Cal/OSHA study, SIRC testimony, and OSHA's site visits show, this is not the case. At the time of ADL's 1980 study, spray booths may have been nonexistent in these facilities, but that is clearly not the case today.

Fourth, ADL underestimates the effectiveness of the methods available to control exposure. For example, the Cal/OSHA study found that many facilities with only minimal levels of control were routinely achieving the 50-ppm limit and that others that were exceeding 50 ppm could achieve compliance by adopting minor engineering improvements, implementing better/maintenance procedures, and instituting improved work practices [Ex. 3–742, Attach. 2, pp. 20, 29–33]. Dr. Daniel Boyd, speaking for SIRC, testified to the effectiveness of improved work practices (training workers to leave the spray booth when not involved in sprayup/layup operations, to position themselves properly during spray operations, etc.). Based on his experience, Dr. Boyd estimated that work practices alone could reduce employees' 8-hour exposures to styrene by 50 percent [Tr. 8/2/88, p. 5–106].

Fifth, ADL ignored control approaches based on substitution, preferring instead to estimate that major revamping of ventilation systems and installation of local exhaust ventilation would be necessary in all facilities. OSHA is aware that other materials cannot be substituted for styrene in all applications; however a costing methodology that relies exclusively on engineering controls ignores the movement in this sector away from styrene and high-emitting resins. A series of methodological problems, which compound each other, seriously undermines the usefulness of the ADL study. The Agency believes it more appropriate to rely on OSHA's industrywide survey as a source of data and to use the cost algorithm as a method of evaluating costs in these sectors. OSHA therefore concludes that the costs reported in the PRIA for SIC 30 are representative and reliable estimates.

The limit of 4 ppm for carbon disulfide may not be achievable with engineering controls in some operations performed during the manufacture of cellulosic food casings (SIC 308). These operations include unloading xanthate from the baratte, aligning of casing strands in the extrusion cabinet, and puncturing casings at the extrusion nozzle. Air-supplied hoods are currently used by workers performing these operations, and OSHA finds that respirators are likely to continue to be needed in these three processes, which require the opening of process machinery. Because employers will be permitted to use respirators to achieve compliance during these three operations, OSHA concludes that the cost estimates presented in the PRIA for SIC 30 accurately reflect costs for this industry.

Leather and Leather Products (SIC 31). One of the lowest costs of compliance in the manufacturing sectors is expected to occur in SIC 31, Leather and Leather Products ($2.4 million). In the leather and leather products industry sector, most of the affected establishments produce manufactured leather goods. The costs in this SIC are predominantly derived from gluing operations.

Stone, Clay, Glass and Concrete Product Manufacturing (SIC 32). The stone, clay, glass and concrete product industry is estimated to incur compliance costs of about $22.5 million. A major part of the annual costs in this industry segment may occur in the concrete, gypsum and plaster products (SIC 327) industries. According to the survey, controls in this sector are primarily expected to control silica generated during large scale crushing, grinding and sizing operations.

Primary Metal Manufacturing (SIC 33). The annual costs of compliance in primary metal manufacturing are estimated to total $71.0 million. The costs of compliance for this sector are heavily weighted by the cost of controls required in large establishments in this segment. Blast furnace establishments and primary foundries have large numbers of hot processes which require controls. Control of emissions from these hot metal processes to the proposed levels will require large increases in the volume of air being moved through the ventilation systems. Additional costs will be incurred to increase capacities of scrubbers and baghouses to remove the contaminants from the air.

The $71.0 million estimate includes engineering controls for processes where none are currently in use, as well as additional control of some already controlled proecesses. The controls for which costs have been estimated are sufficient for essentially all of the facilities in this sector. However, this estimate may somewhat underestimate the compliance costs because it does not take into account the additional costs at a small number of very large facilities where these engineering controls may not be sufficient. This situation arises in SIC 3312 at the blast furnaces and basic oxygen furnaces (BOFs) in the few (about 15) remaining integrated steel mills, which operate on a substantially larger scale than the other facilities in this sector. It is the scale of these approximately 15 operations (which account for about 80 percent of domestic steel production) which requires more extensive controls than other facilities in this sector.

Because engineering controls alone are likely to be insufficient to consistently control exposures to the proposed PELs around the blast furnaces and BOFs at the integrated mills, OSHA anticipates that respirators will be needed in addition to the engineering controls. Engineering controls could possibly be installed to fully meet the proposed exposure levels, but the cost would likely be prohibitive, about $10 million per facility. The estimated cost of $71.0 million for this sector takes into account engineering controls such as improved air purification in control rooms and purified air showers at some work stations. These improvements would help to control exposures, but might not always be sufficient to meet the new standards. Thus, OSHA has also included an annual cost of $7.41 million for respirators at the integrated mills (included in the $71 million total estimate).

Fabricated Metal Products Manufacturing (SIC 34). Plating and coating establishments (SIC 347) and miscellaneous fabricated products (SIC 349) would account for a major portion of the $39.4 million annual costs in SIC 34. Worker exposures in this industry sector result from chemicals used in plating processes, solvents and coatings, metals and dusts. The survey indicated that ventilation systems are not now present at many of the processes with chemical exposure.

Machinery Except Electrical (SIC 35) and Electrical Machinery (SIC 36). The machinery manufacturing sectors together are estimated to incur total annual compliance costs of $65.9 million. Machinery except electrical accounts for $45.2 million of this total. The electrical machinery sector is estimated to require $20.7 million in annual compliance costs. Controls in these sectors would be required for exposures to metals, solvents and welding fumes.

Transportation Equipment Manufacturing (SIC 37). Annual costs of compliance for SIC 37 are estimated at $49.8 million. Costs in the truck and car body and motor vehicle parts sectors (SICs 3711, 3713, 3714) would account for a large percentage of the costs in SIC 37. Controls may be needed in order to control exposures to heavy metals, solvents, welding fumes and a large variety of other chemicals at large scale hot processes. Additionally, costs in small plants in this sector will include compliance activities to control exposures to styrene and other chemicals in small boat construction, as well as trailer and recreational vehicle insulation.

The Styrene Information Research Council (SIRC) presented the results of a study by Arthur D. Little, Inc. (ADL) of the costs of meeting a 50-ppm, 8-hour TWA for styrene in the boat-building industry [Exs. 3–742; 34A; Tr. 8/3/88, pp. 5–117 to 5–130]. The ADL study concluded that capital costs for boat builders would be $714.3 million and operating costs would be $132.1 million per year [Ex. 34A, Table 2]. If these costs are annualized using OSHA's interest and life-of-equipment assumptions, annualized costs for firms in this sector would be $249.2 million per year. OSHA's PRIA estimated a total annualized cost for all of SIC 37 (which includes many other segments in addition to boat-building) of $47 million per year (53 FR 21736). There is thus a substantial disagreement between ADL's chemical- and industry-specific

estimate and OSHA's estimate for the entire sector.

OSHA believes that ADL's estimates grossly overestimate costs for controlling styrene in the boat-building sector. For example, ADL estimates that 50 percent of all workers in this sector are exposed to styrene levels of greater than 50 ppm as 8-hour TWA's. However, Daniel Boyd, testifying for SIRC, estimated that not more than 20 percent of employees engaged in boat building are directly exposed to styrene in the gel-coat and lamination processes; according to Dr. Boyd, the remainder of employees work in assembly and shipping and have little direct exposure to styrene [Tr. 8/3/88, p. 5-100]. OSHA's site visits to boat-building facilities in this sector [Exs. 136A, 136B] confirm that no more than 20 percent of employees in boat building facilities work in jobs having direct exposure to styrene.

ADL also chose to use 1980 exposure data to construct an exposure baseline for costs in this segment. OSHA finds that extensive exposure and control data collected in the Cal/OSHA study [Ex. 3-742, Attach. 2] superior to the 1980 ADL data because they are more recent, more extensive, specifically related to control measures (both engineering and work practice), and reflect good industrial hygiene practice (ADL, for example, calculates 8-hour TWAs on the basis of 1- or 2-hour samples, while Cal/OSHA uses appropriate sampling techniques). The Cal/OSHA study determined that the mean 8-hour TWA exposure for gel-coat and lamination workers (who are the most heavily styrene-exposed employees) were generally lower than reported by ADL.

In addition to overestimating both exposure levels and the number of workers overexposed, the ADL study [Ex. 33-742, Attach. 9, pp. 1-5] assumes that an extensive system of engineering controls and work practices would be required to achieve exposures of 50 ppm or less, i.e., ADL assumes a very low (or nonexistent) baseline level of control. However, both the Cal/OSHA study and OSHA's site visits [Exs. 136A, 136B] show that most gel-coat application is being done today in a spray booth [Ex. 3-742, Attach. 2, p. 20], and that many gel-coat operators have 8-hour TWA exposures of less than 50 ppm.

Further, based on the Agency's feasibility assessment for manual layup and sprayup operations within SIC 37, OSHA is permitting respirators to be used during these operations to achieve the revised limits for styrene. Thus, it is unlikely that employers will incur substantial costs to implement engineering controls for manual layup and sprayup operations.

Finally, ADL did not consider the impact of substitution of lower-emitting styrene resins or of other, less hazardous substances in lieu of styrene on worker exposures. OSHA therefore concludes that the costs reflected in the PRIA for SIC 37, which are based on data from the survey and estimates developed by the cost of algorithm, are an accurate representation of costs to firms in this sector.

Instruments Manufacturing (SIC 38). Annual control costs in SIC 38 are estimated to total $9.6 million. Exposures in this sector are to a large number of chemicals used within instruments and to various metals and solvents.

Miscellaneous Manufacturing (SIC 39). This industry accounts for a wide range of products, processes and chemical exposures. About half of the establishments that would incur the $15.8 million annual cost in the industry are believed to be included in SIC 3999, miscellaneous manufacturing not elsewhere classified.

The Casket Manufacturers Association of America (CMAA) commented that achievement of the proposed hardwood dust limit of 1 mg/m^3 would impose prohibitive costs on casket manufacturers [Ex. 8-78]. The CMAA presented estimates of the costs it anticipates as a result of the proposed limit; these costs were derived by estimating per-machine ventilation costs, multiplying this estimate by the number of machines per plant, and then multiplying by 18 plants [Ex. 8-78]. The CMAA estimated costs from a zero (no control) baseline and from an incremental baseline [Ex. 8-78]. Because the use of a zero-cost baseline is not appropriate when estimating potential compliance costs, OSHA has focused on the CMAA's incremental costs. According to the CMAA, total costs for 18 companies to achieve a 1 mg/m^3 limit for hardwood dust would be $1.32 million, or $73,000 per plant.

OSHA believes that the CMAA has seriously overestimated compliance costs. First, the final rule has adopted a 5 mg/m^3 limit for wood dust (the 2.5 mg/m^3 Western red cedar dust limit does not affect casket manufacturers because they do not use this wood). Second, the CMAA estimates assume that all machines in all facilities will need local exhaust ventilation, when in fact only a few machines would need to be engineered since only hand- and machine-finishing operations present an exposure problem, according to the CMAA [Ex. 8-87]. Finally, the recent exposure data collected and submitted by the CMAA show that, even under a worst-case scenario, seven of nine sample results were below the 5 mg/m^3 limit (see detailed discussion for SIC 39 in the Technological Feasibility section of the preamble). These exposure results demonstrate that most employees and operations are already below the final rule's 5 mg/m^3 limit and will therefore incur no costs.

Thus OSHA finds that the costs projected by the CMAA are unlikely to be incurred by hardwood casket manufacturers. The Agency's PRIA cost estimates for SIC 39 appear to be accurate and take into account costs of the magnitude likely to be encountered by these manufacturers.

One comment was received from a participant concerned about the costs of achieving the proposed limit for styrene in the manufacturing of diving boards, a business that is classified in SIC 3949, Sporting and Athletic Goods (nec). This commenter [Ex. 3-380] was of the opinion that the equipment changes and plant restructuring required to comply with the proposed limit would require a complete shut-down of affected facilities, and that this closure would result in such a substantial loss of revenue that economic feasibility would become an issue [Ex. 3-380].

In response, OSHA notes that a review of the record evidence has shown that the great majority of all exposure samples and reinforced plastics facilities potentially affected by the revised standard are already achieving compliance with this limit or are very close to doing so (see discussion of Technological Feasibility for SICs 30 and 37). In those few cases where compliance is not presently being achieved, OSHA has determined that improved work practices, such as having employees leave the booth when not engaged in manual layup operations and having them stand downwind, and making minor adjustments in ventilation will achieve the final rule's PEL. Thus OSHA finds that the cost impacts projected by this commenter [Ex. 3-380] are not likely to be incurred by diving board manufacturers.

Transportation and Utilities (SICs 40, 45, 47, and 49). The transportation and utilities sectors (SICs 40, 45, 47, and 49) include a large number of establishments. However, operations at Railroad (SIC 40), and Air Transport establishments (SIC 45) are subject to regulation by other Federal agencies in addition to OSHA. Consequently, the number of establishments which would incur costs to comply with the final standard are limited. For railroads, OSHA's standards normally apply to

off-track operations. The estimated cost of compliance for SIC 40 is $1.1 million, while the cost for SIC 45 is $3.7 million.

Transportation Services Sector (SIC 47). The $3.8 million annual costs in SIC 47 will primarily be incurred in SIC 4789, transportation services not elsewhere classified. This sector includes establishments which provide incidental services such as cleaning railroad ballast and other rail car maintenance.

Electric, Gas and Sanitary Service Utilities (SIC 49). Annual costs in the utilities sectors are estimated to total $38.0 million. Costs would result from installation and improvement of controls necessary for activities such as boiler/furnace feed preparation in electric services, odorant addition by natural gas companies and water treatment and purification of water supplies.

Edison Electric Institute (EEI) estimates that in electric utility operations where exposures are intermittent in nature and limited in duration, engineering controls to reduce exposure would likely cost one to two million dollars per generating unit [Ex. 3–831]. However, intermittent activities such as boiler and precipitator cleaning would not require the installation of engineering controls, so these costs would not be incurred. During these intermittent activities, workers do have the option of wearing respirators. OSHA's cost estimate for SIC 49 does reflect costs to control exposures to coal dust generated in material handling operations.

The remaining cost estimate from EEI is $12–46 million per unit to modify the approximately 428 positive pressure boilers currently operating in the United States. EEI contends that if electric utilities lost their flexibility in using personal protective equipment to meet the proposed PELs, the boilers would have to be modified to reduce potential leaks of nitrogen dioxide and sulfur dioxide [Ex. 3–831]. OSHA believes that in most cases where overexposures might occur, they could be corrected by general ventilation or directed blowers and by correcting the most severe emission points. The prediction for such radical and costly modifications of power generating equipment does not appear to be well grounded.

The Interstate Natural Gas Association of America (INGAA) stated that costs to control "exposure to emissions from combustion sources that are ducted to ambient air" would be prohibitively costly [Ex. 3–739]. However, INGAA did not provide any specific explanations as to possible errors in OSHA's cost analysis. Thus OSHA did not have any additional evidence with which to compare its costs. After reviewing its methodology and survey data, OSHA concludes that the costs of compliance for the natural gas industry were adequately represented.

Wholesale Trade (SICs 50, 51). Costs in the wholesale trade sectors (SICs 50, 51), are estimated to total about $17.2 million annually. A large percentage of the total number of establishments which would incur costs to comply with the final rule are in SIC 5093, Scrap and Waste Materials, wholesale.

Several of the commenters who submitted data and information on the technological feasibility of achieving the Agency's proposed grain dust standard of 4 mg/m^3 in SIC 5153, Wholesale Trade and Grain and Field Beans, also expressed concern about the costs of compliance OSHA estimated for this sector in the PRIA. The PRIA estimated that approximately 10 percent of the grain elevators classified in SIC 5153 would incur costs to meet the proposed 4 mg/m^3 PEL and that the average per-elevator annualized costs would be $6,000 per year (Ex. 33). OSHA's estimates were based on data derived from the survey and calculated using the cost algorithm.

The National Grain and Feed Association (NGFA) presented a different estimate of the compliance costs that owners of grain elevators in this SIC category would incur (Ex. 3–752) to reach the proposed 4 mg/m^3 PEL. To derive its estimates, the NGFA used the following assumptions:

(1) All grain elevators processing wheat, oats, or barley will need pneumatic dust control systems and do not now have them;

(2) Eighty-seven percent of all grain elevators process wheat, oats, or barley;

(3) The costs of pneumatic dust control systems are those estimated by Booz Allen in a study done for OSHA in connection with the Agency's grain handling standard (inflated by 15 percent to convert them from 1984 to 1988 dollars).

Using these assumptions, the NGFA estimated total capital costs for all affected grain elevators at $1.9 billion. If these costs are annualized using OSHA's interest and life-of-capital-equipment assumptions and including an operating cost component calculated at 10 percent of capital costs, annualized costs would be $500 million per year, most of which reflect costs for country elevators. If the NGFA's estimated capital costs are used as a starting point, the average annualized per-elevator cost would be $41,125.

OSHA finds that the NGFA's estimates seriously overstate potential compliance costs for two principal reasons:

(1) OSHA has determined, as described for SIC 51 in the Technological Feasibility section of the preamble, that the PEL established in the final rule will be 10 mg/m^3, rather than the proposed PEL of 4 mg/m^3;

(2) The NGFA overestimates the number of grain elevators potentially affected by the new standard.

OSHA believes that no more than 10 percent, rather than the 87 percent projected by the NGFA, of all SIC 51 grain elevators will incur costs to achieve the 10 mg/m^3 PEL, because most elevators are already achieving this level. Data in the record show that:

(1) Only 5 percent of 109 8-hour TWA samples taken in grain elevators in one study were above 10 mg/m^3 [Rankin et al. 1986];

(2) Fewer than 5 percent of 203 8-hour TWA samples from grain handling facilities characterized as "small" were above 10 mg/m^3 [Ex. 3–751, Attach. 2 and Fig. 1, Docket H–0117];

(3) Only 12 percent of all total dust samples taken at 6 elevators in 3 states were above 10 mg/m^3 [Ex. 3–751, Attach., Docket H–0117]; and

(4) Only 6 percent of the employee full-shift exposures taken by NIOSH in a grain elevator were above 10 mg/m^3 [NIOSH HHE 76–13–316].

These data confirm that no more than 10 percent of all SIC 5153 elevators will be affected by the final standard. Further, these data make it clear that controls will be needed only in those instances and areas where the 10 mg/m^3 is not already being achieved, and that the complete, facility-wide installation of pneumatic control systems envisioned by the NGFA to meet a 4 mg/m^3 PEL will rarely, if ever, be required. OSHA has not reduced the compliance costs included in the PRIA for this sector despite the increase in the PEL from 4 to 10 mg/m^3; as such, costs are believed to be conservative and may overstate actual expenditures needed to comply with the new level.

Auto Dealers (SIC 55). The only retail trade sector expected to incur compliance costs, Auto Dealers (SIC 55) is estimated to incur $13.6 million annually. These costs result from the potentially large number of motor vehicle dealers (SIC 5511) which may incur compliance costs to control exposures to paints, coatings and solvents during vehicle spray and coating operations. The costs result from the installation of paint spray booths.

Service Sectors (SICs 72, 73, 75, 76, and 80). The service sectors, SICs 72, 73, 75, 76 and 80 are estimated to total

about $26.7 in annual compliance costs. The major costs in these sectors would result from potential compliance activities in SIC 721, laundry, cleaning and garment services. Establishments in SIC 721 would incur annual operating and annualized capital costs to control exposures for dry cleaning operations.

Because the limit for perchloroethylene was lowered from the proposed level of 50 ppm to 25 ppm, the engineering control designed for dry cleaning was reevaluated. OSHA reevaluated the control design used to project cost in the preliminary regulatory impact analysis. The air flow rate to control exposures at 25 ppm was increased, resulting in a unit cost increase of $910, making the revised unit cost $2,410. OSHA is aware of the improvements in dry cleaning equipment, particularly the increasing use of dry-to-dry machines. Based on information provided by the International Fabricare Institute and the Amalgamated Clothing and Textile Workers Union regarding replacement rates for drycleaning machines, OSHA believes that virtually all machines in use will be dry-to-dry by 1992 [Ex. 3-671]. The average perchloroethylene exposure associated with dry-to-dry machines is 23.9 ppm. Thus, it is anticipated that the PEL will be met largely by the normal rate of retirement of existing equipment.

Additional costs in the service sectors may result from control of solvent chemicals in SIC 734, Building Services, control of welding fumes at Welding Repair operations (SIC 7692), control of solvent and photographic chemicals in Mailing, Reproduction, Commerical Art, Photography and Stenographic Services (SIC 733), and local ventilation for exposure control in SIC 8071, Medical Laboratories.

Per Plant Average Costs

Table G-3 presents the estimated average per plant annual cost of compliance by industry sector. Costs shown in this Table are calculated only for those establishments in a sector which would incur costs. Average per plant annual operating and annualized capital costs for all affected establishments across industry sectors are estimated at $6,000. The per plant cost for large plants is $13,000 and for small plants with fewer than 20 employees, $3,100.

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TABLE G-3

AVERAGE PER PLANT ANNUAL COSTS AND NUMBERS OF AFFECTED PLANTS (a)

SIC (b)	SIC DESCRIPTION	ANNUAL COST	TOTAL # OF PLANTS	# OF AFFECTED PLANTS	% AFFECTED	AVERAGE COST PER AFFTED PLANT	AVERAGE COST PER LARGE AFFTED PLANT	AVERAGE COST PER SMALL AFFTED PLANT
20	FOOD PROD. (c)	$33,493,100	29,000	4,932	16.98%	$6,800	$13,000	$3,600
21	TOBACCO (c)	$19,700	200	3	1.39%	$6,600	$6,600	$0
22	TEXT. MILL (c)	$29,478,400	11,000	2,765	25.08%	$10,700	$21,400	$3,700
23	APPAREL PROD. (c)	$31,744,200	30,000	6,179	20.57%	$5,100	$11,500	$2,000
24	LUMBER & WOOD	$56,720,800	27,100	18,427	68.00%	$3,100	$4,200	$2,700
25	FURNITURE	$21,075,800	12,700	5,062	40.00%	$4,200	$12,400	$1,800
26	PAPER PROD.	$30,998,700	7,000	3,518	50.00%	$8,800	$15,200	$800
27	PRINTING & PUB.	$33,754,500	60,300	3,597	6.88%	$9,400	$6,200	$10,600
28	CHEMICAL PROD.	$35,454,700	16,400	3,007	18.31%	$11,800	$16,200	$5,400
29	PETRO. REFINING	$23,686,000	2,300	306	13.25%	$77,400	$109,600	$700
30	RUBBER & PLASTICS	$111,093,400	15,100	3,562	26.22%	$31,200	$27,000	$35,100
31	LEATHER PROD.	$2,414,700	2,300	300	13.46%	$8,000	$10,400	$6,400
32	STONE & CLAY	$22,457,800	15,900	3,267	22.80%	$6,900	$12,200	$3,400
33	PRIM. METAL	$70,957,600	8,000	2,411	30.03%	$29,400	$41,900	$6,200
34	FAB. METALS	$39,419,700	37,300	4,597	14.50%	$8,600	$15,800	$3,800
35	MACHINERY	$45,206,600	64,400	6,801	10.56%	$7,800	$14,600	$3,000
36	ELEC. MACH.	$20,667,500	21,600	2,359	10.92%	$7,800	$14,500	$3,000
37	TRANS. EQUIP.	$49,792,400	13,600	4,979	36.56%	$10,000	$11,800	$8,800
38	INSTRUMENTS	$9,633,500	12,000	1,289	10.74%	$7,800	$14,500	$3,000
39	MISC. MANUF.	$15,842,600	25,300	2,649	10.47%	$7,800	$14,600	$3,000
40	R.R. TRANS.	$1,083,400	400	93	20.86%	$11,700	$11,700	$0
45	AIR TRANS.	$3,740,500	5,500	320	5.79%	$11,700	$11,700	$0
47	TRANS. SERV.	$3,789,400	26,200	324	1.24%	$11,700	$11,700	$0
49	ELEC. GAS. SAN.	$38,009,300	15,800	3,485	22.24%	$10,900	$17,000	$3,600
50	WHOLESALE TRADE	$2,995,300	5,800	801	13.78%	$3,400	$6,200	$2,900
51	WHOLESALE, NON-DUR	$14,215,800	33,600	4,436	13.22%	$3,400	$6,200	$2,900
55	AUTO DEALERS	$13,550,500	165,800	24,847	14.99%	$360	$2,000	$300
72	PERSONAL SRV.	$10,872,100	95,500	5,217	5.47%	$2,200	$6,000	$1,000
73	BUSINESS SRV.	$2,422,100	12,100	800	6.61%	$2,200	$8,300	$1,500
75	AUTO REPAIR	$6,143,500	91,500	8,351	9.13%	$600	$3,500	$300
76	MISC. REPAIR SRV.	$2,809,900	15,100	1,163	11.56%	$2,400	$12,400	$2,100
80	HEALTH SERV. (c)	$4,439,400	222,800	1,158	0.52%	$3,800	$12,500	$2,100
TOTAL		$787,982,900	1,101,600	131,005	11.89%	$6,000	$13,000	$3,100

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

(a) Costs were calculated by annualizing the capital cost over the projected life of the equipment (10 years) using a 10 percent cost of capital and adding an annual operating and maintenance cost estimated at 10 percent of the capital cost.

(b) Industry sectors not identified in this table include industries with no major cost impact expected, the construction industry, which will be the subject of a separate regulatory analysis, and industries such as mining, over which OSHA has no jurisdiction.

(c) Costs in these sectors were based on expert judgement and secondary data collection.

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The highest costs on an average per plant basis are expected to occur in SIC 29. Averge per plant costs for large plants in SIC 29 may total $109,600 in annual operating and annualized captial costs. Per plant costs in SIC 29 are substantially higher than those in the next highest industry, SIC 30, Rubber and Plastics. The $31,200 per plant costs in this industry result from above average compliance costs estimated for exposure control in molding and vulcanizing in large plants and crushing and grinding operations in small plants.

Although small establishments account for about 73 percent of the 131,005 affected establishments, compliance costs for small establishments are expected to account for only 36 percent of total industry compliance costs.

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TABLE G-4
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SIC 24 = LUMBER AND WOOD

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
ABRASIVE BLASTING	206	0	0	$7,200	$0
ADHESIVE BINDING	2	4	4	$3,070	$12,116
ASSEMBLY	237	3,567	172	$1,140	$196,170
BATCH PROCESS COKE PRODUCTION/REMOVAL	53	0	0	$0	$0
BLEACHING	53	264	264	$2,900	$765,762
BOILERS	57	6	0	$180	$0
CALENDARING/WINDING	2	4	0	$180	$0
CLEANING	205	0	0	$710	$0
COATING/SPRAYING/FINISHING/LAYUP	6,048	22,501	1,775	$3,070	$5,448,216
CRUSHING/GRINDING/CALCINING	2	0	0	$4,740	$0
CUTTING/SAWING/PLANNING	21,525	96,614	15,458	$1,900	$29,370,656
DRYING/BAKING	1,085	3,834	181	$4,740	$858,878
GLUEING/HOT PRESSING	9,070	25,453	1,182	$3,070	$3,628,740
LOADING/OFFLOADING/RECEIVING/HANDLING	2	59	0	$1,120	$0
METAL WORKING (ROLLING, MILLING, SHAPING)	2	197	0	$1,140	$0
OTHER	473	13,799	0	$1,140	$0
PLATE CLEANING	42	77	0	$710	$0
POLISHING (SURFACE)/GRINDING	7	21	0	$1,140	$0
PULP SCREENING/WASHING	2	22	22	$2,900	$62,947
REBLENDING/REMIXING	2	22	0	$1,140	$0
RECOVERY/REPROCESSING/RECLAMATION	2	4	0	$21,900	$0
SANDING/POLISHING/GRINDING	7,574	45,225	7,236	$2,200	$15,919,200
SEPARATION	2	4	0	$1,120	$0
STAMPING/SHAPING/MOLDING/PRESSING	9	53	0	$2,900	$0
WELDING/SOLDERING	2	4	0	$1,140	$0
ZSUBTOTAL	46,664	211,734	26,294	$71,450	$56,262,684
ZZMAINTENANCE	18,116	2,743	148	$520	$457,965
ZZTOTAL	64,780	214,477	26,442	$71,970	$56,720,649

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SIC 25 = FURNITURE

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
ADHESIVE BINDING	5	5	0	$3,070	$0
ASSEMBLY	216	489	0	$1,140	$0
COATING/SPRAYING/FINISHING/LAYUP	6117	13182	685	$3,070	$2,102,014
CUTTING/SAWING/PLANNING	6821	28050	4460	$1,900	$8,474,469
DEBURRING	229	457	0	$7,200	$0
DRILLING/BORING	1040	1197	192	$2,200	$421,344
DRYING/BAKING	704	1555	0	$4,740	$0
GLUEING/HOT PRESSING	5432	10829	0	$3,070	$0
LOADING/OFFLOADING/RECEIVING/HANDLING	14	180	14	$1,120	$15,641
MACHINING/GRINDING/WELDING/BRAZING	51	771	0	$1,140	$0
OTHER	56	1169	0	$1,120	$0
PACKAGING/BAGGING	65	84	0	$1,120	$0
SANDING/POLISHING/GRINDING	7539	28052	4488	$2,200	$9,874,304
STAMPING/SHAPING/MOLDING/PRESSING	99	718	0	$2,900	$0
WELDING/SOLDERING	51	514	0	$1,140	$0
ZSUBTOTAL	28439	87252	9839		$20,887,672
ZZMAINTENANCE	12804	945	361	$520	$187,885
ZZTOTAL	41243	88197	10200		$21,075,557

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SIC 26 = PAPER PRODUCTS

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
ADHESIVE BINDING	37	7	7	$3,070	$20,810
BLEACHING	38	150	73	$2,900	$211,326
BLENDING/MIXING/FORMULATING	135	363	0	$3,070	$0
BOILERS	38	119	11	$180	$2,033
CALENDARING/WINDING	213	739	136	$180	$24,549
COATING/SPRAYING/FINISHING/LAYUP	565	2883	141	$3,070	$433,631
CRUSHING/GRINDING/CALCINING	2	5	0	$4,740	$0
CUTTING/SAWING/PLANNING	1899	9504	1521	$1,900	$2,889,216
DIGESTER	29	138	9	$14,000	$126,535
DON'T KNOW	756	0	0		$0
DRYING/BAKING	1328	9052	305	$4,740	$1,445,970
EXTRUSION	71	140	2	$1,140	$2,575
FLEXOGRAPHIC PRINTING	2	43	0	$1,380	$0
FOAM PROCESSING	69	1718	0	$1,140	$0
GLUEING/HOT PRESSING	1663	8384	873	$3,070	$2,679,276
LINOTYPE SETTING	2	5	0	$1,380	$0
LITHOGRAPHIC PRINTING	7	23	0	$1,380	$0
LOADING/OFFLOADING/RECEIVING/HANDLING	5	9	0	$1,120	$0
OTHER	254	1248	0	$90	$0
PACKAGING/BAGGING	1669	12651	1452	$1,120	$1 626,905
PRESS SECTION	246	962	136	$180	$24,492
PRINTING	716	3666	946	$1,380	$1,306,073
PULP SCREENING/WASHING	38	237	133	$2,900	$386,612
RECOVERY/REPROCESSING/RECLAMATION	38	91	25	$14,000	$352,492
SHEET PROCESS	2	7	0	$90	$0
SHREDDING/WASTE PROCESSING	1347	4309	1198	$14,000	$16,774,349
SIZE PRESS/COATERS	366	990	611	$180	$109,969
STAMPING/SHAPING/MOLDING/PRESSING	46	132	11	$2,900	$32,763
WATER TREATMENT	67	122	74	$14,000	$1,031,668
WET END	233	753	182	$180	$32,802
ZSUBTOTAL	11871	58370	7847		$29,512,044
ZZMAINTENANCE	7022	3357	2858	$520	$1,486,291
ZZTOTAL	18893	61727	10705		$30,998,335

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SIC 27 - PRINTING AND ALLIED INDUSTRIES

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
ADHESIVE BINDING	5667	8109	197	$3,070	$604,003
BLENDING/MIXING/FORMULATING	39	197	0	$4,740	$0
BOILERS	20	20	0	$180	$0
CALENDARING/WINDING	113	678	0	$180	$0
CLEANING	1602	9629	9629	$710	$6,836,424
CUTTING/SAWING/PLANNING	38	283	0	$180	$0
DEGREASING	20	20	0	$710	$0
DRILLING/CUTTING/FLAME-JET LANCING	18	111	111	$1,140	$126,499
DRYING/BAKING	39	39	0	$4,740	$0
FILM PROCESSING	2115	4194	0	$1,240	$0
GRAVURE PLATEMAKING	361	1105	0	$1,380	$0
GRAVURE PRINTING	22	328	0	$1,380	$0
INJECTION MOLDING	20	315	0	$1,140	$0
LETTERPRESS PRINTING	7526	18729	0	$1,380	$0
LINOTYPE SETTING	8372	12642	20	$1,380	$27,150
LITHOGRAPHIC PLATEMAKING	17757	22169	99	$1,380	$136,716
LITHOGRAPHIC PRINTING	27040	87625	17540	$1,380	$24,204,521
MATERIALS MANUFACTURE/FABRICATION	20	236	0	$1,140	$0
METAL PLATING	20	138	0	$710	$0
MONO OR LINOTYPE SETTING	6367	9629	0	$1,380	$0
OTHER	3884	4900	0	$1,140	$0
PHOTOENGRAVING	418	1132	0	$1,380	$0
PHOTOENGRAVING PLATEMAKING	113	0	0	$1,380	$0
PHOTOGRAVURE	1850	5415	678	$1,380	$936,090
PLATE CLEANING	113	0	0	$710	$0
PLATE MAKING	268	764	0	$1,380	$0
PRINTING	3687	6981	0	$1,380	$0
SANDING/POLISHING/GRINDING	20	20	0	$2,200	$0
SCREEN PRINTING	304	1306	0	$1,380	$0
SCREEN STENCIL PLATEMAKING	133	585	0	$1,380	$0
SEMICONDUCTOR PHOTORESIST	20	413	0	$710	$0
STAMPING/SHAPING/MOLDING/PRESSING	18	148	148	$2,900	$429,063
STRIPPING/PAINT REMOVING	113	452	0	$1,140	$0
ZSUBTOTAL	88114	198311	28421		$33,300,466
ZZMAINTENANCE	60282	19919	678	$520	$454,041
ZZTOTAL	148396	218230	29099		$33,754,507

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SIC 28 = CHEMICALS AND ALLIED PRODUCTS

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
ADHESIVE BINDING	104	219	0	$3,070	$0
ASSEMBLY	6	714	0	$1,140	$0
BLENDING/MIXING/FORMULATING	7640	35776	1988	$3,070	$6,104,570
BLOWING/MOLDING	2	6	0	$90	$0
BOILERS	7	89	0	$180	$0
CALCINING KILN	3	5	0	$4,740	$0
CALENDARING/WINDING	8	604	604	$180	$108,707
CLEANING	71	471	26	$710	$18,119
COATING/SPRAYING/FINISHING/LAYUP	137	570	16	$3,070	$47,818
CRUSHING/GRINDING/CALCINING	1196	5740	542	$4,740	$2,570,110
CUTTING/SAWING/PLANNING	80	149	0	$180	$0
DIGESTER	45	360	0	$14,000	$0
DRYING/BAKING	1204	3455	230	$4,740	$1,088,607
EXTRUSION	222	1460	123	$1,140	$140,758
GLUEING/HOT PRESSING	2	9	0	$3,070	$0
IMPREGNATION	53	153	0	$1,140	$0
INJECTION MOLDING	2	28	0	$1,140	$0
LITHOGRAPHIC PRINTING	6	6	0	$1,380	$0
LOADING/OFFLOADING/RECEIVING/HANDLING	6100	14631	2065	$1,120	$2,312,937
MATERIALS MANUFACTURE/FABRICATION	7	35	0	$1,140	$0
MEASUREMENT	11	33	0	$1,120	$0
MONOTYPESETTING	25	25	0	$1,380	$0
OTHER	162	6899	0	$1,120	$0
OTHER	3	15	0	$90	$0
OTHER	261	392	0	$90	$0
OTHER	3	0	0	$710	$0
PACKAGING/BAGGING	5137	23614	4538	$1,120	$5,082,081
PACKAGING/REPACKAGING	29	29	0	$710	$0
PRINTING	2	14	0	$1,380	$0
PROCESS INSP/SUPERVISION/QUAL CONTROL	39	80	0	$1,120	$0
PULP SCREENING/WASHING	5	638	0	$2,900	$0
REACTION/FERMENTATION	1680	7889	3855	$1,120	$4,317,769
RECOVERY/REPROCESSING/RECLAMATION	628	1113	429	$21,900	$9,402,121
SAMPLING	2	0	0	$1,120	$0
SANDING/POLISHING/GRINDING	36	69	0	$2,200	$0
SEPARATION	1521	6026	2269	$1,120	$2,541,828
STAMPING/SHAPING/MOLDING/PRESSING	45	169	20	$2,900	$58,846
STERILIZATION	8	8	6	$710	$4,225
VULCANIZATION/CURING	36	380	0	$90	$0
WATER TREATMENT	13	38	37	$14,000	$511,930
ZSUBTOTAL	26539	111911	16748		$34,310,426
ZZMAINTENANCE	16426	3952	497	$520	$1,144,315
ZZTOTAL	42965	115863	17245		$35,454,741

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SIC 29 = PETROLEUM REFINING AND RELATED PRODUCTS

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
ADHESIVE BINDING	8	15	0	$3,070	$0
BATCH PROCESS COKE PRODUCTION/REMOVAL	47	208	16	$150,000	$2,281,338
BLENDING/MIXING/FORMULATING	649	2004	23	$4,740	$107,388
CALCINING KILN	2	11	0	$4,740	$0
CALENDARING/WINDING	11	13	0	$180	$0
COATING/SPRAYING/FINISHING/LAYUP	24	38	0	$3,070	$0
CUTTING/SAWING/PLANNING	12	14	2	$180	$392
DRYING/BAKING	108	178	0	$4,740	$0
FELTING	2	2	0	$3,070	$0
IMPREGNATION	6	6	0	$3,070	$0
LOADING/OFFLOADING/RECEIVING/HANDLING	745	4065	278	$1,120	$311,593
MEASUREMENT	112	787	0	$1,120	$0
OTHER	2	4	0		$0
OTHER	11	45	0	$1,120	$0
OTHER	2	9	0	$4,740	$0
OTHER	6	0	0	$4,740	$0
OTHER	45	116	0	$4,740	$0
PACKAGING/BAGGING	342	931	83	$1,120	$92,457
PRESS SECTION	2	2	0	$180	$0
PROCESS INSP/SUPERVISION/QUAL CONTROL	214	2299	1441	$1,120	$1,614,469
REACTION/FERMENTATION	37	205	72	$1,120	$80,102
RECOVERY/REPROCESSING/RECLAMATION	25	54	0	$21,900	$0
SAMPLING	267	5790	3065	$1,120	$3,433,000
SAMPLING OF PIPE LINES	16	16	0	$1,120	$0
SANDING/POLISHING/GRINDING	2	7	0	$2,200	$0
SEPARATION	41	73	28	$1,120	$30,912
WATER TREATMENT	215	1646	1103	$14,000	$15,440,626
WELDING/SOLDERING	2	0	0	$1,140	$0
ZSUBTOTAL	2953	18538	6109		$23,392,277
ZZMAINTENANCE	2315	875	318	$520	$293,765
ZZTOTAL	5268	19412	6427		$23,686,042

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SIC 31 = LEATHER AND LEATHER PRODUCTS

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
ASSEMBLY	195	770	0	$1,140	$0
BEAMHOUSE	29	171	0	$2,510	$0
BLENDING/MIXING/FORMULATING	12	35	35	$3,070	$61,419
CALENDARING/WINDING	12	47	47	$180	$8,375
CLEANING	22	111	30	$710	$21,070
COATING/SPRAYING/FINISHING/LAYUP	204	547	0	$3,070	$0
COLORING/DYEING	44	420	0	$0	$0
CUTTING/SAWING/PLANNING	59	232	0	$180	$0
DEGREASING	12	91	0	$710	$0
EXTRUSION	12	105	0	$1,140	$0
FAT LIQUORING	27	281	0	$720	$0
FINISHING	66	148	0	$1,820	$0
GLUEING/HOT PRESSING	1,059	5,540	736	$3,070	$2,258,853
INJECTION MOLDING	10	69	0	$1,140	$0
LOADING/OFFLOADING/RECEIVING/HANDLING	35	54	0	$1,120	$0
OTHER	12	931	0	$1,120	$0
PACKAGING/BAGGING	35	35	0	$1,120	$0
PRESERVATION/DEFESTATION/DISINFECTION	34	58	41	$720	$29,466
SPLITTING/SHAVING	22	77	0	$720	$0
STAMPING/SHAPING/MOLDING/PRESSING	22	241	0	$2,900	$0
TANNING/RETANNING	51	158	10	$2,510	$25,887
ZSUBTOTAL	1,971	10,120	999		$2,652,183
ZZMAINTENANCE	2,326	0	0	$520	$9,646
ZZTOTAL	4,297	10,120	999		$2,414,719

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SIC 32 = STONE, CLAY, GLASS AND CONCRETE PRODUCT MANUFACTURING

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
ANNEALING/QUENCH/TEMPER	92	641	0	$710	$0
ASSEMBLY	30	281	0	$1,140	$0
BATCH MAKING	1,205	1,262	0	$4,740	$0
BISCUIT FIRING	110	110	0	$4,740	$0
BLENDING/MIXING/FORMULATING	4,005	22,404	284	$4,740	$1,346,994
BLOWING/MOLDING	112	1,672	544	$90	$48,937
BONDING	1,157	2,463	2,200	$3,070	$6,753,534
CALCINING KILN	139	241	11	$4,740	$54,320
CALENDARING/WINDING	11	92	0	$180	$0
CASTING	1,319	1,756	382	$3,890	$1,484,491
CHIPPING GRINDING	1,466	2,108	276	$1,140	$313,322
COATING/ETCHING	226	645	11	$3,070	$35,182
COATING/SPRAYING/FINISHING/LAYUP	298	1,512	92	$3,070	$281,257
COLD ROLLING MILL	92	183	0	$1,820	$0
CRUSHING/GRINDING/CALCINING	900	2,033	1,168	$4,740	$5,538,889
CUTTING/SAWING/PLANNING	1,349	3,618	1,269	$180	$228,376
DECORATION	133	810	11	$710	$8,136
DRILLING/BORING	18	73	73	$2,200	$159,501
DRILLING/CUTTING/FLAME-JET LANCING	1,466	2,474	0	$1,140	$0
DRYING/BAKING	341	636	54	$4,740	$257 739
EXTRUSION	183	550	0	$1,140	$0
FIBER FORMING	30	138	0	$90	$0
FINISHING	110	1,924	1,924	$1,820	$3,501,522
GLAZE APPLICATION	187	276	91	$3,070	$278,220
GLOSS FIRING	164	200	36	$4,740	$171,826
IMPREGNATION	11	115	0	$1,140	$0
INJECTION MOLDING	11	138	0	$1,140	$0
LOADING/OFFLOADING/RECEIVING/HANDLING	241	558	23	$1,120	$25,670
MACHINING/GRINDING/WELDING/BRAZING	110	1,134	0	$1,140	$0
MATERIALS MANUFACTURE/FABRICATION	18	0	0	$1,140	$0
MELTING	249	1,256	36	$3,890	$141,013
METAL MELTING/POURING/CASTING	11	92	0	$1,820	$0
METAL PLATING	30	229	0	$710	$0
MOLDMAKING	11	57	0	$2,520	$0
OTHER	110	182	0	$90	$0
PACKAGING/BAGGING	352	958	23	$1,120	$25,670
PACKAGING/REPACKAGING	11	23	0	$1,820	$0
POLISHING (SURFACE)/GRINDING	1,466	1,833	0	$1,140	$0
SANDING/POLISHING/GRINDING	18	0	0	$2,200	$0
SCREEN PRINTING	238	695	0	$1,380	$0
SCREEN STENCIL PLATEMAKING	18	36	0	$1,380	$0
SIZING	157	673	127	$4,740	$601,392
SLIP HOUSE (BLENDING)	341	341	0	$1,140	$0
STAMPING/SHAPING/MOLDING/PRESSING	82	749	138	$2,900	$398,807
VULCANIZATION/CURING	11	57	0	$90	$0
WELDING/SOLDERING	201	769	403	$1,140	$459,089
ZSUBTOTAL	18,843	57,897	10,863		$22,112,897
ZZMAINTENANCE	15,920	8,620	344	$520	$344,949
ZZTOTAL	34,763	66,516	11,207		$22,457,846

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SIC 33 = PRIMARY METAL MANUFACTURING

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
ABRASIVE BLASTING	1504	4674	660	$7,200	$4,035,395
ACID WASHING	78	103	0	$710	$0
ANNEALING/QUENCH/TEMPER	1381	7106	1315	$710	$933,430
BLENDING/MIXING/FORMULATING	12	12	0	$3,070	$0
CALCINING KILN	10	10	10	$4,740	$46,198
CLEANING	18	18	0	$710	$0
COATING & DRAWING	287	389	0	$7,200	$0
COATING/SPRAYING/FINISHING/LAYUP	31	283	0	$3,070	$0
COKE MANUFACTURE	22	35	0	$0	$0
COLD ROLLING MILL	403	2620	391	$1,820	$712,061
COLORING/DYEING	18	37	0	$0	$0
CORE MAKING	945	3466	945	$2,520	$2,380,446
COSTING/PAINTING	192	628	0	$3,070	$0
DEGREASING	320	453	41	$710	$28,828
DEMAGGING	28	48	24	$1,140	$27,164
ELECTROD PRODUCTION	37	100	50	$1,820	$90,555
ELECTROPLATE/ELECTRCAL DISCHRGE MACHING	109	6737	0	$710	$0
EXTRUSION	10	39	0	$1,140	$0
EXTRUSION COATING	236	1325	297	$7,200	$2,139,242
FINISHING	748	5746	656	$1,820	$1,193,680
FORGING PRESS	10	0	0	$1,820	$0
GLUEING/HOT PRESSING	94	188	0	$3,070	$0
HOT DIP GALVANIZING	12	12	0	$710	$0
HOT METAL WORKING	28	112	19	$7,200	$140,348
HOT SHAPING	342	1003	109	$2,520	$274,999
IMPREGNATION	180	728	150	$1,140	$171,537
MACHINING/GRINDING/WELDING/BRAZING	508	1479	305	$1,140	$347,508
MAINTENANCE ACTIVITIES	12	25	0	$7,200	$0
MELTING	77	192	0	$3,890	$0
METAL CASTING	1299	5649	686	$2,520	$1,727,898
METAL MELTING/POURING/CASTING	3522	14024	2767	$1,820	$5,036,591
METAL WORKING (ROLLING, MILLING, SHAPING	74	145	0	$1,140	$0
MOLDMAKING	656	2170	145	$2,520	$365 710
ORE HANDLING	110	19631	19589	$1,820	$35,651,199
OTHER	89	387	0	$4,740	$0
PC BOARDS SOLDERING	25	62	50	$1,140	$56,721
PICKELING	357	749	5	$710	$3,743

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

POLISHING (SURFACE)/GRINDING	1532	4834	1668	$1,140	$1,901,789
POURING	608	3298	131	$1,820	$239,227
PRESSING	38	1382	0	$3,070	$0
RAW MATERIALS PREPARATION	115	230	0	$2,520	$0
SAMPLING	12	149	0	$1,120	$0
SAND RECLAMATION	115	152	0	$2,520	$0
SANDING	47	47	0	$1,140	$0
SEPARATION	28	139	203	$1,120	$226 914
SHAKEOUT	832	3337	468	$2,520	$1,179,773
SINTERING	166	412	85	$21,900	$1,865,656
SOLDERING	47	94	0	$1,140	$0
STAMPING/SHAPING/MOLDING/PRESSING	102	626	0	$2,900	$0
STRIP ANNEALING	94	939	469	$1,820	$854,157
TORCH CUTTING	131	734	56	$2,520	$140,453
WELDING/BRAZING	65	214	141	$1,140	$160,506
WELDING/SOLDERING	285	888	237	$1,140	$270,505
WIRE DRAWING	177	1917	289	$1,140	$329,715
ZSUBTOTAL	18083	99477	31862		$62,531,948
ZZMAINTENANCE	64224	54448	1784	$520	$8,425,657
ZZTOTAL	26111	106283	33646		$70,957,605

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SIC 34 = FABRICATED METAL PRODUCTS MANUFACTURING

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
ABRASIVE BLASTING	3523	3957	138	$7,200	$993,370
ACID WASHING	1175	2679	0	$710	$0
ANNEALING/QUENCH/TEMPER	609	659	0	$710	$0
ASSEMBLY	1189	7018	618	$1,140	$704,382
BISCUIT FIRING	24	0	0	$4,740	$0
BLENDING/MIXING/FORMULATING	178	1069	0	$2,200	$0
COATING & DRAWING	177	148	0	$7,200	$0
COATING/SPRAYING/FINISHING/LAYUP	1119	940	0	$3,070	$0
COLD ROLLING MILL	144	2290	0	$1,820	$0
COSTING/PAINTING	12164	22825	1411	$3,070	$4,332,852
CUTTING/SAWING/PLANNING	422	2139	0	$180	$0
DEBURRING	129	271	247	$7,200	$1,779,493
DEGREASING	6315	15464	55	$710	$39,384
DRILLING/BORING	406	406	0	$2,200	$0
DRILLING/CUTTING/FLAME-JET LANCING	24	97	0	$1,140	$0
ELECTROD PRODUCTION	89	89	0	$1,820	$0
ELECTROPLATE/ELECTRCAL DISCHRGE MACHING	3103	18617	1505	$710	$1,068,543
ENGRAVING/ETCHING	406	812	0	$710	$0
EXTRUSION COATING	62	510	0	$7,200	$0
FINISHING	501	958	0	$1,820	$0
FORGING PRESS	15	463	463	$1,820	$843,405
HOT DIP GALVANIZING	466	5871	93	$710	$65,804
HOT METAL WORKING	89	891	0	$7,200	$0
HOT SHAPING	240	926	0	$2,520	$0
LITHOGRAPHIC PRINTING	15	124	124	$1,380	$170,534
LOADING/OFFLOADING/RECEIVING/HANDLING	89	356	0	$1,120	$0
MACHINING/GRINDING/WELDING/BRAZING	5051	29045	1219	$1,140	$1,389,146
MAKING OF DENTAL APPLIANCES	15	463	0	$1,140	$0
METAL MELTING/POURING/CASTING	15	46	0	$1,820	$0
METAL WORKING (ROLLING, MILLING, SHAPING	1363	6551	3656	$1,140	$4,167,440
MOLDMAKING	89	267	0	$2,520	$0
OTHER	602	1691	0	$4,740	$0
PACKAGING/BAGGING	178	624	0	$1,120	$0
PICKELING	64	112	0	$710	$0
POLISHING (SURFACE)/GRINDING	8600	23188	2192	$1,140	$2,498,617
PRESSING	2294	15729	0	$3,070	$0
PROCESS INSP/SUPERVISION/QUAL CONTROL	446	1280	121	$1,120	$135,841

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

REACTION/FERMENTATION	15	46	0	$1,120	$0
RECEIVING/SHIPPING	89	356	0	$1,120	$0
SANDING/POLISHING/GRINDING	89	89	0	$2,200	$0
SOLDERING	24	97	0	$1,140	$0
STAMPING/SHAPING/MOLDING/PRESSING	1343	15761	1236	$2,900	$3,683,702
TORCH CUTTING	89	891	0	$2,520	$0
WATER TREATMENT	24	24	24	$14,000	$339,603
WELDING/SOLDERING	11788	46728	13256	$1,140	$15,111,924
WIRE DRAWING	247	2605	0	$1,140	$0
ZSUBTOTAL	64903	235074	26358		$37,224,040
ZZMAINTENANCE	37315	13302	1698	$520	$2,195,645
ZZTOTAL	102218	248377	28056		$39,419,685

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SICs 35,36,38,39 = MACHINERY, INSTRUMENTS, AND MISC. MANUFACTURING

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
ABRASIVE BLASTING	12863	13588	407	$7,200	$2,928,488
ACID WASHING	2809	2809	0	$710	$0
ASSEMBLY	5841	64324	5054	$1,140	$5,762,058
BLENDING/MIXING/FORMULATING	1573	11016	731	$3,070	$2,243,008
BRISTLE/FIBER CLEANING/RECEIVING	49	49	0	$1,140	$0
CLEANING	2015	3071	0	$710	$0
COATING/SPRAYING/FINISHING/LAYUP	7478	19943	1162	$3,070	$3,567,133
COIL PRODUCTION	49	49	0	$2,520	$0
CORE MAKING	203	1627	0	$2,520	$0
COSTING/PAINTING	20560	45995	2052	$3,070	$6,300,780
CRUSHING/GRINDING/CALCINING	314	1905	0	$4,740	$0
CUTTING/SAWING/PLANNING	6902	25579	1017	$180	$183,030
DEGREASING	17198	28737	1927	$710	$1,367,926
DRILLING/BORING	2886	26868	2034	$2,200	$4,474,080
DRYING/BAKING	49	98	0	$4,740	$0
ELECTROPLATE/ELECTRCAL DISCHRGE MACHING	877	877	0	$710	$0
EPOXY COATING	456	407	0	$3,070	$0
EXTRUSION COATING	203	813	0	$7,200	$0
FILM & PRINT PAPER MAKING	49	196	0	$1,240	$0
FINISHING	81	101	0	$1,820	$0
FOAM PROCESSING	61	61	61	$1,140	$69,828
GLASSMAKING	203	203	0	$90	$0
GLUEING/HOT PRESSING	6672	12260	4871	$3,070	$14,953,587
HOT SHAPING	27	27	0	$2,520	$0
INJECTION MOLDING	1763	9317	0	$1,140	$0
LOADING/OFFLOADING/RECEIVING/HANDLING	1272	2014	0	$1,120	$0
MACHINING/GRINDING/WELDING/BRAZING	6176	65357	959	$1,140	$1,092,764
MATERIALS MANUFACTURE/FABRICATION	3090	29519	6799	$1,140	$7,750,921
METAL MELTING/POURING/CASTING	1321	3862	1419	$1,820	$2,582,353
METAL WORKING (ROLLING, MILLING, SHAPING	2876	6710	0	$1,140	$0
MOLDMAKING	375	3708	2361	$2,520	$5,949,315
MONO OR LINOTYPE SETTING	110	110	0	$1,380	$0
ORE HANDLING	203	203	0	$1,820	$0
OTHER	1301	15739	0	$90	$0
PACKAGING/BAGGING	1740	5971	203	$1,120	$227,771
PAINTING/COATING	49	0	0	$3,070	$0
PC BOARDS - ETCHING	486	645	110	$2,520	$277,652

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

PC BOARDS - SOLDERING	1370	1494	0	$1,140	$0
PHOTOFINISHING	252	960	0	$1,240	$0
PICKELING	301	554	0	$710	$0
PLATE PROCESS	265	1446	0	$90	$0
POLISHING (SURFACE)/GRINDING	31073	108851	1728	$1,140	$1,969,642
POTTING	1136	2880	0	$2,520	$0
PRESSING	8518	36907	3182	$3,070	$9,770,026
PRINTING	380	1268	0	$1,380	$0
PROCESS INSP/SUPERVISION/QUAL CONTROL	1463	6382	0	$1,120	$0
REACTION/FERMENTATION	456	2132	0	$1,120	$0
RECEIVING/SHIPPING	61	61	0	$1,120	$0
REFRIGERANT CHARGING	203	0	0	$2,520	$0
SAMPLING	49	489	0	$1,120	$0
SANDING	3829	4820	0	$1,140	$0
SANDING/POLISHING/GRINDING	1475	5134	0	$2,200	$0
SCREEN PRINTING	314	362	61	$1,380	$84,528
SEMICONDUCTOR PHOTORESIST	282	282	0	$2,520	$0
SEMICONDUCTOR PHOTORESIST STRIPPING	171	233	0	$2,520	$0
SEMICONDUCTOR WAFER CLEANING	343	1028	0	$2,520	$0
SEMICONDUCTOR-CHEMICAL ETCHENTS	171	233	0	$2,520	$0
SEMICONDUCTOR-DIFFUSION & ION IMPLANT	49	7339	0	$2,520	$0
SEPARATION	49	49	0	$1,120	$0
SOLDERING	22901	96171	9730	$1,140	$11,092,750
STAMPING/SHAPING/MOLDING/PRESSING	3238	17434	1607	$2,900	$4,660,122
STERILIZATION	1480	957	0	$710	$0
WELDING/BRAZING	1153	2316	0	$1,140	$0
WELDING/SOLDERING	13988	62094	2024	$1,140	$2,307,300
ZSUBTOTAL	205154	765639	49499		$89,615,064
ZZMAINTENANCE	123365	25313	3338	$520	$1,735,608
ZZTOTAL	328519	790953	52837		$91,350,672

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SICs 40,45,47 = TRANSPORTATION

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
ASSEMBLY	7	372	0	$1,140	$0
CLEANING	213	213	0	$710	$0
DEICING	15	30		$2,410	$0
ENGINE FUELING/OPERATION	272	4,361	7	$560	$4,161
HANDLING SPILLS/LEAKS	58	58	0	$0	$0
LOADING/OFFLOADING/RECEIVING/HANDLING	471	39,011	7	$1,120	$8,322
MACHINING/GRINDING/WELDING/BRAZING	7	0	0	$1,140	$0
MAINTENANCE ACTIVITIES	352	401	0	$0	$0
PAINTING/COATING	15	7	0	$3,070	$0
RECEIVING/SHIPPING	242	2,586	2,558	$1,120	$2,864,591
REFUELING	22	171	22	$560	$12,483
SPECIAL CARE OF LADING SERVICES	36	29	0	$560	$0
WELDING/BRAZING	242	455	0	$1,140	$0
ZSUBTOTAL	1,953	47,694	2,595		$2,889,559
ZZMAINTENANCE	42,025	1,403	132	$520	$5,723,634
ZZTOTAL	43,979	49,097	2,727		$8,613,193

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SIC 49 = ELECTRIC, GAS, AND SANITARY SERVICE UTILITIES

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
ASSEMBLY	5	3	0	$1,140	$0
BOILER/FURNACE PREP/OPERATION	630	1,240	368	$710	$262,310
BREAKING UP WASTE	68	136	0	$14,000	$0
CHEMICAL PREPARATION/APPLICATION	72	72	0	$14,000	$0
CLEANING	14	0	0	$710	$0
COLLECTION/TRANSPORT	524	10,414	6,111	$520	$8,403,203
CONDENSATE COLLECTION	151	917	584	$520	$1,027,855
DETOXIFICATION	5	15	3	$2,410	$6,034
ENGINE FUELING/OPERATION	1,022	2,124	130	$560	$72,676
HANDLING SPILLS/LEAKS	754	2,906	0	$0	$0
INCINERATION	14	55	0	$14,000	$0
LAB PROCEDURES: TISSUE STAINING & FIXING	3	3	0	$0	$0
LOADING/OFFLOADING/RECEIVING/HANDLING	14	28	0	$1,120	$0
MAINTENANCE ACTIVITIES	4,781	18,397	0	$0	$0
ODORANT ADDITION	384	1,334	1,170	$0	$2,059,194
OTHER	167	246	0	$1,120	$0
PAINTING/COATING	88	90	0	$3,070	$0
PRINTING	3	15	0	$1,380	$0
PROCESS INSP/SUPERVISION/QUAL CONTROL	99	498	68	$1,120	$75,713
PUMP STATION FUELING/ENGINE FUMES	65	65	65	$560	$36,338
RECEIVING/SHIPPING	3	5	0	$1,120	$0
RECYCLING/RECLAMATION	44	47	14	$21,900	$310,010
SAMPLING	79	230	0	$1,120	$0
SAMPLING OF PIPE LINES	295	1,474	1,474	$1,120	$1,651,346
USE OF CHEMICAL ADDITIVES	1,145	4,382	0	$1,120	$0
USE OF DISINFECTANTS & SOLVENTS	228	228	0	$2,410	$0
VENTING	14	1,380	0	$0	$0
WATER PURIFICATION	540	1,251	0	$14,000	$0
WATER TREATMENT	655	2,167	92	$14,000	$1,281,486
WELDING	111	212	5	$1,140	$5,708
WELDING/BRAZING	162	239	0	$1,140	$0
WOOD PRESERVATION	65	0	0	$3,070	$0
ZSUBTOTAL	12,200	50,171	10,083		$15,191,877
ZZMAINTENANCE	15 812	11,056	1,345	$520	$22,817,441
ZZTOTAL	28 012	61,226	11,428		$38,009,318

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SIC 50,51 = WHOLESALE TRADE

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
BALING/COMPACTING	217	295	60	$14,000	$838,812
BLENDING/MIXING/FORMULATING	2,264	4,048	954	$4,740	$1,844,592
BREAKING UP WASTE	38	109	0	$14,000	$0
CHEMICAL PREPARATION/APPLICATION	684	1,654	182	$14,000	$321,174
CHIPPING GRINDING	8	60	0	$1,140	$0
COATING/SPRAYING/FINISHING/LAYUP	457	1,066	913	$3,070	$2,804,276
COLLECTING	518	4,687	0	$520	$0
CRUSHING/GRINDING/CALCINING	30	30	0	$4,740	$0
DRILLING/CUTTING/FLAME-JET LANCING	4	11	0	$1,140	$0
DRYING/BAKING	34	131	0	$4,740	$0
MAINTENANCE ACTIVITIES	4	15	0	$520	$0
METAL MELTING/POURING/CASTING	4	11	0	$1,820	$0
OTHER	67	116	0	$1,140	$0
PACKAGING/REPACKAGING	3,144	5,071	1,752	$1,760	$3,084,385
PESTICIDE PREPARATION/APPLICATION	30	0	0	$520	$0
POLISHING (SURFACE)/GRINDING	30	30	0	$1,140	$0
PROCESS INSP/SUPERVISION/QUAL CONTROL	4	95	0	$1,120	$0
REACTION/FERMENTATION	8	15	0	$1,120	$0
RECEIVING/SHIPPING	8,725	13,696	6,864	$1,120	$7,470,731
RECYCLING/RECLAMATION	4	4	4	$21,900	$82,794
SAMPLING	152	152	0	$1,120	$0
SANDING/POLISHING/GRINDING	152	304	0	$2,200	$0
SORTING	794	1,472	30	$14,000	$419,406
WASHING	152	152	0	$0	$0
WELDING	4	11	0	$1,140	$0
ZSUBTOTAL	17,626	33,237	10,759		$16,866,172
ZZMAINTENANCE	39,371	800	19	$520	$344,858
ZZTOTAL	56,897	34,037	10,778		$17,211,030

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SIC 55,75 = AUTO DEALERS AND REPAIR

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
ASSEMBLY	9	66	0	$1,140	$0
CLEANING	83012	342202	2458	$710	$1,745,425
COATING/SPRAYING/FINISHING/LAYUP	3	0	0	$3,070	$0
CONFINED SPACE EXPOSURE TO EXHAUST FUME	57023	224767	23623	$80	$1,889,810
COSTING/PAINTING	23623	0	0	$3,070	$0
FLOAT PROCESS	3	0	0	$90	$0
MACHINING/GRINDING/WELDING/BRAZING	3	47	0	$1,140	$0
MAINTENANCE ACTIVITIES	49086	155359	0	$80	$0
MATERIALS MANUFACTURE/FABRICATION	3	3	0	$1,140	$0
OTHER	11811	0	0	$90	$0
OTHER SOLVENT USE	14592	50937	0	$710	$0
PAINT STRIPPING	14795	41509	96	$710	$68,069
PAINTING/COATING	33997	127958	0	$3,070	$0
PUMP STATION FUELING/ENGINE FUMES	23623	94491	0	$560	$0
REACTION/FERMENTATION	11811	0	0	$2,900	$0
RECEIVING/SHIPPING	70	211	0	$1,120	$0
REFRIGERANT CHARGING	11811	0	0	$3,070	$0
STAMPING/SHAPING/MOLDING/PRESSING	3	22	0	$2,900	$0
TORCH CUTTING	11811	0	0	$2,520	$0
WELDING	45594	164157	7081	$1,140	$8,072,535
ZSUBTOTAL	392685	1201729	33258		$11,775,839
ZZMAINTENANCE	257267	76073	3400	$520	$7,918,216
ZZTOTAL	649952	1277801	36658		$19,694,055

TABLE G-4 (CONT.)
DESCRIPTIVE INFORMATION ON PROCESSES AND RELATED COSTS

SIC 76 - MISCELLANEOUS REPAIR SERVICE

PROCESS	FREQUENCY OF PROCESS	TOTAL WORK STATIONS	COSTED WORK STATIONS	UNIT COST	TOTAL COST FOR PROCESS
CLEANING	2	4	0	$710	$0
GLUEING/HOT PRESSING	3,982	4,837	16	$3,070	$48,168
OTHER	13	13	0	$2,200	$0
PAINT STRIPPING	2,348	2,820	0	$710	$0
PAINTING/COATING	2,381	2,492	0	$3,070	$0
SANDING/POLISHING/GRINDING	3,876	6,151	0	$2,200	$0
WELDING/BRAZING	4,052	7,830	2,120	$1,140	$2,417,367
ZSUBTOTAL	16,653	24,145	2,836		$2,465,536
ZZMAINTENANCE	15,095	4,260	509	$520	$344,257
ZZTOTAL	31,747	28,405	3,345		$2,809,793

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References

1. Dun and Bradstreet, Inc., 1985 Count of Establishments. (Database)

H. Economic Impact, Regulatory Flexibility Analysis and Environmental Impact Assessment

Economic Impact

The economic impacts discussed in this chapter have been estimated following an analysis of data collected through a nationwide sample survey of about 5,700 establishments. Two alternative polar assumptions were used in this analysis.

- *Perfectly Elastic Demand or Zero Cost-Passthrough:* All compliance costs are absorbed by the firm in the form of reduced profits. This assumption is the "worst case" scenario, where the maximum reduction in pre-tax profits to the firm (and industry) results.
- *Perfectly Inelastic Demand or Total Cost-Passthrough:* All compliance costs are passed on to the consumer sector in the form of higher prices. From the perspective of the firm, this is the "best case" scenario. The resulting price increase would be the maximum theoretically possible.

Two points should be noted. First, for the majority of industry sectors, neither assumed market structure would be accurate. In practice, the impacts will almost always produce a price increase smaller than the inelastic demand projection and a reduction in profits smaller than that predicted under perfectly elastic demand conditions. Second, increased firm productivity would mitigate any adverse economic effects of the final standard. Productivity effects would be related to reduced worker illness, absence and turnover. In addition, knowledge of improved workplace health conditions could result in higher workforce morale and productivity. The firm would enjoy lower employee training costs (due to the reduced turnover rate) and lower medical benefit and worker compensation claims. Overall productivity increases would be realized by firms that use a relatively fixed-factor production process (i.e., low elasticities of substitution between labor and other factors of production). It is difficult to estimate the magnitude of these productivity and cost reducing effects. Any estimated economic costs of compliance would have to be adjusted downward to reflect these effects. Since data were not available to make any offset estimates, the economic effects of the standard identified in this chapter are overstated.

In addition, OSHA is allowing a phase-in period, up to five years, for engineering controls. Respirator use will be allowed during this period. Economic costs presented in this chapter will be overstated to the extent that capital expenditures are delayed during the phase-in period.

For this analysis, OSHA used a percentage reduction in profits approach to obtain estimates of the short-run economic impacts under the assumption of perfect demand elasticity. These estimates were obtained by using the following formula:

$$\text{Percentage Reduction} = \frac{\text{New Profits} - \text{Old Profits}}{\text{Old Profits}}$$

where:
New Profits = (Old Pre-tax Profits − Compliance Costs) × (1 − Old Profits = (Return on Sales)*(Total Sales).

These calculations were performed at the two-digit SIC level for firms in large and small size-class stratifications (above and below 20 employees). The data used to obtain these estimates was based on Dun and Bradstreet company files [1;2].

The potential impact on prices was used to estimate the market consequences under the second assumption of inelastic demand. Total sales values for 1985 were used, the year for which the compliance costs were estimated. (Total sales represent the totality of production that leaves the establishment, whether it is sold to customers or sent to a parent company in a captive transaction. For industries in the service and trade sectors, total sales data were also used. The rate of return percentage for each industry sector corrected and transformed gross sales data into more accurate and relevant industry profit estimates.)

For a given firm-size class, the potential price increase was estimated by dividing the total estimated compliance costs for a firm by the sales of that firm. These estimated price effects were then compared to recent industry price series. The intent of this comparison was to evaluate the impact of the compliance cost-generated price increase in light of recent industry price increase experience.

In this scenario, the potential for international trade implications of the standard was explored. It is anticipated that any international trade effects will not be significant given the small value of domestically produced goods and services which are exported (about seven percent of GDP). Also, the U.S. dollar has recently experienced a sharp decline in value relative to the yen and European Currencies. Between February 1985 and December 1987, the trade-weighted value of the U.S. dollar fell 46 percent [3]. This depreciation will likely overwhelm any potential adverse international economic effect of the standard.

In Tables H–1 and H–2, the estimated domestic economic impacts are reported for the two polar methodologies. To derive the percentage change in profits and the costs as a percent of sales, industry sales and rate of return (R.o.R) on sales data were obtained from Dun and Bradstreet. The total sales data are the best estimates for industry sectors potentially impacted by the rule.

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TABLE H-1

ECONOMIC EFFECTS: NO-COST PASSTHROUGH SCENARIO[1]

SIC	Industry	Annual Costs[2] ($ millions)	Total Sales[3] ($ millions)	R.o.R. on Sales (%)[4]	Pre-Reg Profits ($ m)	Post-Reg Profits ($ m)	% Change in Profits
20	FOOD PROD.	33.49	353,780.38	1.9	8,008.04	7,986.29	- 0.2715
21	TOBACCO	0.02	74,030.13	5.3	3,923.60	3,923.59	- 0.0003
22	TEXT. MILL	29.48	60,735.22	2.7	1,765.42	1,747.59	- 1.0100
23	APPAREL PROD.	31.74	74,474.65	2.8	1,813.22	1,793.56	- 1.0845
24	LUMBER & WOOD	56.72	57,994.48	3.9	1,974.51	1,931.92	- 2.1574
25	FURNITURE	21.08	37,648.27	3.5	1,411.02	1,398.82	- 0.8645
26	PAPER PROD.	31.00	103,694.14	3.7	3,778.20	3,761.12	- 0.4519
27	PRINTING & PUB.	33.75	134,830.21	4.8	6,471.85	6,444.77	- 0.4185
28	CHEMICAL PROD.	35.45	272,759.67	3.7	11,738.80	11,717.79	- 0.1790
29	PETRO. REFINING	23.69	196,400.57	2.7	4,964.85	4,952.04	- 0.2579
30	RUBBER & PLASTICS	111.09	86,538.58	4.3	3,423.75	3,343.76	2.3361
31	LEATHER PROD.	2.41	15,449.56	2.6	401.69	400.03	- 0.4127
32	STONE & CLAY	22.46	46,094.04	4.1	1,954.99	1,940.97	- 0.7170
33	PRIMARY METALS	70.96	112,564.26	3.3	3,714.62	3,674.83	- 1.0712
34	FAB. METALS	39.42	150,146.41	4.0	6,005.86	5,981.33	- 0.4084
35	MACHINERY	45.21	345,144.89	5.1	17,602.39	17,573.57	- 0.1637
36	ELEC. MACH.	20.67	245,982.70	5.0	12,299.14	12,286.86	- 0.0998
37	TRANS. EQUIP.	49.79	365,427.20	3.9	14,520.25	14,485.24	- 0.2411
38	INSTRUMENTS	9.63	83,359.57	4.9	3,373.26	3,367.32	- 0.1763
39	MISC. MANUF.	15.84	41,870.30	4.4	1,788.56	1,778.14	- 0.5825
40	R.R. TRANS.	1.08	43,869.14	10.0	3,969.62	3,969.04	- 0.0147
45	AIR TRANS.	3.74	109,538.08	3.6	3,251.40	3,249.38	- 0.0621
47	TRANS. SERVICES	3.79	12,254.96	2.7	582.18	580.13	- 0.3515
49	ELEC., GAS & SAN.	38.01	300,254.83	7.0	21,017.84	20,994.71	- 0.1100
50	WHOLESALE TRADE[5]	3.00	13,853.52	2.0	277.07	274.56	- 0.9048
51	WHOLESALE, NON-DUR	14.22	113,848.20	1.5	1,726.26	1,718.59	- 0.4447
55	AUTO DEALERS	13.55	341,574.50	1.9	6,489.92	6,480.69	- 0.1422
72	PERSONAL SERV.	10.87	24,270.74	7.3	1,771.76	1,763.60	- 0.4606
73	BUSINESS SERV.	2.42	22,165.94	6.6	1,462.95	1,460.94	- 0.1375
75	AUTO REPAIR	6.14	45,750.92	5.1	2,492.19	2,488.29	- 0.1563
76	MISC. REPAIR SERV.	2.81	2,665.52	5.5	146.60	144.36	- 1.5298
80	HEALTH SERVICES	4.44	170,234.25	4.5	7,807.72	7,804.54	- 0.0406

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

Notes:
1. All values in 1985 dollars.
2. Reproduced from Table G-1.
3. Dun and Bradstreet, Dun's Marketing Identifiers (DMI) Database.
4. Rate of Return on Sales, Dun and Bradstreet, Industry Norms Database.
5. Consists of SIC 5093 (scrap and waste materials) only.

TABLE H-2

ECONOMIC EFFECTS: TOTAL-COST PASSTHROUGH

SIC	Industry	Annual Costs ($ millions)	Total Sales ($ millions)	Costs as a Percent of Sales
20	FOOD PROD.	33.49	353,780.38	0.0095
21	TOBACCO	0.02	74,030.13	0.0000
22	TEXT. MILL	29.48	60,735.22	0.0485
23	APPAREL PROD.	31.74	74,474.65	0.0426
24	LUMBER & WOOD	56.63	57,994.48	0.0978
25	FURNITURE	26.28	37,648.28	0.0560
26	PAPER PROD.	33.00	103,694.14	0.0299
27	PRINTING & PUB	34.39	134,830.21	0.0250
28	CHEMICAL PROD.	38.87	272,759.67	0.0130
29	PETRO. REFINING	23.91	196,400.57	0.0121
30	RUBBER & PLASTICS	121.93	86,538.58	0.1284
31	LEATHER PRODUCTS	2.66	15,449.56	0.0156
32	STONE & CLAY	25.83	46,094.04	0.0487
33	PRIM. METALS	78.24	112,564.26	0.0630
34	FAB. METALS	53.51	150,146.41	0.0263
35	MACHINERY	50.00	345,144.89	0.0131
36	ELEC. MACH.	23.30	245,982.70	0.0084
37	TRANS. EQUIP.	49.79	365,427.20	0.0136
38	INSTRUMENTS	10.75	83,359.57	0.0116
39	MISC. MANUF.	17.29	41,870.30	0.0378
40	R.R. TRANS.	1.09	43,869.14	0.0025
45	AIR TRANS.	3.76	109,538.08	0.0034
47	TRANS. SERVICES	3.81	12,254.96	0.0309
49	ELEC., GAS & SAN.	37.83	300,254.83	0.0127
50	WHOLESALE TRADE[1]	3.13	13,853.52	0.0216
51	WHOLESALE, NON-DUR.	14.80	113,848.20	0.0125
55	AUTO DEALERS	22.72	341,574.50	0.0040
72	PERSONAL SERVICES	10.87	24,270.74	0.0448
73	BUSINESS SERVICES	2.42	22,165.94	0.0109
75	AUTO REPAIRS	10.25	45,750.92	0.0134
76	MISC. REPAIR SERV.	4.86	2,665.52	0.1054
80	HEALTH SERVICES	4.44	170,234.25	0.0026

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

Notes: 1. Consists of SIC 5093 (scrap and waste materials) only.

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Dun and Bradstreet provided OSHA with this information. The R.o.R. on sales were obtained from summary statistics found in the Dun and Bradstreet Industry Norms Database.

Industry Effects. The estimated economic impact of the rule for firms potentially affected is summarized in Table H–1. These estimates represent the maximum industry impact within a market scenario where none of the costs can be passed onto consumers, and there is no productivity offset to costs.

Data in Table H–1 indicate that the rule will not have a significant impact on profits in most industry sectors. The estimated average change in profits is less than one percent; this amount of profit reduction should not represent a significant economic burden.

The most adversely affected industry sector is SIC 30, Rubber and Plastics, with an estimated 2.3 percent reduction in profits. The only other industry with an impact greater than 2 percent is SIC 24, Lumber and Wood (2.2 percent). However, even in the worst case, OSHA believes the standard is economically feasible. In reality, the reduction in profits will be less because part of the costs will be passed on to consumers, and because profitability in these industries since 1985 (the year for which the cost impact was estimated) has improved [9].

Consumer effects were estimated using a "full cost passthrough" scenario. As demonstrated by the estimates summarized in Table H–2, the impacts on market prices will not be significant. No price increase would exceed one-half of one percent. Changes of this magnitude are within general price movements recorded by producer price and other price indices.

During the public hearing and comment period OSHA received comments concerning the economic impact of the standard. Comments were primarily concerned with the following industries and substances.

SIC 20: The meat packing and processing industries (SICs 2011 and 2013) expressed concern over the impact a carbon disulfide PEL of 1 ppm would have on the cellulosic casings industry (SIC 3079). Industry representatives in all three of these SICs believe that if OSHA sets a PEL which is economically difficult to meet, domestic production of the meat casings will dramatically decrease [Ex. 3–659; 3–756; 3–757; 3–898]*. Although foreign markets already supply casings to the U.S. meat industry, this industry believes that foreign markets will not be able to absorb a dramatic change in demand for casings [Ex. 3–898; 3–897; 3–756].

OSHA has decided to increase the PEL for carbon disulfide from the proposed limit of 1 ppm to a standard of 4 ppm. Compliance with a 4 ppm PEL will be easier for casing manufacturers. OSHA believes that domestic production will be unaffected by a carbon disulfide PEL of 4 ppm, particularly since respirators may be used in difficult to control operations.

SIC 23: See SIC 72.

SICs 24 and 25: The wood and furniture industries argued that to comply with the proposed PEL of 5 mg/m^3 for softwood and 1 mg/m^3 for hardwood, the majority of establishments will have to engineer to 1 mg/m^3 for all wood dust since most establishments process both soft and hard wood. Most commenters concluded that they, as well as most members of their industry, would go out of business if they had to control wood dust exposures to 1 mg/m^3.

Several industry organizations submitted comments and information concerning the potential economic impact of these PELs on their industry [Ex. 3–626; 3–627; 3–748; 3–899; 3–951; 3–627]. The U.S. Small Business Administration (SBA) believes "it is likely that the wood dust PELs of 1 mg/m^3 for hard wood dust and 5 mg/m^3 for soft wood dust are not economically feasible for small facilities with fewer than 20 employees," and that a "5 mg/m^3 standard . . . may be more economically feasible for affected industries" [Ex. 3–951]. This recommendation was supported by many comments from industry [Ex. 3–627; 3–768; 3–750; 3–917].

OSHA's final rule establishes a 5 mg/m^3 PEL for all wood dust except Western red cedar, for which a 2.5 mg/m^3 limit is established. OSHA believes that the 2.5 mg/m^3 standard for this allergenic wood is feasible since 90% of the firms using Western red cedar are located in the state of Washington which has already adopted a 2.5 mg/m^3 PEL.

OSHA's estimated costs for compliance with the final PELs are significantly lower than those corresponding to the proposed PELs of 1 and 5 mg/m^3. The economic impact of the standard on these wood processing industries reflects this decrease in cost. These reduced costs amount to 2% of profits. (The actual effects on profits will be even less since some costs will be passed on to consumers.)

Industries in SIC 24 had strong domestic and foreign markets in 1987. Price increases averaged 5% over 1986. As a result of increasing prices, demand, and output, profits increased for firms in this industry. The 1988 U.S. Industrial Outlook predicted a 41% growth (in constant dollars) of shipments of wood products in 1988. These recent developments indicate that the economic impact of complying with a 5 mg/m^3 PEL for wood dust will be less than the impact presented in Table H–1 [9].

In addition, furniture product shipments in SIC 251 increased 4.5% in 1987 (constant dollars) [9]. This will make it easier for firms in this SIC to absorb the costs imposed by this rule, and the economic impact will actually be less than that estimated by OSHA.

SIC 30: Establishments in SIC 308 submitted comments and information concerning OSHA's proposed standard of 50 ppm for styrene. The Styrene Information Research Council (SIRC) submitted cost and impact estimates of the proposed PEL on SIC 30 and SIC 37 [Ex. 3–742]. OSHA has examined this information and, based on reasons outlined in the cost chapter of this document (Chpt. G), has determined that the survey performed for this rulemaking provides the most accurate and up-to-date information on employee exposures and cost of controls. The costs OSHA estimates are less than .44% of profits and are economically feasible. In addition, the value of shipments of rubber and plastics products have been increasing since 1985 (for which costs and impacts were estimated), and growth in shipments is projected to be 2% in 1988 [9]. Increased profitability should offset the economic consequences of compliance.

Comments were received from the manufacturers of cellulosic casings (SIC 3079) concerning OSHA's proposed PEL of 1 ppm for carbon disulfide. There are three companies which currently produce these casings. A study of engineering controls required to comply with this PEL, as well as the costs of these controls, was submitted by industry [Ex. 8–45]. This study indicated high costs to control to 1 ppm. OSHA has subsequently adopted a 4 ppm PEL for the final rule. OSHA concludes that industry can comply with this level. (According to docket evidence, at least one company already operates at 10 ppm or less [Ex. 3–945].) The costs are clearly feasible.

SIC 33: Representatives of the American Iron and Steel Institute

* All Exhibit [Ex.] numbers refer to the material in Docket H–020, the official record of this rulemaking. References to the transcripts of the public hearings, available in the docket, are identified as "Tr." followed by the date of the hearing and the page numbers of the transcript.

indicated in their comments that because of the "fragile financial condition" of the industry, capital investment for equipment such as engineering controls has been limited [Ex. 72, pg. 32]. Representatives expressed concern over the costs to comply with proposed OSHA PELs for several different substances and the economic impact on competition with foreign steel producers [Ex. 72, pg. 3, and pp. 33–34]. Although the dollar has recently depreciated in value relative to the yen and European currencies, depreciation of the dollar relative to the value of currencies of steel-producing countries has been gradual [9]. It is likely that the dollar's depreciation will not be as beneficial to the steel industry as it will for other industries. However, exports of steel-intensive products (excluding motor vehicles) has increased due to the dollar's depreciation (by June of 1987, the volume of exports was 24% over the 1986 level) [9]. Although the steel industry is not growing rapidly, it is certainly not experiencing the downturn of the early 1980's, and the impact of compliance costs should not be as detrimental as the industry predicted [Ex. 72].

In addition, the PELs for iron oxide and aluminum metal dust, two substances which constituted a significant part of the costs estimated for this industry in the 1987 proposal, will not be changed from the OSHA standard currently in effect. OSHA recognizes the special feasibility problems of complying with the proposed PELs for hazardous substance exposures in the steel industry, and is allowing the use of respirators in operations where carbon monoxide and sodium dioxide are present. These changes will substantially decrease costs to the industry, and hence will lessen the economic impact.

SIC 37: Styrene exposures in the manual layup/sprayup operations in the boat building industry are difficult to control through engineering methods due to the nature of the operation and small space within which the styrene is applied [Ex. 3–742]. Evidence submitted to the docket suggests typical exposures in this industry are below 50 ppm except in the layup/sprayup operation [Ex. 3–742]. OSHA is permitting respirator use in these operations in this industry in view of special compliance problems. The costs are low (.24%) in relation to profits. OSHA concludes there will be no adverse economic impact on the industry.

SIC 51: Concern was expressed by the National Grain and Feed Association (NGFA) on behalf of the grain elevator operators/grain handlers in SIC 5153 and the National Cotton Council regarding the feasibility of the proposed PEL of 4 mg/m^3 for grain dust [Ex. 3–752 and Ex. 3–1080]. For the final rule, OSHA has established a PEL of 10 mg/m^3. Most employee exposures are at or below 10 mg/m^3, (see Chapt. G, pg. 19).

OSHA's assessment of the economic impact of the proposal by two-digit SIC was criticized as being too general an approach for estimating the economic consequences of the rule on industry subsectors [Ex. 3–752]. The economic impact of the standard is based on the costs presented above in Chapter G. These costs are based on an industry survey conducted by OSHA for this rulemaking which gathered data at the four digit level. However, the survey was designed to be statistically meaningful at the cell level (two or three digit SIC level). There would be more uncertainty at the four digit level. Much four digit data were in the record and OSHA developed more when requested by participants. As discussed above, OSHA concludes that its cost estimate for SIC 51 (which includes many grain elevators) is accurate. The costs demonstrate economic feasibility even if all costs were borne by SIC 5153 (grain elevators).

SIC 72: In the proposed standard, OSHA indicated an intention to change the PEL for perchloroethylene to 50 ppm. Employees are exposed to this chemical during a wet-to-dry industrial process used in the dry cleaning, laundry, and garment sector (SIC 721). Comments received from the International Fabricare Institute indicated that by 1992, almost all machines used by dry cleaners will be dry-to-dry, a process which has reduced exposures to perchloroethylene [Ex. 3–671]. OSHA believes that industry can comply with a lower PEL of 25 ppm within the four year phase-in period through the normal course of capital replacement as dry-to-dry process equipment is substituted for wet-to-dry process equipment.

OSHA is sympathetic to the circumstances of the number of small businesses in this SIC. OSHA has stated in the Preamble discussion that a phase-in period, up to five years, will be allowed for engineering controls. If it appears that there will be a significant economic difficulty for small dry cleaning operations to convert to new equipment or to retrofit within the time period permitted by the Standard, OSHA will consider extending the phase-in period for firms in this industry.

Regulatory Flexibility Analysis

In accordance with the Regulatory Flexibility Act (P.L. 96–353, 94 Stat. 1164 [5 U.S.C. 601 et seq.]), OSHA has assessed the impact of the rulemaking on large and small establishments. For this assessment, large establishments are defined as those with 20 or more employees and small establishments as those with 19 or fewer employees. The results of this assessment are summarized in Table H–3.

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TABLE H-3

ECONOMIC IMPACTS BY ESTABLISHMENT SIZE

SIC	Industry	Percentage Change in Profits	
		Large	Small
20	FOOD PROD.	- 0.1526	- 3.0770
21	TOBACCO	- 0.0003	0.0000
22	TEXT. MILL	- 0.7442	- 7.0804
23	APPAREL PROD.	- 0.7916	- 3.4067
24	LUMBER & WOOD	- 0.5895	-10.4061
25	FURNITURE	- 0.7701	- 1.8987
26	PAPER PRODUCTS	- 0.4440	- 0.6384
27	PRINTING & PUB.	- 0.0536	- 1.9402
28	CHEMICAL PROD.	- 0.1390	- 0.9853
29	PETRO. REFINING	- 0.2619	- 0.0458
30	RUBBER & PLASTICS	- 0.7788	-28.5020
31	LEATHER PROD.	- 0.1838	- 3.4971
32	STONE & CLAY	- 0.5564	- 1.2857
33	PRIMARY METALS	- 1.0084	- 2.1953
34	FAB. METALS	- 0.2889	- 1.5027
35	MACHINERY	- 0.0988	- 1.8448
36	ELEC. MACH.	- 0.0766	- 1.1400
37	TRANS. EQUIP.	- 0.0883	-21.2970
38	INSTRUMENTS	- 0.1201	- 1.6686
39	MISC. MANUF.	- 0.3489	- 1.9125
40	R.R. TRANS.	n/a	n/a
45	AIR TRANS.	n/a	n/a
47	TRANS. SERVICES	n/a	n/a
49	ELEC., GAS & SAN.	n/a	n/a
50	WHOLESALE, TRADE[1]	- 0.6786	- 1.2044
51	WHOLESALE, NON-DUR	- 0.1638	- 0.7812
55	AUTO DEALERS	- 0.0948	- 0.2293
72	PERSONAL SERV.	- 0.2395	- 0.6369
73	BUSINESS SERV.	- 0.1374	- 0.1376
75	AUTO REPAIR	- 0.4836	- 0.0786
76	MISC. REPAIR SERV.	- 0.6105	- 1.8934
80	HEALTH SERVICES	- 0.0736	- 0.0295

Source: U.S. Department of Labor, Occupational Safety and Health Administration, Office of Regulatory Analysis.

Notes: 1. Consists of SIC 5093 (scrap and waste materials) only.

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Industry sales and profit estimates were based on data from Dun and Bradstreet and the Department of Commerce 1982 Census of Manufactures [5], Wholesalers [6], Retailers [7] and Services [8]. Sales and profit data for selected transportation sector industries (SIC 40, 45, 47 and 49) were not available for use in this assessment.

The information summarized in Table H–3 indicates that with three exceptions small firms will not have important adverse impacts.

Data for small establishments in SIC 24 (Lumber and Wood), SIC 30 (Rubber and Plastics), and SIC 37 (Transportation Equipment Manufacturers), show the potential for more significant changes in profits. In the case of SIC 24, many small businessmen and their representatives testified and supported the final standard. This suggests that the impact will be manageable.

It should be noted that these negative effects result in part from the extreme assumption of perfectly elastic demand. An important ameliorating factor for each firm will be its ability to pass through additional costs to the consumer. The ability of individual firms to do this will be dependent upon product demand elasticities. It is expected that most impacted firms will be able to pass through some portion of their increased costs.

Environmental Impact Assessment

This assessment has been prepared in accordance with provisions of the National Environmental Policy Act (NEPA) (42 U.S.C. 4325 et seq.) as well as the regulations of the Council on Environmental Quality (40 CFR Part 1500), and DOL–NEPA Compliance Procedures (29 CFR Part 11).

OSHA has reviewed the standard and the information contained in the secondary data bases, as well, as the information submitted by the contractors' industry experts and submissions by the public to the record during the course of this rulemaking, and has concluded that no significant environmental impacts are likely to occur as a result of this action.

Two environments may be affected by an OSHA regulatory action: (1) The workplace environment; and (2) the general human environment external to the workplace, including impacts on air and water pollution, solid waste, energy, and land use. Usually OSHA regulations have their most significant impacts on the workplace environment since this environment is under the Agency's jurisdiction. Lower and new PELs will benefit the workplace environment because they will reduce worker exposure to toxic substances.

In most cases, the effects of previous OSHA regulations on the exterrnal environment have been negligible because of their limited scope and application. Similarly, there is no evidence to indicate that there would be any significant adverse impacts to the external environment as a result of this standard. As with other OSHA regulations in the past, however, there may be a potential benefit to the environment. The potential benefits and other impacts are briefly summarized here.

Air Pollution. Because of the nature of the emission standards of the Environmental Protection Agency (EPA) (40 CFR Part 61), many industry operations already use engineering controls to reduce the amount of emissions to the atmosphere. This practice is not expected to change as a result of the rule. OSHA anticipates that controls already in place will continue to operate effectively in reducing emissions under the revised standard. Fourteen of the chemicals addressed in this standard have been recognized by EPA as air pollutants. These are listed below:

- Beryllium
- Carbon Monoxide
- Epichlorohydrin
- Ethylene dichloride
- Hexachlorocyclopentadiene
- Mercury
- Methyl chloroform
- Nitrogen dioxide
- Ozone
- Perchloroethylene
- Sulfur dioxide
- Toluene
- Trichloroethylene

Water Pollution. EPA regulates over 100 of the chemicals addressed in this standard under the Clean Water Act of 1977 (33 USC 1251 et seq.). EPA's effluent limitation guidelines (40 CFR Part 427) include (1) standards of performance for all new point sources within specified categories and (2) pretreatment standards for new plants discharging to municipal sewer systems. These limitations would serve to prevent the discharge of effluents into the environment without prior treatment. Moreover, the Federal Water Pollution Control Act Amendments of 1972 required that wastewater effluents be treated by the best practicable technology (BPT) by December 31, 1977 and that the best available technology (BAT) economically achievable be used by December 31, 1983. The EPA effluent limitations establish the degree of effluent quality necessary to meet the BPT and BAT requirements. The BAT and pretreatment standards would essentially mean no discharge of process wastewater to navigable waters and no discharge of incompatible pollutants. These requirements will not change as the result of this proposal and where they continue to be met, effluent quality will not be altered.

Solid Waste Disposal. It does not appear that there would be any significant change in present waste disposal practices for over 80 chemicals addressed by this rule, or in the maintenance of waste disposal sites. EPA's national emissions standards will continue to provide for the control and maintenance of active and inactive disposal sites and require no visible emissions from these sites.

Energy And Land Use. The implementation of required engineering controls could result in an increase in total energy requirements or costs for general industry. This would be particularly true where controls are not in place. Where general exhaust ventilation is used, there is the expense of heating or cooling the replacement air brought in from the outside. These costs, plus the cost of vacuuming, where necessary, have been included in the annual costs estimated in Chapter G. In terms of land use, OSHA does not project any significant impact on land use plans, policies or controls. OSHA does not anticipate any significant impact on the short term uses of man's environment or upon the maintenance of long-term productivity.

Other Impacts. The standard could also have other impacts that may affect the external environment. The standard could encourage the further use, research, and development of suitable substitutes for hazardous chemicals. This, in turn, would result in a positive environmental effect because fewer hazardous chemicals would be used, emitted to the air, discharged as wastewater effluent, or disposed of as solid waste. The magnitude or probability of these impacts, however, is impossible to quantify.

Overall, the projected impacts of the standard on the external environment are not expected to be significant in view of EPA's regulation of air emissions, water effluents, and solid waste disposal methods.

Summary

Based on the data summarized in Tables H–1 and H–2 and historical information, and information submitted by the public during this rulemaking procedure, OSHA has concluded that the economic impacts of the standard

are clearly feasible for industry sectors and subsectors. However, the estimates indicate that some small establishments in SICs 24, 30, and 37 may experience a greater impact than larger entities. The rule is not expected to have an adverse effect on the environment.

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Supplement 1—Technical Description of the Sample Survey

1. Introduction

This appendix contains a description of the statistical methodology employed to design and implement the PEL survey. The following topics will be discussed:

- Survey objectives;
- Sampling frame selection;
- Stratification;
- Sample size determination;
- Estimation procedures;
- Data collection method;
- Variance estimation;
- Treatment of non-sampling errors; and
- Survey instrument.

2. Survey Objectives

Surveys are frequently designed to produce a set of estimates at a predefined level of accuracy. This requires defining the set of quantities to be estimated and specifying their levels of accuracy. Since many variables may ultimately be estimated from the survey, and since no single design can be optimal for all estimates simultaneously, it is customary to define the most important variables for estimation. For this survey, the following variables were identified as those motivating the survey design:

- Cost to industry of the proposed set of new permissible exposure limits (as a group);
- Number of workers potentially exposed to toxic substances; and
- Number of workers affected by the proposed regulations.

Statistical theory dictates that responses be concentrated both in groups which have the highest variability with respect to these variables and in groups representing the majority of establishments in the population. No hard information relating to the variability of the variables mentioned above by industry sector or other relevant breakdown was available at the outset of the survey. Hence, the variability in the number of employees was used as a variability measure. Consistent with the notion that the variability of numbers exposed as well as the variability of cost required to remedy an overexposure are highest in the largest companies, the sample was designed to include a higher proportion of larger establishments.

The sample was drawn so as to insure that the relative standard errors (RSE) estimates (the ratio of the sample standard error to the mean) was within predetermined bounds. The relative standard error is a measure of the accuracy of each estimate. A relative standard error of 5 percent means that the standard error of the estimate is equal to 5 percent of that estimate. This can be interpreted as saying that the estimate is within two standard errors or 10 percent of the true value with 95 percent probability. Since risks were judged to be different in different sectors, OSHA selected a 5 percent relative standard error in the industries using the most chemicals, 7.5 percent in industries with moderate use of chemicals and 10 percent in the service sectors. A table of design specifications is included in Section A.5 below.

3. Sampling Frame Selection

The Dun and Bradstreet (D&B) listing was chosen for the PEL survey sampling frame (a listing of establishments from which sample units are selected). This is a nationally based list, containing establishment names as well as each establishment's address, telephone number, SIC code, and number of employees. The Dun and Bradstreet database is regularly refined (every six months) thus minimizing the probability of obtaining out of business or out of scope (e.g., wrong SIC code) establishments when using the frame. The D&B is a commercial listing and its use does not violate any confidentiality requirement associated with other frames available to particular agencies in the government.

4. Stratification

Thirty-four groupings of industries (estimation cells) were chosen to be examined for the PEL study. The cell definitions were determined by grouping together industry sectors defined by Standard Industrial Classifications (SICs) which share similar processes and procedures. The cell definitions used for the PEL survey are given in TABLE 1–1.

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TABLE 1-1

Definitions of Estimation Cells

Cell Number	SIC Codes Included	Description
1	243	Millwork,Veneer,Plywood
2	245	Wood Bldgs & Mobile Homes
3	249	Misc. Wood Products
4	25	Furniture
5	26	Paper Products
6	27	Printing & Publishing
7	281	Indust. Inorganic Chems
8	282	Plastics & Syn. Resins
9	283	Drugs
10	284	Soaps, Detergents & Cleaning
11	285	Paints, Varnishes, Lacquers
12	286	Indust. Organic Chems
13	287	Agricultural Chemicals
14	289	Misc. Chemical Products
15	291	Petroleum Refining
16	295	Paving & Roofing Materials
17	299	Misc. Petroleum Products
18	308	Misc. Plastic Products
19	30 (not 308)	Plastics & Rubber
20	311	Leather Tanning
21	31 (not 311)	Leather & Leather Products
22	32	Stone & Clay

TABLE 1-1, cont'd

Definitions of Estimation Cells

Cell Number	SIC Codes Included	Description
23	33	Primary Metals
24	34	Fabricated Metals
25	35	Machinery
	36	Electrical Machinery
	38	Instruments
	39	Misc. Manufacturing
26	40	R.R. Transportation
	44	Water Transportation
	45	Air Transportation
	47	Transportation Services
27	46	Pipelines
28	49	Electrical,Gas & Sanitary
30	5093	Misc. Durable Goods
	5153	Grain
	5161	Chemicals & Allied Products
	5191	Misc. Farm Supplies
	5198	Misc. Paints, Varnishes
31	55	Auto Dealers
	75	Auto Repair
32	7211	Power Laundries, Family & Commercial
	7213	Linen Supply
	7215	Coin-operated Laundries & Cleaning

TABLE 1-1, cont'd

Definitions of Estimation Cells

Cell Number	SIC Codes Included	Description
32 cont.	7216	Drycleaning Plants, except Rug
	7218	Industrial Launderers
	7219	Laundry & Garment Services, nec
	7221	Photographic Studios, Portrait
	7231	Beauty Shops
	7241	Barber Shops
	7251	Shoe Repair & Shoeshine Parlors
	7261	Funeral Service & Crematories
	7299	Miscellaneous Services, nec
	7332	Blueprinting & Photocopying Services
	7342	Disinfecting & Pest Control Services
	7395	Photofinishing Laboratories
33	7641	Furniture Repair
	7692	Welding Repair
34	80	Health Services
99	37	Transportation Equipment

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For each estimation cell, units on the Dun and Bradstreet sampling frame were classified into one of the four size classes listed below:

Size:	Number of employees
1	1 to 19.
2	20 to 99.
3	100 to 249.
4	250 and above.

For each size class stratum within a cell, the establishments on the frame were further identified by their four digit SIC classification (within the two or three digit sample cell). A separate systematic sample was then selected in each estimation cell/size class stratum. This procedure was accomplished by first selecting one case at random in the size class from the first K units on the frame—where K is the reciprocal of the sampling fraction—and then selecting every Kth unit in the stratum thereafter. Note, from the size class definitions that establishments having zero employees were not included in this survey. Such units were assumed to be out of the scope of the survey.

5. Sample Size Determination and Allocation Within Strata

The total number of establishments selected from the Dun and Bradstreet sampling frame was determined using two stages. The first stage was to compute the target number of respondents for each estimation cell using the standard sample size formula. The required specifying a target relative standard error (RSE) for the cell estimates. The RSE's for this survey were set at the following levels:

	Relative standard error (percent)
SIC range:	
24 through 29	5.0
30 through 39	7.5
40 through 80	10.0

The units were then allocated to size classes within the estimation cells using Neyman allocation. This method allocates based on the number of establishments in each stratum and on the stratum variability in the key design variable (in this case employment). Size class strata having a large number of establishments on the frame or a high variability in employment (as defined by the population variance) received a greater number of sample units than other strata in the sample. Because the larger size classes often have a high variability in employment, this allocation resulted in "oversampling" the larger size classes in a cell. The required number of cases for each stratum are shown in TABLE 1–2 in the column labeled "Target Number of Respondents."

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TABLE 1-2

Number of Firms, Required Sample Sizes, Calls Made and Completes

SIC GROUPS	Size	Total Plants	Target Number Respondents	Total Cases Called	Number Completed May 1988
243	1-19	10,986	39	78	41
	20-99	1,995	32	64	40
	100-249	346	12	24	15
	>250	147	48	98	64
	Total	13,474	131	264	160
245	1-19	864	15	25	9
	20-99	385	15	40	26
	100-249	220	17	45	19
	>250	43	30	43	20
	Total	1,512	77	153	74
249	1-19	4,301	37	74	43
	20-99	888	35	70	45
	100-249	129	11	22	12
	>250	44	12	24	15
	Total	5,362	95	190	115
25	1-19	11,505	20	40	21
	20-99	3,254	26	52	34
	100-249	858	13	26	13
	>250	449	73	146	86
	Total	16,066	132	264	154
26	1-19	3,485	20	44	13
	20-99	2,830	30	62	34
	100-249	1,307	30	54	34
	>250	576	184	384	227
	Total	8,198	264	544	308
27	1-19	64,922	45	60	30
	20-99	10,656	34	122	77
	100-249	1,850	12	126	87
	>250	869	88	50	40
	Total	78,297	179	358	234
281	1-19	1,721	20	52	25
	20-99	735	20	52	33
	100-249	189	20	52	29
	>250	157	96	157	68
	Total	2,802	156	313	155

TABLE 1-2, cont'd

SIC GROUPS	Size	Total Plants	Target Number Respondents	Total Cases Called	Number Completed May 1988
282	1-19	700	20	40	25
	20-99	499	20	40	25
	100-249	184	20	40	21
	>250	175	58	116	52
	Total	1,558	118	236	123
283	1-19	1,289	25	50	28
	20-99	544	25	50	30
	100-249	179	25	50	26
	>250	205	92	184	92
	Total	2,217	167	334	176
284	1-19	3,065	20	40	23
	20-99	767	20	40	31
	100-249	184	20	40	22
	>250	155	70	140	61
	Total	4,171	130	260	137
285	1-19	1,092	20	50	36
	20-99	549	15	40	20
	100-249	100	10	30	15
	>250	45	37	45	33
	Total	1,786	82	165	104
286	1-19	860	20	54	28
	20-99	346	15	44	29
	100-249	95	15	44	28
	>250	92	50	62	39
	Total	1,393	100	204	124
287	1-19	1,306	8	31	14
	20-99	338	9	33	19
	100-249	57	4	23	17
	>250	43	44	44	21
	Total	1,744	65	131	71
289	1-19	2,562	16	38	26
	20-99	918	23	52	31
	100-249	162	9	24	15
	>250	85	51	85	38
	Total	3,727	99	199	110

TABLE 1-2 , cont'd

SIC GROUPS	Size	Total Plants	Target Number Respondents	Total Cases Called	Number Completed May 1988
291	1-19	606	20	50	23
	20-99	227	20	50	23
	100-249	85	20	50	28
	>250	130	59	90	48
	Total	1,048	119	240	122
295	1-19	862	21	46	27
	20-99	237	26	56	38
	100-249	45	10	24	20
	>250	9	8	9	3
	Total	1,153	65	135	88
299	1-19	516	15	32	24
	20-99	186	22	46	35
	100-249	23	4	11	11
	>250	5	5	5	3
	Total	730	46	94	73
308	1-19	8,062	16	32	18
	20-99	4,249	13	26	22
	100-249	1,162	7	14	17
	>250	388	18	36	23
	Total	13,861	54	108	80
30 (not 308)	1-19	1,983	25	50	31
	20-99	722	25	50	33
	100-249	239	25	50	33
	>250	234	48	96	61
	Total	3,178	123	246	158
311	1-19	283	5	24	3
	20-99	125	9	21	11
	100-249	29	4	16	3
	>250	16	5	10	5
	Total	453	23	71	22
31 (not 311)	1-19	2,232	8	16	6
	20-99	610	13	26	20
	100-249	220	13	26	16
	>250	176	13	26	17
	Total	3,238	47	94	59

TABLE 1-2, cont'd

SIC GROUPS	Size	Total Plants	Target Number Respondents	Total Cases Called	Number Completed May 1988
32	1-19	14,499	15	28	10
	20-99	4,207	34	62	40
	100-249	873	35	64	44
	>250	448	29	53	33
	Total	20,027	113	207	127
33	1-19	4,983	67	201	81
	20-99	2,803	67	201	139
	100-249	1,006	42	126	86
	>250	711	25	75	54
	Total	9,503	201	603	360
34	1-19	29,005	62	113	56
	20-99	11,849	110	200	117
	100-249	2,394	86	157	86
	>250	1,080	62	113	66
	Total	44,328	320	583	325
35,36, 38,39	1-19	117,005	100	200	90
	20-99	30,820	126	188	123
	100-249	7,468	93	137	98
	>250	5,657	80	133	84
	Total	160,950	399	658	395
40,44, & 45	1-19	45,323	20	37	20
	20-99	5,612	20	37	15
	100-249	799	20	37	15
	>250	533	50	91	32
	Total	52,267	110	202	82
46	1-19	439	15	28	20
	20-99	162	15	28	21
	100-249	18	8	18	16
	>250	5	5	5	6
	Total	624	43	79	63
49	1-19	12,982	40	73	47
	20-99	4,046	40	73	57
	100-249	844	40	73	58
	>250	558	150	273	206
	Total	18,430	270	492	368

TABLE 1-2, cont'd

SIC GROUPS	Size	Total Plants	Target Number Respondents	Total Cases Called	Number Completed May 1988
50 & 51~	1-19	45,422	200	364	233
	20-99	3,464	65	142	106
	100-249	205	30	79	48
	>250	57	57	57	31
	Total	49,148	352	642	418
55 & 75	1-19	284,632	10	30	20
	20-99	20,846	10	30	23
	100-249	1,523	10	30	18
	>250	116	20	60	31
	Total	307,117	50	150	92
72 & 73~~	1-19	139,889	120	240	119
	20-99	5,511	30	60	33
	100-249	527	20	40	28
	>250	108	25	50	23
	Total	146,035	195	390	203
7641 & 7692	1-19	18,098	60	110	67
	20-99	289	20	48	32
	100-249	12	10	9	8
	>250	1	1	1	0
	Total	18,400	91	168	107
80	1-19	233,984	50	91	50
	20-99	17,174	30	55	34
	100-249	6,310	30	55	39
	>250	3,912	220	400	279
	Total	261,380	330	601	402
37	1-19	9,863	10	19	5
	20-99	2,997	10	19	12
	100-249	1,026	10	19	13
	>250	1,072	70	128	72
	Total	14,958	100	185	102

~ Refers to SIC Codes: 5093, 5193, 5161, 5191, 5198
~~Specifically Sic Codes: 7211,7213,7215,7216,7218,7231, 7241,7251,7261,7299,7732,7342, and 7395

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The number of units actually selected from the D&B frame in each stratum was based on the number of completed cases required for the stratum and on the expected response rate. Almost all sample surveys, especially voluntary surveys, select some number of cases which do not result in a completed interview. In some instances, these will be establishments which have gone out of business, are duplicate cases, or are companies not in the SIC category shown on the frame. Such cases are "Out of Scope." Other establishments, though in scope, refuse to participate or are not reached in the sampling protocol, defined here as a total of five telephone attempts. Experience on surveys similar to the PEL survey indicated that a completion ratio of 50–60% was expected for this survey (the ratio of completed questionnaires to total cases which must be drawn—both in and out of scope). However, to be safe, a larger number of cases were selected and held in reserve from the D&B frame so that, if additional sample units needed to be included to reach the target sample sizes, the cases could be easily obtained.

In fact, for the vast majority of cells, a 60 percent completion ratio was realized. The total number of establishments called in each of the sample strata are shown in TABLE 1–2 in the column labeled "Total Cases Called." In general, this number is equal to the target sample divided by 0.60. The number of completed survey responses is shown in TABLE 1–2.

6. Data Collection Methodology

The data collection method chosen for the survey was Computer Assisted Telephone Interviewing (CATI). In this method the interviewer talks to the respondent on the telephone while sitting in front of a computer screen. Each question to be asked appears on the screen in the proper sequence. CATI systems allow for the responses to be examined during the data collection process. Answers that are out of the possible range of responses or which are not consistent with other answers received earlier in the questionnaire can be immediately identified. Another advantage is that it frees the interviewer from using a hard copy questionnaire which requires skipping manually to different parts of the questionnaire based upon the responses. Finally, this method saves resources by creating a machine readable record of the responses at the conclusion of the interview, thereby eliminating the need for keypunching.

7. Establishment Count Comparison

Comparison of the survey establishment count is designed to put into relief both consistencies and inconsistencies between the sample results compared with other existing databases. Table II–4 of the RIA compares the estimated number of establishments from the sample survey with the Dun and Bradstreet (D&B) establishment list (the sample frame), the establishment count from County Business Patterns (CBP) and the establishment count from the Bureau of Labor Statistics ES–202 file. In general, the survey produced establishment estimates in between the higher D&B counts and the lower CBP and ES–202 establishment numbers.

8. Variance Estimation

As with any sample survey, quantification of sampling error of estimates is an important function. Errors are quantified by computing the standard error of each estimate produced from the survey. Under certain assumptions, the standard error can be used to make probability statements about estimates. For example, an interval approximately equal to two standard errors on either side of an estimate is a 95 percent confidence interval. Such an interval indicates the expected range into which the estimate would fall 95 percent of the time, were the survey to be replicated many times.

A replication technique was used to determine standard errors for the PEL survey. Such techniques involve resampling the sample data multiple times to calculate its variability. A replication method was chosen because of two characteristics of the survey. First, some of the estimates which were planned to be produced are nonlinear, such as the benchmarked estimates described above. Second, nonresponse adjustment was used to modify the final weights. In both of these situations, replication-type variance estimators are particularly useful.

The PEL survey was designed using employment as a variability measure. The survey results are consistent with this design; those estimates more closely related to employment had lower relative standard errors. Hence, the RSE for the survey estimate for the total number of production workers over all industries was three percent, for the total number of workers potentially exposed, four percent overall, and for the total number of workers overexposed, six percent overall.

Compared to these estimates, the final cost estimates had considerably higher RSE's. This stems from the fact that many establishments were assigned a zero cost, while others in the same stratum were often assigned a very large cost. This "all or none" characteristic of the costing algorithm resulted in an increased RSE for this variable. Even so, for all industries combined, the overall RSE for cost was 11 percent.

9. Treatment of Non-Sampling Errors

An important component to any survey effort is the treatment of nonsampling errors. Examples of such errors are:

- Nonresponse bias—error introduced because some selected respondents either do not respond at all (unit nonresponse) or do not respond to a particular question (item nonresponse);
- Response bias—error introduced due to the way questions are phrased or the way respondents interpret what is being asked (this also includes error due to deliberate misrepresentation of the answers to questions by respondents).

In the PEL survey, the nonresponse problem was dealt with using two standard methodologies. For unit nonresponse, a mean imputation procedure was used. This procedure assumes that there is no fundamental difference between respondents and nonrespondents and, therefore, usable cases can be reweighted to represent the entire universe. For item nonresponse, an imputation scheme which uses related cases in the respondent group to estimate the missing data was used.

The situation for response bias was handled by obtaining information from site visits. OSHA conducted 90 site visits in a cross section of industries. A portion of these visits were performed on establishments which were also in the telephone survey. Data on key variables collected during the telephone survey were compared with information obtained from the site visits. This analysis can be found in Supplement 3.

9.1. Unit nonresponse adjustment

To adjust the sample for those cases selected from the D&B frame which where called but were out of scope (OOS), out of business (OOB), or in scope but unwilling to participate in the survey, the following approach was used.

- All solicited sample units were assigned a response code based on the following categories:

Code and Description

03 Non-working telephone number
04 Incorrect SIC—out of scope
05 Out of Business (OOB)
06 Not a business or wrong business
07 Duplicate record

08 Could not reach respondent after five attempts
09 Communication barrier
10 Initial refusal
11 Mid-interview refusal (did not answer initial chemical and process questions)
12 Completed interview (completed both initial chemical and process questions)
13 Other nonresponse.

• All units having a response code equal to 08, 09, 10, 11, 12, or 13 were classified as viable sample units (in scope, in business). Sample units having a response code equal to 12 were classified as both viable and usable. A nonresponse adjustment weight was assigned to each usable record in the database, based on the ratio of viable to usable sample units in the record's cell and size stratum:

$$NRAF_{ij} = \frac{\sum_{k=1}^{n_1} I(V_k)}{\sum_{k=1}^{n_1} I(U_k)} /$$

where:
i=number of estimation cell
j=number of the size class
$I(V_k)$=1 if the kth sample unit is viable, =O otherwise;
$I(U_k)$=1 if the kth sample unit is usable, =O otherwise.

The use of this weight is equivalent to performing a mean imputation for item nonresponse.

The response rate may be defined as the number of usable cases divided by the number of viables, and the completion rate as the number of usables divided by the total number of cases contacted. Using these definitions, the response and completion rates were as follows:

Response rate=68.7%
Completion rate=60.0%

9.2. Item nonresponse adjustment/ imputation

Often survey respondents do not know the answers to some questions or refuse to answer particular questions. In such cases, it is possible to fill in missing values using an imputation scheme. The idea is to use information from both the respondent (answers to other questions which they did supply) and information from other respondents (those answering the missing question) in order to estimate a reasonable response to the missing datum.

The imputation method chosen for the PEL survey is a hybrid method which combines the concepts of a mean imputation and a "hot-deck" imputation. A mean imputation method replaces the missing values on a certain question with the mean value from those respondents answering that question. A hot deck method attempts to find a respondent who matches the respondent having a missing value (in terms of other survey characteristics) and uses the value of the "twin" to replace the missing value. The method used here is a hybrid in the sense that it employs a mean imputation, but only over a small segment of the population which obviously matches the respondent having a missing value.

In particular, the procedure examines three or four digit SIC subgroups within the estimation cell by size class. The mean values of the responses to a particular question of interest in such sample subgroupings were used to impute the missing values in that grouping. In the case of categorical variables (for example, YES/NO questions), a randomization scheme was used randomly supplied the appropriate set of responses to missing questions based on a probability distribution determined from those who responded.

It should be noted that the values which were placed on the database were not intended to be estimates of the missing responses. Rather, they are meant to be substitute responses which allow the case to be used in the generation of survey estimates. In the aggregate, estimates produced using imputed data make sense for use in aggregate estimates, but may not be useful for the individual establishment. Care was taken in the imputation program to be sure that imputed responses were consistent with other answers for the establishment of interest. Original responses to all questions were retained on the sample record and all responses representing imputed values were identified.

One set of questions which was not imputed for was whether monitoring for the presence of certain toxic chemicals was done at the establishment. The data collected produced an estimate, for those establishments where chemicals or processes were found, that 15.8 percent did monitoring, 71.9 percent did not do monitoring, and 12.3 percent of respondents did not know or refused to answer the question. Of those establishments that did monitor, 25.6 percent provided the requested data.

10. Survey Instrument

As mentioned earlier, data collection for PEL survey was accomplished by Computer Assisted Telephone Interviewing. Prior to calling, a letter was sent to each selected establishment. This letter is shown in Exhibit 1–1. Also, a hard copy version of the PEL questionnaire is given in Exhibit 1–2.

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EXHIBIT 1--1

U.S. Department of Labor

Assistant Secretary for
Occupational Safety and Health
Washington, D.C. 20210

SIC Code 3479
Metal Coating & Allied Serv.
OMB Approval No. 1218-0142

February 25, 1988

Mr. John Q. Sample
Chairman
Anycompany
123 Sample St.
Anytown, US 12345

Dear Mr. Sample:

The Occupational Safety and Health Administration (OSHA) of the U.S. Department of Labor is required by law to set permissible exposure limits for chemical substances in the workplace. Current exposure limits were set 17 years ago using values established by the American Conference of Governmental Industrial Hygienists (ACGIH) and the American National Standards Institute (ANSI).

OSHA has begun a process for revising out-of-date permissible exposure limits. To ensure that any new exposure limits take into account actual workplace conditions, we are conducting a voluntary survey of U.S. business establishments. Included will be questions about specific processes which we believe are performed in your industry and a limited (no more than 10 per process) list of chemicals which we believe are involved in those processes. Your facility was selected to be included in the study.

Decisions regarding new permissible exposure limits will be improved significantly if we have input from as many firms as possible. The interview will take about 30 minutes. Names of responding firms will not be associated with their answers, and all data will be treated as confidential by our contractor.

Please help us expedite the survey process by returning to us, within one week, the enclosed postage paid card with the name and phone number of the person in your organization our contract interviewer should contact. If this card is not received, a representative of our contractor, KCA Research, Inc., will call your office directly to conduct the interview or be directed to the company official designated by you.

Enclosed is a list of the topic areas for the survey. This may help in preparing for the interview.

We appreciate your cooperation and look forward to receiving the information we need from your designated representative.

Sincerely,

John A. Pendergrass
Assistant Secretary for OSHA

Enclosure

EXHIBIT 1-1
(cont.)

TOPICS COVERED BY SURVEY

I. GENERAL FIRM CHARACTERISTICS

- Primary activity at this location
- Approximate numbers of production & maintenance workers
- Number of shifts per day and length of shift

II. IDENTIFICATION OF GENERAL PROCESSES PERFORMED BY FIRM

- Chemicals used in specific processes or operations and estimated quantities involved
- Approximate number of work stations or assembly lines used and number of workers at each
- Description of process engineering controls such as ventilation and enclosures
- Estimated frequency of process or operation performance
- Description of personal protective equipment used, including respirators, eye, face, and skin protection
- Information regarding exposure monitoring

EXHIBIT 1-2

_____, we are conducting a survey on behalf of OSHA to assess the current practices of all types of businesses in the handling of toxic and hazardous chemicals. A letter was sent to you informing you of this survey.

1. Did you receive our letter?

1 = Yes
2 = No

If answer "Yes", begin next paragraph with "As you know,"
If answer " No ", begin next paragraph with "I'm sorry. Let me summarize what the letter said about the survey".

We are interested in understanding all significant operations or processes in your firm that generate dust, mist, fumes, gases or vapor that your employees might potentially encounter. Of course, all responses and trade or technological secrets will be kept strictly confidential and no company-specific information will be released to OSHA.

2. Should I direct my questions to you, or is there someone else in the firm who you feel would be better qualified to answer?

1 = Yes, this person will answer survey
2 = No, call: Name_____
Title _____
Phone _____
C = Call back (Set up time for recontact)
R = Refused to answer (Terminate interview)
D = Don't Know/ No Response

Let me begin by asking some general questions about your facility

3. Our records show your firm to be engaged in _____.? Is this correct? (Interviewer will read title or brief description for this SIC code.)

1 = Yes
2 = No, our function here is _____
C
R
D

EXHIBIT 1-2
(cont.)

4. How many production workers do you have at this location?

1 = _____ production workers
C = Call back
R = Refused to answer
D = Don't know

5. How many maintenance workers (for example: painters, welders & cleaning staff) do you employ?

1 = _____ maintenance workers
2 = Production workers do maintenance functions
3 = None, only clerical, managerial, or sales personnel
C
R
D

5a. Of these maintenance workers, how many do painting as their primary work activity?

1 = _____ do painting as primary activity
2 = None
C
R
D

5b. Of these maintenance workers, how many do welding as their primary work activity?

1 = _____ do welding as primary activity
2 = None
C
R
D

6. How many shifts per day (24 hr. period) do you have at this location?

1 = _____shifts/24 hr.
C
R
D

EXHIBIT 1-2
(cont.)

I now want to ask you some questions about chemicals which we believe are common among firms in your industry. [These chemicals would be selected on the basis of large volume usage, known toxicity, or known exposure problems in excess of permissable limits as identified from NOES or IMIS or from industry expert opinion].

7. Which of the following chemicals are used, processed, or emitted at your facility?

Chemical A 1=Yes 2=No C R D

[The interviewer will read chemical list specified for this 4-digit SIC If "Don't Know" (D) is the response, the interviewer will then attempt to clarify the question by reading a list of common synonyms for the chemical. The subsequent answer can then be reassessed as "Yes" or "No".

8. Are there any other chemicals in major use in your operations that I did not list?

1 = Yes (Skip to #8 and add to list)
2 = No
C
R
D

9. What is the approximate quantity of chemical A that your facility purchases each week or month?

1 = _____ lbs. per week purchased
2 = _____ gals. per week purchased
3 = _____ lbs. per month purchased
4 = _____ gals. per month purchased
C
R
D

Repeat Question #9 until all identified chemicals are quantified.

EXHIBIT 1-2
(cont.)

10. Have exposure limits been adopted by your firm for these chemicals?

1 = Yes
2 = No
C
R
D
(Skip to #12)

11. What exposure limits have been adopted?

1 = OSHA PEL's
2 = NIOSH REL's
3 = ACGIH TLV's
4 = Other _____
C
R
D

The next questions are about processes/operations which we believe are common among firms in your industry

12. Are any of the following processes/operations performed in your facility?

Operation #1 1=Yes 2=No C R D

[Interviewer would read list of up to 6 processes or operations specified for this 4-digit SIC code . This list would be identified from secondary data sources and industry experts. If information regarding relevant processes was not available or sufficient, then this question would be rephrased to elicit process/operation identification from the respondent].

13. Are there any other processes/operations at your facility that I did not list?

1 = Yes (Skip to #12 and add to list)
2 = No
C
R
D

EXHIBIT 1-2
(cont.)

For each identified process/operation, ask questions 14 - 26

14. In Process/Operation 1:

Is Chemical A used? 1=Yes 2=No C R D

REPEAT UNTIL ALL IDENTIFIED CHEMICALS HAVE BEEN ASKED ABOUT USAGE IN THIS PROCESS

15. How many work stations (or assembly lines) are involved in this process/operation?

1 = _____ work stations
2 = _____ assembly lines
C
R
D

16. On average, how many workers are directly involved in this process/operation at each work station (or assembly line)?

1 = _____ workers/work station
2 = _____ workers/assembly line
C
R
D

17. Of these workers, what percent work exclusively at this process/operation?

1 = 100% (Go to #18)
2 = _____%
C
R } (Go to #18)
D

17a. For those workers who do not work exclusively at this process/operation, in what other processes/operations are they also employed?

1 = _____ _____
C
R
D

EXHIBIT 1-2
(cont.)

18. Is this process/operation a completely enclosed activity?

1 = Yes (Skip to #14)
2 = No
C
R
D

19. Is this process/operation located outdoors?

1 = Yes (Skip to #21)
2 = No
C
R
D

20. Is this process/operation ventilated?

1 = Yes
2 = No
C
R
D
(Skip to # 21)

20a. What is the type of ventilation?

1 = Local exhaust 1 = Yes 2 = No C R D
2 = General dilution
3 = Natural ventilation
4 = Other (specify type)
_____

21. How often is this process/operation performed during each shift?

1 = Continuously over entire shift, every shift
2 = Daily (specify #/day) _____
3 = Weekly (specify #/week) _____
4 = Monthly (specify #/month) _____
5 = Yearly (specify #/year) _____
6 = Other (specify #/period) _____
C
R
D

EXHIBIT 1-2
(cont.)

22. Are respirators routinely used by workers?

1 = Yes
2 = No) (Skip to # 23)
C
R
D

22a. What type of respirator?

1 = Single use
2 = Half-Mask cartridge
3 = Half-mask canister
4 = Full-face cartridge
5 = Full-face canister
6 = Powered air purifying respirator
7 = Air supplied respirator
8 = Self-contained breathing apparatus
9 = Escape respirator
10= Other _____
C
R
D

23. Do you provide maintenance workers who have exposure to this process with respirators?

1 = Yes
2 = No
C
R
D

24. Is skin, face, or eye protection used?

1 = Yes
2 = No) (Skip to #25)
C
R
D

24a. What type(s) of skin, face, or eye protection?

1 = Long sleeve shirt
2 = Coverall
3 = Apron
4 = Gloves
5 = Chemical Protective Clothing
6 = Goggles
7 = Face Shield
8 = Other _____
C
R
D

EXHIBIT 1-2
(cont.)

25. Do you have a hazard communications training program for these workers?

1 = Yes
2 = No
C
R
D

26. Has environmental monitoring been done at or near this process/operation?

1 = Yes
2 = No
C
R
D

(Skip to # 14 until all processes surveyed)

26a. Has this monitoring been designed to evaluate control of:

1 = potential short term (< 15 min.) exposures? (STEL)
2 = potential 15 minute - 4 hour exposures?
3 = potential 4 - 8 hour exposures? (TWA)
C
R
D

26b. During this monitoring, were any chemicals found to be in excess of your adopted exposure guidelines?

1 = Yes
2 = No
C
R
D

(Skip to #27)

26c. Which chemical(s) were found to exceed adopted guidelines?

1 = _____
C
R
D

(Skip to #27)

26d. What activity, work process or operation do you feel is most responsible for the exposures above your adopted guidelines?

1 = _____
2 = Not able to specify
C
R
D

EXHIBIT 1-2
(cont.)

27. Can you give us your monitoring data for Process 1?

1 = Yes
2 = No
C
R
D
} (Skip to #14)

27a. What is the name of the first (next) chemical for which you have monitoring data?

1 = _____
C
R
D

27b. Is the data based on actual monitoring readings or is it estimated?

1 = Actual
2 = Estimated
C
R
D

27c. Is the data for the work area or for the person (worker)?

1 = Area
2 = Person
C
R
D

27d. Is the data recorded for the individual worker or the work process?

1 = Worker
2 = Process
C
R
D

EXHIBIT 1-2
(cont.)

27f. Is the unit of measurement parts per million or milligrams per cubic meter?

1 = PPM
2 = Mg/M3
C
R
D

27g. What is the exposure data for this chemical

1 = _____
C
R
D

27h. Do you have exposure estimates for other chemicals used in this process?

1 = Yes (Skip to 27a)
2 = No
C
R (Skip to #14 until all processes surveyed)
D

28. What do you estimate to be the market value of plant and equipment at your facility?

1 = Less than $50,000
2 = $50,000 - $500,000
3 = $501,000 - $1,000,000
4 = $1 to $5 million
5 = $5 to 50 million
6 = More than $50 million
C
R
D

29. Can you estimate the annual value of shipments from your facility?

1 = Less than $50,000
2 = $50,000 - $500,000
3 = $500,000 - $1,000,000
4 = $ 1 - 5 million
5 = $ 5 - 50 million
6 = More than $50 million
C
R
D

Thank you for cooperating with us in our survey.

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VIII. Summary and Explanation of the Standard

A. Scope and Selection of PELs

On the basis of all evidence, OSHA has concluded that the TLVs published by the ACGIH constitute the best available starting point for determining the substances included in this Rulemaking. Thus, the boundaries of the substances to be considered in this standard were established to include all of the substances included in the 1987–88 ACGIH TLV listing. There was widespread support for OSHA's selection of these substances for regulation which increased the Agency's confidence that the substances selected for this generic rulemaking are both necessary and appropriate. See also the discussion in Section I.D. of the Proposal and IV. D. of this preamble.

NIOSH (Ex. 8–47) additionally recommended as a potential source the Nordic Expert Group for Documentation of Occupational Exposure Limits. NIOSH stated:

> No single source should be expected to stand alone as a comprehensive list of candidates for regulation. OSHA should construct its own comprehensive list by drawing information from all available sources (Ex. 8–47, p. 20).

OSHA agrees with NIOSH in general, although it determined that it was necessary to select a single, comprehensive list as the starting point for consideration for regulation.

As described in the Proposal, the Agency used both the NIOSH RELs and ACGIH TLVs as starting points for establishing PEL's, and then carefully reviewed all of the literature, comments and testimony submitted in the course of this rulemaking. After careful review and evaluation of this body of information on any given substance and in conformance with Agency policy and statutory requirements, OSHA then determined the appropriate PEL or PELs for each substance.

U.V. Henderson, Jr., Director of Environmental Affairs for the Texaco Company, endorsed OSHA's choice of regulatory candidates by stating: "No substances are included in the listings which should be excluded from the rulemaking" (Ex. 3–593). In this regard, NIOSH also expressed support for the inclusion of the proposed substances but urged OSHA to take further action "immediately upon completion of this rulemaking . . . to establish PELs for all substances that are excluded from this rulemaking" and for which NIOSH has made a recommendation to OSHA (Ex. 8–47, p. 19). NIOSH stated that OSHA should initiate "consolidated rulemaking . . . to adopt all NIOSH RELs pending (the initiation of) chemical-specific Section 6(b) rulemaking . . ." (Ex. 8–47, p. 17). In the future OSHA will review those RELs for which there are not PELs. Based on that analysis, other priorities and resources, OSHA will determine the need to develop PELs for these substances.

For its discussion of health effects, OSHA grouped substances on the basis of the TLV documentation. The substances were divided into fifteen generic health effects groups. These were: neuropathic effects, narcotic effects, sensory irritants, liver and kidney effects, ocular effects, adverse respiratory effects, cardiovascular effects, systemic effects, no observed effects, physical irritants and other effects, odor and taste effects, analogy, biochemical and metabolic effects, sensitizers, and carcinogenic effects. The OSHA analysis also considered three special categories concerned with: change only to the STEL; change regarding skin designation in the TLV; and situations where the TLV is greater than the existing PEL.

OSHA is establishing these new limits for general industry only at this time. In the future, consideration will be given to applying these limits to construction, maritime and agriculture. To attempt to consider these sectors in this rulemaking would have delayed this important process. See also the discussion in Section IV. F. of this Preamble.

B. Start-Up Schedule

OSHA intends that the effective date of the new exposure limits shall be March 1, 1989, in conformance with provisions set forth in Section 6(b)(4) of the OSH Act.

In addition, OSHA has set forth start-up dates for most of its health standards acknowledging that it takes time for employers to evaluate exposures as well as to purchase, install and make operable equipment to control such exposures.

In the case of this standard, OSHA has considered the need for start-up dates to allow sufficient time to take into account the fact that many employers will have to evaluate and make operable controls for several different chemicals. This will undoubtedly require more time than would be necessary for only one chemical.

OSHA believes that September 1, 1989, is a reasonable time by which to evaluate exposures and come into compliance with any reasonable combination of engineering, work practice and respirator control methods. OSHA standards generally have had a period of approximately this length or shorter to come into compliance with an exposure limit with any reasonable combination of controls. See for example, the benzene standard, 29 CFR 1910.1028 (m)(2), 52 FR 34460, 345676 (September 11, 1987) and the formaldehyde standard, 29 CFR 1910.1048 (p)(2)(iv), 52 FR 46168, 46296 (December 4, 1987). OSHA experience has indicated that the six-month period following the effective date is appropriate and sufficient to come into compliance with any reasonable combination of controls.

The proposed rule (53 FR 20960 et seq.) suggested six months from the publication date of the final regulation as a reasonable time for employers to evaluate the exposures of their employees and to come into compliance using any combination of respirators, work practices and engineering controls. Several commenters, such as the Texaco Company (Ex. 3–593) and the Synthetic Organic Chemical Manufacturers Association (SOCMA) (Ex. 3–891), indicated that such an approach was appropriate. The Kerr McGee Corporation (Ex. 3–623) was more specific in its comments and contended that the initial 6-month period should be extended to a 24-month period to allow industry sufficient time to monitor and develop the necessary control measures. The American Paper Institute (Ex. 3–685) was also of the opinion that an initial 6-month compliance period would be too short. OSHA believes that the September 1, 1989, date is adequate based on all of the comments received and OSHA's past experience.

OSHA has generally provided a more extended period to come into compliance using the hierarchy of controls contained in 29 CFR 1910.1000 (e), with its preference for engineering and work practice controls. It takes more time, in general, to plan, purchase equipment, install and make operational engineering controls than to implement other types of control strategies. Examples of representative start-up periods include: 1 to 10 years (depending upon the sector) for the lead standard, 29 CFR 1910.1025 (e); 4 years for the cotton dust standard, 29 CFR 1910.1043 (m); 2 years for the benzene standard, 29 CFR 1910.1028 (m)(2)(ii); and 14 months for the formaldehyde standard, 29 CFR 1910.1048 (p)(2)(v). These dates have varied depending upon OSHA estimates of the difficulties involved. OSHA's experience has been that generally the times for these standards have been sufficient.

In the Proposal, OSHA also estimated that all employers, including those who

would have to control exposures for several different chemicals, could achieve compliance within four years using the hierarchy of controls specified in 29 CFR 1910.1000 (e) (i.e., engineering controls, work practices, and if these are not feasible, personal protective equipment). Regarding the four-year engineering controls implementation date schedule, OSHA received a number of comments. Generally industry supported the four-year period. NIOSH (Ex. 8–47) suggested that two years was a reasonable time for compliance, and a number of unions supported that period. The Fibre Box Association, however, recommended ten years (Ex. 3–823).

In testimony July 15, 1988, related to the experience of the Washington State Occupational Safety and Health Administration with respect to updating permissible exposure limits, Stephen M. Cant stated (Ex. 20):

> Washington's PELs became effective thirty days after adoption and did not include a lengthy phase-in for engineering controls. No protests, no complaints, and no observable difficulties have been encountered; however, use of good judgment is always critical to successful enforcement. Engineering controls are not always feasible, although significant improvement, if not total control, is often obtained. In practical terms, longer interim times between implementation and full engineering control tends to occur with lowered PELs and in some cases respirators provide the only control or are used in combination with engineering.

OSHA has evaluated the data from various industries regarding the time needed to come into compliance with the hierarchy of controls set forth in 1910.1000 (e), and has determined that it is feasible for employers in nearly all operations to achieve compliance using engineering controls by December 31, 1992. OSHA's experience is that for substances of normal difficulty, one to two years is sufficient. The longer approximately four-year period takes into account that some employers will have to control several substances and also considers those few substances where compliance may take greater efforts for some employers. Because of the large number of employers and types of industry OSHA covers, OSHA does not believe a very short period similar to that used by the State of Washington would be feasible. For a very small number of specific operations (involving 4 substances—carbon monoxide, carbon disulfide, sulfur dioxide and styrene—which are discussed in this preamble in Section VII.), OSHA has indicated that employers may use any combination of controls and that the burden of proof that the final rule's limits can be achieved in these designated operations using engineering controls will rest with the Secretary of Labor, rather than the employer.

Since OSHA is in the process of reviewing regulations relating to the hierarchy of controls, it asked in the Proposal whether the phase-in period should be based on the final decisions in that rulemaking. Most of those who commented supported fixed dates. The Dow Chemical Company (Ex. 3–741) urged the Agency not to wait to set a start-up date for this rule. A few companies (Exs. 3–669 and 3–527) suggested that the Agency delay the coming-into-compliance period until after publication of any new regulations on this subject; these commenters cited costs of compliance as a major concern.

OSHA concludes that fixed compliance dates are, in general, more appropriate. The times set are reasonable. The additional protection for many workers is a very important goal. Only a small number of participants supported the alternate approach. However, OSHA is setting forth the possibility of a one-year extension as discussed below.

OSHA did not raise the issue of methods of compliance in this rulemaking. The exposure limits required after the Transitional Period, are to be achieved with the then current hierarchy of controls set forth in 29 CFR 1910.1000 (e).

In a separate Rulemaking OSHA will be requesting public comment on methods of compliance shortly. The results of that review may lead to change or no change in the OSHA hierarchy of controls as set forth in 29 CFR 1910.1000 (e).

As discussed, OSHA has concluded that 4 years is a reasonable period for coming into compliance with the new exposure limit through the methods of compliance set forth in 29 CFR 1910.1000 (e) with its preference for engineering and work practice controls. If, however, the rulemaking on methods of compliance has not been completed and published in the **Federal Register** by December 31, 1991, either with a determination to modify or not to modify, then some added flexibility is appropriate.

Accordingly para. 1910.1000 (f)(2)(ii) provides that if the methods of compliance rulemaking is completed by December 31, 1991, then compliance with paragraph (e) to lower exposures to the new limits is to be achieved by December 31, 1992. If, however, the methods of compliance rulemaking is not completed by December 31, 1991, then compliance with paragraph (e) to the new limits is to be achieved by December 31, 1993.

OSHA proposed that in the Transitional Period, the existing exposure limits are to be achieved with the hierarchy of controls specified in 1910.1000 (e). That has been the requirement since 1971. Participants did not object to this provision. OSHA is maintaining this provision in the final standard. Between September 1, 1989, and December 31, 1992, the existing limits from Tables Z–1 (which have been placed in the Transitional Limits Columns of Table Z–1–A), Z–2, and Z–3 are to be achieved by the hierarchy of controls specified in 1910.1000 (e). This is a protective approach and no evidence has been presented to contradict it.

C. Analytical Methods

In the proposal, OSHA included an appendix of analytic methods. It requested comments on those methods and on other methods, OSHA identified seven substances for which it was not aware of acceptable analytical methods. OSHA requested comments on how it should handle substances with no analytical method. It suggested that one approach was to issue a new limit but stay enforcement until a new method was developed.

OSHA received few comments on the methods it proposed, alternate methods, or the approach to be followed for those few substances where OSHA was not aware of a practical method. Both NIOSH (Ex. 8–47) and the Los Alamos National Laboratory (Ex. 3–741) expressed concern about promulgating limits for substances without existing or adequate sampling and analytical methods, i.e., substances requiring special attention because of the lack or inadequacy of methods to measure them in airborne concentrations (53 Fr 20978). For the substances identified by OSHA as lacking an available method, Los Alamos representatives stated that rulemaking "should be delayed until adequate and validated procedures are developed" (Ex. 3–1095). NIOSH agreed with OSHA that substances without existing or adequate sampling and analytical methods should receive special attention (Ex. 8–47). According to NIOSH, "it is important that NIOSH and OSHA work together on a method development scheme that will allow the appropriate validated methods to be developed in a prioritized fashion . . ."; however, NIOSH was not in favor of delaying the implementation date of the final rule because of sampling and analytical deficiencies (Ex. 8–47).

OSHA has reviewed the few comments and the methods identified. OSHA concludes that, for all but the

seven substances identified below, there is an adequate sampling and analytic method for enforcement purposes.

For a few of the substances where OSHA believes there are adequate methods, NIOSH points out that there has not been extensive inter-laboratory cross checking. This procedure (which is known by the technical term "validation") does improve analytic techniques but is not necessary for typical enforcement purposes.

Therefore, OSHA finds that it is appropriate to adopt PELs for all of those substances identified in the Appendix of Section XI as having available in-house sampling and analytical methods. Copies of information on these methods have been submitted to the docket for this rulemaking (Ex. 12) and are available to all parties. Industry and union participants have not criticized these techniques.

In the Proposal, OSHA identified seven substances as not having adequate sampling and analytic techniques for enforcement. OSHA subsequently was informed of reasonable techniques for two of these. However, it also determined that two other substances with inadequate sampling methods were not listed in the Proposal. The list of seven substances now includes aluminum alkyls, ethylidene norbornene, hexafluoracetone, mercury [alkyl compounds], oxygen difluoride, phenylphosphine and sulfur pentafluoride).

OSHA believes it is appropriate to adopt PELs but stay enforcement of these PELs until adequate sampling and analytical methods are available. At such time OSHA will publish in the **Federal Register** its determination that such methods exist (together with a copy of the method), and indicate the proposed effective date for enforcement of the PEL for the substance in question.

OSHA notes the overwhelming success of the private sector and the joint efforts of NIOSH and OSHA to develop sampling and analytical methods in this area in the past. In 1971, at the time of the promulgation of OSHA's original Z-Tables, sampling and analytical methods were available for only a few of the hundreds of substances on these Tables. In the intervening years, NIOSH, OSHA and the private sector have developed and tested hundreds of methods and have made these available to the industrial hygiene community in several volumes of documented methods (*OSHA Analytical Methods Manual* and *NIOSH Manual of Analytical Methods*). OSHA is confident that the two agencies and the private sector will work together to develop rapidly methods for these substances.

D. Content of Standard

The present 29 CFR 1910.1000 contains three Tables and 5 paragraphs. The Tables Z-1, Z-2 and Z-3 express exposure limits for approximately 450 substances in various formats. Paragraph (a) states how Table Z-1 is to be complied with, paragraph (b) how Z-2 is to be complied with and paragraph (c) how Table Z-3 is to be complied with.

Paragraph (d) states the rule to be followed if there are exposures to more than one substance covered by the standard. Paragraph (e) states the hierarchy of controls to be followed in achieving the limit.

In OSHA's Proposal, it opened the rulemaking only to the appropriate exposure limits for 260 substances already included in Tables Z-1, Z-2, and Z-3 and 168 substances with no prior exposure limits. OSHA did not open any substantive issues as to Paragraphs 1910.1000 (a) through (e), or as to the approximately 169 substances in Tables Z-1, Z-2, and Z-3 for which OSHA did not propose to consider changes. However, the need for format changes was recognized since there would be the need to integrate conveniently for the public both the old and new exposure limits.

OSHA proposed a new Table Z-4 which included all of the 428 substances which OSHA proposed to consider for new exposure limits. A new paragraph (d) was proposed to indicate how Table Z-4 was to be complied with including Time-Weighted average (TWA), short term exposure limits (STEL), ceiling limits and skin designations. The provisions of proposed paragraph (d) were opened for public comment. The other paragraphs were proposed only for format changes so that the new limits could be incorporated without confusion. The existing paragraph (d) was redesignated paragraph (f).

There were a number of recommendations by the public on how the exposure limits could be formatted so they would be more convenient for the public to use. OSHA has carefully considered how to present the exposure limits in a manner most convenient for the public. The format of this final standard and Tables reflects that effort.

OSHA is deleting Table Z-1 and inserting Table Z-1-A. (The change in nomenclature is designed to avoid confusion between the two Tables). For the convenience of the public, Table Z-1-A includes every substance regulated by OSHA in subpart Z.

Therefore, Table Z-1-A includes all new substances regulated for the first time in this rulemaking, all substances regulated before in Tables Z-1, Z-2, and Z-3 for which OSHA is promulgating new exposure limits and also those substances regulated before in Tables Z-1, Z-2, and Z-3 for which OSHA considered changing exposure limits but concluded that the exposure limit should remain unchanged. All of these exposure limts were substantively considered and were at issue in the rulemaking. They have been issued or reissued as section 6(b) standards.

Secondly, Table Z-1-A includes several groups of substances which were not considered for change or opened for comment in this rulemaking. They have been placed in Table Z-1-A for the convenience of the public and reformatted but no substantive changes have been made. These include 169 substances which had been located in Tables Z-1, Z-2 and Z-3 which OSHA did not propose to consider changes for and which are carried over substantively unchanged.

For some of those substances located before in Tables Z-2 and Z-3, the format of presentation could not fit into the columns of Table Z-1-A. In that case Table Z-1-A references the fact that those substances' limits appear in Table Z-2 or Z-3.

These substances which were not opened for rulemaking and which appeared before in Tables Z-1, Z-2, or Z-3 can be identified by having identical limits in both the Transitional Limits Columns and the Final Rule Limits Columns of Table Z-1-A. The identical nature of both limits may require examination of a cross reference to Table Z-2 or Z-3. All of these substances were originally issued as Section 6(a) standards.

Also listed in Table Z-1-A are all substances which have individual standards in Sections 1910.1001 through 1910.1048. In those cases the exposure limit is not listed in Table Z-1-A, but there is a cross reference to the section where the complete standard for that substance is located.

There are also three substances (benzene, cotton dust and formaldehyde) which have single substance standards in 1910.1001 through 1910.1048, for which exposure limits in Tables Z-1 or Z-2 were retained for certain sectors, operations or circumstances not covered by the single substance standard. These limits are either presented directly in Table Z-1-A, or are cross referenced to Table Z-2. An explanatory note indicates where these situations apply.

Through these formatting changes, all substances regulated in Subpart Z are listed in alphabetical order in Table Z-1-A. Also included (where possible) is a CAS number to help identify each substance. This formatting will facilitate the use of these Z Tables.

As discussed above, there will be a transition period. Until September 1, 1989, the existing limits of Tables Z-1, Z-2 and Z-3 continue to apply. These are presented, or cross referenced in the Transitional Limits columns of Table Z-1-A. The methods of compliance hierarchy, as set forth in Sec. 1910.1000(e) applies to these limits. For substances where there has been no change in limits, the methods of compliance specified in para. 1910.1000(e) have been applicable to achieve the limit specified since 1971 and will remain applicable without gap into future unless subsequently amended. The September 1, 1989, and December 30, 1992, dates do not affect the methods of compliance or exposure limit for substances whose exposure limits have not been changed. Substances which fit into the unchanged limits category can be recognized because the limits specified in both Transitional Limits column and Final Rule Limits columns are the same.

Between September 1, 1989, and December 31, 1992, two limits will be applicable for substances which had an OSHA limit and for which OSHA changed the limits in this Rulemaking. The methods of compliance hierarchy set forth in 1910.1000(e) will apply to the limits noted in the Transitional Limits columns. The additional protection to achieve the more protective limits noted in the Final Rule Limits columns can be achieved using any reasonable control methods as set forth in para. (f)(2)(ii).

An example may assist in explaining this requirement. Chemical A has a limit of 100 ppm in the Transitional Limits columns and 50 ppm in the Final Rule Limits columns. Between September 1, 1989, and December 30, 1992, 100 ppm must be achieved with the hierarchy of controls specified in Para. 1910.1000(e) with its preference for feasible engineering and work practice controls. During this period, the additional protection from 100 ppm down to 50 ppm must be achieved by any reasonable combination of engineering controls, work practices and personal protective equipment as specified in para. 1910.1000(f)(2)(i).

After December 30, 1992, the methods of compliance specified in para. 1910.1000(e) shall apply to the limits specified in the Final Rule Limits column for all substances with changed limits. The limits specified in the Transitional Limits column shall no longer be applicable.

New substances not previously regulated by OSHA have their exposure limits appear only in the Final Rule Limits columns. For those substances, the methods of compliance specified in para. 1910.1000(f)(2)(i) apply between September 1, 1989, and December 30, 1992, to achieve the airborne exposure limits specified. After December 30, 1992, the methods of compliance specified in para. 1910.1000(e) apply.

If no final rule has been published in the Federal Register by December 31, 1991, amending or determining not to amend paragraph (e) of this section, then the permissible limits specified in the Final Rule Limits columns of Table Z-1-A shall be achieved by the methods of compliance specified by paragraph (e) of this section effective December 31, 1993, and paragraph (f)(2)(i) of this section shall remain in effect through December 30, 1993.

As discussed above, some substances are listed in the Transitional Limits or Final Rule Limits columns by cross reference to Table Z-2 or Z-3. Those substances are considered to be in the Transitional Limits columns or Final Rule Limits columns just the same as if the exposure limits were presented in those columns. Consequently, the methods of compliance apply the same, whether the exposure limit is listed directly or listed by cross reference to Table Z-2 or Z-3.

The operational language for Table Z-1 is in 29 CFR 1910.1000(a) (1988), for Z-2 is in 1910.1000(b) and Z-3 in 1910.1000(c). The language of each was not identical because they had different historical sources. It is and has always been OSHA's interpretation that the language, though slightly different, had the same meaning.

In this rulemaking the Table Z-1 has been integrated into the Transitional Limits columns of Table Z-1-A. The operational language that had been in 29 CFR 1910.1000(a) (1988) becomes paras. 1910.1000(a) (1) and (2). Paragraphs 1910.1000 (b) and (c) are carried over. Some word changes are necessary to these paragraphs to integrate Table Z-1-A into the regulatory framework and to cover the transitional period. These are just formal changes and no substantive changes in the regulations are intended by the formal changes in the language of paras. 1910.1000 (a)(1), (a)(2), (b) and (c).

Paragraphs 1910.1000 (a)(3), (a)(4) and (a)(5) are new. Necessary explanation of them is given below.

Paragraph 1910.1000(d) contains the computation formulas when employees are exposed to more than one toxic substance at the same time and 1910.1000(e) is the hierarchy of controls. OSHA did not open the issue of whether substantive changes should be made to these paragraphs in the proposal. A few comments were received recommending substantive changes. OSHA has not considered them in this proceeding. This rulemaking is sufficiently broad so that resources were not available to consider those recommendations and, of course, no notice was given that OSHA was considering changes to these paragraphs. Accordingly there are no substantive changes to these paragraphs and that was not an issue in the rulemaking. No changes at all are made to para. (e). It is reprinted unchanged for the convenience of the public.

OSHA is making only format changes to para. (d). Those are needed to incorporate Table Z-1-A. They also make clear OSHA's existing position that para. (d) applies to all of Subpart Z. See 53 FR 21241. The names of chemicals in the example are changed to A, B, and C since the exposure limits for the named chemicals have been changed. This should prevent confusion. All of para. (d) is reprinted for the convenience of the public. It should be noted that paragraph (d) had been proposed to be redesignated as paragraph (f) in the Proposal. In the final rule that has not been necessary because of the change in format.

In addition, since OSHA is proposing no changes to Part 1917, Marine Terminals, which references the existing Z-1, Z-2 and Z-3 Tables, the limits shown in the Transitional Limits columns of Table Z-1-A or the limits columns of Z-2 and Z-3 will remain in effect for Marine Terminals. (OSHA in a follow-up rulemaking will consider adoption of new limits for the Construction and Maritime Industries.)

For some substances, OSHA proposed using the 10-hour TWA given in the NIOSH RELs as a new PEL. It should be noted that NIOSH generally refers in its criteria documents to airborne concentrations of a substance as a "time-weighted average (TWA) exposure for up to a 10-hour work shift in a 40-hour work week." OSHA has concluded that this is equivalent to the OSHA definition of an 8-hour work shift for a 40-hour work week. OSHA received limited comments regarding this question. NIOSH (Ex. 8-47) provided the most detailed response explaining the history of the 10-hour TWA and why the same TWA REL was intended to be applied to 8-hour and 10-hour work days in a 40-hour work week. NIOSH explained that the 10-hour REL originated during the energy crisis of the

1970s, when many employers began to use 10-hour/4-day work schedules to conserve energy (Ex. 8–47, p. 25). Thus, the 40-hour work week rather than the length of a workday is, in NIOSH's view, the important time element in the (concentration) X (time) equation: any given REL can be applied to either four 10-hour days or five 8-hour days without being exceeded. NIOSH supports OSHA's proposal to apply 10-hour NIOSH RELs to 8-hour days by stating:

> The action proposed by OSHA in this rulemaking relative to these RELs is consistent with that original intent (Ex. 8–47, p. 26).

In this final rule, OSHA is therefore applying certain values derived from 10-hour NIOSH RELs as 8-hour TWA PELs.

NIOSH REL ceiling values are based on time intervals which range from instantaneous to 120 minutes. OSHA asked in the Proposal whether, for convenience of enforcement fewer time limits could be used. There were a few comments which gave support to this possibility. After consideration of the record, OSHA has concluded that PELs based on REL ceilings of 10, 15 and 20 minutes shall be made 15-minute STEL's in order to achieve greater uniformity and simplicity in the standard. However, OSHA has decided that the 30-minute, 60-minute and 120-minute ceilings, if adopted, shall remain as specified since those times are so different.

The ceiling limits in Table Z–1–A are consistent with the ACGIH definition. If instantaneous measuring devices are available, then the ceiling limit shall not be exceeded in an instantaneous measurement. If instantaneous measuring devices are not available, then the exposure is to be measured over a 15-minute period. Therefore, some of the ceiling limits are equivalent to STELs.

OSHA proposed PELs for some substances where the basis for the proposal also included a carcinogenicity designation (e.g., TLV with an A1 or A2 designation; REL with a Ca designation). OSHA asked in the Proposal whether such chemicals should have a cancer designation included in the table. Some commenters (Exs. 3–741 and 3–891) indicated that OSHA's Hazard Communication Standard already requires employers to inform employees about the carcinogenic hazards of any substances listed as carcinogens by IARC or NTP. According to these respondents, identification of substances as carcinogens in the Z-Tables would therefore be duplicative and could cause confusion (Ex. 3–891). Other commenters (Exc. 3–593, 3–1095, 8–16 and 8–47) favored the addition of a cancer designation to carcinogenic substances included in the Z Tables. For examples, the American Industrial Hygiene Association (AIHA) stated:

> AIHA would support the inclusion of a designation on carcinogenicity . . . provided that such designation reflects the weight of evidence for carcinogenic effects . . . . (Ex. 8–16, p. 14).

NIOSH (Ex. 8–47) concurred in recommending the inclusion of such a designation in the final rule's Z-Tables.

OSHA has carefully reviewed the record evidence on this issue and has investigated the various evaluative criteria used by scientific and regulatory bodies to determine the classification of a substance as a carcinogen. The Agency notes that each organization has a different system and that the criteria used rarely coincide. Thus, the ACGIH uses two designations, A1 and A2, to reflect the strength of the evidence for a substance's carcinogenicity while the EPA has 5 classifications that represent different kinds of evidence. OSHA believes that the inclusion of a cancer designation on the Z-Tables would further complicate this already complex situation by adding yet another classification system to those already in use. OSHA is also concerned that adding cancer designation to the Z-Table limits would require frequent updating and revision as additional substances are identified as carcinogens in the future. Therefore, OSHA has determined not to add a cancer designation to the Tables.

Paragraphs 1910.1000 (a)(3), (a)(4) and (a)(5) are new. Paragraph (a)(3) requires an employer to maintain an employee's exposure below the Time Weighted Average (TWA), Short Term Exposure Limit (STEL) and/or Ceiling specified in the Final Rule Limits Columns of Table Z–1–A. Paragraph (a)(5) defines those limits. The language of these two paragraphs is consistent with OSHA past practices and good industrial hygiene. The record of this rulemaking supports the approach taken to the language. OSHA intends this language to be interpreted consistent with similar language in 1910.1000 (a)(1), (a)(2), (b) and (c).

Paragraph (a)(4) puts limits on skin exposure. It states:

> *Skin Designation.* To prevent or reduce skin absorption, an employee's skin exposure to substances listed in Table Z–1–A with an X in the skin column under the Final Rule Limits column shall be prevented or reduced to the extent necessary under the circumstances through the use of gloves, coveralls, goggles or other appropriate personal protective equipment, engineering controls or work practices.

This reflects both format and substantive changes from the language proposed. This preamble discussion also reflects a substantive change from the discussion in the proposal. The substantive changes are in response to many comments in the record.

The ACGIH gave skin designation to substances which could be absorbed through the skin. The proposal preamble stated that the skin notation was used to indicate both substances absorbed through the skin and those which might cause skin irritation. There was much public comment pointing out that the underlying documentation considered only skin absorption and not skin irritation. It also pointed out that the two concepts should not be confused because a substance that could be absorbed might not irritate, and conversely.

OSHA agrees with these comments and their reasoning. Accordingly a skin designation for the final rule is only given to a substance which may be absorbed through the skin.

The use of skin designation does not indicate that the substance may irritate the skin. Similarly, lack of a skin designation does not mean that the substance will not irritate the skin.

The purpose of having the skin designation is to prevent the same toxic effects that the chemical causes through inhalation. The inhalation limit is based on keeping exposure below the limit which will create a significant risk of material impairment of health. If skin absorption is possible, an employee might be below the inhalation limit; however, the additional body burden through skin absorption may create the material impairment which the limit attempts to reduce.

The revised language permits compliance with personal protective equipment such as gloves, goggles and coveralls, engineering controls or work practices. No specific hierarchy is stated. An employer must take appropriate actions to prevent routine or regular exposures. However, except when there is the reasonable possibility of slight, a severe reaction through absorption, the methods need not be such as to prevent the possibility of slight infrequent exposure. This language reflects comments in the record that preventing the possibility of exposure is not always necessary to prevent material impairment of health.

Many existing substances have a skin designation which is indicated in the Transitional Limits columns. Para. (f)(2)(iii) states that they shall remain in effect until August 31, 1989. The skin designations in the Final Rule Limits

columns take effect on September 1, 1989. This is sufficient time for employers to institute control practices.

Para. (f)(3)(iii) states that if any of the revised limits are stayed then the limits existing prior to this final rule remain effective until the stay is lifted. If a revised limit is vacated, then the limit existing prior to this final rule remains effective.

Para. (f)(4) stays the enforcement of PELs for seven substances for which OSHA is not aware of a practical sampling and analytic technique as of November 10, 1988, the close of the record. When a suitable method becomes available OSHA will publish a notice in the **Federal Register** notifying the public of the method and setting a date ending the stay.

E. State Plan Applicability

The 25 states with their own OSHA-approved occupational safety and health plans must adopt a comparable standard within six months of the publication date of this final standard. These States include: Alaska, Arizona, California, Connecticut, (for State and local government employees only), Hawaii, Indiana, Iowa, Kentucky, Maryland, Michigan, Minnesota, Nevada, New Mexico, New York, (for state and local government employees only), North Carolina, Oregon, Puerto Rico, South Carolina, Tennessee, Utah, Vermont, Virginia, Virgin Islands, Washington, Wyoming. Until such time as a State Standard is promulgated, Federal OSHA will provide interim enforcement assistance, as appropriate.

List of Subjects in 29 CFR Part 1910

Air contaminants, Occupational safety and health, Permissible exposure limits, Health, Risk assessment.

IX. Authority: This document has been prepared under the direction of John A. Pendergrass, Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, 200 Constitution Avenue, NW., Washington, DC 20210. Pursuant to Section 6 of the Occupational Safety and Health Act of 1970 (29 U.S.C. 655), Section 4 of the Administrative Procedures Act (5 U.S.C. 553), 29 CFR Part 1911 and Secretary of Labor's Order 9–83 (48 FR 35736), it is proposed to amend 29 CFR Part 1910 by revising § 1910.1000 as set forth below.

Signed at Washington, DC, this 11th day of January 1989.

John A. Pendergrass,

Assistant Secretary of Labor.

X. Standard

OSHA is amending Part 1910 of Title 29 of the Code of Federal Regulations as follows:

PART 1910 [AMENDED]

1. The authority citation for Subpart Z of Part 1910 is revised to read as follows:

Authority: Secs. 6, 8. Occupational Safety and Health Act, 29 U.S.C. 655, 657; Secretary of Labor's Orders 12–71 (36 FR 8754), 8–76 (41 FR 25059), or 9–83 (48 FR 35736) as applicable; and 29 CFR Part 1911.

All of Subpart Z issued under Sec 6(b) of the Occupational Safety and Health Act, 29 U.S.C. 655(b) except those substances listed in the Final Rule Limits columns of Table Z–1–A, which have identical limits listed in the Transitional Limits columns of Table Z–1–A, Table Z–2 or Table Z–3. The latter were issued under Sec. 6(a) (5 U.S.C. 655 (a)).

Section 1910.1000, the Transitional Limits columns of Table Z–1–A, Table Z–2 and Table Z–3 also issued under 5 U.S.C. 533. Section 1910.1000, Tables Z–1–A, Z–2 and Z–3 not issued under 29 CFR 1911 except for the arsenic, benzene, cotton dust, and formaldehyde listings.

Section 1910.1001 also issued under Sec. 107 of Contract Work Hours and Safety Standards Act, 40 U.S.C. 333.

Section 1910.1002 not issued under 29 U.S.C. 655 or 29 CFR Part 1911; also issued under 5 U.S.C. 553.

Sections 1910.1003 through 1910.1018 also issued under 29 U.S.C. 653.

Section 1910.1025 also issued under 29 U.S.C. 653 and 5 U.S.C. 553.

Section 1910.1028 also issued under 29 U.S.C. 653.

Section 1910.1043 also issued under 5 U.S.C. 551 et seq.

Sections 1910.1045 and 1910.1047 also issued under 29 U.S.C. 653.

Section 1910.1048 also issued under 29 U.S.C. 653.

Sections 1910.1200, 1910.1499 and 1910.1500 also issued under 5 U.S.C. 553.

2. Section 1910.1000 is amended by revising the introductory text and paragraphs (a) through (d), republishing paragraph (e), adding a new paragraph (f), removing Table Z–1 and adding Table Z–1–A, and republishing Tables Z–2 and Z–3. As revised, § 1910.1000 reads as follows:

§ 1910.1000 Air contaminants.

An employees's exposure to any substance listed in Tables Z–1–A, Z–2 or Z–3 of this section shall be limited in accordance with the requirements of the following paragraphs of this section.

(a) *Table Z–1–A*—(1) *Substances in Transitional Limits Columns with limits preceded by "C"—Ceiling Values.* An employee's exposure to any substance in Table Z–1–A under the Transitional Limits columns, the exposure limit of which is preceded by a "C", shall at no time exceed the exposure limit given for that substance in Table Z–1–A under the Transitional Limits columns.

(2) *Other Substances in Transitional Limits Columns—8-hour Time Weighted Average.* An employee's exposure to any substance in Table Z–1–A under the Transitional Limits columns, the exposure limit of which is not preceded by a "C", shall not exceed the 8-hour Time Weighted Average given for that substance in Table Z–1–A under the Transitional Limits columns in any 8-hour work shift of a 40-hour work week.

(3) *Final Rule Limits Columns.* An employee's exposure to any substance listed in Table Z–1–A shall not exceed the Time Weighted Average (TWA), Short Term Exposure Limit (STEL) and Ceiling Limit specified for that substance in Table Z–1–A under the Revised Limits columns.

(4) *Skin Designation.* To prevent or reduce skin absorption, an employee's skin exposure to substances listed in Table Z–1–A with an "X" in one or both of the Skin Designation columns following the substance name shall be prevented or reduced to the extent necessary in the circumstances through the use of gloves, coveralls, goggles, or other appropriate personal protective equipment, engineering controls or work practices.

(5) *Definitions.* The following definitions are applicable to the Final Rule Limits columns of Table Z–1–A:

(i) Time weighted average (TWA) is the employee's average airborne exposure in any 8-hour work shift of a 40-hour work week which shall not be exceeded.

(ii) Short term exposure limit (STEL) is the employee's 15-minute time weighted average exposure which shall not be exceeded at any time during a work day unless another time limit is specified in a parenthetical notation below the limit. If another time period is specified, the time weighted average exposure over that time period shall not be exceeded at any time during the working day.

(iii) Ceiling is the employee's exposure which shall not be exceeded during any part of the work day. If instantaneous monitoring is not feasible, then the ceiling shall be assessed as a 15-minute time weighted average exposure which shall not be exceeded at any time over a working day.

(6) *Additional Definition.* The terms "substance", "air contaminant," and "material" are equivalent in meaning for 29 CFR 1910.1000.

(b) *Table Z-2.* Table Z-2 is applicable for the transitional period and to the extent set forth in paragraph (f) of this section.

(1) *8-hour time weighted averages.* An employee's exposure to any material listed in table Z-2, in any 8-hour work shift of a 40-hour work week, shall not exceed the 8-hour time weighted average limit given for that material in Table Z-2.

(2) *Acceptable ceiling concentrations.* An employee's exposure to a material listed in table Z-2 shall not exceed at any time during an 8-hour shift the acceptable ceiling concentration limit given for the material in the table, except for a time period, and up to a concentration not exceeding the maximum duration and concentration allowed in the column under "acceptable maximum peak above the ceiling concentration for an 8-hour shift."

(3) *Example.* During an 8-hour work shift, an employee may be exposed to a concentration of Substance A (with a 10 ppm TWA, 25 ppm ceiling and 50 ppm peak) above 25 ppm (but never above 50 ppm) only for a maximum period of 10 minutes. Such exposure must be compensated for by exposures to concentrations less than 10 ppm so that the cumulative exposure for the entire 8-hour work shift does not exceed a weighted average of 10 ppm.

(c) *Table Z-3.* Table Z-3 is applicable for the transitional period and to the extent set forth in paragraph (f) of this section. An employee's exposure to any substance listed in Table Z-3 in any 8-hour work shift of a 40-hour work week shall not exceed the 8-hour time weighted average limit given for that substance in the table.

(d) *Computation formulae.* The computation formula which shall apply to employee exposure to more than one substance for which 8-hour time weighted averages are listed in subpart Z of 29 CFR Part 1910 in order to determine whether an employee is exposed over the regulatory limit is as follows:

(1)(i) The cumulative exposure for an 8-hour work shift shall be computed as follows:

$$E=(C_aT_a+C_bT_b+...C_nT_n)\div 8$$

Where:

E is the equivalent exposure for the working shift.

C is the concentration during any period of time T where the concentration remains constant.

T is the duration in hours of the exposure at the concentration C.

The value of E shall not exceed the 8-hour time weighted average specified in Subpart Z or 29 CFR Part 1910 for the material involved.

(ii) To illustrate the formula prescribed in paragraph (d)(1)(i) of this section, assume that Substance A has an 8-hour time weighted average limit of 100 ppm noted in Table Z-1-A. Assume that an employee is subject to the following exposure:

Two hours exposure at 150 p/m
Two hours exposure at 75 p/m
Four hours exposure at 50 p/m

Substituting this information in the formula, we have

$$(2\times 150+2\times 75+4\times 50)\div 8=81.25 \text{ p/m}$$

Since 81.25 ppm is less than 100 p.p.m., the 8-hour time weighted average limit, the exposure is acceptable.

(2)(i) in case of a mixture of air contaminants an employer shall compute the equivalent exposure as follows:

$$E_m=(C_1\div L_1+C_2\div L_2)+...(C_n\div L_n)$$

Where:

E_m is the equivalent exposure for the mixture.

C is the concentration of a particular contaminant.

L is the exposure limit for that substance specified in Subpart Z of 29 CFR Part 1910.

The value of E_m shall not exceed unity (1).

(ii) To illustrate the formula prescribed in paragraph (d)(2)(i) of this section, consider the following exposures:

Substance	Actual concentration of 8 hour exposure (ppm)	8 hr. TWA PEL (ppm) 8
B	500	1000
C	45	200
D	40	200

Substituting in the formula, we have:
$Em=500\div 1,000+45\div 200+40\div 200$
$Em=0.500+0.225+0.200$
$Em=0.925$

Since Em is less than unity (1), the exposure combination is within acceptable limits.

(e) To achieve compliance with paragraphs (a) through (d) of this section, administrative or engineering controls must first be determined and implemented whenever feasible. When such controls are not feasible to achieve full compliance, protective equipment or any other protective measures shall be used to keep the exposure of employees to air contaminants within the limits prescribed in this section. Any equipment and/or technical measures used for this purpose must be approved for each particular use by a competent industrial hygienist or other technically qualified person. Whenever respirators are used, their use shall comply with § 1910.134.

(f) *Effective dates, start-up dates and transitional provisions*—(1) *Effective date.* The effective date for the permissible exposure limits specified in the Final Rule Limits columns of Table Z-1-A is March 1, 1989.

(2) *Start-up dates.* (i) The permissible exposure limits specified in the Final Rule Limits columns of Table Z-1-A shall be achieved by any reasonable combination of engineering controls, work practices and personal protective equipment effective September 1, 1989, through December 30, 1992.

(ii)(A) The permissible exposure limits specified in the Final Rule Limits columns of Table Z-1-A shall be achieved by the method of compliance specified in paragraph (e) of this section effective December 31, 1992, if by December 31, 1991 a final rule has been published in the **Federal Register** amending or determining not to amend paragraph (e) of this section.

(B) If no final rule has been published in the **Federal Register** by December 31, 1991, amending or determining not to amend paragraph (e) of this section, then the permissible limits specified in the Final Rule Limits columns of Table Z-1-A shall be achieved by the methods of compliance specified by paragraph (e) of this section effective December 31, 1993, and paragraph (f)(2)(i) of this section shall remain in effect through December 30, 1993.

(iii) The skin designations in the Final Rule Limits columns become effective September 1, 1989. The skin designations in the Transitional Limits columns are in effect from March 1, 1989, through August 31, 1989.

(3) *Transitional provisions.* (i) The permissible exposure limits specified in the Transitional Limits columns of Table Z-1-A, Table Z-2 and Table Z-3 shall continue to be achieved by the methods of compliance specified in paragraph (e) of this section through December 30, 1992. If paragraph (f)(2)(ii)(B) of this section takes effect, this provision is extended through December 30, 1993.

(ii) The permissible exposure limits specified in the Transitional Limits columns of Table Z-1-A, Z-2 and Z-3 shall be applicable to the extent cross referenced in 29 CFR Parts 1915, 1917 and 1918.

(iii) If any new or amended provisions or new or revised limits for any substance or substances are either administratively stayed or judicially stayed or vacated, then the existing provisions or limits for those substances specified in the Transitional Limits columns of Table Z-1-A, Table Z-2 or

Table Z-3 shall remain in effect until such stay is lifted, or indefinitely, if the limit is vacated.

(4) Enforcement of the limits are indefinitely stayed for: aluminum alkyls; ethylidene norbornene; hexafluoracetone; mercury (alkyl compounds); oxygen difluoride; phenylphosphine; and sulfur pentafluoride; until OSHA publishes in the **Federal Register** a notice that a sampling and analytical technique is available.

BILLING CODE 4510-26-M

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	CEILING Skin Designation
Acetaldehyde	75-07-0	200	360	-	100	180	150	270	-	-	-
Acetic acid	64-19-7	10	25	-	10	25	-	-	-	-	-
Acetic anhydride	108-24-7	5	20	-	-	-	-	-	5	20	-
Acetone	67-64-1	1000	2400	-	750	1800	1000	2400	-	-	-
Acetonitrile	75-05-8	40	70	-	40	70	60	105	-	-	-
2-Acetylaminofluorine; see 1910.1014	53-96-3										
Acetylene dichloride; see 1,2-Dichloroethylene											
Acetylene tetrabromide	79-27-6	1	14	-	1	14	-	-	-	-	-
Acetylsalicylic acid (Aspirin)	50-78-2	-	-	-	-	5	-	-	-	-	-
Acrolein	107-02-8	0.1	0.25	-	0.1	0.25	0.3	0.8	-	-	-
Acrylamide	79-06-1	-	0.3	X	-	0.03	-	-	-	-	X
Acrylic acid	79-10-7	-	-	-	10	30	-	-	-	-	X
Acrylonitrile; see 1910.1045	107-13-1										
Aldrin	309-00-2	-	0.25	X	-	0.25	-	-	-	-	X
Allyl alcohol	107-18-6	2	5	X	2	5	4	10	-	-	X
Allyl chloride	107-05-1	1	3	-	1	3	2	6	-	-	-
Allyl glycidyl ether (AGE)	106-92-3	(C)10	(C)45	-	5	22	10	44	-	-	-
Allyl propyl disulfide	2179-59-1	2	12	-	2	12	3	18	-	-	-
alpha-Alumina	1344-28-1										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits PEL* Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	CEILING Skin Designation
Aluminum (as Al) Metal	7429-90-5										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Pyro powders		-	-	-	-	5	-	-	-	-	-
Welding fumes***		-	-	-	-	5	-	-	-	-	-
Soluble salts		-	-	-	-	2	-	-	-	-	-
Alkyls		-	-	-	-	2	-	-	-	-	-
4-Aminodiphenyl; see 1910.1011	92-67-1										
2-Aminoethanol; see Ethanolamine											
2-Aminopyridine	504-29-0	0.5	2	-	0.5	2	-	-	-	-	-
Amitrole	61-82-5	-	-	-	-	0.2	-	-	-	-	-
Ammonia	7664-41-7	50	35	-	-	-	35	27	-	-	-
Ammonium chloride fume	12125-02-9	-	-	-	-	10	-	20	-	-	-
Ammonium sulfamate	7773-06-0										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
n-Amyl acetate	628-63-7	100	525	-	100	525	-	-	-	-	-
sec-Amyl acetate	626-38-0	125	650	-	125	650	-	-	-	-	-
Aniline and homologs	62-53-3	5	19	X	2	8	-	-	-	-	X
Anisidine (o-,p-isomers)	29191-52-4	-	0.5	-	-	0.5	-	-	-	-	-
Antimony and compounds (as Sb)	7440-36-0	-	0.5	-	-	0.5	-	-	-	-	-
ANTU (Alpha naphthyl-thiourea)	86-88-4	-	0.3	-	-	0.3	-	-	-	-	-
Arsenic, organic compounds (as As)	7440-38-2	-	0.5	-	-	0.5	-	-	-	-	-
Arsenic, inorganic compounds (as As); see 1910.1018	Varies with compound	See 1910.1018(a) for applications excluded									

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Arsine	7784-42-1	0.05	0.2	-	0.05	0.2	-	-	-		-
Asbestos; see 1910.1001	Varies										
Atrazine	1912-24-9	-	-	-	-	5	-	-	-	-	-
Azinphos-methyl	86-50-0	-	0.2	X	-	0.2	-	-	-	-	X
Barium, soluble compounds (as Ba)	7440-39-3	-	0.5	-	-	0.5	-	-	-	-	-
Barium sulfate	7727-43-7										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Benomyl	17804-35-2										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Benzene; see 1910.1028	71-43-2	See Table Z-2 for the limits applicable in the operations or sectors excluded in 1910.1028[d]									
Benzidine; see 1910.1010	92-87-5										
p-Benzoquinone; see Quinone											
Benzo(a)pyrene; see Coal tar pitch volatiles											
Benzoyl peroxide	94-36-0	-	5	-	-	5	-	-	-	-	-
Benzyl chloride	100-44-7	1	5	-	1	5	-	-	-	-	-
Beryllium and beryllium compounds (as Be)	7440-41-7	See Table Z-2			0.002	-	0.005 (30 min)	-	0.025	-	-
Biphenyl; see Diphenyl											
Bismuth telluride, Undoped	1304-82-1										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Bismuth telluride, Se-doped		-	-	-	-	5	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	CEILING Skin Designation
Borates, tetra, sodium salts											
Anhydrous	1330-43-4	-	-	-	-	10	-	-	-	-	-
Decahydrate	1303-96-4	-	-	-	-	10	-	-	-	-	-
Pentahydrate	12179-04-3	-	-	-	-	10	-	-	-	-	-
Boron oxide	1303-86-2										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Boron tribromide	10294-33-4	-	-	-	-	-	-	-	1	10	-
Boron trifluoride	7637-07-2	(C)1	(C)3	-	-	-	-	-	1	3	-
Bromacil	314-40-9	-	-	-	1	10	-	-	-	-	-
Bromine	7726-95-6	0.1	0.7	-	0.1	0.7	0.3	2	-	-	-
Bromine pentafluoride	7789-30-2	-	-	-	0.1	0.7	-	-	-	-	-
Bromoform	75-25-2	0.5	5	X	0.5	5	-	-	-	-	X
Butadiene (1,3-Butadiene)	106-99-0	1000	2200	-	In process of 6(b) rulemaking						
Butane	106-97-8	-	-	-	800	1900	-	-	-	-	-
Butanethiol; see Butyl mercaptan											
2-Butanone (Methyl ethyl ketone)	78-93-3	200	590	-	200	590	300	885	-	-	-
2-Butoxyethanol	111-76-2	50	240	X	25	120	-	-	-	-	X
n-Butyl-acetate	123-86-4	150	710	-	150	710	200	950	-	-	-
sec-Butyl acetate	105-46-4	200	950	-	200	950	-	-	-	-	-
tert-Butyl acetate	540-88-5	200	950	-	200	950	-	-	-	-	-
Butyl acrylate	141-32-2	-	-	-	10	55	-	-	-	-	-
n-Butyl alcohol	71-36-3	100	300	-	-	-	-	-	50	150	X
sec-Butyl alcohol	78-92-2	150	450	-	100	305	-	-	-	-	-
tert-Butyl alcohol	75-65-0	100	300	-	100	300	150	450	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Butylamine	109-73-9	(C)5	(C)15	X	-	-	-	-	5	15	X
tert-Butyl chromate (as CrO_3)	1189-85-1	-	(C)0.1	X	-	-	-	-	-	0.1	X
n-Butyl glycidyl ether (BGE)	2426-08-6	50	270	-	25	135	-	-	-	-	-
n-Butyl lactate	138-22-7	-	-	-	5	25	-	-	-	-	-
Butyl mercaptan	109-79-5	10	35	-	0.5	1.5	-	-	-	-	-
o-sec-Butylphenol	89-72-5	-	-	-	5	30	-	-	-	-	X
p-tert-Butyltoluene	98-51-1	10	60	-	10	60	20	120	-	-	-
Cadmium fume (as Cd)	7440-43-9	See Table Z-2			In process of 6(b) rulemaking						
Cadmium dust (as Cd)	7440-43-9	See Table Z-2			In process of 6(b) rulemaking						
Calcium carbonate	1317-65-3										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Calcium cyanamide	156-62-7	-	-	-	-	0.5	-	-	-	-	-
Calcium hydroxide	1305-62-0	-	-	-	-	5	-	-	-	-	-
Calcium oxide	1305-78-8	-	5	-	-	5	-	-	-	-	-
Calcium silicate	1344-95-2										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Calcium sulfate	7778-18-9										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Camphor, synthetic	76-22-2	-	2	-	-	2	-	-	-	-	-
Caprolactam	105-60-2										
Dust		-	-	-	-	1	-	3	-	-	-
Vapor		-	-	-	5	20	10	40	-	-	-
Captafol (Difolatan®)	2425-06-1	-	-	-	-	0.1	-	-	-	-	-
Captan	133-06-2	-	-	-	-	5	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m^3 [b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m^3 [b]	STEL[c] ppm[a]	STEL mg/m^3 [b]	CEILING ppm[a]	CEILING mg/m^3 [b]	Final Rule Limits Skin Designation
Carbaryl (Sevin[R])	63-25-2	-	5	-	-	5	-	-	-	-	-
Carbofuran (Furadan[R])	1563-66-2	-	-	-	-	0.1	-	-	-	-	-
Carbon black	1333-86-4	-	3.5	-	-	3.5	-	-	-	-	-
Carbon dioxide	124-38-9	5000	9000	-	10,000	18,000	30,000	54,000	-	-	-
Carbon disulfide	75-15-0		See Table Z-2		4	12	12	36	-	-	X
Carbon monoxide	630-08-0	50	55	-	35	40	-	-	200	229	-
Carbon tetrabromide	558-13-4	-	-	-	0.1	1.4	0.3	4	-	-	-
Carbon tetrachloride	56-23-5		See Table Z-2		2	12.6	-	-	-	-	-
Carbonyl fluoride	353-50-4	-	-	-	2	5	5	15	-	-	-
Catechol (Pyrocatechol)	120-80-9	-	-	-	5	20	-	-	-	-	X
Cellulose	9004-34-6										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Cesium hydroxide	21351-79-1	-	-	-	-	2	-	-	-	-	-
Chlordane	57-74-9	-	0.5	X	-	0.5	-	-	-	-	X
Chlorinated camphene	8001-35-2	-	0.5	X	-	0.5	-	1	-	-	X
Chlorinated diphenyl oxide	55720-99-5	-	0.5	-	-	0.5	-	-	-	-	-
Chlorine	7782-50-5	(C)1	(C)3	-	0.5	1.5	1	3	-	-	-
Chlorine dioxide	10049-04-4	0.1	0.3	-	0.1	0.3	0.3	0.9	-	-	-
Chlorine trifluoride	7790-91-2	(C)0.1	(C)0.4	-	-	-	-	-	0.1	0.4	-
Chloroacetaldehyde	107-20-0	(C)1	(C)3	-	-	-	-	-	1	3	-
a-Chloroacetophenone (Phenacyl chloride)	532-27-4	0.05	0.3	-	0.05	0.3	-	-	-	-	-
Chloroacetyl chloride	79-04-9	-	-	-	0.05	0.2	-	-	-	-	-
Chlorobenzene	108-90-7	75	350	-	75	350	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³ [b]	Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³ [b]	STEL[c] ppm[a]	STEL mg/m³ [b]	CEILING ppm[a]	CEILING mg/m³ [b]	Skin Designation
o-Chlorobenzylidene malononitrile	2698-41-1	0.05	0.4	-	-	-	-	-	0.05	0.4	X
Chlorobromomethane	74-97-5	200	1050	-	200	1050	-	-	-	-	-
2-Chloro-1,3-butadiene; see b-Chloroprene											
Chlorodifluoromethane	75-45-6	-	-	-	1000	3500	-	-	-	-	-
Chlorodiphenyl (42% Chlorine) (PCB)	53469-21-9	-	1	X	-	1	-	-	-	-	X
Chlorodiphenyl (54% Chlorine) (PCB)	11097-69-1	-	0.5	X	-	0.5	-	-	-	-	X
1-Chloro,2,3-epoxypropane; see Epichlorohydrin											
2-Chloroethanol; see Ethylene chlorohydrin											
Chloroethylene; see Vinyl chloride											
Chloroform (Trichloromethane)	67-66-3	(C)50	(C)240	-	2	9.78	-	-	-	-	-
bis(Chloromethyl) ether; see 1910.1008	542-88-1										
Chloromethyl methyl ether; see 1910.1006	107-30-2										
1-Chloro-1-nitropropane	600-25-9	20	100	-	2	10	-	-	-	-	-
Chloropentafluoro-ethane	76-15-3	-	-	-	1000	6320	-	-	-	-	-
Chloropicrin	76-06-2	0.1	0.7	-	0.1	0.7	-	-	-	-	-
beta-Chloroprene	126-99-8	25	90	X	10	35	-	-	-	-	X
o-Chlorostyrene	2039-87-4	-	-	-	50	285	75	430	-	-	-
o-Chlorotoluene	95-49-8	-	-	-	50	250	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
2-Chloro-6-trichloromethyl pyridine	1929-82-4										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Chlorpyrifos	2921-88-2	-	-	-	-	0.2	-	-	-	-	X
Chromic acid and chromates (as CrO_3)	Varies with compound		See Table Z-2		-	-	-	-	0.1	-	-
Chromium (II) compounds (as Cr)	Varies with compound	-	0.5	-	-	0.5	-	-	-	-	-
Chromium (III) compounds (as Cr)	Varies with compound	-	0.5	-	-	0.5	-	-	-	-	-
Chromium metal (as Cr)	7440-47-3	-	1	-	-	1	-	-	-	-	-
Chrysene; see Coal tar pitch volatiles	218-01-9										
Clopidol	2971-90-6										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Coal dust (less than 5% SiO_2), Respirable quartz fraction	—		See Table Z-3			2	-	-	-	-	-
Coal dust (greater than or equal to 5% SiO_2), Respirable quartz fraction	—		See Table Z-3			0.1	-	-	-	-	-
Coal tar pitch volatiles (benzene soluble fraction), anthracene, BaP, phenanthrene, acridine, chrysene, pyrene	8007-45-2	-	0.2	-	-	0.2	-	-	-	-	-
Cobalt metal, dust, and fume (as Co)	7440-48-4	-	0.1	-	-	0.05	-	-	-	-	-

TABLE Z-1-A. Limits for Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Cobalt carbonyl (as Co)	10210-68-1	-	-	-	-	0.1	-	-	-	-	-
Cobalt hydrocarbonyl (as Co)	16842-03-8	-	-	-	-	0.1	-	-	-	-	-
Coke oven emissions; see 1910.1029	†										
Copper	7440-50-8										
Fume (as Cu)		-	0.1	-	-	0.1	-	-	-	-	-
Dusts and mists (as Cu)		-	1	-	-	1	-	-	-	-	-
Cotton dust (raw),	--	1	-		1	-	-	-	-	-	-
This 8-hour TWA applies to respirable dust as measured by a vertical elutriator cotton dust sampler or equivalent instrument. The time-weighted average applies to the cotton waste processing operations of waste recycling (sorting, blending, cleaning, and willowing) and garretting. See also 1910.1043 for cotton dust limits applicable to other sectors.											
Crag herbicide (Sesone)	136-78-7										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Cresol, all isomers	1319-77-3; 95-48-7; 108-39-4; 106-44-5	5	22	X	5	22	-	-	-	-	X
Crotonaldehyde	123-73-9; 4170-30-3	2	6	-	2	6	-	-	-	-	-
Crufomate	299-86-5	-	-	-	-	5	-	-	-	-	-
Cumene	98-82-8	50	245	X	50	245	-	-	-	-	X
Cyanamide	420-04-2	-	-	-	-	2	-	-	-	-	-
Cyanides (as CN)	151-50-8	-	5	-	-	5	-	-	-	-	-
Cyanogen	460-19-5	-	-	-	10	20	-	-	-	-	-
Cyanogen chloride	506-77-4	-	-	-	-	-	-	-	0.3	0.6	-
Cyclohexane	110-82-7	300	1050	-	300	1050	-	-	-	-	-
Cyclohexanol	108-93-0	50	200	-	50	200	-	-	-	-	X

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Cyclohexanone	108-94-1	50	200	-	25	100	-	-	-	-	X
Cyclohexene	110-83-8	300	1015	-	300	1015	-	-	-	-	-
Cyclohexylamine	108-91-8	-	-	-	10	40	-	-	-	-	-
Cyclonite	121-82-4	-	-	-	-	1.5	-	-	-	-	X
Cyclopentadiene	542-92-7	75	200	-	75	200	-	-	-	-	-
Cyclopentane	287-92-3	-	-	-	600	1720	-	-	-	-	-
Cyhexatin	13121-70-5	-	-	-	-	5	-	-	-	-	-
2,4-D (Dichlorylphenoxy-acetic acid)	94-75-7	-	10	-	-	10	-	-	-	-	-
Decaborane	17702-41-9	0.05	0.3	X	0.05	0.3	0.15	0.9	-	-	X
Demeton (Systox[R])	8065-48-3	-	0.1	X	-	0.1	-	-	-	-	X
Dichlorodiphenyltri-chloroethane (DDT)	50-29-3	-	1	X	-	1	-	-	-	-	X
Dichlorvos (DDVP)	62-73-7	-	1	X	-	1	-	-	-	-	X
Diacetone alcohol (4-Hydroxy-4-methyl-2-pentanone)	123-42-2	50	240	-	50	240	-	-	-	-	-
1,2-Diaminoethane; see Ethylenediamine											
Diazinon	333-41-5	-	-	-	-	0.1	-	-	-	-	X
Diazomethane	334-88-3	0.2	0.4	-	0.2	0.4	-	-	-	-	-
Diborane	19287-45-7	0.1	0.1	-	0.1	0.1	-	-	-	-	-
1,2-Dibromo-3-chloropropane; see 1910.1044	96-12-8										
2-N-Dibutylamino-ethanol	102-81-8	-	-	-	2	14	-	-	-		
Dibutyl phosphate	107-66-4	1	5	-	1	5	2	10	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Dibutyl phthalate	84-74-2	-	5	-	-	5	-	-	-	-	-
Dichloroacetylene	7572-29-4	-	-	-	-	-	-	-	0.1	0.4	-
o-Dichlorobenzene	95-50-1	(C)50	(C)300	-	-	-	-	-	50	300	-
p-Dichlorobenzene	106-46-7	75	450	-	75	450	110	675	-	-	-
3,3'-Dichlorobenzidine; see 1910.1007	91-94-1										
Dichlorodifluoromethane	75-71-8	1000	4950	-	1000	4950	-	-	-	-	-
1,3-Dichloro-5,5-dimethyl hydantoin	118-52-5	-	0.2	-	-	0.2	-	0.4	-	-	-
1,1-Dichloroethane	75-34-3	100	400	-	100	400	-	-	-	-	-
1,2-Dichloroethylene	540-59-0	200	790	-	200	790	-	-	-	-	-
Dichloroethyl ether	111-44-4	(C)15	(C)90	X	5	30	10	60	-	-	X
Dichloromethane; see Methylene chloride											
Dichloromonofluoromethane	75-43-4	1000	4200	-	10	40	-	-	-	-	-
1,1-Dichloro-1-nitroethane	594-72-9	(C)10	(C)60	-	2	10	-	-	-	-	-
1,2-Dichloropropane; see Propylene dichloride											
1,3-Dichloropropene	542-75-6	-	-	-	1	5	-	-	-	-	X
2,2-Dichloropropionic acid	75-99-0	-	-	-	1	6	-	-	-	-	-
Dichlorotetrafluoroethane	76-14-2	1000	7000	-	1000	7000	-	-	-	-	-
Dicrotophos	141-66-2	-	-	-	-	0.25	-	-	-	-	X
Dicyclopentadiene	77-73-6	-	-	-	5	30	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Dicyclopentadienyl iron	102-54-5										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Dieldrin	60-57-1	-	0.25	X	-	0.25	-	-	-	-	X
Diethanolamine	111-42-2	-	-	-	3	15	-	-	-	-	-
Diethylamine	109-89-7	25	75	-	10	30	25	75	-	-	-
2-Diethylaminoethanol	100-37-8	10	50	X	10	50	-	-	-	-	X
Diethylene triamine	111-40-0	-	-	-	1	4	-	-	-	-	-
Diethyl ether; see Ethyl ether											
Diethyl ketone	96-22-0	-	-	-	200	705	-	-	-	-	-
Diethyl phthalate	84-66-2	-	-	-	-	5	-	-	-	-	-
Difluorodibromomethane	75-61-6	100	860	-	100	860	-	-	-	-	-
Diglycidyl ether (DGE)	2238-07-5	(C)0.5	(C)2.8	-	0.1	0.5	-	-	-	-	-
Dihydroxybenzene; see Hydroquinone											
Diisobutyl ketone	108-83-8	50	290	-	25	150	-	-	-	-	-
Diisopropylamine	108-18-9	5	20	X	5	20	-	-	-	-	X
4-Dimethylaminoazobenzene; see 1910.1015	60-11-7										
Dimethoxymethane; see Methylal											
Dimethyl acetamide	127-19-5	10	35	X	10	35	-	-	-	-	X
Dimethylamine	124-40-3	10	18	-	10	18	-	-	-	-	-
Dimethylaminobenzene; see Xylidine											

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Dimethylaniline (N-Dimethyl-aniline)	121-69-7	5	25	X	5	25	10	50	-		X
Dimethylbenzene; see Xylene											
Dimethyl-1,2-dibromo-2,2-dichloroethyl phosphate	300-76-5	-	3	-	-	3	-	-	-	-	X
Dimethylformamide	68-12-2	10	30	X	10	30	-	-	-	-	X
2,6-Dimethyl-4-heptanone; see Diisobutyl ketone											
1,1-Dimethylhydrazine	57-14-7	0.5	1	X	0.5	1	-	-	-	-	X
Dimethylphthalate	131-11-3	-	5	-	-	5	-	-	-	-	-
Dimethyl sulfate	77-78-1	1	5	X	0.1	0.5	-	-	-	-	X
Dinitolmide (3,5-Dinitro-o-toluamide)	148-01-6	-	-	-	-	5	-	-	-	-	-
Dinitrobenzene (all isomers)	528-29-0 99-65-0 100-25-4	-	1	X	-	1	-	-	-	-	X
Dinitro-o-cresol	534-52-1	-	0.2	X	-	0.2	-	-	-	-	X
Dinitrotoluene	121-14-2	-	1.5	X	-	1.5	-	-	-	-	X
Dioxane (Diethylene dioxide)	123-91-1	100	360	X	25	90	-	-	-	-	X
Dioxathion (Delnav)	78-34-2	-	-	-	-	0.2	-	-	-	-	X
Diphenyl (Biphenyl)	92-52-4	0.2	1	-	0.2	1	-	-	-	-	-
Diphenylamine	122-39-4	-	-	-	-	10	-	-	-	-	-
Diphenylmethane diisocyanate; see Methylene bisphenyl isocyanate											
Dipropylene glycol methyl ether	34590-94-8	100	600	X	100	600	150	900	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits PEL* Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	CEILING Skin Designation
Dipropyl ketone	123-19-3	-	-	-	50	235	-	-	-	-	-
Diquat	85-00-7	-	-	-	-	0.5	-	-	-	-	-
Di-sec octyl phthalate (Di-2-ethylhexyl-phthalate)	117-81-7	-	5	-	-	5	-	10	-	-	-
Disulfiram	97-77-8	-	-	-	-	2	-	-	-	-	-
Disulfoton	298-04-4	-	-	-	-	0.1	-	-	-	-	X
2,6-Di-tert-butyl-p-cresol	128-37-0	-	-	-	-	10	-	-	-	-	-
Diuron	330-54-1	-	-	-	-	10	-	-	-	-	-
Divinyl benzene	108-57-6	-	-	-	10	50	-	-	-	-	-
Emery	112-62-9										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Endosulfan	115-29-7	-	-	-	-	0.1	-	-	-	-	X
Endrin	72-20-8	-	0.1	X	-	0.1	-	-	-	-	X
Epichlorohydrin	106-89-8	5	19	X	2	8	-	-	-	-	X
EPN	2104-64-5	-	0.5	X	-	0.5	-	-	-	-	X
1,2-Epoxypropane; see Propylene oxide											
2,3-Epoxy-1-propanol; see Glycidol											
Ethanethiol; see Ethyl mercaptan											
Ethanolamine	141-43-5	3	6	-	3	8	6	15	-	-	-
Ethion	563-12-2	-	-	-	-	0.4	-	-	-	-	X
2-Ethoxyethanol	110-80-5	200	740	X	In process of 6(b) rulemaking						
2-Ethoxyethyl acetate (Cellosolve acetate)	111-15-9	100	540	X	In process of 6(b) rulemaking						

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL[c] mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Ethyl acetate	141-78-6	400	1400	-	400	1400	-	-	-	-	-
Ethyl acrylate	140-88-5	25	100	X	5	20	25	100	-	-	X
Ethyl alcohol (Ethanol)	64-17-5	1000	1900	-	1000	1900	-	-	-	-	-
Ethylamine	75-04-7	10	18	-	10	18	-	-	-	-	-
Ethyl amyl ketone (5-Methyl-3-heptanone)	106-68-3	25	130	-	25	130	-	-	-	-	-
Ethyl benzene	100-41-4	100	435	-	100	435	125	545	-	-	-
Ethyl bromide	74-96-4	200	890	-	200	890	250	1110	-	-	-
Ethyl butyl ketone (3-Heptanone)	106-35-4	50	230	-	50	230	-	-	-	-	-
Ethyl chloride	75-00-3	1000	2600	-	1000	2600	-	-	-	-	-
Ethyl ether	60-29-7	400	1200	-	400	1200	500	1500	-	-	-
Ethyl formate	109-94-4	100	300	-	100	300	-	-	-	-	-
Ethyl mercaptan	75-08-1	(C)10	(C)25	-	0.5	1	-	-	-	-	-
Ethyl silicate	78-10-4	100	850	-	10	85	-	-	-	-	-
Ethylene chlorohydrin	107-07-3	5	16	X	-	-	-	-	1	3	X
Ethylenediamine	107-15-3	10	25	-	10	25	-	-	-	-	-
Ethylene dibromide	106-93-4		See Table Z-2		In process of 6(b) rulemaking						
Ethylene dichloride	107-06-2		See Table Z-2		1	4	2	8	-	-	-
Ethylene glycol	107-21-1	-	-	-	-	-	-	-	50	125	-
Ethylene glycol dinitrate	628-96-6	(C)0.2	(C)1	X	-	-	-	0.1	-	-	X
Ethylene glycol methyl acetate; see Methyl cellosolve acetate											
Ethyleneimine; see 1910.1012	151-56-4										

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Final Rule Limits Skin Designation
Ethylene oxide; see 1910.1047	75-21-8	See 1910.1047(a)(2) for operations excluded									
Ethylidene chloride; see 1,1-Dichloroethane											
Ethylidene norbornene	16219-75-3	-	-	-	-	-	-	-	5	25	-
N-Ethylmorpholine	100-74-3	20	94	X	5	23	-	-	-	-	X
Fenamiphos	22224-92-6	-	-	-	-	0.1	-	-	-	-	X
Fensulfothion (Dasanit)	115-90-2	-	-	-	-	0.1	-	-	-	-	-
Fenthion	55-38-9	-	-	-	-	0.2	-	-	-	-	X
Ferbam	14484-64-1										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Ferrovanadium dust	12604-58-9	-	1	-	-	1	-	3	-	-	-
Fluorides (as F)	Varies with compound	See Table Z-2			2.5	-	-	-	-	-	-
Fluorine	7782-41-4	0.1	0.2	-	0.1	0.2	-	-	-	-	-
Fluorotrichloromethane (Trichlorofluoromethane)	75-69-4	1000	5600	-	-	-	-	-	1000	5600	-
Fonofos	944-22-9	-	-	-	-	0.1	-	-	-	-	X
Formaldehyde; see 1910.1048;	50-00-0	See Table Z-2 for operations or sectors excluded or for which limit(s) is(are) stayed.									
Formamide	75-12-7	-	-	-	20	30	30	45	-	-	-
Formic acid	64-18-6	5	9	-	5	9	-	-	-	-	-
Furfural	98-01-1	5	20	X	2	8	-	-	-	-	X
Furfuryl alcohol	98-00-0	50	200	-	10	40	15	60	-	-	X
Gasoline	8006-61-9	-	-	-	300	900	500	1500	-	-	-
Germanium tetrahydride	7782-65-2	-	-	-	0.2	0.6	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL[a] ppm[a]	Transitional Limits PEL[a] mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	CEILING Skin Designation
Glutaraldehyde	111-30-8	-	-	-	-	-	-	-	0.2	0.8	
Glycerin (mist)	56-81-5										
Total dust		-	15	-	-	10	-	-	-	-	
Respirable fraction		-	5	-	-	5	-	-	-	-	
Glycidol	556-52-5	50	150	-	25	75	-	-	-	-	-
Glycol monoethyl ether; see 2-Ethoxyethanol											
Grain dust (oat, wheat, barley)	—	-	-	-	-	10	-	-	-	-	-
Graphite, natural respirable dust	7782-42-5	See Table Z-3			-	2.5	-	-	-	-	-
Graphite, synthetic	—										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Guthion[R]; see Azinphos methyl											
Gypsum	7778-18-9										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Hafnium	7440-58-6	-	0.5	-	-	0.5	-	-	-	-	-
Heptachlor	76-44-8	-	0.5	X	-	0.5	-	-	-	-	X
Heptane (n-Heptane)	142-82-5	500	2000	-	400	1600	500	2000	-	-	-
Hexachlorobutadiene	87-68-3	-	-	-	0.02	0.24	-	-	-	-	-
Hexachlorocyclopentadiene	77-47-4	-	-	-	0.01	0.1	-	-	-	-	-
Hexachloroethane	67-72-1	1	10	X	1	10	-	-	-	-	X
Hexachloronaphthalene	1335-87-1	-	0.2	X	-	0.2	-	-	-	-	X
Hexafluoroacetone	684-16-2	-	-	-	0.1	0.7	-	-	-	-	X
n-Hexane	110-54-3	500	1800	-	50	180	-	-	-	-	-
Hexane isomers	Varies with compound	-	-	-	500	1800	1000	3600	-	-	

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
2-Hexanone (Methyl n-butyl ketone)	591-78-6	100	410	-	5	20	-	-	-	-	-
Hexone (Methyl isobutyl ketone)	108-10-1	100	410	-	50	205	75	300	-	-	-
sec-Hexyl acetate	108-84-9	50	300	-	50	300	-	-	-	-	-
Hexylene glycol	107-41-5	-	-	-	-	-	-	-	25	125	-
Hydrazine	302-01-2	1	1.3	X	0.1	0.1	-	-	-	-	X
Hydrogenated terphenyls	61788-32-7	-	-	-	0.5	5	-	-	-	-	-
Hydrogen bromide	10035-10-6	3	10	-	-	-	-	-	3	10	-
Hydrogen chloride	7647-01-0	(C)5	(C)7	-	-	-	-	-	5	7	-
Hydrogen cyanide	74-90-8	10	11	X	-	-	4.7	5			X
Hydrogen fluoride (as F)	7664-39-3	See Table Z-2		-	3	-	6	-	-	-	-
Hydrogen peroxide	7722-84-1	1	1.4	-	1	1.4	-	-	-	-	-
Hydrogen selenide (as Se)	7783-07-5	0.05	0.2	-	0.05	0.2	-	-	-	-	-
Hydrogen sulfide	7783-06-4	See Table Z-2		-	10	14	15	21	-	-	-
Hydroquinone	123-31-9	-	2	-	-	2	-	-	-	-	-
2-Hydroxypropyl acrylate	999-61-1	-	-	-	0.5	3	-	-	-	-	X
Indene	95-13-6	-	-	-	10	45	-	-	-	-	-
Indium and compounds (as In)	7440-74-6	-	-	-	-	0.1	-	-	-	-	-
Iodine	7553-56-2	(C)0.1	(C)1	-	-	-	-	-	0.1	1	-
Iodoform	75-47-8	-	-	-	0.6	10	-	-	-	-	-
Iron oxide dust and fume (as Fe) Total particulate	1309-37-1	-	10	-	-	10	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³ [b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³ [b]	STEL[c] ppm[a]	STEL mg/m³ [b]	CEILING ppm[a]	CEILING mg/m³ [b]	Skin Designation
Iron pentacarbonyl (as Fe)	13463-40-6	-	-	-	0.1	0.8	0.2	1.6	-	-	-
Iron salts (soluble) (as Fe)	Varies with compound	-	-	-	-	1	-	-	-	-	-
Isoamyl acetate	123-92-2	100	525	-	100	525	-	-	-	-	-
Isoamyl alcohol (primary and secondary)	123-51-3	100	360	-	100	360	125	450	-	-	-
Isobutyl acetate	110-19-0	150	700	-	150	700	-	-	-	-	-
Isobutyl alcohol	78-83-1	100	300	-	50	150	-	-	-	-	-
Isooctyl alcohol	26952-21-6	-	-	-	50	270	-	-	-	-	X
Isophorone	78-59-1	25	140	-	4	23	-	-	-	-	-
Isophorone diisocyanate	4098-71-9	-	-	-	0.005	-	0.02	-	-	-	X
2-Isopropoxyethanol	109-59-1	-	-	-	25	105	-	-	-	-	-
Isopropyl acetate	108-21-4	250	950	-	250	950	310	1185	-	-	-
Isopropyl alcohol	67-63-0	400	980	-	400	980	500	1225	-	-	-
Isopropylamine	75-31-0	5	12	-	5	12	10	24	-	-	-
N-Isopropylaniline	768-52-5	-	-	-	2	10	-	-	-	-	X
Isopropyl ether	108-20-3	500	2100	-	500	2100	-	-	-	-	-
Isopropyl glycidyl ether (IGE)	4016-14-2	50	240	-	50	240	75	360	-	-	-
Kaolin	—										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Ketene	463-51-4	0.5	0.9	-	0.5	0.9	1.5	3	-	-	-
Lead inorganic (as Pb); see 1910.1025	7439-92-1	For independent battery breaking, non-ferrous foundries, secondary copper, lead pigments, lead chemical, ship building, stevedoring, and brass and bronze ingot manufacturing, paragraph (e)(1) is under court remand									

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Limestone	1317-67-3										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Lindane	58-89-9	-	0.5	X	-	0.5	-	-	-	-	X
Lithium hydride	7580-67-8	-	0.025	-	-	0.025	-	-	-	-	-
L.P.G. (Liquefied petroleum gas)	68476-85-7	1000	1800	-	1000	1800	-	-	-	-	-
Magnesite	546-93-0										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Magnesium oxide fume	1309-48-4										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Malathion	121-75-5										
Total dust		-	15	X	-	10	-	-	-	-	X
Respirable fraction		-	5	X	-	5	-	-	-	-	X
Maleic anhydride	108-31-6	0.25	1		0.25	1	-	-	-	-	-
Manganese compounds (as Mn)	7439-96-5	-	(C)5	-	-	-	-	-	-	5	-
Manganese fume (as Mn)	7439-96-5	-	(C)5	-	-	1	-	3	-	-	-
Manganese cyclopentadienyl tricarbonyl (as Mn)	12079-65-1	-	-	-	-	0.1	-	-	-	-	X
Manganese tetroxide (as Mn)	1317-35-7	-	-	-	-	1	-	-	-	-	-
Marble	1317-65-3										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Mercury (aryl and inorganic)(as Hg)	7439-97-6	See Table Z-2		-	-	-	-	-	-	0.1	X
Mercury (organo) alkyl compounds (as Hg)	7439-97-6	See Table Z-2		-	-	0.01	-	0.03	-	-	X
Mercury (vapor) (as Hg)	7439-97-6	See Table Z-2		-	-	0.05	-	-	-	-	X

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Mesityl oxide	141-79-7	25	100	-	15	60	25	100	-	-	-
Methacrylic acid	79-41-4	-	-	-	20	70	-	-	-	-	X
Methanethiol; see Methyl mercaptan											
Methomyl (Lannate)	16752-77-5	-	-	-	-	2.5	-	-	-	-	-
Methoxychlor	72-43-5										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
2-Methoxyethanol; see Methyl cellosolve											
4-Methoxyphenol	150-76-5	-	-	-	-	5	-	-	-	-	-
Methyl acetate	79-20-9	200	610	-	200	610	250	760	-	-	-
Methyl acetylene (Propyne)	74-99-7	1000	1650	-	1000	1650	-	-	-	-	-
Methyl acetylene propadiene mixture (MAPP)	--	1000	1800	-	1000	1800	1250	2250	-	-	-
Methyl acrylate	96-33-3	10	35	X	10	35	-	-	-	-	X
Methylacrylonitrile	126-98-7	-	-	-	1	3	-	-	-	-	X
Methylal (Dimethoxy-methane)	109-87-5	1000	3100	-	1000	3100	-	-	-	-	-
Methyl alcohol	67-56-1	200	260	-	200	260	250	310	-	-	X
Methylamine	74-89-5	10	12	-	10	12	-	-	-	-	-
Methyl amyl alcohol; see Methyl isobutyl carbinol											
Methyl n-amyl ketone	110-43-0	100	465	-	100	465	-	-	-	-	-
Methyl bromide	74-83-9	(C)20	(C)80	X	5	20	-	-	-	-	X

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³ [b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³ [b]	STEL[c] ppm[a]	STEL mg/m³ [b]	CEILING ppm[a]	CEILING mg/m³ [b]	CEILING Skin Designation
Methyl butyl ketone; see 2-Hexanone											
Methyl cellosolve (2-Methoxyethanol)	109-86-4	25	80	X	In process of 6(b) rulemaking						
Methyl cellosolve acetate (2-Methoxyethyl acetate)	110-49-6	25	120	X	In process of 6(b) rulemaking						
Methyl chloride	74-87-3	See Table Z-2			50	105	100	205	-	-	-
Methyl chloroform (1,1,1-Trichloroethane)	71-55-6	350	1900	-	350	1900	450	2450	-	-	-
Methyl 2-cyanoacrylate	137-05-3	-	-	-	2	8	4	16	-	-	-
Methylcyclohexane	108-87-2	500	2000	-	400	1600	-	-	-	-	-
Methylcyclohexanol	25639-42-3	100	470	-	50	235	-	-	-	-	-
o-Methylcyclohexanone	583-60-8	100	460	X	50	230	75	345	-	-	X
Methylcyclopentadiehyl manganese tricarbonyl (as Mn)	12108-13-3	-	-	-	-	0.2	-	-	-	-	X
Methyl demeton	8022-00-2	-	-	-	-	0.5	-	-	-	-	X
4,4'-Methylene bis (2-chloroaniline) (MBOCA)	101-14-4	-	-	-	0.02	0.22	-	-	-	-	
Methylene bis(4-cyclohexylisocyanate)	5124-30-1	-	-	-	-	-	-	-	0.01	0.11	
Methylene chloride	75-09-2	See Table Z-2			In process of 6(b) rulemaking						
Methyl ethyl ketone (MEK); see 2-Butanone											
Methyl ethyl ketone peroxide (MEKP)	1338-23-4	-	-	-	-	-	-	-	0.7	5	
Methyl formate	107-31-3	100	250	-	100	250	150	375	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Methyl hydrazine (Monomethyl hydrazine)	60-34-4	(C)0.2	(C)0.35	X	-	-	-	-	0.2	0.35	X
Methyl iodide	74-88-4	5	28	X	2	10	-	-	-	-	X
Methyl isoamyl ketone	110-12-3	-	-	-	50	240	-	-	-	-	-
Methyl isobutyl carbinol	108-11-2	25	100	X	25	100	40	165	-	-	X
Methyl isobutyl ketone; see Hexone											
Methyl isocyanate	624-83-9	0.02	0.05	X	0.02	0.05	-	-	-	-	X
Methyl isopropyl ketone	563-80-4	-	-	-	200	705	-	-	-	-	-
Methyl mercaptan	74-93-1	(C)10	(C)20	-	0.5	1	-	-	-	-	-
Methyl methacrylate	80-62-6	100	410		100	410	-	-	-	-	
Methyl parathion	298-00-0	-	-	-	-	0.2	-	-	-	-	X
Methyl propyl ketone; see 2-Pentanone											
Methyl silicate	681-84-5	-	-	-	1	6	-	-	-	-	-
alpha-Methyl styrene	98-83-9	(C)100	(C)480	-	50	240	100	485	-	-	-
Methylene bisphenyl isocyanate (MDI)	101-68-8	(C)0.02	(C)0.2	-	-	-	-	-	0.02	0.2	-
Metribuzin	21087-64-9	-	-	-	-	5	-	-	-	-	-
Mica; see Silicates											
Molybdenum (as Mo)	7439-98-7										
Soluble compounds		-	5	-	-	5	-	-	-	-	-
Insoluble compounds											
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Monocrotophos (Azodrin®)	6923-22-4	-	-	-	-	0.25	-	-	-	-	-
Monomethyl aniline	100-61-8	2	9	X	0.5	2	-	-	-	-	X

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	CEILING Skin Designation
Morpholine	110-91-8	20	70	X	20	70	30	105	-	-	X
Naphtha (Coal tar)	8030-30-6	100	400	-	100	400	-	-	-	-	-
Naphthalene	91-20-3	10	50	-	10	50	15	75	-	-	-
alpha-Naphthylamine; see 1910.1004	134-32-7										
beta-Naphthylamine; see 1910.1009	91-59-8										
Nickel carbonyl (as Ni)	13463-39-3	0.001	0.007	-	0.001	0.007	-	-	-	-	-
Nickel, metal and insoluble compounds (as Ni)	7440-02-0	-	1	-	-	1	-	-	-	-	-
Nickel, soluble compounds (as Ni)	7440-02-0	-	1	-	-	0.1	-	-	-	-	-
Nicotine	54-11-5	-	0.5	X	-	0.5	-	-	-	-	X
Nitric acid	7697-37-2	2	5	-	2	5	4	10	-	-	-
Nitric oxide	10102-43-9	25	30	-	25	30	-	-	-	-	-
p-Nitroaniline	100-01-6	1	6	X	-	3	-	-	-	-	X
Nitrobenzene	98-95-3	1	5	X	1	5	-	-	-	-	X
p-Nitrochlorobenzene	100-00-5	-	1	X	-	1	-	-	-	-	X
4-Nitrodiphenyl; see 1910.1003	92-93-3										
Nitroethane	79-24-3	100	310	-	100	310	-	-	-	-	-
Nitrogen dioxide	10102-44-0	(C)5	(C)9	-	-	-	1	1.8	-	-	-
Nitrogen trifluoride	7783-54-2	10	29	-	10	29	-	-	-	-	-
Nitroglycerin	55-63-0	(C)0.2	(C)1	X	-	-	-	0.1	-	-	X
Nitromethane	75-52-5	100	250	-	100	250	-	-	-	-	-
1-Nitropropane	108-03-2	25	90	-	25	90	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
2-Nitropropane	79-46-9	25	90	-	10	35	-	-	-	-	-
N-Nitrosodimethylamine; see 1910.1016	62-75-9										
Nitrotoluene											
o-isomer	88-72-2;										
m-isomer	99-08-1;	5	30	X	2	11	-	-	-	-	X
p-isomer	99-99-0										
Nitrotrichloromethane; see Chloropicrin											
Nonane	111-84-2	-	-	-	200	1050	-	-	-	-	-
Octachloronaphthalene	2234-13-1	-	0.1	X	-	0.1	-	0.3	-	-	X
Octane	111-65-9	500	2350	-	300	1450	375	1800	-	-	-
Oil mist, mineral	8012-95-1	-	5	-	-	5	-	-	-	-	-
Osmium tetroxide (as Os)	20816-12-0	-	0.002	-	0.0002	0.002	0.0006	0.006	-	-	-
Oxalic acid	144-62-7	-	1	-	-	1	-	2	-	-	-
Oxygen difluoride	7783-41-7	0.05	0.1	-	-	-	-	-	0.05	0.1	-
Ozone	10028-15-6	0.1	0.2	-	0.1	0.2	0.3	0.6	-	-	-
Paraffin wax fume	8002-74-2	-	-	-	-	2	-	-	-	-	-
Paraquat, respirable dust	4685-14-7	-	0.5	X	-	0.1	-	-	-	-	X
Parathion	56-38-2	-	0.1	X	-	0.1	-	-	-	-	X
Particulates not otherwise regulated	—										
Total dust	—	-	15	-	-	15	-	-	-	-	-
Respirable fraction	—	-	5	-	-	5	-	-	-	-	-
Pentaborane	19624-22-7	0.005	0.01	-	0.005	0.01	0.015	0.03	-	-	-
Pentachloronaphthalene	1321-64-8	-	0.5	X	-	0.5	-	-	-	-	X
Pentachlorophenol	87-86-5	-	0.5	X	-	0.5	-	-	-	-	X
Pentaerythritol	115-77-5										
Total dust		-	15	-	-	10	-	-	-	-	
Respirable fraction		-	5	-	-	5	-	-	-	-	

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits PEL* Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	CEILING Skin Designation
Pentane	109-66-0	1000	2950	-	600	1800	750	2250	-	-	-
2-Pentanone (Methyl propyl ketone)	107-87-9	200	700	-	200	700	250	875	-	-	-
Perchloroethylene (Tetrachloroethylene)	127-18-4	See Table Z-2			25	170	-	-	-	-	-
Perchloromethyl mercaptan	594-42-3	0.1	0.8	-	0.1	0.8	-	-	-	-	-
Perchloryl fluoride	7616-94-6	3	13.5	-	3	14	6	28	-	-	-
Perlite	—										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Petroleum distillates (Naphtha)	8002-05-9	500	2000	-	400	1600	-	-	-	-	-
Phenol	108-95-2	5	19	X	5	19	-	-	-	-	X
Phenothiazine	92-84-2	-	-	-	-	5	-	-	-	-	X
p-Phenylene diamine	106-50-3	-	0.1	X	-	0.1	-	-	-	-	X
Phenyl ether, vapor	101-84-8	1	7	-	1	7	-	-	-	-	-
Phenyl ether-biphenyl mixture, vapor	—	1	7	-	1	7	-	-	-	-	-
Phenylethylene; see Styrene											
Phenyl glycidyl ether (PGE)	122-60-1	10	60	-	1	6	-	-	-	-	-
Phenylhydrazine	100-63-0	5	22	X	5	20	10	45	-	-	X
Phenyl mercaptan	108-98-5	-	-	-	0.5	2	-	-	-	-	-
Phenylphosphine	638-21-1	-	-	-	-	-	-	-	0.05	0.25	-
Phorate	298-02-2	-	-	-	-	0.05	-	0.2	-	-	X
Phosdrin (Mevinphos®)	7786-34-7	-	0.1	X	0.01	0.1	0.03	0.3	-	-	X

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits PEL* Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	CEILING Skin Designation
Phosgene (Carbonyl chloride)	75-44-5	0.1	0.4	-	0.1	0.4	-	-	-	-	-
Phosphine	7803-51-2	0.3	0.4	-	0.3	0.4	1	1	-		-
Phosphoric acid	7664-38-2	-	1	-	-	1	-	3	-	-	-
Phosphorus (yellow)	7723-14-0	-	0.1	-	-	0.1	-	-	-	-	-
Phosphorus oxychloride	10025-87-3	-	-	-	0.1	0.6	-	-	-	-	-
Phosphorus pentachloride	10026-13-8	-	1	-	-	1	-	-	-	-	-
Phosphorus pentasulfide	1314-80-3	-	1	-	-	1	-	3	-	-	-
Phosphorus trichloride	7719-12-2	0.5	3	-	0.2	1.5	0.5	3	-	-	-
Phthalic anhydride	85-44-9	2	12	-	1	6	-	-	-	-	-
m-Phthalodinitrile	626-17-5	-	-	-	-	5	-	-	-	-	-
Picloram	1918-02-1										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Picric acid	88-89-1	-	0.1	X	-	0.1	-	-	-	-	X
Piperazine dihydrochloride	142-64-3	-	-	-	-	5	-	-	-	-	-
Pindone (2-Pivalyl-1,3-indandione)	83-26-1	-	0.1	-	-	0.1	-	-	-	-	-
Plaster of Paris	7778-18-9										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Platinum (as Pt)	7440-06-4										
Metal		-	-	-	-	1	-	-	-	-	-
Soluble salts		-	0.002	-	-	0.002	-	-	-	-	-
Portland cement	65997-15-1										
Total dust			See Table Z-3		-	10	-	-	-	-	-
Respirable fraction			See Table Z-3		-	5	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Potassium hydroxide	1310-58-3	-	-	-	-	-	-	-	-	2	-
Propane	74-98-6	1000	1800	-	1000	1800	-	-	-	-	-
Propargyl alcohol	107-19-7	-	-	-	1	2	-	-	-	-	X
beta-Propriolactone; see 1910.1013	57-57-8										
Proplonic acid	79-09-4	-	-	-	10	30	-	-	-	-	-
Propoxur (Baygon)	114-26-1	-	-	-	-	0.5	-	-	-	-	-
n-Propyl acetate	109-60-4	200	840	-	200	840	250	1050	-	-	-
n-Propyl alcohol	71-23-8	200	500	-	200	500	250	625	-	-	-
n-Propyl nitrate	627-13-4	25	110	-	25	105	40	170	-	-	-
Propylene dichloride	78-87-5	75	350	-	75	350	110	510	-	-	-
Propylene glycol dinitrate	6423-43-4	-	-	-	0.05	0.3	-	-	-	-	-
Propylene glycol monomethyl ether	107-98-2	-	-	-	100	360	150	540	-	-	-
Propylene imine	75-55-8	2	5	X	2	5	-	-	-	-	X
Propylene oxide	75-56-9	100	240	-	20	50	-	-	-	-	-
Propyne; see Methyl acetylene											
Pyrethrum	8003-34-7	-	5	-	-	5	-	-	-	-	-
Pyridine	110-86-1	5	15	-	5	15	-	-	-	-	-
Quinone	106-51-4	0.1	0.4	-	0.1	0.4	-	-	-	-	-
Resorcinol	108-46-3	-	-	-	10	45	20	90	-	-	-
Rhodium (as Rh), metal fume and insoluble compounds	7440-16-6	-	0.1	-	-	0.1	-	-	-	-	-
Rhodium (as Rh), soluble compounds	7440-16-6	-	0.001	-	-	0.001	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Ronnel	299-84-3	-	15	-	-	10	-	-	-	-	-
Rosin core solder pyrolysis products, as formaldehyde	—	-	-	-	-	0.1	-	-	-	-	-
Rotenone	83-79-4	-	5	-	-	5	-	-	-	-	-
Rouge	—										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Selenium compounds (as Se)	7782-49-2	-	0.2	-	-	0.2	-	-	-	-	-
Selenium hexafluoride (as Se)	7783-79-1	0.05	0.4	-	0.05	0.4	-	-	-	-	-
Silica, amorphous, precipitated and gel	—	See Table Z-3			-	6	-	-	-	-	-
Silica, amorphous, diatomaceous earth, containing less than 1% crystalline silica	68855-54-9	See Table Z-3			-	6	-	-	-		-
Silica, crystalline cristobalite (as quartz), respirable dust	14464-46-1	See Table Z-3			-	0.05	-	-	-	-	-
Silica, crystalline quartz (as quartz), respirable dust	14808-60-7	See Table Z-3			-	0.1	-	-	-	-	-
Silica, crystalline tripoli (as quartz), respirable dust	1317-95-9	See Table Z-3			-	0.1	-	-	-	-	-
Silica, crystalline tridymite (as quartz), respirable dust	15468-32-3	See Table Z-3			-	0.05	-	-	-	-	-
Silica, fused, respirable dust	60676-86-0	See Table Z-3			-	0.1	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Silicates (less than 1% crystalline silica)											
Mica (respirable dust)	12001-26-2	See Table Z-3			-	3	-	-	-	-	-
Soapstone, total dust	--	See Table Z-3			-	6	-	-	-	-	-
Soapstone, respirable dust	--	See Table Z-3			-	3	-	-	-	-	-
Talc (containing asbestos): use asbestos limit	--	See Table Z-3			See 29 CFR 1910.1001						
Talc (containing no asbestos), asbestos), respirable dust	14807-96-6	See Table Z-3			-	2	-	-	-	-	-
Tremolite		See Table Z-3			See 29 CFR 1910.1101						
Silicon	7440-21-3										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Silicon carbide	409-21-2										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Silicon tetrahydride	7803-62-5	-	-	-	5	7	-	-	-	-	-
Silver, metal dust and fume (as Ag)	7440-22-4	-	0.01	-	-	0.01	-	-	-	-	-
Soapstone; see Silicates											
Sodium azide	26628-22-8										
(as NN_3)		-	-	-	-	-	-	-	0.1	-	X
(as NaN_3)		-	-	-	-	-	-	-	-	0.3	X
Sodium bisulfite	7631-90-5	-	-	-	-	5	-	-	-	-	-
Sodium fluoroacetate	62-74-8	-	0.05	X	-	0.05	-	0.15	-	-	X
Sodium hydroxide	1310-73-2	-	2	-	-	-	-	-	-	2	-
Sodium metabisulfite	7681-57-4	-	-	-	-	5	-	-	-	-	-
Starch	9005-25-8										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	
Stibine	7803-52-3	0.1	0.5	-	0.1	0.5	-	-	-	-	-
Stoddard solvent	8052-41-3	500	2900	-	100	525	-	-	-	-	-
Strychnine	57-24-9	-	0.15	-	-	0.15	-	-	-	-	-
Styrene	100-42-5	See Table Z-2			50	215	100	425	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³ [b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³ [b]	STEL[c] ppm[a]	STEL mg/m³ [b]	CEILING ppm[a]	CEILING mg/m³ [b]	CEILING Skin Designation
Subtilisins (Proteolytic enzymes)	1395-21-7	-	-	-	-	-	-	-	-	0.00006	-
Sucrose	57-50-1										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Sulfur dioxide	7446-09-5	5	13	-	2	5	5	10	-	-	-
Sulfur hexafluoride	2551-62-4	1000	6000	-	1000	6000	-	-	-	-	-
Sulfuric acid	7664-93-9	-	1	-	-	1	-	-	-	-	-
Sulfur monochloride	10025-67-9	1	6	-	-	-	-	-	1	6	-
Sulfur pentafluoride	5714-22-7	0.025	0.25	-	-	-	-	-	0.01	0.1	-
Sulfur tetrafluoride	7783-60-0	-	-	-	-	-	-	-	0.1	0.4	-
Sulfuryl fluoride	2699-79-8	5	20	-	5	20	10	40	-	-	-
Sulprofos	35400-43-2	-	-	-	-	1	-	-	-	-	-
Systox®, see Demeton											
2,4,5-T	93-76-5	-	10	-	-	10	-	-	-	-	-
Talc; see Silicates											
Tantalum, metal and oxide dust	7440-25-7	-	5	-	-	5	-	-	-	-	-
TEDP (Sulfotep)	3689-24-5	-	0.2	X	-	0.2	-	-	-	-	X
Tellurium and compounds (as Te)	13494-80-9	-	0.1	-	-	0.1	-	-	-	-	-
Tellurium hexafluoride (as Te)	7783-80-4	0.02	0.2	-	0.02	0.2	-	-	-	-	-
Temephos	3383-96-8										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
TEPP	107-49-3	-	0.05	X	-	0.05	-	-	-	-	X
Terphenyls	26140-60-3	(C)1	(C)9	-	-	-	-	-	0.5	5	-
1,1,1,2-Tetrachloro-2,2-difluoroethane	76-11-9	500	4170	-	500	4170	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL ppm[a]	Transitional Limits PEL mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits[aa] TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
1,1,2,2-Tetrachloro-1,2-difluoroethane	76-12-0	500	4170		500	4170					
1,1,2,2-Tetrachloroethane	79-34-5	5	35	X	1	7					X
Tetrachoroethylene; see Perchloroethylene											
Tetrachloromethane; see Carbon tetrachloride											
Tetrachloronaphthalene	1335-88-2		2	X		2					X
Tetraethyl lead (as Pb)	78-00-2		0.075	X		0.075					X
Tetrahydrofuran	109-99-9	200	590		200	590	250	735			
Tetramethyl lead, (as Pb)	75-74-1		0.075	X		0.075	--	--			X
Tetramethyl succinonitrile	3333-52-6	0.5	3	X	0.5	3		--	--		X
Tetranitromethane	509-14-8	1	8		1	8					
Tetrasodium pyrophosphate	7722-88-5					5					
Tetryl (2,4,6-Trinitrophenyl-methyl-nitramine)	479-45-8		1.5	X		0.1					X
Thallium, soluble compounds (as Tl)	7440-28-0		0.1	X		0.1					X
4,4'-Thiobis(6-tert, Butyl-m-cresol)	96-69-5										
Total dust			15			10					
Respirable fraction			5			5					
Thioglycolic acid	68-11-1				1	4					X
Thionyl chloride	7719-09-7								1	5	
Thiram	137-26-8		5			5					
Tin, inorganic compounds (except oxides) (as Sn)	7440-31-5					2					
Tin, organic compounds (as Sn)	7440-31-5		0.1			0.1					X

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits PEL* Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL[c] mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Tin oxide (as Sn)	7440-31-5	-	-	-	-	2	-	-	-	-	-
Titanium dioxide	13463-67-7										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Toluene	108-88-3	See Table Z-2			100	375	150	560	-	-	-
Toluene-2,4-diisocyanate (TDI)	584-84-9	(C)0.02	(C)0.14	-	0.005	0.04	0.02	0.15	-	-	-
m-Toluidine	108-44-1	-	-	-	2	9	-	-	-	-	X
o-Toluidine	95-53-4	5	22	X	5	22	-	-	-	-	X
p-Toluidine	106-49-0	-	-	-	2	9	-	-	-	-	X
Toxaphene; see Chlorinated camphene											
Tremolite; see Silicates											
Tributyl phosphate	126-73-8	-	5	-	0.2	2.5	-	-	-	-	-
Trichloroacetic acid	76-03-9	-	-	-	1	7	-	-	-	-	-
1,2,4-Trichlorobenzene	120-82-1	-	-	-	-	-	-	-	5	40	-
1,1,1-Trichloroethane; see Methyl chloroform											
1,1,2-Trichloroethane	79-00-5	10	45	X	10	45	-	-	-	-	X
Trichloroethylene	79-01-6	See Table Z-2			50	270	200	1080	-	-	-
Trichloromethane; see Chloroform											
Trichloronaphthalene	1321-65-9	-	5	X	-	5	-	-	-	-	X
1,2,3-Trichloropropane	96-18-4	50	300	-	10	60	-	-	-	-	-
1,1,2-Trichloro-1,2,2-trifluoroethane	76-13-1	1000	7600	-	1000	7600	1250	9500	-	-	-
Triethylamine	121-44-8	25	100	-	10	40	15	60	-	-	-
Trifluorobromomethane	75-63-8	1000	6100	-	1000	6100	-	-	-	-	-
Trimellitic anhydride	552-30-7	-	-	-	0.005	0.04	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL[c] mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Trimethylamine	75-50-3	-	-	-	10	24	15	36	-	-	-
Trimethyl benzene	25551-13-7	-	-	-	25	125	-	-	-	-	-
Trimethyl phosphite	121-45-9	-	-	-	2	10	-	-	-	-	-
2,4,6-Trinitrophenyl; see Picric acid											
2,4,6-Trinitrophenylmethyl nitramine; see Tetryl											
2,4,6-Trinitrotoluene (TNT)	118-96-7	-	1.5	X	-	0.5	-	-	-	-	X
Triorthocresyl phosphate	78-30-8	-	0.1	-	-	0.1	-	-	-	-	X
Triphenyl amine	603-34-9	-	-	-	-	5	-	-	-	-	-
Triphenyl phosphate	115-86-6	-	3	-	-	3	-	-	-	-	-
Tungsten (as W)	7440-33-7										
Insoluble compounds		-	-	-	-	5	-	10	-	-	-
Soluble compounds		-	-	-	-	1	-	3	-	-	-
Turpentine	8006-64-2	100	560	-	100	560	-	-	-	-	-
Uranium (as U)	7440-61-1										
Soluble compounds		-	0.05	-	-	0.05	-	-	-	-	-
Insoluble compounds		-	0.25	-	-	0.2	-	0.6	-	-	-
n-Valeraldehyde	110-62-3	-	-	-	50	175	-	-	-	-	-
Vanadium	1314-62-1										
Respirable dust (as V_2O_5)		—	(C)0.5	-	-	0.05	-	-	-	-	-
Fume (as V_2O_5)		—	(C)0.1	-	-	0.05	-	-	-	-	-
Vegetable oil mist	—										
Total dust		-	15	-	-	15	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Vinyl acetate	108-05-4	-	-	-	10	30	20	60	-	-	-
Vinyl benzene; see Styrene											
Vinyl bromide	593-60-2	-	-	-	5	20	-	-	-	-	-
Vinyl chloride; see 1910.1017	75-01-4										

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No.	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³ [b]	Transitional Limits PEL* Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³ [b]	STEL[c] ppm[a]	STEL mg/m³ [b]	CEILING ppm[a]	CEILING mg/m³ [b]	CEILING Skin Designation
Vinylcyanide; see Acrylonitrile											
Vinyl cyclohexene dioxide	106-87-6	-	-	-	10	60	-	-	-	-	X
Vinylidene chloride (1,1-Dichloroethylene)	75-35-4	-	-	-	1	4	-	-	-	-	-
Vinyl toluene	25013-15-4	100	480	-	100	480	-	-	-	-	-
VM & P Naphtha	8032-32-4	-	-	-	300	1350	400	1800	-	-	-
Warfarin	81-81-2	-	0.1	-	-	0.1	-	-	-	-	-
Welding fumes (total particulate)***	—	-	-	-	-	5	-	-	-	-	-
Wood dust, all soft and hard woods, except Western red cedar	—	-	-	-	-	5	-	10	-	-	-
Wood dust, Western red cedar	—	-	-	-	-	2.5	-	-	-	-	-
Xylenes (o-, m-, p-isomers)	1330-20-7	100	435	-	100	435	150	655	-	-	-
m-Xylene alpha, alpha - diamine	1477-55-0	-	-	-	-	-	-	-	-	0.1	X
Xylidine	1300-73-8	5	25	X	2	10	-	-	-	-	X
Yttrium	7440-65-5	-	1	-	-	1	-	-	-	-	-
Zinc chloride fume	7646-85-7	-	1	-	-	1	-	2	-	-	-
Zinc chromate (as CrO_3)	Varies with Compound	See Table Z-2			-	-	-	-	-	0.1	-
Zinc oxide fume	1314-13-2	-	5	-	-	5	-	10	-	-	-
Zinc oxide	1314-13-2										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-
Zinc stearate	557-05-1										
Total dust		-	15	-	-	10	-	-	-	-	-
Respirable fraction		-	5	-	-	5	-	-	-	-	-

TABLE Z-1-A. Limits For Air Contaminants

Substance	CAS No	Transitional Limits PEL* ppm[a]	Transitional Limits PEL* mg/m³[b]	Transitional Limits Skin Designation	Final Rule Limits** TWA ppm[a]	TWA mg/m³[b]	STEL[c] ppm[a]	STEL mg/m³[b]	CEILING ppm[a]	CEILING mg/m³[b]	Skin Designation
Zirconium compounds (as Zr)	7440-6	-	5	-	-	5	-	10	-	-	-

* The transitional PELs are 8-hour TWAs unless otherwise noted; a (C) designation denotes a ceiling limit.

** Unless otherwise noted, employers in General Industry (i.e., those covered by 29 CFR 1910) may use any combination of controls to achieve these limits for a period not to exceed 4 years from the effective date of this standard; for employers operating Marine Terminals (i.e., those covered by 29 CFR 1918), any combination of controls may be used until further notice.

*** As determined from breathing-zone air samples.

a Parts of vapor or gas per million parts of contaminated air by volume at 25°C and 760 torr.

b Approximate milligrams of substance per cubic meter of air.

c Duration is for 15 minutes, unless otherwise noted.

d The final benzene standard in 1910.1028 applies to all occupational exposures to benzene except some subsegments of industry where exposures are consistently under the action level (i.e., distribution and sale of fuels, sealed containers and pipelines, coke production, oil and gas drilling and production, natural gas processing, and the percentage exclusion for liquid mixtures); for the excepted subsequents, the benzene limits in Table Z-2 apply.

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TABLE Z-2

Material	8-hour time-weighted average	Acceptable ceiling concentration	Acceptable maximum peak above the acceptable ceiling concentration for an 8-hour shift	
			Concentration	Maximum duration
Benzene (Z37.40-1969) [1]	10 ppm	25 ppm	50 ppm	10 minutes.
Beryllium and Beryllium compounds (Z37.29-1970)	2 μg/m³	5 μg/m³	25 μg/m³	30 minutes.
Cadmium fume (Z37.5-1970)	0.1 mg/m³	0.3 mg/m³		
Cadmium dust (Z37.5-1970)	0.2 mg/m³	0.6 mg/m³		
Carbon disulfide (Z37.3-1968)	20 ppm	30 ppm	100 ppm	30 minutes.
Carbon Tetrachloride (Z37.17-1967)	10 ppm	25 ppm	200 ppm	5 minutes in any 4 hours.
Chromic acid and chromates (Z37.7-1971)		1 mg/10 m³		
Ethylene dibromide (Z37.31-1970	20 ppm	30 ppm	50 ppm	5 minutes.
Ethylene dichloride (Z37.21-1969)	50 ppm	100 ppm	200 ppm	5 minutes in any 3 hours.
Formaldehyde (Z37.16-1967) [2]	3 ppm	5 ppm	10 ppm	30 minutes.
Hydrogen fluoride (Z37.28-1969)	3 ppm			
Hydrogen sulfide (Z37.2-1966)		20 ppm	50 ppm	10 minutes once only if no other measurable exposure occurs.
Fluoride as dust (Z37.38-1969)	2.5 mg/m³			
Mercury (Z37.8-1971)		1 mg/10 m³		
Methyl chloride(Z37.18-1969	100 ppm	200 ppm	300 ppm	5 minutes in any 3 hours.
Methylene chloride (Z37.23-1969)	500 ppm	1,000 ppm	2,000 ppm	5 minutes in any 2 hours.
Organo (alkyl) mercury (Z37.30-1969)	0.01 mg/m³	0.04 mg/m³		
Styrene (Z37.15-1969)	100 ppm	200 ppm	600 ppm	5 minutes in any 3 hours.
Tetrachloroethylene (Z37.22-1967)	100 ppm	200 ppm	300 ppm	5 minutes in any 3 hours.
Toluene (Z37.12-1967)	200 ppm	300 ppm	500 ppm	10 minutes.
Trichloroethylene (Z37.19-1967)	100 ppm	200 ppm	300 ppm	5 minutes in any 2 hours.

[1] This standard applies to the industry segments exempt from the 1 ppm 8-hour TWA and 5 ppm STEL of the benzene standard at 1910.1028. This standard also applies to any industry for which 1910.1028 is stayed or otherwise not in effect.

[2] This standard applies to any industry for which 1910.1048 is stayed or otherwise not in effect.

TABLE Z-3

Substance	mppcf*	mg/m³
SILICA:		
CRYSTALLINE		
QUARTZ (RESPIRABLE)*	250[f]	10 mg/m³[m]
QUARTZ (TOTAL DUST)	%SiO_2+5	%SiO_2+2 30 mg/m³
CRISTOBALITE*: Use ½ the value calculated from the count or mass formulae for quartz		%SiO_2+2
TRIDYMITE*: Use ½ the value calculated from the formulae for quartz		
AMORPHOUS, including natural diatomaceous earth*	20	80 mg/m³
SILICATES (less than 1% crystalline silica):		%SiO_2
Mica*	20	
Soapstone*	20	
Talc (not containing asbestos)*	20[n]	
Talc (containing asbestos). Use asbestos limit		
Tremolite (see 29 CFR 1910.1101)		
Portland cement*	50	
GRAPHITE (NATURAL)*	15	
COAL DUST (respirable fraction less than 5% SiO_2)*		2.4 mg/m³ or
For more than 5% SiO_2*		10 mg/m³
INERT OR NUISANCE DUST:		%SiO_2+2
Respirable fraction*	15	5 mg/m³
Total dust	50	15 mg/m³

Note.—Conversion factors—mmpcf×35.3=million particles per cubic meter=particles per c.c.

*Substances that are in Table Z-4 in this proposal.

*Millions of particles per cubic foot of air, based on impinger samples counted by light-field techniques.

[f] The percentage of crystalline silica in the formula is the amount determined from airborne samples, except in those instances in which other methods have been shown to be applicable.

[m] Both concentration and percent quartz for the application of this limit are to be determined from the fraction passing a size-selector with the following characteristics:

Aerodynamic diameter (unit density sphere)	Percent passing selector	Aerodynamic diameter (unit density sphere)	Percent passing selector
2	90	3.5	50
2.5	75	5.0	25

Aerodynamic diameter (unit density sphere)	Percent passing selector
10	0

[n] Containing less than 1% quartz; if 1% quartz, use quartz limit.

The measurements under this note refer to the use of an AEC (now NRC) instrument. The respirable fraction of coal dust is determined with an MRE: the figure corresponding to that of 2.4 mg/m³ in the table for coal dust is 4.5 mg/m³.

Editorial note: This Appendix will not appear in the Code of Federal Regulations.

XI. Appendix—Sampling and Analytical Methods

The sampling and analytical methods for the substances listed in Section II of this preamble are categorized into three groups: (1) Fully Validated Methods, (2) Other Methods, and (3) No Methods. These methods are indicated in the tables in this Appendix. The first table details fully validated methods, other methods, substances for which there are no identified methods, and detection limits. The second table identifies the most recent NIOSH Analytical methods.

A. Fully Validated Methods

Fully Validated methods were developed by either NIOSH or OSHA. The criteria used in validating these procedures were developed independently by each agency. There are some differences in validation protocol, but in general similar testing procedures were followed. These methods are widely accepted by the scientific community.

B. Other Methods

Methods in this category have not been subjected to all of the testing procedures required of fully validated methods. Some of these procedures have been taken directly from scientific literature and may not have been used by OSHA. Some are methods that were validated for a specific analyte and OSHA believes are applicable for similar analytes. OSHA has reviewed these methods and has concluded that they are of adequate quality to be used for assessing exposures and for enforcement.

C. No Methods

These analytes do not have an adequate sampling method available at OSHA, nor has an appropriate method been found in the available scientific literature.

D. Detection Limits

The values listed under Detection Limits are the lowest air concentrations that can be monitored, based on recommended sample air volumes. Detection limits for the OSHA validated methods are determined during the evaluation. The detection limits listed for the in-house methods are the estimates of OSHA.

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H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1001.	Acetaldehyde	OSHA 68	—	—	0.58 ppm	
1002.	Acetic Acid	OSHA-NIOSH 1603	—	—	6 ppb	DL based on 240 L air vol
1003.	Acetic Anhydride	NIOSH 3506	—	—	5 mg/M^3	DL based on 100 L air vol
1004.	Acetone	OSHA 69	—	—	0.3 ppm	
1005.	Acetonitrile	NIOSH 1606	—	—	1 ppm	
1006.	Acetylsalicylic Acid (Aspirin)	—	In-house[2]	—	2 ug/M^3	DL based on 120 L air vol
1007.	Acrolein	OSHA 52	—	—	2.7 ppb	
1008.	Acrylamide	OSHA 21	—	—	1.3 ppb	
1009.	Acrylic Acid	OSHA 28	—	—	0.014 ppb	
1010.	Allyl Alcohol	NIOSH 1402	—	—	0.5 ppm	
1011.	Allyl Chloride	NIOSH 1000	—	—	0.2 ppm	
1012.	Allyl Glycidyl Ether (AGE)	NIOSH S346	—	—	0.1 ppm	
1013.	Allyl Propyl Disulfide	—	In-house	—	0.16 ppm	
1014.	alpha-Alumina	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1015.	Aluminum					
1016.	Metal & oxide- \					
1017.	Pyro powders- \					/ DL based on 480 L air vol
1018.	Soluble Salts- >	GRAV & In-house	—	—	0.1 mg/M^3	< Method does not differentiate
1019.	Welding Fumes- _/					\ different forms of Al.
—	Alkyls-	—	—	XXXX		
1020.	Amitrole (3-Amino-1,2,4-triazole)	—	In-house	—	0.004 mg/M^3	DL based on 60 L air vol
1021.	Ammonia	OSHA ID188, ID164	—	—	1 ppm	DL based on 24 L air vol
1022.	Ammonium Chloride (fume)	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1024.	Ammonium Sulfamate (Ammate)	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1025.	Aniline	NIOSH 2002	—	—	0.05 ppm	
—	ANTU (alpha-Naphthyl-Thiourea)	NIOSH S276	—	—	0.01 mg/M^3	DL based on 480 L air vol
—	Arsenic	OSHA ID105	—	—	0.0005 mg/M^3	DL based on 480 L air vol
1028.	Asphalt (Petroleum) Fumes	—	OSHA 58	—	0.006 mg/M^3	Benzene soluble portion

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H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1029.	Atrazine	—	In-house	—	0.05 mg/M^3	DL based on 120 L air vol
—	Azinphos-Methyl	—	In-house	—	0.01 mg/M^3	DL based on 300 L air vol
1031.	Barium Sulfate	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1032.	Benomyl	—	In-house	—	0.03 mg/M^3	DL based on 60 L air vol
1033.	Beryllium & Compounds-	OSHA ID125G	—	—	0.04 ug/M^3	DL based on 480 L air vol
1034.	Bismuth Telluride (Selenium doped)	—	In-house	—	0.02 mg/M^3	DL based on 480 L air vol
1035.	Bismuth Telluride (Undoped)	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
—	Borates, Tetra, Sodium Salts					
1036.	Anhydrous- \				/	DL based on 480 L air vol. Method is for
1037.	Decahydrate- >	—	In-house	—	0.01 mg/M^3 <	total Boron and does not differentiate
1038.	Pentahydrate- /				\	different forms of Boron.
1039.	Boron Oxide	GRAVIMETRIC	—	—	0.02 mg/sample	Same as nuisance particulate TLV
1040.	Boron Tribromide	—	In-house	—	0.01 mg/M^3	Based on total Br^- & 15 L air vol
1041.	Bromacil	—	In-house	—	0.1 mg/M^3	DL based on 50 L air vol
1042.	Bromine	OSHA ID108	—	—	0.09 ppm	DL based on 30 L air vol
1043.	Bromine Pentafluoride	—	In-house	—	0.01 mg/M^3	Based on total Br^- & 48 L air vol
1044.	Butane	—	In-house	—	1 ppm	DL based on 10 L air vol
1045.	2-Butanone (MEK)	OSHA 16	—	—	1.5 ppm	
1046.	2-Butoxy Ethanol	NIOSH 1403	—	—	0.02 ppm	DL based on 10 L air vol
1047.	n-Butyl Acetate	NIOSH 1450	—	—	0.04 ppm	DL based on 10 L air vol
1048.	Butyl Acrylate	—	In-house	—	0.3 ppm	DL based on 3 L air vol
1049.	sec-Butyl Alcohol	NIOSH 1401	—	—	0.5 ppm	DL based on 10 L air vol
1050.	tert-Butyl Alcohol	NIOSH 1400	—	—	0.5 ppm	DL based on 10 L air vol
1051.	n-Butyl Alcohol	NIOSH 1401	—	—	0.5 ppm	DL based on 10 L air vol
1052.	n-Butyl Glycidyl ether (BGE)	NIOSH S81	—	—	0.5 ppm	DL based on 10 L air vol
1053.	n-Butyl Lactate	—	OSHA 7	—	0.2 ppm	Adapt OSHA method to this compound
1054.	Butyl Mercaptan	NIOSH S350	—	—	0.2 ppm	DL based on 10 L air vol
1055.	o-sec-Butylphenol	—	In-house	—	0.1 ppm	DL based on 10 L air vol
1056.	p-tert-Butyltoluene	NIOSH 1501	—	—	0.1 ppm	DL based on 10 L air vol

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H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1057.	Calcium Carbonate (Limestone, Marble)	GRAVIMETRIC	—	—	0.02 mg/Sample	Same as nuisance particulate TLV
1058.	Calcium Cyanamide	—	In-house	—	0.02 mg/M^3	DL based on sol. Ca & 480 L air vol
1059.	Calcium Hydroxide	—	OSHA ID121	—	0.002 mg/M^3	DL based on total Ca
1060.	Calcium Oxide	—	OSHA ID121	—	0.002 mg/M^3	DL based on total Ca
1061.	Calcium Silicate, Total Dust-	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1062.	Calcium Sulfate	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1063.	Camphor, Synthetic	NIOSH 1301	—	—	0.5 ppm	DL based on 10 L air vol
1064.	Caprolactam (Vapor & Aerosol)	—	In-house	—	0.007 mg/M^3	DL based on 100 L air vol
1065.	Caprolactam (Vapor only)	—	In-house	—	0.01 mg/M^3	DL based on 100 L air vol
1066.	Captafol (Difolatan)	—	In-house	—	0.02 mg/M^3	DL based on 120 L air vol
1067.	Captan	—	In-house	—	0.02 mg/M^3	DL based on 60 L air vol
1068.	Carbofuran (Furadan)	—	In-house	—	0.02 mg/M^3	DL based on 300 L air vol
1069.	Carbon Dioxide	OSHA ID172	—	—	500 ppm	
1070.	Carbon Disulfide	OSHA-NIOSH 1600	—	—	0.1 ppm	DL based on 5 L air vol
1071.	Carbon Monoxide	—	Field Test	—	0.5 ppm	Use direct reading instrument
1072.	Carbon Tetrabromide	—	In-house	—	0.01 ppm	DL based on 10 L air vol
1073.	Carbon Tetrachloride (Tetrachloromethane)	NIOSH 1003	—	—	0.1 ppm	DL based on 15 L air vol
1074.	Carbonyl Fluoride	—	In-house	—	0.2 mg/M^3	Based on total F^- & 240 L air vol
1075.	Catechol (Pyrocatechol)	—	OSHA 32	—	0.5 ppm	Adapt OSHA method to this compound
1076.	Cellulose (paper fiber)	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1077.	Cesium Hydroxide	—	In-house	—	0.02 mg/M^3	DL based on Total Cs & 480 L air vol
1078.	Chlorinated Camphene (Toxaphene)	NIOSH S67	—	—	0.05 mg/M^3	DL based on 15 L air vol
1079.	Chlorine	OSHA ID101	—	—	0.13 ppm	
1080.	Chlorine Dioxide	—	In-house	—	0.05 ppm	DL based on 29 L air vol

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H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1081.	1-Chloro-1-Nitropropane	NIOSH S211	—	—	0.5 ppm	
1082.	2-Chloro-6-Trichloro-Methyl Pyridine (Nitrapyrin)	—	In-house	—	0.08 mg/M^3	DL based on 60 L air vol
1083.	Chloroacetyl Chloride	—	In-house	—	0.05 ppm	DL based on 10 L air vol
1084.	o-Chlorobenzylidene-Malononitrile	—	NIOSH 304	—	0.003 mg/M^3	DL based on 90 L air vol
1085.	Chlorodifluoromethane	—	NIOSH 1020	—	1 ppm	Adapt NIOSH method to this compound
1086.	Chloroform	OSHA 5	—	—	0.11 ppm	
1087.	Chloropentafluoroethane	—	NIOSH 1020	—	1 ppm	Adapt NIOSH method to this compound
1088.	beta-Chloroprene	NIOSH 1002	—	—	0.5 ppm	
1089.	o-Chlorostyrene	—	NIOSH 1003	—	0.5 ppm	Adapt NIOSH method to this compound
1090.	o-Chlorotoluene	—	NIOSH 1003	—	0.5 ppm	Adapt NIOSH method to this compound
1091.	Chlorpyrifos (Dursban)	OSHA 62	—	—	0.23 ppb	DL based on 480 L air vol
1092.	Chromic Acid & Chromates	OSHA ID103	—	—	<0.4 ug/M^3 as Cr^{+6}.	DL based on 480 L air vol
1093.	Chromium, metal	ID121, ID125G	—	—	2 ug/M^3	DL based on 480 L air vol
1094.	Chromyl Chloride	—	In-house	—	0.01 mg/M^3	Based on Cr^{+6} & 240 L air vol
1095.	Clopidol (Coyden)	—	In-house	—	0.08 mg/M^3	DL based on 60 L air vol
1096.	Coal Dust (<5% Quartz)	GRAV & ID142	—	—	0.02 mg/M^3	Quartz analysis using 400 L air vol
1097.	Coal Dust (>5% Quartz)	GRAV & ID142	—	—	0.02 mg/M^3	Quartz analysis using 400 L air vol
1098.	Cobalt Carbonyl, as Co	—	In-house	—	0.002 mg/M^3	Based on total Co & 480 L air vol
1099.	Cobalt Hydrocarbonyl, as Co	—	In-house	—	0.002 mg/M^3	Based on total Co & 480 L air vol
1100.	Cobalt, as Co (Metal Dust & Fume)	ID121, ID125G	—	—	2 ug/M^3	DL based on 480 L air vol
1101.	Copper, Fume-	ID121, ID125G	—	—	2 ug/M^3	DL based on 480 L air vol
1102.	Crag Herbicide (Sesone)	NIOSH S356	—	—	1.5 mg/M^3	DL based on 90 L air vol
1103.	Crufomate	—	In-house	—	0.03 mg/M^3	DL based on 60 L air vol
1104.	Cyanamide	—	In-house			
1105.	Cyanogen	—	In-house	—	0.08 ppm	DL based on 10 L air vol
1106.	Cyanogen Chloride	—	In-house	—	0.05 ppm	DL based on 10 L air vol
1107.	Cyclohexanol	NIOSH 1402	—	—	0.5 ppm	DL based on 10 L air vol
1108.	Cyclohexanone	OSHA 1	—	—	0.05 ppm	

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H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1109.	Cyclohexylamine	—	In-house	—	0.5 ppm	DL based on 10 L air vol
1110.	Cyclonite (RDX)	—	In-house	—	0.08 mg/M^3	DL based on 80 L air vol
1111.	Cyclopentane	—	NIOSH 1500	—	0.5 ppm	Adapt NIOSH method to this compound
1112.	Cyhexatin	—	In-house	—	0.01 mg/M^3	Solvent extract, Sn anal & 480 L air vol
1113.	DDT (Dichlorodiphenyl-trichloroethane)	NIOSH S274	—	—	0.01 mg/M^3	DL based on 90 L air vol
1114.	Decaborane	—	In-house	—	0.002 mg/M^3	Hot H_2O extract, B anal & 480 L air vol
—	Demeton (Systox)	NIOSH 5514	—	—	0.006 mg/M^3	DL based on 480 L air vol
1116.	Di-sec-Octyl Phthalate	—	In-house	—	0.01 mg/M^3	DL based on 100 L air vol
1117.	2,6-Di-tert-Butyl-p-Cresol	—	NIOSH 226	—	0.5 ppm	
1118.	Diazinon	OSHA 62	—	—	0.24 ppb_3	DL based on 480 L air vol
1119.	Dibutyl Phosphate	—	NIOSH 5017	—	0.3 mg/M^3	DL based on 250 L air vol
1120.	2-N-Dibutylaminoethanol	—	OSHA-NIOSH 2007	—	0.5 ppm	
1121.	1,1-Dichloro-1-Nitroethane	NIOSH 1601	—	—	0.5 ppm	
1122.	1,3-Dichloro-5,5-Dimethylhydantoin	—	In-house	—	0.2 mg/mL	Field anal by Chemiluminescence
1123.	Dichloroacetylene	—	NIOSH 1003	—	0.05 ppm	Adapt NIOSH method to this compound
—	o-Dichlorobenzene	NIOSH 1003	—	—	0.5 ppm	
1125.	p-Dichlorobenzene	NIOSH 1003	—	—	0.5 ppm	
1126.	1,1-Dichloroethane	NIOSH 1003	—	—	0.5 ppm	
1127.	Dichloroethyl Ether	NIOSH 1004	—	—	1 ppm	
1128.	Dichlorofluoromethane	NIOSH 2516	—	—	1 ppm	
1129.	1,3-Dichloropropene	—	NIOSH 1003	—	0.5 ppm	Adapt NIOSH method to this compound
1130.	2,2-Dichloropropionic Acid	—	In-house	—	0.5 ppm	DL based on 10 L air vol
1131.	Dicrotophos (Bidrin)	—	In-house	—	0.01 mg/M^3	DL based on 240 L air vol
1132.	Dicyclopentadiene	—	In-house	—	0.04 ppm	DL based on 10 L air vol
1133.	Dicyclopentadienyl Iron	—	ID121, ID125	—		
1134.	Diethanolamine	—	In-house	—	0.05 ppm	DL based on 10 L air vol
1135.	Diethyl Ketone	—	In-house	—	0.5 ppm	DL based on 10 L air vol
1136.	Diethyl Phthalate	—	In-house	—	0.07 mg/M^3	DL based on 180 L air vol
1137.	Diethylamine	OSHA 41	—	—	0.053 ppm	
1138.	Diethylenetriamine	OSHA 60	—	—	4 ppb	

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H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1139.	Diglycidyl Ether (DGE)	—	—	—	—	Not present in air - Sample for Epichlorohydrin & Bisphenol A
1140.	Diisobutyl Ketone	NIOSH 1300	—	—	0.5 mg/M^3	
1141.	Dimethyl-1,2-Dibromo-2,2-Dichloroethyl Phosphate	—	In-house	—	0.01 mg/M^3	DL based on 480 L air vol
1142.	Dimethyl Sulfate	—	OSHA-NIOSH 301	—	0.1 mg/M^3	DL based on 20 L air vol
1143.	Dimethylaniline (N,N-Dimethylaniline)	NIOSH 2002	—	—	0.003 ppm	DL based on 20 L air vol
1144.	Dinitolmide (3,5-Dinitro-o-Toluamide)	—	In-house	—	0.05 mg/M^3	DL based on 240 L air vol
1145.	Dioxane (Diethylene Dioxide)	NIOSH 1602	—	—	0.5 ppm	
1146.	Dioxathion (Delnav)	—	In-house	—	0.03 mg/M^3	DL based on 10 L air vol
1147.	Diphenylamine	OSHA 22	—	—	1 ug/M^3	
1148.	Dipropyl Ketone	—	OSHA 7	—	0.5 ppm	Adapt OSHA method to this compound
1149.	Dipropylene Glycol Methyl Ether	NIOSH S69	—	—	0.5 ppm	DL based on 10 L air vol
1150.	Diquat	—	In-house	—	2.8 mg/M^3	DL based on 100 L air vol
1151.	Disulfiram	—	In-house	—	0.02 mg/M^3	DL based on 25 L air vol
1152.	Disulfoton (Disyston)	—	In-house	—	0.002 mg/M^3	DL based on 480 L air vol
1153.	Diuron	—	In-house	—	0.03 mg/M^3	DL based on 60 L air vol
1154.	Divinyl Benzene	—	OSHA 9	—	0.5 ppm	Adapt OSHA method to this compound
1155.	Emery	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1156.	Endosulfan	—	In-house	—	3.4 ug/M^3	DL based on 60 L air vol
1158.	Epichlorohydrin	NIOSH 1010	—	—	0.1 ppm	DL based on 20 L air vol
1159.	Ethanolamine	—	In-house	—	0.08 ppm	DL based on 10 L air vol
1160.	Ethion (Nialate)	—	In-house	—	0.01 mg/M^3	DL based on 240 L air vol
1161.	Ethyl Acrylate	NIOSH 1450	—	—	0.5 ppm	
1162.	Ethyl Benzene	NIOSH 1501	—	—	0.05 ppm	DL based on 10 L air vol
1163.	Ethyl Bromide	NIOSH 1011	—	—	0.5 ppm	
1164.	Ethyl Ether	NIOSH 1610	—	—	0.5 ppm	
1165.	Ethyl Mercaptan	—	In-house	—	0.1 ppm	DL based on 20 L air vol

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H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1166.	Ethyl Silicate	NIOSH S264	—	—	0.5 ppm	
1167.	Ethylene Chlorohydrin	NIOSH 2513	—	—	0.1 ppm	
1168.	Ethylene Dichloride (1,2-Dichloroethane)	OSHA 3	—	—	0.05 ppm	
1169.	Ethylene Glycol	—	NIOSH 5500	—	0.8 ppm	
1170.	Ethylene Glycol Dinitrate	OSHA 43	—	—	2 ppb	DL based on 15 L air vol
1171.	Ethylidene Norbornene	—	—	XXXX		
1172.	N-Ethylmorpholine	NIOSH S146	—	—	0.1 ppm	
1173.	Fenamiphos	—	In-house	—	0.004 mg/M^3	DL based on 480 L air vol
1174.	Fensulfothion (Dasanit)	—	In-house	—	0.02 mg/M^3	DL based on 200 L air vol
1175.	Fenthion (Tiguvon)	—	In-house	—	0.004 mg/M^3	DL based on 480 L air vol
1176.	Ferbam	—	In-house	—	0.08 mg/M^3	DL based on 60 L air vol
1177.	Ferrovanadium Dust	ID121, ID125G	—	—	2 ug/M^3	DL based on Fe or V & 480 L air vol
1178.	Fibrous Glass Dust	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1179.	Fluorine	—	In-house	—	0.1 mg/M^3	As F^-, DL based on 240 L air vol
1180.	Fluorotrichloromethane	NIOSH 1006	—	—	1 ppm	DL based on 3 L air vol
1181.	Fonofos (Dyfonate)	—	In-house	—	0.004 mg/M^3	DL based on 480 L air vol
1182.	Formamide	—	In-house	—	0.5 ppm	DL based on 10 L air vol
1183.	Furfural	OSHA 72	—	—	0.1 ppm	DL based on 10 L air vol
1184.	Furfuryl Alcohol	NIOSH S365	—	—	0.5 ppm	
1185.	Gasoline	—	In-house	—	0.9 mg/M^3	DL based on 10 L air vol
1186.	Germanium Tetrahydride	—	In-house	—	0.001 mg/M^3	As Ge by (HGA AAS) & 240 L air vol
1187.	Glutaraldehyde	OSHA 64	—	—	4.4 ppb	
1188.	Glycerin (Mist)	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1189.	Glycidol (2,3-Epoxy-1-Propanol)	NIOSH 1608	—	—	1 ppm	DL based on 10 L air vol
1190.	Grain Dust (oat, wheat, barley)	GRAVIMETRIC	—	—	0.02 mg/M^3	
1191.	Graphite (Natural, respirable)	GRAV & ID142	—	—	0.02 mg/M^3	
1191A.	Graphite (Synthetic)	GRAVIMETRIC	—	—	0.02 MG/M^3	Same as nuisance particulate TLV
1192.	Gypsum, Total Dust-	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV

SAMPLING AND ANALYTICAL METHODS

H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1194.	n-Heptane	NIOSH 1500	—	—	0.2 ppm	
1195.	Hexachlorobutadiene	NIOSH 307	—	—	0.2 ppm	DL based on 100 L air vol
1196.	Hexachlorocyclopentadiene	—	NIOSH 2518	—	1 ppb	
1197.	Hexachloroethane	NIOSH 1003	—	—	0.5 ppm	DL based on 10 L air vol
1198.	Hexafluoroacetone	—	—	XXXX		
1200.	n-Hexane	NIOSH 1500	—	—	0.05 ppm	
1201.	Hexane Isomers	—	NIOSH 1500	—	0.5 ppm	Adapt NIOSH method to this compound
1202.	2-Hexanone	NIOSH 1300	—	—	0.5 ppm	
1203.	Hexone (Methyl Isobutyl Ketone)	NIOSH 1300	—	—	0.05 ppm	DL based on 10 L air vol
1204.	Hexylene Glycol	—	In-house	—	0.5 ppm	DL based on 10 L air vol
1205.	Hydrazine	OSHA 20	—	—	1.2 ppb	
1206.	Hydrogen Bromide	—	In-house	—	0.6 ppb	DL based on 96 L air vol
1207.	Hydrogen Cyanide	OSHA ID120	—	—	0.03 ppm	DL based on 90 L air vol
1208.	Hydrogen Fluoride	—	In-house	—	0.1 ppm	DL based on 240 L air vol
1209.	Hydrogen Sulfide	OSHA ID141	—	—	0.9 ppm	DL based on 2 L air vol
1210.	Hydrogenated Terphenyls	—	NIOSH 5021	—	0.1 mg/M^3	Adapt NIOSH method to this compound
1211.	2-Hydroxypropyl Acrylate	—	NIOSH S43	—	1 ppm	Adapt NIOSH method to this compound
1212.	Indene	—	In-house	—	0.01 ppm	DL based on 10 L air vol
1213.	Indium & Compounds	—	In-house	—	0.02 mg/M^3	As In. DL based on 480 L air vol
1214.	Iodoform	—	In-house	—	0.01 ppm	DL based on 10 L air vol
1215.	Iron Oxide, (Dust & Fume)	ID121, ID125G	—	—	3 ug/M^3	As Fe_2O_3. DL based on 480 L air vol
1216.	Iron Pentacarbonyl (as Fe)	—	COLORIMETRIC	—	0.04 mg/M^3	DL based on 240 L air vol
1217.	Iron Salts, Soluble, as Fe-	OSHA ID121	—	—	2 ug/M^3	As Fe water soluble. DL based on 480 L
1218.	Isoamyl Alcohol	NIOSH 1402	—	—	0.5 ppm	
1219.	Isobutyl Alcohol	NIOSH 1401	—	—	0.03 ppm	
1220.	Isooctyl Alcohol	—	NIOSH 1400	—	0.5 ppm	Adapt NIOSH method to this compound
1221.	Isophorone	NIOSH 2508	—	—	0.07 ppm	DL based on 12 L air vol
1222.	Isophorone Diisocyanate	—	OSHA 42	—	0.02 mg/M^3	DL based on 15 L air vol
1223.	2-Isopropoxyethanol	—	OSHA 53	—	0.5 ppm	Adapt OSHA method to this compound
1224.	Isopropyl Acetate	NIOSH S50	—	—	0.5 ppm	

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H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1225.	Isopropyl Alcohol	NIOSH 1400	—	—	0.2 ppm	
1226.	Isopropyl Ether	NIOSH S368	—	—	0.5 ppm	
1227.	Isopropyl Glycidyl Ether (IGE)	NIOSH S77	—	—	0.5 ppm	
1228.	Isopropylamine	NIOSH S147	—	—	0.5 ppm	
1229.	N-Isopropylaniline	—	NIOSH 2002	—	0.5 ppm	Adapt NIOSH method to this compound
1230.	Kaolin, Total Dust	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1231.	Ketene	NIOSH S92	—	—	0.2 mg/M^3	DL based on 50 L air vol
1232.	Limestone, Total Dust	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1233.	Magnesite, Total Dust	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1234.	Magnesium Oxide fume	OSHA ID121	—	—	4 ug/M^3	As total Mg, DL based on 480 L air vol
1235.	Malathion	OSHA 62	—	—	2.2 ppb	DL based on 60 L air vol
—	Manganese, as Mn					
—	Dust & Compounds-	ID121, ID125G	—	—	0.07 mg/M^3 \	As Mn, Method does not distinguish
1236A.	Fume-	ID121, ID125G	—	—	0.004 mg/M^3 /	fume from dust.
1237.	Manganese Cyclopentadienyl Tricarbonyl, as Mn-	—	In-house	—	0.004 mg/M^3	Total Mn by AAS, 240 L air vol
1238.	Manganese Tetroxide	ID121, ID125G	—	—	0.004 mg/M^3	DL based on total Mn & 30 L air vol
1239.	Marble, Total Dust	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
—	Mercury, as Hg					
1240.	Aryl & Inorganic Compounds-	—	In-house	—	0.001 mg/M^3	DL based on 15 L air vol
1241.	Vapor-	—	In-house	—	0.001 mg/M^3	DL based on 15 L air vol
1242.	Alkyl Compounds-	—	—	XXXX		
1243.	Mesityl Oxide	NIOSH 1301	—	—	0.5 ppm	
1244.	Methacrylic Acid	—	In-house	—	0.03 ppm	DL based on 20 L air vol
1245.	Methomyl (Lannate)	—	In-house	—	0.03 mg/M^3	DL based on 60 L air vol & OVS-2 Sampler
1246.	Methoxychlor	—	OSHA-NIOSH S371	—	0.5 mg/M^3	DL based on 100 L air vol
1247.	4-Methoxyphenol	—	OSHA 32	—	0.5 ppm	Use OSHA method for Cresol
1248.	Methyl-2-Cyanoacrylate	OSHA 55	—	—	0.01 ppm	
1249.	Methyl Acetate	NIOSH S42	—	—	0.5 ppm	
1250.	Methyl Acetylene/Propadiene Mixture (MAPP)-	—	In-house	—	2.5 ppm	DL based on 5 L air vol

SAMPLING AND ANALYTICAL METHODS

H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1251.	Methyl Acrylonitrile	—	OSHA 37	—	0.1 ppm	Adapt OSHA method to this compound
1252.	Methyl Alcohol	OSHA-NIOSH 2000	—	—	1.5 ppm	
1253.	Methyl Bromide	NIOSH 2520	—	—	0.5 ppm	
1254.	Methyl Chloride	NIOSH 1001	—	—	0.5 ppm	DL based on 10 L air vol
1255.	Methyl Chloroform (1,1,1-Trichloroethane)	OSHA 14	—	—	0.07 ppm	
1256.	Methyl Demeton	—	In-house	—	0.03 mg/M^3	DL based on 60 L air vol
1257.	Methyl Ethyl Ketone Peroxide	—	In-house	—	0.05 mg/M^3	DL based on 15 L air vol
1258.	Methyl Formate	NIOSH S291	—	—	1 ppm	DL based on 10 L air vol
1259.	Methyl Iodide	NIOSH 1014	—	—	0.5 ppm	
1260.	Methyl Isoamyl Ketone	—	In-house	—	0.5 ppm	DL based on 10 L air vol
1261.	Methyl Isobutyl Carbinol (Methyl Amyl Alcohol)	NIOSH 1402	—	—	0.5 ppm	
1262.	Methyl Isopropyl Ketone	—	OSHA 7	—	0.5 ppm	Adapt OSHA method to this compound
1263.	Methyl Mercaptan (Methanethiol)	OSHA 26	—	—	0.03 ppm	
1264.	Methyl-(n-amyl) Ketone	NIOSH 1301	—	—	0.5 ppm	
1265.	Methyl Parathion	—	In-house	—	0.002 mg/M^3	DL based on 480 L air vol
1266.	Methyl Silicate	—	NIOSH S264	—	0.5 ppm	Adapt NIOSH method to this compound
1267.	alpha-Methyl Styrene	NIOSH 1501	—	—	1 ppm	
1268.	Methylcyclohexane	NIOSH 1500	—	—	0.5 ppm	
1269.	Methylcyclohexanol	NIOSH S374	—	—	0.5 ppm	
1270.	o-Methylcyclohexanone	NIOSH 2521	—	—	0.5 ppm	
1271.	Methylcyclopentadienyl -Mn-Tricarbonyl, as Mn-	—	In-house	—	0.004 mg/M^3	Total Mn by AAS, & 240 L air vol
1272.	Methylene Bis (4-Cyclo-hexylisocyanate)	—	In-house	—	0.02 mg/M^3	DL based on 15 L air vol
1273.	4,4'-Methylene-Bis-(2-Chloroaniline)	OSHA 71	—	—	3.6 ug/M^3	DL based on 100 L air vol
—	Methylene Bisphenyl Isocyanate (MDI)	OSHA 47	—	—	2.6 ug/M^3	DL based on 15 L air vol

SAMPLING AND ANALYTICAL METHODS

H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1275.	Metribuzin	—	In-house	—	0.1 mg/M^3	DL based on 120 L air vol
1276.	Mica	GRAV & ID142	In-house	—	0.02 mg/M^3	Regulated as Quartz if >1% Quartz, 400 L
1277.	Mineral Wool Fiber	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1278.	Molybdenum Insoluble Compounds, as Mo-	ID121, ID125G	—	—	4 ug/M^3	as Mo, DL based on 480 L air vol
1279.	Monocrotophos	—	In-house	—	0.01 mg/M^3	DL based on 240 L air vol
1280.	Monomethyl Aniline	NIOSH S153	—	—	0.1 ppm	
1281.	Morpholine	NIOSH S150	—	—	0.5 ppm	
1282.	Naphthalene	OSHA 35	—	—	80 ppb	
1283.	Nickel Soluble Compounds as Ni-	ID121, ID125G	—	—	4 ug/M3	As Ni, DL based on 480 L air vol
1284.	Nickel Carbonyl	—	In-house	—	0.002 mg/M^3	Based on total Ni & 240 L air vol
1286.	Nitric Acid	OSHA ID127	OSHA ID165SG	—	0.5 ppb	DL based on 96 L air vol
1287.	p-Nitroaniline	NIOSH S7	—	—	0.5 ppm	
1288.	p-Nitrochlorobenzene	NIOSH 2005	—	—	0.1 ppm	
1289.	Nitrogen Dioxide	OSHA ID182	—	—	0.2 ppm	DL based on 3 L air vol
1290.	Nitroglycerin (NG)	OSHA 43	—	—	2 ppb	DL based on 15 L air vol
1291.	2-Nitropropane	OSHA 46	—	—	0.025 ppm	
1292.	Nitrotoluene	NIOSH 2005	—	—	0.1 ppm	
1293.	Nonane	—	NIOSH 1500	—	0.2 ppm	Adapt NIOSH method to this compound
1294.	Nuisance Particulates, Total Dust-	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1295.	Octachloronaphthalene	NIOSH S97	—	—	0.05 mg/M^3	
1296.	Octane	NIOSH 1500	—	—	0.5 ppm	
1297.	Oil Mist, (Mineral)	GRAV & In-house	—	—	0.1 mg/M^3	DL varies based on oil & 480 L air vol
1298.	Osmium Tetroxide	—	In-house	—	0.002 mg/M^3	Neutron Activation Analysis, Total Os
1299.	Oxalic Acid	—	In-house	—	0.02 mg/M^3	DL based on 480 L air vol
1300.	Oxygen Difluoride	—	—	XXXX		
1301.	Ozone	—	In-house	—	0.01 ppm	Chemiluminescence, direct read
1302.	Paraffin Wax fume	—	In-house	—	0.5 mg/M^3	DL based on 120 L air vol

SAMPLING AND ANALYTICAL METHODS

H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1303.	Paraquat, Respirable Dust-	NIOSH 5003	—	—	0.1 mg/M^3	DL based on 90 L air vol
1304.	Pentaborane	—	In-house	—	0.01 mg/M^3	Based on B & 240 L air vol
1305.	Pentaerythritol, Total Dust-	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1306.	Pentane	NIOSH 1500	—	—	0.5 ppm	DL based on 10 L air vol
1307.	2-Pentanone (Methyl Propyl Ketone)	NIOSH 1300	—	—	0.5 ppm	DL based on 10 L air vol
1308.	Perchloroethylene (Tetrachloroethylene)	NIOSH 1003	—	—	0.05 ppm	DL based on 10 L air vol
1309.	Perchloryl Fluoride	—	In-house	—	0.6 mg/M^3	Based on F^- & 240 L air vol
1310.	Perlite	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1312.	Petroleum Distillates (Naphtha)	OSHA 48	—	—	$\leq$260 mg/M^3	DL based on 3 L air vol
1313.	Phenothiazine	—	In-house	—	0.1 mg/M^3	DL based on 240 L air vol
1314.	Phenyl Ether (Vapor)	NIOSH S72	—	—	0.1 ppm	
1315.	Phenyl Glycidyl Ether	NIOSH S74	—	—	0.1 ppm	
1316.	Phenylmercaptan	—	OSHA 26	—	0.1 ppm	Adapt OSHA method to this compound
1317.	Phenylhydrazine	NIOSH S160	—	—	5 mg/M^3	DL based on 120 L air vol
1318.	Phenylphosphine	—	—	XXXX		
1319.	Phorate (Thimet)	—	In-house	—	0.003 mg/M^3	DL based on 480 L air vol
1320.	Phosdrin (Mevinphos)	NIOSH 2503	—	—	0.001 mg/M^3	DL based on 240 L air vol
1321.	Phosphine	OSHA ID180	—	—	0.015 ppm_3	DL based on 36 L air vol
1322.	Phosphoric Acid	OSHA ID111	OSHA ID165SG	—	0.01 mg/M^3	DL based on 480 L air vol
1323.	Phosphorus Oxychloride	—	In-house	—	0.002 mg/M^3	DL based on PO_4^{-3} & 240 L air vol
1324.	Phosphorus Pentasulfide	—	In-house	—	0.02 mg/M^3	DL based on 60 L air vol
1325.	Phosphorus Trichloride	—	NIOSH 6402	—	1.1 mg/M^3	DL based on 24 L air vol
1326.	Phthalic Anhydride	—	In-house	—	0.38 mg/M^3	DL based on 27 L air vol
1327.	m-Phthalodinitrile	—	OSHA 37	—	0.5 mg/M^3	Adapt OSHA method to this compound
1328.	Picloram (Tordom)	—	In-house	—	0.03 mg/M^3	DL based on 60 L air vol
1329.	Picric Acid (2,4,6-Trinitrophenol)	NIOSH S228	—	—	0.04 mg/M^3	DL based on 180 L air vol

SAMPLING AND ANALYTICAL METHODS

H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1330.	Piperazine Dihydrochloride	—	In-house	—	1 ppm	DL based on 10 L air vol
1331.	Plaster of Paris Total Dust-	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1332.	Platinum, metal-	—	In-house	—	0.2 ug/M^3	DL based on 480 L air vol
1333.	Portland Cement	GRAV & ID142	—	—	0.02 mg/M^3	Use Quartz std if >1% Quartz
1334.	Potassium Hydroxide	—	OSHA ID121	—	0.03 mg/M^3	Bases on water soluble K^+ & 480 L air
1335.	Propargyl Alcohol	—	NIOSH 1400	—	0.1 ppm	Adapt NIOSH method to this compound
1336.	Propionic Acid	—	In-house	—	0.03 ppm	DL based on 10 L air vol
1337.	Propoxur (Baygon)	—	In-house	—	0.003 mg/M^3	DL based on 60 L air vol
1338.	n-Propyl Acetate	NIOSH 1450	—	—	0.5 ppm	DL based on 10 L air vol
1339.	Propyl Alcohol	NIOSH 1401	—	—	0.5 ppm	
1340.	n-Propyl Nitrate	NIOSH S227	—	—	1 ppm	
1341.	Propylene Dichloride	NIOSH 1003	—	—	0.5 ppm	
1342.	1,2-Propylene Glycol Dinitrate	—	OSHA 43	—	0.2 mg/M^3	Use OSHA 43 for EGDN, & 15 L air vol
1343.	Propylene Glycol Monomethyl Ether	—	OSHA 53	—	0.5 ppm	Adapt OSHA method to this compound
1344.	Propylene Oxide	NIOSH 1612	—	—	0.1 ppm	
—	Pyridine	NIOSH 1613	—	—	0.5 ppm	
1346.	Resorcinol	—	In-house	—	0.02 mg/M^3	DL based on 120 L air vol
—	Rhodium, metal-	—	In-house	—	0.1 ug/M^3	DL based on 480 L air vol
1347.	Insoluble Compounds as Rh-	—	In-house	—	0.1 ug/M^3	DL based on 480 L air vol
1348.	Soluble Compounds as Rh-	—	In-house	—	0.1 ug/M^3	DL based on 480 L air vol
1349.	Ronnel	NIOSH S299	—	—	0.002 mg/M^3	DL based on 120 L air vol
1350.	Rosin Core Solder- (Pyrolysis Products, as Formaldehyde)-	OSHA 52	In-house	—	—	/Sample for Formaldehyde (OSHA 52) and < Abietic Acid (OSHA In-house) as per \ Chemical Information File
1351.	Rouge, (Total Dust)	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV

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H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1352.	Silica - Amorphous \ Diatomaceous Earth \ (Uncalcined)- >	GRAV & ID142	—	—	0.02 mg/M^3	If >1% Quartz, use Quartz standard
1353.	Percipitated Silica- /					
—	Silica Gel- /					
—	Silica - Crystalline					
1354.	Cristobalite-	OSHA ID142	—	—	0.05 mg/M^3	DL based on 816 L air vol
1355.	Quartz-	OSHA ID142	—	—	0.02 mg/M^3	DL based on 480 L air vol
1356.	Tridymite-	OSHA ID142	—	—	0.05 mg/M^3	
1357.	Tripoli-	OSHA ID142	—	—	0.02 mg/M^3	Quartz analysis
1358.	Silica, Fused-	OSHA ID142	—	—	0.02 mg/M^3	If >1% Quartz, use Quartz standard
1359.	Silicon	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1360.	Silicon Carbide	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1361.	Silicon Tetrahydride (Silane)	—	In-house	—	0.002 mg/M^3	DL based on Si anal & 240 L air vol
1362.	Silver metal-	OSHA ID121	—	—	0.5 ug/M^3	DL based on 480 L air vol
	Soluble Compounds as Ag	OSHA ID121	—	—	0.5 ug/M^3	DL based on 480 L air vol
1363.	Soapstone, Respirable & Total-	GRAV & ID142	—	—	0.02 mg/M^3	If >1% Quartz, use Quartz standard
1364.	Sodium Azide	—	In-house	—	0.06 mg/M^3	DL based on 15 L air vol
1365.	Sodium Bisulfite	—	In-house	—	0.04 mg/M^3	Based on Na & 480 L air vol
1366.	Sodium Fluoroacetate	—	In-house	—	0.04 mg/M^3	Based on Na & 480 L air vol
1367.	Sodium Hydroxide	—	OSHA ID121	—	0.04 mg/M^3	Based on Na & 480 L air vol
1368.	Sodium Metabisulfite	—	In-house	—	0.04 mg/M^3	Based on Na & 480 L air vol
1369.	Starch, Total Dust	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1371.	Stoddard Solvent	OSHA 48	—	—	<260 mg/M^3	DL based on 3 L air vol
1372.	Styrene, Monomer	OSHA 9	—	—	3.1 ppm	
1373.	Subtilisins (Proteolytic Enzymes)	—	In-house	—	10 pg/M^3	DL based on 15 M^3 air vol
1374.	Sucrose, Total Dust	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1375.	Sulfur Dioxide	OSHA ID107	—	—	0.07 mg/M^3	DL based on 24 L air vol
1376.	Sulfur Monochloride	—	In-house	—	0.01 mg/M^3	Based on Cl^- & 15 L air vol

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H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1377.	Sulfur Pentafluoride	—	—	XXXX		
1378.	Sulfur Tetrafluoride	—	In-house	—	0.05 mg/M^3	Based on F^- & 15 L air vol
1379.	Sulfuryl Fluoride	—	Field test	—	0.1 ppm	Miran 1A & 1B: MIN. Detectable Conc. = 0.1 ppm at 11.5 um
1380.	Sulprofos	—	In-house	—	0.03 mg/M^3	DL based on 60 L air vol
1381.	Talc (non-Asbestiform)	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1382.	Tantalum, Metal & Oxide Dust-	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1383.	Temephos	—	In-house	—	0.02 mg/M^3	DL based on 60 L air vol
1384.	Terphenyls	NIOSH 5021	—	—	0.1 ppm	
1385.	1,1,2,2-Tetrachloroethane	NIOSH 1019	—	—	0.1 ppm	
1386.	Tetraethyl Lead (as Pb)	—	In-house	—	0.004 mg/M^3	DL based on 240 L air vol & total Pb
1387.	Tetrahydrofuran	NIOSH 1609	—	—	1 ppm	
1388.	Tetramethyl Lead (as Pb)	—	In-house	—	0.004 mg/M^3	DL based on 240 L air vol & total Pb
1389.	Tetrasodium Pyrophosphate	—	In-house	—	0.02 mg/M^3	Based on Na & 480 L air vol
—	Thallium, Soluble Compounds-	—	OSHA ID121	—	0.03 mg/M^3	DL based on 480 L air vol
1391.	4,4'-Thio-bis(6-tert-Butyl-m-Cresol)	—	In-house	—	0.04 mg/M^3	DL based on 120 L air vol
1392.	Thioglycolic Acid	—	In-house	—	0.07 ppm	DL based on 10 L air vol
1393.	Thionyl Chloride	—	In-house	—	0.01 mg/M^3	Based on Cl^- & 15 L air vol
—	Tin					
1394.	Organic Compounds, as Sn-	—	In-house	—	0.01 mg/M^3	Only Org-Sn's listed in Chem Info File
1395.	Oxide-	—	In-house	—	0.03 mg/M^3	DL based on 480 L air vol
1396.	Titanium Dioxide	GRAVIMETRIC	—	—	0.02 mg/M^3	Same as nuisance particulate TLV
1397.	Toluene	NIOSH 4000	—	—	0.02 ppm	DL based on 10 L air vol
1398.	Toluene-2,4-Diisocyanate (TDI)	OSHA 42	—	—	0.36 ppb	DL based on 15 L air vol
1399.	o-Toluidine	OSHA 73	—	—	0.1 ppm	
1400.	p-Toluidine	OSHA 73	—	—	0.1 ppm	
1401.	m-Toluidine	OSHA 73	—	—	0.1 ppm	

SAMPLING AND ANALYTICAL METHODS

H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1402.	Tributyl Phosphate	OSHA-NIOSH S208	—	—	0.5 mg/M^3	DL based on 100 L air vol
1403.	1,1,2-Trichloro-1,2,2-Trifluoroethane	NIOSH 1020	—	—	1.4 ppm	DL based on 1.5 L air vol
1404.	Trichloroacetic Acid	—	NIOSH 1603	—	0.1 ppm	Adapt NIOSH method to this compound
1405.	1,2,4-Trichlorobenzene	—	NIOSH 5517	—	0.5 ppm	DL based on 10 L air vol
1406.	Trichloroethylene	NIOSH 1022, 3701	—	—	0.2 ppm	DL based on 3 L air vol
1407.	1,2,3-Trichloropropane	NIOSH 1003	—	—	0.5 ppm	DL based on 10 L air vol
1408.	Triethylamine	NIOSH S152	—	—	0.5 ppm	
1409.	Trimellitic Anhydride	—	In-house	—	0.001 mg/M^3	DL based on 480 L air vol
1410.	Trimethyl Phosphite	—	In-house	—	0.5 ppm	DL based on 120 L air vol
1411.	Trimethylamine	—	In-house	—	0.5 ppm	DL based on 10 L air vol
1412.	Trimethylbenzene	—	In-house	—	0.02 ppm	DL based on 10 L air vol
1413.	2,4,6-Trinitrotoluene (TNT)	OSHA 44	—	—	0.021 mg/M^3	
1414.	Triorthocresyl Phosphate	OSHA-NIOSH S209	—	—	0.01 mg/M^3	DL based on 100 L air vol
1415.	Triphenylamine	—	In-house	—	0.5 ppm	DL based on 10 L air vol
—	Tungsten, as W					
1416.	Insoluble Compounds-	—	In-house	—	0.002 mg/M^3	480 L & Neutron Activation Analysis
1417.	Soluble Compounds-	—	In-house	—	0.002 mg/M^3	480 L & Neutron Activation Analysis
—	Uranium					
1418.	Insoluble Compounds-	—	In-house	—	0.8 ug/M^3	DL based on 480 L air vol
1419.	Soluble Compounds-	—	OSHA ID170SG	—	0.8 ug/M^3	DL based on 480 L air vol
1420.	N-Valeraldehyde	—	OSHA 68	—	1 ppm	Adapt OSHA method to this compound
1421.	Vanadium, as (V_2O_5)					
1421.	Respirable Dust-	OSHA ID125G	OSHA ID185	—	0.004 mg/M^3	Based on total V analysis & 480 L
1422	Fume-	OSHA ID125G	OSHA ID185	—	0.004 mg/M^3	Based on total V analysis & 480 L
1423.	Vegetable Oil Mist	GRAVIMETRIC	—	—	—	Same as nuisance particulate TLV
1424.	Vinyl Acetate	OSHA 51	—	—	0.01 ppm	
1425.	Vinyl Bromide	OSHA 8	—	—	0.2 ppm	
1426.	Vinyl Cyclohexene Dioxide	—	In-house	—	0.5 ppm	DL based on 10 L air vol
1427.	Vinyl Toluene	NIOSH 1501	—	—	0.5 ppm	
1428.	Vinylidene Chloride	OSHA 19	—	—	0.05 ppm	

SAMPLING AND ANALYTICAL METHODS

H.S. NO.	ANALYTE	VALIDATED METHOD	OTHER METHOD	NO METHOD	DET LIMIT[1]	COMMENTS
1429.	VM&P Naphtha	OSHA 48	—	—	$\leq$260 mg/M^3	DL based on 3 L air vol
1430.	Welding Fumes (Total Particulate)	GRAV & ID125G	—	—	—	See Det. Lim. for specific metals
1430A.	Wood Dust	GRAVIMETRIC	—	—	0.02 mg/M^3	
1431.	Xylene (o-,m-,p-isomers)	NIOSH 1501	—	—	0.02 ppm	DL based on 12 L air vol
1432.	m-Xylene-alpha,alpha'-Diamine	—	In-house	—	0.1 ppm	DL based on 10 L air vol
1433.	Xylidine	NIOSH 2002	—	—	2.5 mg/M^3	DL based on 20 L air vol
1434.	Zinc Stearate	—	OSHA ID121	—	0.003 mg/M^3	Based on Zn & 480 L air vol
1435.	Zinc Chloride fume	—	OSHA ID121	—	0.006 mg/M^3	Based on Zn & 480 L air vol
1436.	Zinc Chromates, as (CrVI)-	OSHA ID103	—	—	0.4 ug/M^3	As Cr^{+6}. DL based on 480 L air vol
—	Zinc Oxide					
1437.	Fume-	GRAV & ID121,			0.003 mg/M^3 \	Total Zn. Does not differentiate
1438.	Dust-	ID125G	ID143	—	0.003 mg/M^3 /	fume & dust
1439.	Zirconium Compounds	GRAV & ID121	—	—	1 mg/M^3	DL based on 480 L air vol

1. Detection Limits (DL) are approximate values based on the analytical procedures recommended air volume or the air volume cited in the "comments" section.

2. "In-house" refers to analytical methods used by OSHA that have not been fully validated by either NIOSH or OSHA. These procedures have been developed by OSHA or were taken from the literature. Some literature methods may not have been used by OSHA as yet.

NIOSH ANALYTICAL METHODS FOR PEL UPDATE

H.S. No.	ANALYTE	VALIDATED METHOD	OTHER METHOD
1001	Acetaldehyde	NIOSH 3507	
1002	Acetic acid	NIOSH 1603	
1003	Acetic anhydride	NIOSH 3506	
1004	Acetone	NIOSH 1400	
1005	Acetonitrile	NIOSH 1606	
1007	Acrolein	NIOSH 2501	
1010	Allyl alcohol	NIOSH 1402	
1011	Allyl chloride	NIOSH 1000	
1012	Allyl glycidyl ether (AGE)	NIOSH S346	
1021	Ammonia	NIOSH 6701	
1024	Ammonium Sulfamate (Ammate)	NIOSH 5348	
1025	Aniline	NIOSH 2002	
----	ANTU (Alpha-Naphthyl Thiourea)	NIOSH S276	
----	Arsenic	NIOSH 7900	
1033	Beryllium & compounds	NIOSH 7102	
1039	Boron Oxide	NIOSH 500, 600	
1045	2-Butanone (MEK)	NIOSH 2500	
1046	2-Butoxy ethanol	NIOSH 1403	
1047	n-Butyl acetate	NIOSH 1450	
1049	sec-Butyl alcohol	NIOSH 1401	
1050	tert-Butyl alcohol	NIOSH 1400	
1051	n-Butyl alcohol	NIOSH 1401	
1052	n-Butyl glycidyl ether (BGE)	NIOSH S81	
1054	Butyl mercaptan	NIOSH S350	
1056	p-tert-Butyltoluene	NIOSH 1501	
1057	Calcium carbonate (Limestone, Marble)	NIOSH 500, 600	
1059	Calcium hydroxide	NIOSH 7020	
1060	Calcium oxide	NIOSH 7020	
1062	Calcium Sulfate (Plaster of Paris)	NIOSH 500, 600	
1063	Camphor, synthetic	NIOSH 1301	
1069	Carbon Dioxide	NIOSH 5249	
1070	Carbon Disulfide	NIOSH 1600	
1071	Carbon Monoxide	NIOSH S340	
1073	Carbon Tetrachloride (Tetrachloromethane)	NIOSH 1003	
1076	Cellulose (paper fiber)	NIOSH 500, 600	
1078	Chlorinated Camphene (Toxaphene)	NIOSH S67	
1081	1-Chloro-1-nitropropane	NIOSH S211	
1084	o-Chlorobenzylidene Malononitrile		NIOSH 304

NIOSH ANALYTICAL METHODS FOR PEL UPDATE

H.S. No.	ANALYTE	VALIDATED METHOD	OTHER METHOD
1085	Chlorodifluoromethane		NIOSH 1020
1086	Chloroform	NIOSH 1003	
1087	Chloropentafluoroethane		NIOSH 1020
1088	beta-Chloroprene	NIOSH 1002	
1089	o-Chlorostyrene		NIOSH 1003
1090	o-Chlorotoluene		NIOSH 1003
1092	Chromic Acid & Chromates	NIOSH 7600	
1093	Chromium, metal	NIOSH 7024, 7300	
1100	Cobalt, as Co Metal dust & fume-	NIOSH 7027, 7300	
1101	Copper, Fume-	NIOSH 7029,7200,7300	
1102	Crag Herbicide (Sesone)	NIOSH S356	
1107	Cyclohexanol	NIOSH 1402	
1108	Cyclohexanone	NIOSH 1300	
1111	Cyclopentane		NIOSH 1500
1113	DDT (Dichlorodiphenyl-trichloroethane)	NIOSH S274	
----	Demeton (Systox)	NIOSH 5514	
1117	2,6-Di-tert-Butyl-p-cresol		NIOSH 226
1119	Dibutyl Phosphate		NIOSH 5017
1120	2-N-Dibutylaminoethanol		NIOSH 2007
1121	1,1-Dichloro-1-nitroethane	NIOSH 1601	
1123	Dichloroacetylene		NIOSH 1003
----	o-Dichlorobenzene	NIOSH 1003	
1125	p-Dichlorobenzene	NIOSH 1003	
1126	1-1-Dichloroethane	NIOSH 1003	
1127	Dichloroethyl Ether	NIOSH 1004	
1128	Dichlorofluoromethane	NIOSH 2516	
1129	1,3-Dichloropropene		NIOSH 1003
1140	Diisobutyl ketone	NIOSH 1300	
1142	Dimethyl Sulfate		NIOSH 301
1143	Dimethylaniline (N,N-Dimethylaniline)	NIOSH 2002	
1145	Dioxane (Diethylene Dioxide)	NIOSH 1602	
1149	Dipropylene Glycol Methyl Ether	NIOSH S69	
1158	Epichlorohydrin	NIOSH 1010	
1159	Ethanolamine	NIOSH 2007	
1161	Ethyl Acrylate	NIOSH 1450	
1162	Ethyl Benzene	NIOSH 1501	
1163	Ethyl Bromide	NIOSH 1011	
1164	Ethyl Ether	NIOSH 1610	
1166	Ethyl Silicate	NIOSH S264	
1167	Ethylene Chlorohydrin	NIOSH 2513	

NIOSH ANALYTICAL METHODS FOR PEL UPDATE

H.S. No.	ANALYTE	VALIDATED METHOD	OTHER METHOD
1168	Ethylene Dichloride (1 2-Dichloroethane)	NIOSH 1003	
1169	Ethylene Glycol	NIOSH 5500	
1170	Ethylene Glycol Dinitrate	NIOSH 2507	
1172	N-Ethylmorpholine	NIOSH S146	
1180	Fluorotrichloromethane	NIOSH 1006	
1183	Furfural	NIOSH 2529	
1184	Furfuryl Alcohol	NIOSH S365	
1189	Glycidol (2,3 Epoxy-1-Propanol)	NIOSH 1608	
1191	Graphite (Natural, Respirable)	NIOSH 500, 600	
1192	Gypsum, Total dust-	NIOSH 500, 600	
1194	n-Heptane	NIOSH 1500	
1195	Hexachlorobutadiene	NIOSH 307	
1196	Hexachlorocyclopentadiene		NIOSH 308
1197	Hexachloroethane	NIOSH 1003	
1200	n-Hexane	NIOSH 1500	
1201	Hexane Isomers		NIOSH 1500
1202	2-Hexanone	NIOSH 1300	
1203	Hexone (Methyl Isobutyl Ketone)	NIOSH 1300	
1205	Hydrazine	NIOSH 3503	
1206	Hyrogen Bromide	NIOSH 7903	
1207	Hydrogen Cyanide	NIOSH 7904	
1208	Hydrogen Fluoride	NIOSH 7903	
1210	Hydrogenated Terphenyls		NIOSH 5021
1211	2-Hydroxypropyl Acrylate		NIOSH S43
1218	Isoamyl Alcohol	NIOSH 1402	
1219	Isobutyl Alcohol	NIOSH 1401	
1220	Isooctyl Alcohol		NIOSH 1400
1221	Isophorone	NIOSH 2508	
1224	Isopropyl Acetate	NIOSH S50	
1225	Isopropyl Alcohol	NIOSH 1400	
1226	Isopropyl Ether	NIOSH S368	
1227	Isopropyl Glycidyl Ether (IGE)	NIOSH S77	
1228	Isopropylamine	NIOSH S147	
1229	N-Isopropylaniline		NIOSH 2002
1231	Ketene	NIOSH S92	
1232	Limestone, Total dust	NIOSH 500, 600	
1233	Magnesite, total dust	NIOSH 500, 600	
1239	Marble, Total Dust	NIOSH 500, 600	
1243	Mesityl Oxide	NIOSH 1301	
1246	Methoxychlor		NIOSH S371

NIOSH ANALYTICAL METHODS FOR PEL UPDATE

H.S. No.	ANALYTE	VALIDATED METHOD	OTHER METHOD
1249	Methyl Acetate	NIOSH S42	
1250	Methyl Acetylene/Propadiene Mixture (MAPP)-	NIOSH S85	
1252	Methyl Alcohol	NIOSH S59	
1253	Methyl Bromide	NIOSH 2520	
1254	Methyl Chloride	NIOSH 1001	
1255	Methyl Chloroform (1,1,1-Trichloroethane)	NIOSH 1003	
1257	Methyl Ethyl Ketone Peroxide		NIOSH 3508
1258	Methyl Formate	NIOSH S291	
1259	Methyl Iodide	NIOSH 1014	
1261	Methyl Isobutyl Carbinol (Methyl Amyl Alcohol)	NIOSH 1402	
1264	Methyl (n-Amyl)Ketone	NIOSH 1301	
1266	Methyl Silicate		NIOSH S264
1267	alpha-Methyl Styrene	NIOSH 1501	
1268	Methylcyclohexane	NIOSH 1500	
1269	Methylcyclohexanol	NIOSH S374	
1270	o-Methylcyclohexanone	NIOSH 2521	
1277	Mineral Wool Fiber	NIOSH 500, 600	
1280	Monomethyl Aniline	NIOSH S153	
1281	Morpholine	NIOSH S150	
1282	Naphthalene	NIOSH 1501	
1284	Nickel Carbonyl		NIOSH 6007
1286	Nitric Acid	NIOSH 7903	
1287	p-Nitroaniline	NIOSH S7	
1288	p-Nitrochlorobenzene	NIOSH 2005	
1289	Nitrogen Dioxide		NIOSH 6700
1290	Nitroglycerin (NG)	NIOSH 2507	
1291	2-Nitropropane	NIOSH 2528	
1292	Nitrotoluene	NIOSH 2005	
1293	Nonane		NIOSH 1500
1294	Nuisance Particulates, Total dust-	NIOSH 500, 600	
1295	Octachloronaphthalene	NIOSH S97	
1296	Octane	NIOSH 1500	
1303	Paraquat Respirable Dust-	NIOSH 5003	
1306	Pentane	NIOSH 1500	
1307	2-Pentanone (Methyl Propyl Ketone)	NIOSH 1300	
1308	Perchloroethylene (Tetrachloroethylene)	NIOSH 1003	
1310	Perlite	NIOSH 500, 600	

NIOSH ANALYTICAL METHODS FOR PEL UPDATE

H.S. No.	ANALYTE	VALIDATED METHOD	OTHER METHOD
1312	Petroleum Distilates (Naphtha)	NIOSH 1550	
1314	Phenyl Ether (Vapor)	NIOSH S72	
1315	Phenyl Glycidyl Ether (PGE)	NIOSH S74	
1317	Phenylhydrazine	NIOSH S160	
1320	Phosdrin (Mevinphos)	NIOSH 2503	
1322	Phosphoric Acid	NIOSH 7903	
1325	Phosphorus Trichloride		NIOSH 305
1329	Picric Acid (2,4,6-Trinitrophenol)	NIOSH S228	
1331	Plaster of Paris Total Dust-	NIOSH 500, 600	
1335	Propargyl Alcohol		NIOSH S65
1338	n-Propyl Acetate	NIOSH 1450	
1339	Propyl Alcohol	NIOSH 1401	
1340	n-Propyl Nitrate	NIOSH S227	
1341	Propylene Dichloride	NIOSH 1003	
1344	Propylene Oxide	NIOSH 1612	
----	Pyridine	NIOSH 1613	
1351	Rouge, Total Dust-	NIOSH 500, 600	
1352	Silica - Amorphous \ Diatomaceous earth \ (uncalcined)- > Percipitated silica- / Silica gel- /		NIOSH 7501
----	Silica - Crystalline		
1354	Cristobalite	NIOSH 7500	
1355	Quartz	NIOSH 7500	
1356	Tridymite-	NIOSH 7500	
1357	Tripoli-	NIOSH 7500	
1359	Silicon	NIOSH 500, 600	
1360	Silicon Carbide	NIOSH 500, 600	
1367	Sodium Hydroxide		NIOSH 7401
1369	Starch, Total Dust	NIOSH 500, 600	
1371	Stoddard Solvent	NIOSH 1550	
1374	Sucrose, Total Dust	NIOSH 500 600	
1375	Sulfur Dioxide		NIOSH 6004
1381	Talc (Non-asbestiform)	NIOSH 500, 600	
1384	Terphenyls	NIOSH 5021	
1385	1 1 2,2-Tetrachloroethane	NIOSH 1019	
1386	Tetraethyl Lead (as Pb)	NIOSH 2533	
1387	Tetrahydrofuran	NIOSH 1609	
1388	Tetramethyl Lead (as Pb)	NIOSH 2534	
1394	Tin, as Sn-		NIOSH 5504
1397	Toluene	NIOSH 1500 1501	

NIOSH ANALYTICAL METHODS FOR PEL UPDATE

H.S. No.	ANALYTE	VALIDATED METHOD	OTHER METHOD
1398	Toluene-2,4-Diisocyanate (TDI)	NIOSH 2535	
1399	o-Toluidine	NIOSH 2002	
1400	p-Toluidine		NIOSH 2002
1401	m-Toluidine		NIOSH 2002
1402	Tributyl Phosphate	NIOSH S208	
1403	1,1,2-Trichloro-1,2,2-Trifluoroethane	NIOSH 1020	
1404	Trichloroacetic Acid		NIOSH 1603
1405	1,2,4 Trichlorobenzene		NIOSH 5517
1406	Trichloroethylene	NIOSH 1022, 3701	
1407	1,2,3-Trichloropropane	NIOSH 1033	
1408	Triethylamine	NIOSH S152	
1414	Triorthocresyl Phosphate	NIOSH S209	
	Tungsten, as W		
1416	Insoluble compounds-	NIOSH 7074	
1417	Soluble compounds-	NIOSH 7074	
1421	Vanadium, as (V_2O_5)		
	Respirable dust-		NIOSH 7504
	fume-		NIOSH 7504
1424	Vinyl Acetate	NIOSH 278	
1425	Vinyl Bromide	NIOSH 1009	
1427	Vinyl Toluene	NIOSH 1501	
1428	Vinylidene Chloride	NIOSH 1015	
1429	VM&P Naphtha	NIOSH 1550	
1430	Welding fumes, Total particulate-	NIOSH 7200	
1431	Xylene (o-,m-,p-isomers)	NIOSH 1501	
1433	Xylidine	NIOSH 2002	
	Zinc Oxide		
1437	Fume-	NIOSH 7502	
1438	Dust-	NIOSH 7502	