

Table VI-12

Comparison of Gerin et al. SMRs and OSHA risk model predictions

Gerin et al. cohort*		OSHA risk model	
Cumulative exposure range ( $\mu\text{g}/\text{m}^3\text{-yrs}$ )	Ratio of observed to expected lung cancer deaths (SMR) (95% C.I.)	Cumulative exposure ( $\mu\text{g}/\text{m}^3\text{-yrs}$ )	Ratio of predicted to background lung cancer deaths (95% C.I.)
50 - 500	214 - 230 (44 - 589)	275	119 - 194 (111 - 260)
500 - 1500	252 - 258 (69 - 661)	1000	168 - 441 (140 - 677)
> 1500	130 - 133 (36 - 339)	2500	270 - 941 (201 - 1510)

\* restricted to workers with individual work histories, to reduce exposure misclassification

Table VI-12 shows that the range of risk ratios predicted by OSHA's model is higher than the ratios reported for the highest exposure group in the Gerin *et al.* cohort, consistent with EPRI's observations (Ex. 38-8, p. 25). However, the risk ratios predicted by OSHA's model are consistent with the Gerin SMRs for the 500-1500  $\mu\text{g}\text{-yrs}/\text{m}^3$  cumulative exposure range. For the 50-500  $\mu\text{g}\text{-yrs}/\text{m}^3$  cumulative exposure range, the OSHA prediction falls slightly below the lung cancer mortality ratio observed for the Gerin *et al.* cohort. The OSHA predictions for each group overlap with the 95% confidence intervals of the Gerin *et al.* SMRs, suggesting that sampling error may partly account for the discrepancies between the observed and predicted risk ratios in the lowest and highest exposure groups.

As previously discussed, OSHA believes that the lack of a clear exposure-response trend in the Gerin *et al.* study may be partly explained by exposure misclassification. As shown in Table VI-12, the highest exposure group has lower risk than might be expected based on OSHA's preferred risk models, while the lowest exposure group appears to have higher risk than OSHA's models would predict. This overall pattern of generally elevated but non-increasing SMRs across the three larger exposure groups in the Gerin study is consistent with potentially severe exposure misclassification. The higher-than-predicted risks among welders in the lowest exposure group could similarly reflect misclassification. However, it is not possible to determine with certainty that exposure misclassification is the cause of the differences between the risk predicted by OSHA's model and that observed in the Gerin cohort.

Finally, EPRI cites the generally similar relative risks found among stainless steel and mild steel welders as further evidence that exposure to Cr(VI) may not carry the same risk of lung cancer in welding operations as it does

in the chromate production industry. EPRI states:

[I]t is reasonable to expect that if Cr(VI) were a relevant risk factor for welders in the development of lung cancer, and certain types of welding involve Cr(VI) more than other types, then subgroups of welders who are more exposed to Cr(VI) by virtue of the type of welding they do should have higher rates of lung cancer than welders not exposed to Cr(VI) in their welding occupation;

in particular, " \* \* \* stainless steel welders should have a higher risk of lung cancer than welders of mild steel" (Ex. 38-8, p. 13). OSHA believes that EPRI's point would be correct if the subgroups in question are similar in terms of other important risk factors for lung cancer, such as smoking, co-exposures, and overall population health. However, no analysis comparing stainless steel welders with mild steel welders has properly controlled for these factors, and in fact there have been indications that mild steel welders may be at greater risk of lung cancer than stainless steel welders from non-occupational causes. As discussed by EPRI, "[r]esults from cohort studies of stainless steel welders with SMRs much less than 100 support an argument that the healthy worker effect might be more marked among stainless steel workers compared to mild steel welders"; also " \* \* \* stainless steel welders are generally more qualified and paid more than other welders" (Ex. 38-8, p. 16), a socioeconomic factor that suggests possible differences in lung cancer risk due to smoking, community exposures, or occupational exposures from employment other than welding.

Comments submitted by Exponent (Ex. 38-233-4) and EPRI (Ex. 38-8) compare the Cr(VI) compounds found in welding fumes and those found in the chromate production environments of the Gibb and Luippold cohorts. Exponent stated that "[t]he forms of Cr(VI) to which chromate production workers were historically exposed are primarily the soluble potassium and sodium chromates" found in stainless

steel welding fumes. Less soluble forms of Cr(VI) are also found in stainless steel welding fumes in limited amounts, as discussed in the 1990 IARC monograph on welding (Ex. 35-242, p. 460), and are believed to have been present in limited amounts at the plants where the Gibb and Luippold workers were employed (Ex. 38-233-4, p. 4). Exponent concludes that, while it is difficult to compare the exposures of welders to chromate production workers, " \* \* \* there is no obvious difference \* \* \* in solubility \* \* \* " that would lead to a significantly lesser risk from Cr(VI) exposure in welding as compared to the Gibb and Luippold cohort exposures (Ex. 38-233-4, p. 3, p. 11). OSHA believes that the similarity in the solubility of Cr(VI) exposures to welders and chromate production workers supports the Agency's use of its risk model to describe Cr(VI)-related risks to welders.

Exponent and others (Exs. 38-8; 39-25) commented on the possibility that the bioavailability of Cr(VI) may nevertheless differ between welders and chromate production workers, stating that " \* \* \* bioavailability of Cr(VI)-containing particles from welding fumes may not be specifically related to solubility of the Cr(VI) chemical species in the fume" (Ex. 38-233-4, p. 11). In this case, Exponent argues,

delivered doses of Cr(VI) to the lung could be quite dissimilar among welders as compared to chromate production industry workers exposed to the same Cr(VI) chemical species at the same Cr(VI) airborne concentrations (Ex. 38-233-4, p. 11).

However, Exponent provided no data or plausible rationale that would support a Cr(VI) bioavailability difference between chromate production and welding. The low proportion of respirable Cr(VI) particles that apparently limits bioavailability of inhaled Cr(VI) during aircraft spray priming operations described previously is not an issue with welding. High temperature welding generates fumes of small