members having a median annual average exposure value below 10 μg/m³ Cr(VI), 69% below 20 μg/m³, and 91% below the previous PEL (Ex. 35–295). In addition, Dr. Gibb indicated that exposures in general were lower than suggested by some commenters (Tr. 1856, Ex. 38–215–2, p. 17). For example, about half of the total time that workers were exposed was estimated to be below 14 μg/m³ Cr(VI) from 1960–1985 (Ex. 47–8, p. 1).

Exponent calculated SMRs for six groups of workers in the Gibb cohort, classified according to the level of their highest average annual exposure estimates. They found that only the group of workers whose highest exposure estimates were above approximately 95 μg/m³ Cr(VI) had statistically significantly elevated lung cancer risk when Baltimore reference rates were used (Ex. 31–18–15–1, p. 33). Exponent's results are presented in Table VI–8 below, adapted from Table 10 in their report (Ex. 31–18–15–1, p. 33).

Table VI-8

Exponent SMR Analysis of Peak Exposures in Gibb Cohort

Group	Peak Exposure (μg Cr(VI)/m³)	Observed Cancer Deaths	Person-years of Observation	SMR (95% CI) Maryland	SMR (95% CI) Baltimore
1	0.000 - 3.7	50	36,733	1.18 (0.87 - 1.55)	0.91 (0.67 - 1.20)
2	3.7 - 10.0	21	10,401	1.97 (1.22 - 3.01)	1.51 (0.94 - 2.31)
3	10.0 - 25.0	19	9,800	2.07 (1.24 - 3.23)	1.56 (0.94 - 2.43)
4	25.0 - 54.9	12	6,707	2.06 (1.07 - 3.60)	1.54 (0.80 - 2.69)
5	54.9 - 94.6	7	3,462	2.20 (0.88 - 4.53)	1.66 (0.67 - 3.43)
6	94.6 - 419.3	13	3,664	3.00 (1.60 - 5.13)	2.35 (1.25 - 4.02)

OSHA does not believe that Exponent's analysis of the Gibb data provides convincing evidence of a threshold in exposure-response. While the lower-exposure groups do not have statistically significantly elevated lung cancer risk ($p > 0.05$) when compared with a Baltimore reference population, the SMRs for all groups above 3.7 μg/m³ are consistently elevated. Moreover, the increased risk approaches statistical significance, especially for those subgroups with higher power (Groups 2 and 3). This can be seen by the lower 95% confidence bound on the SMR for these groups, which is only slightly below 1. The analysis suggests a lack of power to detect excess risk in Groups 2–5, rather than a lack of excess risk at these exposure levels.

Analyses of the Luippold cohort by Crump *et al.* (Ex. 35–58) and Proctor *et al.* (Ex. 38–216–10) used exposure estimates they called ''highest average monthly exposure'' to explore the effects of exposure intensity on lung cancer risk. They reported that lung cancer risk was elevated only for individuals with exposure estimates higher than the previous PEL of 52 μg/m³ Cr(VI). Crump *et al.* additionally found ''statistically significant evidence of a dose-related increase in the relative risk of lung cancer mortality'' only for groups above four times the previous PEL, using a series of Poisson regressions modeling the increase in risk across the first two subgroups and with the successive addition of higher-exposed subgroups (Ex. 35–58, p. 1154).

As with the Gibb data, OSHA does not believe that the subgroup of workers exposed at low levels is large enough to provide convincing evidence of a threshold in exposure-response. In the Crump *et al.* and Proctor *et al.* analyses, the groups for which no statistically significant elevation or dose-related trends in lung cancer risk were observed are quite small by the standards of cancer epidemiology (*e.g.*, the Luippold cohort had only about 100 workers below the previous PEL and about 40 workers within 1–3 times the previous PEL). Crump *et al.* emphasized that ''* * * this study had limited power to detect increases [in lung cancer risk] at these low exposure levels'' (Ex. 35–58, p. 1147). The authors did not conclude that their results indicate a threshold. They stated that their cancer potency estimates based on a linear relative risk model using the cumulative exposure metric ''* * * are comparable to those developed by U.S. regulatory agencies and should be useful for assessing the potential cancer hazard associated with inhaled Cr(VI)'' (Ex. 35–58, p. 1147).

OSHA discussed the Exponent, Crump *et al.* and Luippold *et al.* SMR analyses of the Gibb and Luippold cohorts in the preamble to the proposed rule, stating that the lack of a statistically significant result for a subset of the entire cohort should not be construed to imply a threshold (69 FR at 59382). During the hearing, Robert Park of NIOSH expressed agreement with OSHA's preliminary interpretation, adding that:

> [W]e think that any interpretation of threshold in these studies is basically a statistical artifact * * * It is important I think to understand that any true linear or even just monotonic exposure response that doesn't have a threshold will exhibit a threshold by the methods that they used. If you stratify the exposure metric fine enough and look at the lower levels, they will be statistically insignificant in any finite study * * * telling you nothing about whether or not in fact there is a threshold (Tr. 351).

To further explore the effects of highly exposed individuals on OSHA's risk model, The Chrome Coalition suggested that OSHA should base its exposure-response model on a subcohort of workers excluding those who were exposed to ''* * * an extraordinary exposure level for some extended period of time* * *'', *e.g.*, estimated exposures greater than the previous PEL for more than one year (Ex. 38–231, p. 21). The Chrome Coalition stated,

> We are not aware of any study that has performed this type of analysis but we believe that it should be a way of better estimating the risk for exposures in the range that OSHA is considering for the PEL (Ex. 38–231, p. 21).

To gauge the potential utility of such an analysis, OSHA examined the subset of the Gibb cohort that was exposed for more than 365 days and had average annual exposure estimates above the previous PEL of 52 μg/m³ Cr(VI). The Agency found that the subcohort includes only 82 such individuals, of whom 37 were reported as deceased at the end of follow-up and five had died of lung cancer. In a cohort of 2357