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Occupational Exposure to Hexavalent Chromium; Proposed Rule

DEPARTMENT OF LABOR

Occupational Safety and Health Administration

29 CFR Parts 1910, 1915, 1917, 1918, and 1926

[Docket No. H054A]

RIN 1218–AB45

Occupational Exposure to Hexavalent Chromium

AGENCY: Occupational Safety and Health Administration (OSHA), Department of Labor.

ACTION: Proposed rule; request for comments and scheduling of informal public hearings.

SUMMARY: The Occupational Safety and Health Administration (OSHA) proposes to amend its existing standard for employee exposure to hexavalent chromium (Cr(VI)). The basis for issuance of this proposal is a preliminary determination by the Assistant Secretary that employees exposed to Cr(VI) face a significant risk to their health at the current permissible exposure limit and that promulgating this proposed standard will substantially reduce that risk. The information gathered so far in this rulemaking indicates that employees exposed to Cr(VI) well below the current permissible exposure limit are at increased risk of developing lung cancer. Occupational exposures to Cr(VI) may also result in asthma, and damage to the nasal epithelia and skin.

This document proposes an 8-hour time-weighted average permissible exposure limit of one microgram of Cr(VI) per cubic meter of air (1 mg/m^3) for all Cr(VI) compounds. OSHA also proposes other ancillary provisions for employee protection such as preferred methods for controlling exposure, respiratory protection, protective work clothing and equipment, hygiene areas and practices, medical surveillance, hazard communication, and recordkeeping. OSHA is proposing separate regulatory texts for general industry, construction, and shipyards in order to tailor requirements to the circumstances found in each of these sectors.

DATES: *Written comments.* The Agency invites interested persons to submit written comments regarding the proposed rule, including comments on the information collection determination described in Section X of the preamble (OMB Review under the Paperwork Reduction Act of 1995), by mail, facsimile, or electronically. All comments, whether submitted by mail, facsimile, or electronically through the Internet, must be sent by January 3, 2005.

Informal public hearings. The Agency plans to hold an informal public hearing in Washington, DC, beginning on February 1, 2005. OSHA expects the hearing to last from 9:30 a.m. to 5:30 p.m.; however, the exact daily schedule is at the discretion of the presiding administrative law judge.

Notice of intention to appear to provide testimony at the informal public hearing. Interested persons who intend to present testimony at the informal public hearing in Washington, DC, must notify OSHA of their intention to do so no later than December 3, 2004.

Hearing testimony and documentary evidence. Interested persons who request more than 10 minutes to present their testimony, or who will be submitting documentary evidence at the hearing, must provide the Agency with copies of their full testimony and all documentary evidence they plan to present by January 3, 2005. See Section XVI below for details on the format and how to file a notice of intention to appear, submit documentary evidence at the hearing, and request an appropriate amount of time to present testimony.

ADDRESSES: *Written comments.* Interested persons may submit three copies of written comments to the Docket Office, Docket H054A, Room N–2625, OSHA, U.S. Department of Labor, 200 Constitution Avenue, NW., Washington, DC 20210; telephone (202) 693–2350. If written comments are 10 pages or fewer, they may be faxed to the OSHA Docket Office, facsimile number (202) 693–1648. Comments may also be submitted electronically through the Internet at *http://ecomments.osha.gov.* Supplemental information such as studies and journal articles cannot be attached to electronic submissions. Instead, three copies of each study, article, or other supplemental document must be sent to the OSHA Docket Office at the address above. These materials must clearly identify the associated electronic comments to which they will be attached in the docket by the following information: Name of person submitting comments; date of comment submission; subject of comments; and docket number to which comments belong.

Informal public hearings. The informal public hearing to be held in Washington, DC, will be held in the Frances Perkins Building, U.S. Department of Labor, 200 Constitution Avenue, NW., Washington, DC 20210.

Notice of intention to appear to provide testimony at the informal public hearing. Interested persons who intend to present testimony at the informal public hearing in Washington, DC, may submit three copies of their notice of intention to appear to the Docket Office, Docket H054A, Room N–2625, OSHA, U.S. Department of Labor, 200 Constitution Avenue, NW., Washington, DC 20210. Notices may also be submitted electronically through the Internet at *http://ecomments.osha.gov.* OSHA Docket Office and Department of Labor hours of operation are 8:15 a.m. to 4:45 p.m.

Hearing testimony and documentary evidence. Interested persons who request more than 10 minutes in which to present their testimony, or who will be submitting documentary evidence at the informal public hearing must submit three copies of the testimony and the documentary evidence to the Docket Office, Docket H054A, Room N–2625, OSHA, U.S. Department of Labor, 200 Constitution Avenue, NW., Washington, DC 20210. Written testimony may also be submitted electronically through the Internet at *http://ecomments.osha.gov.*

Please note that security-related problems may result in significant delays in receiving comments and other materials by regular mail. Telephone the OSHA Docket Office at (202) 693–2350 for information regarding security procedures concerning delivery of materials by express delivery, hand delivery, and messenger service.

All comments and submissions will be available for inspection and copying in the OSHA Docket Office at the address above. Most comments and submissions will be posted on OSHA's Web page (*http://www.osha.gov*). Contact the OSHA Docket Office at (202) 693–2350 for information about materials not available on the OSHA Web page and for assistance in using this Web page to locate docket submissions. Because comments sent to the docket or to OSHA's Web page are available for public inspection, the Agency cautions interested parties against including in these comments personal information such as social security numbers and birth dates.

FOR FURTHER INFORMATION CONTACT: For general information and press inquiries, contact Mr. George Shaw, Office of Communications, Room N–3647, OSHA, U.S. Department of Labor, 200 Constitution Avenue, NW., Washington, DC 20210; telephone (202) 693–1999. For technical inquiries, contact Ms. Amanda Edens, Directorate of Standards and Guidance, Room N–3718, OSHA, U.S. Department of Labor, 200 Constitution Avenue, NW., Washington, DC 20210; telephone (202) 693–2093 or

fax (202) 693–1678. For hearing information contact Ms. Veneta Chatmon, Office of Communications, Room N–3647, OSHA, U.S. Department of Labor, 200 Constitution Avenue, NW., Washington, DC 20210; telephone (202) 693–1999.

SUPPLEMENTARY INFORMATION: For additional copies of this **Federal Register** document, contact the Office of Publications, Room N–3101, OSHA, U.S. Department of Labor, 200 Constitution Avenue, NW., Washington, DC 20210; telephone (202) 693–1888. Electronic copies of this **Federal Register**, as well as news releases and other relevant documents, are available at OSHA's Home page at *http://www.osha.gov.*

I. General

The preamble to the proposed standard on occupational exposure to chromium (VI) discusses events leading to the proposal, health effects of exposure, the degree and significance of the risk presented, a summary of the analysis of technological and economic feasibility, regulatory impact, and regulatory flexibility, and the rationale behind the specific provisions set forth in the proposed standard. The discussion follows this outline:

I. General
II. Issues
III. Pertinent Legal Authority
IV. Events Leading to the Proposed Standards
V. Chemical Properties and Industrial Uses
VI. Health Effects
VII. Preliminary Quantitative Risk Assessment
VIII. Significance of Risk
IX. Summary of the Preliminary Economic Analysis and Initial Regulatory Flexibility Analysis
X. OMB Review under the Paperwork Reduction Act of 1995
XI. Federalism
XII. State Plans
XIII. Unfunded Mandates
XIV. Protecting Children from Environmental Health and Safety Risks
XV. Environmental Impacts
XVI. Public Participation—Notice of Hearing
XVII. Summary and Explanation of the Standards
XVIII. Authority and Signature
XIX. Proposed Standards

II. Issues

OSHA requests comment on all relevant issues, including health effects, risk assessment, significance of risk determination, technological and economic feasibility, and the provisions of the proposed regulatory text. OSHA is especially interested in responses, supported by evidence and reasons, to the following questions:

Health Effects

1. OSHA has described a variety of studies addressing the major adverse health effects that have been associated with exposure to Cr(VI). Has OSHA adequately identified and documented all critical health impairments associated with occupational exposure to Cr(VI)? Are there any additional studies or other data that would controvert the information discussed or significantly enhance the determination of material health impairment or the assessment of exposure-response relationships? Submit any relevant information, and explain your reasoning for recommending the inclusion of any studies you suggest.

2. Using currently available epidemiologic and experimental studies, OSHA has made a preliminary determination that all Cr(VI) compounds (*e.g.*, water soluble, insoluble and slightly soluble) possess carcinogenic potential and thus present a lung cancer risk to exposed workers. Is this determination correct? Are there additional data OSHA should consider in evaluating the carcinogenicity or relative carcinogenic potencies of different Cr(VI) compounds?

Risk Assessment

3. In its preliminary assessment of risk, OSHA has relied primarily on two epidemiologic cohort studies of chromate production workers to estimate the lung cancer risk to workers exposed to Cr(VI) (Exs. 31–22–11; 33–10). Are there any other studies that you believe are better suited to estimating the risk to exposed workers; if so, please provide the studies and explain why you believe they are better.

4. OSHA is aware of two cohorts (i.e., Alexander cohort, Ex. 31–16–3, and Pastides cohort, Ex. 35–279) in which a sizable number of workers were probably exposed to low Cr(VI) air levels (*e.g.*, <10 $\mu g/m^3$) more consistent with concentrations found in the workplace today. However, OSHA believes the period of follow-up observation (median <10 yr), the young age (<45 yr at end of follow-up) and the low number of observed lung cancers (≤15 lung cancers) severely limits these cohorts as primary data sets for quantitative risk analysis. Other limitations to the Alexander study include a lack of data on workers who were employed between 1940 and 1974, but whose employment ended prior to 1974, and on exposures prior to 1974. Are there updated analyses available for the Alexander and Pastides cohorts? How many years do these cohorts need to be followed and how many lung cancers need to be observed in order for these data sets to provide insight into the shape of the exposure-response curve at lower levels of Cr(VI) exposure (*e.g.*, 0.5 to 5 $\mu g/m^3$)? In the case of the Alexander cohort, is there additional information on cohort members' exposures prior to 1974 or workers who left prior to 1974 that could improve the analysis? Are there other cohorts available to look at low exposures?

5. OSHA has relied upon a linear relative risk model and cumulative Cr(VI) exposure for estimating the lifetime occupational lung cancer risk among Cr(VI)-exposed workers. In particular, OSHA has made a preliminary determination that a threshold model is not appropriate for estimating the lung cancer risk associated with Cr(VI). However, there is some evidence that pathways (*e.g.*, extracellular reduction, DNA repair, cell apoptosis, etc.) may exist within the lung that protect against Cr(VI)-induced respiratory carcinogenesis, and may potentially introduce non-linearities into the Cr(VI) exposure-cancer response. Is there convincing scientific evidence of a non-linear exposure-response relationship in the range of occupational exposures of interest to OSHA? If so, are there sufficient data to define a non-linear approach that would provide more reliable predictions of risk than the linear relative risk model used by OSHA?

6. OSHA's estimates of lung cancer risk are based on workers primarily exposed to highly water-soluble sodium chromate and sodium dichromate. OSHA has preliminarily concluded that the risk for workers exposed to equivalent levels of other Cr(VI) compounds will be of a similar magnitude or, in the case of some Cr(VI) compounds, possibly greater than the risks projected in the OSHA quantitative risk assessment. Is this determination appropriate? Are there sufficient data to reliably quantify the risk from occupational exposure to specific Cr(VI) compounds? If so, explain how the risk could be estimated.

7. The preliminary quantitative risk assessment relies on two (Gibb and Luippold) cohort studies in which most workers were exposed higher Cr(VI) levels than the PEL proposed by OSHA, for shorter durations than a working lifetime exposure. The risks estimated by OSHA for lifetime exposure to the proposed PEL, therefore, carry the assumption that a cumulative exposure achieved by short duration exposure to higher Cr(VI) air levels (*e.g.*, exposed 3 years to 15 $\mu g/m^3$) leads to the same risk as an equivalent cumulative exposure achieved by longer duration exposure to

lower Cr(VI) exposure (e.g, exposed 45 years to 1 $\mu g/m^3$). OSHA preliminarily finds this assumed exposure equivalency to represent an uncertainty in the estimates of risk but does not have information that indicates this uncertainty introduces serious error in its predictions of risk. Does the OSHA exposure-response assessment based on the higher Cr(VI) air levels and/or shorter durations experienced by the Gibb and Luippold cohorts lead to a serious underprediction or overprediction in estimated risks for the occupational exposure scenarios of interest to OSHA? Please provide any data to support your rationale.

8. OSHA has made a preliminary determination that suitable data are not available for making quantitative risk estimates for the non-cancer adverse health effects associated with exposure to Cr(VI) (*e.g.*, nasal septum ulcerations and perforations, asthma, irritant and allergic contact dermatitis). Are there suitable data for a quantitative estimation of risk for non-cancer adverse effects that OSHA should include in its final quantitative risk assessment? If so, what models or approaches should be used?

9. Are there other factors OSHA should take into consideration in its final quantitative risk assessment to better characterize the risks associated with exposure to Cr(VI)?

Technologic and Economic Feasibility

10. In its Preliminary Economic Analysis of the proposed standard, OSHA presents a profile of the affected worker population. In that profile are estimates of the number of affected workers by application group and job category and the distribution of exposures by job category. Are there additional data that will enable the Agency to refine its profile of the worker population exposed to Cr(VI)? If so, how should OSHA use these data in making such revisions?

11. What are the job categories in which employees are potentially exposed to Cr(VI) in your company or industry? For each job category, provide a brief description of the operation and describe the job activities that may lead to Cr(VI) exposure. How many employees are exposed, or have the potential for exposure, to Cr(VI) in each job category in your company or industry? What are the frequency, duration and levels of exposures to Cr(VI) at each job category in your company or industry? Where commenters are able to provide exposure data, OSHA requests that, where possible, exposure data be personal samples with clear descriptions of the length of the sample and analytical method. Exposure data that provide information concerning the controls in place are more valuable than exposure data without such information.

12. Have there been technological changes within your industry that have influenced the magnitude, frequency, or duration of exposure to Cr(VI) or the means by which employers attempt to control exposures? Describe in detail these technological changes and their effects on Cr(VI) exposures and methods of control.

13. Has there been a trend within your industry to eliminate Cr(VI) from production processes, products and services? If so, comments are requested on the success of substitution efforts. Commenters should estimate the percentage reduction in Cr(VI), and the extent to which Cr(VI) is still necessary in their processes within product lines or production activities. OSHA also requests that commenters describe any technical, economic or other deterrents to substitution.

14. Does any job category or employee in your workplace have exposures to Cr(VI) that raw air monitoring data do not adequately portray due to the short duration, intermittent or non-routine nature, or other unique characteristics of the exposure? Please explain your response and indicate peak levels, duration and frequency of exposures for employees in these job categories.

15. OSHA requests the following information regarding engineering and work practice controls in your workplace or industry:

a. Describe the operations in which the proposed PEL is being achieved most of the time by means of engineering and work practice controls.

b. What engineering and work practice controls have been implemented in these operations?

c. For all operations in facilities where Cr(VI) is used, what engineering and work practice controls have been implemented? If you have installed engineering controls or adopted work practices to reduce exposure to Cr(VI), describe the exposure reduction achieved and the cost of these controls. Where current work practices include the use of regulated areas and hygiene facilities, provide data on the implementation of these controls, including data on the costs of installation, operation, and maintenance associated with these controls.

d. Describe additional engineering and work practice controls which could be implemented in each operation where exposure levels are currently above the proposed PEL to further reduce exposure levels.

e. When these additional controls are implemented, to what levels can exposure be expected to be reduced, or what per cent reduction is expected to be achieved?

f. What are the costs and amount of time needed to develop, install and implement these additional controls? Will the added controls affect productivity?

g. Are there any processes or operations for which it is not reasonably possible to implement engineering and work practice controls within two years to achieve the proposed PEL? If so, would allowing additional time for employers to implement engineering and work practice controls make compliance possible? How much additional time would be necessary?

16. OSHA requests information on whether there are any limited or unique conditions or job tasks in Cr(VI) manufacture or use where engineering and work practice controls are not available or are not capable of reducing exposure levels to or below the proposed PEL most of the time. Provide data and evidence to support your response.

17. In its Preliminary Economic Analysis, OSHA presents estimated baseline levels of use of personal protective equipment (PPE) and the incremental costs associated with the proposed standard. Are OSHA's estimated compliance rates reasonable? Are OSHA's estimates of PPE costs, and the assumptions underlying these estimates, consistent with current industry practice? Comments are solicited on OSHA's analysis of PPE costs.

18. In its Preliminary Economic Analysis, OSHA presents estimated baseline levels of communication of Cr(VI)-related hazards and the incremental costs associated with the additional requirements for communication in the proposed standard. OSHA requests information on hazard communication programs addressing Cr(VI) that are currently being implemented by employers and any necessary additions to those programs that are anticipated in response to the proposed standard. Are OSHA's baseline estimates and unit costs for training reasonable and consistent with current industry practice?

Effects on Small Entities

19. Will difficulties be encountered by small entities when attempting to comply with requirements of the proposed standard? Can any of the

proposal's requirements be deleted or simplified for small entities, while still protecting the health of employees? Would a longer time allowed for compliance for small entities make a difference to their ability to comply, and if so, why? (Information submitted in the SBREFA process is part of the record and need not be resubmitted).

Economic Impacts and Economic Feasibility

20. OSHA, in its Preliminary Economic Analysis, has estimated, by application group, compliance costs per affected entity and the likely impacts on revenues and profits under alternative market scenarios. OSHA requests that affected employers provide comment on OSHA's estimate of revenue, profit, and the impacts of costs for their industry or application group. Are there special circumstances—such as unique cost factors, foreign competition, or pricing constraints—that OSHA needs to consider when evaluating economic impacts for particular application groups? Comments are requested on OSHA's analysis of economic feasibility in the PEA.

Overlapping and Duplicative Regulations

21. Do any federal regulations duplicate, overlap, or conflict with the proposed Cr(VI) standard?

22. In some facilities, adjustments in ventilation systems to comply with the proposed PEL may require additional time and expense to retest these systems to ensure compliance with EPA requirements or state requirements. OSHA requests information and comment indicating how frequently retesting would be required, and the time and costs involved in such retesting.

Environmental Impacts

23. Submit any data, information, or comments pertaining to possible environmental impacts of adopting this proposal, such as the following:

a. Any positive or negative environmental effects that could result;

b. Any irreversible commitments of natural resources which could be involved; and

c. Estimates of the effect of the proposed standard on the levels of Cr(VI) in the environment.

In particular, consideration should be given to the potential direct or indirect impacts of the proposal on water and air pollution, energy use, solid waste disposal, or land use.

d. Some small entity representatives noted that OSHA PELs are sometimes used to set ''fence line'' standards for air pollutants. OSHA is unable to find evidence of states formally using this procedure, though some states may use such a procedure informally. Do any states or other air pollution authorities base standards on OSHA PELs? What effects might this have on the environment and on environmental compliance?

Provisions of the Standard

24. OSHA's safety and health advisory committees for Construction and Maritime advised the Agency to take into consideration the unique nature of their work environments by either settings separate standards or making accommodations for the differences in work environments in construction and maritime. To account for differences in the workplace environment for these different sectors OSHA has proposed separate standards for general industry, construction, and shipyards. Is this approach appropriate? What other approaches should the Agency consider? Please provide a rationale for your response.

25. OSHA has not proposed to cover agriculture, because the Agency is not aware of significant exposures to Cr(VI) in agriculture. Is this determination correct?

26. OSHA has proposed to regulate exposures to all Cr(VI) compounds. As discussed in the health effects section of this preamble, the Agency has made a preliminary determination that the existing data support coverage of all Cr(VI) compounds in the scope of the proposed standard. Is this an appropriate determination or are there additional data that support the exclusion of certain compounds from the scope of the final standard? If so, describe specifically how these data would support a decision to exclude certain compounds from the scope of the final rule.

27. OSHA has made a preliminary determination to exclude Cr(VI) exposures due to work with portland cement from the scope of the construction standard. OSHA believes that guidance efforts by the Agency may be more suitable for addressing the dermal hazards associated with portland cement use in construction settings. OSHA's Advisory Committee for Construction Safety and Health (ACCSH) advised OSHA to include construction cement work under the proposed standard because of the known hazards associated with wet cement and the large number of workers exposed to wet cement in construction work settings. In particular ACCSH advised OSHA that only certain provisions might be necessary for workers exposed to wet cement (*e.g.*, protective work clothing, hygiene areas and practices, medical surveillance for signs and symptoms of adverse health effects only, communication of hazards and recordkeeping for medical surveillance and training). Other provisions, ACCSH advised, might not be necessary (*e.g.*, permissible exposure levels, exposure assessment, methods of compliance and respiratory protection). Should OSHA expand the scope of the construction proposal to include Cr(VI) exposures from portland cement? If so, what would be the best approach for addressing the dermal hazards from Cr(VI) faced by these workers? If Cr(VI) exposure from portland cement work in construction is included in the final standard, should only certain provisions such as those outlined by ACCSH be considered?

28. OSHA has proposed to include exposure to Cr(VI) from portland cement in the scope of the standard for general industry. The Agency believes that the potential for airborne exposure to Cr(VI) in general industry due to work with portland cement, as indicated by the profile of exposed workers presented in Table IX–2 of this preamble, is higher than in the construction industry. OSHA acknowledges, however, that the exposure profile indicates that no workers are exposed to Cr(VI) at levels over the proposed action level. Given the low level of airborne exposure among cement workers in general industry, should OSHA exclude exposures to Cr(VI) from portland cement from the scope of the general industry standard? OSHA seeks data to help inform this issue, and solicits comments on particular provisions of the general industry and construction standards that may or may not be appropriate for cement workers.

29. OSHA has proposed to exempt from coverage Cr(VI) exposures occurring in the application of pesticides in general industry (such as the treatment of wood with chromium copper arsenate (CCA)) because pesticide application is regulated by EPA, and section 4(b)(1) of the OSH Act precludes OSHA from regulating where other Federal agencies exercise their statutory authority to do so. OSHA has proposed to cover exposures resulting from use of treated materials. Is this approach appropriate? Are there any instances where EPA-regulated pesticide application occurs in construction or shipyard workplaces?

30. Describe any additional industries, processes, or applications that should be exempted from the Cr(VI) standard and provide detailed reasons for any requested exemption. In

particular, are the epidemiologic and experimental studies sufficient to support OSHA's the inclusion of various industries or processes under the scope of the proposed standard? Please provide the rationale and supporting data for your response.

31. Can the proposed Cr(VI) standard for the construction industry be modified in any way to better account for the workplace conditions in that industry, while still providing appropriate protection to Cr(VI)-exposed workers in that industry? Would an alternative approach similar to that used in OSHA's asbestos standard, where the application of specified controls in certain situations would be considered adequate to meet the requirements of the standard, be useful? Is there enough information available to define such technology specifications?

32. Can the proposed Cr(VI) standard for shipyards be modified in any way to better account for the workplace conditions in that industry, while still providing appropriate protection to Cr(VI)-exposed workers in that industry?

33. OSHA has proposed a TWA PEL for Cr(VI) of 1.0 $\mu g/m^3$. The Agency has made a preliminary determination that this is the lowest level that is both technologically and economically feasible and is necessary to reduce significant risks of material health impairment from exposure to Cr(VI). Is this PEL appropriate and is it adequately supported by the existing data? If not, what PEL would be more appropriate or would more adequately protect employees from Cr(VI)-associated health risks? Provide evidence to support your response.

34. Should different PELs be established for different Cr(VI) compounds? If so, how should they be established? Where possible, provide specific detail about how different PELs could be established and how the Agency should apply those PELs in instances where workers may be exposed to more than one Cr(VI) compound.

35. OSHA has proposed an action level for Cr(VI) exposure in general industry, but not in construction or shipyards. Is this an appropriate approach? Should OSHA set an action level for exposure to Cr(VI) in construction and shipyards? Should the proposed action level in general industry be retained in the final rule?

36. If an action level is included in the final rule, is the proposed action level for general industry (0.5 $\mu g/m^3$) the appropriate level for the PEL under consideration? If not, at what level should the action level be set?

37. If an action level is included in the final rule, which provisions should be triggered by exposure above the action level? Indicate the basis for your position and include any supporting information.

38. If no action level is included in the final rule, which provisions should apply to all Cr(VI)-exposed workers? Which provisions should be triggered by the PEL? Are there any other appropriate triggers for the requirements of the standard?

39. Should OSHA set a short-term exposure limit (STEL) or ceiling for exposure to Cr(VI)? If so, please specify the appropriate air concentration and the rationale for its selection.

40. Do you conduct initial air monitoring or do you rely on objective data to determine Cr(VI) exposures? Describe any other approaches you have implemented for assessing an employee's initial exposure to Cr(VI).

41. Describe any follow-up or subsequent exposure assessments that you conduct. How often do you conduct such follow-up or subsequent exposure assessments? Please comment on OSHA's estimate of baseline industry practice and the projected costs for initial and periodic exposure assessment. Are OSHA's estimates consistent with current industry practice?

42. Do shipyard employers presently measure their employees' exposure to Cr(VI)? If not, do they use some alternative method of identifying which employees may be over-exposed to Cr(VI)?

43. OSHA has proposed specific requirements for exposure assessment in general industry, but has not proposed that these requirements apply to construction or shipyard employers. Should requirements for exposure assessment in construction or shipyards be included in the final Cr(VI) standard? Are there any advantages to requiring construction or shipyard employers to measure their employees' exposures to Cr(VI)? If so, would the exposure assessment requirements proposed for general industry be appropriate? Would construction or shipyard employers encounter situations where monitoring would be infeasible if they were required to follow the exposure assessment requirements proposed for employers in general industry? Indicate the basis for your position and include any supporting information. What types of exposure assessment strategies are effective for assessing worker exposures at construction and shipyard worksites?

44. Should requirements for exposure assessment in general industry be included in the final Cr(VI) standard, or would the performance-oriented requirement proposed for construction and shipyards be more appropriate? Indicate the basis for your position and include any supporting information.

45. OSHA has proposed that exposure monitoring in general industry be conducted at least every six months if exposures are above the action level but below the PEL, and at least every three months if exposures are at or above the PEL. Are these proposed frequencies appropriate? If not, what frequency of monitoring would be more appropriate, and why?

46. OSHA has proposed that regulated areas be established in general industry wherever an employee's exposure to airborne concentrations of Cr(VI) is, or can reasonably be expected to be, in excess of the PEL. OSHA seeks comments on this provision and in particular:

a. Describe any work settings where establishing regulated areas could be problematic or infeasible. If establishing regulated areas is problematic, what approaches might be used to warn employees in such work settings of high risk areas (i.e., areas where the airborne concentrations of Cr(VI) exceed the PEL?).

b. Should OSHA add hazards from eye or skin contact as a trigger for establishing regulated areas? Explain the basis for your position, and include any supporting information. c. Describe any methods currently used that have been found to be effective in establishing regulated areas.

47. OSHA has not proposed requirements for establishment of regulated areas in construction or shipyards. Should requirements for regulated areas for construction or shipyards be included in the final Cr(VI) standard? If so, would the requirements for regulated areas proposed for general industry be appropriate? Are there any particular problems in construction or shipyard settings that make regulated areas problematic or infeasible? If requirements for regulated areas for construction or shipyards are not included in the final Cr(VI) standard, should OSHA include requirements for warning signs or other measures to alert employees of the presence of Cr(VI)? If so, what practical means could be used to determine where and when such labeling would be required? What potential difficulties might be encountered by using such an approach? Indicate the basis for your position and include any supporting information.

48. Under the proposed standard, employers are required to use engineering and work practice controls

to reduce and maintain employee exposure to Cr(VI) to or below the PEL unless the employer can demonstrate that employees are not exposed above the PEL for 30 or more days per year, or the employer can demonstrate that such controls are not feasible. Is this approach appropriate for Cr(VI)? Indicate the basis for your position and include any supporting information.

49. In OSHA's Cadmium standard (29 CFR 1010.1027), the Agency established separate engineering control air limits (SECALs) for certain processes in selected industries. SECALs were established where compliance with the PEL by means of engineering and work practice controls was infeasible. For these industries, a SECAL was established at the lowest feasible level that could be achieved by engineering and work practice controls. The PEL was set at a lower level, and could be achieved by any allowable combination of controls. SECALs thus allowed OSHA to establish a lower PEL for cadmium than would otherwise have been possible, given technological feasibility constraints. Should OSHA establish SECALs for Cr(VI) in any industries or processes? If so, in what industries or processes, and at what levels? Provide rationale to support your position.

50. The proposed standard prohibits the use of job rotation for the sole purpose of lowering employee exposures to Cr(VI). Are there any circumstances where this practice should be allowed in order to meet the proposed PEL?

51. OSHA is proposing that employers provide appropriate protective clothing and equipment when a hazard is present or is likely to be present from skin or eye contact with Cr(VI). OSHA would expect an employer to exercise common sense and appropriate expertise to determine if a hazard is present or likely to be present. Is this approach appropriate? Are there other approaches that would be better for characterizing eye and skin contact with Cr(VI)? For example, are there methods to measure dermal exposure that could be used to routinely monitor worker exposure to Cr(VI) that OSHA should consider including in the final standard?

52. For employers whose employees are exposed to Cr(VI), what approaches do you currently use to assess potential hazards from eye or skin contact with Cr(VI)? What protective clothing and equipment do you use to protect employees from eye or skin contact with Cr(VI)? What does this protective clothing and equipment cost? Who pays for the protective clothing and equipment?

53. Should OSHA require the use of protective clothing and equipment for those employees who are exposed to airborne concentrations of Cr(VI) in excess of the PEL? If so, what type of protective clothing and equipment might be necessary?

54. OSHA has proposed to require that employers pay for protective clothing and equipment provided to employees. The Agency seeks comment on this provision, in particular:

a. Should OSHA refrain from requiring employer payment, and follow the outcome of the rulemaking addressing employer payment for personal protective equipment (64 FR 15401 (3/31/99))?

b. Are there circumstances where employers should not be required to pay for clothing and equipment used to protect employees from Cr(VI) hazards, such as situations where it is customary for employees to provide their own protective clothing and equipment (i.e., ''tools of the trade'')?

c. OSHA realizes that there is frequent turnover in the construction industry, where employees frequently move from jobsite to jobsite. This is an important factor because an employer with a high-turnover workplace would have to buy protective clothing and equipment for more employees if the protective clothing and equipment could only be used by one employee. The Agency requests comment on whether this proposal's requirement for employer payment for protective clothing and equipment is appropriate in the construction industry. Are there any alternative approaches that would be responsive to the turnover situation and would also be protective of construction workers? Are there any other issues specific to the construction industry that OSHA should be consider in this rulemaking?

d. At some ports, employees are hired for jobs in shipyards, longshoring, and marine terminals through a labor pool, and a single employee may work for five different employers in the same week. How do these factors affect who is required to pay for protective clothing and equipment? Are there any other issues specific to shipyards, longshoring, or marine terminals that OSHA should consider in this rulemaking?

55. OSHA is proposing that washing facilities capable of removing Cr(VI) from the skin be provided to affected employees, but does not propose that showers be required. Should OSHA include requirements to provide showers to employees exposed to Cr(VI)? If so, under what circumstances should showers be required? Describe work situations where showers are either unnecessary for employee protection or that present obstacles to their implementation and describe any such obstacles.

56. OSHA has not included housekeeping provisions in the proposed Cr(VI) standard for construction or shipyards. The Agency has made a preliminary determination that the housekeeping requirements proposed for general industry are likely to be difficult to implement in the construction and shipyard environments. Is this an appropriate determination? If not, what practicable housekeeping measures can construction and shipyard employers take to reduce employee exposure to Cr(VI) at the work site? What housekeeping activities are currently being performed?

57. Is medical surveillance being provided to Cr(VI)-exposed employees at your worksite? If so,

a. What exposure levels or other factors trigger medical surveillance?

b. What tests or evaluations are included in the medical surveillance program?

c. What benefits have been achieved from the medical surveillance program?

d. What are the costs of the medical surveillance program? How do your current costs compare with OSHA's estimated unit costs for the physical examination and employee time involved in the medical surveillance program? Please comment on OSHA's baseline assumptions and cost estimates for medical surveillance.

e. How many employees are included in your medical surveillance program?

f. In what North American Industry Classification System (NAICS) code does your workplace fall?

58. OSHA has proposed that medical surveillance be triggered in general industry in the following circumstances: (1) When exposure to Cr(VI) is above the PEL for 30 days or more per year; (2) after an employee experiences signs or symptoms of the adverse health effects associated with Cr(VI) exposure (*e.g.*, dermatitis, asthma); or (3) after exposure in an emergency. OSHA seeks comments as to whether or not these are appropriate triggers for offering medical surveillance and whether there are additional triggers that should be included. Should OSHA require that medical surveillance be triggered in general industry only upon an employee experiencing signs and symptoms of disease or after exposure in an emergency, as in the construction and maritime standards? OSHA also solicits comment on the optimal frequency of medical surveillance.

59. OSHA has proposed that medical surveillance be triggered in construction and shipyards in the following circumstances: (1) after an employee experiences signs or symptoms of the adverse health effects associated with Cr(VI) exposure (*e.g.*, dermatitis, asthma); or (2) after exposure in an emergency. Should medical surveillance in construction or shipyards be triggered by exposure to Cr(VI) above the PEL for 30 days or more per year, as proposed for general industry? OSHA seeks comments as to whether or not the proposed triggers are appropriate for offering medical surveillance and whether there are additional triggers that should be included.

60. OSHA has not included certain biological tests (*e.g.*, blood or urine monitoring, skin patch testing for sensitization, expiratory flow measurements for airway restriction) as a part of the medical evaluations required to be provided to employees offered medical surveillance under the proposed standard. OSHA has preliminarily determined that the general application of these tests is of uncertain value as an early indicator of potential Cr(VI)-related health effects. However, the proposed standard does allow for the provision of any tests (which could include urine or blood tests) that are deemed necessary by the physician or other licensed health care professional. Are there any tests (*e.g.*, urine tests, blood tests, skin patch tests, airway flow measurements, or others) that should be included under the proposed standard's medical surveillance provisions? If there are any that should be included, explain the rationale for their inclusion, including the benefit to worker health they might provide, their utility and ease of use in an occupational health surveillance program, and associated costs.

61. OSHA has not included requirements for medical removal protection (MRP) in the proposed standard. OSHA has made a preliminary determination that there are few instances where temporary worker removal and MRP will be useful. The Agency seeks comment as to whether the final Cr(VI) standard should include provisions for the temporary removal and extension of MRP benefits to employees with certain Cr(VI)-related health conditions. In particular, what endpoints should be considered for temporary removal and for what maximum amount of time should MRP benefits be extended? OSHA also seeks information on whether or not MRP is currently being used by employers with Cr(VI)-exposed workers, and the costs of such programs.

62. OSHA has proposed that employers provide hazard information to employees in accordance with the Agency's Hazard Communication standard (29 CFR 1910.1200), and has also proposed additional requirements regarding signs, labels, and additional training specific to work with Cr(VI). Should OSHA include these additional requirements in the final rule, or are the requirements of the Hazard Communication standard sufficient?

63. OSHA has proposed that bags or containers of laundry contaminated with Cr(VI) bear warning labels. Will this cause you to alter your current laundry practices? Are there laundries in your area that would accept such laundry? Would laundering costs increase? If so, by how much?

64. OSHA requests comment on the time allowed for compliance with the provisions of the proposed standard. Is the time proposed sufficient, or is a longer or shorter phase-in of requirements appropriate? Identify any industries, processes, or operations that have special needs for additional time, the additional time required and the reasons for the request.

65. Some other OSHA health standards have included appendices that address topics such as the hazards associated with the regulated substance, health screening considerations, occupational disease questionnaires, and PLHCP obligations. OSHA has not proposed to include any appendices with the Cr(VI) rule because the Agency has made a preliminary determination that such topics would be best addressed with guidance materials. What would be the advantage of including such appendices in the final rule? If you believe they should be included, what information should be included? What would be the disadvantage of including these appendices in the final rule?

III. Pertinent Legal Authority

The purpose of the Occupational Safety and Health Act, 29 U.S.C. 651 et seq. (''the Act'') 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.'' 29 U.S.C. 651(b). To achieve this goal Congress authorized the Secretary of Labor to promulgate and enforce occupational safety and health standards. 29 U.S.C. 655(a)(authorizing summary adoption of existing consensus and federal standards within two years of Act's enactment), 655(b)(authorizing promulgation of standards pursuant to notice and comment), 654(b)(requiring employers to comply with OSHA standards).

A safety or health standard is a standard ''which requires conditions or the adoption of or use of one or more practices, means, methods, operations or processes, reasonably necessary or appropriate to provide safe or healthful employment or places of employment 29 U.S.C. 652(8).

A standard is reasonably necessary or appropriate within the meaning of Section 652(8) if it substantially reduces or eliminates significant risk, and is economically feasible, technologically feasible, cost effective, consistent with prior Agency action or supported by a reasoned justification for departing from prior Agency actions, supported by substantial evidence, and is better able to effectuate the Act's purpose than any national consensus standard it supersedes. See 58 Fed. Reg. 16612–16616 (March 30, 1993).

OSHA has generally considered, at minimum, fatality risk of 1/1000 over a 45-year working lifetime to be a significant health risk. *See* the Benzene standard, *Industrial Union Dep't* v. *American Petroleum Institute,* 448 U.S. 607, 646 ((1980); the Asbestos standard, *International Union, UAW* v. *Pendergrass,* 878 F.2d 389, 393 (D.C. Cir. 1989).

A standard is technologically feasible if the protective measures it requires already exist, can be brought into existence with available technology, or can be created with technology that can reasonably be expected to be developed. *American Textile Mfrs. Institute* v. *OSHA,* 452 U.S. 490, 513 (1981)(''ATMI'') American Iron and Steel Institute v. OSHA, 939 F.2d 975, 980 (D.C. Cir. 1991)(''AISI'').

A standard is economically feasible if industry can absorb or pass on the costs of compliance without threatening its long-term profitability or competitive structure. *See* ATMI, 452 U.S. at 530 n. 55; AISI, 939 F. 2d at 980.

A standard is cost effective if the protective measures it requires are the least costly of the available alternatives that achieve the same level of protection. ATMI, 453, U.S, at 514 n. 32; International *Union, UAW* v. *OSHA,* 37 F.3d 665, 668 (D.C., Cir 1994)(''LOTO III'').

All standards must be highly protective. See 58 FR 16614–16615; LOTO III, 37 F. 3d at 669. However, health standards must also meet the ''feasibility mandate'' of Section 6(b)(7) of the Act, 29 U.S.C. 655(b)(5). Section 6(b)(5) requires OSHA to select ''The most protective standard consistent with feasibility'' that is needed to reduce significant risk when regulating health standards. ATMI, 452 U.S. at 509.

Section 6(b)(5) also directs OSHA to base health standard on ''the best available evidence,'' including research, demonstrations, and experiments. 29 U.S.C. 655(b)(5). OSHA shall consider ''in addition to the attainment of the highest degree of health and safety protection * * * feasibility and experience gained under this and other health and safety laws.'' *Id.*

Section 6(b)(7) authorizes OSHA to include among a standard's requirements labeling, monitoring, medical testing and other information gathering and transmittal provisions. 29 U.S.C. 655(b)(7).

Finally, whenever practical, standards shall ''be expressed in terms of objective criteria and of the performance desired.'' *Id.*

IV. Events Leading to the Proposed Standards

OSHA's present standards for workplace exposure to Cr(VI) were adopted in 1971, pursuant to section 6(a) of the OSH Act, from a 1943 American National Standards Institute (ANSI) recommendation originally established to control irritation and damage to nasal tissues (Ex. 20–3). OSHA's general industry standard set a permissible exposure limit (PEL) of 1 mg chromium trioxide per 10 m^3 air in the workplace (1 mg/10 m^3 CrO_3) as a ceiling concentration, which corresponds to a concentration of 52 µg/m^3 Cr(VI). A separate rule promulgated for the construction industry set an eight-hour time-weighted-average PEL of 1 mg/10 m^3 CrO_3, also equivalent to 52 µg/m^3 Cr(VI), adopted from the American Conference of Governmental Industrial Hygienists (ACGIH) 1970 Threshold Limit Value (TLV) (36 FR 7340 (4/17/71)).

Following the ANSI standard of 1943, other occupational and public health organizations evaluated Cr(VI) as a workplace and environmental hazard and formulated recommendations to control exposure. The ACGIH first recommended control of workplace exposures to chromium in 1946, recommending a time-weighted average Maximum Allowable Concentration (later called a Threshold Limit Value) of 100 µg/m^3 for chromic acid and chromates as Cr_2O^3 (Ex. 5–37), and classified certain Cr(VI) compounds as class A1 (confirmed human) carcinogens in 1974. In 1975, the NIOSH Criteria for a Recommended Standard recommended that occupational exposure to Cr(VI) compounds should be limited to a 10-hour TWA of 1 µg/m^3, except for some forms of Cr(VI) then believed to be noncarcinogenic (Ex. 3–92). The National Toxicology Program's First Annual Report on Carcinogens identified calcium chromate, chromium chromate, strontium chromate, and zinc chromate as carcinogens in 1980 (Ex. 35–157).

During the 1980s, regulatory and standards organizations came to recognize Cr(VI) compounds in general as carcinogens. The Environmental Protection Agency (EPA) Health Assessment Document of 1984 stated that ''using the IARC [International Agency for Research on Cancer] classification scheme, the level of evidence available for the combined animal and human data would place hexavalent chromium Cr(VI) compounds into Group 1, meaning that there is decisive evidence for the carcinogenicity of those compounds in humans'' (Ex. 19–1, p. 7–107). In 1988 IARC evaluated the available evidence regarding Cr(VI) carcinogenicity, concluding in 1990 that ''There is sufficient evidence in humans for the carcinogenicity of chromium[VI] compounds as encountered in the chromate production, chromate pigment production and chromium plating industries'', and ''sufficient evidence in experimental animals for the carcinogenicity of calcium chromate, zinc chromates, strontium chromate and lead chromates'(Ex. 18–3, p. 213). In September 1988, NIOSH advised OSHA to consider all Cr(VI) compounds as potential occupational carcinogens (Ex. 31–22–22, p. 8). ACGIH now classifies water-insoluble and water-soluble Cr(IV) compounds as class A1 carcinogens (Ex. 35–207). Current ACGIH standards include specific 8-hour time-weighted average TLVs for calcium chromate (1 µg/m^3), lead chromate (12 µg/m^3), strontium chromate (0.5 µg/m^3), and zinc chromates (10 µg/m^3), and generic TLVs for water soluble (50 µg/m^3) and insoluble (10 µg/m^3) forms of hexavalent chromium not otherwise classified, all measured as chromium (Ex. 35–207).

In July 1993, OSHA was petitioned for an emergency temporary standard to reduce occupational exposures to Cr(VI) compounds (Ex. 1). The Oil, Chemical, and Atomic Workers International Union (OCAW) and Public Citizen's Health Research Group (HRG), citing evidence that occupational exposure to Cr(VI) increases workers' risk of lung cancer, petitioned OSHA to promulgate an emergency temporary standard to lower the PEL for Cr(VI) compounds to 0.5 µg/m^3 as an eight-hour, time-weighted average (TWA). Upon review of the petition, OSHA agreed that there was evidence of increased cancer risk from exposure to Cr(VI) at the existing PEL, but found that the available data did not show the ''grave danger'' required to support an emergency temporary standard (Ex. 1–C). The Agency therefore denied the request for an emergency temporary standard, but initiated section 6(b)(5) rulemaking and began performing preliminary analyses relevant to the rule. In 1997, OSHA was sued by HRG for unreasonable delay in issuing a Cr(VI) standard. The U.S. Court of Appeals for the Third Court ruled in OSHA's favor and the Agency continued its data collection and analytic efforts on Cr(VI) (Ex. 35–208, p. 3). OSHA was sued again in 2002 by HRG for continued unreasonable delay in issuing a Cr(VI) standard (Ex. 31–24–1). In August 2002, OSHA published a Request for Information on Cr(VI) to solicit additional information on key issues related to controlling exposures to Cr(VI)(67 FR 54389 (8/22/02)), and on December 4, 2002 announced its intent to proceed with developing a proposed standard (Ex. 307). The Court ruled in favor of HRG on December 24, 2002, ordering the Agency to proceed expeditiously with a Cr(VI) standard (Ex. 35–208). On April 2, 2003 the Court set deadlines of October 4, 2004 for publication of a proposed standard and January 18, 2006 for publication of a final standard (Ex. 35–306).

OSHA initiated Small Business Regulatory Enforcement Act (SBREFA) proceedings in 2003, seeking the advice of small business representatives on the proposed rule. The SBREFA panel, including representatives from OSHA, the Small Business Administration (SBA), and the Office of Management and Budget (OMB), was convened on December 23, 2003. The panel conferred with representatives from small entities in chemical, alloy, and pigment manufacturing, electroplating, welding, aerospace, concrete, shipbuilding, masonry, and construction on March 16–17, 2004, and delivered its final report to OSHA on April 20, 2004. The Panel's report, including comments from the small entity representatives (SERS) and recommendations to OSHA for the proposed rule, is available in the Cr(VI) rulemaking docket (Ex. 34).

OSHA provided the Advisory Committee on Construction Safety and Health (ACCSH) and the Maritime Advisory Committee on Occupational Safety and Health (MACOSH) with copies of the draft proposed rule for review in early 2004. OSHA representatives met with ACCSH in February 2004 and May 2004 to discuss the rulemaking and receive their comments and recommendations. On February 13, ACCSH recommended that portland cement should be included

within the scope of the proposed standard (Ex. 35–308, pp. 288–293) and that identical PELs should be set for the construction, maritime, and general industries (Ex. 35–308, pp. 293–297). The Committee recommended on May 18 that the construction industry should be included in the current rulemaking, and affirmed its earlier recommendation regarding portland cement. OSHA representatives met with MACOSH in March 2004. On March 3, MACOSH decided to collect and forward additional exposure monitoring data to OSHA to help the Agency better evaluate exposures to Cr(VI) in shipyards (Ex. 310, p. 208). MACOSH also recommended a separate Cr(VI) standard for the maritime industry, arguing that maritime involves different exposures and requires different means of exposure control than general industry and construction (Ex. 310, p. 227).

V. Chemical Properties and Industrial Uses

Chromium is a metal that exists in several oxidation or valence states, ranging from chromium (−II) to chromium (+VI). The elemental valence state, chromium (0), does not occur in nature. Chromium compounds are very stable in the trivalent state and occur naturally in this state in ores such as ferrochromite, or chromite ore ($FeCr_2O_4$). The hexavalent, Cr(VI) or chromate, is the second most stable state. It rarely occurs naturally; most Cr(VI) compounds are man made.

Chromium compounds in higher valence states are able to undergo ''reduction'' to lower valence states; chromium compounds in lower valence states are able to undergo ''oxidation'' to higher valence states. Thus, Cr(VI) compounds can be reduced to Cr(III) in the presence of oxidizable organic matter. Chromium can also be reduced in the presence of inorganic chemicals such as iron.

Chromium does exist in less stable oxidation (valence) states such as Cr(II), Cr(IV), and Cr(V). Anhydrous Cr(II) salts are relatively stable, but the divalent state (II, or chromous) is generally relatively unstable and is readily oxidized to the trivalent (III or chromic) state. Compounds in valence states such as (IV) and (V) usually require special handling procedures as a result of their instability. Cr(IV) oxide (CrO_2) is used in magnetic recording and storage devices, but very few other Cr(IV) compounds have industrial use. Evidence exists that both Cr(IV) and Cr(V) are formed as transient intermediates in the reduction of Cr(VI) to Cr(III) in the body.

Chromium (III) is also an essential nutrient that plays a role in glucose, fat, and protein metabolism by causing the action of insulin to be more effective. Chromium picolinate, a trivalent form of chromium combined with picolinic acid, is used as a dietary supplement, because it is claimed to speed metabolism.

Elemental chromium and the chromium compounds in their different valence states have various physical and chemical properties, including differing solubilities. Most chromium species are solid. Elemental chromium is a steel gray solid, with high melting and boiling points (1857 °C and 2672 °C, respectively), and is insoluble in water and common organic solvents. Chromium (III) chloride is a violet or purple solid, with high melting and sublimation points (1150 °C and 1300 °C, respectively), and is slightly soluble in hot water and insoluble in common organic solvents. Ferrochromite is a brown-black solid; chromium (III) oxide is a green solid; and chromium (III) sulfate is a violet or red solid, insoluble in water and slightly soluble in ethanol. Chromium (III) picolinate is a ruby red crystal soluble in water (1 part per million at 25 °C). Chromium (IV) oxide is a brown-black solid that decomposes at 300 °C and is insoluble in water.

Cr(VI) compounds have mostly lemon yellow to orange to dark red hues. They are typically crystalline, granular, or powdery although one compound (chromyl chloride) exists in liquid form. They range from very soluble to insoluble in water. For example, chromyl chloride is a dark red liquid that decomposes into chromate ion and hydrochloric acid in water. Chromic acids are dark red crystals that are very soluble in water. Other examples of soluble chromates are potassium chromate (lemon yellow crystals), sodium chromate (yellow crystals), and sodium dichromate (reddish to bright orange crystals). Nickel chromate, lead chromate oxide, and zinc chromate are completely insoluble in water. The nickel chromate (black crystals) dissolves in nitric acid and hydrogen peroxide. Lead chromate oxide is a red crystalline powder. The zinc chromate (lemon yellow crystals) decomposes in hot water and is soluble in acids and liquid ammonia. Examples of slightly soluble Cr(VI) compounds are barium (light yellow), calcium (yellow), lead (yellow to orange-yellow), and strontium (yellow) chromates, and zinc chromate hydroxide (yellow). They all exist in solid form as crystals or powder. Potassium zinc chromate hydroxide (greenish-yellow crystals) is also slightly soluble in water.

Some major users of chromium are the metallurgical, refractory, and chemical industries. Chromium is used by the metallurgical industry to produce stainless steel, alloy steel, and nonferrous alloys. Chromium is alloyed with other metals and plated on metal and plastic substrates to improve corrosion resistance and provide protective coatings for automotive and equipment accessories. Welders use stainless steel welding rods when joining metal parts.

Cr(VI) compounds are widely used in the chemical industry in pigments, metal plating, and chemical synthesis as ingredients and catalysts. Chromates are used as high quality pigments for textile dyes, paints, inks, glass, and plastics. Cr(VI) can be produced during welding operations even if the chromium was originally present in another valence state. While Cr(VI) is not intentionally added to portland cement, it is often present as an impurity.

Occupational exposures to Cr(VI) can occur from inhalation of mists (*e.g.*, chrome plating, painting), dusts (*e.g.*, inorganic pigments), or fumes (*e.g.*, stainless steel welding), and from dermal contact (cement workers).

There are about thirty major industries and processes where Cr(VI) is used. These include producers of chromates and related chemicals from chromite ore, electroplating, welding, painting, chromate pigment production and use, steel mills, and iron and steel foundries. A detailed discussion of the uses of Cr(VI) in industry is found in Section IX of this preamble.

VI. Health Effects

The studies of adverse health effects resulting from exposure to hexavalent chromium (Cr(VI)) in humans and experimental animals are summarized in the section below. Section VI includes information on the fate of Cr(VI) in the body and laboratory research that relates to its toxic mode of action. The primary health impairments from workplace exposure to Cr(VI) are lung cancer, asthma, and damage to the nasal epithelia and skin. This chapter on health effects will not attempt to describe every study ever conducted on Cr(VI) toxicity. Instead, only the most important articles and reviews of studies will be evaluated.

A. Absorption, Distribution, Metabolic Reduction and Elimination

Chromium can exist in a number of valence states from −2 to +6 valence. The most common forms are the elemental metal Cr(0), trivalent Cr(III), and hexavalent Cr(VI). Chromium exists naturally in the environment in

chromite ore as Cr(III). Cr(0) and Cr(VI), as well as Cr(III) are produced during industrial processes. Cr(VI) is the form considered to be the greatest health risk. A small amount of Cr(III) is needed for optimal insulin receptor function in human tissues but much larger amounts may be harmful. Much less is known about the toxicity of Cr(0), but it is believed to be converted to Cr(III) in the body and is not considered to be a serious health risk. Cr(VI) enters the body by inhalation, ingestion, or absorption through the skin. For occupational exposure, the airways and skin are the primary routes of uptake.

1. Deposition and Clearance of Inhaled Cr(VI) From the Respiratory Tract

Various anatomical, physical and physiological factors determine both the fractional and regional deposition of inhaled particulate matter. Due to the airflow patterns in the lung more particles tend to deposit at certain preferred regions in the lung. Schlesinger and Lippman have shown a high degree of correlation between sites of greatest particle deposition in the tracheobronchial airways and increased incidence of bronchial tumors (Ex. 35–102). It is possible to have a buildup of chromium at certain sites in the bronchial tree that could create areas of very high chromium concentration. This would especially be true for occupational environments that are particularly dusty or contain other irritating aerosols.

Large inhaled particles (>5 μm) are efficiently removed from the air-stream in the extrathoracic region (Ex. 35–175). Particles greater than 2.5 μm are generally deposited in the tracheobronchial regions, whereas particles less than 2.5 μm are generally deposited in the pulmonary region. Some larger particles (>2.5 μm) can reach the pulmonary region. The mucociliary escalator predominantly clears particles that deposit in the extrathoracic and the tracheobronchial region of the lung. Individuals exposed to high particulate levels of Cr(VI) may also have altered respiratory mucociliary clearance. Particulates that reach the alveoli can be absorbed into the bloodstream cleared by phagocytosis.

2. Absorption of Inhaled Cr(VI) Into the Bloodstream

The absorption of inhaled chromium compounds depends on a number of factors, including physical and chemical properties of the particles (oxidation state, size, solubility) and the activity of alveolar macrophages (Ex. 35–41). The hexavalent chromate anion $(CrO_4)^{2-}$ enter cells via facilitated diffusion through non-specific anion channels (similar to phosphate and sulfate anions). Suzuki *et al.* have demonstrated that Cr(VI) is rapidly and extensively transported to the bloodstream in rats (Ex. 35–97). They exposed rats to 7.3–15.9 mg Cr(VI)/m^3 as potassium dichromate for 2–6 hours. Following exposure to Cr(VI), the ratio of blood chromium/lung chromium was 1.44±0.30 at 0.5 hours, 0.81±0.10 at 18 hours, 0.85±0.20 at 48 hours, and 0.96±0.22 at 168 hours after exposure.

Once the Cr(VI) particles reach the alveoli, absorption into the bloodstream is greatly dependent on solubility. Bragt and van Dura demonstrated that more soluble chromates are absorbed faster than less soluble chromates (Ex. 35–56). Insoluble chromates are poorly absorbed and therefore have longer resident time in the lungs. They studied the kinetics of three Cr(VI) compounds: Sodium chromate, zinc chromate and lead chromate. They instilled ^{51}chromium-labeled compounds (0.38 mg Cr(VI)/kg as sodium chromate, 0.36 mg Cr(VI)/kg as zinc chromate, or 0.21 mg Cr(VI)/kg as lead chromate) intratracheally in rats. Peak blood levels of ^{51}chromium were reached after 30 minutes for sodium chromate (0.35 μg chromium/ml), and after 24 hours for zinc chromate (0.60 μg chromium/ml) and lead chromate (0.007 μg chromium/ml). At 30 minutes after administration, the lungs contained 36, 25, and 81% of the respective dose of the sodium, zinc, and lead chromate. On day six, >80% of the dose of all three compounds had been cleared from the lungs, during which time the disappearance from lungs followed linear first-order kinetics. The residual amount left in the lungs on day 50 or 51 was 3.0, 3.9, and 13.9%, respectively. From these results authors concluded that zinc chromate, which is less soluble than sodium chromate, is more slowly absorbed from the lungs. Lead chromate was more poorly and slowly absorbed, as indicated by very low levels in blood and greater retention in the lungs. The authors also noted that the kinetics of sodium and zinc chromates were very similar. Zinc chromate, which is less soluble than sodium chromate, was slowly absorbed from the lung, but the maximal blood levels were higher than those resulting from an equivalent dose of sodium chromate. The authors believe that this was probably due to irritative properties of the zinc chromate used, as it caused hemorrhages in the lungs which were macroscopically visible as early as 24 hours after intratracheal administration.

The studies by Langard *et al.* and Adachi *et al.* provide further evidence of absorption of chromates from the lungs (Exs. 35–93; 189). Rats exposed to 2.1 mg Cr(VI)/m^3 as zinc chromate for 6 hours/day achieved steady state concentrations in the blood after 4 days of exposure (Ex. 35–93). Adachi *et al.* studied rats that were subject to a single inhalation exposure to chromic acid mist generated from electroplating at a concentration of 3.18 mg Cr(VI)/m^3 for 30 minutes which was then rapidly absorbed from the lungs (Ex. 189). The amount of chromium in the lungs of these rats declined from 13.0 mg immediately after exposure to 1.1 mg after 4 weeks, with an overall half-life of five days.

Several other studies have reported absorption of chromium from the lungs after intratracheal instillation (Exs. 7–9; 9–81; Visek *et al.* 1953 as cited in Ex. 35–41). These studies indicated that 53–85% of Cr(VI) compounds (particle size <5 μm) were cleared from the lungs by absorption into the bloodstream or by mucociliary clearance in the pharynx; the rest remained in the lungs. Absorption of Cr(VI) from the respiratory tract of workers has been shown in several studies that identified chromium in the urine, serum and red blood cells following occupational exposure (Exs. 5–12; 35–294; 35–84).

Evidence indicates that even chromates that are encapsulated in a paint matrix may be released in the lungs (Ex. 31–15, p. 2). LaPuma *et al.* measured the mass of Cr(VI) released from particles into water originating from three types of paint particles: solvent-borne expoxy (25% strontium chromate ($SrCrO_4$)), water-borne expoxy (30% $SrCrO_4$) and polyurethane (20% $SrCrO_4$) (Ex. 31–2–1). The mean fraction of Cr(VI) released into the water after one and 24 hours for each primer averaged: 70% and 85% (solvent epoxy), 74% and 84% (water epoxy), and 94% and 95% (polyurethane). Correlations between particle size and the fraction of Cr(VI) released indicated that smaller particles (<5 m) release a larger fraction of Cr(VI) versus larger particles (>5 μm). This study demonstrates that the paint matrix only modestly hinders Cr(VI) release into a fluid, especially with smaller particles. Larger particles, which contain the majority of Cr(VI) due to their size, appear to release proportionally less Cr(VI) (as a percent of total Cr(VI)) than smaller particles.

A number of questions remain unanswered regarding encapsulated Cr(VI) and bioavailability from the lung. There is a lack of detailed information on the encapsulation process. The efficiency of encapsulation and whether all of the chromate molecules are

encapsulated is not known. The stability of the encapsulated product in physiological and environmental conditions has not been demonstrated. It would be useful to know if any processes can break the encapsulation during its use. Finally, the fate of inhaled encapsulated and unencapsulated Cr(VI) in the respiratory tract as well as the systemic tissues needs to be more thoroughly studied.

3. Dermal Absorption of Cr(VI)

Both human and animal studies demonstrate that Cr(VI) compounds are absorbed after dermal exposure. Dermal absorption depends on the oxidation state of chromium, the vehicle and the integrity of the skin. Cr(VI) readily traverses the epidermis to the dermis (Exs. 9–49; 309). The histological distribution of Cr(VI) within intact human skin was studied by Liden and Lundberg (Ex. 35–80). They applied test solutions of potassium dichromate in petrolatum or in water as occluded circular patches of filter paper to the skin. Results with potassium dichromate in water revealed that Cr(VI) penetrated beyond the dermis and penetration reached steady state with resorption by the lymph and blood vessels by 5 hours. About 10 times more chromium penetrated when potassium dichromate was applied in petrolatum than when applied in water, indicating that organic solvents facilitate the absorption of Cr(VI) from the skin. Baranowska-Dutkiewicz also demonstrated that the absorption rates of sodium chromate solutions from the occluded forearm skin of volunteers increase with increasing concentration (Ex. 35–75). The rates were 1.1 μg Cr(VI)/cm^2/hour for a 0.01 molar solution, 6.4 μg Cr(VI)/cm^2/hour for a 0.1 molar solution, and 10 μg Cr(VI)/cm^2/hour for a 0.2 molar solution.

Using volunteers, Mali found that potassium dichromate penetrates the intact epidermis (Exs. 9–49; 35–41). Wahlberg and Skog demonstrated the presence of chromium in the blood, spleen, bone marrow, lymph glands, urine and kidneys of guinea pigs exposed to ^{51}chromium labeled Cr(VI) compounds (Ex. 35–81). In this study radiolabeled sodium chromate solution was dermally applied to guinea pigs and ^{51}Cr was monitored by scintillation counting in tissues. These studies demonstrate that the absorption of Cr(VI) compounds can take place through the dermal route. Also, the absorption of Cr(VI) can be facilitated by organic solvents.

4. Absorption of Cr(VI) by the Oral Route

Inhaled Cr(VI) can enter the digestive tract as a result of mucocilliary clearance and swallowing. Studies indicate Cr(VI) is absorbed from the gastrointestinal tract. The six-day fecal and 24-hour urinary excretion patterns of radioactivity in groups of six volunteers given Cr(VI) as sodium chromate labeled with ^{51}chromium indicated that at least 2.1% of the Cr(VI) was absorbed. After intraduodenal administration at least 10% of the Cr(VI) compound was absorbed. These studies also demonstrated that Cr(VI) compounds are reduced to Cr(III) compounds in the stomach, thereby accounting for the relatively poor gastrointestinal absorption of orally administered Cr(VI) compounds (Exs. 35–96; 35–41).

In the gastrointestinal tract, Cr(VI) can be reduced to Cr(III) by gastric juices, which is then poorly absorbed (Underwood, 1971 as cited in Ex. 19–1; Ex. 35–85). The mechanism by which Cr(VI) is carried across the intestinal wall and the site of absorption are not known and may well depend upon the efficiency of defense mechanisms (Mertz, 1969 as cited in Ex. 19–1).

Kuykendall *et al.* studied the absorption of Cr(VI) in human volunteers after oral administration of potassium dichromate (Ex. 35–77). They reported the bioavailability based on 14-day urinary excretion to be 6.9% (range 1.2–18%) for Cr(VI). Other investigators have also reported absorption of Cr(VI) compounds after oral administration (Exs. 35–76; 31–22–13; 35–91).

Studies with ^{51}chromium in animals also indicate that chromium and its compounds are poorly absorbed from the gastrointestinal tract after oral exposure. When radioactive sodium chromate (Cr(VI)) was given orally to rats, the amount of chromium in the feces was greater than that found when sodium chromate was injected directly into the small intestine. These results are consistent with evidence that the gastric environment has a capacity to reduce Cr(VI) to Cr(III) and therefore decrease the amount of Cr(VI) absorbed from the GI tract.

Treatment of rats by gavage with an unencapsulated lead chromate pigment or with a silica-encapsulated lead chromate pigment resulted in no measurable blood levels of chromium (measured as Cr(III), detection limit=10 μg/L) after two or four weeks of treatment or after a two-week recovery period. However, kidney levels of chromium (measured as Cr(III)) were significantly higher in the rats that received the unencapsulated pigment when compared to the rats that received the encapsulated pigment, indicating that silica encapsulation may reduce the gastrointestinal bioavailability of chromium from lead chromate pigments (Ex. 11–5). This study does not address the bioavailability of encapsulated chromate pigments from the lung where residence time could be different.

5. Distribution of Cr(VI) in the Body

Once in the bloodstream, Cr(VI) is taken up into erythrocytes, where it is reduced to lower oxidation states and forms chromium protein complexes during reduction (Ex. 35–41). Once complexed with protein, chromium cannot leave the cell. The binding of chromium compounds by proteins in the blood has been studied in some detail (Exs. 5–24; 35–41; 35–52). It was found that intravenously injected anionic Cr(VI) passes through the membrane of red blood cells and binds to the globin fraction of hemoglobin. It has been hypothesized that before Cr(VI) is bound by hemoglobin, it is reduced to Cr(III) by an enzymatic reaction within red blood cells. Once inside the blood cell, chromium ions are unable to repenetrate the membrane and move back into the plasma (Exs. 7–6; 7–7; 19–1; 35–41; 35–52). According to Aaseth *et al.*, the intracellular Cr(VI) reduction depletes Cr(VI) concentration in the red blood cell (Ex. 35–89). This serves to enhance diffusion of Cr(VI) from the plasma into the erythrocyte resulting in very low plasma levels of Cr(VI). It is also believed that the rate of uptake of Cr(VI) by red blood cells may not exceed the rate at which they reduce Cr(VI) to Cr(III) (Ex. 35–99). The higher tissue levels of chromium after administration of Cr(VI) than after administration of Cr(III) reflect the greater tendency of Cr(VI) to traverse plasma membranes and bind to intracellular proteins in the various tissues, which may explain the greater degree of toxicity associated with Cr(VI) (MacKenzie *et al.* 1958 as cited in 35–52; Maruyama 1982 as cited in 35–41; Ex. 35–71).

Examination of autopsy tissues from chromate workers who were occupationally exposed to Cr(VI) showed that the highest chromium levels were in the lungs. The liver, bladder, and bone also had chromium levels above background. Mancuso examined tissues from three individuals with lung cancer who were exposed to chromium in the workplace (Ex. 124). One was employed for 15 years as a welder, the second and third worked for 10.2 years and 31.8 years, respectively, in ore milling and preparations and boiler operations. The cumulative

chromium exposures for the three workers were estimated to be 3.45, 4.59, and 11.38 mg/m ^{3}–years, respectively. Tissues from the first worker were analyzed 3.5 years after last exposure, the second worker 18 years after last exposure, and the third worker 0.6 years after last exposure. All tissues from the three workers had elevated levels of chromium, with the possible exception of neural tissues. Levels were orders of magnitude higher in the lungs when compared to other tissues. The highest lung level reported was 456 mg/10 g tissue in the first worker, 178 in the second worker, and 1,920 for the third worker. There were significant chromium levels in the tissue of the second worker even though he had not been exposed to chromium for 18 years. Similar results were also reported in autopsy studies of people who may have been exposed to chromium in the workplace as well as chrome platers and chromate refining workers (Exs. 35–92; 21–1; 35–74; 35–88).

Animal studies have shown similar distribution patterns after inhalation exposure. The distribution of Cr(VI) compared with Cr(III) was investigated in guinea pigs after intratracheal instillation of potassium dichromate or chromium trichloride (Ex. 7–8). At 24 hours after instillation, 11% of the original dose of chromium from potassium dichromate remained in the lungs, 8% in the erythrocytes, 1% in plasma, 3% in the kidney, and 4% in the liver. The muscle, skin, and adrenal glands contained only a trace. All tissue concentrations of chromium declined to low or nondetectable levels in 140 days, with the exception of the lungs and spleen. After chromium trichloride instillation, 69% of the dose remained in the lungs at 20 minutes, while only 4% was found in the blood and other tissues, with the remaining 27% cleared from the lungs and swallowed. The only tissue that contained a significant amount of chromium two days after instillation of chromium trichloride was the spleen. After 30 and 60 days, 30 and 12%, respectively, of the Cr(III) was retained in the lungs, while only 2.6 and 1.6%, respectively, of the Cr(VI) dose was retained in the lungs.

6. Metabolic Reduction of Cr(VI)

Cr(VI) is reduced to Cr(III) in the lungs by a variety of reducing agents. This serves to limit uptake into lung cells and absorption into the bloodstream. Cr(V) and Cr(IV) are transient intermediates in this process. The genotoxic effects produced by the Cr(VI) are related to the reduction process and are further discussed in the section on Mechanistic Considerations.

In vivo and *in vitro* experiments in rats indicated that, in the lungs, Cr(VI) can be reduced to Cr(III) by ascorbate and glutathione. The reduction of Cr(VI) by glutathione is slower than the reduction by ascorbate (Ex. 35–65). Other studies have reported the reduction of Cr(VI) to Cr(III) by epithelial lining fluid (ELF) obtained from the lungs of 15 individuals by bronchial lavage. The average overall reduction capacity was 0.6 µg Cr(VI)/mg of ELF protein. In addition, cell extracts made from pulmonary alveolar macrophages derived from five healthy male volunteers were able to reduce an average of 4.8 µg Cr(VI)/10 ^{6} cells or 14.4 µg Cr(VI)/mg protein (Ex. 35–83). Postmitochondrial (S12) preparations of human lung cells (peripheral lung parenchyma and bronchial preparations) were also able to reduce Cr(VI) to Cr(III) (De Flora *et al.* 1984 as cited in Ex. 35–41). As discussed earlier, Cr(VI) is also reduced to Cr(III) in the gastric environment by the gastric juice (Ex. 35–85) and ascorbate after oral exposure (Ex. 35–82).

7. Elimination of Cr(VI) From the Body

Excretion of chromium from Cr(VI) compounds is predominantly in the urine, although there is some biliary excretion into the feces. In both urine and feces, the chromium is present as low molecular weight Cr(III) complexes. Absorbed chromium is excreted from the body in a rapid phase representing clearance from the blood and at least two slower phases representing clearance from tissues. Urinary excretion accounts for over 50% of eliminated chromium (Ex. 35–41). Although chromium is excreted in urine and feces, the intestine plays only a minor part in chromium elimination, representing only about 5% of elimination from the blood (Ex. 19–1). Normal urinary levels of chromium in humans have been reported to range from 0.24–1.8 µg/L with a median level of 0.4 µg/L (Ex. 35–79). Humans exposed to 0.05–1.7 mg Cr(III)/m ^{3} as chromium sulfate and 0.01–0.1 mg Cr(VI)/m ^{3} as potassium dichromate (8-hour time-weighted average) had urinary excretion levels from 0.0247 to 0.037 mg Cr(III)/L. Workers exposed mainly to Cr(VI) compounds had higher urinary chromium levels than workers exposed primarily to Cr(III) compounds. An analysis of the urine did not detect Cr(VI), indicating that Cr(VI) was rapidly reduced before excretion (Exs. 35–294; 5–48).

A half-life of 15–41 hours has been estimated for chromium in urine for four welders using a linear one-compartment kinetic model (Exs. 35–73; 5–52; 5–53). Limited work on modeling the absorption and deposition of chromium indicates that adipose and muscle tissue retain chromium at a moderate level for about two weeks, while the liver and spleen store chromium for up to 12 months. The estimated half-life for whole body chromium retention is 22 days for Cr(VI) and 92 days for Cr(III) (Ex. 19–1). The half-life of chromium in the human lung is 616 days, which is similar to the half-life in rats (Ex. 7–5).

Elimination of chromium was shown to be very slow in rats exposed to 2.1 mg Cr(VI)/m ^{3} as zinc chromate six hours/day for four days. Urinary levels of chromium remained almost constant for four days after exposure and then decreased (Ex. 35–93). After intratracheal administration of sodium dichromate to rats, peak urinary chromium concentrations were observed at six hours, after which the urinary concentrations declined rapidly (Ex. 35–94). The more prolonged elimination of the less soluble zinc chromate as compared to the more soluble sodium dichromate is consistent with the influence of Cr(VI) solubility on absorption from the respiratory tract discussed earlier.

Information regarding the excretion of chromium in humans after dermal exposure to chromium or its compounds is limited. Fourteen days after application of a salve containing potassium chromate, which resulted in skin necrosis and sloughing at the application site, chromium was found at 8 mg/L in the urine and 0.61 mg/100 g in the feces of one individual (Brieger 1920 as cited in Ex. 19–1). A slight increase over background levels of urinary chromium was observed in four subjects submersed in a tub of chlorinated water containing 22 mg Cr(VI)/L as potassium dichromate for three hours (Ex. 31–22–6). For three of the four subjects, the increase in urinary chromium excretion was less than 1 µg/day over the five-day collection period. Chromium was detected in the urine of guinea pigs after radiolabeled sodium chromate solution was applied to the skin (Ex. 35–81).

8. Physiologically-based Pharmacokinetic Modeling

O'Flaherty developed physiologically-based pharmacokinetic (PBPK) models that simulate absorption, distribution, metabolism, and excretion of Cr(VI) and Cr(III) compounds in humans (Ex. 35–95) and rats (Exs. 35–86; 35–70). The original model (Ex. 35–86) evolved from a similar model for lead, and contained compartments for the lung, GI tract, skin, blood, liver, kidney, bone, well-

perfused tissues, and slowly perfused tissues. The model was refined to include two lung subcompartments for chromium, one of which allowed inhaled chromium to enter the blood and GI tract and the other only allowed chromium to enter the GI tract (Ex. 35–70). Reduction of Cr(VI) to Cr(III) was considered to occur in every tissue compartment except bone.

The model was developed from several data sets in which rats were dosed with Cr(VI) or Cr(III) intravenously, orally or by intratracheal instillation, because different distribution and excretion patterns occur depending on the route of administration. In most cases, the model parameters (*e.g.*, tissue partitioning, absorption, reduction rates) were estimated by fitting model simulations to experimental data. The optimized rat model was validated against the 1978 Langard inhalation study (Ex. 35–93). Chromium blood levels were overpredicted during the four-day inhalation exposure period, but blood levels during the post-exposure period were well predicted by the model. The model-predicted levels of liver chromium were high, but other tissue levels were closely estimated.

A human PBPK model recently developed by O'Flaherty *et al.* is able to predict tissue levels from ingestion of Cr(VI) (Ex. 35–95). The model incorporates differential oral absorption of Cr(VI) and Cr(III), rapid reduction of Cr(VI) to Cr(III) in major body fluids and tissues, and concentration-dependent urinary clearance. The model does not include a physiologic lung compartment, but can be used to estimate an upper limit on pulmonary absorption of inhaled chromium. The model was calibrated against blood and urine chromium concentration data from a group of controlled studies in which adult human volunteers drank solutions of soluble Cr(III) or Cr(VI).

PBPK models are increasingly used in risk assessments, primarily to predict the concentration of a potentially toxic chemical that will be delivered to any given target tissue following various combinations of route, dose level, and test species. Further development of the respiratory tract portion of the model, specific Cr(VI) rate data on extracellular reduction and uptake into lung cells, and more precise understanding of critical pathways inside target cells would improve the model value for risk assessment purposes.

9. Summary

Based on the studies presented above, evidence exists in the literature that shows Cr(VI) can be systemically absorbed by the respiratory tract. The absorption of inhaled chromium compounds depends on a number of factors, including physical and chemical properties of the particles (oxidation state, size, and solubility), the reduction capacity of the ELF and alveolar macrophages and clearance by the mucocliary escalator and phagocytosis. Soluble Cr(VI) compounds enter the bloodstream more readily than highly insoluble Cr(VI) compounds. However, insoluble compounds may have longer residence time in lung. Absorption of Cr(VI) can also take place after oral and dermal exposure, particularly if the exposures are high.

The chromate $(CrO_4)^{2-}$ enters cells via facilitated diffusion through non-specific anion channels (similar to phosphate and sulfate anions). Following absorption of Cr(VI) compounds from various exposure routes, chromium is taken up by the blood cells and is widely distributed in tissues as Cr(VI). Inside blood cells and tissues, Cr(VI) is rapidly reduced to lower oxidation states and bound to macromolecules which may result in genotoxic or cytotoxic effects. However, in the blood a substantial proportion of Cr(VI) is taken up into erythrocytes, where it is reduced to Cr(III) and becomes bound to hemoglobin and other proteins.

Inhaled Cr(VI) is reduced to Cr(III) *in vivo* by a variety of reducing agents. Ascorbate and glutathione in the ELF and macrophages have been shown to reduce Cr(VI) to Cr(III) in the lungs. After oral exposure, gastric juices are also responsible for reducing Cr(VI) to Cr(III). This serves to limit the amount of Cr(VI) systemically absorbed.

Absorbed chromium is excreted from the body in a rapid phase representing clearance from the blood and at least two slower phases representing clearance from tissues. Urinary excretion is the primary route of elimination, accounting for over 50% of eliminated chromium. Although chromium is excreted in urine and feces, the intestine plays only a minor part in chromium elimination representing only about 5% of elimination from the blood.

B. Carcinogenic Effects

There has been extensive study on the potential for Cr(VI) to cause carcinogenic effects, particularly cancer of the lung. OSHA reviewed epidemiologic data from several industry sectors including chromate production, chromate pigment production, chromium plating, stainless steel welding, and ferrochromium production. Supporting evidence from animal studies and mechanistic considerations are also evaluated in this section.

1. Evidence from Chromate Production Workers

The epidemiologic literature of workers in the chromate production industry represents the earliest and best-documented relationship between exposure to chromium and lung cancer. The earliest study of chromate production workers in the United States was reported by Machle and Gregorius in 1948 (Ex.7–2). In the United States, two chromate production plants, one in Baltimore, Maryland and one in Painesville, Ohio have been the subject of multiple studies. Both plants were included in the 1948 Machle and Gregorius study and again in the study conducted by the Public Health Service and published in 1953 (Ex. 7–3). Both of these studies reported the results in aggregate. The Baltimore chromate production plant was studied by Hayes *et al.* (Ex. 7–14) and more recently by Gibb *et al.* (Ex. 31–22–11). The chromate production plant in Painesville, Ohio has been followed since the 1950s by Mancuso with his most recent follow-up published in 1997. The most recent study of the Painesville plant was published by Luippold *et al.* (Ex. 31–18–4). The studies by Gibb and Luippold present historical exposure data for the time periods covered by their respective studies. The Gibb exposure data are especially interesting since the industrial hygiene data were collected on a routine basis and not for compliance purposes. These routine air measurements may be more representative of those typically encountered by the exposed workers. In Great Britain, three plants have been studied repeatedly, with reports published between 1952 and 1991. Other studies of cohorts in the United States, Germany, Italy and Japan are also reported. The consistently elevated lung cancer mortality reported in these cohorts and the significant upward trends with duration of employment and cumulative exposure provide some of the strongest evidence that Cr(VI) be regarded as carcinogenic to workers. A summary of selected human epidemiologic studies in chromate production workers is presented in Table VI–1.

TABLE VI–1.—SUMMARY OF SELECTED EPIDEMIOLOGIC STUDIES OF LUNG CANCER IN WORKERS EXPOSED TO HEXAVALENT CHROMIUM—CHROMATE PRODUCTION

Reference/exhibit number	Study population	Reference population	Chromium (VI) exposure	Lung Cancer Risk
Hayes *et al.* (1979, Ex. 7–14) Braver *et al.* (1985, Ex. 7–17).	1803 male workers initially employed 3 or more months 1945–1974 at old and new Baltimore MD production facility; follow-up through 1977.	Baltimore City mortality	Primarily sodium chromate and dichromate production. Avg Cr(VI) of 21 to 413 μg/m^3 and avg duration 1.6 yr to 13 yr depending on subcohort, plant, and year employed.	—O/E of 2.0 (p<0.01) based on 59 lung cancer deaths. —Increased risk with duration of employment.
Gibb *et al.* (2000, Ex. 31–22–11).	2357 male workers initially employed 1950–1974 only at new Baltimore MD production facility; follow-up through 1992.	U.S. mortality	Primarily sodium chromate and dichromate. Mean cumulative Cr(VI) of 0.070 mg/m^3 − yr and work duration of 3.1 yr.	—O/E of 1.86 (p<0.01) based on 71 lung cancer deaths. —Significant upward mortality trend with cumulative Cr(VI) exposure.
Mancuso (1997, Ex. 23) ..... Mancuso (1975, Ex. 7–11). Mancuso and Heuper (1951, Ex. 7–13). Bourne and Yee (1950, Ex. 7–98).	332 male workers employed at Painesville OH facility 1931–1937; follow-up through 1993.	Mortality rate directly calculated using the distribution of person years by age group for the entire exposed population as the standard.	Primarily sodium chromate and dichromate production with some calcium chromate as a result of using high lime process. Most cumulative soluble Cr(VI) between 0.25 and 4.0 mg/m^3 − yr based on 1949 survey.	O/E not calculated but significant increase in age-adjusted lung cancer death rate with cumulative chromium exposure based on 66 deaths.
Luippold *et al.* (2003, Ex. 31–18–4).	492 male workers employed one year between 1940 and 1972 at Painesville OH facility; follow-up through 1997.	U.S. and Ohio Mortality Rates	Primarily sodium chromate and dichromate production with minor calcium chromate. Mean cumulative soluble Cr(VI) of 1.58 mg/m^3 − yr.	—O/E of 2.41(p<0.01) based on Ohio rates and 51 deaths. —Significant upward mortality trend with cumulative Cr(VI) exposure
Davies *et al.* (1991, Ex. 7–99) Alderson *et al.* (1981, Ex. 7–22). Bistrup and Case (1956, Ex. 7–20).	2298 male chromate production workers employed for one year between 1950 and 1976 at three different UK plants; follow-up through 1989.	Cancer mortality of England, Wales and Scotland and unexposed local workers.	Principally sodium chromate and dichromate production with some calcium chromate before switch from high lime to no lime process. Avg soluble Cr(VI) in early 1950s from 2 to 880 μg/m^3 depending on job.	—O/E of 1.97 (p<0.01) pre-process change based on 175 deaths. —SMR of 1.02 (NS) post-process change based on 14 deaths. —Increased risk for high exposed compared with less exposed.
Korallus *et al.* (1993, Ex. 7–91).. Korallus *et al.* (1982, Ex. 7–26).	1417 chromate production workers employed for one year between 1948 and 1987 at two different German plants; follow-up through 1988.	Mortality rates for North Rhine-Westphalia region of Germany where plants located.	Principally sodium chromate and dichromate production with some calcium chromate before switch from high lime to no lime process. Annual mean Cr(VI) between 6.2 and 38 μg/m^3 after 1977. Cr(VI) exposure not reported before 1977.	—O/E of 2.27 (p<0.01) pre-process change based on 66 deaths. —O/E of 1.25 (NS) post-process change based on 9 deaths.

Observed/Expected (O/E)
Relative Risk (RR)
Not Statistically Significant (NS)
Odds Ratio (OR)

The basic hexavalent chromate production process involves milling and mixing trivalent chromite ore with soda ash, sometimes in the presence of lime (Exs. 7–103; 35–61). The mixture is ''roasted'' at a high temperature, which oxidizes much of the chromite to hexavalent sodium chromate. Depending on the lime content used in the process, the roast also contains other chromate species, especially calcium chromate under high lime conditions. The highly water-soluble sodium chromate is water-extracted from the water-insoluble trivalent chromite and the less water-soluble chromates (*e.g.*, calcium chromate) in the ''leaching'' process. The sodium chromate leachate is reacted with sulfuric acid and sodium bisulfate to form sodium dichromate. The sodium dichromate is prepared and packaged as a crystalline powder to be sold as final product or sometimes used as the starting material to make other chromates such as chromic acid and potassium dichromate.

a. *Cohort Studies of the Baltimore Facility.* The Hayes *et al.* study of the Baltimore, Maryland chromate production plant was designed to determine whether changes in the industrial process at one chromium chemical production facility were associated with a decreased risk of cancer, particularly cancer of the respiratory system (Ex. 7–14). Four thousand two hundred and seventeen (4,217) employees were identified as newly employed between January 1, 1945 and December 31, 1974. Excluded from this initial enumeration were employees who: (1) were working as of 1945, but had been hired prior to 1945 and (2) had been hired since 1945 but who had previously been employed at the plant. Excluded from the final cohort were those employed less than 90 days; women; those with unknown length of employment; those with no work history; and those of unknown age. The final cohort included 2,101 employees (1,803 hourly and 298 salaried).

Hayes divided the production process into three departments: (1) The mill and roast or ''dry end'' department which consists of grinding, roasting and leaching processes; (2) the bichromate department which consists of the acidification and crystallization processes; and (3) the special products department which produces secondary products including chromic acid. The bichromate and special products departments are referred to as the ''wet end''.

The construction of a new mill and roast and bichromate plant that opened during 1950 and 1951 and a new chromic acid and special products plant that opened in 1960 were cited by Hayes as ''notable production changes'' (Ex. 7–

14). The new facilities were designed to ''obtain improvements in process technique and in environmental control of exposure to chromium bearing dusts * * *'' (Ex. 7–14).

Plant-related work and health histories were abstracted for each employee from plant records. Each job on the employee's work history was characterized according to whether the job exposure occurred in (1) a newly constructed facility, (2) an old facility, or (3) could not be classified as having occurred in the new or the old facility. Those who ever worked in an old facility or whose work location(s) could not be distinguished based upon job title were considered as having a high or questionable exposure. Only those who worked exclusively in the new facility were defined for study purposes as ''low exposure''. Data on cigarette smoking was abstracted from plant records, but was not utilized in any analyses since the investigators thought it ''not to be of sufficient quality to allow analysis.''

One thousand one hundred and sixty nine (1,169) cohort members were identified as alive, 494 not individually identified as alive and 438 as deceased. Death certificates could not be located for 35 reported decedents. Deaths were coded to the 8th revision of the *International Classification of Diseases.*

Mortality analysis was limited to the 1,803 hourly employees calculating the standardized mortality ratios (SMRs) for specific causes of death. The SMR is a ratio of the number of deaths observed in the study population to the number that would be expected if that study population had the same specific mortality rate as a standard reference population (*e.g.*, age-, gender-, calendar year adjusted U.S. population). The SMR is typically multiplied by 100, so a SMR greater than 100 represents an elevated mortality in the study cohort relative to the reference group. In the Hayes study, the expected number of deaths was based upon Baltimore, Maryland male mortality rates standardized for age, race and time period. For those where race was unknown, the expected numbers were derived from mortality rates for whites. Cancer of the trachea, bronchus and lung accounted for 69% of the 86 cancer deaths identified and was statistically significantly elevated (O = 59; E = 29.16; SMR = 202; 95% CI: 155–263).

Analysis of lung cancer deaths among hourly workers by year of initial employment (1945–1949; 1950–1959 and 1960–1974), exposure category (low exposure or questionable/high exposure) and duration of employment (short term defined as 90 days–2 years; long term defined as 3 years +) was also conducted. For those workers characterized as having questionable/high exposure, the SMRs were significantly elevated for the 1945–1949 and the 1950–1959 hire periods and for both short- and long-term workers (not statistically significant for the short-term workers initially hired 1945–1949). For those characterized as low exposure, there was an elevated SMR for the long-term workers hired between 1950 and 1959, but based only on three deaths (not statistically significant). No lung cancer cases were observed for workers hired 1960–1974.

Case-control analyses of (1) a history of ever having been employed in selected jobs or combinations of jobs or (2) a history of specified morbid conditions and combinations of conditions reported on plant medical records were conducted. Cases were defined as decedents (both hourly and salaried were included in the analyses) whose underlying or contributing cause of death was lung cancer. Controls were defined as deaths from causes other than malignant or benign tumors. Cases and controls were matched on race (white/non-white), year of initial employment (+/−3 years), age at time of initial employment (+/−5 years) and total duration of employment (90 days–2 years; 3–4 years and 5 years +). An odds ratio (OR) was determined where the ratio is the odds of employment in a job involving Cr(VI) exposure for the cases relative to the controls.

Based upon matched pairs, analysis by job position showed significantly elevated odds ratios for special products (OR = 2.6) and bichromate and special products (OR = 3.3). The relative risk for bichromate alone was also elevated (OR = 2.1, not statistically significant).

The possible association of lung cancer and three health conditions (skin ulcers, nasal perforation and dermatitis) as recorded in the plant medical records was also assessed. Of the three medical conditions, only the odds ratio for dermatitis was statistically significant (OR = 3.0). When various combinations of the three conditions were examined, the odds ratio for having all three conditions was statistically significantly elevated (OR = 6.0).

Braver *et al.* used data from the Hayes study discussed above and the results of 555 air samples taken during the period 1945–1950 by the Baltimore City Health Department, the U.S. Public Health Service, and the companies that owned the plant, in an attempt to examine the relationship between exposure to Cr(VI) and the occurrence of lung cancer (Ex. 7–17). According to the authors, methods for determining the air concentrations of Cr(VI) have changed since the industrial hygiene data were collected at the Baltimore plant between 1945 and 1959. The authors asked the National Institute for Occupational Safety and Health (NIOSH) and the Occupational Safety and Health Administration (OSHA) to review the available documents on the methods of collecting air samples, stability of Cr(VI) in the sampling media after collection and the methods of analyzing Cr(VI) that were used to collect the samples during that period.

Air samples were collected by both midget impingers and high volume samplers. According to the NIOSH/OSHA review, high volume samplers could have led to a ''significant'' loss of Cr(VI) due to the reduction of Cr(VI) to Cr(III) by glass or cellulose ester filters, acid extraction of the chromate from the filter, or improper storage of samples. The midget impinger was ''less subject'' to loss of Cr(VI) according to the panel since neither filters nor acid extraction from filters was employed. However, if iron was present or if the samples were stored for too long, conversion from Cr(VI) to Cr(III) may have occurred. The midget impinger can only detect water soluble Cr(VI). The authors noted that, according to a 1949 industrial hygiene survey by the U.S. Public Health Service, very little water insoluble Cr(VI) was found at the Baltimore plant. One NIOSH/OSHA panel member characterized midget impinger results as ''reproducible'' and ''accuracy * * * fairly solid unless substantial reducing agents (*e.g.*, iron) are present'' (Ex. 7–17, p. 370). Based upon the panel's recommendations, the authors used the midget impinger results to develop their exposure estimates even though the panel concluded that the midget impinger methods ''tend toward underestimation'' of Cr(VI).

The authors also cite other factors related to the industrial hygiene data that could have potentially influenced the accuracy of their exposure estimates (either overestimating or underestimating the exposure). These include: measurements may have been taken primarily in ''problem'' areas of the plant; the plants may have been cleaned or certain processes shut down prior to industrial hygiene monitoring by outside groups; respirator use; and periodic high exposures (due to infrequent maintenance operations or failure of exposure control equipment) which were not measured and therefore not reflected in the available data.

The authors estimated exposure indices for cohorts rather than for specific individuals using hire period (1945–1949 or 1950–1959) and duration

of exposure, defined as short (at least 90 days but less than three years) and long (three years or more). The usual exposure to Cr(VI) for both the short- and long-term workers hired 1945–1949 was calculated as the average of the mean annual air concentration for 1945–1947 and 1949 (data were missing for 1948). This was estimated to be 413 μg/m^3. The usual exposure to Cr(VI) was estimated to be 218 μg/m^3 for the short and long employees hired between 1950 and 1959 based on air measurements in the older facility in the early 1950s.

Cumulative exposure was calculated as the usual exposure level × average duration. Short-term workers, regardless of length of employment, were assumed to have received 1.6 years of exposure regardless of hire period. For long-term workers, the average length of exposure was 12.3 years. Those hired 1945–1949 were assigned five years at an exposure of 413 μg/m^3 and 7.3 years at an exposure of 218 μg/m^3. For the long-term workers hired 1950–1959, the average length of exposure was estimated to be 13.4 years. The authors estimated that the cumulative exposures at which ''significant increases in lung cancer mortality'' were observed in the Hayes study were 0.35, 0.67, 2.93 and 3.65 μg/m^3-years. The association seen by the authors appears more likely to be the result of duration of employment rather than the magnitude of exposure since the variation in the latter was small.

Gibb *et al.* relied upon the Hayes study to investigate mortality in a second cohort of the Baltimore plant (Ex. 31–22–11). The Hayes cohort was composed of 1,803 hourly and 298 salaried workers newly employed between January 1, 1945 and December 31, 1974. Gibb excluded 734 workers who began work prior to August 1, 1950 and included 990 workers employed after August 1, 1950 who worked less than 90 days, resulting in a cohort of 2,357 males followed for the period August 1, 1950 through December 31, 1992. Fifty-one percent (1,205) of the cohort was white; 36% (848) nonwhite. Race was unknown for 13% (304) of the cohort. The plant closed in 1985.

Deaths were coded according to the 8th revision of the *International Classification of Diseases*. Person years of observation were calculated from the beginning of employment until death or December 31, 1992, whichever came earlier. Smoking data (yes/no) were available for 2,137 (93.3%) of the cohort from company records.

Between 1950 and 1985, approximately 70,000 measurements of airborne Cr(VI) were collected utilizing several different sampling methods. The program of routine air sampling for Cr(VI) was initiated to ''characterize 'typical/usual exposures' of workers'' (Ex. 31–22–11, p.117). Area samples were collected during the earlier time periods, while both area and personal samples were collected starting in 1977. Exposure estimates were derived from the area sampling systems and were adjusted to ''an equivalent personal exposure estimate using job-specific ratios of the mean area and personal sampling exposure estimates for the period 1978–1985 * * *.'' (Ex. 31–22–11, p.117). According to the author, comparison of the area and personal samples showed ''no significant differences'' for about two-thirds of the job titles. For several job titles with a ''significant point source of contamination'' the area sampling methods ''significantly underestimated'' personal exposure estimates and were adjusted ''by the ratio of the two'' (Ex. 31–22–11, p.118).

A job exposure matrix (JEM) was constructed, where air sampling data were available, containing annual average exposure for each job title. Data could not be located for the periods 1950–1956 and 1960–1961. Exposures were modeled for the missing data using the ratio of the measured exposure for a job title to the average of all measured job titles in the same department. For the time periods where ''extensive'' data were missing, a simple straight line interpolation between years with known exposures was employed.

In an attempt to estimate airborne Cr(III) concentrations, 72 composite dust samples were collected at or near the fixed site air monitoring stations about three years after the facility closed. The dust samples were analyzed for Cr(VI) content using ion chromatography. Cr(III) content was determined through inductively coupled plasma spectroscopic analysis of the residue. The Cr(III):Cr(VI) ratio was calculated for each area corresponding to the air sampling zones and the measured Cr(VI) air concentration adjusted based on this ratio. Worker exposures were calculated for each job title and weighted by the fraction of time spent in each air-monitoring zone. The Cr(III):Cr(VI) ratio was derived in this manner for each job title based on the distribution of time spent in exposure zones in 1978. Cr(VI) exposures in the JEM were multiplied by this ratio to estimate Cr(III) exposures.

A total of 855 observed deaths (472 white; 323 nonwhite and 60 race unknown) were reported. SMRs were calculated using U.S. rates for overall mortality. Maryland rates (the state in which the plant was located) were used to analyze lung cancer mortality in order to better account for regional differences in disease fatality.

A statistically significant lung cancer SMR, based on the national rate, was found for whites (O=71; SMR=186; 95% CI: 145–234); nonwhites (O=47; SMR=188; 95% CI: 138–251) and the total cohort (O=122; SMR=180; 95% CI: 149–214). Of the 122 lung cancer cases, 116 were smokers and four were non smokers at the time of hire. Smoking status was unknown for two lung cancer cases. SMRs were not adjusted for smoking.

The ratio of observed to expected lung cancer deaths (O/E) for the entire cohort stratified by race and cumulative exposure quartile were computed. Cumulative exposure was lagged five years (only exposure occurring five years before a given age was counted). The cut point for the quartiles divided the cohort into four equal groups based upon their cumulative exposure at the end of their working history (0–0.00149 mgCrO_3/m^3-yr; 0.0015–0.0089 mgCrO_3/m^3-yr; 0.009–0.0769 mgCrO_3/m^3-yr; and 0.077–5.25 mgCrO_3/m^3-yr). For whites, the relative risk of lung cancer was significantly elevated for the second through fourth exposure quartiles with O/E values of 0.8, 2.1, 2.1 and 1.7 for the four quartiles, respectively. For nonwhites, the O/E values by exposure quartiles were 1.1, 0.9, 1.2 and 2.9, respectively. Only the highest exposure quartile was significantly elevated. For the total cohort, a significant exposure-response trend was observed such that lung cancer mortality increased with increasing cumulative Cr(VI) exposure.

Proportional hazards models were used to assess the relationship between chromium exposure and the risk of lung cancer. The lowest exposure quartile was used as the reference group. The median exposure in each quartile was used as the measure of cumulative Cr(VI) exposure. When smoking status was included in the model, relative lung cancer risks of 1.83, 2.48 and 3.32 for the second, third and fourth exposure quartiles respectively were estimated. Smoking, Cr(III) exposure, and work duration were also significant predictors of lung cancer risk in the model.

The analysis attempted to separate the effects into two multivariate proportionate hazards models (one model incorporated the log of cumulative Cr(VI) exposure, the log of cumulative Cr(III) exposure and smoking; the second incorporated the log of cumulative Cr(VI), work duration and smoking). In either regression model, lung cancer mortality remained significantly associated ($p < .05$) with

cumulative Cr(VI) exposure even after controlling for the combination of smoking and Cr(III) exposure or the combination of smoking and work duration. On the other hand, lung cancer mortality was not significantly associated with cumulative Cr(III) or work duration in the multivariate analysis indicating lung cancer risk was more strongly correlated with cumulative Cr(VI) exposure than the other variables.

Exponent, as part of a larger submission from the Chrome Coalition, submitted comments on the Gibb paper asking that OSHA review methodological issues believed by Exponent to impact upon the usefulness of the Gibb data in a risk assessment analysis. While Exponent states that the Gibb study offers data that ''are substantially better for cancer risk than the Mancuso study* * *'' they believe that further scrutiny of some of the methods and analytical procedures are necessary (Ex. 31–18–15–1, p. 5).

The issues raised by Exponent and the Chrome Coalition (Ex. 31–18–14) concerning the Gibb paper are: selection of the appropriate reference population for compilation of expected numbers for use in the SMR analysis; inclusion of short term workers (<1 year); expansion of the number of exposure groupings to evaluate dose response trends; analyzing dose response by peak JEM exposure levels; analyzing dose-response at exposures above and below the current PEL and calculating smoking-adjusted SMRs for use in dose-response assessments. Exponent obtained the original data from the Gibb study. The data were reanalyzed to address the issues cited above. Exponent's findings are presented in Exhibit 31–18–15–1 and are discussed below.

Exponent suggests that Gibb's use of U.S. and Maryland mortality rates for developing expectations for the SMR analysis was inappropriate and suggested that Baltimore city mortality rates would have been the appropriate standard to select since those mortality rates would more accurately reflect the mortality experience of those who worked at the plant. Exponent reran the SMR analysis to compare the SMR values reported by Gibb (U.S. mortality rates for SMR analysis) with the results of an SMR analysis using Maryland mortality rates and Baltimore mortality rates. Gibb reported a lung cancer SMR of 1.86 (95% CI: 1.45–2.34) for white males based upon 71 lung cancer deaths using U.S. mortality rates. Reanalysis of the data produced a lung cancer SMR of 1.85 (95% CI: 1.44–2.33) for white males based on U.S. mortality rates, roughly the same value obtained by Gibb. When Maryland and Baltimore rates are used, the SMR drops to 1.70 and 1.25 respectively.

Exponent suggested conducting sensitivity analysis that excludes short-term workers (defined as those with one year of employment) since the epidemiologic literature suggests that the mortality of short-term workers is different than long-term workers. Short-term workers in the Gibb study comprise 65% of the cohort and 54% of the lung cancers. The Coalition also suggested that data pertaining to short-term employee's information are of ''questionable usefulness for assessing the increased cancer risk from chronic occupational exposure to Cr(VI)'' (Ex. 31–18–15–1, p. 5).

Lung cancer SMRs were calculated for those who worked <1 year and for those who worked one year or more. Exponent defined short-term workers as those who worked a minimum of one year ''because it is consistent with the inclusion criteria used by others studying chromate chemical production worker cohorts'' (Ex. 31–18–15–1, p. 12). Exponent also suggested that Gibb's breakdown of exposure by quartile was not the most ''appropriate'' way of assessing dose-response since cumulative Cr(VI) exposures remained near zero until the 50th to 60th percentile, ''so there was no real distinction between the first two quartiles * * *'' (Ex. 31–18–15–1, p. 24). They also suggested that combining ''all workers together at the 75th quartile * * * does not properly account for the heterogeneity of exposure in this group'' (Ex. 31–18–15–1, p. 24). The Exponent reanalysis used six cumulative exposure levels of Cr(VI) compared with the four cumulative exposure levels of Cr(VI) in the Gibb analysis. The lower levels of exposure were combined and ''more homogeneous'' categories were developed for the higher exposure levels.

Using these re-groupings and excluding workers with less than one year of employment, Exponent reported that the highest SMRs are seen in the highest exposure group (1.5<5.25 mg CrO_3/m^3-years) for both white and nonwhite, based on either the Maryland or the Baltimore mortality rates. The authors did not find ''that the inclusion of short-term workers had a significant impact on the results, especially if Baltimore rates are used in the SMR calculations'' (Ex. 31–18–15–1, p. 28).

Analysis of length of employment and ''peak'' (i.e., highest recorded mean annual) exposure level to Cr(VI) was conducted. Exponent reported that approximately 50% of the cohort had ''only very low'' peak exposure levels (<07.2 μg CrO_3/m^3 or approximately 3.6 μg/m^3 of Cr(VI)). The ''majority'' of the short-term workers had peak exposures of <100 μg CrO_3/m^3. There were five peak Cr(VI) exposure levels (<7.2 μg CrO_3/m^3; 7.2<19.3 μg CrO_3/m^3; 19.3<48.0 μg CrO_3/m^3; 48.0<105 μg CrO_3/m^3; 105<182 μg CrO_3/m^3; and 182<806 μg CrO_3/m^3) included in the analyses. Overall, the lung cancer SMRs for the entire cohort grouped according to the six ''peak'' exposure categories were slightly higher using Maryland reference rates compared to Baltimore reference rates.

The Exponent analysis of workers who were ever exposed above the current PEL versus those never exposed above the current PEL produced slightly higher SMRs for those ever exposed, with the SMRs higher using the Maryland standard rather than the Baltimore standard. The only statistically significant result was for all lung cancer deaths combined.

Assessment was made of the potential impact of smoking on the lung cancer SMRs since Gibb did not adjust the SMRs for smoking. Exponent stated that the smoking-adjusted SMRs are more appropriate for use in the risk assessment than the unadjusted SMRs. It should be noted that smoking adjusted SMRs could not be calculated using Baltimore reference rates. As noted by the authors, the smoking adjusted SMRs produced using Maryland reference rates are, by exposure, ''reasonably consistent with the Baltimore-referenced SMRs'' (Ex. 31–18–15–1, p. 41).

Gibb *et al.* included workers regardless of duration of employment, and the cohort was heavily weighted by those individuals who worked less than 90 days. In an attempt to clarify this issue, Exponent produced analyses of short-term workers, particularly with respect to exposures. Exponent redefined short-term workers as those who worked less than one year, to be consistent with the definition used in other studies of chromate producers. OSHA finds this reanalysis excluding short-term workers to be useful. It suggests that including cohort workers employed less than one year did not substantively alter the conclusions of Gibb *et al.* with regard to the association between Cr(VI) exposure and lung cancer mortality. It should be noted that in the Hayes study of the Baltimore plant, the cohort is defined as anyone who worked 90 days or more.

Hayes *et al.* used Baltimore mortality rates while Gibb *et al.* used U.S. mortality rates to calculate expectations for overall SMRs. To calculate

expectations for the analysis of lung cancer mortality and exposure, Gibb *et al.* used Maryland state mortality rates. The SMR analyses provided by Exponent using both Maryland and Baltimore rates are useful. The data showed that using Baltimore rates raised the expected number lung cancer deaths and, thus, lowered the SMRs. However, there remained a statistically significant increase in lung cancer risk among the exposed workers and a significant upward trend with cumulative Cr(VI) exposure. The comparison group should be as similar as possible with respect to all other factors that may be related to the disease except the determinant under study. Since the largest portion of the cohort (45%) died in the city of Baltimore, and even those whose deaths occurred outside of Baltimore (16%) most likely lived in proximity to the city, the use of Baltimore mortality rates as an external reference population is preferable.

Gibb's selection of the cut points for the exposure quartiles is accomplished by dividing the workers in the cohort into four equal groups based on their cumulative exposure at the end of their working history. Using the same method but excluding the short-term workers would have resulted in slightly different cumulative exposure quartiles. Exponent expressed a preference for a six-tiered exposure grouping. The impact of using different exposure groupings is further discussed in preamble section VII.C of the preliminary quantitative risk assessment.

The exposure matrix of Gibb *et al.* does utilize a unique set of industrial hygiene data. Over 70,000 samples taken to characterize the ''typical/usual'' working environment is more extensive industrial hygiene data then is commonly available for most exposure assessments. However, there are several unresolved issues regarding the exposure assessment, including the impact of the different industrial hygiene sampling techniques used over the sampling time frame, how the use of different sampling techniques was taken into account in developing the exposure assessment and the use of area vs. personal samples.

Exponent and the Chrome Coalition also suggested that the SMRs should have been adjusted for smoking. According to Exponent, smoking adjusted SMRs based upon the Maryland mortality rates produced SMRs similar to the SMRs obtained using Baltimore mortality rates (Ex. 31–18–15–1). The accuracy of the smoking data is still questionable since it represents information obtained at the time of hire. Hayes abstracted the smoking data from the plant medical records, but ''found it not to be of sufficient quality to allow analysis.'' One advantage to using the Baltimore mortality data may be to better control for the potential confounding of smoking.

Despite the potential methodological limitations of the Gibb study, this is one of the better cohort mortality studies of workers in the chromium production industry. The quality of the available industrial hygiene data and its characterization as ''typical/usual'' makes the Gibb study useful for risk assessment.

b. *Cohort Studies of the Painesville Facility.* The Ohio Department of Health conducted epidemiological and environmental studies at a plant in Painesville that manufactured sodium bichromate from chromite ore. Mancuso and Hueper (Ex. 7–12) reported an excess of respiratory cancer among chromate workers when compared to the county in which the plant was located. Among the 33 deaths in males who had worked at the plant for a minimum of one year, 18.2% were from respiratory cancer. In contrast, the expected frequency of respiratory cancer among males in the county in which the plant was located was 1.2%. Although the authors did not include a formal statistical comparison, the lung cancer mortality rate among the exposed workers would be significantly greater than the county rate.

Mancuso (Ex. 7–11) updated his 1951 study of 332 chromate production workers employed during the period 1931–1937. Age adjusted mortality rates were calculated by the direct method using the distribution of person years by age group for the total chromate population as the standard. Vital status follow-up through 1974 found 173 deaths. Of the 66 cancer deaths, 41 (62.1%) were lung cancers. A cluster of lung cancer deaths was observed in workers with 27–36 years since first employment.

Mancuso used industrial hygiene data collected in 1949 to calculate weighted average exposures to water-soluble (presumed to be Cr(VI)), insoluble (presumed to be principally Cr(III)) and total chromium (Ex. 7–98). The age-adjusted lung cancer death rate increased from 144.6 (based upon two deaths) to 649.6 (based upon 14 deaths) per 100,000 in five exposure categories ranging from a low of 0.25–0.49 to a high of 4.0+ mg/m^3 – years for the insoluble Cr(III) exposures. For exposure to soluble Cr(VI), the age adjusted lung cancer rates ranged from 80.2 (based upon three deaths) to 998.7 (based upon 12 deaths) in five exposure categories ranging from <0.25 to 2.0+ mg/m^3 – years. For total chromium, the age-adjusted death rates ranged from 225.7 (based upon three deaths) to 741.5 (based upon 16 deaths) for exposures ranging from 0.50–0.99 mg/m^3 – years to 6.0+ mg/m^3 – years.

Age-adjusted lung cancer death rates also were calculated by classifying workers by the levels of insoluble Cr(III) and total chromium exposure. From the data presented, it appears that for a fixed level of insoluble Cr(III), the lung cancer risk appears to increase as the total chromium increases (Ex. 7–11).

Mancuso (Ex. 23) updated the 1975 study. As of December 31, 1993, 283 (85%) cohort members had died and 49 could not be found. Of the 102 cancer deaths, 66 were lung cancers. The age-adjusted lung cancer death rate per 100,000 ranged from 187.9 (based upon four deaths) to 1,254.1 (based upon 15 deaths) for insoluble Cr(III) exposure categories ranging from 0.25–0.49 to 4.00–5.00 mg/m^3 years. For the highest exposure to insoluble Cr(III) (6.00+ mg/m^3 years) the age-adjusted lung cancer death rate per 100,000 fell slightly to 1,045.5 based upon seven deaths.

The age-adjusted lung cancer death rate per 100,000 ranged from 99.7 (based upon five deaths) to 2,848.3 (based upon two deaths) for soluble Cr(VI) exposure categories ranging from <0.25 to 4.00+ mg/m^3 years. For total chromium, the age-adjusted lung cancer death rate per 100,000 ranged from 64.7 (based upon two deaths) to 1,106.7 (based upon 21 deaths) for exposure categories ranging from <0.50 to 6.00+ mg/m^3 years.

To investigate whether the increase in the lung cancer death rate was due to one form of chromium compound (presumed insoluble Cr(III) or soluble Cr(VI)), age-adjusted lung cancer mortality rates were calculated by classifying workers by the levels of exposure to insoluble Cr(III) and total chromium. For a fixed level of insoluble Cr(III), the lung cancer rate appears to increase as the total chromium increases for each of the six total chromium exposure categories, except for the 1.00–1.99 mg/m^3 – years category. For the fixed exposure categories for total chromium, increasing exposures to levels of insoluble Cr(III) showed an increased age-adjusted death rate from lung cancer in three of the six total chromium exposure categories.

For a fixed level of soluble Cr(VI), the lung cancer death rate increased as total chromium categories of exposure increased for three of the six gradients of soluble Cr(VI). For the fixed exposure categories of total chromium, the increasing exposure to specific levels of

soluble Cr(VI) led to an increase in two of the six total chromium exposure categories. Mancuso concluded that the relationship of lung cancer is not confined solely to either soluble or insoluble chromium. Unfortunately, it is difficult to attribute these findings specifically to Cr(III) [as insoluble chromium] and Cr(VI) [as soluble chromium] since it is likely that some slightly soluble and insoluble Cr(VI) as well as Cr(III) contributed to the insoluble chromium measurement.

Luippold *et al.* conducted a retrospective cohort study of 493 former employees of the chromate production plant in Painesville, Ohio (Ex. 31–18–4). This Painesville cohort does not overlap with the Mancuso cohort and is defined as employees hired beginning in 1940 who worked for a minimum of one year at Painesville and did not work at any other facility owned by the same company that used or produced Cr(VI). An exception to the last criterion was the inclusion of workers who subsequently were employed at a company plant in North Carolina (number not provided). Four cohort members were identified as female. The cohort was followed for the period January 1, 1941 through December 31, 1997. Thirty-two percent of the cohort worked for 10 or more years.

Information on potential confounders was limited. Smoking status (yes/no) was available for only 35% of the cohort from surveys administered between 1960 and 1965 or from employee medical files. For those employees where smoking data were available, 78% were smokers (responded yes on at least one survey or were identified as smokers from the medical file). Information on race also was limited, the death certificate being the primary source of information.

Results of the vital status follow-up were: 303 deaths; 132 presumed alive and 47 vital status unknown. Deaths were coded to the 9th revision of the *International Classification of Diseases.* Cause of death could not be located for two decedents. For five decedents the cause of death was only available from data collected by Mancuso and was recoded from the 7th to the 9th revision of the ICD. There were no lung cancer deaths among the five recoded deaths.

SMRs were calculated based upon two reference populations: the U.S. (white males) and the state of Ohio (white males). Lung cancer SMRs stratified by year of hire, duration of exposure, time since first employment and cumulative exposure group also were calculated.

Proctor *et al.* analyzed airborne Cr(VI) levels throughout the facility for the years 1943 to 1971 (the plant closed April 1972) from 800 area air sampling measurements from 21 industrial hygiene surveys (Ex. 35–61). A job exposure matrix (JEM) was constructed for 22 exposure areas for each month of plant operation. Gaps in the matrix were completed by computing the arithmetic mean concentration from area sampling data, averaged by exposure area over three time periods (1940–1949; 1950–1959 and 1960–1971) which coincided with process changes at the plant (Ex. 31–18–1).

The production of water-soluble sodium chromate was the primary operation at the Painesville plant. It involved a high lime roasting process that produced a water insoluble Cr(VI) residue (calcium chromate) as byproduct that was transported in open conveyors and likely contributed to worker exposure until the conveyors were covered during plant renovations in 1949. The average airborne *soluble* Cr(VI) from industrial hygiene surveys in 1943 and 1948 was 0.72 mg/m^3 with considerable variability among departments. During these surveys, the authors believe the reported levels may have underestimated total Cr(VI) exposure by 20 percent or less for some workers due to the presence of *insoluble* Cr(VI) dust.

Reductions in Cr(VI) levels over time coincided with improvements in the chromate production process. Industrial hygiene surveys over the period from 1957 to 1964 revealed average Cr(VI) levels of 270 $\mu g/m^3$. Another series of plant renovations in the early 1960s lowered average Cr(VI) levels to 39 $\mu g/m^3$ over the period from 1965 to 1972. The highest Cr(VI) concentrations generally occurred in the shipping, lime and ash, and filtering operations while the locker rooms, laboratory, maintenance shop and outdoor raw liquor storage areas had the lowest Cr(VI) levels.

The average cumulative Cr(VI) exposure (mg/m^3-yrs) for the cohort was 1.58 mg/m^3-yrs and ranged from 0.006 to 27.8 mg/m^3-yrs. For those who died from lung cancer, the average Cr(VI) exposure was 3.28 mg/m^3-yrs and ranged from 0.06 to 27.8 mg/m^3-yrs. According to the authors, 60% of the cohort accumulated an estimated Cr(VI) exposure of 1.00 mg/m^3-yrs or less.

Sixty-three per cent of the study cohort was reported as deceased at the end of the follow-up period (December 31, 1997). There was a statistically significant increase for the all causes of death category based on both the national and Ohio state standard mortality rates (national: O=303; E=225.6; SMR=134; 95% CI: 120–150; state: O=303; E=235; SMR=129; 95% CI: 115–144). Fifty-three of the 90 cancer deaths were cancers of the respiratory system with 51 coded as lung cancer. The SMR for lung cancer is statistically significant using both reference populations (national O=51; E=19; SMR 268; 95% CI: 200–352; state O=51; E=21.2; SMR 241; 95% CI: 180–317).

SMRs also were calculated by year of hire, duration of employment, time since first employment and cumulative Cr(VI) exposure, $mg/m^3-years$. The highest lung cancer SMRs were for those hired during the earliest time periods. For the period 1940–1949, the lung cancer SMR was 326 (O=30; E=9.2; 95% CI: 220–465); for 1950–1959, the lung cancer SMR was 275 (O=15; E=5.5; 95% CI: 154–454). For the period 1960–1971, the lung cancer SMR was just under 100 based upon six deaths with 6.5 expected.

Lung cancer SMRs based upon duration of employment (years) increased as duration of employment increased. For those with one to four years of employment, the lung cancer SMR was 137 based upon nine deaths (E=6.6; 95% CI: 62–260); for five to nine years of employment, the lung cancer SMR was 160 (O=8; E=5.0; 95% CI: 69–314). For those with 10–19 years of employment, the lung cancer SMR was 169 (O=7; E=4.1; 95% CI: 68–349) and for those with 20 or more years of employment, the lung cancer SMR was 497 (O=27; E=5.4; 95% CI: 328–723).

Analyses of cumulative Cr(VI) exposure found the lung cancer SMR (based upon the Ohio standard) in the highest exposure group (2.70–27.80 mg/m^3-yrs) was 463 (O=20; E=4.3; 95% CI: 183–398). In the 1.05–2.69 mg/m^3-yrs cumulative exposure group, the lung cancer SMR was 365 based upon 16 deaths (E=4.4; 95% CI: 208–592). For the cumulative exposure groups 0.49–1.04, 0.20–0.48 and 0.00–0.19, the lung cancer SMRs were 91 (O=4; E=4.4; 95% CI: 25–234; 184 (O=8; E=4.4; 95% CI: 79–362) and 67 (O=3; E=4.5; 95% CI: 14–196). A test for trend showed a strong relationship between lung cancer mortality and cumulative Cr(VI) exposure (p=0.00002). The authors claim that the SMRs are also consistent with a threshold effect since there was no statistically significant trend for excess lung cancer mortality with cumulative Cr(VI) exposures less than about 1 mg/m^3-yrs. The issue of whether the cumulative Cr(VI) exposure-lung cancer response is best represented by a threshold effect is discussed further in preamble section VII on the preliminary quantitative risk assessment.

The Painesville cohort is small (482 employees). Excluded from the cohort were six employees who worked at other chromate plants after Painesville closed. However, exceptions were made for employees who subsequently worked at the company's North Carolina plant (number not provided) because exposure data were available from the North Carolina plant. Subsequent exposure to Cr(VI) by other terminated employees is unknown and not taken into account by the investigators. Therefore, the extent of the bias introduced is unknown.

The 10% lost to follow-up (47 employees) in a cohort of this size is striking. Four of the forty-seven had ''substantial'' follow-up that ended in 1997 just before the end date of the study. For the remaining 43, most were lost in the 1950s and 1960s (most is not defined). Since person-years are truncated at the time individuals are lost to follow up, the potential implication of lost person years could impact the width of the confidence intervals.

The authors used U.S. and Ohio mortality rates for the standards to compute the expectations for the SMRs, stating that the use of Ohio rates minimizes bias that could occur from regional differences in mortality. It is unclear why county rates were not used to address the differences in regional mortality.

c. *Other Cohort Studies.* The first study of cancer of the respiratory system in the U.S. chromate producing industry was reported by Machle and Gregorius (Ex. 7–2). The study involved a total of 11,000 person-years of observation between 1933 and 1947. There were 193 deaths; 42 were due to cancer of the respiratory system. The proportion of respiratory cancer deaths among chromate workers was compared with proportions of respiratory cancer deaths among Metropolitan Life Insurance industrial policyholders. A non-significant excess respiratory cancer among chromate production workers was found. No attempt was made to control for confounding factors (*e.g.*, age). While some exposure data are presented, the authors state that one cannot associate tumor rates with tasks (and hence specific exposures) because of ''shifting of personnel'' and the lack of work history records.

Baetjer reported the results of a case-control study based upon records of two Baltimore hospitals (Ex. 7–7). A history of working with chromates was determined from these hospital records and the proportion of lung cancer cases determined to have been exposed to chromates was compared with the proportion of controls exposed. Of the lung cancer cases, 3.4% had worked in a chromate manufacturing plant, while none of the controls had such a history recorded in the medical record. The results were statistically significant and Baetjer concluded that the data confirmed the conclusions reached by Machle and Gregorius that ''the number of deaths due to cancer of the lung and bronchi is greater in the chromate-producing industry than would normally be expected'' (Ex. 7–7, p. 516).

As a part of a larger study carried out by the U.S. Public Health Service, the morbidity and mortality of male workers in seven U.S. chromate manufacturing plants during the period 1940–1950 was reported (Exs. 7–1; 7–3). Nearly 29 times as many deaths from respiratory cancer (excluding larynx) were found among workers in the chromate industry when compared to mortality rates for the total U.S. for the period 1940–1948. The lung cancer risk was higher at the younger ages (a 40-fold risk at ages 15–45; a 30-fold risk at ages 45–54 and a 20-fold risk at ages 55–74). Analysis of respiratory cancer deaths (excluding larynx) by race showed an observed to expected ratio of 14.29 for white males and 80 for nonwhite males.

Taylor conducted a mortality study in a cohort of 1,212 chromate workers followed over a 24 year (1937–1960) period (Ex. 7–5). The workers were from three chromate plants that included approximately 70% of the total population of U.S. chromate workers in 1937. In addition, the plants had been in continuous operation for the study period (January 1, 1937 to December 31, 1960). The cohort was followed utilizing records of Old Age and Survivors Disability Insurance (OASDI). Results were reported both in terms of SMRs and conditional probabilities of survival to various ages comparing the mortality experience of chromate workers to the U.S. civilian male population. No measures of chromate exposure were reported although results are provided in terms of duration of employment. Taylor concluded that not only was there an excess in mortality from respiratory cancer, but from other causes as well, especially as duration of employment increased.

In a reanalysis of Taylor's data, Enterline excluded those workers born prior to 1989 and analyzed the data by follow-up period using U.S. rates (Ex. 7–4). The SMR for respiratory cancer for all time periods showed a nine-fold excess (O=69 deaths; E=7.3). Respiratory cancer deaths comprised 28% of all deaths. Two of the respiratory cancer deaths were malignant neoplasms of the maxillary sinuses, a number according to Enterline, ''greatly in excess of that expected based on the experience of the U.S. male population.'' Also slightly elevated were cancers of the digestive organs (O=16; E=10.4) and non-malignant respiratory disease (O=13; E=8.9).

Pastides *et al.* conducted a cohort study of workers at a North Carolina chromium chemical production facility (Ex. 7–93). Opened in 1971, this facility is the largest chromium chemical production facility in the United States. Three hundred and ninety eight workers employed for a minimum of one year between September 4, 1971 and December 31, 1989 comprised the study cohort. A self-administered employee questionnaire was administered to collect data concerning medical history, smoking, plant work history, previous employment and exposure to other potential chemical hazards. Personal air monitoring results for Cr(VI) were available from company records for the period February 1974 through April 1989 for 352 of the 398 cohort members. A job matrix utilizing exposure area and calendar year was devised. The exposure means from the matrix were linked to each employee's work history to produce the individual exposure estimates by multiplying the mean Cr(VI) value from the matrix by the duration (time) in a particular exposure area (job). Annual values were summed to estimate total cumulative exposure.

Personal air monitoring indicated that TWA Cr(VI) air concentrations were generally very low. Roughly half the samples were less than 1 $\mu g/m^3$, about 75 percent were below 3 $\mu g/m^3$, and 96 percent were below 25 $\mu g/m^3$. The average age was 42 years and mean duration of employment was 9.5 years. Two thirds of the workers had accumulated less than 0.01mg/m^3-yr cumulative Cr(VI) exposure. SMRs were computed using national, state (not reported) and county mortality rates (eight adjoining North Carolina counties, including the county in which the plant is located). Two of the 17 recorded deaths in the cohort were from lung cancers. The SMRs for lung cancer were 127 (95% CI: 22–398) and 97 (95% CI: 17–306) based on U.S. and North Carolina county mortality rates, respectively. The North Carolina cohort is still relatively young and not enough time has elapsed to reach any conclusions regarding lung cancer risk and Cr(VI) exposure.

A study of four chromate producing facilities in New Jersey was reported by Rosenman (Ex. 35–104). A total of 3,408 individuals were identified from the four facilities over different time periods (plant A from 1951–1954; plant B from

1951–1971; plant C from 1937–1964 and plant D 1937–1954). No Cr(VI) exposure data was collected for this study. Proportionate mortality ratios (PMRs) and proportionate cancer mortality ratios (PCMRs), adjusted by race, age, and calendar year, were calculated for the three companies (plants A and B are owned by one company). Unlike SMRs, PMRs are not based on the expected mortality rates in a standardized population but, instead, merely represent the proportional distribution of deaths in the cohort relative to the general U.S. population. Analyses were done evaluating duration of work and latency from first employment.

Significantly elevated PMRs were seen for lung cancer among white males (170 deaths, PMR=1.95; 95% CI: 1.67–2.27) and black males (54 deaths, PMR=1.88; 95% CI: 1.41–2.45). PMRs were also significantly elevated (regardless of race) for those who worked 1–10, 11–20 and >20 years and consistently higher for white and black workers 11–20 years and >20 years since first hire. The results were less consistent for those with 10 or fewer years since first hire.

Bidstrup and Case reported the mortality experience of 723 workers at three chromate producing factories in Great Britain (Ex. 7–20). Lung cancer mortality was 3.6 times that expected (O=12; E=3.3) for England and Wales. Alderson *et al.* conducted a follow-up of workers from the three plants in the U.K. (Bolton, Rutherglen and Eaglescliffe) originally studied by Bidstrup (Ex. 7–22). Until the late 1950s, all three plants operated a ''high-lime'' process. This process potentially produced significant quantities of calcium chromate as a by-product as well as the intended sodium dichromate. Process changes occurred during the 1940s and 1950s. The major change, according to the author, was the introduction of the ''no-lime'' process, which eliminated unwanted production of calcium chromate. The no-lime process was introduced at Eaglescliffe 1957–1959 and by 1961 all production at the plant was by this process. Rutherglen operated a low-lime process from 1957/1959 until it closed in 1967. Bolton never changed to the low-lime process. The plant closed in 1966. Subjects were eligible for entry into the study if they had received an X-ray examination at work and had been employed for a minimum of one year between 1948 and 1977. Of the 3,898 workers enumerated at the three plants, 2,715 met the cohort entrance criteria, (alive: 1,999; deceased: 602; emigrated: 35; and lost to follow-up: 79). Those lost to follow-up were not included in the analyses. Eaglescliffe contributed the greatest number of subjects to the study (1,418). Rutherglen contributed the largest number of total deaths (369, or 61%). Lung cancer comprised the majority of cancer deaths and was statistically significantly elevated for the entire cohort (O=116; E=47.96; SMR=240; p <0.001). Two deaths from nasal cancer were observed, both from Rutherglen.

SMRs were computed for Eaglescliffe by duration of employment, which was defined, based upon plant process updates (those who only worked before the plant modification, those who worked both before and after the modifications, or those who worked only after the modifications were completed). Of the 179 deaths at the Eaglescliffe plant, 40 are in the pre-change group; 129 in the pre-/post-change and 10 in the post-change. A total of 36 lung cancer deaths occurred at the plant, in the pre-change group O=7; E=2.3; SMR=303; in the pre-/post-change group O=27; E=13; SMR=2.03 and in the post-change group O=2; E=1.07; SMR=187.

In an attempt to address several potential confounders, regression analysis examined the contributions of various risk factors to lung cancer. Duration of employment, duration of follow-up and working before or after plant modification appear to be greater risk factors for lung cancer, while age at entry or estimated degree of chromate exposure had less influence.

Davies updated the work of Alderson, *et al.* concerning lung cancer in the U.K. chromate producing industry (Ex. 7–99). The study cohort included payroll employees who worked a minimum of one year during the period January 1, 1950 and June 30, 1976 at any of the three facilities (Bolton, Eaglescliffe or Rutherglen). Contract employees were excluded unless they later joined the workforce, in which case their contract work was taken into account.

Based upon the date of hire, the workers were assigned to one of three groups. The first, or ''early'' group, consists of workers hired prior to January 1945 who are considered long term workers, but do not comprise a cohort since those who left or died prior to 1950 are excluded. The second group, ''pre-change'' workers, were hired between January 1, 1945 to December 31, 1958 at Rutherglen or to December 31, 1960 at Eaglescliffe. Bolton employees starting from 1945 are also termed pre-change. The cohort of pre-change workers is considered incomplete since those leaving 1946–1949 could not be included and because of gaps in the later records. For those who started after 1953 and for all men staying 5+ years, this subcohort of pre-change workers is considered complete. The third group, ''post-change'' workers, started after the process changes at Eaglescliffe and Rutherglen became fully effective and are considered a ''complete'' cohort. A ''control'' group of workers from a nearby fertilizer facility, who never worked in or near the chromate plant, was assembled.

A total of 2,607 employees met the cohort entrance criteria. As of December 31, 1988, 1,477 were alive, 997 dead, 54 emigrated and 79 could not be traced (total lost to follow-up: 133). SMRs were calculated using the mortality rates for England and Wales and the mortality rates for Scotland. Causes of death were ascertained for all but three decedents and deaths were coded to the revision of the *International Classification of Diseases* in effect at the time of death. Lung cancer in this study is defined as those deaths where the underlying cause of death is coded as 162 (carcinoma of the lung) or 239.1 (lung neoplasms of unspecified nature) in the 9th revision of the ICD. Two deaths fell into the latter category. The authors attempted to adjust the national mortality rates to allow for differences based upon area and social class.

There were 12 lung cancer deaths at Bolton, 117 at Rutherglen, 75 at Eaglescliffe and one among staff for a total of 205 lung cancer deaths. A statistically significant excess of lung cancer deaths (175 deaths) among early and pre-change workers is seen at Rutherglen and Eaglescliffe for both the adjusted and unadjusted SMRs. For Rutherglen, for the early period based upon 68 observed deaths, the adjusted SMR was 230 while the unadjusted SMR was 347 (for both SMRs p<0.001). For the 41 pre-change lung cancer deaths at Rutherglen, the adjusted SMR was 160 while the unadjusted SMR was 242 (for both SMRs p<0.001). At Eaglescliffe, there were 14 lung cancer deaths in the early period resulting in an adjusted SMR of 196 and an unadjusted SMR of 269 (for both SMRs p<0.05). For the pre-change period at Eaglescliffe, the adjusted SMR was 195 and the unadjusted was 267 (p<0.001 for both SMRs). At Bolton there is a non-significant excess among pre-change men. There are no apparent excesses in the post-change groups, the staff groups or in the non-exposed fertilizer group.

There is a highly significant overall excess of nasal cancers with two cases at Eaglescliffe and two cases at Rutherglen (O=4, Eadjusted=0.26; SMR=1538). All four men with nasal

cancer had more than 20 years of exposure to chromates.

Aw reported on two case-control studies conducted at the previously studies Eaglescliffe plant (Ex. 35–245). In 1960, the plant, converted from a ''high-lime'' to a 'no-lime' process, reducing the likelihood of calcium chromate formation. As of March 1996, 2,672 post-change workers had been employed, including 891 office personnel. Of the post-change plant personnel, 56% had been employed for more than one year. Eighteen lung cancer cases were identified among white male post-change workers (13 deceased; five alive). Duration of employment for the cases ranged from 1.5 to 25 years with a mean of 14.4. Sixteen of the lung cancer cases were smokers.

In the first case-control study reported, the 15 lung cancer cases identified up to September 1991 were matched to controls by age and hire date (five controls per case). Cases and controls were compared based upon their job categories within the plant. The results showed that cases were more likely to have worked in the kiln area than the controls. Five of the 15 cases had five or more years in the kiln area where Cr(VI) exposure occurred vs. six of the 75 controls. A second case-control study utilized the 18 lung cancer cases identified in post change workers up to March 1996. Five controls per case were matched by age (+/−5 years), gender and hire date. Both cases and controls had a minimum of one year of employment. A job exposure matrix was being constructed that would allow the investigators to ''estimate exposure to hexavalent chromates for each worker in the study for all the jobs done since the start of employment at the site until 1980.'' Starting in 1970 industrial hygiene sampling was performed to determine exposure for all jobs at the plant. Cr(VI) exposure levels for the period between 1960 and 1969 were being estimated based on the recall of employees regarding past working conditions relative to current conditions from a questionnaire. The author stated that preliminary analysis suggests that the maximum recorded or estimated level of exposure to Cr(VI) for the cases was higher than that of the controls. However, specific values for the estimated Cr(VI) exposures were not reported.

Korallus *et al.* conducted a study of 1,140 active and retired workers with a minimum of one year of employment between January 1, 1948 and March 31, 1979 at two German chromate production plants (Ex. 7–26). Workers employed prior to January 1, 1948 (either active or retired) and still alive at that date were also included in the cohort. The primary source for determining cause of death was medical records. Death certificates were used only when medical records could not be found. Expected deaths were calculated using the male population of North Rhineland-Westphalia. Elevated SMRs for cancer of the respiratory system (50 lung cancers and one laryngeal cancer) were seen at both plants (O=21; E=10.9; SMR=192 and O=30; E=13.4; SMR=224).

Korallus *et al.* reported an update of the study. The cohort definition was expanded to include workers with one year of employment between January 1, 1948 and December 31, 1987 (Ex. 7–91). One thousand four hundred and seventeen workers met the cohort entrance criteria and were followed through December 31, 1988. While death certificates were used, where possible, to obtain cause of death, a majority of the cause of death data was obtained from hospital, surgical and general practitioner reports and autopsies because of Germany's data protection laws. Smoking data for the cohort were incomplete.

Process modifications at the two plants eliminated the high-lime process by January 1, 1958 at one location and January 1, 1964 at the second location. In addition, technical measures were introduced which led to reductions in the workplace air concentrations of chromate dusts. Cohort members were divided into pre- and post-change cohorts, with subcohorts in the pre-change group. SMRs were computed with the expected number of deaths derived from the regional mortality rates (where the plants are located). One plant had 695 workers (279 in the pre-change group and 416 in the post change group). The second plant had 722 workers (460 in the pre-change group and 262 in the post-change group). A total of 489 deaths were ascertained (225 and 264 deaths). Of the cohort members, 6.4% were lost to follow-up.

Lung cancer is defined as deaths coded 162 in the 9th revision of the *International Classification of Diseases.* There were 32 lung cancer deaths at one plant and 43 lung cancer deaths at the second plant. Lung cancer SMRs by date of entry (which differ slightly by plant) show elevated but declining SMRs for each plant, possibly due to lower Cr(VI) exposure as a result of improvements in production process. The lung cancer SMR for those hired before 1948 at Plant 1 is statistically significant (O=13; SMR=225; 95% CI: 122–382). The overall lung cancer SMR for Plant 1 is also statistically significantly elevated based upon 32 deaths (SMR=175; 95% CI: 120–246). At Plant 2, the only lung cancer SMR that is not statistically significant is for those hired after 1963 (based upon 1 death). Lung cancer SMRs for those hired before 1948 (O=23; SMR=344; 95% CI: 224–508) and for those hired between 1948 and 1963 (O=19; SMR=196; 95% CI: 1.24–2.98) are statistically significantly elevated. The overall lung cancer SMR at Plant 2 based upon 43 deaths is 239 (95% CI: 177–317). No nasal cavity neoplasms were found. A statistically significant SMR for stomach cancer was observed at Plant 2 (O=12; SMR=192; 95% CI: 104–324).

DeMarco *et al.* conducted a cohort study of chromate production workers in northern Italy to assess the existence of excess risk of respiratory cancer, specifically lung cancer (Ex. 7–54). The cohort was defined as males who worked for a minimum of one year from 1948 to 1985 and had at least 10 years of follow-up. Five hundred forty workers met the cohort definition. Vital status follow-up, carried out through June 30, 1985, found 427 cohort members alive, 110 dead and three lost to follow-up. Analysis utilizing SMRs based on Italian national rates was conducted. Of the 110 deaths, 42 were cancer deaths. The statistically significant SMR for lung cancer based upon 14 observed deaths with 6.46 expected was 217 (95% CI: 118–363

Exposure estimates were based upon the duration of cumulative exposure and upon a risk score (low, medium, high and not assessed) assigned to the department in which the worker was primarily employed. A committee assigned the scores, based upon knowledge of the production process or on industrial hygiene surveys taken in 1974, 1982 and 1984. The risk score is a surrogate for the workplace concentrations of Cr(VI) in the different plant departments. Since no substantial changes had been made since World War II, the assumption was made that exposures remained relatively stable. Lung cancer SMRs based upon type of exposure increased with level of exposure (Low: O=1; E=1.43; SMR=70; Medium: O=5; E=202; SMR=2.48; High: O=6; E=1.4; SMR=420; Not Assessed: O=2; E=1.6; SMR=126). Only the SMR for those classified as having worked in departments characterized as high exposure was statistically significant at the $p<0.05$ level.

A cohort study of workers at a chromium compounds manufacturing plant in Tokyo, Japan by Satoh *et al.* included males employed between 1918 and 1975 for a minimum of one year and for whom the necessary data were

available (Ex. 7–27). Date and cause of death data were obtained from the death certificate (85%) or from other ''reliable'' written testimony (15%). Of the 1,061 workers identified, 165 were excluded from the study because information was missing. A total of 896 workers met the cohort inclusion criteria and were followed through 1978. The causes of 120 deaths were ascertained. SMRs based on age-cause specific mortality for Japanese males were calculated for four different time periods (1918–1949; 1950–1959; 1960–1969 and 1970–1978) and for the entire follow-up period (1918–1978). An elevated SMR for lung cancer is seen for the entire follow-up period (O=26; E=2.746; SMR=950). A majority of the lung cancer deaths (20) occurred during the 1970–1978 interval.

Results from the many studies of chromate production workers from different countries indicate a relationship between exposure to chromium and malignant respiratory disease. The epidemiologic studies done between 1948 and 1952 by Machle and Gregorius (Ex. 7–2), Mancuso and Hueper (Ex. 7–12) and Brinton, *et al.* (Ex. 7–1) suggest a risk for respiratory cancer among chromate workers between 15 and 29 times expectation. Despite the potential problems with the basis for the calculations of the expectations or the particular statistical methods employed, the magnitude of the difference between observed and expected is powerful enough to overcome these potential biases.

It is worth noting that the magnitude of difference in the relative risks reported in a mortality study among workers in three chromate plants in the U.K. (Ex.7–20) were lower than the relative risks reported for chromate workers in the U.S. during the 1950s and 1960s. The observed difference could be the result of a variety of factors including different working conditions in the two countries, a shorter follow-up period in the British study, the larger lost-to-follow-up in the British study or the different statistical methods employed. While the earlier studies established that there was an excess risk for respiratory cancer from exposure to chromium, they were unable to specify either a specific chromium compound responsible or an exposure level associated with the risk. Later studies were able to use superior methodologies to estimate standardized lung cancer mortality ratios between chromate production cohorts and appropriate reference populations (Exs. 7–14; 7–22; 7–26; 7–99; 7–91). These studies generally found statistically increased lung cancer risk of around two-fold. The studies usually found trends with duration of employment, year of hire, or some production process change that tended to implicate chromium exposure as the causative agent.

The most recent studies were able to use industrial hygiene data to reconstruct historical Cr(VI) exposures and show statistically significant associations between cumulative airborne Cr(VI) and lung cancer mortality (Exs. 23; 31–22–11; Ex. 31–18–4). Gibb *et al.* found the significant association between Cr(VI) and lung cancer was evident in models that accounted for smoking. The exposure'response relationship from these chromate production cohorts provide strong evidence that occupational exposure to Cr(VI) dust can increase cancer in the respiratory tract of workers.

The Davies, Korallus, and Luippold studies examine mortality patterns at chromate producing facilities where one production process modification involved conversion from a high-lime to a low-lime or a lime-free process (Exs. 7–99; 7–91; 31–18–4). In addition to process modification, technical improvements also were implemented that lowered Cr(VI) exposure. One of the plants in the Davies study retained the high-lime process and is not discussed. The lung cancer SMRs for one British plant and both of the German plants declined from early, to pre-change to post change time periods. In the remaining British plants, the lung cancer SMR is basically identical for the early and pre-change period, but does decline in the post-change time period. The lung cancer SMR in the Luippold cohort also declined over time as the amount of lime was reduced in the roasting process. Other modifications at the Painesville plant that reduced airborne Cr(VI) exposure, such as installation of covered conveyors and conversion from batch to continuous process occurred at the same time (Ex. 35–61). It is not clear whether reduced levels of the high-lime byproduct, calcium chromate, or the roasting/leaching end product, sodium dichromate that resulted from the various process changes is the reason for the decrease in lung cancer SMRs in these cohorts. However, it should be noted increased lung cancer risk was experienced by workers at the Baltimore plant (*e.g.*, Hayes and Gibb cohorts) even though early air monitoring studies suggest that a lime-free process was probably used at this facility (Ex. 7–17).

2. Evidence From Chromate Pigment Production Workers

Chromium compounds are used in the manufacture of pigments to produce a wide range of vivid colors. Lead and zinc chromates have historically been the predominant hexavalent chromium pigments, although others such as strontium and barium chromate have also been produced. These chromates vary considerably in their water solubility with lead and barium chromates being the most water insoluble. All of the above chromates are less water-soluble than the highly water-soluble sodium chromate and dichromate that usually serve as the starting material for chromium pigment production. The reaction of sodium chromate or dichromate with the appropriate zinc or lead compound to form the corresponding lead or zinc chromate takes place in solution. The chromate pigment is then precipitated, separated, dried, milled, and packaged. Worker exposures to chromate pigments are greatest during the milling and packaging stages.

There have been a number of cohort studies of chromate pigment production workers from the United States, the United Kingdom, France, Germany, the Netherlands, Norway and Japan. Most of the studies found significantly elevated lung cancers in workers exposed to Cr(VI) pigments over many years when compared against standardized reference populations. In general, the studies of chromate pigment workers lack the historical exposure data found in some of the chromate production cohorts. The consistently higher lung cancers across several worker cohorts exposed to the less water-soluble Cr(VI) compounds complements the lung cancer findings from the studies of workers producing highly water soluble chromates and adds to the further evidence that occupational exposure to Cr(VI) compounds should be regarded as carcinogenic. A summary of selected human epidemiologic studies in chromate production workers is presented in Table VI–2.

TABLE VI–2.—SUMMARY OF SELECTED EPIDEMIOLOGIC STUDIES OF LUNG CANCER IN WORKERS EXPOSED TO HEXAVALENT CHROMIUM—CHROMATE PIGMENT PRODUCTION

Reference/exhibit No.	Study population	Reference population	Chromium (VI) exposure	Lung cancer risk
Langard & Vigander (1983, Ex. 7–36). Langard & Vigander (1975, Ex. 7–33).	133 Norwegian chromium pigment production workers employed between 1948 and 1972; 24 workers with 3+ years exposure to chromate dust; follow up through 1980.	Cancer incidence from Norwegian Cancer Registry 1955–1976.	Lead and zinc chromates with some sodium dichromate as starting material; Cr(VI) levels between 10 and 30 μg/m^3 1975–1980. No reporting <1975.	–O/E of 44 for subcohort of 24 workers based on 6 cancer cases. –5 of 6 cases were exposed primarily to zinc chromate.
Davies (1984, Ex. 7–42) ..... Davies (1979, Ex. 7–41).	1152 British chromate pigment workers from 3 plants with a minimum of 1 year employment between 1930–June, 1975; follow up through 1981.	Mortality of England and Wales	Factory A: chromates—primarily lead; some zinc; minor barium Factory B: mostly lead and zinc chromates; minor strontium. Factory C: lead chromate only No Cr(VI) levels reported.	—O/E of 2.2 (p<0.05) for high exposed in Factory A 1932–1954; 21 deaths. —O/E of 4.4 (p<0.05) for high exposed in Factory B 1948–1967; 11 deaths. —O/E of 1.1 (NS) for exposed Factory C 1946–1967; 7 deaths.
Hayes *et al.* (1989, Ex. 7–46) Sheffet *et al.* (1982, Ex. 7–48).	1,946 male pigment workers from New Jersey facility employed for a minimum of one month between 1940 and 1969; follow up through March, 1982.	U.S. Mortality	–Primarily lead chromate with some zinc chromate. –Cr(VI) levels in later years reported to be >500 μg/m$_3$ for exposed workers.	—O/E of 1.2 (NS) for entire cohort based on 41 deaths. —O/E of 1.5 (p<0.5) for workers employed >10 yr based on 23 deaths. —Upward trend (p<0.01) with duration of exposure.
Equitable Environmental Health (1983, Ex. 2–D–1). Equitable Environmental Health (1976, Ex. 2–D–3)	574 male chromate workers from three plants (West Virginia, New Jersey or Kentucky) with a minimum of 6 months of exposure to lead chromate prior to 1974.	U.S. white male mortality rates	West Virginia: lead chromates Kentucky: chromates—mostly lead, some zinc, minor strontium and barium. —New Jersey; mostly lead and some zinc chromate. —Median Cr(VI) in 1975 reported to equal or exceed 52 μg/m^3	—O/E of 1.30 (NS) for West Virginia plant based on 3 deaths. —O/E of 2.16 (NS) for Kentucky plant based on 3 deaths. —O/E of 2.31 (p<.05) for New Jersey plant based on 9 deaths.
Deschamps *et al.* (1995, 35–234). Haguenoer *et al.* (1981, Ex. 7–44)	294 male pigment workers from French facility employed for a minimum of six months between 1958 and 1987.	Death rates from northern France.	—Mostly lead chromate with some zinc chromate. —Cr(VI) levels in 1981 between 2 and 180 μg/m^3	—O/E of 3.6 (p<0.01) based on 18 deaths. —Upward trend (p<0.01) with duration of exposure.

Observed/Expected (O/E).
Relative Risk (RR).
Not Statistically Significant (NS).
Odds Ratio (OR).

Langard and Vigander updated a cohort study of lung cancer incidence in 133 workers employed by a chromium pigment production company in Norway (Ex. 7–36). The cohort was originally studied by Langard and Norseth (Ex. 7–33). Twenty-four men had more than three years of exposure to chromate dust. From 1948, when the company was founded, until 1951, only lead chromate pigment was produced. From 1951 to 1956, both lead chromate and zinc chromate pigments were produced and from 1956 to the end of the study period in 1972 only zinc chromate was produced. Workers were exposed to chromates both as the pigment and its raw material, sodium dichromate.

The numbers of expected lung cancers in the workers were calculated using the age-adjusted incidence rates for lung cancer in the Norwegian male population for the period 1955–1976. Follow-up using the Norwegian Cancer Registry through December 1980, found the twelve cancers of which seven were lung cancers. Six of the seven lung cancers were observed in the subcohort of 24 workers who had been employed for more than three years before 1973. There was an increased lung cancer incidence in the subcohort based on an observed to expected ratio of 44 (O=6; E=0.135). Except for one case, all lung cancer cases were exposed to zinc chromates and only sporadically to other chromates. Five of the six cases were known to be smokers or ex-smokers. Although the authors did not report any formal statistical comparisons, the extremely high age-adjusted standardized incidence ratio suggests that the results would likely be statistically significant.

Davies reported on a cohort study of English chromate pigment workers at three factories that produced chromate pigments since the 1920s or earlier (Ex. 7–41). Two of the factories produced both zinc and lead chromate. Both products were made in the same sheds and all workers had mixed exposure to both substances. The only product at the third factory was lead chromate.

Cohort members are defined as males with a minimum of one year of employment first hired between 1933 and 1967 at plant A; 1948 and 1967 at plant B and 1946–1961 at plant C. The analysis excludes men who entered employment later than 1967 because of the short follow-up period. Three hundred and ninety-six (396) men from Factory A, 136 men from Factory B and 114 men from Factory C were followed to mid-1977. Ninety-four workers with 3–11 months employment during 1932–1945 at Factory A were also included. Expectations were based upon calendar time period-, gender- and age-specific national cancer death rates for England and Wales. The author adjusted the death rates for each factory for local differences, but the exact methods of adjustment were not explicit.

Exposure to chromates was assigned as high for those in the dry departments where pigments were ground, blended and packed; medium for those in the wet departments where precipitates were washed, pressed and stove dried and in maintenance or cleaning which required time in various departments; or low for those jobs which the author states involved ''slight exposure to chromates such as most laboratory jobs, boiler stoking, painting and bricklaying'' (Ex. 7–41, p. 159). The high and

medium exposure categories were combined for analytical purposes.

For those entering employment from 1932 to 1954 at Factory A, there were 18 lung cancer deaths in the high/medium exposure group, with 8.2 deaths expected. The difference is significant at p<. 01. In the low exposure group, the number of observed and expected lung cancer deaths was equal (two deaths). There were no lung cancer deaths at Factory A for those hired between 1955–1960 and 1961–1967.

For those entering employment between 1948 and 1967 at Factory B, there were seven observed lung cancer deaths in the high/medium exposure group with 1.4 expected which is statistically significant at p<. 001. At Factory C (which manufactured only lead chromate), there was one death in the high/medium exposure group and one death in the low exposure group for those beginning employment between 1946 and 1967.

The author points out that:

> There has been no excess lung cancer mortality amongst workers with chromate exposure rated as ''low'', nor among those exposed only to lead chromate. High and medium exposure-rated workers who in the past had mixed exposure to both lead and zinc chromate have experienced a marked excess of lung cancer deaths, even if employed for as little as one year'' (Ex. 7–41, p. 157).

It is the author's opinion that the results ''suggest that the manufacture of zinc chromate may involve a lung cancer hazard'' (Ex. 7–41, p. 157).

Davies updated the lung cancer mortality at the three British chromate pigment production factories (Ex. 7–42). The follow-up was through December 31, 1981. The cohort was expanded to include all male workers completing one year of service by June 30, 1975 but excluded office workers.

Among workers at Factory A with high and medium exposure, mortality was statistically significantly elevated over the total follow-up period among entrants hired from 1932 to 1945 (O/E=2.22). A similar, but not statistically significant, excess was seen among entrants hired from 1946 to 1954 (O/E=2.23). The results for Factory B showed statistically significantly elevated lung cancer mortality among workers classified with medium exposures entering service during the period from 1948 to 1960 (O/E=3.73) and from 1961 to 1967 (O/E=5.62). There were no lung cancer deaths in the high exposure group in either time period. At Factory C, analysis by entry date (early entrant and the period 1946–1960) produced no meaningful results since the number of deaths was small. When the two periods are combined, the O/E was near unity. The author concluded that in light of the apparent absence of risk at Factory C, ''it seems reasonable to suggest that the hazard affecting workers with mixed exposures at factories A and B * * * is attributable to zinc chromates'' (Ex. 7–42, p. 166).

Davies also studied a subgroup of 57 chromate pigment workers, mostly employed between 1930 and 1945, who suffered clinical lead poisoning (Ex. 7–43). Followed through 1981, there was a statistically significantly elevated SMR for lung cancer based upon four cases (O=4; E=2.8; SMR=145).

Haguenoer studied 251 French zinc and lead chromate pigment workers employed for six months or more between January 1, 1958 and December 31, 1977 (Ex. 7–44). As of December 31, 1977, 50 subjects were identified as deceased. Cause of death was obtained for 30 of the 50 deaths (60%). Lung cancer mortality was significantly elevated based on 11 fatalities (SMR=461; 95% CI: 270–790). The mean time from first employment until detection of cancer was 17 years. The mean duration of employment among cases was 15 years.

The Haguenoer cohort was followed up in a study by Deschamps *et al.* (Ex. 234). Both lead and zinc chromate pigments were produced at the plant until zinc chromate production ceased in 1986. The cohort consisted of 294 male workers employed for at least six months between 1958 and 1987. At the end of the follow-up, 182 cohort members were alive, 16 were lost to follow-up and 96 were dead. Because of French confidentiality rules, the cause of death could not be obtained from the death certificate; instead physicians and hospital records were utilized. Using cause of death data from sources other than death certificates raises the potential for misclassification bias. Cause of death could not be obtained for five decedents. Data on smoking habits was not available for a number of workers and was not used in the analysis.

Since individual work histories were not available, the authors made the assumption that the exposure level was the same for all workers during their employment at the plant. Duration of employment was used as a surrogate for exposure. Industrial hygiene measurements taken in 1981 provide some idea of the exposure levels at the plant. In the filtration department, Cr(VI) levels were between 2 and 3 μg/m^3; in the grinding department between 6 and 165 μg/m^3; in the drying and sacking department between 6 and 178 μg/m^3; and in the sacks marking department more than 2000 μg/m^3.

The expected number of deaths for the SMR analysis was computed from age-adjusted death rates in the northern region of France where the plant was located. There was a significant increase in lung cancer deaths based on 18 fatalities with five expected (SMR=360; 95% CI: 213–568). Using duration of employment as a surrogate for exposure, statistically significant SMRs were seen for the 10–15 years of exposure (O=6, SMR=720, 95% CI: 264–1568), 15–20 years (O=4, SMR=481, 95% CI: 131–1231), and 20+ years (O=6, SMR=377, 95% CI: 1.38–8.21) time intervals. There was a significantly elevated SMR for brain cancer based upon two deaths (SMR=844, 95% CI: 102–3049). There was a non-statistically significant increase for digestive tract cancer (O=9, SMR=130) consisting of three esophageal cancers, two stomach cancers and four colon cancers.

Equitable Environmental Health, Inc., on behalf of the Dry Color Manufacturers Association, undertook a historical prospective mortality study of workers involved in the production of lead chromate (Exs. 2–D–3; 2–D–1). The cohort was defined as male employees who had been exposed to lead chromate for a minimum of six months prior to December 1974 at one of three facilities in West Virginia, Kentucky or New Jersey. The New Jersey facility had a unit where zinc chromate was produced dating back to 1947 (Ex. 2–D–3). Most workers rotated through this unit and were exposed to both lead and zinc chromates. Two men were identified at the New Jersey facility with exposure solely to lead chromate; no one with exposure only to zinc chromate was identified.

Subsequent review of the data found that the Kentucky plant also produced zinc chromates from the late 1930s to early 1964. During the period 1961–1962, zinc chromates accounted for approximately 12% of chromate production at the plant. In addition, strontium chromate and barium chromate also were produced at the plant.

The cohort consisted of 574 male employees from all three plants (Ex. 2–D–1). Eighty-five deaths were identified with follow up through December 1979. Six death certificates were not obtained. SMRs were reported based on U.S. white male death rates. There were 53 deaths from the New Jersey plant including a statistically significant SMR for cancer of the trachea, bronchus and lung based upon nine deaths (E=3.9; SMR=231; 95% CI: 106–438). One lung cancer decedent worked solely in the

production of lead chromates. Three of the lung cancer deaths were black males. In addition, there were six deaths from digestive system cancers, five of which were stomach cancers reported at the New Jersey plant. The SMR for stomach cancer was statistically significantly elevated (O=5; E=0.63; SMR=792; 99% CI: 171–2243). There were 21 deaths from the West Virginia plant, three of which were cancer of the trachea, bronchus and lung (E=2.3; SMR=130; 95% CI: 27–381). There were 11 deaths at the Kentucky plant, two of which were cancer of the trachea, bronchus and lung (E=0.9; SMR=216; 95% CI: 26–780).

Sheffet *et al.* examined the lung cancer mortality among 1,946 male employees in a chromate pigment factory in Newark, New Jersey who were exposed to both lead chromate and zinc chromate pigments (Ex. 7–48). The men worked for a minimum of one month between January 1, 1940 and December 31, 1969. As of March 31, 1979, a total of 321 cohort members were identified as deceased (211 white males and 110 non-white males). Cause of death could not be ascertained for 37 white males and 12 non-white males. The proportion of the cohort lost to follow up was high (15% of white males and 20% of non-white males).

Positions at the plant were classified into three categories according to intensity of exposure: high (continuous exposure to chemical dust), moderate (occasional exposure to chemical dust or to dry or wet pigments) and low (infrequent exposure by janitors or office workers). Positions were also classified by type of chemical exposure: chromates, other inorganic substances, and organics. The authors' state that in almost all positions individuals ''who were exposed to any chemicals were also exposed to hexavalent chromium in the form of airborne lead and zinc chromates (Ex. 7–48, p. 46).'' The proportion of lead chromate to zinc chromate was approximately nine to one. Calculations, based upon air samples during later years, give an estimate for the study period of more than 2000 µg airborne chromium/m^3 for the high exposure category, between 500 and 2000 µg airborne chromium/m^3 and less than 100 µg airborne chromium/m^3 for the low exposure category. Other suspected carcinogens present in the workplace air at much lower levels were nickel sulfate and nickel carbonate.

Because of the large proportion of workers lost to follow-up (15% of white males and 20% of non-white males) and the large numbers of unknown cause of death (21% of white males and 12% of non-white males), the authors calculated three separate mortality expectations based upon race-, gender-, age- and time-specific U.S. mortality ratios. The first expectation was calculated upon the assumption that those lost to follow-up were alive at the end of the study follow-up period. The second expectation was calculated on the assumption that those whose vital status was unknown were lost to follow-up as of their employment termination date. The third expectation was calculated excluding those of unknown vital status from the cohort. Deaths with unknown cause were distributed in the appropriate proportions among known causes of death which served as an adjustment to the observed deaths. The adjusted deaths were used in all of the analyses.

A statistically significant ratio for lung cancer deaths among white males (O/E=1.6) was observed when using the assumption that either the lost to follow-up were assumed lost as of their termination date or were excluded from the cohort (assumptions two and three above). The ratio for lung cancer deaths for non-white males results in an identical O/E of 1.6 for all three of the above scenarios, none of which was statistically significant.

In addition, the authors also conducted Proportionate Mortality Ratio (PMR) and Proportionate Cancer Mortality Ratio (PCMR) analyses. For white males, the lung cancer PMR was 200 and the lung cancer PCMR was 160 based upon 25.5 adjusted observed deaths (21 actual deaths). Both were statistically significantly elevated at the $p<.05$ level. For non-white males, the lung cancer PMR was 200 and the lung cancer PCMR was 150 based upon 11.2 adjusted observed deaths (10 actual deaths). The lung cancer PMR for non-white males was statistically significantly elevated at the $p<.05$ level. Statistically significantly elevated PMRs and PCMRs for stomach cancer in white males were reported (PMR=280; PCMR=230) based upon 6.1 adjusted observed deaths (five actual).

The Sheffet cohort was updated in a study by Hayes *et al.* (Ex. 7–46). The follow up was through December 31, 1982. Workers employed as process operators or in other jobs which involved direct exposure to chromium dusts were classified as having exposure to chromates. Airborne chromium concentrations taken in ''later years'' were estimated to be >500 µg g/m^3 for ''exposed'' jobs and >2000 µg /m^3 for ''highly exposed'' jobs.

The cohort included 1,181 white and 698 non-white males. Of the 453 deaths identified by the end of the follow-up period, 41 were lung cancers. For the entire study group, no statistically significant excess was observed for lung cancer (SMR=116) or for cancer at any other site. Analysis by duration of employment found a statistically significant trend (p=. 04) for lung cancer SMRs (67 for those employed < 1 year; 122 for those employed 1–9 years and 151 for those employed 10+ years).

Analysis of lung cancer deaths by duration of employment in chromate dust associated jobs found no elevation in risk for subjects who never worked in these jobs (SMR=92) or for subjects employed less than one year in these jobs (SMR=93). For those with cumulative employment of 1–9 and 10+ years in jobs with chromate dust exposure, the SMRs were 176 (nine deaths) and 194 (eight deaths) respectively.

Frentzel-Beyme studied the mortality experience of 1,396 men employed for more than six months in one of five factories producing lead and zinc chromate pigments located in Germany and the Netherlands (Ex. 7–45). The observed deaths from the five factories were compared with the expected deaths calculated on the basis of mortality figures for the region in which the plant was located. Additional analysis was conducted on relevant cohorts which included workers with a minimum of 10 years exposure, complete records for the entire staff, and exclusion of foreign nationals. Jobs were assigned into one of three exposure categories: high (drying and milling of the filtered pigment paste), medium (wet processes including precipitation of the pigment, filtering and maintenance, craftsmen and cleaning) and low or trivial exposure (storage, dispatch, laboratory personnel and supervisors).

There were 117 deaths in the entire cohort of which 19 were lung cancer deaths (E=9.3). The lung cancer SMRs in the relevant cohort analyses were elevated at every plant; however, in only one instance was the increased lung cancer SMR statistically significant, based upon three deaths (SMR=386, $p<0.05$). Analysis by type of exposure is not meaningful due to the small number of lung cancer death per plant per exposure classification.

Kano *et al.* conducted a study of five Japanese manufacturers who produced lead chromates, zinc chromate, and/or strontium chromate to assess if there was an excess risk of lung cancer (Ex. 7–118). The cohort consisted of 666 workers employed for a minimum of one year between 1950 and 1975. At the end of 1989, 604 subjects were alive, five lost to follow-up and 57 dead. Three lung cancer deaths were observed

in the cohort with 2.95 expected (SMR=102; 95% CI: 0.21–2.98). Eight stomach cancer deaths were reported with a non-statistically significant SMR of 120.

In response to OSHA's August 2002 Request for Information, the Color Pigment Manufacturers Association suggested that OSHA consider reviewing the Davies (Ex. 7–43), Cooper [Equitable Environmental Health, Inc.] (Ex. 2–D–1) and Kano (Ex. 14–1–B) epidemiologic studies with respect to the health effects of lead chromate color pigments. The Equitable Environmental Health and the Kano *et al.* studies each report three deaths from lung cancer among chromate pigment production workers. The number of lung cancer deaths is too small to be meaningful. Even if there were a sufficient number of deaths for analysis, no quantitative exposure data are provided. In the case of the Davies study, there were seven lung cancer deaths at the one manufacturing facility that made only lead chromate pigments. When analyzed by period (early, 1946–1967) and high/medium and low exposure category, the numbers are too small in any category to be meaningful. Studies of lead and zinc chromate pigment worker cohorts that experienced a greater number of lung cancer deaths (*e.g.*, >10 deaths) consistently found significant elevations in lung cancer risk, particularly those workers with the longest latency and durations of exposure (Exs. 234; 7–46; 7–42).

3. Evidence From Workers in Chromium Plating

Chrome plating is the process of depositing chromium metal onto the surface of an item using a solution of chromic acid. The items to be plated are suspended in a diluted chromic acid bath. A fine chromic acid mist is produced when gaseous bubbles, released by the dissociation of water, rise to the surface of the plating bath and burst. There are two types of chromium electroplating. Decorative or ''bright'' involves depositing a thin (0.5–1 μm) layer of chromium over nickel or nickel-type coatings to provide protective, durable, non-tarnishable surface finishes. Decorative chrome plating is used for automobile and bicycle parts. Hard chromium plating produces a thicker (exceeding 5 μm) coating which makes it resistant and solid where friction is usually greater, such as in crusher propellers and in camshafts for ship engines. Limited air monitoring indicates that Cr(VI) levels are five to ten times higher during hard plating than decorative plating (Ex. 35–116).

There are fewer studies that have examined the lung cancer mortality of chrome platers than of soluble chromate production and chromate pigment production workers. The largest and best described cohort studies investigated chrome plating cohorts in the United Kingdom (Exs. 7–49; 7–57; 271; 35–62). They generally found elevated lung cancer mortality among the chrome platers, especially those engaged in chrome bath work, when compared to various reference populations. The studies of British chrome platers are summarized in Table VI–3.

TABLE VI–3.—SUMMARY OF SELECTED EPIDEMIOLOGIC STUDIES OF LUNG CANCER IN WORKERS EXPOSED TO HEXAVALENT CHROMIUM—CHROMIUM PLATING

Reference/exhibit No.	Study population	Reference population	Chromium (VI) exposure	Lung cancer risk
Sorahan & Harrington (2000, Ex. 35–62). Royle (1975, Ex. 7–49)	920 male platers employed in 54 plants in Yorkshire, UK for a minimum of three months between 1969 and 1972; follow up through 1997.	—Mortality rates for the general population of England and Wales. —Age-, sex-matched comparison group unexposed to CR(VI).	—Chromic acid mist with some nickel and cadmium co-exposure. —Cr(VI) levels in 1970 reported to range from <30 μg/m^3 to >100 μg/m^3.	—O/E of 1.85 (p=0.001) based on 60 deaths and general pop. —O/E of 1.39 (p=0.06) based on unexposed comparison group. —No upward trend w/duration of exposure.
Sorahan *et al.* (1998, Ex. 35–271). Sorahan *et al.* (1987, Ex. 7–57).	1,762 platers employed for a minimum of six months between 1946 and 1975 from a Midlands, UK plant; follow up through 1995.	—Mortality rates for the general population of England and Wales.	—Chromic acid mist with nickel co-exposure. —No reported Cr(VI) exposure levels.	—O/E of 1.6 (p<0.01) for male chrome bath workers based on 40 deaths. —O/E of 0.66 (NS) for other chrome workers based on 9 deaths. —Upward trend (p<0.05) with duration of chrome bath work.

Observed/Expected (O/E).
Relative Risk (RR).
Not Statistically Significant (NS).
Odds Ratio (OR).

Cohort studies of chrome platers in Italy, the United States, and Japan are also discussed in this subsection. Co-exposure to nickel, another suspected carcinogen, during plating operations can complicate evaluation of an association between Cr(VI) and an increased risk of lung cancer in chrome platers. Despite this, the International Agency for Research on Cancer concluded that the epidemiological studies provide sufficient evidence for carcinogenicity of Cr(VI) as encountered in the chromium plating industry; the same conclusion reached for chromate production and chromate pigment production (Exs. 18–1; 35–43). The findings implicate the highly water-soluble chromic acid as an occupational carcinogen. This adds to the weight of evidence that water-soluble (*e.g.*, sodium chromates, chromic acid) and water-insoluble forms (*e.g.*, lead and zinc chromates) of Cr(VI) are able to cause cancer of the lower respiratory tract.

Royle reported on a cohort mortality study of 1,238 chromium platers employed for a minimum of three consecutive months between February 20, 1969 and May 31, 1972 in 54 plating plants in West Riding, Yorkshire, England (Ex. 7–49). A control population was enumerated from other departments of the larger companies where chromium plating was only a portion of the companies' activities and from the former and current employees of two industrial companies in York where information on past workers was available. Controls were matched for gender, age (within two years) and date last known alive. In addition, 229 current workers were matched for smoking habits.

As of May 1974, there were 142 deaths among the platers (130 males and 12 females) and 104 deaths among the controls (96 males and 8 females). Among the male platers, there were 24 deaths from cancer of the lung and pleura compared to 13 deaths in the control group. The difference was not statistically significant. There were eight deaths from gastrointestinal cancer among male platers versus four deaths in the control group. The finding was not statistically significant.

The Royle cohort was updated by Sorahan and Harrington (Ex. 35–62). Chrome plating was the primary activity at all 54 plants, however 49 of the plants used nickel and 18 used cadmium. Also used, but in smaller quantities according to the authors, were zinc, tin, copper, silver, gold, brass or rhodium. Lead was not used at any of the plants. Four plants, including one of the largest, only used chromium. Thirty-six chrome platers reported asbestos exposure versus 93 comparison workers.

Industrial hygiene surveys were carried out at 42 plants during 1969–1970. Area air samples were done at breathing zone height. With the exception of two plants, the chromic acid air levels were less than 30 $\mu g/m^3$. The two exceptions were large plants, and in both the chromic acid levels exceeded 100 $\mu g/m^3$.

The redefined cohort consisted of 1087 platers (920 men and 167 women) from 54 plants employed for a minimum of three months between February 1969 and May 31, 1972 who were alive on May 31, 1972. Mortality data were also available for a comparison group of 1,163 workers (989 men and 174 women) with no chromium exposure. Both groups were followed for vital status through 1997.

The lung cancer SMR for male platers was statistically significant (O=60; E=32.5; SMR=185; 95% CI: 141–238). The lung cancer SMR for the comparison group, while elevated, was not statistically significant (O=47; E=36.9; SMR=127; 95% CI: 94–169). The only statistically significant SMR in the comparison group was for cancer of the pleura (O=7; E=0.57; SMR=1235; 95% CI: 497–2545).

Internal regression analyses were conducted comparing the mortality rates of platers directly with those of the comparison workers. For these analyses, lung cancers mentioned anywhere on the death certificate were considered cases. The redefinition resulted in four additional lung cancer cases in the internal analyses. There was a statistically significant relative risk of 1.44 ($p<0.05$) for lung cancer mortality among chrome platers that was slightly reduced to 1.39 after adjustment for smoking habits and employment status. There was no clear trend between lung cancer mortality and duration of Cr(VI) exposure. However, any positive trend may have been obscured by the lack of information on worker employment post-1972 and the large variation in chromic acid levels among the different plants.

Sorahan reported the experience of a cohort of 2,689 nickel/chromium platers from the Midlands, U.K. employed for a minimum of six months between 1946 and 1975 and followed through December 1983 (Ex. 7–57). There was a statistically significant lung cancer SMR for males (O=63; E=40; SMR=158; $p<0.001$). The lung cancer SMR for women, while elevated (O=9; E=8.1; SMR=111), was not statistically significant. Other statistically significant cancer SMRs for males included: stomach (O=21; E=11.3; SMR=186; $p<0.05$); liver (O=4; E=0.6; SMR=667; $p<0.01$); and nasal cavities (O=2; E=0.2; SMR=1000; $p<0.05$). While there were several elevated SMRs for women, none were statistically significant. There were nine lung cancers and one nasal cancer among the women.

Analysis by type of first employment (i.e., chrome bath workers vs. other chrome work) resulted in a statistically significant SMR for lung cancer of 199 (O=46; E=23.1; $p<0.001$) for chrome bath workers and a SMR of 101 for other chrome work. The SMR for cancer of the stomach for male chrome bath workers was also statistically significantly elevated (O=13; E=6.3; SMR=206; $p<0.05$); for stomach cancer in males doing other chrome work, the SMR was 160 with 8 observed and 5 expected. Both of the nasal cancers in males and the one nasal cancer in women were chrome bath workers. The nasal cancer SMR for males was statistically significantly elevated (O=2; E=0.1; SMR=2000; $p<0.05$).

Regression analysis was used to examine evidence of association of several types of cancers and Cr(VI) exposure duration among the cohort. There was a significant positive association between lung cancer mortality and exposure duration as a chrome bath worker controlling for gender as well as year and age at the start of employment. There was no evidence of an association between other cancer types and duration of Cr(VI) exposure. There was no positive association between duration of exposure to nickel bath work and cancer of the lung. The two largest reported SMRs were for chrome bath workers 10–14 years (O=13; E=3.8; SMR=342; $p<0.001$) and 15–19 years (O=12; E=4.9; SMR=245; $p<0.01$) after starting employment. The positive associations between lung cancer mortality and duration of chrome bath work suggests Cr(VI) exposure may be responsible for the excess cancer risk.

Sorahan *et al.* reported the results of a follow-up to the nickel/chromium platers study discussed above (Ex. 271). The cohort was redefined and excluded employees whose personnel records could not be located (650); those who started chrome work prior to 1946 (31) and those having no chrome exposure (236). The vital status experience of 1,762 workers (812 men and 950 women) was followed through 1995. The expected number of deaths was based upon the mortality of the general population of England and Wales.

There were 421 deaths among the men and 269 deaths among the women, including 52 lung cancers among the men and 17 among the women. SMRs were calculated for different categories of chrome work: period from first chrome work; year of starting chrome work, and cumulative duration of chrome work categories. Poisson regression modeling was employed to investigate lung cancer in relation to type of chrome work and cumulative duration of work.

A significantly elevated lung cancer SMR was seen for male workers with some period of chrome bath work (O=40; E=25.4; SMR=157; 95% CI: 113–214, $p<0.01$) that was not the case for male workers engaged in other chrome work away from the chromic acid bath (O=9; E=13.7; SMR=66; 95% CI: 30–125). Similar lung cancer mortality results were found for female chrome bath workers (O=15; E=8.6; SMR=175; 95% CI: 98–285; $p<0.06$). After adjusting for sex, age, calendar year, year starting chrome work, period from first chrome work, and employment status, regression modeling showed a statistically significant positive trend ($p<0.05$) between duration of chrome bath work and lung cancer mortality risk. The relative lung cancer risk for chrome bath workers with more than five years of Cr(VI) exposure (i.e., relative to the risk of those without any chrome bath work) was 4.25 (95% CI: 1.83–9.37).

Since the Sorahan cohort consists of nickel/chromium workers, the question arises of the potential confounding of nickel. In the earlier study, 144 of the 564 employees with some period of chrome bath work had either separate or simultaneous periods of nickel bath employment. According to the authors, there was no clear association between cancer deaths from stomach, liver, respiratory system, nose and larynx, and

lung and bronchus and the duration of nickel bath employment. In the follow-up report, the authors re-iterate this result stating, ''findings for lung cancer in a cohort of nickel platers (without any exposure to chrome plating) from the same factory are unexceptional'' (Ex. 271, p. 241).

Silverstein *et al.* reported the results of a cohort study of hourly employees and retirees with at least 10 years of credited pension service in a Midwestern plant manufacturing hardware and trim components for use primarily in the automobile industry (Ex. 7–55). Two hundred thirty eight deaths occurred between January 1, 1974 and December 31, 1978. Proportional Mortality Ratio (PMR) analysis adjusted for race, gender, age and year of death was conducted. For white males, the PMR for cancer of the lung and pleura was 1.91 ($p<0.001$) based upon 28 deaths. For white females, the PMR for cancer of the lung and pleura was 3.70 ($p<0.001$) based upon 10 deaths.

White males who worked at the plant for less than 15 years had a lung cancer PMR of 1.65. Those with 15 or more years at the plant had a lung cancer PMR of 2.09 ($p<0.001$). For white males with less than 22.5 years between hire and death (latency) the lung cancer PMR was 1.78 ($p<0.05$) and for those with 22.5 or more years, the PMR was 2.11 ($p<0.01$).

A case-control analysis was conducted on the Silverstein cohort to examine the association of lung cancer risk with work experience. Controls were drawn from cardiovascular disease deaths (ICD 390–458, 8th revision). The 38 lung cancer deaths were matched to controls for race and gender. Odds ratios (ORs) were calculated by department depending upon the amount of time spent in the department (ever/never; more vs. less than one year; and more vs. less than five years). Three departments showed increasing odds ratios with duration of work; however, the only statistically significant result was for those who worked more than five years in department 5 (OR=9.17, $p=0.04$, Fisher's exact test). Department 5 was one of the major die-casting and plating areas of the plant prior to 1971.

Franchini *et al.* conducted a mortality study of employees and retirees from nine chrome plating plants in Parma, Italy (Ex. 7–56). Three plants produced hard chrome plating. The remaining six plants produced decorative chromium plates. A limited number of airborne chromium measurements were available. Out of a total of 10 measurements at the hard chrome plating plants, the air concentrations of chromium averaged 7 $\mu g/m^3$ (range of 1–50 $\mu g/m^3$) as chromic acid near the baths and 3 $\mu g/m^3$ (range of 0–12 $\mu g/m^3$) in the middle of the room.

The cohort consisted of 178 males (116 from the hard chromium plating plants and 62 from the bright chromium plating plants) who had worked for at least one year between January 1, 1951 and December 31, 1981. In order to allow for a 10 year latency period, only those employed before January 1972 were included in further analysis. There were three observed lung cancer deaths among workers in the hard chrome plating plants, which was significantly greater than expected (O=3; E=0.6; $p<0.05$). There were no lung cancer deaths among decorative chrome platers.

Okubo and Tsuchiya conducted a study of plating firms with five or more employees in Tokyo (Exs. 7–51; 7–52). Five hundred and eighty nine firms were sent questionnaires to ascertain information regarding chromium plating experience. The response rate was 70.5%. Five thousand one hundred seventy platers (3,395 males and 1,775 females) met the cohort entrance criteria and were followed from April 1, 1970 to September 30, 1976. There were 186 deaths among the cohort; 230 people were lost to follow-up after retirement. The cohort was divided into two groups: chromium platers who worked six months or more and a control group with no exposure to chromium (clerical, unskilled workers). There were no deaths from lung cancer among the chromium platers.

The Okubo cohort was updated by Takahashi and Okubo (Ex. 265). The cohort was redefined to consist of 1,193 male platers employed for a minimum of six months between April 1970 and September 1976 in one of 415 Tokyo chrome plating plants and who were alive and over 35 years of age on September 30, 1976. The only statistically significant SMR was for lung cancer for all platers combined (O=16; E=8.9; SMR=179; 95% CI: 102–290). The lung cancer SMR for the chromium plater subcohort was 187 based upon eight deaths and 172 for the nonchromium plater subcohort, also based upon eight deaths. The cohort was followed through 1987. Itoh *et al.* updated the Okubo metal plating cohort through December 1992 (Ex. 35–163). They reported a lung cancer SMR of 118 (95% CI: 99–304).

4. Evidence From Stainless Steel Welders

Welding is a term used to describe the process for joining any materials by fusion. The fumes and gases associated with the welding process can cause a wide range of respiratory exposures which may lead to an increased risk of lung cancer. The major classes of metals most often welded include mild steel, stainless and high alloy steels and aluminum. The fumes from stainless steel, unlike fumes from mild steel, contain nickel and Cr(VI). There are several cohort and case-control studies as well as two meta analyses of welders potentially exposed to Cr(VI). In general, the studies found an excess number of lung cancer deaths among stainless steel welders. However, few of studies found clear trends with Cr(VI) exposure duration or cumulative Cr(VI). In most studies, the reported excess lung cancer mortality among stainless steel welders was no greater than mild steel welders, even though Cr(VI) exposure is much greater during stainless steel welding. This weak association between lung cancer and indices of exposure limits the evidence provided by these studies. Another limitation was the co-exposures to other potential lung carcinogens, such as nickel, asbestos, and cigarette smoke. Nevertheless, these studies add some further support to the much stronger link between Cr(VI) and lung cancer found in soluble chromate production workers, chromate pigment production workers, and chrome platers. The key studies are summarized in Table VI–4.

TABLE VI–4.– SUMMARY OF SELECTED EPIDEMIOLOGIC STUDIES OF LUNG CANCER IN WORKERS EXPOSED TO HEXAVALENT CHROMIUM—STAINLESS STEEL WELDING

Reference/Exhibit No.	Study population	Reference population	Chromium (VI) exposure	Lung cancer risk
Moulin (1997, Ex. 35–285)	Meta analysis of epidemiological studies of lung cancer risk among welders in five categories including stainless steel welding and mild steel welding.	Stainless steel welding cohort studies: Simonato *et al.*, 1991; Polednak *et al.*, 1981 case control studies: Hull *et al.*, 1989; Gerin *et al.*, 1984; Kjuus *et al.* 1986.	Stainless steel welders exposed to higher Cr(VI) than mild steel welders.	—RR of 1.50 ($p<0.05$) for stainless steel welders based on combined 114 deaths from five studies —RR of 1.50 ($p<0.05$) for mild steel welders based on combined 137 deaths from four studies.
Sjogren *et al.* (1994, Ex. 7–113).	Meta analysis of epidemiological studies of exposure to stainless steel welding fumes and lung cancer.	Stainless steel welding cohort studies: Moulin et al., 1993; Sjogren *et al.*, 1987 case control studies: Lauritsen *et al.*, 1996; Gerin *et al.*, 1984; Kjuus *et al.* 1986.	Cr(VI) exposure was not part of the analysis.	RR of 1.94 ($p<0.05$) for stainless steel welders based on combined 70 deaths from five studies.
Simonato *et al.* (1991, Ex.7–114). Gerin *et al.* (1993, Ex. 35–220)	Cohort of 11,092 male welders from 135 companies in nine European countries. Cohort entrance criteria varied by country.	Age and sex specific mortality rates computed using the WHO mortality data bank.	Avg cumulative Cr(VI) exposures estimated between 0.05 to 1.5 mg/ m^3-yr based on job process matrix.	—O/E of 1.23 (NS) for primarily stainless steel welders based on 20 deaths. —Upward trend ($p<0.05$) with time since first exposure. —No trend with cumulative exposure
Moulin *et al.* (1993, Ex. 7–92)	Cohort of 2,721 French male welders from 13 factories with a minimum of one year of employment from 1975 to 1988.	6,683 unexposed manual workers from 13 factories with a minimum of one year of employment from 1975 to 1988.	—Primarily manual metal arc welding. —Cr(VI) exposures not recorded	—O/E of 1.03 (NS) for primarily stainless steel welders based on 2 deaths. —No trend with exposure duration.
Hansen *et al.* (1996, Ex. 35–247).	Cohort of 10,059 male welders and other steel workers from 79 Danish companies employed for a minimum of one year between 1964 and 1984.	National cancer incidence rates from the Danish Cancer Registry.	Cr(VI) exposure not recorded ..	—O/E of 2.38 (NS) for stainless steel only welders based on 5 deaths. No trend with exposure duration.
Lauritsen *et al.* (1996, Ex. 35–291).	Nested case-control study of 94 lung cancer deaths from Hansen study.	439 eligible controls who were not cases and did not have respiratory disease or unknown malignancy as cause of death.	Cr(VI) exposure not recorded ..	—OR of 1.3 (NS) for stainless steel only welders. —No trend with exposure duration.
Sjogren *et al.* (1987, Ex. 795)	Cohort of 234 male stainless steel welders and 208 male railway track welders. Minimum employment was 5 years between 1950 and 1965. Follow-up through 1984.	Mortality rates for Swedish males.	Median Cr level for stainless steel welding was 57 $\mu g/m^3$ and for gas shielded welding [railway welders] was 5 $\mu g/m^3$ in Sweden during 1975.	—O/E of 2.5 (NS) for stainlesssteel welders based on 5 deaths. —O/E of 0.3 (NS) for railway welders based on 1 death.
Kjuus *et al.* (1986, Ex. 7–72) ...	A hospital-based case-control study of 176 male incident lung cancer cases admitted to two hospitals in Norway during 1979–1983.	186 controls admitted to the same hospitals in Norway during 1979–1983 and matched to cases for age +/- 5 years.	Cr(VI) exposure not recorded ..	—OR of 3.0 ($p<0.05$, adjusted for smoking) for stainless steel welding based on 16 deaths. —Welding not significant in logistic model with smoking, asbestos.
Hull, *et al.* (1989, Ex. 35–243)	Case-control study of 85 lung cancer cases in white male welders identified through the LA County tumor registry (1972–1987).	Controls were 74 welders with non-pulmonary malignancies.	No direct Cr(VI) exposure measurements recorded.	—OR of 0.9 (NS) for stainless steel welding based on 34 cases. —OR of 1.3 (NS) for manual metal arc welding on stainless steel based on 61 cases.

Observed/Expected (O/E)
Relative Risk (RR)
Not Statistically Significant (NS)
Odds Ratio (OR)

Sjogren *et al.* reported on the mortality experience in two cohorts of welders (Ex. 7–95). The cohort characterized as ''high exposure'' consisted of 234 male stainless steel welders with a minimum of five years of employment between 1950 and 1965. An additional criterion for inclusion in the study was assurance from the employer that asbestos had not been used or had been used only occasionally and never in a dust-generating way. The cohort characterized as ''low exposure'' consisted of 208 male railway track welders working at the Swedish State Railways for at least five years between 1950 and 1965. In 1975, air pollution in stainless steel welding was surveyed in Sweden. The median time weighted average (TWA) value for Cr(VI) was 110 $\mu g\ CrO_3/m^3$ (57 $\mu g/m^3$ measured as CrVI). The highest concentration was 750 $\mu g\ CrO_3/m^3$ (390 $\mu g/m^3$ measured as CrVI) found in welding involving coated electrodes. For gas-shielded welding, the median Cr(VI) concentration was 10 $\mu g\ CrO_3/m^3$ (5.2 $\mu g/m^3$ measured as CrVI) with the highest concentration measured at 440 $\mu g\ CrO_3/m^3$ (229 $\mu g/m^3$ measured as CrVI). Follow-up for both cohorts was through December 1984. The expected number of deaths was based upon Swedish male death rates. Of the 32 deaths in the ''high exposure'' group, five were cancers of the trachea, bronchus and lung (E=2.0; SMR=249;

95% CI: 0.80–5.81). In the low exposure group, 47 deaths occurred, one from cancer of the trachea, bronchus and lung.

Polednak compiled a cohort of 1,340 white male welders who worked at the Oak Ridge nuclear facilities from 1943 to 1977 (Ex. 277). One thousand fifty-nine cohort members were followed through 1974. The cohort was divided into two groups. The first group included 536 welders at a facility where nickel-alloy pipes were welded; the second group included 523 welders of mild steel, stainless steel and aluminum materials. Smoking data were available for 33.6% of the total cohort. Expectations were calculated based upon U.S. mortality rates for white males. There were 17 lung cancer deaths in the total cohort (E=11.37; SMR=150; 95% CI: 87–240). Seven of the lung cancer deaths occurred in the group which routinely welded nickel-alloy materials (E=5.65; SMR=124; 95% CI: 50–255) versus 10 lung cancer deaths in the ''other'' welders (E=6.12; SMR=163; 95% CI: 78–300).

Becker *et al.* compiled a cohort of 1,213 stainless steel welders and 1,688 turners from 25 German metal processing factories who had a minimum of six months employment during the period 1950–1970 (Exs. 227;250;251). The data collected included the primary type of welding (*e.g.*, arc welding, gas-shielded welding, etc.) used by each person, working conditions, average daily welding time and smoking status. The most recent follow-up of the cohort was through 1995. Expected numbers were developed using German mortality data. There were 268 deaths among the welders and 446 deaths among the turners. An elevated, but non-statistically significant, lung cancer SMR (O=28; E=23; SMR=121.5; 95% CI: 80.7–175.6) was observed among the welders. There were 38 lung cancer deaths among the turners with 38.6 expected, resulting in a SMR slightly below unity. Seven deaths from cancer of the pleura (all mesotheliomas) occurred among the welders with only 0.6 expected (SMR=1,179.9; 95% CI: 473.1–2,430.5), compared to only one death from cancer of the pleura among the turners, suggesting that the welders had exposure to asbestos. Epidemiological studies have shown that asbestos exposure is a primary cause of pleural mesotheliomas.

The International Agency for Research on Cancer (IARC) and the World Health Organization (WHO) cosponsored a study on welders. IARC and WHO compiled a cohort of 11,092 male welders from 135 companies in nine European countries to investigate the relationship between the different types of exposure occurring in stainless steel, mild steel and shipyard welding and various cancer sites, especially lung cancer (Ex. 7–114). Cohort entrance criteria varied by country. The expected number of deaths was compiled using national mortality rates from the WHO mortality data bank.

Results indicated the lung cancer deaths were statistically significant in the total cohort (116 cases; E=86.81; SMR=134; 95% CI: 110–160). Cohort members were assigned to one of four subcohorts based upon type of welding activity. While the lung cancer SMRs were elevated for all of the subcohorts, the only statistically significant SMR was for the only mild steel welders (O=40; E=22.42; SMR=178; 95% CI: 127–243). Results for the other subgroups were: shipyard welders (O=36; E=28.62; SMR=126; 95% CI: 88–174); ever stainless steel welders (O=39; E=30.52; SMR=128; 95% CI: 91–175); and predominantly stainless steel welders (O=20; E=16.25; SMR=123; 95% CI: 75–190). When analyzed by subcohort and time since first exposure, the SMRs increased over time for every group except shipyard welders. For the predominantly stainless steel welder subcohort, the trend to increase with time was statistically significant ($p < .05$).

An analysis was conducted of lung cancer mortality in two stainless steel welder subgroups (predominantly and ever) with a minimum of five years of employment. Cumulative Cr(VI) was computed from start of exposure until 20 years prior to death. A lung cancer SMR of 170, based upon 14 cases, was observed in the stainless steel ever subgroup for those welders with >0.5 μg-years/m^3 Cr(VI) exposure; the lung cancer SMR for those in the <0.5 μg-years/m^3 Cr(VI) exposure group was 123 (based upon seven cases). Neither SMR was statistically significant. For the predominantly stainless steel welders, which is a subset of the stainless steel ever subgroup, the corresponding SMRs are 167 (>0.5 μg-years/m^3 Cr(VI) exposure) based upon nine cases and 191 (<0.5 μg-years/m^3 Cr(VI) exposure) based upon three cases. Neither SMR is statistically significant.

In conjunction with the IARC/WHO welders study, Gerin *et al.* reported the development of a welding process exposure matrix relating 13 combinations of welding processes and base metals used to average exposure levels for total welding fumes, total chromium, Cr(VI) and nickel (Ex. 7–120). Quantitative estimates were derived from the literature supplemented by limited monitoring data taken in the 1970s from only eight of the 135 companies in the IARC/WHO mortality study. An exposure history was constructed which included hire and termination dates, the base metal welded (stainless steel or mild steel), the welding process used and changes in exposure over time. When a detailed welding history was not available for an individual, the average company welding practice profile was used. In addition, descriptions of activities, work force, welding processes and parameters, base metals welded, types of electrodes or rods, types of confinement and presence of local exhaust ventilation were obtained from the companies.

Cumulative dose estimates in mg/m^3 years were generated for each welder's profile (number of years and proportion of time in each welding situation) by applying a welding process exposure matrix associating average concentrations of welding fumes (mg/m^3) to each welding situation. The corresponding exposure level was multiplied by length of employment and summed over the various employment periods involving different welding situations. No dose response relationship was seen for exposure to Cr(VI) for either those who were ''ever stainless steel welders'' or those who were ''predominantly stainless steel welders''. The authors note that if their exposure estimates are correct, the study had the power to detect a significant result in the high exposure group for Cr(VI).

The IARC/WHO multicenter study is the sole attempt to undertake even a semi-quantified exposure analysis of stainless steel welders' potential exposure to nickel and Cr(VI) for <5 and ≥0.5 mg-years/m^3 Cr(VI) exposures. The IARC/WHO investigators noted that there was more than a twofold increase in SMRs between the long (≥20 years since first exposure) and short (<20 years since first exposure) observation groups for the predominantly stainless steel welders ''suggesting a relation of lung cancer mortality with the occupational environment for this group'' (Ex. 7–114, p. 152). The authors conclude that the increase in lung cancer mortality does not appear to be related to either duration of exposure or cumulative exposure to total fume, chromium, Cr(VI) or nickel.

Moulin compiled a cohort of 2,721 French male welders and an internal comparison group of 6,683 manual workers employed in 13 factories (including three shipyards) with a minimum of one year of employment from 1975 to 1988 (Ex. 7–92). Three

controls were selected at random for each welder. Smoking data were abstracted from medical records for 86.6% of welders and 86.5% of the controls. Smoking data were incorporated in the lung cancer mortality analysis using methods suggested by Axelson. Two hundred and three deaths were observed in the welders and 527 in the comparison group. A non-statistically significant increase was observed in the lung cancer SMR (O=19; E=15.33; SMR=124; 95% CI: 0.75–1.94) for the welders. In the control group, the lung cancer SMR was in deficit (O=44; E=46.72; SMR=94; 95% CI: 0.68–1.26). The resulting relative risk was a non-significant 1.3. There were three deaths from pleural cancer in the comparison group and none in the welders suggesting asbestos exposure in the comparison group. The welders were divided into four subgroups (shipyard welders, mild steel only welders, ever stainless steel welders and stainless steel predominantly Cr(VI) welders). The highest lung cancer SMR was for the mild steel welders O=9; SMR of 159). The lowest lung cancer SMRs were for ever stainless steel welders (O=3; SMR= 92) and for stainless steel predominantly Cr(VI) welders (O=2; SMR=103). None of the SMRs are statistically significant.

Hansen conducted a study of cancer incidence among 10,059 male welders, stainless steel grinders and other metal workers from 79 Danish companies (Ex. 9–129). Cohort entrance criteria included: Alive on April 1, 1968; born before January 1, 1965; and employed for at least 12 months between April 1, 1964 and December 31, 1984. Vital status follow-up found 9,114 subjects alive, 812 dead and 133 had emigrated. A questionnaire was sent to subjects and proxies for decedents/emigrants in an attempt to obtain information about lifetime occupational exposure, smoking and drinking habits. The overall response rate was 83%. The authors stated that no major differences in smoking habits were found between exposure groups with or without a significant excess of lung cancer.

The expected number of cancers was based on age-adjusted national cancer incidence rates from the Danish Cancer Registry. There were statistically significantly elevated Standardized Incidence Ratios (SIRs) for lung cancer in the welding (any kind) group (O=51; E=36.84; SIR=138; 95% CI: 103–181) and in the mild steel only welders (O=28; E=17.42; SIR=161; 95% CI: 107–233). The lung cancer SIR for mild steel ever welders was 132 (O=46; E=34.75; 95% CI: 97–176); for stainless steel ever welders 119 (O=23; E=19.39; 95% CI: 75–179) and for stainless steel only welders 238 (O=5; E=2.10; 95% CI: 77–555).

Laurtitsen reported the results of a nested case-control conducted in conjunction with the Hansen cancer incidence study discussed above (Exs. 291; 9–129). Cases were defined as the 94 lung cancer deaths. Controls were defined as anyone who was not a case, but excluded deaths from respiratory diseases other than lung cancer (either as an underlying or a contributing cause of death), deaths from ''unknown malignancies'' and decedents who were younger than the youngest case. There were 439 decedents eligible for use as controls.

The crude odds ratio (OR) for welding ever (yes/no) was 1.7 (95% CI: 1.0–2.8). The crude OR for mild steel welding only was 1.3 (95% CI: 0.8–2.3) and for stainless steel welding only the crude OR was 1.3 (95% CI: 0.3–4.3). When analyzed by number of years exposed, ''ever'' stainless steel welding showed no relationship with increasing number of years exposed. The highest odds ratio (2.9) was in the lowest category (1–5 years) based upon seven deaths; the lowest odds ratio was in the highest category (21+ years) based upon three deaths.

Kjuus *et al.* conducted a hospital-based case-control study of 176 male incident lung cancer cases and 186 controls (matched for age, +/− 5 years) admitted to two county hospitals in southeast Norway during 1979–1983 (Ex. 7–72). Subjects were classified according to exposure status of main occupation and number of years in each exposure category and assigned into one of three exposure groups according to potential exposure to respiratory carcinogens and other contaminants. A statistically significantly elevated risk ratio for lung cancer (adjusted for smoking) for the exposure factor ''welding, stainless, acid proof'' of 3.3 ($p<0.05$) was observed based upon 16 lung cancer deaths. The unadjusted odds ratio is not statistically significant (OR=2.8). However, the appropriateness of the analysis is questionable since the exposure factors are not discrete (a case or a control may appear in multiple exposure factors and therefore is being compared to himself). In addition, the authors note that several exposure factors were highly correlated and point out specifically that one-half of the cases ''exposed to either stainless steel welding fumes or fertilizers also reported moderate to heavy asbestos exposure.'' When put into a stepwise logistic regression model, exposure to stainless steel fumes, which was initially statistically significant, loses its significance when smoking and asbestos are first entered into the model.

Hull *et al.* conducted a case-control study of lung cancer in white male welders aged 20–65 identified through the Los Angeles County tumor registry (Southern California Cancer Surveillance Program) for the period 1972 to 1987 (Ex. 243). Controls were welders 40 years of age or older with non-pulmonary malignancies. Interviews were conducted to obtain information about sociodemographic data, smoking history, employment history and occupational exposures to specific welding processes, metals welded, asbestos and confined space welding. Interviews were completed for 90 (70%) of the 128 lung cancer cases and 116 (66%) of the controls. Analysis was conducted using 85 deceased cases and 74 deceased controls after determining that the subject's vital status influenced responses to questions concerning occupational exposures. The crude odds ratio (ever vs. never exposed) for stainless steel welding, based upon 34 cases, was 0.9 (95% CI: 0.3–1.4). For manual metal arc welding on stainless steel, the crude odds ratio was 1.3 (95% CI: 0.6–2.3) based upon 61 cases.

While the relative risk estimates in both cohort and case-control of stainless steel welders are elevated, none are statistically significant. However, when combined in two meta-analyses, a small but statistically significant increase in lung cancer risk was reported. Two meta-analyses of welders have been published. Moulin carried out a meta-analysis of epidemiologic studies of lung cancer risk among welders, taking into account the role of asbestos and smoking (Ex. 285). Studies published between 1954 and 1994 were reviewed. The inclusion criteria were clearly defined: only the most recent updates of cohort studies were used and only the mortality data from mortality/morbidity studies were included. Studies that did not provide the information required by the meta-analysis were excluded.

Five welding categories were defined (shipyard welding, non-shipyard welding, mild steel welding, stainless steel welding and all or unspecified welding). The studies were assigned to a welding category (or categories) based upon the descriptions provided in the paper's study design section. The combined relative risks (odds ratios, standardized mortality ratios, proportionate mortality ratios and standardized incidence ratios) were calculated separately for the population-based studies, case-control studies and

cohort studies and for all the studies combined.

Three case-control studies (Exs. 243; 7–120; 7–72) and two cohort studies (Exs. 7–114; 277) were included in the stainless steel welding portion of the meta-analysis. The combined relative risk was 2.00 (O=87; 95% CI: 1.22–3.28) for the case-control studies and 1.23 (O=27; 95% CI: 0.82–1.85) for the cohort studies. When all five studies were combined, the relative risk was 1.50 (O=114; 95% CI: 1.10–2.05).

By contrast, the combined risk ratio for the case-control studies of mild steel welders was 1.56 (O=58; 95% CI: 0.82–2.99) (Exs. 7–120; 243). For the cohort studies, the risk ratio was 1.49 (O=79; 95% CI: 1.15–1.93) (Exs. 270; 7–114). For the four studies combined, the risk ratio was 1.50 (O=137; 95% CI: 1.18–191). The results for the stainless steel welders and the mild steel welders are basically the same.

The meta-analysis by Sjogren of exposure to stainless steel welding fumes and lung cancer included studies published between 1984 and 1993, which took smoking and potential asbestos exposure into account (Ex. 7–113). Five studies met the author's inclusion criteria and were included in the meta-analysis: two cohort studies, Moulin *et al.* (Ex. 283) and Sjogren *et al.* (Ex. 7–95); and three case-control studies, Gerin, *et al.* (Ex. 7–120, Hansen *et al.* (Ex. 9–129) and Kjuus *et al.* (Ex. 7–72). The calculated pooled relative risk for welders exposed to stainless steel welding fumes was 1.94 (95% CI: 1.28–2.93).

5. Evidence From Ferrochromium Workers

Ferrochromium is produced by the electrothermal reduction of chromite ore with coke in the presence of iron in electric furnaces. Some of the chromite ore is oxidized into Cr(VI) during the process. However, most of the ore is reduced to chrome metal. The manufacture of ferroalloys results in a complex mixture of particles, fumes and chemicals including nickel, Cr(III) and Cr(VI). Polycyclic aromatic hydrocarbons (PAH) are released during the manufacturing process. The co-exposure to other potential lung carcinogens combined with the lack of a statistically significant elevation in lung cancer mortality among ferrochromium workers were limitations in the key studies. Nevertheless, the observed increase in the relative risks of lung cancer add some further support to the much stronger link between Cr(VI) and lung cancer found in soluble chromate production workers, chromate pigment production workers, and chrome platers. The key studies are summarized in Table VI–5.

TABLE VI–5.—SUMMARY OF SELECTED EPIDEMIOLOGIC STUDIES OF LUNG CANCER IN WORKERS EXPOSED TO HEXAVALENT CHROMIUM—FERROCHROMIUM PRODUCTION

Reference/Exhibit No.	Study population	Reference population	Chromium (VI) exposure	Lung cancer risk
Axelsson *et al.* (1980, Ex. 7–62).	1932 Swedish males employed at least one year in a ferrochromium between 1930 to 1975.	Swedish county mortality and incidence rates.	"Recent" job-specific Cr(VI) levels estimated at 10 to 250 μg/m^3.	—O/E of 0.7 (NS) for ferrochromium workers based on 5 cases. —No trend with job-specific Cr(VI).
Langard *et al.* (1990, Ex. 7–37)	1235 males employed at least one year who started working prior to 1965 in a Norway ferrochromium plant. Follow-up was through 1985.	—Norwegian Cancer Registry —Subcohort of ferrosilicon workers at same plant not exposed to Cr(VI).	Avg total Cr exposure was 50 μg/m^3 in 1975 with 11 to 33% soluble Cr(VI).	—O/E of 1.5 (NS) for ferrochromium workers based on 10 cases. —O/E of 0.3 for ferrosilicon workers based on 2 cases.

Observed/Expected (O/E).
Relative Risk (RR).
Not Statistically Significant (NS).
Odds Ratio (OR).

Langard *et al.* conducted a cohort study of male workers producing ferrosilicon and ferrochromium for more than one year between 1928 and 1977 at a plant located on the west coast of Norway (Exs. 7–34; 7–37). The cohort and study findings are summarized in Table VI.5. Excluded from the study were workers who died before January 1, 1953 or had an unknown date of birth. The cohort was defined in the 1980 study as 976 male employees who worked for a minimum of one year prior to January 1, 1960. In the 1990 study, the cohort definition was expanded to include those hired up to 1965.

Production of ferrosilicon at the plant began in 1928 and ferrochromium production began in 1932. Job characterizations were compiled by combining information from company personnel lists and occupational histories contained in medical records and supplemented with information obtained via interview with long-term employees. Ten occupational categories were defined. Workers were assigned to an occupational category based upon the longest time in a given category.

Industrial hygiene studies of the plant from 1975 indicated that both Cr(III) and Cr(VI) were present in the working environment. The ferrochromium furnance operators were exposed to measurements of 0.04–0.29 mg/m^3 of total chromium. At the charge floor the mean concentration of total chromium was 0.05 mg/m^3, 11–33% of which was water soluble. The water soluble chromium was considered to be in the hexavalent state.

Both observed and expected cases of cancer were obtained via the Norwegian Cancer Registry. The observation period for cancer incidence was January 1, 1953 to December 31, 1985. Seventeen incident lung cancers were reported in the 1990 study (E=19.4; SIR=88). A deficit of lung cancer incidence was observed in the ferrosilicon group (O=2; E=5.8; SIR=35). In the ferrochromium group there were a significant excess of lung cancer; 10 observed lung cancers with 6.5 expected (SIR=154).

Axelsson *et al.* conducted a study of 1,932 ferrochromium workers to examine whether exposure in the ferrochromium industry could be associated with an increased risk of developing tumors, especially lung cancer (Ex. 7–62). The study cohort and findings are summarized in Table VI.5. The study cohort was defined as males employed at a ferrochromium plant in Sweden for at least one year during the period January 1, 1930 to December 31, 1975.

The different working sites within the industry were classified into four groups with respect to exposure to Cr(VI) and Cr(III). Exposure was primarily to metallic and trivalent chromium with estimated levels ranging from 0–2.5 mg/m^3. Cr(VI) was also present in certain operations with estimated levels ranging from 0–0.25 mg/m^3. The highest exposure to Cr(VI) was in the arc-furnace operations. Cr(VI) exposure also

occurred in a chromate reduction process during chromium alum production from 1950–1956. Asbestos-containing materials had been used in the plant. Cohort members were classified according to length and place of work in the plant.

Death certificates were obtained and coded to the revision of the *International Classification of Diseases* in effect at the time of death. Data on cancer incidence were obtained from the Swedish National Cancer Registry. Causes of death in the cohort for the period 1951–1975 were compared with causes of death for the age-adjusted male population in the county in which the plant was located.

There were seven cases of cancers of the trachea, bronchus and lung and the pleura with 5.9 expected (SIR=119) for the period 1958–1975. Four of the seven cases in the lung cancer group were maintenance workers and two of the four cases were pleural mesotheliomas. In the arc furnace group, which was thought to have the highest potential exposure to both Cr(III) and Cr(VI), there were two cancers of the trachea, bronchus and lung and the pleura. One of the cases was a mesothelioma. Of the 380 deaths that occurred during the period 1951–1975, five were from cancer of the trachea, bronchus and lung and the pleura (E=7.2; SMR=70). For the ''highly'' exposed furnace workers, there was one death from cancer of the trachea, bronchus and lung and the pleura.

Moulin *et al.* conducted a cohort mortality study in a French ferrochromium/stainless steel plant to determine if exposure to chromium compounds, nickel compounds and polycyclic aromatic hydrocarbons (PAHs) results in an increased risk of lung cancer (Ex. 282). The cohort was defined as men employed for at least one year between January 1, 1952 and December 31, 1982; 2,269 men met the cohort entrance criteria. No quantitative exposure data were available and no information on the relative amounts of Cr(VI) and Cr(III) was provided. In addition, some workers were also exposed to other carcinogens, such as silica and asbestos. The authors estimated that 75.7% of the cohort had been exposed to combinations of PAH, nickel and chromium compounds. Of the 137 deaths identified, the authors determined 12 were due to cancer of the trachea, bronchus and lung (E=8.56; SMR=140; 95% CI: 0.72–2.45). Eleven of the 12 lung cancers were in workers employed for at least one year in the ferrochromium or stainless steel production workshops (E=5.4; SMR=204; 95% CI: 1.02–3.64).

Pokrovskaya and Shabynina conducted a cohort mortality study of male and female workers employed ''some time'' between 1955 and 1969 at a chromium ferroalloy production plant in the U.S.S.R (Ex. 7–61). Workers were exposed to both Cr(III) and Cr(VI) as well as to benzo [a] pyrene. Neither the number of workers nor the number of cancer deaths by site were provided. Death certificates were obtained and the deaths were compared with municipal mortality rates by gender and 10 year age groups. The investigators state that they were able to exclude those in the comparison group who had chromium exposures in other industries. The lung cancer SMR for male chromium ferroalloy workers was 440 in the 30–39 year old age group and 660 in the 50–59 year old age group (p=0.001). There were no lung cancer deaths in the 40–49 and the 60–69 year old age groups. The data suggest that these ferrochromium workers may have been had an excess risk of lung cancer.

The association between Cr(VI) exposure in ferrochromium workers and the incidence of respiratory tract cancer these studies is difficult to assess because of co-exposures to other potential carcinogens (*e.g.*, asbestos, PAHs, nickel, etc.), absence of a clear exposure-response relationship and lack of information on smoking. There is suggestive evidence of excess lung cancer mortality among Cr(VI)-exposed ferrochromium workers in the Norwegian (Langard) cohort when compared to a similar unexposed cohort of ferrosilicon workers. However, there is little consistency for this finding in the Swedish (Axelsson) or French (Moulin) cohorts.

6. Evidence From Workers in Other Industry Sectors

There are several other epidemiological studies that do not fit into the five industry sectors previously reviewed. These include worker cohorts in the aerospace industry, paint manufacture, and leather tanning operations, among others. The two cohorts of aircraft manufacturing workers are summarized in Table VI–6. All of the cohorts had some Cr(VI) exposure but, certain cohorts may have included a sizable number of workers with little or no exposure to Cr(VI). This creates an additional complexity in assessing whether the study findings support a Cr(VI) etiology for cancer of the respiratory system.

TABLE VI–6.—SUMMARY OF SELECTED EPIDEMIOLOGIC STUDIES OF LUNG CANCER IN WORKERS EXPOSED TO HEXAVALENT CHROMIUM—AIRCRAFT MANUFACTURE

Reference/Exhibit No.	Study population	Reference population	Chromium (VI) exposure	Lung Cancer risk
Alexander *et al.* (1996, Ex. 31–16–3).	2429 aerospace workers with a minimum six months employment in Washington State from 1974 to 1994. Median age at end of study was 42 years with median 9 years follow-up.	Incidence rates from regional cancer surveillance system registry.	Painters/sanders exposed to zinc strontium and lead chromates. Platers/tank tenders exposed primarily to chromic acid Median cumulative chromate exposure between 0.01 and 0.18 mg/m^3-yr based on 1974 to 1994 data.	—O/E of 0.8 (NS) for aerospace cohort based on 15 deaths. —No clear trend with chromate exposure.
Boice *et al.* (1999, Ex. 31–16–4).	77,965 workers employed for minimum of one year in California aircraft manufacturing plant on or after 1960. Follow-up through 1996.	Mortality rates for white population of California and for non-white U.S. population.	8 percent of cohort had potential for routine Cr(VI) exposure as painters and platers. No Cr(VI) exposure levels reported.	—O/E of 1.02 (NS) for workers with routine Cr(VI) exposures based on 87 deaths. —Upward trend (NS) with duration of exposure. —O/E of 0.71 (p<0.05) for non-factory workers.

Observed/Expected (O/E)
Relative Risk (RR)
Not Statistically Significant (NS)
Odds Ratio (OR)

Alexander *et al.* conducted a cohort study of 2,429 aerospace workers with a minimum of six months of cumulative employment in jobs involving chromate exposure during the period 1974 through 1994 (Ex. 31–16–3). Exposure estimates were based on industrial hygiene measurements and work history records. Jobs were classified into categories of ''high'' (spray painters, decorative painters), ''moderate'' (sanders/maskers, maintenance painters) and ''low'' (chrome platers, surface processors, tank tenders, polishers, paint mixers) exposure. Each exposure category was assigned a summary TWA exposure based upon the weighted TWAs and information from industrial hygienists. The use of respiratory protection was accounted for in setting up the job exposure matrix. The index of cumulative total chromium exposure (reported as $\mu g/m^3$ chromate TWA-years) was computed by multiplying the years in each job by the summary TWAs for each exposure category.

In addition to cumulative chromate exposure, chromate exposure jobs were classified according to the species of chromate. According to the authors, in painting operations the exposure is to chromate pigments with moderate and low solubility such as zinc chromate, strontium chromate and lead chromate; in sanding and polishing operations the same chromate pigments exist as dust; while platers and tank tenders are exposed to chromium trioxide, which is highly soluble.

Approximately 26% of the cohort was lost to follow-up. The cohort was followed for a relatively short 8.9 years per cohort member. Cases were identified through the Cancer Surveillance System (CSS) at the Fred Hutchinson Cancer Research Center in Seattle, Washington. CSS records primary cancer diagnoses in 13 counties in western Washington. Expected numbers were calculated using race-, gender-, age- and calendar-specific rates from the Puget Sound reference population for 1974 through 1994. Fifteen lung cancer cases were identified with an overall standardized incidence ratio (SIR) of 80 (95% CI: 0.4–1.3). The SIRs for lung cancer by cumulative years of employment in the ''high exposure'' painting job category were based upon only three deaths in each of the cumulative years categories (<5 and ≥5); years of employment was inversely related to the risk of lung cancer. For those in the ''low exposure'' category, the SIRs were 130 for those who worked less than five years in that category (95% CI: 0.2–4.8) and 190 for those who worked five years or more (95% CI: 0.2–6.9). However, there were only two deaths in each category. The SIR for those who worked ≥5 years was 270 (95% CI: 0.5–7.8), but based only on three deaths.

Boice *et al.* conducted a cohort mortality study of 77,965 workers employed for a minimum of one year on or after January 1960 in aircraft manufacturing (Ex. 31–16–4). Routine exposures to Cr(VI) compounds occurred primarily while operating plating and coating process equipment or when using chromate based primers or paints. According to the authors, 3,634 workers, or 8% of the cohort, had the potential for routine exposure to chromate and 3,809 workers, or 8.4%, had the potential for intermittent exposure to chromate. Estimates of chromate exposure were not provided in the study.

Follow up of the cohort was through 1996. Expectations were calculated based on the general population of California for white workers, while general population rates for the U.S. were used for non-white workers. For the 3,634 cohort members who had potential for routine exposure to chromates, the lung cancer SMR (race and gender combined) was 102 based upon 87 deaths (95% CI: 0.82–1.26). There was a slight non-significant positive trend (p value>2.0) for lung cancer with duration of potential exposure. The SMR was 108 (95% CI: 0.75–1.57) for workers exposed to chromate for ≥5 years. Among the painters, there were 41 deaths from lung cancer yielding a SMR of 111 (95% CI: 0.80–1.51). For those who worked as a process operator or plater the SMR for lung cancer was 103 based upon 38 deaths (95% CI: 0.73–1.41).

OSHA believes the Alexander (Ex. 31–16–3) and the Boice *et al.* (Ex. 31–16–4) studies have several limitations. The Alexander cohort is small and lacks smoking data. In addition, the study's authors cite the relatively young age of the population. Considering these three factors, the authors note, ''limits the overall power of the study and the stability of the risk estimates, especially in exposure-related subanalyses'' (Ex. 31–16–3, p. 1256). Another limitation of the study is the 26.3% of cohort members lost to follow-up. Boice *et al.* is a well conducted study of workers in the aircraft manufacturing industry, but lacks information on Cr(VI) exposure (Ex. 31–16–4).

Dalager *et al.* conducted a proportionate mortality study of 977 white male spray painters potentially exposed to zinc chromate in the aircraft maintenance industry who worked at least three months and terminated employment within ten years prior to July 31, 1959 (Ex. 7–64). Follow-up was through 1977. The expected numbers of deaths were obtained by applying the cause-specific proportionate mortality of U.S. white males to the total numbers of deaths in the study group by five year age groups and five year time intervals. Two hundred and two deaths were observed. There were 21 deaths from cancer of the respiratory system (PMR=184), which was statistically significant. The Proportionate Cancer Mortality Ratio for cancer of the respiratory system was not statistically significant (PCMR=146). Duration of employment as a painter with the military as indicated on the service record was used as an estimate of exposure to zinc chromate pigments, which were used as a metal primer. The PMRs increased as duration of employment increased (<5 years, O=9, E=6.4, PMR=141; 5–9 years, O=6, E=3, PMR=200; and 10+ years, O=6, E=2, PMR=300) and was statistically significant for those who worked 10 or more years.

Bertazzi *et al.* studied the mortality experience of 427 workers employed for a minimum of six months between 1946 and 1977 in a plant manufacturing paint and coatings (Ex. 7–65). According to the author, chromate pigments represented the ''major exposure'' in the plant. The mortality follow-up period was 1954–1978. There were eight deaths from lung cancer resulting in a SMR of 227 on the local standard (95% CI: 156–633) and a SMR of 334 on the national standard (95% CI: 106–434). The authors were unable to differentiate between exposures to different paints and coatings. In addition, asbestos was used in the plant and may be a potential confounding exposure.

Morgan conducted a cohort study of 16,243 men employed after January 1, 1946 for at least one year in the manufacture of paint or varnish (Ex. 8–4). Analysis was also conducted for seven subcohorts, one of which was for work with pigments. Expectations were calculated based upon the mortality experience of U.S. white males. The SMR for cancer of the trachea, bronchus and lung was below unity based upon 150 deaths. For the pigment subcohort, the SMR for cancer of the trachea, bronchus and lung was 117 based upon 43 deaths. In a follow-up study of the subcohorts, case-control analyses were conducted for several causes of death including lung cancer (Ex. 286). The details of matching were not provided. The authors state that no significant excesses of lung cancer risk by job were found. No odds ratios were presented.

Pippard *et al.* conducted a cohort mortality study of 833 British male tannery workers employed in 1939 and followed through December 31, 1982 (Ex. 278). Five hundred and seventy three men worked in tanneries making vegetable tanned leathers and 260 men worked in tanneries that made chrome tanned leathers. The expected number of deaths was calculated using the mortality rates of England and Wales as a whole. The lung cancer SMR for the vegetable tanned leather workers was in deficit (O=31; E=32.6; 95% CI: 65–135), while the lung cancer SMR for the chrome tanned leather workers was slightly elevated but not statistically significant (O=13; E=12; SMR=108; 95% CI: 58–185).

In a different study of two U.S. tanneries, Stern *et al.* investigated mortality in a cohort of all production workers employed from January 1, 1940 to June 11, 1979 at tannery A (N=2,807) and from January 1, 1940 to May 1, 1980 at tannery B (N=6,558) (Ex. 7–68). Vital status was followed through December 31, 1982. There were 1,582 deaths among workers from the two tanneries. Analyses were conducted employing both U.S. mortality rates and the mortality rates for the state in which the plant is located. There were 18 lung/pleura cancer deaths at tannery A and 42 lung/pleura cancer deaths at tannery B. The lung cancer/pleura SMRs were in deficit on both the national standard and the state standard for both tanneries. The authors noted that since the 1940s most chrome tanneries have switched to the one-bath tanning method in which Cr(VI) is reduced to Cr(III).

Blot *et al.* reported the results of a cohort study of 51,899 male workers of the Pacific Gas & Electric Company alive in January 1971 and employed for at least six months before the end of 1986 (Ex. 239). A subset of the workers were involved in gas generator plant operations where Cr(VI) compounds were used in open and closed systems from the 1950s to early 1980s. One percent of the workers (513 men) had worked in gas generator jobs, with 372 identified from post-1971 listing at the company's three gas generator plants and 141 from gas generator job codes. Six percent of the cohort members (3,283) had trained at one of the gas generator plants (Kettleman).

SMRs based on national and California rates were computed. Results in the paper are based on the California rates, since the overall results reportedly did not differ substantially from those using the national rates. SMRs were calculated for the entire cohort and for subsets defined by potential for gas generator plant exposure. No significant cancer excesses were observed and all but one cancer SMR was in deficit. There were eight lung cancer deaths in the gas generator workers (SMR=81; 95% CI: 0.35–1.60) and three lung cancer deaths among the Kettleman trainees (SMR=57; 95% CI: 0.12–1.67). There were no deaths from nasal cancer among either the gas generator workers or the Kettleman trainees. The risk of lung cancer did not increase with length of employment or time since hire.

Rafnsson and Johannesdottir conducted a study of 450 licensed masons (cement finishers) in Iceland born between 1905 and 1945, followed from 1951 through 1982 (Ex. 7–73). Stonecutters were excluded. Expectations were based on the male population of Iceland. The SMR for lung cancer was 314 and is statistically significant based upon nine deaths (E=2.87; 95% CI: 1.43–5.95). When a 20 year latency was factored into the analysis, the lung cancer SMR remained statistically significant (O=8; E=2.19; SMR=365; 95% CI: 1.58–7.20).

Svensson *et al.* conducted a cohort mortality study of 1,164 male grinding stainless steel workers employed for three months or more during the period 1927–1981 (Ex.266). Workers at the facility were reportedly exposed to chromium and nickel in the stainless steel grinding process. Records provided by the company were used to assign each worker to one of three occupational categories: Those considered to have high exposure to chromium, nickel as well as total dust, those with intermediate exposure, and those with low exposure. Mortality rates for males in Blekinge County, Sweden were used as the reference population. Vital status follow-up was through December 31, 1983. A total of 194 deaths were observed (SMR= 91). No increased risk of lung cancer was observed (SMR=92). The SMR for colon/rectum cancer was 2.47, but was not statistically significant.

Cornell and Landis studied the mortality experience of 851 men who worked in 26 U.S. nickel/chromium alloy foundries between 1968 and 1979 (Ex. 7–66). Standardized Proportionate Mortality Ratio (SPMR) analyses were done using both an internal comparison group (foundry workers not exposed to nickel/chromium) and the mortality experience of U.S. males. The SPMR for lung cancer was 105 (O=60; E=56.9). No nasal cancer deaths were observed.

Brinton *et al.* conducted a case-control study of 160 patients diagnosed with primary malignancies of the nasal cavity and sinuses at one of four hospitals in North Carolina and Virginia between January 1, 1970 and December 31, 1980 (Ex. 8–8). For each case determined to be alive at the time of interview, two hospital controls were selected matched on vital status, hospital, year of admission (+/− 2 years), age (+/− 5 years), race and state economic area or county or usual residence. Excluded from control selection were malignant neoplasms of the buccal cavity and pharynx, esophagus, nasal cavity, middle ear and accessory sinuses, larynx, and secondary neoplasms. Also excluded were benign neoplasms of the respiratory system, mental disorders, acute sinusitis, chronic pharyngitis and nasopharyngitis, chronic sinusitis, deflected nasal septum or nasal polyps. For those cases who were deceased at the time of interview, two different controls were selected. One control series consisted of hospital controls as described previously. The second series consisted of decedents identified through state vital statistics offices matched for age (+ /− 5 years), sex, race, county of usual residence and year of death. A total of 193 cases were identified and 160 case interviews completed. For those exposed to chromates, the relative risk was not significantly elevated (OR=5.1) based upon five cases. According to the authors, chromate exposure was due to the use of chromate products in the building industry and in painting, rather than the manufacture of chromates.

Hernberg *et al.* reported the results of a case-control study of 167 living cases of nasal or paranasal sinus cancer diagnosed in Denmark, Finland and Sweden between July 1, 1977 and December 31, 1980 (Exs. 8–7; 7–71). Controls were living patients diagnosed with malignant tumors of the colon and rectum matched for country, gender and age at diagnosis (+ /− 3 years) with the cases. Both cases and controls were interviewed by telephone to obtain occupational histories. Patients with work-related exposures during the ten years prior to their illness were excluded. Sixteen cases reported exposure to chromium, primarily in the ''stainless steel welding'' and ''nickel'' categories, versus six controls (OR=2.7l; 95% CI: 1.1–6.6).

7. Evidence From Experimental Animal Studies

Most of the key animal cancer bioassays for chromium compounds were conducted before 1988. These studies have been critically reviewed by the IARC in the Monograph *Chromium, Nickel, and Welding* (Ex. 35–43) and by ATSDR in their toxicological profile for chromium (Ex. 35–41). OSHA reviewed

the critical studies from both the IARC Monograph and the ATSDR toxicological profile on chromium and conducted its own literature search to update and supplement the review.

In the experimental studies, Cr(VI) compounds were administered by various routes including inhalation, intratracheal instillation, intrabronchial implantation, and intrapleural injection, as well as intramuscular and subcutaneous injection. For assessing human health effects from occupational exposure, the most relevant route is inhalation. However, as a whole, there were very few inhalation studies. In addition to inhalation studies, OSHA is also relying on intrabronchial implantation and intratracheal instillation studies for hazard identification because these studies examine effects directly administered to the respiratory tract, the primary target organ of concern, and they give insight into the relative potency of different Cr(VI) compounds. In comparison to studies examining inhalation, intrabronchial implantation, and intratracheal instillation, studies using subcutaneous injection and intramuscular administration of Cr(VI) compounds were of lesser significance but were still considered for hazard identification.

In its evaluation, OSHA took into consideration the exposure regimen and experimental conditions under which the experiments were performed, including the exposure level and duration; route of administration; number, species, strain, gender, and age of the experimental animals; the inclusion of appropriate control groups; and consistency in test results. Some studies were not included if they did not contribute to the weight of evidence, lacked adequate documentation, were of poor quality, or were less relevant to occupational exposure conditions (*e.g.*, some intramuscular injection studies).

The summarized animal studies are organized by Cr(VI) compound in order of water solubility (*i.e.,* compounds that are considered highly soluble in water, followed by those considered slightly soluble in water, and then those considered insoluble in water) since it has been suggested that solubility may be an important factor in determining the carcinogenic potency of Cr(VI) compounds (Ex 35–47). Solubility characteristics described in this section are based on those cited in the IARC Monograph (as cited in Ex. 35–43, pages 56–59).

a. *Highly Water Soluble Cr(VI) Compounds.* Multiple animal carcinogenicity studies have been conducted on highly water soluble sodium dichromate and chromic acid. The key studies are summarized in Table VI–7.

TABLE VI–7.—SUMMARY OF SELECTED CARCINOGENICITY STUDIES IN EXPERIMENTAL ANIMALS ADMINISTERED HEXAVALENT CHROMIUM—HIGHLY WATER SOLUBLE CHROMATES

Compound	Route	Sex/species/strain (# in exposed groups)	Dose administered [1] and observation periods	Tumor incidence	Reference/exhibit #
Chromic acid (Chromium trioxide).	Inhalation	Female ICR mice (50 per exposed group.	3.6 mg Cr(VI)/m^3 for 30 min per day, 2 d/wk up to 12 mo. Histopatholoical evaluation at periods up to 18 mo.	—Lung tumors: 7/48 vs 2/20 for control. —5 benign adenomas and 2 adenocarcinomas.	Adachi *et al.* (1986, Ex. 35–26–1).
	Inhalation	Female C57BL mice (23 examined at 12 mo; 20 examined at 18 mo).	1.8 mg Cr(VI)/m^3 120 min 2 x week for 12 months; Histopatholoical evaluation at 12 and 18 mo.	Nasal papilloma: 6/20 (<0.05) at 18 mo; Lung adenoma: 1/20 (NS) at 18 mo.	Adachi (1987, Ex. 35–219).
	Intrabronchial	Male/female Porton-Wistar rats (50 per exposed group).	1.0 mg Cr(VI) as single dose mixed w cholesterol in steel pellet and evaluated at 2 years.	Bronchial carcinoma (M/F combined): 2/100 (N.S.).	Levy *et al.* (1986, Ex. 11–2).
Sodium dichromate	Inhalation	Male Wistar rats (20 per exposed group).	0.025, 0.050 and 0.10 mg Cr(VI)m^3 22–23 hr/day, 7 d/wk for 18 months; evaluated at up to 30 months.	Lung tumors: 0.025 mg/m^3—0/18; 0.05 mg/m^3—0/018; 0.1 mg/m^3—3/19(NS).	Glaser *et al.* (1986, Ex. 10–11).
	Intrabronchial	Male/female Porton-Wistar rats (50 per exposed group).	0.8 mg Cr(VI) as a single dose mixed w cholesterol in steel pellet and evaluated at 2 years.	Bronchial carcinoma (M/F combined): 1/100 (NS).	Levy *et al.* (1986, 11–2).
	Intratracheal	Male/female Sprague Dawley rats (40 per exposed group).	5 x weekly: 0.0034, 0.017, 0.086 mg Cr(VI)/kg bw for 30 mo; 1 x weekly: 0.017, 0.086, 0.43 mg Cr(VI)/kg bw for 30 mo.	Lung tumors (M/F combined)— 5 x weekly: 0/80 in all groups; 1 x weekly: 0.017 mg/kg–0/80; 0.086 mg/kg–1/80; 0.043 mg/kg–14/80 (p<0.01).	Steinhoff *et al.* (1986, Ex. 11–7).

[1] Doses calculated and recorded as mg of Cr(VI), rather than specific chromate compound, where possible.
Not Statistically Significant—NS
Male/Female M/F.

Sodium dichromate. Glaser *et al.* exposed male Wistar rats to aerosolized sodium dichromate by inhalation for 22–23 hours per day, seven days per week for 18 months (Exs. 10–10; 10–11). The rats were held for an additional 12 months at which point the study was terminated. Lung tumor incidences among groups exposed to 25, 50, and 100 µg Cr(VI)/m^3 were 0/18, 0/18, and 3/19, respectively, vs. 0/37 for the control animals. Histopathology revealed one adenocarcinoma and two adenomas in the highest group. The slightly elevated tumor incidence at the highest dose was not statistically significant. As noted by IARC, a small number of animals (20 per group) were used in this study. In addition, the administered doses used in this study were fairly low, such that the maximum tolerated dose (i.e., the maximum dose level that does not lead to moderate reduction in body weight gain) may not have been achieved. Together, these factors limit the interpretation of the study.

In an analysis prepared by Exponent and submitted by the Chrome Coalition in response to OSHA's RFI, Exponent stated that ''inhalation studies of Glaser *et al.* support a position that exposures to soluble Cr(VI) at concentrations at least as high as the current PEL (i.e., 52 μg/m^3) do not cause lung cancer'' (Ex. 31–18–1, page 2). However, it should be noted that the Glaser *et al.* studies found that 15% (3/19) of the rats exposed to an air concentration just above the current PEL developed lung tumors, and that the elevated tumor incidence was not statistically significant in the highest dose group because the study used a small number of animals. OSHA believes the Glaser study lacks the statistical power to state with sufficient confidence that Cr(VI) exposure does not cause lung cancer at the current PEL, especially when given the elevated incidence of lung tumors at the next highest dose level.

Steinhoff *et al.* studied the carcinogenicity of sodium dichromate in Sprague-Dawley rats (Ex. 11–7). Forty male and 40 female Sprague-Dawley rats were divided into two sets of treatment groups. In the first set, doses of 0.01, 0.05 or 0.25 mg/kg body weight in 0.9% saline were instilled intratracheally five times per week. In the second set of treatment groups, 0.05, 0.25 or 1.25 mg/kg body weight in 0.9% saline doses were instilled intratracheally once per week. Duration of exposure in both treatment groups was 30 months. The total cumulative dose for the lowest treatment group of animals treated once per week was the same as the lowest treatment group treated five times per week. Similarly, the medium and high dose groups treated once per week had total doses equivalent to the medium and high dose animals treated five times per week, respectively. No increased incidence of lung tumors was observed in the animals dosed five times weekly. However, in the animals dosed once per week, tumor incidences were 0/80 in control animals, 0/80 in 0.05 mg/kg exposure group, 1/80 in 0.25 mg/kg exposure group and 14/80 in 1.25 mg/kg exposure group ($p < 0.01$). The tumors were malignant in 12 of the 14 animals in the 1.25 mg/kg exposure group. The authors believe that the results of this study suggest that the dose-rate for sodium dichromate is a significant factor in its carcinogenic potency and that limiting occasional high dose exposures may be critical to reducing the risk of carcinogenicity in humans occupationally exposed to sodium dichromate.

In separate but similar studies, Levy *et al.* and Levy and Venitt implanted stainless steel mesh pellets filled with a single dose of 2 mg sodium dichromate (0.80 mg Cr(VI)) mixed 50:50 with cholesterol in the bronchi of male and female Porton-Wistar rats (Exs. 11–2; 11–12). Control groups (males and females) received blank pellets or pellets loaded with cholesterol. The rats were observed for two years. Levy *et al.* and Levy and Venitt reported a bronchial tumor incidence of 1/100 and 0/89, respectively, for exposed rats. However, the latter study reported a statistically significant increase in squamous metaplasia, a lesion believed capable of progressing to carcinoma, among exposed rats when compared to unexposed rats. The earlier Levy *et al.* study did not report the incidence of squamous metaplasia. There were no bronchial tumors or squamous metaplasia in any of the control animals and no significant increases in lung tumors were observed in the two studies.

In the Hueper study, 26 rats (sex, age, and strain not specified) were given intrapleural implantation for 27 months (Ex. 10–4). Dosage was not specified. No significant increases in tumor incidence were observed in rats exposed to sodium dichromate or in the control group (0/26 vs. 0/34 in control).

Chromic acid (Chromium trioxide). In a study by Adachi et al, ICR/JcI mice were exposed by inhalation to 3.63 mg/m^3 for 30 minutes per day, two days per week for up to 12 months (Ex. 35–26–1). The mice were observed for an additional six months. The authors used a miniaturized chromium electroplating system to generate chromic acid for the study. The authors found there were elevations in lung adenomas at 10–14 months (3/14 vs. 0/10) and lung adenocarcinomas at 15–18 months (2/19 vs. 0/10), but the results were not statistically significant. Statistically significant increases in nasal papillomas were observed in another study by Adachi *et al.*, in which 43 C57B1 mice were exposed by inhalation to 1.81 mg/m^3 chromic acid for 120 min per day, two days per week for up to 12 months (Ex. 35–26). At 18 months, the tumor incidence was 6/20 in exposed animals vs. 0/20 in the control animals ($p < 0.05$).

In separate but similar studies, Levy *et al.* and Levy and Venitt, using similar exposure protocol, conducted bronchial implantation experiments in which 100 male and female Porton-Wistar rats were dosed with single intrabronchial implantations of 2 mg chromic acid (1.04 mg Cr(VI)) mixed 50:50 with cholesterol in stainless steel mesh pellets (Exs. 11–2; 11–12). The authors found no statistically significant increases in lung tumors, although Levy *et al.* found a bronchial carcinoma incidence of 2/100 in exposed rates compared with 0/100 in control rats. Levy and Venitt found a bronchial carcinoma incidence of 1/100 accompanied by a statistically significant increase in squamous metaplasia, a lesion believed capable of progressing to carcinoma. There was no statistically significant increase in the incidence of squamous metaplasia in control rats or rats treated with Cr(III) compounds in the same study. This finding suggests that squamous metaplasia is specific to Cr(VI) and is not evoked by a non-specific stimuli, the implantation procedure itself, or a treatment with Cr(III) containing materials. The incidence of squamous metaplasia was not investigated in the 1986 Levy *et al.* study.

Similar to Levy *et al.* and Levy and Venitt studies, Laskin *et al.* gave a single intrabronchial implantation of 3–5 mg chromic acid mixed 50:50 with cholesterol in stainless steel mesh pellets to 100 male and female Porton-Wistar rats (Ex. 10–1). The rats were observed for 2 years. No tumors were identified in the treated or control animals (0/100 vs. 0/24).

Potassium chromate. No studies were found that administered this compound by way of the respiratory tract. Borneff *et al.* exposed mice to potassium chromate in drinking water for three generations at a dose of 9 mg Cr(VI)/kg/day (as cited in ATSDR, Ex. 35–41, Pages 108 and 345). In treated mice, two of 66 females developed forestomach carcinoma and 10/66 females and 1/35 males developed forestomach papillomas. The controls also developed forestomach papillomas (2/79 females, 3/47 males), but no carcinomas were observed. The incidence of forestomach tumors was not statistically significant.

b. *Slightly Water Soluble Cr(VI) Compounds.* Animal carcinogenicity studies have been conducted on slightly water soluble calcium chromate and strontium chromate. The key studies are summarized in Table VI–8.

TABLE VI–8: SUMMARY OF SELECTED CARCINOGENICITY STUDIES IN EXPERIMENTAL ANIMALS ADMINISTERED HEXAVALENT CHROMIUM—SLIGHTLY WATER SOLUBLE CHROMATES

Compound	Route	Sex/species/strain (# in exposed groups)	Dose administered [1] and observation periods	Tumor incidence	Reference/exhibit
Calcium chromate	Inhalation	Male/female C57BL/6 mice (136 per group).	4.3 mg Cr(VI)/m^3, 5 hr/d, 5d/wk over animal lifetime.	Lung adenoma (M/F combined): 14/272 vs 5/272 for controls.	Nettesheim *et al.* (1971, Ex. 10–8).
	Intrabronchial	Male/female Porton-Wistar rats (100 per group).	0.67 mg Cr(VI) as a single dose mixed w cholesterol in steel pellet and evaluated at 2 years.	Bronchial carcinoma (M/F combined): 25/100 (p<0.01).	Levy *et al.* (1986, Ex. 11–2).
	Intratracheal	Male/female Sprague Dawley rats (40 per group).	5 x weekly: 0.083 mg Cr(VI)/kg bw for 30 mo; 1 x weekly: 0.41.mg Cr(VI)/kg bw for 30 mo.	Lung tumors (M/F combined)—5 x weekly: 0.083 mg/kg-6/80 (p<0.01); 1 x weekly: 0.41 mg/kg-13/80 (p<0.01).	Steinhoff *et al.* (1986, Ex. 11–7).
	Intratracheal	Male Sprague Dawley rats (50 per exposed group).	0.67 mg Cr(VI)/kg bw x 13 installations over 20 wks and evaluated at 2 to 2.5 yr.	Lung tumors: 1/44 (NS)	Snyder *et al.* (1997, Ex. 31–18–12).
Strontium chromates (two different compounds).	Intrabronchial	Male/female Porton-Wistar rats (50 per exposed group).	0.48 mg Cr(VI) as a single dose mixed w cholesterol in steel pellet and evaluated at 2 years.	Bronchial carcinoma (M/F combined): 43/99 & 62/99 (p<0.01).	Levy *et al.* (1986, Ex. 11–2).

[1] Doses calculated and recorded as mg of Cr(VI), rather than specific chromate compound, where possible.
Not Statistically significant—NS.
Male/Female—M/F.

Calcium chromate. Nettesheim *et al.* conducted the only available inhalation carcinogenicity study with calcium chromate showing borderline statistical significance for increased lung adenomas in C57B1/6 mice exposed to 13 mg/m^3 for 5 hours per day, 5 days per week over the life of the mice. The tumor incidences were 6/136 in exposed male mice vs. 3/136 in control male mice and 8/136 in exposed female mice vs. 2/136 in control female mice (Ex. 10–8).

Steinhoff *et al.* observed a statistically significant increase in lung tumors in Sprague-Dawley rats exposed by intratracheal instillation to 0.25 mg/kg body weight calcium chromate in 0.9% saline five times weekly for 30 months (Ex. 11–7). Tumors were found in 6/80 exposed animals vs. 0/80 in unexposed controls (p<0.01). Increased incidence of lung tumors was also observed in those rats exposed to 1.25 mg/kg calcium chromate once per week (14/80 vs. 0/80 in controls) for 30 months. At the highest dose, the authors observed 11 adenomas, one adenocarcinoma, and two squamous carcinomas. The total administered doses for both groups of dosed animals (1×1.25 mg/kg and 5×0.25 mg/kg) were equal, but the tumor incidence in the rats exposed once per week was approximately double the incidence in rats exposed to the same weekly dose divided into five smaller doses. The authors suggested that the dose-rate for calcium chromate compounds may be important in determining carcinogenic potency and that limiting higher single exposures may offer greater protection against carcinogenicity than reducing the average exposure alone.

Snyder *et al.* administered Cr(VI)-contaminated soil of defined aerodynamic diameter (2.9 to 3.64 micron) intratracheally to male Sprague-Dawley rats (Ex. 31–18–12). For the first six weeks of treatment, the rats were instilled with weekly suspensions of 1.25 mg of material per kg body weight, followed by 2.5 mg/kg every other week, until treatments were terminated after 44 weeks. The investigation included four exposure groups: Control animals (50 rats), rats administered Cr(VI)-contaminated soil (50 rats), rats administered Cr(VI)-contaminated soil supplemented with calcium chromate (100 rats), and rats administered calcium chromate alone (100 rats). The total Cr(VI) dose for each group was: Control group (0.000002 mg Cr(VI)/kg), soil alone group (0.324 mg Cr(VI)/kg), soil plus calcium chromate group (7.975 mg Cr(VI)/kg), and calcium chromate alone group (8.700 mg Cr(VI)/kg). No primary tumors were observed in the control group or the chromium contaminated soil group. Four primary tumors of the lung were found in the soil plus calcium chromate group and one primary lung tumor was observed in the group treated with calcium chromate alone; however, these incidences did not reach statistical significance.

In the analysis submitted to OSHA by the Chrome Coalition, Exponent stated that the ''intratrachael instillation data of Steinhoff *et al.* 1986 and Snyder *et al.* 1997 indicates there is a likely threshold for lung cancer'' (Ex. 31–18–1, page 2). OSHA believes the results of the Steinhoff *et al.* 1986 study show that the rate at which Cr(VI) is administered may be an important determinant for carcinogenic potency and thus useful for hazard identification purposes. However, in accordance with the Agency's long standing cancer policy, OSHA believes it is inappropriate to establish a threshold or ''no effect'' level of exposure to a carcinogen (*see* 29 CFR 1990.143). Moreover, the Snyder 1997 study, in particular, used contaminated soil samples and an irregular dosing protocol, creating additional complexities in relating the results to workplace inhalation exposures.

Statistically significant increases in the incidence of bronchial carcinoma in rats exposed to calcium chromate through intrabronchial instillation were reported by Levy *et al.* (Ex. 11–2) and Levy and Venitt (Ex. 11–12). These studies, using a similar protocol, implanted a single dose of 2 mg calcium chromate (0.67 mg Cr(VI)) mixed 50:50 with cholesterol in stainless steel pellets into the bronchi of Porton-Wistar rats. Levy *et al.* and Levy and Venitt found bronchial carcinoma incidences of 25/100 and 8/84, respectively, following a 24-month observation. The increased incidences were statistically significant when compared to the control group. Levy and Venitt also reported statistically significant increases in squamous metaplasia in the calcium chromate-treated rats (Ex. 11–12).

Laskin *et al.* observed 8/100 tumors in rats exposed to a single dose of 3–5 mg calcium chromate mixed with cholesterol in stainless steel mesh

pellets implanted in the bronchi (Ex. 10–1). Animals were observed for a total of 136 weeks. The sex, strain, and species of the rats were not specified in the study. Tumor incidence in control animals was 0/24. Although tumor incidence did not reach statistical significance in this study, OSHA agrees with IARC that the incidences are due to calcium chromate itself rather than background variation.

Strontium chromate. Strontium chromate was tested by intrabronchial implantation and intrapleural injection. In a study by Levy *et al.,* two strontium chromate compounds mixed 50:50 with cholesterol in stainless steel mesh pellets were administered by intrabronchial instillation of a 2 mg (0.48 mg Cr(VI)) dose into 100 male and female Porton-Wistar rats (Ex. 11–2). Animals were observed for up to 136 weeks. The strontium chromate compounds induced bronchial carcinomas in 43/99 (Sr, 42.2%; CrO_4, 54.1%) and 62/99 rats (Sr, 43.0%; Cr, 24.3%), respectively, compared to 0/100 in the control group. These results were statistically significant. The strontium chromates produced the strongest carcinogenic response out of the 20 Cr(VI) compounds tested by the intrabronchial implantation protocol.

In the study by Hueper, strontium chromate was administered by intrapleural injection (doses unspecified) lasting 27 months (Ex. 10–4). Local tumors were observed in 17/28 treated rats vs. 0/34 for the untreated rats. Although the authors did not examine the statistical significance of tumors, the results clearly indicate a statistical significance.

c. *Water Insoluble Cr(VI) Compounds.* There have been a number of animal carcinogenicity studies involving implantation or injection of principally water insoluble zinc, lead, and barium chromates. The key studies are summarized in Table VI–9.

TABLE VI–9.—SUMMARY OF SELECTED CARCINOGENICITY STUDIES IN EXPERIMENTAL ANIMALS ADMINISTERED HEXAVALENT CHROMIUM—WATER INSOLUBLE CHROMATES

Compound	Route	Sex/species/strain (# in exposed groups)	Dose administered [1] and observation periods	Tumor incidence	Reference/exhibit #
Zinc chromates (three different compounds).	Intrabronchial	Male/female Porton-Wistar rats (50 per exposed group).	0.42 to 0.52 mg Cr(VI) as a single dose mixed w cholesterol in steel pellet and evaluated at 2 years.	Bronchial carcinoma (M/F combined): 3/61 ($p<0.05$), 5/100 ($p<0.05$), 3/100 ($p=0.07$).	Levy *et al.* (1986, Ex. 11–2); Levy and Venitt (1986, Ex. 11–12).
Zinc tetroxychromate	Intrabronchial	Male/female Porton-Wistar rats (50 per exposed group).	0.18 mg Cr(VI) as a single dose mixed w cholesterol in steel pellet and evaluated at 2 years.	Bronchial carcinoma (M/F combined): 1/100 (NS).	Levy *et al.* (1986, Ex. 11–2).
Lead chromates (seven different compounds).	Intrabronchial	Male/female Porton-Wistar rats (50 per exposed group).	0.25 to 0.32 mg Cr(VI) as single dose mixed w cholesterol in steel pellet and evaluated at 2 years.	Bronchial carcinoma (M/F combined): 0–1/100 (N.S.).	Levy *et al.* (1986, Ex. 11–2).
Lead chromates (three different compounds).	Subcutaneous	Male/female Sprague Dawley rats (20 per exposed group).	1.5 to 4.8 mg Cr(VI) as a single dose in water and evaluated after 2 years.	Sarcomas at injection site (M/F combined): 26–36/40 vs 0/40 for controls.	Maltoni *et al.* (1974, Ex. 8–25); Maltoni (1976, Ex. 5–2).
Lead chromate	Intramuscular	Male/female Fischer 344 rats (25 per exposed group).	1.29 mg Cr(VI) in trioctyanoin 1 x mo for 9 mo and evaluated at up to 2 yr.	Sarcomas at injection site (M/F combined): 31/47 vs 0/44 for controls.	Furst *et al.* (1976, Ex. 10–2).
		Female NIH-Swiss mice (25 per exposed group).	0.72 mg Cr(VI) in trioctyanoin 1 x mo for 4 mo and evaluated at up to 2 yr.	Sarcomas at injection site: 0/22 (NS).	
Barium chromate	Intrabronchial	Male/female Porton-Wistar rats (50 per exposed group).	0.37 mg Cr(VI) as a single dose mixed w cholesterol in steel pellet and evaluated at 2 years.	Bronchial carcinoma (M/F combined): 0/100 (NS).	Levy *et al.* (1986, Ex. 11–2).

[1] Doses calculated and recorded as mg of Cr(VI), rather than specific chromate compound, where possible.
Not Statistically significant—NS.
Male/Female—M/F.

Zinc chromate compounds. Animal studies have been conducted to examine several zinc chromates that range from water insoluble to slightly water soluble compounds depending on the form and composition. In separate, but similarly conducted studies, Levy *et al.* and Levy and Venitt studied two water-insoluble compounds (zinc chromate—lW and zinc tetroxychromate) and two slightly water-soluble compounds (zinc chromate—Norge composition and zinc potassium chromate) (Exs. 11–2; 11–12). Two milligrams of the compounds were administered by intrabronchial implantation to 100 male and female Porton-Wistar rats. The slightly water soluble zinc potassium chromate (0.52 mg Cr(VI)) produced a bronchial tumor incidence of 3/61 which was statistically significant ($p<0.05$) when compared to a control group (Ex. 11–12). There was also a statistically significant increase in bronchial tumors in rats receiving water-insoluble zinc chromate—lW (5/100; $p=0.04$). The bronchial tumor incidence with slightly water soluble zinc chromate—Norge (3/100; $p= 0.068$) and water-insoluble zinc tetroxychromate (1/100) were not statistically significant when compared to a control group. Zinc potassium chromate (slightly water soluble) was administered at doses of 0.42 mg Cr(VI), zinc chromate—Norge (slightly water soluble) was administered at doses of 0.45 mg Cr(VI), and zinc tetroxychromate (insoluble in water) was administered at doses of 0.18 mg Cr(VI). These studies show that insoluble to slightly water soluble zinc chromate compounds may produce statistically significant elevated incidences of tumors in rats.

Basic potassium zinc chromate (slightly water soluble) was administered to mice, guinea pigs and rabbits via intratracheal instillation (Ex. 35–46). Sixty-two Strain A mice were given six injections of 0.03 ml of a 0.2% saline suspension of the zinc chromate at six week intervals and observed until death. A statistically significant increase in tumor incidence was observed in exposed animals when compared to controls (31/62 vs. 7/18). Statistically significant effects were not observed

among guinea pigs or rabbits. Twenty-one guinea pigs (sex and strain not given) received six injections of 0.3 ml of a 1% suspension of zinc chromate at three monthly intervals and observed until death. Results showed pulmonary adenomas in only 1/21 exposed animals vs. 0/18 in controls. Seven rabbits (sex and strain not given) showed no increase in lung tumors when given 3–5 injections of 1 ml of a saline suspension of 10 mg zinc chromate at 3-month intervals. However, as noted by IARC, the small numbers of animals used in the guinea pig and rabbit experiments (as few as 13 guinea pigs and 7 rabbits per group) limit the power of the study to detect increases in cancer incidence.

Hueper found that intrapleural injection of slightly water soluble zinc yellow (doses were unspecified) resulted in statistically significant increases in local tumors in rats (sex, strain, and age of rat unspecified; dose was unspecified). The incidence of tumors in exposed rats was 22/33 vs. 0/34 in controls (Ex. 10–4).

Maltoni *et al.* observed increases in the incidence of local tumors after subcutaneous injection of slightly water soluble zinc yellow in 20 male and 20 female Sprague-Dawley rats (statistical significance was not evaluated) (Ex. 8–37). Tumor incidences were 6/40 in 20% CrO_3 dosed animals at 110 weeks and 17/40 in 40% CrO_3 dosed animals at 137 weeks compared to 0/40 in control animals.

Lead chromate and lead chromate pigments. Levy *et al.* examined the carcinogenicity of lead chromate and several lead chromate-derived pigments in 100 male and female Porton-Wistar rats after a single intrabronchial implantation followed by a two year observation period (Ex. 11–12). The rats were dosed with two mg of a lead chromate compound and lead chromate pigments, which was mixed 50:50 with cholesterol in stainless steel mesh pellets and implanted in the bronchi of experimental animals. The lead chromate and lead chromate pigment compositions consisted of the following: lead chromate (35.8% CrO_4; 0.32 mg Cr(VI)), primrose chrome yellow (12.6% Cr; 0.25 mg Cr(VI)), molybdate chrome orange (12.9% Cr; 0.26 mg Cr(VI)), light chrome yellow (12.5% Cr; 0.25 mg Cr(VI)), supra LD chrome yellow (26.9% CrO_3; 0.28 mg Cr(VI)), medium chrome yellow (16.3% Cr; 0.33 mg Cr(VI)) and silica encapsulated medium chrome yellow (10.5% Cr; 0.21 mg Cr(VI)). No statistically significant tumors were observed in the lead chromate group compared to controls (1/98 vs. 0/100), primrose chrome yellow group (1/100 vs. 0/100), and supra LD chrome yellow group (1/100 vs. 0/100). The authors also noted no tumors in the molybdate chrome orange group, light chrome yellow group, and silica encapsulated medium chrome yellow group.

Maltoni (Ex. 8–25), Maltoni (Ex. 5–2), and Maltoni *et al.* (Ex. 8–37) examined the carcinogenicity of lead chromate, basic lead chromate (chromium orange) and molybdenum orange in 20 male and 20 female Sprague-Dawley rats by a single subcutaneous administration of the lead chromate compound in water. Animals were observed for 117 to 150 weeks. After injection of 30 mg lead chromate, local injection site sarcomas were observed in 26/40 exposed animals vs. 0/60 and 1/80 in controls. Although the authors did not examine the statistical significance of sarcomas, the results clearly indicate a statistical significance. Animals injected with 30 mg basic lead chromate (chromium orange) were found to have an increased incidence of local injection site sarcomas (27/40 vs. 0/60 and 1/80 in controls). Animals receiving 30 mg molybdenum orange in 1 ml saline were also found to have an increased incidence of local injection site sarcomas (36/40 vs. 0/60 controls).

Carcinogenesis was observed after intramuscular injection in a study by Furst *et al.* (Ex. 10–2). Fifty male and female Fischer 344 rats were given intramuscular injections of 8 mg lead chromate in trioctanoin every month for nine months and observed up to 24 months. An increase in local tumors at the injection site (fibrosarcomas and rhabdomyosarcomas) was observed (31/47 in treated animals vs. 0/22 in controls). These rats also had an increased incidence of renal carcinomas (3/23 vs. 0/22 in controls), but IARC noted that the renal tumors may be related to the lead content of the compound. In the same study, 3 mg lead chromate was administered to 25 female NISH Swiss weanling mice via intramuscular injection every 4 months for up to 24 months. In the exposed group, the authors observed three lung alveologenic carcinomas after 24 months of observation and two lymphomas after 16 months of observation. Two control groups were used: an untreated control group (22 rats) and a vehicle injected control group (22 rats). The authors noted one alveologenic carcinoma and one lymphoma observed in each control group.

In response to OSHA's RFI, the Color Pigments Manufacturers Association (CPMA) stated that the lack of carcinogenic response in two studies (Levy *et al.* 1986 and Furst *et al.* 1976) upon exposure to lead chromate and lead chromate pigments in animals indicate these Cr(VI) compounds are not carcinogenic to workers (Ex. 31–15). As described above, the results of the Levy *et al.* 1986 study showed little tumor development (0–1 tumor observed per 100 rats studied in each experiment) after receiving a single dose of 2 mg of lead chromate or a lead chromate compound by an intrabronchial implantation procedure in which the compounds were imbedded in a metal mesh mixed with cholesterol (Ex. 11–2). The total administered dose of the Levy *et al.* study was relatively low at 0.67 mg Cr(VI)/kg when administered only one time (body weight of the rat was around 0.5 kg). A small, single total dose (*e.g.*, 1.6 mg Cr(VI)/kg) of sodium dichromate implanted in the lung also did not result in tumors. However, repeated weekly intratracheal instillations of a lower dose level (0.43 mg Cr(VI)/kg) of sodium dichromate over 30 months for a cumulative total dose of about 56 mg Cr(VI)/kg produced a 17.5 percent lung cancer incidence. Thus, a greater *total* dose of lead chromate instilled in the respiratory tract may also produce a significant tumor incidence. The lack of tumors in the Levy *et al.* study may also have resulted from the inability of water insoluble lead chromate to leach out of the highly non-polar cholesterol environment and gain entry into target lung cells. OSHA, therefore, does not believe that the findings of this study establish that lead chromate and lead chromate pigments are not carcinogenic. OSHA does not believe the results of the Furst *et al.* study show a lack of carcinogenic effect. The study found a 66 percent tumor incidence at the site of injection after multiple intramuscular administrations of lead chromate in rats (Ex. 10–2). Although the route of exposure is not comparable to that found in occupational settings, the carcinogenic potential of lead chromate is supported by the results of several studies showing that pigment workers exposed to lead chromate have significantly elevated lung cancer mortality (see section V.B.2). Several short-term tests have also linked lead chromate with genotoxicity and neoplastic transformation (see section VI.B.8).

Barium chromate. In the studies reviewed by IARC, barium chromate was tested in rats via intrabronchial, intrapleural and intramuscular administration. No excess lung or local tumors were observed (Ex. 11–2; Ex. 10–4; Ex. 10–6).

d. *Summary.* Several Cr(VI) compounds produced tumors in

laboratory animals under a variety of experimental conditions using different routes of administration. The animals were generally given the test material(s) by routes other than inhalation (*e.g.*, intratracheal administration, intramuscular injection, intrabronchial implantation, and subcutaneous injection). Although the route of administration may have differed from that found in an occupational setting, these studies have value in the identification of potential health hazards associated with Cr(VI) and in assessing the relative potencies of various Cr(VI) compounds.

OSHA believes that the results from Adachi *et al.* (Ex. 35–26–1), Adachi *et al.* (Ex. 35–26), Glaser *et al.* (Ex. 10–4), Glaser *et al.* (Ex. 10–10), Levy *et al.* (Ex. 11–2), Steinhoff *et al.* (Ex. 11–7), and Snyder *et al.* (Ex. 31–18–12) studies provide valuable insight on the carcinogenic potency of Cr(VI) compounds in laboratory animals. Total dose administered, dose rate, amount of dosage, dose per administration, number of times administered, exposure duration and the type of Cr(VI) compound are major influences on the observed tumor incidence in animals. It was found that slightly water soluble calcium, strontium, and some zinc chromates showed the highest incidence of lung tumors, as indicated in the results of the Steinhoff, Snyder, and Levy studies, even when compared to similar doses of the more water soluble sodium chromates and chromic acid compounds. The highly insoluble lead chromates did not produce lung tumors by the intrabronchial implantation procedure but did produce tumors by subcutaneous injection and intramuscular injection.

8. Mechanistic Considerations

Mechanistic information can provide insight into the biologically active form(s) of chromium, its interaction with critical molecular targets, and the resulting cellular responses that trigger neoplastic transformation. There has been considerable scientific study in recent years of Cr(VI)-initiated cellular and molecular events believed to impact development of respiratory carcinogenesis. Much of the research has been generated using *in vitro* techniques, cell culture systems, and animal administrations. The early mechanistic data were reviewed by IARC in 1990 (Ex. 35–43). More recent reviews have been done by Singh *et al.* in 1998 (Ex. 35–149), ATSDR in 2000 (Ex. 35–41), and K.S. Crump Group in 2000 (Ex. 35–47).

Recent experimental research has identified several biological steps critical to the mode of action by which Cr(VI) transforms normal lung cells into a neoplastic phenotype. These are: (a) Cellular uptake of Cr(VI) and its extracellular reduction, (b) intracellular Cr(VI) reduction to produce biologically active products, (c) damage to DNA, and (d) activation of signaling pathways in response to cellular stress. Each step will be described in detail below.

a. *Cellular Uptake and Extracellular Reduction.* The ability of different Cr(VI) particulate forms to be taken up by the bronchoalveolar cells of the lung is an essential early step in the carcinogenic process. Particle size and solubility are key physical factors that influence uptake into these cells. Large particulates (>10 μm) are generally deposited in the upper nasopharygeal region of the respiratory tract and do not reach the bronchoalveolar region of the lungs. Smaller Cr(VI) particulates will increasingly reach these lower regions and come into contact with target cells.

Once deposited in the lower respiratory tract, solubility of Cr(VI) particulates becomes a major influence on disposition. Aqueous Cr(VI), such as sodium chromate and chromic acid, rapidly dissolves in the fluids lining the lung epithelia and can be taken up by lung cells via facilitated diffusion mediated by sulfate/phosphate anion transport channels (Ex. 35–148). This is because Cr(VI) exists in a tetrahedral configuration as a chromate oxyanion similar to the physiological anions, sulfate and phosphate (Ex. 35–231). Using cultured human epithelial cells, Liu *et al.* showed that soluble Cr(VI) uptake was time- and dose-dependant over a range of 1 to 300 μM in the medium with 30 percent of the Cr(VI) transported into the cells within two hours and 67 percent at 16 hours at the lowest concentration (Ex. 31–22–18).

Aqueous insoluble Cr(VI) particulates do not readily dissolve into epithelial lining fluids of the bronchoalveolar region. This has led to claims that insoluble chromates, such as lead chromate pigments, are not bioavailable and, therefore, are unable to cause carcinogenesis (Ex. 31–15). However, several scientific studies indicate that insoluble Cr(VI) particulates can come in close contact with the bronchoalveolar epithelial cell surface, allowing enhanced uptake into cells. Wise *et al.* showed that respirable lead chromate particles adhere to the surface of rodent cells in culture causing cell-enhanced dissolution of the chromate ion as well as phagocytosis of lead chromate particles (Exs. 35–68; 35–67). The intracellular accumulation was both time- and dose-dependant. Cellular uptake resulted in damage to DNA, apoptosis (i.e., form of programmed cell death), and neoplastic transformation (Ex. 35–119). Singh *et al.* showed that treatment of normal human lung epithelial cells with insoluble lead chromate particulates (0.4 to 2.0 μg/cm^2) or soluble sodium chromate (10 μM) for 24 hours caused Cr(VI) uptake, Cr-DNA adduct formation, and apoptosis (Ex. 35–66). The proximate genotoxic agent in these cell systems was determined to be the chromate rather than the lead ions (Ex. 35–327). Elias *et al.* reported that cell-enhanced particle dissolution and uptake was also responsible for the cytotoxicity and neoplastic transformation in Syrian hamster embryo cells caused by Cr(VI) pigments, including several complex industrial chrome yellow and molybdate orange pigments (Ex. 125).

Reduction to the poorly permeable Cr(III) in the epithelial lining fluid limits cellular uptake of Cr(VI). Ascorbic acid and glutathione (GSH) are believed to be the key molecules responsible for the extracellular reduction. Cantin *et al.* reported high levels of GSH in human alveolar epithelial lining fluid and Susuki *et al.* reported significant levels of ascorbic acid in rat lung lavage fluids (Exs. 35–147; 35–143). Susuki and Fukuda studied the kinetics of soluble Cr(VI) reduction with ascorbic acid and GSH *in vitro* and following intratracheal instillation (Ex. 35–90). They reported that the reduction was pseudo-first order (i.e., rate of Cr(VI) reduction appeared to be proportional to metal concentration rather than concentration of reductant) with respect to Cr(VI), with a half-life of just under one minute to several hours. They found the greatest reduction rates with higher levels of reductants. Ascorbic acid was more active than GSH. Cr(VI) reduction was slower *in vivo* than predicted from *in vitro* and principally involved ascorbic acid, not GSH. This research indicates that extracellular Cr(VI) reduction to Cr(III) is variable depending on the concentration and nature of the reductant in the epithelial fluid lining regions of the respiratory tract. De Flora *et al.* determined the amount of soluble Cr(VI) reduced *in vitro* by human bronchiolar alveolar fluid and pulmonary alveolar macrophage fractions over a short period and used these specific activities to estimate an ''overall reducing capacity'' of 0.9–1.8 mg Cr(VI) and 136 mg Cr(VI) per day per individual, respectively (Ex. 35–140).

De Flora, Jones, and others have interpreted the extracellular reduction data to mean that very high levels of Cr(VI) are required to ''overwhelm'' the reductive defense mechanism before target cell uptake can occur and, as

such, impart a ''threshold'' character to the exposure-response (Exs. 35–139; 31–22–7). However, the threshold capacity concept does not consider that facilitated lung cell uptake and extracellular reduction are dynamic and parallel processes that happen concurrently. If their rates are comparable then some cellular uptake of Cr(VI) would be expected, even at levels that do not ''overwhelm'' the reductive capacity. Based on the *in vitro* kinetic data, it would appear that such situations are plausible, especially when concentrations of ascorbic acid are low. Unfortunately, there has been little systematic study of the dose-dependence of Cr(VI) uptake in the presence of physiological levels of ascorbate and GSH using experimental systems that possess active anion transport capability.

Wise *et al.* did study uptake of a single concentration of insoluble lead chromate particles (0.8 μg/cm^2) and soluble sodium chromate (1.3 μM) in Chinese hamster ovary cells co-treated with a physiological concentration (1mM) of ascorbate (Ex. 35–68). They found that the ascorbate substantially reduced, but did not eliminate, chromate ion uptake over a 24 hour period. Interestingly, ascorbate did not affect phagocytic uptake of lead chromate particles, although it eliminated the Cr(VI)-induced clastogenesis (*e.g.*, DNA strand breakage and chromatid exchange) as measured under their experimental conditions.

Singh *et al.* suggested that cell surface interactions with insoluble lead chromate particulates created a concentrated microenvironment of chromate ions resulting in higher intracellular levels of chromium than would occur from soluble Cr(VI) (Ex. 35–149). The evidence for cell membrane mediated uptake of Cr(VI) is consistent with the intratracheal and intrabronchial instillation studies in rodents that show greater carcingenicity with sparingly soluble (*e.g.*, calcium chromate) than insoluble chromate (*e.g.*, lead chromate) particulates and soluble chromates (*e.g.*, sodium chromate) (Ex. 11–2).

Finally, Cr(VI) deposited in the tracheobronchial and alveolar regions of the respiratory tract is cleared by the mucocilliary escalator (soluble and particulate Cr(VI)) and macrophage phagocytosis (particulate Cr(VI) only). In most instances, these clearance processes take hours to days to completely clear Cr(VI) from the lung, but it can take considerably longer for particulates deposited at certain sites. For example, Ishikawa *et al.* showed that some workers had substantial amounts of chromium particulates at the bifurcations of the large bronchii for more than two decades after cessation of exposure (Ex. 35–81). Mancuso reported chromium in the lungs of six chromate production workers who died from lung cancer (as cited in Ex. 35–47). The interval between last exposure to Cr(VI) until autopsy ranged from 15 months to 16 years. Using hollow casts of the human tracheobronchial tree and comparing particle deposition with reported occurrence of bronchogenic tumors, Schlesinger and Lippman were able to show good correlations between sites of greatest deposition and increased incidence of bronchial tumors (Ex. 35–102).

b. *Intracellular Reduction of Cr(VI).* Once inside the cell, the hexavalent chromate ion is rapidly reduced to intermediate oxidation states, Cr(V) and Cr(IV), and the more chemically stable Cr(III). Unlike Cr(VI), these other chromium forms are able to react with DNA and protein to generate a variety of adducts and complexes. In addition, reactive oxygen species (ROS) are produced during the intracellular reduction of Cr(VI) that are also capable of damaging DNA. These reactive intermediates, and not Cr(VI) itself, are considered to be the ultimate genotoxic agents that initiate the carcinogenic process.

After crossing the cell membrane, Cr(VI) compounds can be non-enzymatically converted to Cr(III) by several intracellular reducing factors (Ex. 35–184). The most plentiful electron donors in the cell are GSH, and other thiols, such as cysteine, and ascorbate. Connett and Wetterhahn showed that a Cr(VI)-thioester initially forms in the presence of GSH (Ex. 35–206). A two-phase reduction then occurs with rapid conversion to Cr(V) and glutathionyl radical followed by relatively slower reduction to Cr(III) that requires additional molecules of GSH. Depletion of cellular GSH and other thiols is believed to retard complete reduction of Cr(VI) to Cr(III), allowing buildup of intermediates Cr(V) and Cr(IV). The molecular kinetics of the Cr(VI) to Cr(III) reduction with ascorbate is less well understood but can also involve intermediate formation of Cr(V) and free radicals (Ex. 35–184).

Another important class of intracellular Cr(VI) reductions are catalyzed by flavoenzymes, such as GSH reductase, lipoyl dehydrogenase, and ferredoxin-NADP oxidoreductase. The most prominent among these is GSH reductase that uses NADPH as a cofactor in the presence of molecular oxygen (O_2) to form Cr(V)-NADPH complexes. During the reaction, O_2 undergoes one electron reduction to the superoxide radical (O_2^-) which produces hydrogen peroxide (H_2O_2) through the action of the enzyme superoxide dismutase. The Cr(V)-NADPH can then react with H_2O_2 to regenerate Cr(VI) giving off hydroxyl radicals, a highly reactive oxygen species, by a Fenton-like reaction. It is, therefore, possible for a single molecule of Cr(VI) to produce many molecules of potentially DNA damaging ROS through a repeated reduction/oxidation cycling process. Shi and Dalal used electron spin resonance (ESR) to establish formation of Cr(V)-NADPH and hydroxyl radical in an *in vitro* system (Ex. 35–169; 35–171). Sugiyama *et al.* reported Cr(V) formation in cultured Chinese hamster cells treated with soluble Cr(VI) (Ex.35–133). Using a low frequency ESR, Liu *et al.* provided evidence of Cr(V) formation *in vivo* in mice injected with soluble Cr(VI) (Ex. 35–141–28). Several studies have documented that Cr(VI) can generate Cr(V) and ROS in cultured human lung epithelial cells and that this reduction/oxidation pathway leads to DNA damage, activation of the p53 tumor suppressor gene and stress-induced transcription factor NF–κB, cell growth arrest, and apptosis (Exs. 35–125; 35–142; 31–22–18; 35–135). Leonard *et al.* used ESR spin trapping, catalase, metal chelators, free radical scavengers, and O_2-free atmospheres to show that hydroxyl radical generation involves a Fenton-like reaction with soluble potassium dichromate (Ex. 31–22–17) and insoluble lead chromate (Ex. 35–137) *in vitro*. Liu *et al.* showed that the Cr(IV)/Cr(V) compounds are also able to generate ROS with H_2O_2 in a Fenton reduction/oxidation cycle *in vitro* (Ex. 35–183).

Although most intracellular reduction of Cr(VI) is believed to occur in the cytoplasm, Cr(VI) reduction can also occur in mitochondria and the endoplasmic reticulum. Cr(VI) reduction can occur in the mitochondria through the action of the electron transport complex (Ex. 35–230). The microsomal cytochrome P–450 system in the endoplasmic reticulum also enzymatically reduces Cr(VI) to Cr(V), producing ROS through reduction/oxidation cycling as described above (Ex. 35–171).

c. *Genotoxicity and Damage to DNA.* A large number of studies have examined multiple types of genotoxicity in a wide range of experimental test systems. Many of the specific investigations have been previously reviewed by IARC (Ex. 35–43), Klein (Ex. 35–134), ATSDR (Ex. 35–41), and the K.S. Crump Group (Ex. 35–47) and will only be briefly summarized here.

The body of evidence establishes that both soluble and insoluble forms of Cr(VI) cause structural DNA damage that can lead to genotoxic events such as mutagenisis, inhibition of DNA replication and transcription, and altered gene expression, all of which probably play a role in neoplastic transformation. The reactive intermediates and products that occur from intracellular reduction of Cr(VI) cause a wide variety of DNA lesions. At this time, it is not clear which types of DNA damage are the most critical to the carcinogenic process.

Cr(VI) compounds are mutagenic in most bacterial and mammalian test systems (Ex. 35–118). In the bacterial *Salmonella typhimurium* strains, soluble Cr(VI) caused base pair substitutions at A–T sites as well as frame shift mutations (Ex. 35–161). Nestmann *et al.* also reported forward and frame shift mutations in *Salmonella typhimurium* with insoluble Cr(VI) (Ex. 35–162). Several Cr(VI) compounds have produced mutagenic responses at various genetic loci in mammalian cells (Ex. 12–7). Clastogenic damage, such as sister chromatid exchange and chromosomal aberrations, have also been reported for insoluble Cr(VI) and soluble Cr(VI) (Exs. 35–132; 35–115). Mammalian cells undergo neoplastic transformation following treatment with soluble Cr(VI) or insoluble Cr(VI), including a number of zinc and lead chromate pigments (Exs. 12–5; 35–186).

Genotoxicity has been reported from Cr(VI) administration to animals *in vivo*. Soluble Cr(VI) induced micronucleated erythrocytes in mice following intraperitoneal (IP) administration (Ex. 35–150). It also increased the mutation frequency in liver and bone marrow following IP administration to *lacZ* transgenic mice (Exs. 35–168; 35–163). Izzotti *et al.* reported DNA damage in the lungs of rats exposed to soluble Cr(VI) by intratracheal instillation (Ex. 35–170). Intratracheal instillation of soluble Cr(VI) produced a time- and dose-dependant elevation in mutant frequency in the lung of Big Blue transgenic mice (Ex. 35–174). Oral administration of soluble Cr(VI) in animals did not produce genotoxicity in several studies probably due to route-specific differences in absorption. OSHA is not aware of genotoxicity studies from *in vivo* administration of insoluble Cr(VI).

Studies of chromosomal and DNA damage in workers exposed to Cr(VI) vary in their findings. Some studies reported higher levels of chromosomal aberrations, sister chromatid exchanges, or DNA strand breaks in peripheral lymphocytes of stainless steel welders (Exs. 35–265; 35–160) and electroplaters (Ex. 35–164). Other studies were not able to find excess damage in DNA from the blood lymphocytes of workers exposed to Cr(VI) (Exs. 35–185; 35–167). These reports are difficult to interpret since co-exposure to other genotoxic agents (*e.g.*, other metals, cigarette smoke) likely existed and the extent of Cr(VI) exposures were not known.

Because of the consistent positive response across multiple assays in a wide range of experimental systems from prokaryotic organisms (*e.g.*, bacteria) to human cells *in vitro* and animals *in vivo*, OSHA regards Cr(VI) as an agent able to induce carcinogenesis through a genotoxic mode of action. Both soluble and insoluble forms of Cr(VI) are reported to cause mutagenisis, clastogenesis, and neoplastic transformation. On the other hand, Cr(III) compounds do not easily cause mutations or chromosomal damage in intact cellular systems, presumably due to the inability of Cr(III) to penetrate cell membranes (Exs. 12–7; 35–186).

There has been a great deal of research to identify the types of damage to DNA caused by Cr(VI), the reactive intermediates that are responsible for the damage, and the specific genetic lesions critical to carcinogenesis. It was shown that Cr(VI) was inactive in DNA binding assays with isolated nuclei or purified DNA (Ex. 35–47). However, Cr(III) was able to produce DNA protein cross-links, sister chromatid exchanges, and chromosomal aberrations in an acellular system. Zhitkovich *et al.* showed that incubation of Chinese hamster ovary cells with soluble Cr(VI) produced ternary complexes of Cr(III) cross-linked to cysteine, other amino acids, or glutathione and the DNA phosphate backbone (Ex. 312). Utilizing the pSP189 shuttle vector plasmid, they showed these DNA-Cr(III)-amino acid cross-links were mutagenic when introduced in human fibroblasts (Ex. 35–131).

Another research group showed that plasmid DNA treated with Cr(III) produced intrastrand crosslinks and the production of these lesions correlated with DNA polymerase arrest (Ex. 35–126). The same intrastrand crosslinks and DNA polymerase arrest could also be induced by Cr(VI) in the presence of ascorbate as a reducing agent to form Cr(III) (Ex. 35–263). These results were confirmed in a cell system by treating human lung fibroblasts with soluble Cr(VI), isolating genomic DNA, and demonstrating dose-dependant guanine-specific arrest in a DNA polymerase assay (Ex. 35–188). Cr(V) may also form intrastrand crosslinks since Cr(V) interacts with DNA *in vitro* (Ex. 35–178). The Cr(V)-DNA crosslinks are probably readily reduced to Cr(III) in cell systems. Intrastrand crosslinks have also been implicated in inhibition of RNA polymerase and DNA topoisomerase, leading to cell cycle arrest, apoptosis and possibly other disturbances in cell growth that contribute to the carcinogenic pathway (Ex. 35–149).

DNA strand breaks and oxidative damage result from the one electron reduction/oxidation cycling of Cr(VI), Cr(V), and Cr(IV). Shi *et al.* showed that soluble Cr(VI) in the presence of ascorbate and H_2O_2 caused DNA double strand breaks and 8-hydroxy deoxyguanine (8-OHdG, a marker for oxidative DNA damage) *in vitro* (Ex. 35–129). Leonard *et al.* showed that the DNA strand breaks were reduced by several experimental conditions including an O_2-free atmosphere, catabolism of H_2O_2 by catalase, ROS depletion by free radical scavangers, and chelation of Cr(V). They concluded that the strand breaks and 8-OHdG resulted from DNA damage caused by hydroxyl radicals from Cr(VI) reduction/oxidation cycling (Ex. 31–22–17). Generation of ROS-dependant DNA damage could also be shown with insoluble Cr(VI) (Ex. 35–137). DNA strand breaks and related damage caused by soluble Cr(VI) have been reported in Chinese hamster cells (Ex. 35–128), human fibroblasts (Ex. 311), and human prostate cells (Ex. 35–255). Pretreatment of Chinese hamster cells with a metal chelator suppressed Cr(V) formation from Cr(VI) and decreased DNA strand breaks (Ex. 35–197). Chinese hamster cells that developed resistance to H_2O_2 damage also had reduced DNA strand breaks from Cr(VI) treatment compared to the normal phenotype (Ex. 35–176).

Several researchers have been able to modulate Cr(VI)-induced DNA damage using cellular reductants such as ascorbate, GSH and the free radical scavenger tocopherol (vitamin E). This has provided insight into the relationships between DNA damage, reduced chromium forms and ROS. Sugiyama *et al.* showed that Chinese hamster cells pretreated with ascorbate decreased soluble Cr(VI)-induced DNA strand damage (*e.g.*, alkali-labile sites), but enhanced DNA-amino acid crosslinks (Ex. 35–133). Standeven and Wetterhahn reported that elimination of ascorbate from rat lung cytosol prior to *in vitro* incubation with soluble Cr(VI) completely inhibited Cr-DNA binding (Ex. 35–180). However, not all types of Cr-DNA binding are enhanced by ascorbate. Bridgewater *et al.* found that high ratios of ascorbate to Cr(VI)

actually decreased intrastrand crosslinks *in vitro* while low ratios induced their formation (Ex. 35–263). This finding is consistent with research by Stearns and Watterhahn who showed that excessive ascorbate relative to Cr(VI) leads to two-electron reduction of Cr(III) and formation of Cr(III)-DNA monoadducts and DNA-Cr(III)-amino acid crosslinks (Ex. 35–166). Low amounts of ascorbate primarily cause one-electron reduction to intermediates Cr(V) and Cr(IV) that form crosslinks with DNA and ROS responsible for DNA strand breaks, alkali-labile sites, and clastogenic damage. This explains the apparent paradox that *extracellular* Cr(VI) reduction by ascorbate to Cr(III) reduces Cr(VI)-induced DNA binding but *intracellular* Cr(VI) reduction by ascorbate to Cr(III) enhances Cr-DNA binding. The aforementioned studies used soluble forms of Cr(VI), but Blankenship *et al.* showed that ascorbate pretreatment inhibited chromosomal aberrations in Chinese hamster ovary cells caused by both insoluble lead chromate particles as well as soluble Cr(VI) (Ex. 35–115). Pretreatment with the free radical scavenger tocopherol also inhibits chromosomal aberrations and alkali-labile sites in Cr(VI)-treated cells (Exs. 35–115; 35–128).

Studies of the different types of DNA damage caused by Cr(VI) and the modulation of that damage inside the cell demonstrate that Cr(VI) itself is not biologically active. Cr(VI) must undergo intracellular reduction to Cr(V), Cr(IV), and Cr(III) before the damage to DNA can occur. The evidence suggests that Cr(III) can cause DNA-Cr-amino acid, DNA-Cr-DNA crosslinks and Cr-DNA monoadducts. Cr(V) and possibly Cr(IV) contribute to intrastrand crosslinks and perhaps other Cr-DNA binding. ROS generated during intracellular reduction of Cr(VI) lead to lesions such as chromosomal aberrations, DNA strand breaks, and oxidative DNA damage. The specific DNA lesions responsible for neoplastic transformation have yet to be firmly established so all forms of DNA damage should, at this time, be regarded as potential contributors to carcinogenicity.

d. *Cr(VI)-induced Disturbances in the Regulation of Cell Replication.* Recent research has begun to elucidate how Cr(VI)-induced oxidative stress and DNA lesions trigger cell signaling pathways that regulate the cell growth cycle. The complex regulation of the cell growth cycle by Cr(VI) involves activation of the p53 protein and other transcription factors that respond to oxidative stress and DNA damage. The cellular response ranges from a temporary pause in the cell cycle to terminal growth arrest (i.e., viable cells that have lost the ability to replicate) and a programmed form of cell death, known as apoptosis. Apoptosis involves alterations in mitochondrial permeability, release of cytochrome c and the action of several kinases and caspases. Less is known about the molecular basis of terminal growth arrest. Terminal growth arrest and apoptosis serve to eliminate further growth of cells with unrepaired Cr(VI)-induced genetic damage. However, it is believed that cells which escape these protective mechanisms and regain replicative competence eventually become resistant to normal growth regulation and can transform to a neoplastic phenotype (Exs. 35–121; 35–122; 35–120).

Blankenship *et al.* first described apoptosis as the primary mode of cell death following a two hour treatment of Chinese hamster ovary cells with high concentrations (>150 μM) of soluble Cr(VI) (Ex. 35–144). Apoptosis also occurs in human lung cells following short-term treatment with soluble Cr(VI) (Ex. 35–125) as well as longer term treatment (*e.g.*, 24 hours) with lower concentrations of soluble Cr(VI) (*e.g.*, 10 μM) and insoluble Cr(VI) in the form of lead chromate (Ex. 35–166). Ye *et al.* found that the Cr(VI) treatment that caused apoptosis also activated expression of p53 protein (Ex. 35–125). This apoptotic response was substantially reduced in a p53-deficient cell line treated with Cr(VI), suggesting that the p53 activation was required for apoptosis. Other studies using p53 null cells from mice and humans confirmed that Cr(VI)-induced apoptosis is p53-dependent (Ex. 35–225).

The p53 protein is a transcription factor known to be activated by DNA damage, lead to cell cycle arrest, and regulate genes responsible for either DNA repair or apoptosis. Therefore, it is likely that the p53 activation is a response to the Cr(VI)-induced DNA damage. Apoptosis (i.e., programmed cell death) is triggered once the Cr(VI)-induced DNA damage becomes too extensive to successfully repair. In this manner, apoptosis serves to prevent replication of genetically damaged cells. Several researchers have gone on to further elucidate the molecular pathways involved in Cr(VI)-induced apoptosis. ROS produced by intracellular Cr(VI) reduction/oxidation cycling have been implicated in the activation of p53 and apoptosis (Exs. 35–255; 35–122). Using specific inhibitors, Pritchard *et al.* showed that mitochondrial release of cytochrome c is critical to apoptotic death from Cr(VI) (Ex. 35–159). Cytochrome c release from mitochondria could potentially result from either direct membrane damage caused by Cr(VI)-induced ROS or indirectly by enhanced expression of the p53-dependent apoptotic proteins, Bax and Nova, known to increase mitochondrial membrane permeability.

Cr(VI) causes cell cycle arrest and reduces clonogenic potential (i.e., normal cell growth) at very low concentrations (*e.g.*, 1 μM) where significant apoptosis is not evident. Xu *et al.* showed that human lung fibroblasts treated with low doses of Cr(VI) caused guanine-guanine intrastrand crosslinks, guanine-specific polymerase arrest, and inhibited cell growth at the G_1/S phase of the cell cycle (Ex. 35–188). Zhang et al. described a dose-dependent increase in growth arrest at the G_2/M phase of the cell cycle in a human lung epithelial cell line following 24 hour Cr(VI) treatment over a concentration range of 1 to 10 μM (Ex. 35–135). The cell cycle arrest could be partially eliminated by reducing production of Cr(VI)-induced ROS. Apoptosis was not detected in these cells until a concentration of 25 μM Cr(VI) had been reached. These data suggest that low cellular levels of Cr(VI) are able to cause DNA damage and disrupt the normal cell growth cycle.

Pritchard *et al.* studied the clonogenicity over two weeks of human fibroblasts treated 24 hours with soluble Cr(VI) concentrations from 1 to 10 μM (Ex. 35–120). They reported a progressive decline in cell growth with increasing Cr(VI) concentration. Terminal growth arrest (i.e., viable cells that have lost the ability to replicate) was primarily responsible for the decrease in clonogenic survival below 4 μM Cr(VI). At higher Cr(VI) concentrations, apoptosis was increasingly responsible for the loss in clonogenicity. Pritichard *et al.* and other research groups have suggested that a subset of cells that continue to replicate following Cr(VI) exposure could contain unrepaired genetic damage or could have become intrinsically resistant to processes (*e.g.*, apoptosis, terminal growth arrest) that normally control their growth (Exs. 35–121; 35–122; 35–120). These surviving cells would then be more prone to neoplastic progression and have greater carcinogenic potential.

e. *Summary.* Respirable chromate particulates are taken up by target cells in the bronchoalveolar region of the lung, become intracellularly reduced to several reactive genotoxic species able to damage DNA, disrupt normal regulation of cell division and cause neoplastic transformation. Scientific studies indicate that both aqueous

insoluble and soluble Cr(VI) can be transported into the cell. In fact, cell surface interactions with sparingly soluble and some insoluble chromates likely create a concentrated microenvironment of chromate ion resulting in higher intracellular levels of Cr(VI) than would occur from soluble chromates. This is consistent with the studies of respiratory tract carcinogenesis in animals that indicate the most tumorigenic chromates had low to moderate water solubility. Once inside the cell, Cr(VI) is converted to several lower oxidation forms able to bind to and crosslink DNA. ROS are produced during intracellular reduction/oxidation of Cr(VI) that further damage DNA. This genotoxicity is functionally translated into impaired DNA replication, mutagenesis, and altered gene expression that ultimately lead to neoplastic transformation.

9. Preliminary Conclusions

OSHA preliminarily concludes that the study data summarized in the previous sections support the determination that Cr(VI) compounds should be regarded as carcinogenic to workers. The strongest evidence comes from the many cohort studies reporting excess lung cancer mortality in workers exposed to Cr(VI) during production of chromates and chromate pigments. Additional evidence comes from the less consistent elevations in lung cancer mortality found in workers exposed to Cr(VI) in other occupations, increased tumor incidence in experimental animals treated with Cr(VI), and cellular and molecular data on mode of action.

Studies of chromate production workers in several countries have consistently found significantly greater mortality from lung cancer than expected. In the earliest studies of chromate workers in whom Cr(VI) exposures were believed to be highest, the risk for respiratory cancer was between 15 and 29 times expectation (Exs. 7–2; 7–13; 7–1). Lung cancer risks of this magnitude cannot be explained by potential confounders and other biases.

Later studies that were able to reconstruct exposure histories in workers from production plants located in Baltimore, MD and Painesville, OH found significant trends between lung cancer mortality and both cumulative exposure to Cr(VI) and duration of employment (Exs. 31–22–11; 33–10). Workers were predominantly exposed to the highly water soluble sodium chromate and sodium dichromate at these plants, although probable exposure to other chromates also occurred. Gibb *et al.* showed that a significant association between lung cancer and Cr(VI) was evident, even in models that accounted for smoking (Ex. 31–22–11). Other studies documented declines in lung cancer mortality rates with reduced Cr(VI) exposures due to improvements in the production process (Exs. 7–99; 7–91; 31–18–4). These trends serve to strengthen the evidence for causal association between Cr(VI) and lung cancer.

Studies of workers in the chromate pigment production industry also consistently show significantly elevated lung cancer mortality. These include cohorts from Norway, Great Britain, U.S., and France. The workers were principally exposed to zinc and lead chromate pigments, but the levels of Cr(VI) exposure were not well characterized. Some studies presented data that suggested excess lung cancer was more strongly associated with zinc chromate, although workers were exposed to several chromium pigments (Exs. 7–41; 7–42).

Significantly elevated lung cancer mortality was found in two British chromium electroplating cohorts (Exs. 35–62; 271). The workers were exposed to Cr(VI) in the form of chromic acid mist as well as nickel, another potential lung carcinogen. The association between lung cancer and Cr(VI) in stainless steel welders and ferrochromium production workers are confounded by substantial exposures to other potential carcinogens and Cr(III). However, the generally elevated lung cancer mortality in these workers supports the stronger evidence from the soluble chromate and chromate pigment production cohorts.

A number of the epidemiological studies cited above were evaluated by the IARC in 1990 (Ex. 35–43). IARC found ''sufficient evidence in humans for the carcinogenicity of chromium [VI] compounds as encountered in chromate production, chromate pigment production and chromate plating industries'' (Ex. 35–43, p. 213). IARC gave Cr(VI) compounds their highest Group 1 classification for agents considered carcinogenic to humans. The EPA and ACGIH have designated Cr(VI) compounds as known and confirmed human carcinogens, respectively (Exs. 35–52; 35–207). NIOSH considers Cr(VI) compounds to be potential occupational carcinogens (Ex. 31–22–22, p. 8).

Experimental animals have generally been administered Cr(VI) compounds by routes other than inhalation. A number of studies in which Cr(VI) compounds were directly instilled in the respiratory tract of rodents produced a significant incidence of lung tumors (Exs. 11–2; 11–12; 11–7). The findings indicate different tumorigenic potencies among Cr(VI) compounds. The less water soluble calcium chromate, strontium chromates, and zinc chromates cause higher numbers of lung tumors at similar doses than the more water soluble sodium dichromate and chromic acid. Experimental research suggests that cellular uptake of the water-insoluble lead chromate is enhanced by the ability to achieve a high local concentration at the lung cell surface that does not occur during uptake of soluble chromates (Ex. 35–149). Because of the greater cancer potency in animal studies, ACGIH has recommended a lower occupational TLV for insoluble Cr(VI) compounds (10 $\mu g/m^3$) than for water-soluble Cr(VI) compounds (50 $\mu g/m^3$).

The few available inhalation studies are limited by abbreviated exposure durations, low exposure levels, or small number of animals per dose group. These studies report slightly elevated lung tumor incidence that are not statistically significant (Exs. 10–11; 35–26–1) or marginally significant (Exs. 10–8; 35–26). Cr(VI) administered to animals by intramuscular, subcutaneous, and other routes of administration have consistently produced a high incidence of tumors, usually near the site of administration.

Evidence from *in vitro* research shows that Cr(VI) enters the cell and is rapidly converted to several lower oxidation forms able to bind to and crosslink DNA. ROS (reactive oxygen species) are produced during intracellular reduction/oxidation of Cr(VI) that can further damage DNA. Soluble and insoluble Cr(VI) compounds are reported to cause mutagenesis, clastogenesis, and neoplastic transformation across multiple assays in a wide range of experimental systems from prokaryotic organisms to human cells *in vitro* and animals *in vivo*. Therefore, OSHA regards all Cr(VI) compounds as agents able to induce carcinogenesis through a genotoxic mode of action.

The rate, as well as the magnitude of the Cr(VI) dose, that reaches the lung has been shown to influence carcinogenic outcome in experimental animals (Ex. 11–7). Less frequent, but higher dose levels of Cr(VI) instilled in the tracheas of rats caused greater tumor incidence than the same total amount of Cr(VI) instilled more frequently but at lower dose levels. This may result from a proliferation of neoplastic cells triggered by lung inflammation at the high Cr(VI) dose levels or from overwhelming any of a number of molecular pathways that serve to protect against Cr(VI)-induced respiratory

carcinogenesis, including extracellular reduction to poorly absorbed Cr(III), intracellular binding of reactive forms to non-critical macromolecules, or repair of DNA damage. The existence of dose rate effects could potentially introduce non-linearities in the Cr(VI) exposure-cancer response. As discussed in the quantitative risk assessment section (section VII), OSHA is not aware of reliable data on which to confidently predict the range of Cr(VI) air levels at which presumed non-linearities might occur or empirical data that convincingly establishes the existence of a threshold exposure for carcinogenicity.

C. Non-Cancer Respiratory Effects

The following sections describe the evidence from the literature on nasal irritation, nasal ulcerations, nasal perforations, asthma, and bronchitis following inhalation exposure to water soluble Cr(VI) compounds. The evidence clearly demonstrates that workers can develop impairment to the respiratory system (nasal irritation, nasal ulceration, nasal perforation, and asthma) after work place exposure by inhalation exposure to Cr(VI) compounds below the current PEL.

It is very clear from the evidence that workers may develop nasal irritation, nasal septum ulcerations, and nasal septum perforations at occupational exposures level at or below the current PEL of 52 μg/m^3. However, it is not clear what occupational exposure levels lead to the development of occupational asthma or bronchitis.

1. Nasal Irritation, Nasal Septum Ulcerations and Nasal Septum Perforations

Occupational exposure to Cr(VI) can lead to nasal septum ulcerations and nasal septum perforations. The nasal septum separates the nostrils and is composed of a thin strip of cartilage with an overlying mucous membrane known as the mucosa. The initial lesion after Cr(VI) exposure is characterized by localized inflammation or a reddening of the affected mucosa, which can later lead to atrophy. This may progress to an ulceration of the mucosa layer (Ex. 35–1; Ex. 7–3). If exposure is discontinued, the ulcer progression will stop and a scar may form. However, if exposure continues, the ulcer may break through the septum, resulting in a nasal septum perforation sometimes referred to chrome hole. Individuals with nasal perforations may experience a range of signs and symptoms, such as a whistling sound, bleeding, nasal discharge, and infection. Some individuals may experience no noticeable effects. It is currently not known precisely what level would trigger such nasal problems, but, as stated earlier, it is evident that workers are developing nasal problems at levels at or below the current PEL.

Several cohort and cross-sectional studies have described nasal lesions from airborne exposure to Cr(VI) at various electroplating and chrome production facilities. Most of these studies have been reviewed by the Center for Disease Control's Agency for Toxic Substances and Disease Registry (ATSDR) toxicological profile for chromium (Ex. 35–41). OSHA reviewed the studies summarized in the profile and conducted its own literature search to update and supplement the review. In its evaluation, OSHA took into consideration the exposure regimen and experimental conditions under which the studies were performed, including exposure levels, duration of exposure, number, and the inclusion of appropriate control groups. Studies were not included if they did not contribute to the weight of evidence either because of inadequate documentation or because of poor quality. This section only covers some of the key studies and reviews. OSHA has also identified two case reports demonstrating the development of nasal irritation and nasal septum perforations, and these case reports are summarized as well. One case report shows how a worker can develop the nasal perforations from direct contact (i.e., touching the inner surface of the nose with contaminated fingers).

Lindberg and Hedenstierna examined the respiratory symptoms and effects of 104 Swedish electroplaters (Ex. 9–126). Of the 104 electroplaters, 43 were exposed to chromic acid by inhalation. The remaining 61 were exposed to a mixture of chromic acid and nitric acid, hydrochloric acid, boric acid, nickel, and copper salts. The workers were evaluated for respiratory symptoms, changes in the nasal septum, and lung function. All workers were asked to fill out a detailed questionnaire on their history of respiratory symptoms and function. Physicians performed inspections of the nasal passages of each worker. Workers were given a pulmonary function test to assess lung function. For those 43 workers exposed exclusively to chromic acid, the median exposure time was 2.5 years, ranging from 0.2 to 23.6 years. The workers were divided into two groups, a low exposure group (19 workers exposed to eight-hour time weighted average levels below 2 μg/m^3) and a high exposure group (24 workers exposed to eight-hour time weighted average levels above 2 μg/m^3). Personal air sampling was conducted on 11 workers for an entire week and at stations close to the chrome baths to evaluate peak exposures and variations in exposure on different days over the week. Nineteen office employees were not exposed to Cr(VI) used as controls for nose and throat symptoms, and 119 auto mechanics (no car painters or welders) whose lung function had been evaluated using similar techniques to those used on Cr(VI) exposed workers were used as controls for lung function.

The investigators reported nasal ulcerations and perforations in a group of workers exposed at the highest peak exposure levels (ranging from 20 μg/m^3/day to peak levels of 46 μg/m^3/day) to chromic acid as Cr(VI); prevalence of ulceration/perforation was statistically higher than the control group. Of the 14 individuals in the 20–46 μg/m^3 exposure group, seven developed nasal ulcerations. In addition to nasal ulcerations, 2 of the 7 also had progressed to nasal perforations. Furthermore, three individuals developed nasal perforations only, at the same exposure levels. At average exposure levels from 2 μg/m^3 to 20 μg/m^3, half of the workers complained of ''constantly running nose,'' ''stuffy nose,'' or ''there was a lot to blow out.'' (Authors do not provide details of each complaint). Atrophy, which is a precursor to ulcerations and perforations, was only observed in occupationally exposed workers at relatively low peak levels ranging from 2.5 μg/m^3 to 11 μg/m^3. No one exposed to levels below 1 μg/m^3 (time-weighted average, TWA) complained of respiratory symptoms or developed lesions.

The authors also reported that in the exposed workers, both forced vital capacity and forced expiratory volume in one second were reduced by 0.2 L, when compared to controls. The forced mid-expiratory flow diminished by 0.4 L/second from Monday morning to Thursday afternoon in workers exposed to chromic acid as Cr(VI) daily TWA average levels of 2 μg/m^3 or higher. The effects were small, not outside the normal range and transient (recovery after 2 days). There was no difference between the control and exposed group after the weekend. The workers exposed to lower levels (2 μg/m^3 or lower, TWA) showed no significant changes.

Kuo *et al.* evaluated nasal septum ulcerations and perforations in 189 electroplaters in 11 electroplating factories (three factories used chromic acid, six factories used nickel-chromium, and two factories used zinc) in Taiwan (Ex. 35–10). Of the 189 workers, 26 used Cr(VI), 129 used nickel-chromium, and 34 used zinc. The

control group consisted of electroplaters who used nickel and zinc. All workers were asked to fill out a questionnaire and were given a nasal examination including a lung function test by a certified otolaryngologist. The authors determined that 30% of the workers (8/26) that used chromic acid developed nasal septum perforations and ulcerations and 38% (10/26) developed nasal septum ulcers. Using the Mantel Extension Test for Trends, the authors also found that chromium electroplaters had an increased likelihood of developing nasal ulcers and perforations compared to electroplating workers using nickel-chromium and zinc. Personal sampling of airborne Cr(VI) results indicated the highest levels (32 $\mu g/m^3 \pm 35$ $\mu g/m^3$, ranging from 0.1 $\mu g/m^3 - 119$ $\mu g/m^3$) near the electroplating tanks of the Cr(VI) electroplating factories (Ex. 35–11). Much lower personal sampling levels were reported in the ''other areas in the manufacturing area'' and the ''administrative area'' (TWA 0.16 ± 0.10 $\mu g/m^3$) of the Cr(VI) electroplating plant. The duration of sampling was not indicated. The results of the lung function tests showed significantly lower values among Cr(VI) electroplaters compared to the other two exposure groups in regards to vital capacity, forced vital capacity, and forced expiratory volume in one second.

Cohen *et al.* examined respiratory symptoms of 37 electroplaters following inhalation exposure to chromic acid (Ex. 9–18). The mean length of employment for the 37 electroplaters was 26.9 months (range from 0.3 to 132 months). Fifteen workers employed in other parts of the plant were randomly chosen for the control group (mean length of employment was 26.1 months; range from 0.1 to 96). All workers were asked to fill out a questionnaire on their respiratory history, including providing details on their symptoms. An otolaryngologist then examined each individual's nasal passages and identified ulcerations and perforations. Air samples to measure Cr(VI) were collected for electroplaters. The air sampling results of chromic acid as Cr(VI) concentrations for electroplaters was a mean of 2.9 $\mu g/m^3$ (range from non-detectable to 9.1 $\mu g/m^3$). The authors found that 95% of the electroplaters developed pathologic changes in nasal mucosa. Thirty-five of the 37 workers, who were employed for more than 1 year had nasal tissue damage. None of these workers reported any previous job experience involving Cr(VI) exposure. Four workers developed nasal perforations, 12 workers developed ulcerations and crusting of the septal mucosa, 11 workers developed discoloration of the septal mucosa, and eight workers developed shallow erosion of septal mucosa. The control group consisted of 15 workers who were not exposed to Cr(VI) at the plant. All but one had normal nasal mucosa. The one individual with abnormal finding was discovered to have a previous Cr(VI) exposure while working in a garment manufacturing operation as a fabric dyer for three years. In addition to airborne exposure, the authors observed employees frequently wiping their faces and picking their noses with contaminated hands and fingers. Many did not wear any protective gear, such as gloves, glasses, or coveralls.

Lucas and Kramkowsi conducted a Health Hazard Evaluation (HHE) on 11 chrome platers in an industrial electroplating facility (Ex. 3–84). The electroplaters worked for about 7.5 years on average. Physicians evaluated each worker for chrome hole scars, nasal septum ulceration, mucosa infection, nasal redness, perforated nasal septum, and wheezing. Seventeen air samples for Cr(VI) exposure were collected in the chrome area. Cr(VI) air concentrations ranged from 1 to 20 $\mu g/m^3$, with an average of 4 $\mu g/m^3$. In addition to airborne exposure, the authors observed workers being exposed to Cr(VI) by direct ''hand to nose'' contact, such as touching the nose with contaminated hands. Five workers had nasal mucosa that became infected, two workers had nasal septum ulcerations, two workers had atrophic scarring (author did not provide explanation), possibly indicative of presence of past ulcerations, and four workers had nasal septum perforations.

Gomes evaluated 303 employees from 81 electroplating operations in Sao Paulo, Brazil (Ex. 9–31). Results showed that more than two-thirds of the workers had nasal septum ulcerations and perforations following exposure to chromic acid at levels greater than 100 $\mu g/m^3$, but less than 600 $\mu g/m^3$ (precise duration of exposure was not stated). These effects were observed within one year of employment.

Lin et al. examined nasal septum perforations and ulcerations in 79 electroplating workers from seven different chromium electroplating factories in Taipei, Taiwan (Ex.35–13). Results showed six cases of nasal septum perforations, four having scar formations, and 38 cases of nasal septum ulcerations following inhalation exposure to chromic acid. Air sampling near the electroplating tanks had the highest range of chromic acid as Cr(VI) (mean of 28 $\mu g/m^3$; range from 0.7 to 168.3 $\mu g/m^3$). In addition to airborne exposures, the authors also observed direct ''hand to nose'' contact where workers placed contaminated fingers in their nose. The authors attributed the high number of cases to poor industrial hygiene practices in the facilities. Five of the seven factories did not have adequate ventilation systems in place. Workers did not wear any PPE, including respirators.

Bloomfield and Blum evaluated nasal tissue damage and nasal septum perforations in 23 workers employed at six chromium electroplating plants (Ex. 9–13). They found that daily exposure to chromic acid as Cr(VI) at levels of 52 $\mu g/m^3$ or higher can lead to nasal tissue damage. Three workers developed nasal ulcerations, two workers had nasal perforations, nine workers had nose bleeds, and nine workers had inflamed mucosa.

Kleinfeld and Rosso found seven cases out of nine of chrome electroplaters having nasal septum ulcerations (Ex. 9–41). Workers were exposed to chromic acid as Cr(VI) by inhalation at levels ranging from 93 $\mu g/m^3$ to 728 $\mu g/m^3$. Duration of exposure varied from two weeks to one year. Nasal septum ulcerations were noted as early as one month of employment in some workers.

Royle, using questionnaire responses, reported a significant increase in the prevalence of nasal ulcerations among 997 British electroplaters exposed to chromic acid with an increasing prevalence the longer the worker was exposed to chromic acid (*e.g.*, from 14 cases with exposure less than one year to 62 cases with exposure over five years) (Ex. 7–50). In all but 2 cases, air samples revealed chromic acid was at concentrations of 0.03 mg/m^3 (i.e., 30 $\mu g/m^3$).

Gibb *et al.* reported nasal irritations, nasal septum bleeding, nasal septum ulcerations and perforations among a cohort of 2,350 chrome production workers in a Baltimore plant (Ex. 31–22–12). A description of the cohort is provided in detail in the cancer health effects section V.B. of this preamble. The authors found that more than 60% of the cohort had experienced nasal ulcerations and irritations, and that the workers developed these effects for the first time within the first three months of being hired (median). Gibb *et al.* found the median exposure to Cr(VI) during first diagnosis of irritated and/or ulcerated nasal septum was 10 $\mu g/m^3$. About 17% of the cohort had reported nasal perforations. Based on historical data, the authors believe that the nasal findings are attributed to Cr(VI) exposure.

Gibb *et al.* also used a Proportional Hazard Model to evaluate the relationship between Cr(VI) exposure and first occurrence of each of the clinical findings. Cr(VI) data was entered into the model as a time dependent variable. Other explanatory variables were calendar year of hire and age of hire. Results of model indicated that airborne Cr(VI) exposure was associated with the occurrence of nasal septum ulceration (p = 0.0001). The lack of an association of airborne Cr(VI) exposure to nasal perforation and bleeding nasal septum may reflect the fact that Cr(VI) concentrations used in the model represent annual averages for the job, in which the worker was involved in at the time of the findings, rather than a short-term average. Annual averages do not factor in day-to-day fluctuations or extreme episodic occurrences. Also, the author believes poor housekeeping and hygiene practices may have contributed to these health effects as well as Cr(VI) airborne concentrations.

Based on their hazard model, Gibb *et al.* estimated the relative risks for nasal septum ulcerations would increase 1.2 for each 52 μg of Cr(VI)/m^3 increase in Cr(VI) air levels. They saw a reduction in the incidence of nasal findings in the later years. They found that workers from the earlier years who did not wear any PPE had a greater risk of developing respiratory problems. They believe that the reduction in ulcerations was possibly due to an increased use of respirators and protective clothing and improved industrial hygiene practices at the facility.

The U.S. Public Health Service conducted a study of 897 chrome production workers in seven chromate-producing plants in the early 1950s (Ex. 7–3). The findings of this study were used in part as justification for the current OSHA PEL. Workers were exposed by inhalation to various water soluble chromates and bichromate compounds. The total mean exposure to the workers was a TWA of 68 μg/m^3. Of the 897 workers, 57% (or 509 workers) were found to have nasal septum perforations. Nasal septum perforations were observed even in workers during their first year on the job.

Case reports provide further evidence that airborne exposure to direct ''hand to nose'' contact of Cr(VI) compounds lead to the development of nasal irritation and nasal septum perforations.

For example, a 70-year-old man developed nasal irritation, incrustation, and perforation after continuous daily exposure by inhalation to chromium trioxide (doses were not specified, but most likely quite high given the nature of his duties). This individual inhaled chromium trioxide daily by placing his face directly over an electroplating vessel. He worked in this capacity from 1934 to 1982. His symptoms continued to worsen after he stopped working. By 1991, he developed large perforations of the nasal septum and stenosis (or constriction) of both nostrils by incrustation (Ex. 35–8).

Similarly, a 30-year-old female jigger (a worker who prepares the items prior to electroplating by attaching the items to be plated onto jigs or frames) developed nasal perforation in her septum following continuous exposure (doses in this case were not provided) to chromic acid mists. She worked adjacent to the automated Cr(VI) electroplating shop. She was also exposed to chromic acid from direct contact when she placed her contaminated fingers in her nose. Her hands became contaminated by handling wet components in the jigging and de-jigging processes (Ex. 35–24).

Evidence of nasal septum perforations has also been demonstrated in experimental animals. Adachi exposed 23 C57BL mice to chromic acid by inhalation at concentrations of 1.81 mg Cr(VI)/m^3 for 120 minutes per day, twice a week and 3.63 mg Cr(VI)/m^3 for 30 minutes per day, two days per week for up to 12 months (Ex. 35–26). Three of the 23 mice developed nasal septum perforations in the 12-month exposure group.

Adachi *et al.* also exposed 50 ICR female mice to chromic acid by inhalation at concentrations of 3.18 mg Cr(VI)/m^3 for 30 minutes per day, 2 days per week for 18 months (Ex. 35–26–1). The authors used a miniaturized chromium electroplating system to mimic electroplating processes and exposures similar to working experience. Nasal septum perforations were found in six mice that were sacrificed after 10 months of exposure. Of those mice that were sacrificed after 18 months of exposure, nasal septum perforations were found in three mice.

2. Occupational Asthma

Occupational asthma is considered ''a disease characterized by variable airflow limitation and/or airway hyper responsiveness due to causes and conditions attributable to a particular occupational environment and not to stimuli encountered outside the workplace'' (Ex. 35–15). Asthma is a serious illness that can damage the lungs and in some cases be life threatening. The common symptoms associated with asthma include heavy coughing while exercising or when resting after exercising, shortness of breath, wheezing sound, and tightness of chest. Many workers develop an asthmatic attack. An attack may be triggered by particles in the air (Ex. 35–3; Ex. 35–6). It is not clear what occupational exposure levels of Cr(VI) compounds would lead to the development of occupational asthma.

The strongest evidence of occupational asthma has been demonstrated in four case reports. OSHA chose to focus on these four case reports because the data from other occupational studies do not exclusively implicate Cr(VI), even though the studies generally show an increased prevalence of workers having difficulty breathing and other asthmatic-related symptoms following inhalation of multiple chemicals. The four case reports have the following in common: (1) The worker has a history of occupational exposure exclusively to Cr(VI); (2) a physician has confirmed a diagnosis that the worker has symptoms consistent with occupational asthma; and (3) the worker exhibits functional signs of air restriction (*e.g.*, low forced expiratory volume in one second or low peak expiratory flow rate) upon bronchial challenge with Cr(VI) compounds. These case reports demonstrate, through challenge tests, that exposure to Cr(VI) compounds can cause asthmatic responses. The other general case reports below did not use challenge tests to confirm that Cr(VI) was responsible for the asthma; however, these reports were among workers similarly exposed to Cr(VI) such that Cr(VI) is likely to have been a contributing factor in the development of their asthmatic symptoms.

DaReave reported the case of a 48-year-old cement floorer who developed asthma from inhaling airborne Cr(VI) (Ex. 35–7). This worker had been exposed to Cr(VI) as a result of performing cement flooring activities for more than 20 years. The worker complained of dyspnea, shortness of breath, and wheezing after work, especially after working in enclosed spaces. The Cr(VI) content in cement was about 12 ppm. A bronchial challenge test with potassium dichromate produced a 50% decrease in forced expiratory volume in one second. The occupational physician concluded that the worker's asthmatic condition triggered by exposure to Cr(VI) caused the worker to develop bronchial constriction.

LeRoyer reported a case of a 28-year-old roofer who developed asthma from breathing dust while sawing material made of corrugated fiber cement containing Cr(VI) for nine years (Ex. 35–12). This worker demonstrated

symptoms such as wheezing, shortness of breath, coughing, rhinitis, and headaches while working. Skin prick tests were all negative. Several inhalation challenges were performed by physicians and immediate asthmatic reactions were observed after inhaling nebulization of potassium dichromate. A reduction (by 20%) in the forced expiratory volume in one second after exposure to fiber cement dust was noted.

Novey *et al.* reported a case of a 32-year-old electroplating worker who developed asthma from working with chromium sulfate and nickel salts (Ex. 35–16). He began experiencing coughs, wheezing, and dyspnea within the first week of exposure. Inhalation challenge tests given by physicians using chromium sulfate and nickel salts, in separate challenges, both resulted in positive reactions. The worker immediately had difficulty breathing and started wheezing in both challenges. The forced expiratory volume in 1 second decreased by 22% and the forced expiratory volume in 1 second/forced vital capacity ratio also decreased from 74.5% to 60.4%. The author believes the worker's bronchial asthma was induced from inhaling chromium sulfate and nickel salts, individually. Similar findings were reported in a different individual by Sastre (Ex. 35–20).

Shirakawa and Morimoto reported a case of a 50-year-old worker who developed asthma while working at a metal-electroplating plant (Ex. 35–21). Bronchial challenge by physicians produced positive results when using potassium bichromate, followed by a rapid recovery within 5 minutes, when given no exposures. The worker's forced expiratory volume in 1 second dropped by 37% after inhalation of potassium bichromate. The individual immediately began wheezing, coughing with dyspnea, and recovered without treatment within five minutes. The author believes that the worker developed his asthma from inhaling potassium bichromate.

In addition to the case reports confirming that Cr(VI) is responsible for the development of asthma using inhalation challenge tests, the following are several other case reports of Cr(VI) exposed workers having symptoms consistent with asthma where the symptoms were never confirmed by using inhalation challenge tests.

Lockman reported a case of a 41-year old woman, who was occupationally exposed to potassium dichromate during leather tanning (Ex. 35–14). The worker developed an occupational allergy to potassium dichromate. This allergy involved both contact dermatitis and asthma. The physicians considered other challenge tests using potassium dichromate as the test agent (*i.e.,* peak expiratory flow rate, forced expiratory volume in 1 second and methacholine or bronchodilator challenge), but the subject changed jobs before the physicians could administer these tests. Once the subject changed jobs, all her symptoms disappeared. It was not confirmed whether the occupational exposure to Cr(VI) was the cause of the asthma.

Williams reported a 23-year old textile worker who was occupationally exposed to chromic acid. He worked near two tanks of chromic acid solutions (Ex. 35–23). He inhaled fumes while frequently walking through the room with the tanks. He developed both contact dermatitis and asthma. He believes the tank was poorly ventilated and was the source of the fumes. He stopped working at the textile firm on the advice of his physician. After leaving, his symptoms improved greatly. No inhalation bronchial challenge testing was conducted to confirm that chromic acid was causing his asthmatic attacks. However, as noted above, chromic acid exposure has been shown to lead to occupational asthma, and thus, chromic acid was likely to be a causative agent in the development of asthma.

Park *et al.* reported a case of four workers who worked in various occupations involving exposure to either chromium sulfate or potassium dichromate (Ex. 35–18). Two worked in a metal electroplating factory, one worked at a cement manufacturer, and the other worked in construction. All four developed asthma. One individual had a positive response to bronchial provocation test (with chromium sulfate as the test agent). This individual developed an immediate reaction upon given chromium sulfate as the test agent. He experienced wheezing, coughing and dyspnea. Peak expiratory flow rate decreased by about 20%. His physician determined that exposure to chromium sulfate was contributing to his asthma condition. Two had positive reactions to prick skin tests with chromium sulfate as the test agent. Two had positive responses to patch tests using potassium dichromate as the testing challenge agent. Only one out of four underwent inhalation bronchial challenge testing (with a positive result to chromium sulfate) in this report.

3. Bronchitis

In addition to nasal ulcerations, nasal septum perforations, and asthma, there is also limited evidence from reports in the literature of bronchitis associated with Cr(VI) exposure. It is not clear what occupational exposure levels of Cr(VI) compounds would lead to the development of bronchitis.

Royle found that 28% (104/288) of British electroplaters developed bronchitis upon inhalation exposure to chromic acid, as compared to 23% (90/299) controls (Ex. 7–50). The workers were considered to have bronchitis if they had symptoms of persistent coughing and phlegm production. In all but two cases of bronchitis, air samples revealed chromic acid at levels of 0.03 mg/m^3. Workers were asked to fill out questionnaires to assess respiratory problems. Self-reporting poses a problem in that the symptoms and respiratory health problems identified were not medically confirmed by physicians. Workers in this study believe they were developing bronchitis, but it is not clear from this study whether the development of bronchitis was confirmed by physicians. It is also difficult to assess the bronchitis health effects of chromic acid from this study because the study results for the exposed (28%) and control groups (23%) were similar.

Alderson *et al.* reported 39 deaths of chromate production workers related to chronic bronchitis from three chromate producing factories (Bolton, Eaglescliffe, and Rutherglen) from 1947 to 1977 (Ex. 35–2). The specific Cr(VI) compound, extent, and frequency that the workers were exposed to were not specified. However, workers at all three factories were exposed to sodium chromate, chromic acid, and calcium chromate at one time or another. The authors did not find an excess number of number of bronchitis related deaths at the Bolton and Eaglescliffe factories. At Rutherglen, there was an excess number of deaths (31) from chronic bronchitis with a ratio of observed/expected of 1.8 ($p<0.001$). It is difficult to assess the respiratory health effects of Cr(VI) compounds from this study because there are no exposure data, there are no data on smoking habits, nor is it clear on the extent, duration, and amount of specific Cr(VI) compound the workers were exposed to during the study.

While the evidence for bronchitis is limited, evidence from experimental animals demonstrate that Cr(VI) compounds can cause lung irritation, inflammation in the lungs, and possibly lung fibrosis at various exposure levels. Glaser *et al.* examined the effects of inhalation exposure of chromium (VI) on lung inflammation and alveolar macrophage function in rats (Ex. 31–18–9). Twenty, 5-week old male TNO–W–74 Wistar rats were exposed via

inhalation to 25–200 µg Cr(VI)/m^3 as sodium dichromate for 28 days or 90 days for 22 hours per day, 7 days per week in inhalation chambers. Twenty, 5-week old male TNO–W–74 Wistar rats also served as controls. All rats were killed at the end of the inhalation exposure period. The authors found increased lung weight in the 50–200 µg/m^3 groups after the 90-day exposure period. They also found that 28-day exposure to levels of 25 and 50 µg/m^3 resulted in ''activated'' alveolar macrophages with stimulated phagocytic activities. A more pronounced effect on the activation of alveolar macrophages was seen during the 90-day exposure period of 25 and 50 µg/m^3.

Glaser *et al.* exposed 150 male, 8-week old Wistar rats (10 rats per group) continuously by inhalation to aerosols of sodium dichromate at concentrations of 50, 100, 200, and 400 µg Cr(VI)/m^3 for 22 hours per day, 7 days a week, for continuous exposure for 30 days or 90 days in inhalation chambers (Ex. 31–18–11). Increased lung weight changes were noticeable even at levels as low as 50 and 100 µg Cr(VI)/m^3 following both 30 day and 90 day exposures. Significant accumulation of alveolar macrophages in the lungs was noted in all of the exposure groups. Lung fibrosis occurred in eight rats exposed to 100 µg Cr(VI)/m^3 or above for 30 days. Most lung fibrosis disappeared after the exposure period had ceased. At 50 µg Cr(VI)/m^3 or higher for 30 days, a high incidence of hyperplasia was noted, possibly in response to Cr(VI)—induced damage to the lung and respiratory tract. The total protein in bronchoalveolar lavage (BAL) fluid, albumin in BAL fluid, and lactate dehydrogenase in BAL fluid were significant at elevated levels of 200 and 400 µg Cr(VI)/m^3 in both the 30 day and 90 day exposure groups (as compared to the control group). These responses are indicative of severe injury in the lungs of animals exposed to these Cr(VI) dose levels. At levels of 50 and 100 µg Cr(VI)/m^3, the responses are indicative of inflammatory changes in the lungs. The authors concluded that these results suggest that the severe inflammatory reaction may lead to more chronic and obstructive lesions in the lung, and that inflammation is essential for the induction of most effects observed following inhalation exposure.

4. Summary

Overall, there is convincing evidence to indicate that Cr(VI) exposed workers can develop nasal irritation, nasal ulcerations, nasal perforations, and asthma. There is also some limited evidence that bronchitis may occur when exposed to Cr(VI) compounds at high levels. Most of the studies involved exposure to water-soluble Cr(VI) compounds. It is very clear that workers may develop nasal irritations, nasal ulcerations, and nasal perforations at levels below the current PEL of 52 µg/m^3. However, it is not clear what occupational exposure levels lead to disorders like asthma and bronchitis.

There are numerous studies in the literature showing nasal irritations, nasal perforations, and nasal ulcerations resulting from Cr(VI) inhalation exposure. It also appears that direct hand-to-nose contact (*i.e.,* by touching inner nasal surfaces with contaminated fingers) can contribute to the incidence of nasal damage. Additionally, some studies show that workers developed these nasal health problems because they did not wear any PPE, including respiratory protection. Inadequate area ventilation and sanitation conditions (lack of cleaning, dusty environment) probably contributed to the adverse nasal effects.

There are numerous well documented case reports in the literature describing occupational asthma specifically triggered by Cr(VI) in sensitized workers. However, OSHA is not aware of any data from the literature to determine a Cr(VI) dose in the work place that leads to the asthmatic condition or to determine how many people may be affected by such Cr(VI) exposure.

The evidence that workers breathing Cr(VI) can develop respiratory disease that involve inflammation, such as asthma and bronchitis is supported by experimental animal studies. The 1985 and 1990 Glaser *et al.* studies show that animals experience irritation and inflammation of the lungs following repeated exposure by inhalation to water-soluble Cr(VI) at air concentrations near the current PEL.

D. Dermal Effects

Occupational exposure to Cr(VI) is a well-established cause of adverse health effects of the skin. The effects are the result of two distinct processes: (1) Irritant reactions, such as skin ulcers and irritant contact dermatitis, and (2) delayed hypersensitivity (allergic) reactions. Some evidence also indicates that exposure to Cr(VI) compounds may cause conjunctivitis.

The mildest skin reactions consist of erythema (redness), edema (swelling), papules (raised spots), vesicles (liquid spots), and scaling (Ex. 35–313, p. 295). The lesions are typically found on exposed areas of the skin, usually the hands and forearms (Exs. 9–9; 9–25). These features are common to both irritant and allergic contact dermatitis, and it is generally not possible to determine the etiology of the condition based on histopathologic findings (Ex. 35–314). Allergic contact dermatitis can be diagnosed by other methods, such as patch testing (Ex. 35–321, p. 226). Patch testing involves the application of a suspected allergen to the skin, diluted in petrolatum or some other vehicle. The patch is removed after 48 hours and the skin examined at the site of application to determine if a reaction has occurred.

Cr(VI) compounds can also have a corrosive, necrotizing effect on living tissue, forming ulcers, or ''chrome holes'' (Ex. 35–315). This effect is apparently due to the oxidizing properties of Cr(VI) compounds (Ex. 35–318, p. 623). Like dermatitis, chrome ulcers generally occur on exposed areas of the body, chiefly on the hands and forearms (Ex. 35–316). The lesions are initially painless, and are often ignored until the surface ulcerates with a crust which, if removed, leaves a crater two to five millimeters in diameter with a thickened, hardened border. The ulcers can penetrate deeply into tissue and become painful. Chrome ulcers may penetrate joints and cartilage (Ex. 35–317, p. 138). The lesions usually heal in several weeks if exposure to Cr(VI) ceases, leaving a flat, atrophic scar (Ex. 35–318, p.623). If exposure continues, chrome ulcers may persist for months (Ex. 7–3).

It is generally believed that chrome ulcers do not occur on intact skin (Exs. 35–317, p. 138; 35–315; 35–25). Rather, they develop readily at the site of small cuts, abrasions, insect bites, or other injuries (Exs. 35–315; 35–318, p. 138). In experimental work on guinea pigs, Samitz and Epstein found that lesions were never produced on undamaged skin (Ex. 35–315). The degree of trauma, as well as the frequency and concentration of Cr(VI) application, was found to influence the severity of chrome ulcers.

The development of chrome ulcers does not appear to be related to the sensitizing properties of Cr(VI). Edmundson provided patch tests to determine sensitivity to Cr(VI) in 56 workers who exhibited either chrome ulcers or scars (Ex. 9–23). A positive response to the patch test was found in only two of the workers examined.

Parkhurst first identified Cr(VI) as a cause of allergic contact dermatitis in 1925 (Ex. 9–55). Cr(VI) has since been confirmed as a potent allergen. Kligman (1966) used a maximization test (a skin test for screening possible contact allergens) to assess the skin sensitizing potential of Cr(VI) compounds (Ex. 35–

327). Each of the 23 subjects was sensitized to potassium dichromate. On a scale of one to five, with five being the most potent allergen, Cr(VI) was graded as five (i.e., an extreme sensitizer). This finding was supported by a guinea pig maximization test, which assigned a grade of four to potassium chromate using the same scale (Ex. 35–328).

1. Prevalence of Dermal Effects

Adverse skin effects from Cr(VI) exposure have been known since at least 1827, when Cumin described ulcers in two dyers and a chromate production worker (Ex. 35–317, p. 138). Since then, skin conditions resulting from Cr(VI) exposure have been noted in a wide range of occupations. Work with cement is regarded as the most common cause of Cr(VI)-induced dermatitis (Exs. 35–313, p. 295; 35–319; 35–320). Other types of work where Cr(VI)-related skin effects have been reported include chromate production, chrome plating, leather tanning, welding, motor vehicle assembly, manufacture of televisions and appliances, servicing of railroad locomotives, aircraft production, and printing (Exs. 31–22–12; 7–50; 9–31; 9–100; 9–63; 9–28; 9–95; 9–54; 35–329; 9–97; 9–78; 9–9; 35–330). Some of the important studies on Cr(VI)-related dermal effects in workers are described below.

a. *Cement Dermatitis.* Many workers develop cement dermatitis, including masons, tile setters, and cement workers (Ex. 35–318, p. 624). Cement, the basic ingredient of concrete, may contain several possible sources of chromium (Exs. 35–317, p.148; 9–17). Clay, gypsum, and chalk that serve as ingredients may contain traces of chromium. Ingredients may be crushed using chrome steel grinders that, with wear, contribute to the chromium content of the concrete. Refractory bricks in the kiln and ash residues from the burning of coal or oil to heat the kiln serve as additional sources. Trivalent chromium from these sources can be converted to Cr(VI) in the kiln (Ex. 35–317. p. 148).

Cement dermatitis can be caused by direct irritation of the skin, by sensitization to Cr(VI), or both (Ex. 35–317, p. 147). However, sensitization is considered to be of greater importance than irritation in causing cement dermatitis (Ex. 35–317, p. 147). Burrows (1983) combined the results of 16 separate studies to report that, on average, over 80% of cement dermatitis cases were found to be sensitized to Cr(VI) (Ex. 35–317, p. 148). Cement is alkaline, abrasive, and hydroscopic (water-absorbing), and it is likely that the irritant effect resulting from these properties interferes with the skin's defenses, permitting penetration and sensitization to take place more readily (Ex. 35–318, p. 624). Dry cement is considered relatively innocuous because it is not as alkaline as wet cement (Exs. 35–317, p. 147; 9–17). When water is mixed with cement the water liberates calcium hydroxide, causing a rise in pH (Ex. 35–317, p. 147).

Flyvholm *et al.* (1996) noted a correlation between the Cr(VI) concentration in the local cement and the frequency of allergic contact dermatitis (Ex. 35–326, p. 278). Because the Cr(VI) content depends partially upon the chromium concentration in raw materials, there is a great variability in the Cr(VI) content in cement from different geographical regions. In locations with low Cr(VI) content, the prevalence of Cr(VI)-induced allergic contact dermatitis was reported to be approximately one percent, while in regions with higher chromate concentrations the prevalence was reported to rise to between 9 to 11% of those exposed (Ex. 35–326, p. 278).

The relationship between Cr(VI) content in cement and the prevalence of Cr(VI)-induced allergic contact dermatitis is supported by the findings of Avnstorp (1989) in a study of Danish workers who had daily contact with wet cement during the manufacture of pre-fabricated concrete products (Ex. 9–131). Beginning in September of 1981, low concentrations of ferrous sulfate were added to all cement sold in Denmark to reduce Cr(VI) to trivalent chromium. Two hundred and twenty seven workers were examined in 1987 for Cr(VI)-related skin effects. The findings from these examinations were compared to the results from 190 workers in the same plants who were examined in 1981. The prevalence of hand eczema had declined from 11.7% to 4.4%, and the prevalence of Cr(VI) sensitization had declined from 10.5% to 2.6%. Both of these results were statistically significant. There was no significant change in the frequency of skin irritation.

b. *Dermatitis Associated With Cr(VI) From Sources Other Than Cement.* In 1953 the U.S. Public Health Service reported on hazards associated with the chromium-producing industry in the United States (Ex. 7–3). Workers were examined for skin effects from Cr(VI) exposure. Workers' eyes were also examined for possible effects from splashes of Cr(VI)-containing compounds that had been observed in the plants. Of the 897 workers examined, 451 had skin ulcers or scars of ulcers. Seventeen workers were reported to have skin lesions suggestive of chrome dermatitis. The authors noted that most plants provided adequate washing facilities, and had facilities for providing clean work clothes. A statistically significant increase in congestion of the conjunctiva was also reported in Cr(VI)-exposed workers when compared with non-exposed workers (38.7% vs. 25.8%).

In the Baltimore, Maryland chromate production plant examined by Gibb *et al.* (2000), a substantial number of workers were reported to have experienced adverse skin effects (Ex. 31–22–12). The authors identified a cohort of 2,357 workers first employed at the plant between 1950 and 1974. Clinic and first aid records were examined to identify findings of skin conditions. These clinical findings were identified by a physician as a result of routine examinations or visits to the medical clinic by members of the cohort. Percentages of the cohort with various clinical findings were as follows:

Irritated skin: 15.1%
Dermatitis: 18.5%
Ulcerated skin: 31.6%
Conjunctivitis: 20.0%

A number of factors make these results difficult to interpret. The reported findings are not specifically related to Cr(VI) exposure. They may have been the result of other workplace exposures, or non-workplace factors. The report also indicates the percentage of workers who were diagnosed with a condition during their tenure at the plant; however, no information is presented to indicate the expected incidence of these conditions in a population that is not exposed to Cr(VI).

Measurements of Cr(VI) air concentrations by job title were used to estimate worker exposures. Based on these estimates, the authors used a proportional hazards model to find a statistically significant correlation (p=0.004) between ulcerated skin and airborne Cr(VI) exposure. Statistically significant correlations between year of hire and findings of ulcerated skin and dermatitis were also reported. Exposures to Cr(VI) in the plant had generally dropped over time. Median exposure to Cr(VI) at the time of occurrence for most of the findings was said to be about 10 $\mu g/m^3$ Cr(VI) (reported as 20 $\mu g/m^3$ CrO^3). It is unclear, however, what contribution airborne Cr(VI) exposures may have had to dermal effects. Direct dermal contact with Cr(VI) compounds in the plant may have been a contributing factor in the development of these conditions.

Mean and median times on the job prior to initial diagnosis were also

reported. The mean time prior to diagnosis of skin or eye effects ranged from 373 days for ulcerated skin to 719 days for irritated skin. Median times ranged from 110 days for ulcerated skin to 221 days for conjunctivitis. These times are notable because many workers in the plant stayed for only a short time. Over 40% worked for less than 90 days. Because these short-term workers did not remain in the workplace for the length of time that was typically necessary for these effects to occur, the results of this study may underestimate the incidence that would occur with a more stable worker population.

Lee and Goh (1988) examined the skin condition of 37 workers who maintained chrome plating baths and compared these workers with a group of 37 control subjects who worked in the same factories but were not exposed to Cr(VI) (Ex. 35–316). Mean duration of employment as a chrome plater was 8.1 (SD±7.9) years. Fourteen (38%) of the chrome platers had some occupational skin condition; seven had chrome ulcers, six had contact dermatitis and one had both. A further 16 (43%) of the platers had scars suggestive of previous chrome ulcers. Among the control group, no members had ulcers or scars of ulcers, and three had dermatitis.

Where ulcers or dermatitis were noted, patch tests were administered to determine sensitization to Cr(VI) and nickel. Of the seven workers with chrome ulcers, one was allergic to Cr(VI). Of the six workers with dermatitis, two were allergic to Cr(VI) and one to nickel. The worker with ulceration and dermatitis was not sensitized to either Cr(VI) or nickel. Although limited by a relatively small study population, this report clearly indicates that Cr(VI)-exposed workers face an increased risk of adverse skin effects. The fact that the majority of workers with dermatitis were not sensitized to Cr(VI) indicates that irritant factors play an important role in the development of dermatitis in chrome plating operations.

Royle (1975) also investigated the occurrence of skin conditions among workers involved in chrome plating (Ex. 7–50). A questionnaire survey completed by 997 chrome platers revealed that 21.8% had experienced skin ulcers, and 24.6% had suffered from dermatitis. No information was presented to indicate the expected incidence in a comparable population that was not exposed to Cr(VI). Of the 54 plants involved in the study, 49 used nickel, another recognized cause of allergic contact dermatitis.

The author examined the relationship between the incidence of these conditions and length of exposure. The plater population was divided into three groups: those with less than one year of Cr(VI) exposure, those with one to five years of Cr(VI) exposure, and those with over five years of Cr(VI) exposure. A statistically significant trend was found between length of Cr(VI) exposure and incidence of skin ulcers. The incidence of dermatitis, on the other hand, bore no relationship to length of exposure.

In 1973, researchers from NIOSH reported on the results of a health hazard investigation of a chrome plating establishment (Ex. 3–5). In the plating area, airborne Cr(VI) concentrations ranged from less than 0.71 up to 9.12 $\mu g/m^3$ (mean 3.24 $\mu g/m^3$; SD=2.48 $\mu g/m^3$). Of the 37 exposed workers who received medical examinations, five were reported to have chrome-induced lesions on their hands. Hygiene and housekeeping practices in this facility were reportedly deficient, with the majority of workers not wearing gloves, not washing their hands before eating or leaving the plant, and consuming food and beverages in work areas.

Gomes (1972) examined Cr(VI)-induced skin lesions among electroplaters in Sao Paulo, Brazil (Ex. 9–31). A clinical examination of 303 workers revealed 88 (28.8%) had skin lesions, while 175 (58.0%) had skin and mucus membrane lesions. A substantial number of employers (26.6%) also did not provide personal protective equipment to workers. The author attributed the high incidence of skin ulcers on the hands and arms to inadequate personal protective equipment, and lack of training for employees regarding hygiene practices.

Fleeger and Deng (1990) reported on an outbreak of skin ulcerations among workers in a facility where enamel paints containing chromium were applied to kitchen range parts (Ex. 9–97). A ground coat of paint was applied to the parts, which were then placed on hooks and transported through a curing oven. In some cases, small parts were places on hooks before paint application. Tiny holes in the oven coils apparently resulted in improper curing of the paint, leaving sharp edges and a Cr(VI)-containing residue on the hooks. Most of the workers who handled the hooks reportedly did not wear gloves, because the gloves were said to reduce dexterity and decrease productivity. As a result, cuts from the sharp edges allowed the Cr(VI) to penetrate the skin, leading to ulcerations (Ex. 9–97).

2. Prognosis of Dermal Effects

Cr(VI)-related dermatitis tends to become more severe and persistent with continuing exposure. Once established, the condition may persist even if occupational exposure ceases. Fregert followed up on cases of occupational contact dermatitis diagnosed over a 10-year period by a dermatology service in Sweden. Based on responses to questionnaires completed two to three years after treatment, only 7% of women and 10% of men with Cr(VI)-related allergic contact dermatitis were reported to be healed (Ex. 35–322). Burrows reviewed the condition of patients diagnosed with work-related dermatitis 10–13 years earlier. Only two of the 25 cases (8%) caused by exposure to cement had cleared (Ex. 35–323).

Hogan *et al.* reviewed the literature regarding the prognosis of contact dermatitis, and reported that the majority of patients had persistent dermatitis (Ex. 35–324). Job changes reportedly did not usually lead to a significant improvement for most patients. The authors surveyed contact dermatitis experts around the world to explore their experience with the prognosis of patients suffering from occupational contact dermatitis of the hands. Seventy-eight percent of the 51 experts who responded to the survey indicated that chromate was one of the allergens associated with the worst possible prognosis.

Halbert *et al.* reviewed the experience of 120 patients diagnosed with occupational chromate dermatitis over a 10-year period (Ex. 35–320). The time between initial diagnosis and the review ranged from a minimum of six months to a maximum of nine years. Eighty-four (70%) of patients were reviewed two or more years after initial diagnosis, and 40 (33%) after five years or more. In the majority of cases (78, or 65%), the dermatitis was attributed to work with cement. For the study population as a whole, 76% had ongoing dermatitis at the time of the review.

When the review was conducted, 62 (58%) patients were employed in the same occupation as when initially diagnosed. Fifty-five (89%) of these workers continued to suffer from dermatitis. Fifty-eight patients (48%) changed occupations after their initial diagnosis. Each of these individuals indicated that they had changed occupations because of their dermatitis. In spite of the change, dermatitis persisted in 40 members of this group (69%).

Lips *et al.* found a somewhat more favorable outcome among 88 construction workers with occupational chromate dermatitis who were removed from Cr(VI) exposure (Ex. 35–325). Follow-up one to five years after removal indicated that 72% of the patients no longer had dermatitis. The

authors speculated that this result might be due to strict avoidance of Cr(VI) contact. Nonetheless, the condition persisted in a substantial portion of the affected population.

3. Thresholds for Dermal Effects

In a response to OSHA's RFI submitted on behalf of the Chrome Coalition, Exponent indicated that the findings of Fowler *et al.* (1999) and others provide evidence of a threshold for elicitation of allergic contact dermatitis (Ex. 31–18–1, p. 27). Exponent also stated that because chrome ulcers did not develop in the Fowler *et al.* study, ''more aggressive'' exposures appear to be necessary for the development of chrome ulcers.

The Fowler *et al.* study involved the dermal exposure of 26 individuals previously sensitized to Cr(VI) who were exposed to water containing 25 to 29 mg/L Cr(VI) as potassium dichromate (pH 9.4) (Ex. 31–18–5). Subjects immersed one arm in the Cr(VI) solution, while the other arm was immersed in an alkaline buffer solution as a control. Exposure lasted for 30 minutes and was repeated on three consecutive days. Based on examination of the skin, the authors concluded that the skin response experienced by subjects was not consistent with either irritant or allergic contact dermatitis.

The exposure scenario in the Fowler *et al.* study, however, does not mimic the occupational experience. While active dermatitis, scratches, and skin lesions served as criteria for excluding both initial and continuing participation in the study, it is reasonable to expect that individuals with these conditions will often continue to work. Cr(VI)-containing mixtures and compounds used in the workplace may also pose a greater challenge to the integrity of the skin than the solution used by Fowler *et al.* Wet cement, for example, may have a pH higher than 9.4, and may be capable of abrading or otherwise damaging the skin. As damaged skin is liable to make exposed workers more susceptible to Cr(VI)-induced skin effects, the suggested threshold is likely to be invalid. The absence of chrome ulcers in the Fowler *et al.* study is not unexpected, because subjects with ''fissures or lesions'' on the skin were excluded from the study (Ex. 31–18–5). As discussed earlier, chrome ulcers are not believed to occur on intact skin.

4. Preliminary Conclusions

OSHA believes that adverse dermal effects from exposure to Cr(VI), including irritant contact dermatitis, allergic contact dermatitis, and skin ulceration, have been firmly established. The available evidence is not sufficient to relate these effects to any given Cr(VI) air concentration. Rather, it appears that direct dermal contact with Cr(VI) is the most relevant factor in the development of dermatitis and ulcers. Based on the findings of Gibb *et al.* (Ex. 32–22–12) and U.S. Public Health Service (Ex. 7–3), OSHA also considers it likely that conjunctivitis can result from eye contact with Cr(VI).

OSHA does not believe that the available evidence is sufficient to establish a threshold concentration of Cr(VI) below which dermal effects will not occur in the occupational environment. This preliminary finding is supported not only by the belief that the exposure scenario of Fowler *et al.* is not consistent with occupational exposures, but by experience in the workplace as well. As summarized by Flyvholm *et al.* (1996), numerous reports have indicated that allergic contact dermatitis occurs in cement workers exposed to Cr(VI) concentrations below the threshold suggested by Fowler *et al.* (1999). OSHA considers the evidence of Cr(VI)-induced allergic contact dermatitis in these workers to indicate that the threshold for elicitation of response suggested by Fowler *et al.* (1999) is not applicable to the occupational environment.

E. Other Health Effects

OSHA has examined the possibility of health effect outcomes associated with Cr(VI) exposure in addition to such effects as lung cancer, nasal ulcerations and perforations, occupational asthma, and irritant and allergic contact dermatitis. Unlike the Cr(VI)-induced toxicities cited above, the data on other health effects do not definitively establish Cr(VI)-related impairments of health from occupational exposure at or below the current OSHA PEL.

There is some positive evidence that workplace inhalation to Cr(VI) results in gastritis and gastrointestinal ulcers, especially at high exposures (generally over OSHA's current PEL) (Ex. 7–12). This is supported by ulcerations in the gastrointestinal tract of mice breathing high Cr(VI) concentration for long periods (Ex. 10–8). Other studies reported positive effects but significant information was not reported or the confounders made it difficult to draw positive conclusions (Ex. 3–84; Sassi 1956 as cited in Ex. 35–41). Other studies reported negative results (Exs. 7–14; 9–135).

Likewise, several studies reported increases in renal proteins in the urine of chromate production workers and chrome platers (Exs. 35–107; 5–45; 35–105; 5–57). The Cr(VI) air levels recorded in these workers were usually below the current OSHA PEL (Exs. 35–107; 5–45). Workers with the highest urinary chromium levels tended to also have the largest elevations in renal markers (Ex. 35–107). One study reported no relationship between chromium in urine and renal function parameters, no relationship with age or with duration of exposure, and no relationship between the presence of chromium skin ulcers and chromium levels in urine or renal function parameters (Ex. 5–57). In most studies, the elevations renal protein levels were restricted to only one or two proteins out of several examined per study, generally exhibited small increases (Ex. 35–105) and the effects appeared to be reversible (Ex. 5–45). It has been stated that low molecular weight proteinuria can occur from other reasons and cannot by itself be considered evidence of chronic renal disease (Ex. 35–195). Other studies reported no changes in renal markers (Exs. 7–27; 35–104) and animal inhalation studies did not report kidney damage (Exs. 9–135; 31–18–11; 10–11; 31–18–10; 10–10). Some studies with Cr(VI) administered by drinking water or gavage were positive for increases in renal markers, and some cell and tissue damage (Exs. 9–143; 11–10). However, it is not clear how to extrapolate such findings to workers exposed to Cr(VI) via inhalation. Well designed studies of effects in humans via ingestion were not found.

OSHA did not find information to clearly and sufficiently demonstrate that exposures to Cr(VI) result in significant impairment to the hepatic system. Two European studies, positive for an excess of deaths from cirrhosis of the liver and hepatobiliarity disorders, were not able to separate chromium exposures from exposures to the many other substances present in the workplace. The authors also could not rule out the role of alcohol use as a possible contributor to the disorder (Ex. 7–92; Sassi as cited in Ex. 35–41). Other studies did not report any hepatic abnormalities (Exs. 7–27; 10–11).

The reproductive studies showed mixed results. Some positive reproductive effects occurred in some welding studies. However, it is not clear that Cr(VI) is the causative agent in these studies (Exs. 35–109; 35–110; 35–108; 35–202; 35–203). Other positive studies were seriously lacking in information. Information was not given on exposures, the nature of the reproductive complications, or the women's tasks (Shmitova 1980, 1978 as cited in Ex. 35–41, p. 52). ATSDR states that because these studies were

generally of poor quality and the results were poorly reported, no conclusions can be made on the potential for chromium to produce adverse reproductive effects in humans (Ex. 35–41, p.52). In animal studies, where Cr(VI) was administered through drinking water or diet, positive developmental effects occurred in offspring (Exs. 9–142; 35–33; 35–34; 35–38). However, the doses administered in drinking water or given in the diet were high (i.e., 250, 500, and 750 ppm). Furthermore, strong studies showing reproductive or developmental effects in other situations where employees were working exclusively with Cr(VI) were not found. In fact, the National Toxicology Program (NTP) (Exs. 35–40; 35–42; 35–44) conducted an extensive multigenerational reproductive assessment by continuous breeding where the chromate was administered in the diet. The assessment yielded negative results (Exs. 35–40; 35–42; 35–44). Animal inhalation studies were negative (Exs. 35–199; 9–135; 10–10; Glaser 1984 as cited in Ex. 31–22–33;). Thus, it cannot be concluded that Cr(VI) is a reproductive toxin for normal working situations.

VII. Preliminary Quantitative Risk Assessment

A. Introduction

The Occupational Safety and Health (OSH) Act and some landmark court cases have led OSHA to rely on quantitative risk assessment, where possible, to support the risk determinations required to set a permissible exposure limit (PEL) for a toxic substance in standards under the OSH Act. Section 6(b)(5) of the Act states that ''The Secretary [of Labor], in promulgating standards dealing with toxic materials or harmful 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.'' (29 U.S.C. 651 *et seq.*)

In a further interpretation of the risk requirements for OSHA standard setting, the United States Supreme Court, in the 1980 ''benzene'' decision, (*Industrial Union Department, AFL–CIO* v. *American Petroleum Institute,* 448 U.S. 607 (1980)) ruled that the OSH Act requires that, prior to the issuance of a new standard, a determination must be made that there is a significant risk of material impairment of health at the existing PEL and that issuance of a new standard will significantly reduce or eliminate that risk. 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. 642]. The Court also stated ''that the Act does not limit the Secretary's power to require the elimination of significant risks'' [488 U.S. 644]. While the Court indicated that the use of quantitative risk analysis was an appropriate means to establish significant risk, they made clear that ''OSHA is not required to support its finding that a significant risk exists with anything approaching scientific certainty.''

Although the Court in the Cotton Dust case, (*American Textile Manufacturers Institute* v. *Donovan,* 452 U.S. 490 (1981)) rejected the use of cost-benefit analysis in setting OSHA standards, it reaffirmed its previous position in the ''benzene'' case that a risk assessment is not only appropriate but should be used to identify significant health risk in workers and to determine if a proposed standard will achieve a reduction in that risk. Although the Court did not require OSHA to perform a quantitative risk assessment in every case, the Court implied, and OSHA as a matter of policy agrees, that assessments should be put into quantitative terms to the extent possible.

The determining factor in the decision to perform a quantitative risk assessment is the availability of suitable data for such an assessment. As reviewed in section VI.B. on Carcinogenic Effects, there are a substantial number of occupational cohort studies that reported excess lung cancer mortality in workers exposed to Cr(VI) in several industrial operations. Many of these found that workers exposed to higher levels of airborne Cr(VI) for a longer period of time had greater standardized mortality ratios (SMRs) for lung cancer. OSHA believes two recently studied occupational cohorts have the strongest data sets on which to quantify lung cancer risk from cumulative Cr(VI) exposure (i.e., air concentration x exposure duration). Using a linear relative risk model on these data to predict excess lifetime risk, OSHA preliminarily estimates that the lung cancer risk from a 45 year occupational exposure to Cr(VI) at an 8-hour TWA at the current PEL of 52 μg/m^3 is 106 to 334 excess deaths per 1000. Quantitative lifetime risk estimates from a working lifetime exposure at several lower alternative PELs under consideration by the Agency are also estimated. For example, the projected risk at 0.5 μg/m^3 Cr(VI) is 1.1 to 4.3 per 1000. The sections below discuss the selection of the appropriate data sets and risk models, the estimation of lung cancer risks based on the selected data sets and models, the uncertainty in the risk estimates, the key issues that arise as result of the quantitative risk assessment as well as a summary describing comments from an expert peer review and the OSHA response.

In contrast to the more extensive occupational cohort data on Cr(VI) exposure-response, data from experimental animal studies are less suitable for quantitative risk assessment of lung cancer than human studies. Besides the obvious species difference, most of the animal studies administered Cr(VI) to the respiratory tract by less relevant routes, such as instillation or implantation. The few available inhalation studies in animals were limited by a combination of inadequate exposure levels, abbreviated durations, and small numbers of animals per dose group. Despite these limitations, the animal data do provide semi-quantitative information with regard to the relative carcinogenic potency of different Cr(VI) compounds. A more detailed discussion can be found in section VI.B.7.

The data that relate non-cancer health impairments, such as damage to the respiratory tract and skin, to Cr(VI) exposure are also not well suited for quantitative assessment. There are some data from cross-sectional studies and worker surveys that group the prevalence and severity of nasal damage by contemporary time-weighted average (TWA) Cr(VI) air measurements. However, there are no studies that track either incidence or characterize exposure over time. Nasal damage is also more likely influenced by shorter-term peak exposures that have not been as well characterized. While difficult to quantitate, the data indicate that the risk of damage to the nasal mucosa would be significantly reduced by lowering the current PEL, discussed further in section VIII on Significance of Risk.

There are even less suitable exposure-response data to assess risk for other Cr(VI)-induced impairments (*e.g.*, mild renal damage, gastrointestinal ulceration). With the possible exception of respiratory tract effects (*e.g.*, nasal damage, occupational asthma), the risk of non-cancer adverse effects that result from inhaling Cr(VI) are expected to be very low except as a result of long-term regular airborne exposure around or above the current PEL (52 μg/m^3). Since

the non-cancer effects occur at relatively high Cr(VI) air concentrations, OSHA believes that lowering the PEL to reduce the risk of developing lung cancer over a working lifetime would also eliminate or reduce the risk of developing these other health impairments. As discussed in section VI.E., adverse effects to the skin primarily result from dermal rather than airborne exposure.

B. Study Selection

The more than 40 occupational cohort studies reviewed in Section VI.B on carcinogenic effects were evaluated to determine the adequacy of the exposure-response information for the quantitative assessment of lung cancer risk associated with Cr(VI) exposure. The key criteria were data that allowed for estimation of input variables, specifically levels of exposure and duration of exposure (*e.g.*, cumulative exposure in $mg/m^3 - yr$); observed numbers of cancers (deaths or incident cases) by exposure category; and expected (background) numbers of cancer deaths by exposure category.

Additional criteria were applied to evaluate the strengths and weaknesses of the available epidemiological data sets. Studies needed to have well-defined cohorts with identifiable cases. Features such as cohort size and length of follow-up affect the ability of the studies to detect any possible effect of Cr(VI) exposure. Potential confounding of the responses due to other exposures was considered. Study evaluation also considered whether disease rates from an appropriate reference population were used to derive expected numbers of lung cancers. One of the most important factors in study evaluation was the ascertainment and use of exposure information (i.e., well-documented historical exposure data). Both level and duration of exposure are important in determining cumulative dose, and studies are often deficient with respect to the availability or use of such information. Evidence of exposure-response relationship was also important.

Two recently studied cohorts of chromate production workers were found to be the strongest data sets for quantitative assessment (Exs. 31–22–11; 33–10). Of the various studies, these two had the most extensive and best documented Cr(VI) exposures spanning three or four decades. Both cohort studies characterized observed and expected lung cancer mortality and reported a statistically significant positive association between lung cancer risk and cumulative Cr(VI) exposure. Four other cohorts had less satisfactory data for quantitative assessments of lung cancer risk (Exs. 7–11; 23; 7–14; 7–120; 31–16–3). While the lung cancer response in these cohorts was stratified across multiple exposure groups, there were limitations to these data that affected the certainty of the risk projections. The cohorts include chromate production workers, stainless steel welders, and aerospace manufacturing workers. Risk estimates from these lesser cohorts were used to examine the robustness of the more precise estimates from the Gibb and Luippold cohorts. The strengths and weaknesses of all six cohorts in terms of their use in exposure-response analysis are discussed in more detail below. Emphasis has been placed on the quantitative information available for each cohort.

Three other cohort studies that were used in the past to develop crude risk estimates from worker exposure to Cr(VI) are not being relied upon in the present assessment and therefore are not reviewed below (Exs. 7–37; 7–62; 7–95). In these cohorts, risk estimates were determined from background lung cancer rates and excess lung cancer mortality associated with a single, rather than multiple Cr(VI) exposure levels. There were also a number of other limitations to the study data that required the use of unsupported assumptions and raised uncertainties in the risks. The exposure-response data from the three studies and the resulting assessments are discussed in the 1995 report from the K.S. Crump Division (Ex. 13–5). OSHA believes the recent availability of several higher quality cohort studies cited above eliminates the need to rely on these more problematic cohorts to assess lung cancer risk from occupational Cr(VI) exposure.

1. Gibb Cohort

The Gibb et al. study was one of the stronger studies for quantitative risk assessment, especially in terms of cohort size, historical exposure data, and evidence of exposure-response (Exs. 31–22–11; 33–11). Gibb et al. studied an updated cohort from the same Baltimore chromate production plant previously studied by Hayes et al. (see section VII.B.4). The cohort consisted of 2357 male workers (white and non-white) first employed between 1950 and 1974. Follow-up was through the end of 1992 for a total of 70,736 person-years and an average length of 30 years per member. Smoking status at the start of employment was available for 91% of the cohort members.

A significant advantage of the Gibb data was the sizable amount of personal and area sampling measurements from a variety of locations and job titles collected concurrently over the years during which the cohort members were exposed (from 1950 to 1985, when the plant closed). Using these concentration estimates as the basis, a job exposure matrix was constructed giving annual average exposures by job title. Based on the job exposure matrix and work histories for the cohort members, Gibb et al. computed the person-years of observation, the observed numbers of lung cancer deaths, and the expected numbers of lung cancer deaths categorized by cumulative Cr(VI) exposure and age of death. They found that cumulative Cr(VI) exposure was a significant predictor of lung cancer risk over the exposure range of 0 to 2.76 ($mean \pm SD = 0.70 \pm 2.75$) mg/m^3 - yr, even with models that accounted for the smoking data at hire. This included a greater than expected number of premature lung cancer deaths in some workers. For example, chromate production workers between 40 and 50 years of age with mean cumulative Cr(VI) exposure of 0.41 mg CrO_3/m^3 - yr (equivalent to 0.21 mg Cr(VI)/m^3 - yr) were about four times more likely to die of lung cancer than a State of Maryland resident of similar age (Ex. 31–22–11, Table V).

The detailed reporting of the cumulative exposure, including mean values for four categories defined by the quartiles of cumulative exposure versus age, was another significant advantage. This level of documentation reduced some of the uncertainty associated with the estimation of cumulative exposure. Moreover, the cross-classification of cumulative exposure with age allowed the application of more elaborate models that consider the effect of age on lung cancer risk.

Since the publication of Gibb et al., the data file containing the demographic, exposure, and response data for the individual cohort members was made available (Ex. 295). These data have been used in a recent reanalysis (see subsection VII.C.1). The advantages of the study mentioned above are even greater now that the detailed cohort data can be accessed. Among other things, the exposure groups can be defined in alternative ways, the effect of considering different reference populations can be examined, and additional models can be applied in the dose-response analysis.

2. Luippold Cohort

The other well-documented exposure-response data set comes from a second cohort of chromate production workers. Luippold *et al.* studied a cohort of 482 predominantly white, male employees

who started work between 1940 and 1972 at the same Painesville, Ohio plant studied earlier by Mancuso (Ex. 33–10) (see subsection VII.B.3). Mortality status was followed through 1997 for a total of 14,048 person-years and an average length of 30 years. While the Luippold cohort was smaller and less racially diverse than the Gibb cohort, the workforce contained fewer transient, short-term employees. The Luippold cohort consisted entirely of workers employed over one year. Fifty-five percent worked for more than five years. In comparison, 65 percent of the Gibb cohort worked for less than a year and 15 percent for more than five years at the Baltimore plant. There was more limited information about the smoking behavior (smoking status available for only 35 percent of members) of the Luippold cohort than the Gibb cohort.

One aspect that the Luippold cohort had in common with the Gibb cohort was extensive and well-documented air monitoring of Cr(VI). Cr(VI) exposures for the Luippold cohort were based on 21 industrial hygiene surveys conducted at the plant between 1943 and 1971, yielding a total of more than 800 area samples (Ex. 35–61). A job exposure matrix was computed for 22 exposure areas for each month starting in 1940 and, coupled with detailed work histories available for the cohort members, cumulative exposures were calculated for each person-year of observation. The cumulative Cr(VI) exposures, which ranged from 0.003 to 23 (mean±SD = 1.58±2.50) mg Cr(VI)/m^3 − yr, were generally higher but overlapped those of the Gibb cohort.

Luippold *et al.* found significant dose-related trends for lung cancer SMRs as a function of year of hire, duration of employment, and cumulative Cr(VI) exposure. The data on exposure-response for this cohort are relatively strong. The use of individual work histories to define exposure categories and presentation of mean cumulative doses in the exposure groups provided a strong basis for a quantitative risk assessment. The higher cumulative exposure range and the longer work duration of the Luippold cohort serve to complement quantitative data available on the Gibb cohort. Risk assessments on the Luippold *et al.* study data performed by Crump *et al.* had access to the individual data and, therefore, had the best basis for analyses of this cohort (Exs. 31–18–1; 35–205; 35–58).

3. Mancuso Cohort

Mancuso (Ex. 7–11) studied the lung cancer incidence of an earlier cohort of 332 white male employees drawn from the same plant in Painesville, Ohio that was evaluated by the Luippold group. The Mancuso cohort was first employed at the facility between 1931 and 1937 and followed up through 1972, when the plant closed. Mancuso (Ex. 23) later extended the follow-up period through 1993, yielding a total of 12,881 person-years of observation for an average length of 38.8 years and a total of 66 lung cancer deaths. Since the Mancuso workers were first employed in the 1930s and the Luippold workers were first employed after 1940, the cohorts consisted of a completely different set of individuals.

A major limitation of the Mancuso study is the uncertainty of the exposure data. Mancuso relied exclusively on the air monitoring reported by Bourne and Yee (Ex. 7–98) conducted over a single short period of time during 1949. Bourne and Yee presented monitoring data as airborne insoluble chromium, airborne soluble chromium, and total airborne chromium by production department at the Painesville plant. The insoluble chromium was probably Cr(III) compounds with some slightly water-soluble and insoluble chromates. The soluble chromium was probably highly water-soluble Cr(VI). Mancuso (Exs. 7–11; 23) calculated cumulative exposures (mg/m^3 − yr) for each cohort member based on the 1949 mean chromium concentrations, by production department, under the assumption that those levels reflect exposures during the entire duration of employment for each cohort member, even though employment may have begun as early as 1931 and may have extended to 1972. Due to the lack of air measurements spanning the full period of worker exposure and the lack of adequate methodology to distinguish chromium valence states i.e., Cr(VI) vs. Cr(III)), the exposure data associated with the Mancuso cohort were not as well characterized as data from the Luippold or Gibb cohorts.

Mancuso presented observed lung cancer deaths and age-adjusted death rates stratified by age group and cumulative total, soluble and insoluble chromium exposure groups (Ex. 23). However, the study did not provide the expected numbers of lung cancers for the exposure groupings, making it more difficult to apply appropriate risk models to the data. Approaches that attempt to circumvent this limitation are discussed in subsection VII.E.1. Mancuso (Ex. 7–11; 23) reported cumulative exposure-related increases in age-adjusted lung cancer death rates for soluble, insoluble, or total chromium. Within a particular range of exposures to insoluble chromium, lung cancer death rates also tended to increase with increasing total cumulative chromium. However, the study did not report whether these tendencies were statistically significant, nor did it report the extent to which exposures to soluble and insoluble chromium were correlated. Thus, it is possible that the apparent relationship between insoluble chromium *e.g.*, primarily Cr(III)) and lung cancer may have arisen because both insoluble chromium concentrations and lung cancer death rates were positively correlated with Cr(VI) concentrations.

Although a 1995 risk assessment based on data from the 1975 Mancuso study was prepared for OSHA under contract (Ex. 13–5), it has been superseded by an updated assessment from the more complete 1997 Mancuso data (Ex. 33–15). Specific limitations with respect to quantitative risk estimation from the Mancuso cohort are discussed in section VII.E.1 on supporting risk assessments.

4. Hayes Cohort

Hayes *et al.* (Ex. 7–14) studied a cohort of employees at the same chromate production site in Baltimore examined by Gibb *et al.* The Hayes cohort consisted of 2101 male workers who were first hired between 1945 and 1974, excluding those employed for less than 90 days. The Gibb cohort had different date criteria for first employment (1950–1974) and no 90-day exclusion.

Hayes *et al.* reported SMRs for respiratory tract cancer based on workers grouped by time of hire, employment duration, and high or low exposure groups. Workers who had ever worked at an older plant facility and workers whose location of employment could not be determined were considered to have a high or questionable exposure. Workers known to have been employed exclusively at a newer renovated facility built in 1950 and 1951 were considered to have had low exposure. A dose-response was observed in the sense that higher SMRs for respiratory cancer were observed among long-term workers (workers who had worked for three or more years) than among short-term workers. Hayes *et al.* did not quantify occupational exposure to Cr(VI) at the time the cohort was studied.

Later on, Braver *et al.* (Ex. 7–17) estimated average cumulative soluble chromium, (presumed by the authors to be Cr(VI)) exposures for four subgroups of the Hayes cohort. The TWA Cr(VI) concentrations were determined from a total of 555 midget impinger air measurements that were collected at the older plant from 1945 to 1950. The

cumulative exposure for the subgroups were estimated from the yearly average Cr(VI) exposure for the entire plant and their average duration of employment rather than job-specific Cr(VI) concentrations and individual work histories. Such ''group level'' estimation of cumulative exposure is less appropriate than the estimation based on individual experiences as was done for the Gibb and Luippold cohorts. Another weakness is that exposures attributed to many workers (*e.g.*, those hired after 1950) were based on chromium measurements during an earlier period (i.e., 1949–1950).

Braver *et al.* (Ex. 7–17) discussed a number of other potential sources of uncertainty in the Cr(VI) exposure estimates, such as the possible conversion to Cr(III) during sample collection, the inability to measure insoluble forms of Cr(VI) even though soluble Cr(VI) compounds were primarily produced at the plant, and the likelihood that samples may have been collected mainly in potential problem areas. However, the biggest source of uncertainty was the assumption of rather high Cr(VI) air levels in the newly renovated facility at the Baltimore site throughout the 1950s based on measurements made 1945 to 1950 in an older facility, as explained in section VII.E.2.

5. Gerin Cohort

Gerin *et al.* (Ex. 7–120) developed a job exposure matrix that was used to quantify cumulative Cr(VI) exposures for male stainless steel welders who were part of the International Agency for Research on Cancer's (IARC) multi-center historical cohort study (Ex. 7–114). The IARC cohort included 11,092 welders for a total of 164,077 person-years. This resulted in an average of 14.8 person-years of risk for each member of the cohort. The number cohort members who were stainless steel welders, for which Cr(VI) exposures were estimated, could not be determined from their report. Gerin *et al.* used occupational hygiene surveys reported in the published literature to estimate typical eight-hour TWA Cr(VI) breathing zone concentrations for various combinations of welding processes and base metal. The resulting exposure matrix was then combined with information about individual work history, considering time and length of employment, type of welding, base metal, and ventilation status (*e.g.*, confined area, use of local exhaust ventilation, etc.) to estimate the cumulative Cr(VI) exposure.

Unfortunately, the industrial hygiene data used to develop the Gerin exposure matrix included measurements in the 1970s from only 8 of the 135 companies that employed welders in the cohort. Individual work histories were also not available for about 25 percent of the stainless steel welders. In these cases, information was assumed based on the average distribution of welding practices within the company. The lack of specific Cr(VI) air measurements and work practice information for this cohort raises questions concerning the accuracy of the exposure estimates.

Gerin *et al.* reported lung cancer mortality across four cumulative Cr(VI) exposure categories for two subcohorts of stainless steel welders; each accumulating between 7,000 and 10,000 person-years of observation. The welders were also known to be exposed to nickel, another potential lung carcinogen. There was no upward trend in lung cancer with respect to cumulative Cr(VI) exposure for either subcohort. Because of uncertainties in the exposure estimates, the lack of exposure-response, and possible confounding co-exposure to nickel, the Gerin cohort was not considered a featured data set for exposure-response assessment.

6. Alexander Cohort

Alexander *et al.* (Ex. 31–16–3) conducted a retrospective cohort study of 2429 aerospace workers employed in jobs entailing chromate exposure (*e.g.*, spray painting, sanding/polishing, chrome plating, etc.) between 1974 and 1994. The cohort included workers employed as early as 1940. Follow-up averaged a relatively short 8.9 years per cohort member.

Industrial hygiene data collected between 1974 and 1994 were used to classify jobs in categories of ''high'' exposure, ''moderate'' exposure, or ''low'' exposure to Cr(VI). The use of respiratory protection was accounted for when setting up the job exposure matrix. These exposure categories were assigned summary TWA concentrations and combined with individual job history records to estimate cumulative exposures for each person-year of observation. As further discussed in section VII.E.4, it was not clear from the study whether exposures are expressed in units of Cr(VI) or chromate (CrO_3). Exposures occurring before 1974 were assumed to be at TWA levels assigned to the interval from 1974 to 1985. The importance of the exposure assignments to the quantitative assessment of risk is further discussed in section VII.E.4.

Alexander *et al.* presented lung cancer incidence data for four cumulative chromate exposure categories based on worker duration and the three (high, moderate, low) exposure levels above. Lung cancer incidence rates were determined using a local cancer registry, part of the National Cancer Institute (NCI) Surveillance Epidemiology and End Results (SEER) program. There was no positive trend in lung cancer incidence with increasing Cr(VI) exposure. This cohort study was limited by the relatively young age of the cohort members, the short follow-up time, and lack of information on smoking. The available Cr(VI) air measurement data did not span the entire employment period of the cohort (*e.g.*, no data for 1940 to 1974) and was heavily grouped into a relatively small number of ''summary'' TWA concentrations that may not have fully captured individual differences in workplace exposures to Cr(VI). For the above reasons, the Alexander cohort was not considered as strong a data set for quantitative exposure-response analysis as the Gibb and Luippold cohorts.

7. Studies Selected for the Quantitative Risk Assessment

The epidemiologic database is quite extensive and contains several studies that have adequate data suitable for quantitative risk assessment. OSHA considers certain studies to be better suited for quantitative assessment than others. The Gibb and Luippold cohorts are considered the preferred sources for quantitative estimation because they have larger cohort sizes, extensive follow-up periods, fairly well documented historical Cr(VI) exposure levels, and because analysts have had access to the individual job histories and associated exposure matrices.

The Mancuso cohort and the Hayes cohort were derived from workers at the same plants as Luippold and Gibb, respectively, but have limitations associated with the reporting of quantitative information and exposure estimates that make them less suitable for a risk assessment. Similarly, the Gerin and Alexander cohorts are less suitable either because of the small size of the cohort, the shorter follow-up, or limitations with respect to exposure estimation. For example, the lung cancer status of the Alexander cohort had only been tracked for an average of nine years. This is in contrast to the Gibb, Luippold, and Mancuso cohorts that accumulated an average 30 or more years of observation. Long-term follow-up of cohort members is particularly important for determining the risk of lung cancer, which typically has an extended latency period of roughly twenty years. The Alexander cohort would need additional 20 years of

follow-up to achieve the person-years of observation accumulated by the Gibb cohort of about the same number of workers. The Guerin cohort is also limited by lack of follow-up, since the lung cancer status of the stainless steel welders are believed to have only been observed for an average of about 15 years.

Despite the limitations, the lesser studies each provide independent estimates of risk, albeit with more uncertainty, that can be compared to the estimates derived from the preferred data sets. OSHA believes evaluating consistency in risk among several different worker cohorts adds to the overall quality of the assessment. In light of the extensive worker exposure-response data, there is little additional value in deriving quantitative risk estimates from tumor incidence results in rodents, especially considering the concerns with regard to route of exposure and study design.

The following sections, describing the quantitative estimates of risk, start with the preferred Gibb and Luippold cohorts. The risk estimates from the supporting studies and previous risk assessments are then discussed. A discussion of remaining issues and uncertainties follows the quantitative presentation.

C. Quantitative Risk Assessments Based on the Gibb Cohort

Quantitative risk assessments have recently been performed on the exposure-response data from the Gibb cohort by three groups: Environ International (Exs. 33–15; 33–12) under contract with OSHA; the National Institute for Occupational Safety and Health (Ex. 33–13); and Exponent (Ex. 31–18–15–1) for the Chrome Coalition. All reported similar risks for Cr(VI) exposure over a working lifetime despite using somewhat different modeling approaches. The exposure-response data, risk models, statistical evaluation, and risk estimates reported by each group are discussed below.

1. Environ Risk Assessments

In 2002, Environ International (Environ) prepared a quantitative analysis of the association between Cr(VI) exposure and lung cancer (Ex. 33–15). The Environ analysis relied on a summary of the person-years of observation and observed and expected lung cancer deaths broken down by age and cumulative exposure (Ex. 31–22–11, Table V). These data are presented in Table VII–1. The job exposure matrix was the basis for the calculation of individual cumulative exposure estimates for all 2357 members of the cohort. The cumulative exposure estimates were lagged 5 years (i.e., at any point in time after exposure began, an individual's cumulative exposure would equal the product of chromate concentration and duration of exposure, summed over all jobs held up to five years prior to that point in time). An exposure lag is commonly used in the dose-response analysis of lung cancer since there is a long latency period between first exposure and the development of disease. Gibb et al. found that models using five- and ten-year lags provided better fit to the mortality data than lags of zero, two and twenty years (Ex. 31–22–11). The cross-classification of cumulative exposure with age allowed Environ to evaluate models that considered the effect of age on lung cancer risk. A total of 71,994 person-years summed up from Table V of the Gibb et al. study was slightly greater than the reported 70,736 cited in their publication (Ex. 31–22–11, p. 119).

TABLE VII–1.—DOSE-RESPONSE DATA FROM GIBB *et al.* (EX. 31–22–11): OBSERVED AND EXPECTED NUMBER OF LUNG CANCER DEATHS GROUPED BY AGE AND FOUR CUMULATIVE CR(VI) EXPOSURE CATEGORIES

Cumulative Cr(VI) exposure (μg/m³ – years)		Age						
		20–29	30–39	40–49	50–59	60–69	70–79	80+
0–0.77	Observed	0	1	0	14	8	2	1
	Expected	0.018	0.39	2.5	7.56	10.79	5	0.88
	Person-Years	5003	7684	6509	5184	3104	865	163
	Mean Exposure	0.21	0.21	0.27	0.28	0.26	0.24	0.21
0.78–4.6	Observed	0	0	2	10	10	4	2
	Expected	0.001	0.18	1.97	6.09	7.85	3.25	0.44
	Person-Years	349	3139	4643	3928	2183	558	79
	Mean Exposure	2.2	2.2	2.2	2.2	2.2	2.0	1.9
4.7–40	Observed	0	0	3	10	11	4	2
	Expected	0.002	0.19	1.93	5.7	7.66	3.26	0.38
	Person-Years	457	3520	4732	3720	2128	559	78
	Mean Exposure	16	16	16	16	15	15	14
40–2730	Observed	0	0	8	8	18	3	1
	Expected	0.001	0.17	1.82	5.63	6.71	2.48	0.18
	Person-Years	200	2874	4294	3663	1926	423	29
	Mean Exposure	110	170	210	270	330	410	450

A 5-year lag was used in the calculation of the cumulative exposures. The exposure estimates themselves have been converted from those shown in Gibb *et al.*, Table V, by multiplying by 0.52, to convert from chromate concentration to hexavalent chromium concentration and by 1000 to convert from mg/m³ - years to μg/m³–years

A set of ''externally standardized'' models was applied to the data in Table VII–1. These are externally standardized because they required estimates of expected lung cancer deaths from a standard reference population. The 2002 Environ analysis relied on expected lung cancer deaths from age-specific Maryland rates, as provided in Gibb *et al.* The observed numbers of cancer cases were assumed to have a Poisson distribution, with expected values corresponding to three different dose-related models. A Poisson distribution is assumed because it has been commonly used in statistics to describe the allocation of rare events that occur during a given time period. Regression techniques are then used to link explanatory variables (*e.g.*, cumulative exposure) to responses of interest (*e.g.*, lung cancer deaths).

The set of models used was mathematically described as follows:

E1. $N_i = C_0 * E_i * \exp\{kt_i\} * (1 + C_1D_i + C_2D_i^2)$

E2. $N_i = C_0 * E_i * (1 + C_1D_i * \exp\{kt_i\})$

E3. $N_i = C_0 * E_i + (PY_i * C_1D_i)$

where N_i is the predicted number of lung cancers in i^{th} group PY_i is the

number of person-years for group i; E_i is the expected number of lung cancers in that group, based on the reference population; D_i is the mean cumulative dose for that group; and C_0, C_1, C_2, and k are parameters to be estimated. In equations E1 and E2, t_i the mean age for group i.

Models E1 and E2 are relative risk models that differ with respect to the effect of age. In model E1, the background rates are adjusted for age whereas in E2 the dose coefficient is modified by the age. On the other hand, Model E3 is an additive risk model. In the case of additive risk models, the exposure-related estimate of risk is the same regardless of the age- and race-specific background rate of lung cancer. For relative risk models, a dose term is multiplied by the appropriate background rate of lung cancer to derive an exposure-related estimate of risk, so that excess risk is always relative to background.

Estimation of parameters (i.e., C_0, C_1, C_2, and k) was accomplished by maximum likelihood techniques. For the externally standardized models, likelihood ratio tests were used to determine which of the model parameters contributed significantly to the fit of the model. Parameters were sequentially added to the model, starting with C_1, when they contributed significantly ($p \geq 0.05$) to improving the fit. Parameters that did not contribute significantly were excluded from consideration.

Goodness-of-fit for each model was evaluated by considering the deviance, a likelihood-based statistic for which larger p-values indicate better model fit. In addition, the fits of different models were compared using the Akaike Information Criterion (AIC) value, a statistic based on the model's maximized likelihood and the number of parameters used. For the quadratic model E1, addition of a dose-squared term did not significantly improve the fit of model to the data (i.e., C_2 estimated to be zero) relative to a linear model. For models E1 and E2, the parameter k was not determined to be different from 0, and thus models E1 and E2 defaulted to the same linear relative risk model. The deviance-based test of fit suggested an adequate correspondence between model predictions and the observations ($p \geq 0.13$).

A second set of ''internally standardized'' models, which did not require estimation of the expected number of lung cancers, was also fit to the data in Table VII–1 (Ex. 33–15). Model parameters were estimated by the maximum likelihood procedures described above. The test for goodness-of-fit indicated that these models did not fit the data well ($p \leq 0.01$). The formulation and a more detailed description of these models can be found in the 2002 Environ report (Ex. 33–15).

Lifetable calculations were made of the number of extra lung cancers per 1000 workers exposed to Cr(VI), assuming a constant exposure from age 20 through a maximum of age 65. The lifetime probability of a lung cancer death was cumulated to age 100, resulting in a negligible loss of accuracy since the probability that a person will live longer than that is extremely small. Rates of lung cancer and other mortality for the lifetable calculations were based, respectively, on 1998 U.S. lung cancer and all-cause mortality rates for both sexes and all races.

The lifetable calculation of additional lifetime risk was completed for the maximum likelihood parameter estimates for each model. In addition, 95% confidence intervals for the additional lifetime risk were derived by a likelihood profile method. Details about the procedures used to estimate parameters, model fit, lifetable calculations, and confidence intervals are described in the 2002 Environ report (Ex. 33–15, p. 24–26).

Based on comparison of the models' AIC values, Environ indicated that the linear relative risk model (simplified E1/E2) was preferred over the E3 additive risk model. The relative risk model is also preferred over an additive risk model (fits being adequate in both cases) in the case of lung cancer because of its variable background rate with age. It may not be appropriate to assume, as an additive model does, that increased lung cancer risk at age 25, where background risk is relatively low, would be the same (for the same cumulative dose) as at age 50, where background rates are much higher.

The linear relative risk model predicted an excess lifetime risk of lung cancer associated with an occupational exposure of 45 years to 1 $\mu g/m^3$ Cr(VI) to be 6 per 1000 (95% CI: 0.8 to 14). The additive model predicted a slightly lower lifetime risk of 4.4 per 1000 (95% CI: 0.0 to 11). At the OSHA PEL (52 $\mu g/m^3$), the maximum likelihood estimate (MLE) using the linear relative risk model is 253 per 1000 (95% CI: 39 to 456).

Since the completion of the 2002 Environ analysis, individual data for the 2,357 men in the Gibb *et al.* cohort have become available. The new data included cumulative Cr(VI) exposure estimates, smoking information, date of birth, race, date of hire, date of termination, cause of death, and date of the end of follow-up for each individual (Ex. 35–295). The individual data allowed Environ to do several additional analyses that could not be done previously, including assessments based on (1) redefined exposure categories, (2) alternate background reference rates for lung cancer mortality, and (3) Cox proportional hazards modeling (Ex. 33–12). These are discussed below.

In the 2002 analysis, Environ used the same four-group categorization of cumulative exposure reported by Gibb *et al.* and presented in Table VII–1. The individual data allowed Environ to investigate alternate groupings of cumulative exposure categories. Environ presented two alternate groupings with ten cumulative Cr(VI) exposure groups each, six more than reported by Gibb *et al.* and used in the 2002 analysis. One alternative grouping was designed to divide the person-years of follow-up and, therefore, the expected numbers of lung cancers fairly evenly across groups. The other alternative allocated roughly the same number of observed lung cancers to each group. These two alternatives were designed to remedy the uneven distribution of observed and expected cases in the Gibb *et al.* categories, which may have caused parameter estimation problems due to the small number of cases in some groups. The new groupings assigned adequate numbers of observed and expected lung cancer cases to all groups and are presented in Table VII–2.

TABLE VII–2.—DOSE-RESPONSE DATA FROM ENVIRON (2003, EX. 33–12): OBSERVED AND EXPECTED LUNG CANCER DEATHS FOR GIBB COHORT GROUPED BY TEN CUMULATIVE CR(VI) EXPOSURE CATEGORIES

	Cumulative Cr(VI) exposure μg/m³-years)	Mean Cr(VI) exposure (μg/m³-yr)	Person-years	Observed lung cancers	Expected lung cancers	
					Maryland rates	Baltimore rates
Alternative 1: Roughly Equal Observed Cases per Group						
	0–0.151	0.0246	17982	12	10.3	13.37
	0.151–0.686	0.395	9314	12	13.0	16.80
	0.686–2.08	1.25	8694	12	10.3	13.55
	2.08–4.00	2.96	5963	12	7.38	9.42
	4.00–8.32	5.89	5102	12	5.63	7.32
	8.32–18.2	12.4	5829	13	7.09	9.21
	18.2–52	31.1	6679	13	6.83	9.05
	52–182	105	6194	12	5.77	7.73
	182–572	314	4118	12	5.79	7.66
	>572	979	945	12	2.07	2.62
Alternative 2: Roughly Equal Number of Person-Years per Group						
	0–0.052	0.00052	14282	4	5.08	6.63
	0.052–0.273	0.147	6361	11	9.05	11.58
	0.273–0.65	0.455	6278	7	8.71	11.33
	0.65–1.43	0.996	6194	11	7.30	9.58
	1.43–3.12	2.19	6395	12	8.17	10.52
	3.12–6.89	4.59	6207	11	6.90	8.95
	6.89–16.1	10.7	6296	17	7.77	10.05
	16.1–41.6	25.9	6230	12	6.50	8.57
	41.6–1.43	81.5	6287	10	5.56	7.52
	>143	384	6289	27	9.17	11.99
Total			70819.38	122	74.2	96.7

The lower bounds of the ranges are inclusive; the upper bounds are exclusive.

The 2003 Environ analysis also derived expected cases using lung cancer rates from alternative reference populations. In addition to the State of Maryland lung cancer rates that were used by Gibb *et al.,* Environ used age- and race-specific rates from the city of Baltimore, where the plant was located. Baltimore may represent a more appropriate reference population because most of the cohort members resided in Baltimore and Baltimore residents may be more similar to the cohort members than the Maryland or U.S. populations in their co-exposures and lifestyle characteristics, especially smoking habits and urban-related risk factors. On the other hand, Baltimore may not be the appropriate reference population if the elevated lung cancer rates primarily reflect extensive exposure to industrial carcinogens. This could lead to an under representation of relative risk attributable to Cr(VI) exposure.

The 2003 analysis used two externally standardized models, a quadratic relative risk model (model E1 from above, without the age factor) and a quadratic additive risk model (model E3 from above with the additional term $C_2D_i^2$) defined as follows:

E4. $N_i = C_0 * E_i + PY_i * (C_1D_i + C_2D_i^2)$.

The age factor was dropped from model E1 because the individual data obviated the need to rely on the cross-classifications of cumulative exposure. The availability of individual data also allowed a more refined approach to internally standardized modeling than employed in the 2002 assessment. Two Cox proportional hazards models were fit to the individual exposure-response data that incorporated the individual ages at death of all the lung cancer cases. The model forms were:

C1. $h(t;z;D) = h_0(t)*\exp(\beta_1 z + \beta_2 D)$

C2. $h(t;z;D) = h_0(t)*[\exp(\beta_1 z)][1 + \beta_2 D]$

where h is the hazard function, which expresses the age-specific rate of lung cancer among workers, as estimated by the model. In addition, t is age, z is a vector of possible explanatory variables other than cumulative dose, D is cumulative dose, $h_0(t)$ is the baseline hazard function (a function of age only), β_2 is the cumulative dose coefficient, and β_1 is a vector of coefficients for other possible explanatory variables (Ex. 35–57). Cox modeling is an approach that uses the experience of the cohort to estimate an exposure-related effect, irrespective of an external reference population or exposure categorization. Cox models can sometimes eliminate concerns about choosing an appropriate reference population and may be advantageous when the characteristics of the cohort under study are not well matched against reference populations for which age-related background rates have been tabulated. The two forms of the Cox models are consistent with those originally discussed by Cox. Model C1 assumes the lung cancer response is nonlinear with cumulative Cr(VI) exposure, whereas C2 assumes a linear lung cancer response with Cr(VI) exposure.

All externally standardized models provided a good fit to the data ($p \geq 0.40$). The choice of exposure grouping had little effect on the parameter estimates of either model E1 or E4. However, the choice of reference rates had some effect, notably on the ''background'' parameter, C_0, which was included in the models to adjust for differences in background lung cancer rates between cohort members and the reference population. Such an adjustment was necessary for the Maryland reference population (C_0 was significantly different from its default value, 1), but not for the Baltimore city reference population (C_0 was not significantly different from 1). The inclusion of the C_0 parameter allowed the model to fit the data and yielded a cumulative dose coefficient that reflected the effect of exposure and not the effect of differences in background rates. The model results indicated a relatively consistent cumulative dose coefficient, regardless of reference population. Details about the procedures used to estimate parameters, model fit, lifetable calculations, and confidence intervals

are described in the Environ report (Ex. 33–12, p. 8–9).

The coefficient for cumulative dose in the model ranged from 2.87 to 3.48 per mg/m^3-yr for the relative risk model, E1, and from 0.0061 to 0.0071 per mg/m^3-person-yr for the additive risk model, E4. These coefficients determine the slope of the linear cumulative Cr(VI) exposure-lung cancer response relationship. The cumulative dose coefficients for the relative risk model (E1) were only slightly greater than that obtained from model E1 in the 2002 Environ analysis. For the additive risk model (E4), the dose coefficients were approximately twice the value obtained from model E3 in the 2002 analysis (*i.e.,* 0.0033). In no case did the new analysis suggest that a quadratic model fit the data better than a linear model.

For the internally standardized Cox proportional hazards models, C1 and C2, the other possible explanatory variables considered were cigarette smoking status, race, and calendar year of death. For both models, addition of a term for smoking status significantly improved the fit of the models to the data ($p \leq 0.00001$). The experience with non-linear model C1 indicated that race ($p=0.15$) and year of death ($p=0.4$) were not significant contributors when cumulative dose and smoking status were included in the model. Based on results for model C1, race and year of death were not considered by Environ in the linear model C2. The cumulative dose coefficient, $\beta 2$, was 1.00 for model C1 and 2.68 for model C2. Model C2 provided a slightly better fit to the data than did model C1. A more complete description of the models and variables can be found in the 2003 Environ analysis (Ex. 33–12, p. 10).

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Table VII-3

Environ (2003, Ex. 33-12): Model Predictions of Excess Lung Cancer Deaths per 1000 Workers[a] Exposed to Various Cr(VI) Concentrations, by Model, Reference Population, and Exposure Grouping

Model	Reference Population	Exposure Grouping	Cr(VI) Concentration ($\mu g/m^3$) [95% Confidence Interval]						
			0.25	0.5	1.0	5	10	20	52
Relative Risk Model (E1)	Maryland State	Equal Cases/group	1.9 [0.9-3.6]	3.8 [1.8-7.2]	7.5 [3.7-14]	37 [18-69]	72 [36-132]	137 [57-240]	305 [168-471]
		Equal PYRs/group	2.0 [0.6-4.0]	4.0 [1.3-8.0]	8.0 [2.5-16]	39 [12-77]	76 [25-145]	144 [49-258]	318 [120-500]
	Baltimore City	Equal Cases/group	2.1 [1.0-3.6]	4.3 [1.3-8.0]	8.5 [4.1-14]	42 [20-68]	81 [40-130]	153 [78-237]	334 [186-467]
		Equal PYRs/group	2.3 [1.0-3.9]	4.6 [2.0-7.8]	9.1 [4.0-16]	45 [20-75]	86 [39-142]	163 [76-256]	351 [181-493]
Additive Risk Model (E4)	Maryland State	Equal Cases/group	2.4 [1.0-4.0]	4.7 [2.1-8.0]	9.4 [4.1-16]	46 [20-75]	89 [40-146]	170 [79-268]	373 [189-532]
		Equal PYRs/group	2.1 [0.7-3.7]	4.2 [1.5-7.5]	8.4 [3.0-15]	41 [15-72]	80 [29-137]	152 [58-253]	342 [141-511]
	Baltimore City	Equal Cases/group	2.4 [1.2-4.0]	4.8 [2.4-7.9]	10 [4.7-16]	47 [23-76]	92 [46-145]	174 [91-264]	380 [214-530]
		Equal PYRs/group	2.2 [1.0-3.8]	4.4 [2.0-7.5]	8.8 [3.9-15]	43 [19-72]	84 [38-138]	161 [74-254]	356 [181-513]
Cox Model C1	N/A	N/A	0.66 [0.3-0.9]	1.3 [0.6-1.9]	2.7 [1.3-3.8]	15 [6.7-21]	32 [14-49]	N/A	363 [110-606]
Cox Model C2	N/A	N/A	1.8 [0.7-3.4]	3.5 [1.4-6.8]	7.1 [2.7-14]	35 [13-66]	68 [27-125]	129 [52-229]	290 [128-456]

[a] The workers are assumed to start work at age 20 and continue to work for 45 years, at a constant exposure level.

Table VII–3 shows each model's predictions of excess lifetime lung cancer risk from various occupational exposures. The estimates are very consistent regardless of model, exposure grouping, or reference population. The model that appears to generate results least similar to the others is C1, which yielded one of the higher risk estimates at 52 μg/m^3, but estimated the lowest risks for exposure levels of 10 μg/m^3 or lower. The change in magnitude, relative to the other models, is a result of the nonlinearity of this model (the only nonlinear model among the set being considered). Confidence limits for all models, including C1, tend to overlap, suggesting a fair degree of consistency.

The estimates based on the individual data files were slightly greater than

those reported in the previous Environ analysis (Ex. 33–15). For example, the 2003 Environ analysis estimated additional lifetime risk from 45 years of exposure at the OSHA PEL to be between 290 and 380 per 1000, whereas the previous analysis estimated 253 per 1000 (Ex. 33–12, Table 9). This difference may be partly attributed to the availability of individual data, as opposed to data from summary tables, allowing a better definition of exposure categories. Some of the difference may be attributable to slightly different total person-years of follow-up reported by Gibb *et al.* in their summary table (71,994 from Table V, Ex. 31–22–11) and the total person-years accounted for in the individual data files (70,819 from Ex. 295). The reason for this variation in total person-years is unknown.

2. National Institute for Occupational Safety and Health (NIOSH) Risk Assessment

NIOSH (Ex. 33–13) developed a risk assessment from the Gibb cohort. The NIOSH analysis, like the 2003 Environ assessment, used the cohort individual data files to compute cumulative Cr(VI) exposure. However, NIOSH also explored some other exposure-related assumptions. For example, they performed the dose-response analysis with lag times in addition to the 5-year lag used by Environ. NIOSH also analyzed dose-response using as many as 50 exposure categories, although their report presents data in five cumulative Cr(VI) exposure groupings.

NIOSH incorporated information on the cohort smoking behavior in their quantitative assessments. They estimated (packs/day)-years of cumulative smoking for each individual in the cohort, using information from a questionnaire that was administered at the time of each cohort member's date of hire. To estimate cumulative smoking, NIOSH assumed that the cohort members maintained the level of smoking reported in the questionnaire from the age of 18 through the end of follow-up. Individuals with unknown smoking status were assigned a value equal to the average smoking level among all individuals with known smoking levels (presumably including non-smokers). Individuals who were known to smoke but for whom the amount was unknown were assigned a smoking level equal to the average of all smokers.

NIOSH considered six different relative risk models, fit to the data by Poisson regression methods. They did not consider additive risk models. The six relative risk models were externally standardized using age- and race-specific U.S. lung cancer rates. Their background coefficients, C_0, explicitly included smoking, race, and age terms to adjust for differences between the cohort and the reference population. These models are described as follows:

NIOSH1a: $N_i = C_0 * Ei * \exp(C_1 D_i)$
NIOSH1b: $N_i = C_0 * Ei * \exp(C_1 D_i^{1/2})$
NIOSH1c: $N_i = C_0 * Ei * \exp(1 + C_1 D_i + C_2 D_i^2)$
NIOSH1d: $N_i = C_0 * Ei * (1 + D_i)\alpha$
NIOSH1e: $N_i = C_0 * Ei * (1 + C_1 D_i)$
NIOSH1f: $N_i = C_0 * Ei * (1 + C_1 D_i \alpha)$

where the form of the equation has been modified to match the format used in the Environ reports. In addition NIOSH fit Cox proportional hazard models (not specified) to the lung cancer mortality data using the individual cumulative Cr(VI) exposure estimates.

NIOSH reported that the linear relative risk model 1e generally provided a superior fit to the exposure-response data when compared to the various log linear models, 1a-d. Allowing some non-linearity (*e.g.*, model 1f) did not significantly improve the goodness-of-fit, therefore, they considered the linear relative risk model form 1e (analogous to the Environ model E1) to be the most appropriate for determining their lifetime risk calculations. A similar fit could be achieved with a log-linear power model (model 1d) using log-transformed cumulative Cr(VI) and a piece-wise linear specification for the cumulative smoking term.

The dose coefficient (C_1) for the linear relative risk model 1e was estimated by NIOSH to be 1.444 per mg CrO^3/m^3-yr. (Ex. 33–13, Table 4). If the exposures were converted to units of mg Cr(VI)/m^3-yr, the estimated cumulative dose coefficient would be 2.78 (95% CI: 1.04 to 5.44) per mg/m^3-yr. This value is very close to the estimates derived in the Environ 2003 analysis (maximum likelihood estimates ranging from 2.87 to 3.48 for model E1, depending on the exposure grouping and the reference population). Lifetime risk estimates based on the NIOSH-estimated dose coefficient and the Environ lifetable method using 2000 U.S. rates for lung cancer and all cause mortality are shown in Table VII–4. The values are very similar to the estimates predicted by the Environ 2003 analysis (Table VII–3). The small difference may be due to the NIOSH adjustment for smoking in the background coefficient. NIOSH found that excess lifetime risks for a 45-year occupational exposure to Cr(VI) predicted by the best-fitting power model gave very similar risks to the preferred linear relative risk model at TWA Cr(VI) concentrations between 0.52 and 52 μg/m^3 (Ex. 33–13, Table 5). Although NIOSH did not report the results, they stated that Cox modeling produced risk estimates similar to the Poisson regression. The consistency between Cox and Poisson regression modeling is discussed further in section VII.C.4.

Table VII-4

Model Predictions of Additional Lung Cancer Deaths per 1000 Workers[a] Exposed to Various Cr(VI) Concentrations Based on NIOSH-Estimated Parameters

Cr(VI) Concentration (μg/m^3) [95% Confidence Interval]							
0.25	**0.5**	**1.0**	**2.5**	**5**	**10**	**20**	**52**
1.8 [0.7-3.6]	3.7 [1.4-7.2]	7.3 [2.7-14]	18 [7-35]	36 [14-69]	70 [27-131]	133 [53-238]	297 [130-468]

[a] The workers are assumed to start work at age 20 and continue to work for 45 years, at a constant exposure level.

NIOSH reported a significantly higher dose-response coefficient for nonwhite workers than for white workers. That is, nonwhite workers in the Gibb cohort are estimated to have a higher excess risk of lung cancer than white workers, given equal cumulative exposure to Cr(VI). In contrast, no significant race difference was found in the Cox proportional hazards analysis reported by 2003 Environ.

3. Exponent Risk Assessment

In response to OSHA's Request For Information, Exponent (Ex. 31–18–15–1) prepared an analysis of lung cancer mortality from the Gibb cohort. Like 2003 Environ and NIOSH, the Exponent analysis relied on the individual worker data. Exponent performed their dose-response analyses based on three different sets of exposure categories using two reference populations and 70,808 person-years of follow-up. A total of four analyses were completed, using (1) Maryland reference rates and the four Gibb *et al.* exposure categories; (2) Baltimore reference rates and the four Gibb *et al.* exposure categories; (3) Baltimore reference rates and six exposure groups defined by Exponent; and (4) Baltimore City reference rates and five exposure categories, obtained by removing the highest of the six groups defined by Exponent from the dose-response analysis. A linear relative risk model without a background correction term, C_0, (as was used by Environ and NIOSH) was applied in all of these cases and cumulative exposures were lagged five years (as done by Environ and NIOSH). The analyses showed excess lifetime risk between 6 and 14 per 1000 for workers exposed to 1 μg/m^3 Cr(VI) for 45 years.

The analysis using Maryland reference lung cancer rates and the Gibb *et al.* four-category exposure grouping yielded an excess lifetime risk of 14 per 1000. This risk, which is higher than the excess lifetime risk estimates by Environ and NIOSH for the same occupational exposure, probably results from the absence of a background rate coefficient in Exponent's model. As reported in the Environ 2002 and 2003 analyses, the Maryland reference lung cancer rates require a background rate coefficient greater than 1 to achieve the best fit to the exposure-response data. The unadjusted Maryland rates underestimate the cohort's background lung cancer rate, leading to overestimation of the risk attributable to cumulative Cr(VI) exposure.

The two analyses that used Baltimore reference rates and either Exponent's six-category exposure grouping or the Gibb *et al.* four-category grouping both resulted in an excess lifetime risk of 9 per 1000 for workers exposed to 1 μg/m^3 Cr(VI) for 45 years. This risk is close to estimates reported by Environ using their relative risk model (E1) and Baltimore reference rates for the same occupational exposure (Table VII–3). The Environ analysis showed that, unlike the Maryland-standardized model discussed above, the Baltimore-standardized models had background rate coefficients very close to 1, the ''default'' value assumed by the Exponent relative risk model. This suggests that the Baltimore reference rates may more accurately represent the background lung cancer rate for this cohort.

The lowest excess lifetime risk for workers exposed to 1 μg/m^3 Cr(VI) for 45 years reported by Exponent, at 6 per 1000, was derived from the analysis that excluded the highest of Exponent's six exposure groups. While this risk value is close to the Environ and NIOSH unit risk estimates, the analysis merits some concern. Exponent eliminated the highest exposure group on the basis that most cumulative exposures in this group were higher than exposures usually found in current workplace conditions. However, eliminating this group could exclude possible long-term exposures (*e.g.*, >15 years) below the current OSHA PEL (52 μg/m^3) from the risk analysis. Moreover, no matter what current exposures might be, data on higher cumulative exposures are still relevant for understanding the dose-response relationships.

In addition, the Exponent six category cumulative exposure grouping may have led to an underestimate of the dose effect. The definition of Exponent's six exposure groups was not related to the distribution of cumulative exposure associated with individual person-years, but rather to the distribution of cumulative exposure among the workers at the end of their employment. This division does not result in either a uniform distribution of person-years or observed lung cancer cases among exposure categories. In fact, the six category exposure groupings of both person-years and observed lung cancers were very uneven, with a preponderance of both allocated to the lowest exposure group. This skewed distribution of person-years and observed cases puts most of the power for detecting significant differences from background cancer rates at low exposure levels, where these differences are expected to be small, and reduces the power to detect any significant differences from background at higher exposure concentrations.

Exponent conducted analyses to further explore the dose-response relationship in addition to the assessments described above (Ex. 31–18–1). Of particular interest was an examination of short-term workers' likely impact on the dose-response assessment and an SMR analysis based on peak exposure estimates. A substantial proportion of the Gibb cohort worked less than one year at the Baltimore plant. Inclusion of these workers in the exposure-response assessment could potentially bias the results, if, for example, these workers incurred unrecorded Cr(VI) exposures at other jobs. In brief, Exponent found that excluding these short-term workers would not likely impact the dose-response analysis.

Exponent reported that SMRs for workers with ''peak'' exposures less than 0.18 mg CrO_3/m^3 (0.094 mg Cr(VI)/m^3) were not significantly elevated and that this exposure level may represent a ''threshold'' (i.e., exposure below which the probability of cancer is zero), such that workers exposed to concentrations below the threshold may not have excess cancer risk (Ex. 31–18–1). However, the analysis used peak exposure estimates based on recorded average annual exposures. True peak exposures were unavailable for the Gibb cohort members. The use of the highest recorded average annual Cr(VI) air level as an exposure metric ignores any risk contribution from the duration of exposure. It assumes the same lung cancer risk regardless of whether the worker is exposed at a particular Cr(VI) concentration for one month or ten years. This is clearly inconsistent with the study results.

The validity of the ''peak exposure'' analysis also suffers from Exponent's problematic definition of exposure categories, which is similar to the six-part grouping used in the dose-response assessments. As with Exponent's cumulative exposure groups, the peak exposure grouping allocates most of the observed cancers and person-years to the lowest exposure groups, reducing the power to detect significant differences from background at more moderate exposure concentrations below 0.094 mg Cr(VI)/m^3. The implication that the data indicate a ''threshold'' at 0.094 mg Cr(VI)/m^3 is, therefore, misleading, and not considered a valid analysis for estimating risk of lung cancer to workers exposed to Cr(VI).

4. Summary of Risk Assessments Based on the Gibb Cohort

OSHA finds remarkable consistency among the risk estimates from the

various quantitative analyses of the Gibb cohort. The excess lifetime risks from cumulative Cr(VI) exposure were similar whether the analyses were based on the summary information reported by Gibb *et al.* or on the information provided in the individual data file.

Both Environ and NIOSH determined that linear relative risk models with respect to cumulative exposure generally provided a superior fit to the data when compared to other relative risk models. The Environ 2003 analysis further suggested that a linear additive risk model could adequately describe the observed dose-response data. The risk estimates for NIOSH and Environ's best-fitting models were statistically consistent (compare Tables VI–3 and VI–4).

The choice of reference population had little impact on the risk estimates. NIOSH used the entire U.S. population as the reference, but included adjustment terms for smoking, age and race in its models. The Environ 2003 analysis used both Maryland and Baltimore lung cancer rates, and included a generic background adjustment term. The adjustment was significant in the fitted model when Maryland rates were used for external standardization, but not when Baltimore rates were used. Since no adjustment in the model background term was required to better fit the exposure-response data using Baltimore City lung cancer rates, they may best represent the cohort's true background lung cancer incidence. OSHA considers the inclusion of such adjustment factors, whether specific to smoking, race, and age (as defined by NIOSH), or generic (as defined by Environ), to be appropriate and contribute to accurate risk estimation by helping to correct for confounding risk factors. The internally standardized Cox models, especially the linear Cox model, which also adjusted for smoking yielded risk estimates that were generally consistent with the externally standardized models.

Finally, the number of exposure categories used in the analysis had little impact on the risk estimates. When an appropriate adjustment to the background rates was included, the four exposure groups originally defined by Gibb *et al.* and analyzed in the 2002 Environ report, the six exposure groups defined by Exponent, the two alternate sets of ten exposure categories as defined in the 2003 Environ analysis, and the fifty groups defined and aggregated by NIOSH all gave essentially the same risk estimates. The robustness of the results to various categorizations of cumulative exposure adds to the validity of the risk projections.

Having reviewed the analyses described in this section, OSHA finds that the best estimates of excess lung cancer risk to workers exposed to the current PEL (52 μg Cr(VI)/m^3) for a working lifetime are about 300 to 400 per thousand based on data from the Gibb cohort. The best estimates of excess lung cancer risks to workers exposed to TWA exposure concentrations of 1 μg Cr(VI)/m^3 for a working lifetime range from 7.1 to 9.4 per 1000 with the lowest 95% confidence bound being 2.7, and the highest 95% confidence bound being 16 (Table VII–3). These estimates are consistent with predictions from Environ, NIOSH and Exponent models that applied linear relative and additive risk models based on the full range of cumulative Cr(VI) exposures experienced by the Gibb cohort and used appropriate adjustment terms for the background lung cancer mortality rates.

It is instructive to examine whether the excess lung cancer risk estimated from the mathematical modeling reasonably predicts the risk based on the mortality observed in the Gibb *et al.* study. There were 855 deaths in the Gibb cohort of which 122 were from cancer of the lung (Ex. 31–22–11, Table I). The expected number of lung cancer deaths from the age-, gender-, race-, and calendar year-adjusted reference population in Baltimore was 96.7 (Table VII–2). Therefore, there were about 25 lung cancer deaths (i.e., 122—96.7) presumably attributable to Cr(VI) exposure out of the 855 total deaths, or 29 per 1000 workers (i.e., 25/855 × 1000). If lung cancer were to continue to occur with the same proportionate mortality in this cohort (64 percent of the cohort were still living), their excess lifetime lung cancer risk would be close to three percent.

The mean cumulative exposure for the Gibb cohort was 0.134 mg CrO_3/m^3 - yr with a mean 3.1 years of work (Ex. 31–22–11, Table II). An approximate average Cr(VI) air level of 22.5 μg Cr(VI)/m^3 can be calculated after converting from CrO_3 to Cr(VI). Using the average Cr(VI) air concentration (22.5 μg/m^3), mean exposure duration (3.1 yr), and mean age of hire of 30 years of age (Ex. 31–22–11, Table III), the linear relative risk model E1 (equal PYRs per group, Table VII–3) predicts an excess lifetime lung cancer risk of 14.8 per 1000 (95% CI: 6.97 to 25.1 per 1000) for workers with the mean cumulative exposure of the Gibb cohort. These Cr(VI) levels are below the current PEL for considerably shorter than a full working lifetime.

The model-predicted lung cancer risk is about half the risk calculated from the observed mortality in the Gibb *et al.* study. This is probably due, in part, to the higher cumulative Cr(VI) exposure for the subset of workers who had already died. The mean Cr(VI) exposure of the lung cancer cases was slightly over two-fold higher (i.e., 0.294 mg CrO_3/m^3 - yr) than the cohort as a whole (Ex. 31–22–11, Table II). It also seems likely that the workers who already died of causes other than lung cancer would be older cohort members that may have experienced higher Cr(VI) exposure than the presumably younger cohort members hired more recently and still living. If their mean cumulative Cr(VI) exposure were more like that of the lung cancer cases than the total cohort group, the relative risk model would predict risks close to the three percent excess lung cancer risk derived from the observed mortality data.

D. Quantitative Risk Assessments Based on the Luippold Cohort

As discussed earlier, Luippold *et al.* (Exs. 35–204; 33–10) provided information about the cohort of workers employed in a chromate production plant in Painesville, Ohio. Follow-up for the 482 members of the Luippold cohort started in 1940 and lasted through 1997, with accumulation of person-years for any individual starting one year after the beginning of his first exposure. There were 14,048 total person-years of follow-up for the cohort. The person-years were then divided into five exposure groups that had approximately equal numbers of expected lung cancers in each group. Ohio reference rates were used to compute expected numbers of deaths. White male rates were used because the number of women was small (4 out of 482) and race was known to be white for 241 of 257 members of the cohort who died and for whom death certificates were available. The 1960–64 Ohio rates (the earliest available) were assumed to hold for the time period from 1940 to 1960. Rates from 1990–94 were assumed to hold for the period after 1994. For years between 1960 and 1990, rates from the corresponding five-year summary were used. There were significant dose-related trends for lung cancer SMR as a function of year of hire, duration of employment, and cumulative Cr(VI) exposure. Overall, there was significantly increased SMR for lung cancer deaths of 241 (95% CI: 180 to 317).

TABLE VII.–5—DOSE-RESPONSE DATA FROM LUIPPOLD COHORT AS CITED BY ENVIRON (2002, EX. 33–15): OBSERVED AND EXPECTED NUMBERS OF LUNG CANCER DEATHS GROUPED BY FIVE CUMULATIVE CR(VI) EXPOSURE CATEGORIES

Cumulative Cr(VI) exposure (mg/m^3 – yrs)[a]	Mean Cr(VI) exposure (mg/m^3 – yrs)[a]	Observed lung cancers	Expected lung cancers[b]	Person-years
< 0.20	0.10	3	4.5	2952
0.20 – 0.49	0.36	8	4.4	2369
0.49 – 1.05	0.74	4	4.4	3077
1.05 – 2.70	1.79	16	4.4	3220
2.70 – 27.8	4.81	20	4.3	2482

[a] Note that units mg/m^3 – yrs is 1000 times greater than $\mu g/m^3$ – yrs in data tables for Gibb cohort.
[b] Expected lung cancer deaths derived using Ohio state mortality rates.

Environ conducted a risk assessment based on the cumulative Cr(VI) exposure-lung cancer mortality data from Luippold *et al.* and presented in Table VII–5 (Ex. 33–15). Cumulative Cr(VI) exposures were categorized into five groups with about four expected lung cancer deaths in each group. In the absence of information to the contrary, Environ assumed Luippold *et al.* did not employ any lag time in determining the cumulative exposures. The calculated and expected numbers of lung cancers were derived from Ohio reference rates. Environ applied the relative and additive risk models, E1 and E3, to the data in Table VII–5. Model E1 was applied without the $\exp\{kt_i\}$ term, because no categorization by age was available. Addition of a quadratic term did not improve the fit over that of a linear relative risk model. Model E2 was not applied, because without the $\exp\{kt_i\}$ term model E2 is the same as E1. The background rate parameter, C_0, was assumed to be 1.0 in both models since other values did not significantly improve model fit.

Linear relative and additive risk models fit the Luippold cohort data adequately ($p \geq 0.25$). The maximum likelihood estimates for the Cr(VI) exposure-related parameter, C_1, of the linear relative and additive risk models were 0.88 per mg/m^3 – yr and 0.0014 per mg/m^3 – person-yr, respectively. The C_1 estimates based on the Luippold cohort data were about 2.5-fold lower than the parameter estimates based on the Gibb cohort data. The excess lifetime risk estimate calculated by Environ for a 45-year working-lifetime exposure to 1 μg Cr(VI)/m^3 for both models was 2.2 per 1000 workers (95% confidence intervals from 1.3 to 3.5 per 1000 for the relative risk model and 1.2 to 3.4 per 1000 for the additive risk model) using a lifetable analysis with 1998 U.S. mortality reference rates. These risks were 2.5 to 3-fold lower than the projected risks based on the Gibb data set for equivalent cumulative Cr(VI) exposures.

Crump *et al.* (Exs. 33–15; 35–58; 31–18) also performed an exposure-response analysis from the Painesville data. In a Poisson regression analysis, cumulative exposures were grouped into ten exposure categories with approximately two expected lung cancer deaths in each group. The observed and expected lung cancer deaths by Cr(VI) exposure category are shown in Table VII–6. Ohio reference rates were again used in calculating the expected lung cancer deaths and cumulative exposures were lagged 5 years.

TABLE VII–6.—DOSE-RESPONSE DATA FROM CRUMP *et al.* (EX. 35–58): OBSERVED AND EXPECTED NUMBERS OF LUNG CANCER DEATHS FOR LUIPPOLD COHORT GROUPED BY TEN CUMULATIVE CR(VI) EXPOSURE CATEGORIES

Cumulative Cr(VI) exposure (mg/m^3-yrs)[a]	Mean Cr(VI) exposure (mg/m^3-yrs)[a]	Observed lung cancers	Expected lung cancer[b]	Person-years
0–0.06	0.0098	0	2.09	3112
0.06–0.18	0.11	3	2.19	1546
0.18–0.30	0.23	3	2.21	1031
0.30–0.46	0.38	5	2.13	1130
0.46–0.67	0.56	0	2.22	1257
0.67–1.00	0.80	4	2.23	1431
1.00–1.63	1.25	12	2.23	1493
1.63–2.60	2.10	3	2.18	1291
2.60–4.45	3.27	10	2.18	1248
4.45–29.0	7.55	11	2.12	904

The lower bounds of the ranges are inclusive; the upper bounds are exclusive.
[a] Note that units mg/m^3-yrs is 1000 times greater than $\mu g/m^3$-yrs in data tables for Gibb cohort.
[b] Expected lung cancer deaths derived using Ohio state mortality rates.

The Crump *et al.* analysis used the same linear relative risk and additive risk models as Environ on the individual data categorized into the ten cumulative exposure groups (Ex. 35–58). Tests for systematic departure from linearity were non-significant for both models ($p \geq 0.11$). The cumulative dose coefficient determined by the maximum likelihood method was 0.79 (95% CI: 0.47 to 1.19) per mg/m^3-yr for the relative risk model and 0.0016 (95% CI: 0.00098 to 0.0024) per mg/m^3—person-yr for the relative and additive risk model, respectively. The authors noted that application of the linear models to five and seven exposure groups resulted in no significant difference in dose coefficients, although the data was not presented. The dose coefficients reported by Crump *et al.* were very

similar to those obtained by Environ above, even though different exposure groups were used and the lag for the cumulative exposure calculation was slightly different. The authors noted that the linear models did not fit the exposure data grouped into ten categories very well (goodness-of-fit $p \leq 0.01$) but fit the data much better with seven exposure groups ($p > 0.3$) after eliminating the nonmonotonic (i.e., not progressively increasing with exposure) scatter contributed by the many lower exposure categories where there are few observed and expected cancers. This nonmonotonic pattern is avoided by using more stable exposure groupings with greater number of cancers. The reduction in number of exposure groups did not significantly change the dose coefficient estimates.

The maximum likelihood estimate for the cumulative dose coefficient using the linear Cox regression model (i.e., model C2) was 0.66 (90% CI: 0.11 to 1.21), which was similar to the linear [Poisson regression] relative risk model. When the Cox analysis was restricted to the 197 workers with known smoking status and a smoking variable in the model, the dose coefficient for Cr(VI) was nearly identical to the estimate without controlling for smoking. This led the authors to conclude that ''the available smoking data did not suggest that exposure to Cr(VI) was confounded with smoking in this cohort, or that failure to control for smoking had an appreciable effect upon the estimated carcinogenic potency of Cr(VI)'' (Ex. 35–58, p.1156).

Crump *et al.* also presented benchmark dose estimates (EC_{10}s) of 52 $\mu g/m^3$ (95 percent lower confidence bound, LEC_{10}, of 37 $\mu g/m^3$) and 49 $\mu g/m^3$ (LEC_{10} of 35 $\mu g/m^3$) for the relative risk and additive risk models, respectively. The EC_{10} is an estimate of the dose associated with a ten percent, or 100 in 1000, risk. The EC_{10} and its LEC_{10} are being considered by the U.S. EPA, under certain circumstances, as a reasonable point of departure for extrapolation modeling below the biologically observable range (Ex. 35–53, p. 3–12 to 3–15). These results are very consistent with those predicted by Environ (Ex. 33–15) for the Luippold *et al.* cohort (*e.g.*, approximately 100 lung cancer cases per 1000 workers from estimated working lifetime at the OSHA PEL of 52 $\mu g/m^3$). There were only minor non-significant changes in benchmark dose estimates when exposure lags were varied from 5 to 20 years using Poisson or Cox linear regression models.

Given the similarity in results, OSHA believes it is reasonable to use the dose coefficients reported by Exponent based on their groupings of the individual cumulative exposure data to estimate excess lifetime risk from the Luippold cohort. Table VII–7 presents the excess risk for a working lifetime exposure to various TWA Cr(VI) levels as predicted by the relative and additive risk models using a lifetable analysis with 2000 U.S. rates for all causes and lung cancer mortality. The maximum likelihood estimates and 95 percent confidence limits from the Luippold cohort indicate that working lifetime exposures to the current Cr(VI) PEL would entail excess lifetime lung cancer risks around 100 per 1000 and that risks of 1.2 to 3.3 per 1000 would be expected from TWA exposures of 1 μg Cr(VI)/m^3 for a working lifetime.

Table VII-7

Model Predictions of Additional Lung Cancer Deaths per 1000 Workers[a] Exposed to Various Concentrations of Cr(VI) Based on the Luippold Cohort and Crump Dose Coefficients

Model	Cr(VI) Concentration ($\mu g/m^3$) [95% Confidence Interval]							
	0.25	0.5	1.0	2.5	5	10	20	52
Relative Risk	0.52 [0.31 –0.79]	1.0 [0.62–1.6]	2.1 [1.2–3.1]	5.2 [3.1 – 7.8]	10 [6.2–15]	21 [12– 31]	41 [21-60]	101 [62 -147]
Additive Risk	0.55 [0.36– 0.82]	1.1 [0.67–1.6]	2.2 [1.3–3.3]	5.5 [3.4 – 8.2]	11 [6.7–16]	22 [13– 32]	43 [27-64]	108 [67 – 155]

[a] The workers are assumed to start work at age 20 and continue to work for 45 years, at a constant exposure level.
Maximum likelihood estimates and 95% confidence intervals are shown.
These estimates were derived from the Environ risk models using the dose coefficients reported in preamble section VII.D. and a lifetable representing 2000 U.S. mortality rates for all causes and lung cancer.

The excess lung cancer risk predicted from the mathematical modeling can be compared with the risk expected based on the actual mortality experience of the Luippold cohort. There were 303 observed deaths in the cohort of which 51 were from cancer of the lung (Ex. 33–10, Table 2). The expected number of lung cancer deaths from the age-, gender-, race-, and calendar year-adjusted reference population from Ohio was 21.2. Therefore, there were about 30 lung cancer deaths (51–21.2) presumably attributable to Cr(VI) exposure out of 303 total deaths, or 98 per 1000 workers ($29.8/303 \times 1000$). If lung cancer were to continue to occur with the same proportionate mortality in this cohort (37 percent of the cohort was still living), their excess lifetime lung cancer risk would be about ten percent.

The mean cumulative exposure for the Luippold cohort was 1.58 mg Cr(VI)/

m^3-yr (Ex. 33–10, Table 1), which is about twenty-three times the mean exposure for the Gibb cohort (i.e., 0.0697 mg Cr(VI)/m^3-yr). Although the mean length of employment of the Luippold cohort was not reported, a crude distribution of the years employed is consistent with an average of about ten years (Ex. 33–10, Table 1). If the cohort were exposed an average ten years then their average Cr(VI) air level would be roughly 158 μg Cr(VI)/m^3 (1.58×10 yr ÷ 1000 μg/mg). Using this Cr(VI) air concentration (158 μg/m^3), the estimated mean exposure duration (10 yr), and the mean age of hire of 34 years of age (Ex. 33–10, Table 1), the linear relative risk model E1 predicts an excess lifetime lung cancer risk of 74 per 1000 (95% CI: 46 to 110 per 1000). This is slightly lower than the 98 per 1000 excess lung cancer deaths attributable to Cr(VI) determined from the observed study data. The Luippold cohort workers were exposed to mean Cr(VI) levels about three-fold higher than the current PEL for an average duration that was slightly less than a quarter of a full 45 year working lifetime.

As previously explained, it is not surprising that the relative risk model may underpredict the excess risks calculated from study mortality data. The risk model predicts the probability of lung cancer risk in an individual or set of workers, all with the same cumulative Cr(VI) exposure. The excess lung cancer risk calculated from the observed mortality data were for a group of workers with a wide range of Cr(VI) exposures. Like the Gibb study, the lung cancer cases had a mean cumulative Cr(VI) that was twice that of the entire cohort. Therefore, their risk may be somewhat higher than predicted for the cohort as a whole. Since most of the Luippold cohort had died (i.e., 63 percent), the model-derived lung cancer risk based on the mean exposure of the entire Luippold cohort may better predict the mortality-derived excess risk estimate than was the case for the Gibb cohort, which had a lower percentage of deaths (i.e., 36 percent).

Crump *et al.* reported on tests of trend and of excess lung cancer mortality by highest reported monthly TWA Cr(VI) concentration and cumulative Cr(VI) exposure for the workers in the Luippold cohort. The former analysis examined air concentration irrespective of exposure duration, even though there was a significant positive trend for excess lung cancer mortality with duration of employment (Ex. 33–10, Table 3). They found that a statistically significant excess mortality was not observed in workers exposed to less than the current OSHA PEL (i.e., 52 μg/m^3). An analysis of cumulative Cr(VI) exposure found that a statistically significant exposure-related trend in lung cancer mortality only occurred if cumulative Cr(VI) exposure estimates above 1.0 mg/m^3-yr were included. Crump *et al.* acknowledged that their analysis had limited statistical power (i.e., the magnitude of excess mortality needed to achieve statistical significance) to detect increases in excess mortality at the lower cumulative Cr(VI) exposures (Ex. 35–58, p. 1147).

The lack of statistical significance for the subset of 103 workers in the Luippold cohort whose highest monthly TWA exposure was less than the OSHA PEL is readily explained by a further examination of the data. The highest monthly TWA exposures of those workers averaged 27 μg/m^3 for an average duration of 34 months (Ex. 31–18–3, Table 8). Using the dose coefficient from the linear relative risk model based on cumulative exposure fit to the full Luippold data set in a lifetable analysis, where workers were exposed to this Cr(VI) air concentration and duration starting at age 34 (the average starting age for the Luippold cohort), the additional lifetime risk is predicted to be 4.5 per 1000. This means that less than one additional lung cancer case would be projected for the Luippold subcohort of approximately 100 workers whose highest reported eight-hour TWA (i.e., average 27 μg/m^3) was below the PEL using a linear model without a threshold.

Exponent suggested that the lack of a statistically significant increase in lung cancer mortality observed among workers whose reported average monthly TWA Cr(VI) was not above the PEL was evidence of an absence of increased risk at this level (Ex. 31–18–1). This assertion is not supported by the data. As explained above, the Crump *et al.* analysis lacks the statistical power to support this conclusion. Since exposure at the highest reported TWA accounts for almost all of the cumulative exposure experienced by those workers (Ex. 31–18–3, Table 8), the lack of an observed increase in the lung cancer SMR is entirely consistent with a small, but significant, lung cancer risk as predicted by a linear, non-threshold relative risk model.

E. Supporting Quantitative Risk Assessments

In addition to the preferred data sets analyzed above, there are four other cohorts with available data sets for estimation of additional lifetime risk of lung cancer. These are the Mancuso cohort, the Hayes cohort, the Gerin cohort, and the Alexander cohort. Environ (Ex. 33–15) recently did quantitative risk assessments on study data for all but the Hayes cohort. Several years earlier, the K.S. Crump Division (Ex. 13–5) did quantitative assessments on data from the Mancuso and Hayes cohort, under contract with OSHA. The U.S. EPA (Exs. 19–1; 35–52) developed quantitative risk assessments from the Mancuso cohort data for its Integrated Risk Information System (IRIS). The California EPA (Ex. 35–54), Public Citizen Health Research Group (Ex. 1), and the U.S. Air Force Armstrong Laboratory (AFAL) for the Department of Defense (Ex. 35–51) performed assessments from the Mancuso data using the 1984 U.S. EPA risk estimates as their starting point. The U.S. EPA also published a supporting risk assessment based on the Hayes cohort data (Ex. 7–102). Until the cohort studies of Gibb *et al.* and Luippold *et al.* became available, these earlier assessments provided the most current projected cancer risks from airborne exposure to Cr(VI). While the risk estimates from these data sets are associated with a greater degree of uncertainty, it is nevertheless valuable to compare them to the risk estimates from the preferred Gibb and Luippold cohorts. The cohort data sets and the analyses conducted on them are discussed below.

The Mancuso and Hayes cohorts worked at the Painesville and Baltimore chromate production plants, respectively. Even though the entry date requirements, other cohort selection criteria, and the studied site facilities were different, the lung cancer risk estimated from the Hayes data set may not be completely independent from that estimated from the Gibb data set. A similar situation exists between the Mancuso and Luippold data sets. Unlike the Mancuso and Hayes cohorts, the Gerin and Alexander cohorts were not chromate production workers and lung cancer mortality did not show a statistically significant positive trend with cumulative Cr(VI) exposure. Environ performed quantitative assessments on these data sets to determine if the predicted lung cancer risks had statistical precision that was compatible with those estimated from the preferred Gibb and Luippold cohorts.

1. Mancuso Cohort

As described in subsection VII.B.3, the Mancuso cohort was initially defined in 1975 and updated in 1997. The cohort members were hired between 1931 and 1937 and worked at the same Painesville facility as the Luippold cohort workers. However,

there was no overlap between the two cohorts since all Luippold cohort workers were hired after 1939. The quantitative risk assessment by Environ used data reported in the 1997 update (Ex. 23, Table XII) in which lung cancer deaths and person-years of follow-up were classified into four groups of cumulative exposure to soluble chromium, assumed to represent Cr(VI) (Ex. 33–15). The mortality data and person-years were further broken down by age of death in five year increments starting with age interval 40 to 44 years and going up to >75 years. However, no expected numbers of lung cancers were computed, either for the cohort as a whole or for specific groups of person-years. Environ used two methods for dealing with the lack of expected numbers in order to complete the risk assessment based on this cohort.

In the first method, Environ used the recorded median age and year of entry into the cohort to estimate the calendar years that corresponded to the middle of the age categories for which expected numbers of lung cancers were needed. Data in the Mancuso study indicated that the median age at entry into the cohort was somewhere between 25 and 29 years and that the median year of entry into the cohort was in 1933 or 1934 (Ex. 23). Person-years of observation for the 40–44 age category would have been centered around 1948–49 (i.e., 15 years after 1933–34, where 15 is the difference between the age group under consideration and the median age at entry into the cohort, equal to 40–25 or 44–29). Similar calculations were made for the other age categories. Expected numbers were then derived from the U.S. lung cancer mortality rates for years as close to the target years as could be obtained.

The exposure-response data with the resulting expected number of lung cancer deaths are reported in Table 3 of the 2002 Environ report (Ex. 33–15, p. 39). The mean cumulative exposures to soluble Cr(VI) were assumed to be equal to the midpoints of the tabulated ranges. No lag was assumed for calculating the cumulative exposures. Environ applied three externally standardized models (see models E1–E3 in subsection VII.C.1) to these data. Unlike other data sets modeled by Environ, the age-related parameter k for the Mancuso data set was estimated to be different from 0, so that models E1 and E2 had different dose coefficients (Ex. 33–15, Table 6, p. 42). The quadratic term (i.e., C_2 in model E1) did not significantly improve model fit, so E1 was linear with respect to cumulative exposure.

Since the expected numbers of lung cancers for the Mancuso cohort could only be approximated, Environ also applied a set of internally-standardized models that did not require estimation of expected number of lung cancers to the exposure-response data (Ex. 33–15, p. 24–25). While both externally- and internally-standardized models provided adequate fit to the data ($p \geq 0.13$), the AIC procedure indicated that model E2, the linear relative risk model with an age-dependent exposure term, provided a superior fit over the other models. The next best fitting models, E1 and I2, presented other problems. Model E1 estimated risk predictions that were apparent outliers and the confidence intervals around risk predictions from model I2 were unusually wide (Ex. 33–15, Table 8, p. 43). Further explanation for the inherent instability of these models can be found in the 2002 Environ report (Ex. 33–15, p. 28–29).

The excess risk of lung cancer from a working lifetime exposure to Cr(VI) at the current OSHA PEL using the preferred model E2 is 293 per 1000 workers (95% CI: 188 to 403). The maximum likelihood estimate from working lifetime exposure to 1.0 $\mu g/m^3$ Cr(VI) is 7.0 per 1000 workers (95% CI: 4.1 to 11 per 1000). These estimates are close to those predicted from the Gibb cohort but are higher than predicted from the Luippold cohort. This result indicates that the non-overlapping Painesville worker cohorts (i.e., Mancuso and Luippold cohorts) probably generate independent estimates of risk, even though they were drawn from the same plant.

There are uncertainties associated with both the exposure estimates and the estimates of expected numbers of lung cancer deaths for the 1997 Mancuso data set. The estimates of exposure were derived from a single set of measurements obtained in 1949 (Ex. 7–98). Although little prior air monitoring was available, it is thought that the 1949 air levels probably understate the Cr(VI) concentrations in the plant during some of the 1930s and much of the 1940s when chromate production was high to support the war. The sampling methodology used by Bourne and Yee only measured soluble Cr(VI), but it is believed that the chromate production process employed at the Painesville plant in these early years yielded slightly soluble and insoluble Cr(VI) compounds that would not be fully accounted for in the sampling results (Ex. 35–61). This would imply that risks would be overestimated by use of concentration estimates that were biased low. However, it is possible that the 1949 measurements may not have underestimated the Cr(VI) air levels in the early 1930s prior to the high production years. Some older cohort members were also undoubtedly exposed to less Cr(VI) in the 1950s than measured in 1949 survey.

Another uncertainty in the risk assessment for the Mancuso cohort is associated with the post-hoc estimation of expected numbers of lung cancer deaths. The expected lung cancers were derived based on approximate summaries of the ages and assumed start times of the cohort members. Several assumptions were dictated by reliance on the published groupings of results (*e.g.*, ages at entry, calendar year of entry, age at end of follow-up, etc.) as well as by the particular choices for reference mortality rates (*e.g.*, U.S. rates, in particular years close to the approximated time at which the person-years were accrued). Since the validity of these assumptions could not be tested, the estimates of expected numbers of lung cancer deaths are uncertain.

There is also a potential healthy worker survivor effect in the Mancuso cohort. The cohort was identified as workers first hired in the 1930s based on employment records surveyed in the late 1940s (Ex. 2–16). The historical company files in this time period were believed to be sparse and more likely to only identify employees still working at the plant in the 1940s (Ex. 33–10). If there was a sizable number of unidentified short-term workers who were hired but left the plant in the 1930s or who may have died before 1940 prior to systematic death registration, then there may have been a selection bias (i.e., healthy worker survivor effect) toward longer-term, healthier individuals (Ex. 35–60). Since the mortality of these long-term ''survivors'' is often more strongly represented in the higher cumulative exposures, it can negatively confound the exposure-response and lead to an underestimation of risk, particularly to shorter-term workers (Ex. 35–63). This may be an issue with the Mancuso cohort, although the magnitude of the potential underestimation is unclear.

Several earlier quantitative risk assessments were done on cohort data presented in the 1975 Mancuso report (Ex. 7–11). These assessments did not have access to the 20 additional years of follow-up nor did they have age-grouped lung cancer mortality stratified by cumulative *soluble chromium* (presumed Cr(VI)) exposure), which was presented later in the 1997 update. Instead, age-grouped lung cancer mortality was stratified by cumulative exposure to *total chromium* that

included not only carcinogenic Cr(VI) but substantial amounts of non-carcinogenic Cr(III).

The 1995 risk analysis by K.S. Crump Division, under contract with OSHA, estimated cumulative Cr(VI) exposures by multiplying cumulative total chromium exposure by an adjustment factor of 0.4 (Ex. 13–5). This factor is roughly the average contribution of soluble chromium to the total chromium exposure levels measured across departments in the Painesville plant by Bourne and Yee in 1949 (Ex. 7–98). The K.S. Crump Division used the lung cancer mortality data cross-classified by the eight exposure categories and three age groups reported in Table IX of the 1975 Mancuso report (Ex. 7–11). They estimated the expected number of lung cancer deaths in a manner similar to the Environ assessments in 2002. The median age at entry for the cohort was estimated to be 28.5 years from the 1975 Mancuso study with an estimated median start date of 1934. Average values for cumulative exposure in each group were estimated by the arithmetic mean of the endpoints defining the group.

An externally standardized linear relative risk model was used to fit the exposure-response data. A sensitivity analysis was used to examine the impact of different average cumulative exposure estimates to represent the highest exposure group (>3.0 mg-yr/m^3) since an arithmetic average could not be calculated for this category. The maximum likelihood estimates for the dose coefficient were relatively constant over a wide range of assumed average exposures. However, the best fit occurred when the high-exposure group was excluded from the analysis (p=0.49). This was because the lung cancer mortality ratios observed for workers with the highest cumulative chromium exposure in the Mancuso data set tended to be lower than predicted by linear projections based on the lung cancer mortality data from workers exposed to lower cumulative exposures. The excess lung cancer risks for a working lifetime at the current OSHA PEL (52 μg/m^3) for Cr(VI) range from 246 to 342 per 1000 workers using the different assumptions about the highest exposure group (Ex. 13–5, Table 8). The excess risk estimates from a working lifetime exposures to 0.5 μg/m^3 Cr(VI) ranged from 2.9 to 4.4 per 1000 workers. This was similar to the risk estimated by Environ using the more updated Mancuso data set.

Like Environ, the K.S. Crump Division explored another method of Poisson regression that internally controlled for age, and which consequently alleviated the need to estimate background rates from an external control population. The dose coefficients estimated for the internally standardized linear relative risk model were similar to those from the externally controlled model. However, sensitivity analysis indicated that the internally standardized model may lead to less stable risk estimates, in that relatively minor changes in average exposure assumptions led to bigger changes in the risk estimates.

The U.S. EPA also used exposure-response data presented in Table IX of the 1975 Mancuso report (Ex. 7–11) as the primary data source for calculating its unit risk estimate . The unit risk refers to an incremental lifetime cancer risk over background occurring in a hypothetical population in which all individuals are exposed continuously throughout life to a concentration of 1 μg Cr(VI)/m^3 in the air that they breathe. Like the K.S. Crump Division, the EPA relied on the observed lung cancer deaths cross-classified by age group and cumulative exposure to total chromium. However, rather than estimate the year of cohort death based on age at entry into the study, the EPA chose to determine expected number of lung cancers for the entire cohort, regardless of age at death, using lung cancer mortality statistics for 1964. They estimated that a large proportion of lung cancer deaths in the cohort probably occurred around that year.

The U.S. EPA assessment did not adjust the total cumulative chromium exposure estimates of Mancuso for the contribution of Cr(VI). While the EPA acknowledged that the resulting overestimation of dose would likely lead to an underestimation of risk, they judged that this would be potentially balanced by two factors that tend to overestimate the risk of lung cancer. One factor was the likelihood that the airborne Cr(VI) levels in the 1930s and 1940s were higher than measured by Bourne and Yee in 1949, as mentioned previously. EPA also suggested the possibility that the Mancuso cohort may have smoked more than the general population so that the expected numbers of lung cancer deaths associated with Cr(VI) exposure would be low and the relative risk overestimated for the cohort.

The 1984 U.S. EPA assessment employed an exposure-dependent multistage model of additive risk to fit the 1975 Mancuso cohort data that relied on average chromium exposure, rather than the cumulative workplace exposure (Ex. 19–1). In their review of the U.S. EPA assessment, the K.S. Crump Division pointed out potential flaws in the EPA conversion of cumulative workplace exposure to their ''continuous exposure equivalent'' that resulted in high average chromium exposure estimates and a correspondingly low unit risk (Ex. 13–5, p. 19–21). The U.S. EPA determined that the maximum likelihood estimate of additional lung cancer risk associated with continuous lifetime exposure to 1 μg/m^3 of Cr(VI) was 0.012 (i.e., 12 per 1000). More recently, the EPA corrected its dose conversion for the Mancuso cohort which yielded a higher unit risk estimate of 0.016 per μg Cr(VI)/m^3 (Ex. 35–52).

In 1985, the California Department of Health Services (CDHS) estimated a cancer potency factor for Cr(VI) in support of its Toxic Air Contaminants Program (Ex. 35–54, p. 210–215). They estimated the relative lung cancer risks and continuous total chromium exposure equivalents for the 1975 Mancuso data set using the same assumptions and procedures as the 1984 EPA assessment. An average relative risk and average total chromium exposure level, weighted by the person-years per age and exposure category, were calculated for all groups combined. The average total chromium exposure level was multiplied by one-seventh (0.142) as an assumed adjustment for the fraction of total chromium present as Cr(VI). A linear relative risk model was then used to calculate a ''crude'' approximation of the excess risk from continuous exposure to 1 μg/m^3 of Cr(VI) for a lifetime. The CDHS chose the 95 percent upper confidence limit of 0.15 per μg Cr(VI)/m^3 as their cancer potency factor which is about an order of magnitude greater than the EPA unit risk estimate.

The Public Citizen Health Research Group (PCHRG) attempted to estimate the magnitude of lung cancer risks associated with occupational exposure to Cr(VI) from the 1984 U.S. EPA unit risk for continuous lifetime exposure (Ex. 1). They reported that the excess lung cancer risk from a working lifetime exposure to Cr(VI) at the OSHA PEL (52 μg/m^3) was 220 per 1000 workers. As described in the 1995 report by K.S. Crump Division (Ex. 13–5, p. 27–29), there were several errors in the PCHRG analysis and the correctly calculated excess occupational risk at the OSHA PEL using the EPA unit risk method is 80 cases per 1000 workers. This risk is lower than the estimate from Environ and the K.S. Crump Division, probably as a result of the EPA conversion of occupational cumulative chromium exposure to a continuous average Cr(VI) exposure for an individual lifetime.

The U.S. Air Force Armstrong Laboratory (AFAL) estimated lung cancer risks to U.S. Navy workers from Cr(VI) exposures as a result of welding, abrasive blasting, spray painting, and other operations (Ex. 35–51). They used a cancer potency factor of 41 per mg Cr(VI)/kg-day derived from the 1984 EPA unit risk adjusted for an average breathing rate of 20 m^3/day and body weight of 70 kg. They also reduced their measured airborne Cr(VI) dust concentrations by an assumed respirable fraction of 0.23. The estimated excess lifetime risk from a 45-year occupational exposure to an eight hour TWA 0.5 µg/m^3 using the AFAL methodology and assumptions is about 0.2 per 1000 workers. This is lower than the Environ and K.S Crump Group estimates due to the lower EPA potency factor and the added adjustment for the respirable fraction.

OSHA believes that the Environ quantitative risk assessment is the most credible analysis from the Mancuso cohort. It relied on the updated cohort mortality data and cumulative exposure estimates derived directly from air measurements of soluble chromium. The other assessments used older cohort mortality data with fewer years of follow-up and more problematic exposure estimates and calculations.

2. Hayes Cohort

The K.S. Crump Division (Ex. 13–5) and Gibb *et al.* (Ex. 7–102) assessed risk based on the exposure-response data reported in Table IV by Braver *et al.* (Ex. 7–17) for the cohort studied by Hayes *et al.* (Ex. 7–14). The Hayes cohort overlapped with the Gibb cohort. The Hayes cohort included 734 members, not part of the Gibb cohort, who worked at an older facility from 1945 to 1950 but did not work at the newer production facility built in August 1950. The Hayes cohort excluded 990 members of the Gibb cohort who worked less than 90 days in the new production facility after August 1950. As noted in section VII.B.4, Braver *et al.* derived a single cumulative soluble Cr(VI) exposure estimate for each of four subcohorts of chromate production workers categorized by duration of employment and year of hire by Hayes *et al.* Thus, exposures were not determined for individual workers using a more comprehensive job exposure matrix procedure, as was done for the Gibb and Luippold cohorts. In addition, the exposures were estimated from air monitoring conducted only during the first five of the fifteen years the plant was in operation. Unlike the Mancuso cohort, Hayes *et al.* did not stratify the observed lung cancer deaths by age group. The expected number of lung cancer deaths for each subcohort was based on the mortality statistics from Baltimore.

The K.S. Crump Division applied the externally standardized linear relative risk approach to fit the exposure-response data (Ex. 13–5). The maximum likelihood estimate for the dose coefficient (*e.g.*, projected linear slope of the Cr(VI) exposure-response curve) was 0.75 per mg Cr(VI)/m^3-yr with a 90% confidence bound of between 0.45 and 1.1 per mg Cr(VI)/m^3-yr. These confidence bounds are consistent with the dose coefficient estimate obtained from modeling the Luippold cohort data (0.83, 95% CI: 0.55 to 1.2) but lower than that from the Gibb cohort data (3.5, 95% CI: 1.5 to 6.0). The later result indicates that the two Baltimore chromate production cohorts (i.e., Hayes and Gibb cohorts) probably generate independent estimates of risk, even though they were drawn from facilities at the same site for overlapping periods of time. The linear relative risk model fit the Hayes cohort data well (p=0.50). The K.S. Crump Division predicted the excess risk from occupational exposure to Cr(VI) for a 45 year working lifetime at the OSHA PEL (52 µg/m^3) to be 88 lung cancer cases per 1000 workers (95% CI: 61 to 141). For 1 µg/m^3, about 2 excess lung cancer deaths per 1000 (95% CI: 1.2 to 3.0) were predicted for the same duration of occupational exposure. These estimates are somewhat lower than the corresponding estimates based on the Gibb cohort data, probably because of the rather high average soluble Cr(VI) level (218 µg/m^3) assumed by Braver *et al.* for plant workers throughout the 1950s. If these assumed air levels led to an overestimate of worker exposure, the resulting risks would be underestimated.

Gibb *et al.* provided a risk assessment for the U.S. EPA of the same Braver exposure-response data used by the K.S. Crump Division (Ex. 7–102). In order to determine the EPA unit risk, the cumulative occupational exposures were converted to average lifetime concentration (as discussed in section VII.E.2) and an average age of 55 was assumed at the end of follow-up for members of the Hayes cohort. Gibb *et al.* used the additive risk model E3 with the default value of 1 for C_0, to fit the data. They reported that the maximum likelihood estimate for the dose coefficient was 0.13 per mg/m^3-yr and it yields a unit risk similar to that derived by the EPA from the 1975 Mancuso cohort (Ex. 19–1). Since the excess lung cancer risk from lifetime occupational exposure to Cr(VI) at the OSHA PEL was 80 cases per 1000 workers based on the EPA unit risk from the Mancuso cohort, a similar occupational risk estimate is likely from the Gibb *et al.* unit risk based on the Hayes cohort. This would be consistent with the occupational risk (*e.g.*, 88 cases per 1000 workers) at the OSHA PEL projected from the assessments of the K.S. Crump Division.

3. Gerin Cohort

Environ (Ex. 33–15) did a quantitative assessment of the observed and expected lung cancer deaths in stainless steel welders classified into four cumulative Cr(VI) exposure groups reported in Tables 2 and 3 of Gerin *et al.* (Ex. 7–120). The lung cancer data come from a large combined multi-center welding study in which a statistically significant excess lung cancer risk was observed for the whole cohort and non-statistically significant elevated lung cancer mortality was found for the stainless steel welder subcohorts (Ex. 7–114). A positive relationship with time since first exposure was also observed for the stainless steel welders (the type of welding with the highest exposure to Cr(VI)) but not with duration of employment.

The exposure-response data from the Gerin study was only presented for those stainless steel welders with at least five years employment. Workers were divided into ''ever stainless steel welders'' and ''predominantly stainless steel welders'' groups. The latter group were persons known to have had extended time welding stainless steel only or to have been employed by a company that predominantly worked stainless steel. As mentioned in section VII.B.5, the cumulative exposure estimates were not based on Cr(VI) air levels specifically measured in the cohort workers, and therefore are subject to greater uncertainty than exposure estimates from the chromate production cohort studies. Environ restricted their analysis to the ''ever stainless steel welders'' since that subcohort had the greater number of eligible subjects and person-years of follow-up, especially in the important lower cumulative exposure ranges. The person-years, observed numbers of lung cancers, and expected numbers of lung cancers were computed starting 20 years after the start of employment. Gerin *et al.* provided exposure-response data on welders with individual work histories (about two-thirds of the workers) as well as the entire subcohort. Regardless of subcohort examined, there was no obvious indication of a Cr(VI) exposure-related effect on lung cancer mortality. This may be explained by the

uncertainties in the exposure estimates and presence of co-exposures discussed in section VII.B.5.

Environ used their externally standardized models, E1 to E3, to fit the data (Ex. 33–15). They assumed that the cumulative Cr(VI) exposure for the workers was at the midpoint of the reported range. A value of 2.5 mg/m^3-yr was assumed for the highest exposure group (*e.g.*, >0.5 mg/m^3-yr), since Gerin *et al.* cited it as the mean value for the group, which they noted to also include the ''predominantly stainless steel welders''. All models fit the data adequately (p>0.28) with dose coefficients considerably lower than for the Gibb or Luippold cohorts (Ex. 33–15, Table 6). In fact, the maximum likelihood estimates for the dose coefficients were not statistically different from 0 at the p=0.05 significance level, which would be expected when there is no exposure-related trend.

Environ chose the linear relative risk model, E2, as the best fitting model based on the AIC value. The projected excess risk of lung cancer from a working lifetime exposure to Cr(VI) at the current OSHA PEL using the preferred model E2 was 46 (95% CI: 0 to 130) cases per 1000 workers. The maximum likelihood estimates of excess risk from working lifetime exposure to 1.0 µg Cr(VI)/m^3 was 0.9 (95% CI: 0 to 2.8) cases per 1000 workers, respectively. The rather large 95 percent confidence interval around the maximum likelihood estimate reflects the greater statistical uncertainty associated with risk estimates from the Gerin cohort. The confidence interval overlaps that for equivalent risk estimates from the Luippold cohort but not the Gibb cohort.

4. Alexander Cohort

Environ (Ex. 33–15) did a quantitative assessment of the observed and expected lung cancer incidence in aerospace workers exposed to Cr(VI) classified into four cumulative chromate exposure groups, reported in Table 4 of Alexander *et al.* (Ex. 31–16–3). The lung cancer data come from a retrospective study with a small number (15) of observed lung cancers in a young cohort (median age of 42 years at end of follow-up) with a relatively short follow-up period (median nine years per member). The authors stated that they derived ''estimates of exposure to chromium [VI]'' based on the TWA measurements, but later on referred to ''the index of cumulative total *chromate* exposure (italics added) reported as µg/m^3 chromate TWA-years'' (Ex. 31–16–3, p. 1254). For their analysis, Environ assumed that the cumulative exposures were expressed in µg/m^3-yr of Cr(VI), rather than chromate (CrO_4^{-2}) or chromic acid (CrO_3).

Alexander *et al.* grouped the lung cancer data by cumulative exposure with and without a ten year lag period (Ex. 31–16–3). They found no statistically significant elevation in lung cancer incidence among the chromate-exposed workers or clear trend with cumulative chromate exposure. Environ used the externally standardized linear relative risk model to fit the unlagged data (Ex. 33–15). The additional risk model, E3, could not be applied because no person-years of observation were presented by Alexander *et al.* Environ assumed workers were exposed to a cumulative Cr(VI) exposure at the midpoint of the reported ranges. For the open-ended high exposure category, Environ assumed a cumulative exposure 1.5 times greater than the lower limit of 0.18 mg/m^3 - yr. The model did not fit the data particularly well (p=0.04) and the dose coefficient was considered to be 0 since positive values did not significantly improve the fit. This is not surprising considering the lack of a positive trend between lung cancer incidence and cumulative Cr(VI) exposure for this cohort. Possible reasons for the lack of a positive association between Cr(VI) exposure and lung cancer incidence in this cohort were previously discussed in section VII.B.6.

The best estimate of excess risk of lung cancer from the Alexander cohort was 0 for all exposures to Cr(VI) based on the default dose coefficient. The upper 95 percent confidence bound on the risk was estimated to be 212 cases per 1000 workers from a working lifetime exposure to Cr(VI) at the current OSHA PEL. The upper 95 percent confidence bound on risk from working lifetime exposure to 1.0 mg Cr(VI)/m^3 is 4.8 cases per 1000 workers. The confidence intervals around the risk estimates from the Alexander cohort are greater than those from the Gerin cohort reflecting greater statistical uncertainty. However, the 95 percent confidence intervals for the risk estimates from the Alexander cohort overlap those for equivalent risk estimates from both the Luippold and Gibb cohorts.

If the cumulative exposures from Alexander *et al.* are assumed to be cumulative chromate (CrO_4^{-2}) estimates, then exposures in terms of Cr(VI) would be calculated by dividing by 0.45. As a result, the upper confidence bound on risk would be higher by 1/.45 = 2.2-fold, which would also be statistically consistent with the risk estimates based on the Gibb and Luippold data sets.

F. Summary of Risk Estimates Based on Gibb, Luippold, and Supporting Cohorts

OSHA believes that the best estimates of excess lifetime lung cancer risks are derived from the Gibb and Luippold cohorts. These two cohorts have accumulated a substantial number of lung cancer deaths that were extensively examined in terms of cumulative Cr(VI) exposure. Cohort exposures were reconstructed from air measurements and job histories over three or four decades. The linear relative risk model adequately fitted the Gibb and Luippold data sets, as well as several other supporting data sets. Environ and NIOSH explored a variety of nonlinear dose-response forms, but none provided a statistically significant improvement over the linear relative risk model.

The maximum likelihood estimates from a linear relative risk model fitted to the Gibb data are three-to five-fold higher than estimates based on the Luippold data at equivalent cumulative Cr(VI) exposures and the confidence limits around the projected risks from the two data sets do not overlap. This indicates that the maximum likelihood estimates derived from one data set are unlikely to describe the lung cancer mortality observed in the other data set. Despite this statistical inconsistency between the risk estimates, the differences between them are not unreasonably great given that the cohorts worked in different chromate production facilities and the potential uncertainties involved in estimating cancer risk from the data (see section VII.G). Since the analyses based on these two cohorts are each of high quality and their projected risks are reasonably close (*e.g.*, well within an order of magnitude), OSHA believes the excess lifetime risk of lung cancer from occupational exposure to Cr(VI) is best represented by the range of risks that lie between maximum likelihood estimates of the Gibb and Luippold data sets.

TABLE VII–8.—OSHA ESTIMATES OF EXCESS LUNG CANCER CASES PER 1000 WORKERS[a] EXPOSED TO VARIOUS EIGHT HOUR TWA CR(VI) WITH 95 PERCENT CONFIDENCE INTERVAL COMPARISONS BY COHORT

Cr(VI) (μg/m³)	Best estimates of risk[b]	95% confidence interval on risk estimates by cohort[c]					
		Featured cohorts		Supporting cohorts			
		Gibb	Luippold	Mancuso	Hayes	Guerin	Alexander
0.25	0.52–2.3	1.0–3.9	0.31–0.79	1.0–2.7	0.31–0.75	0.0–0.7	0.0–1.2
0.5	1.0–4.6	2.0–7.8	0.62–1.6	2.0–5.4	0.62–1.5	0.0–1.4	0.0–2.4
1.0	2.1–9.1	4.0–16	1.2–3.1	4.1–11	1.2–3.0	0.0–2.8	0.0–4.8
2.5	5.2–23	10–37	3.1–7.8	10–27	3.1–7.5	0.0–6.9	0.0–12
5.0	10–45	20–75	6.2–15	20–52	6.1–15	0.0–14	0.0–24
10	21–86	39–142	12–31	n/a	12–30	0.0–29	0.0–50
20	41–163	76–256	21–60	n/a	24–51	0.0–54	0.0–91
52	101–351	181–493	62–147	188–403	61–141	0.0–130	0.0–212

[a] The workers are assumed to start work at age 20 and continue to work for 45 years, at a constant exposure level. All estimates were recalculated using year 2000 U.S. reference rates, all races, both sexes, for lung cancer and all causes, except for those from Mancuso, for which 1998 rates were used.

[b] OSHA preliminarily finds that the estimates of risk best supported by the scientific evidence are the ranges bounded by the maximum likelihood estimates from the linear relative risk models presented in Table VII–3 (Baltimore reference population/exposure grouping with equal person-years) for the Gibb cohort and Table VII–7 for the Luippold cohort.

[c] The confidence intervals for the Gibb and Luippold cohorts are from Tables VII–3 and VII–7. The confidence intervals for the Mancuso, Guerin and Alexander cohorts are derived from parameters reported by Environ (2002, Ex. 33–15). All are from the best fitting linear relative risk models and are 95% confidence intervals. The confidence interval for the Hayes cohort was calculated from the 90 percent confidence interval on the dose coefficient for the linear relative risk model reported by the K.S. Crump Division (1995, Ex. 13–5).

OSHA's best estimates of excess lung cancer cases from a 45-year working lifetime exposure to Cr(VI) are presented in Table VII–8. This range of projected risks lie between the maximum likelihood estimates derived from the Gibb and Luippold data sets. As previously discussed, several acceptable assessments of the Gibb data set were performed, with similar results. The 2003 Environ model E1, applying the Baltimore City reference population and ten exposure categories based on a roughly equal number of person-years per group, was selected to represent the range of best risk estimates derived from the Gibb cohort, in part because this assessment employed an approach most consistent with the exposure grouping applied in the Luippold analysis (see Table VII–7). To characterize the statistical uncertainty of OSHA's risk estimates, Table VII–8 also presents the 95% confidence limits associated with the maximum likelihood risk estimates from the Gibb cohort and the Luippold cohort. The confidence interval on the risk estimates from the Luippold data set is smaller (i.e., just over a two-fold range) than those for the Gibb data set (i.e., about a 3.5-fold range) but the Gibb cohort is larger. Therefore, it appears reasonable to consider both analyses jointly in providing estimates of lung cancer risk.

OSHA finds that the most likely lifetime excess risk at the current PEL of 52 μg/m³ Cr(VI) lies between 101 per 1000 and 351 per 1000, as shown in Table VII–8. That is, OSHA predicts that between 101 and 351 of 1000 workers occupationally exposed for 45 years at the current PEL would develop lung cancer as a result of their exposure. The wider range of 62 per 1000 (lower 95% confidence bound, Luippold cohort) to 493 per 1000 (upper 95% confidence bound, Gibb cohort) illustrates the range of risks considered statistically plausible, based on these cohorts and, thus, represents the statistical uncertainty in the estimates of lung cancer risk. This range of risks roughly falls proportionally with exposure so that estimates at 5 μg/m³ are about 10 to 45 cases per 1000 workers and estimates at 0.5 μg/m³ are about 1 to 4.5 cases per 1000 workers.

The 95 percent confidence limits on estimates of risk for the four supporting cohort data sets are also presented in Table VII–8. As discussed previously, the exposure-response data from supporting cohorts are not as strong as those from the two featured cohorts. The cumulative Cr(VI) exposure reconstructions in these data sets were based on more limited air measurements and were frequently not linked to cohort workers on an individual basis. Some of the cohort data sets were weaker in terms of either number of workers, length of follow-up, documented mortality data, and possibility of co-exposures or a healthy worker survivor effect. These features may have introduced bias into the estimates of risk determined from the studies. However, observed lung cancers were grouped across multiple exposure groups in these more problematic cohorts that allowed quantitative assessments to be done and compared against the stronger Gibb and Luippold cohorts.

OSHA believes the supplemental assessments support the range of projected excess lung cancer risks from the Gibb and Luippold cohorts. This is illustrated by the 95 percent confidence intervals shown in Table VII–8. The confidence interval encompasses those risk estimates that are consistent with the cohort data to a certainty of 95 percent. The confidence intervals tend to be smaller for the larger data sets and better model fits. OSHA's range of best risk estimates for a given occupational Cr(VI) exposure overlap the 95 percent confidence bands for each of the four supporting cohorts. This indicates that the range of best estimates includes risks with a statistical precision that is compatible with all the exposure-response data sets, including the smaller Gerin and Alexander cohorts where the lung cancers did not show a clear positive trend with cumulative Cr(VI) exposure.

The 95 percent confidence intervals from the four supporting cohorts overlap those of either the Gibb or Luippold cohorts (or both). The confidence intervals for estimates of the Mancuso cohort overlap with those of the Gibb cohort but are higher than those of the Luippold cohort. The risks projected from the Mancuso data set are likely overestimated because they depend on air monitoring conducted near the end of the study period when exposures were likely lower and because the sampling method only captured highly soluble Cr(VI) compounds. The Mancuso cohort was also probably exposed to significant

amounts of the more potent slightly soluble and insoluble chromates (*e.g.*, calcium chromate). The relative potency of Cr(VI) compounds is further discussed in section VII.G.4. The confidence intervals for estimates from the Hayes cohort overlap the Luippold cohort but are lower than those of the Gibb cohort. The risks projected from the Hayes cohort may be low because the cumulative exposure estimates rely on air monitoring near the beginning of the study period when Cr(VI) levels were likely higher. The confidence intervals for estimates from the Gerin cohort also overlap those from the Luippold but not the Gibb cohort. The confidence intervals for estimates from the Alexander cohort overlap those from both featured cohorts.

While there is statistical consistency between the range of best risk estimates based on the primary studies and those estimated from the supporting data sets, the risk analysis does not account for potential bias introduced by the lack of exposure data, inadequate follow-up and other limitations in these weaker studies. Unfortunately, the magnitude and direction of this potential bias cannot be reasonably assessed and, thus, the impacts on the risk estimates are unclear.

It would be difficult to formally combine the data or the results (*e.g.*, parameter estimates) from the six studies considered for quantitative analysis. The inclusion criteria (*e.g.*, duration of employment required for entry into the cohorts) differed from study to study. Moreover, the reported cumulative exposure categories were based on different lag periods before accumulation of exposure began. Nevertheless, the lung cancer risks derived from all the data sets, as a group, support the range of best estimates derived from the two featured cohorts.

G. Issues and Uncertainties

The risk estimates presented in the previous sections include confidence limits that reflect statistical uncertainty. This statistical uncertainty concerns the limits of precision for statistical inference, given assumptions about the input parameters and risk models (*e.g.*, exposure estimates, observed lung cancer cases, expected lung cancer cases, linear dose-response). However, there are uncertainties with regard to the above input and assumptions, not so easily quantified, that may impact the degree of confidence in the OSHA risk estimates. Some of these uncertainties are discussed below.

1. Uncertainty With Regard to Worker Exposure to Cr(VI)

The uncertainty that may have the greatest impact on risk estimates relates to the assessment of worker exposure. Even for the Gibb cohort, whose exposures were estimated from roughly 70,000 air measurements over a 35-year period, the calculation of cumulative exposure is inherently uncertain. The methods used to measure airborne Cr(VI) did not characterize particle size that determines deposition in the respiratory tract (see section VI.A.). Workers differ from one another with respect to working habits and they may have worked in different areas in relation to where samples are taken. Inter-individual (and intra-facility) variability in cumulative exposure can only be characterized to a limited degree, even with extensive measurement. The impact of such variability is likely less for estimates of long-term average exposures when there were more extensive measurements in the Gibb and Luippold cohorts in the 1960s through 1980s, but could affect the reliability of estimates in the 1940s and 1950s when air monitoring was done less frequently. Exposure estimates that rely on annual average air concentrations are also less likely to reliably characterize the Cr(VI) exposure to workers who are employed for short periods of time. This may be particularly true for the Gibb cohort in which a sizable fraction of cohort members were employed for only a few months.

Like many retrospective cohort studies, the frequency and methods used to monitor Cr(VI) concentrations may also be a source of uncertainty in reconstructing past exposures to the Gibb and Luippold cohorts. Exposures to the Gibb cohort in the Baltimore plant from 1950 until 1961 were determined based on periodic collection of samples of airborne dust using high volume sampling pumps and impingers that were held in the breathing zone of the worker for relatively short periods of time (*e.g.*, tens of minutes) (Ex. 31–22–11). High volume sampling with impingers to collect Cr(VI) samples may have underestimated exposure since the accuracy of these devices depended on an air flow low enough to ensure efficient Cr(VI) capture, the absence of agents capable of reducing Cr(VI) to Cr(III), the proper storage of the collected samples, and the ability of short-term collections to accurately represent full-shift worker exposures. Further, impingers would not adequately capture any insoluble forms of Cr(VI) present, although other survey methods indicated minimal levels of insoluble Cr(VI) were produced at Baltimore facility (Ex. 13–18–14).

In the 1960s, the Baltimore plant expanded its Cr(VI) air monitoring program beyond periodic high volume sampling to include extensive area monitoring in 27 exposure zones around the facility. Multiple short-term samples were collected (*e.g.*, twelve one-hour or eight three-hour samples) on cellulose tape for an entire 24 hour period and analyzed for Cr(VI). Studies have shown that Cr(VI) can be reduced to Cr(III) on cellulose filters under certain circumstances so there is potential for underestimation of Cr(VI) using this collection method. Gibb *et al.* reported that the full set of monitoring data records was not accessible prior to 1971. The area monitoring was supplemented by routine full-shift personal monitoring of workers starting in 1977. The 24-hour area sampling supplemented with personal monitoring was continued until plant closure in 1985.

The Exponent critique of the Gibb cohort suggested that the tape samplers used in the Baltimore plant from the mid-1960s to 1985 resulted in reduction of Cr(VI) to Cr(III) and that Braver *et al.* excluded these measurements from their analyses because of concerns about underestimation of Cr(VI) concentration (Ex. 31–18–14). While there may be some potential for Cr(VI) reduction on these tape samplers, Gibb *et al.* reported that the tape measurements did not significantly differ from personal breathing zone air measurements ''for approximately two-thirds of the job titles with sufficient number of samples to make the comparison'' (Ex. 31–22–11, p. 118). Furthermore, Gibb *et al.* reported that exposure estimates from the area tape sampling system were adjusted to an equivalent personal exposure estimate using job-specific ratios of the mean area and personal breathing estimates determined during the 1978–1985 time period when both were in operation (Ex. 31–22–11, p. 117). Any potential exposure underestimation of Cr(VI) by the tape sampling system should be minimized by this correction procedure. Braver *et al.* considered the usual post-1960 Cr(VI) exposures of 31 ug/m3 to be ''less credible because they were very low'' compared to prior time periods (*e.g.*, pre-1950s) and, therefore, excluded workers exposed after 1960 from their exposure assessment (Ex. 7–17, p. 372). However, this exposure level turned out to be very consistent with the more extensive Cr(VI) concentrations later reported by Gibb *et al.* (Ex. 31–22–11) and Proctor *et al.* (Ex. 35–61) for

chromate production plants in the 1960s and 1970s.

Some of the same uncertainties exist in reconstructing exposures from the Luippold cohort. Exposure monitoring from operations at the Painesville plant in the 1940s and early 1950s was sparse and consisted of industrial hygiene surveys conducted by various groups (Ex. 35–61). The United States Public Health Service (USPHS) conducted two industrial hygiene surveys (1943 and 1951), as did the Metropolitan Life Insurance Company (1945 and 1948). The Ohio Department of Health (ODH) conducted surveys in 1949 and 1950. The most detailed exposure information was available in annual surveys conducted by the Diamond Alkali Company (DAC) from 1955 to 1971. Exponent chose not to consider the ODH data in their analysis since the airborne Cr(VI) concentrations reported in these surveys were considerably lower than values measured at later dates by DAC. Excluding the ODH survey data in the exposure reconstruction process may have led to higher worker exposure estimates and lower predicted lung cancer risks.

There were uncertainties associated with the early Cr(VI) exposure estimates for the Painesville cohort. Like the monitoring in the Baltimore plant, Cr(VI) exposure levels were determined from periodic short-term, high volume sampling with impingers that may have underestimated exposures (Ex. 35–61). Since the Painesville plant employed a ''high-lime'' roasting process to produce soluble Cr(VI) from chromite ore, a significant amount of slightly soluble and insoluble Cr(VI) was formed. It was estimated that up to approximately 20 percent of the airborne Cr(VI) was in the less soluble form in some areas of the plant prior to 1950 (Ex. 35–61). The impingers were unlikely to have captured this less soluble Cr(VI) so some reported Cr(VI) air concentrations may have been slightly underestimated for this reason.

The annual air monitoring program at the Painesville plant was upgraded in 1966 in order to evaluate a full 24 hour period (Ex. 35–61). Unlike the continuous monitoring at the Baltimore plant, twelve area air samples from sites throughout the plant were collected for only 35 minutes every two hours using two in-series midget impingers containing water. The more frequent monitoring using the in-series impinger procedure may be an improvement over previous high-volume sampling and is believed to be less susceptible to Cr(VI) reduction than cellulose filters. While the impinger collection method at the Painesville plant may have reduced one source of potential exposure uncertainty, another source of potential uncertainty was introduced by failure to collect air samples for more than 40 percent of the work period. Also, personal monitoring of workers was not conducted at any time.

Another type of uncertainty is associated with extrapolation from one exposure pattern to another (*e.g.*, different combinations of exposure duration and Cr(VI) air concentrations). Both Gibb *et al.* and Luippold *et al.* found that lung cancer mortality showed a significant trend with cumulative Cr(VI) exposure, which is being employed by OSHA as the exposure metric of choice in its quantitative risk assessments. However, the Cr(VI) exposure levels experienced by the cohorts were higher (*e.g.*, 5 to 10,000 $\mu g/m^3$) than for some of the lower exposure scenarios (*e.g.*, 0.25 to 2.5 $\mu g/m^3$) of interest to OSHA. The cohorts were also exposed for a considerably shorter duration than a 45-year working lifetime. Uncertainties arise when extrapolating risks for Cr(VI) concentrations and exposure durations outside the experience of the cohort data, even when cumulative exposures are similar.

There are several examples in which an increasing relative risk of chronic disease has been observed to attenuate (*e.g.*, the slope of the exposure-response lessens) at high cumulative exposures (Ex. 35–55). A variety of reasons can cause this behavior including the healthy worker survivor effect previously discussed, a limit on the relative risk that can be achieved for diseases with a high background rate (*e.g.*, lung cancer), and misclassification of exposure. Since the cumulative exposure for a full working lifetime at the current OSHA PEL is higher than observed in almost all workers from the Gibb cohort and most of the Luippold cohort, it is possible that a linear relative risk model might overpredict the excess risk at this exposure if there were a significant attenuation in the slope of the exposure-response.

In order to evaluate the likelihood of an attenuated relative risk of lung cancer at high cumulative Cr(VI) exposures, Environ fit the Gibb and Luippold data sets to a power model of the form:

$$\text{Relative Risk} = E(1 + bd^C)$$

where E was the expected number of lung cancer deaths, d is the cumulative exposure, and b and c were parameters to be estimated (Ex. 36–2). The parameter, c, was allowed to be less than 1, which would accommodate a decreasing slope in the exposure-response with increasing cumulative exposure. Of course, the power model assumes a linear shape, if c = 1. The power model fit to the two primary data sets produced maximum likelihood estimates of 0.61 and 0.66 for the Gibb and Luippold data sets, respectively. However, the power models did not significantly improve the fit compared to the linear model (p = 0.41 and 0.14 for Gibb and Luippold, respectively). This is consistent with the conclusions of NIOSH and Exponent who also reported that departure from linearity in the exposure-response was not significant for these data sets (Exs. 33–13; 33–12). In light of the above analyses, OSHA does not find adequate reason to believe a linear relative risk model overpredicts the lung cancer risk for a full working lifetime at the OSHA PEL. This is especially true since this Cr(VI) exposure is well within the range of cumulative exposures experienced by workers in the Luippold cohort.

While the cumulative Cr(VI) exposure estimates determined from the Gibb and Luippold cohorts are much more extensive than usually available for a cancer cohort, they are still a primary source of uncertainty in the assessment of risk. As occurs in many retrospective cancer epidemiologic studies, it was difficult to reconstruct worker exposure in the 1950s from the limited air monitoring data available from the Painesville and Baltimore plants. It appears that the usual airborne Cr(VI) exposure levels in some chromate production and processing areas at these facilities dropped five to ten-fold from the late 1940s to the mid-1960s with little documentation in the intervening years. This required more indirect methods to complete the job-exposure matrices for these cohorts. The need to reconstruct cohort exposure in the absence of extensive air measurements combined with the different procedures used to collect air samples at the two plants could partially explain the slight but statistically different exposure-specific risks between the Gibb and Luippold cohorts. Finally, some uncertainty in risk is introduced when extrapolating cohort exposures to higher Cr(VI) levels for shorter periods to an equivalent cumulative exposure of lower intensity for a longer duration (*e.g.*, 45 year exposure to 0.25 $\mu g/m^3$). Despite the uncertainties, the exposure estimates from the Gibb *et al.* and Luippold *et al.* studies are derived from the best available data and better than is generally found in retrospective cohort studies. They are more than adequate to assess occupational risk to

Cr(VI) and OSHA does not believe the potential inaccuracies in the exposure assessment for either cohort are large enough to result in serious overprediction or underprediction of risk.

2. Model Uncertainty, Exposure Threshold, and Dose Rate Effects

The models used to fit the observed data may also introduce uncertainty into the quantitative predictions of risk. Linear and non-linear risk models based on a Poisson distribution were applied to the exposure-response data sets. Both Environ (Ex. 33–12) and NIOSH (Ex. 33–13) evaluated nonlinear models among the suite of models fit to the Gibb *et al.* cohort data. These included quadratic, log-linear, log-square-root, and log-quadratic models as well as models that included cumulative dose raised to some power. Cox proportional hazard models were also applied to the data. Linear models generally fit the exposure-response data better than the nonlinear models. For most data sets, there was no indication that any model more elaborate than a linear model was necessary to describe the exposure-response patterns observed in these cohorts.

The linear relative risk model was used to estimate excess lung cancer risks at cumulative Cr(VI) exposures in the range of 0.01 to 2.3 mg/m^3-yr (*i.e.,* $0.25-52\ \mu g/m^3$ for 45 years) which, to a large extent, overlap the cumulative exposures experienced of workers in either the Gibb or Luippold cohorts. Certainly, cumulative exposures above 0.1 mg/m^3-yrs (*e.g.,* 2.5 $\mu g/m^3$ for 45 years) are within the exposure range of both studies. Since risks were estimated at cumulative exposures generally within the range of the data represented in the preferred cohorts, they are less susceptible to dose-extrapolation uncertainties and less susceptible to model misspecification. Thus, OSHA believes that the use of a linear model is a reasonable and appropriate basis on which to calculate lung cancer risks at the cumulative occupational exposures of interest, especially given the consistency in the results from fitting the linear model across most of the studies.

In their response to the OSHA Request For Information regarding occupational exposure to Cr(VI), the Chrome Coalition submitted comments, prepared by Exponent, suggesting that a threshold dose-response model is an appropriate approach to estimate lung cancer risk from Cr(VI) exposures (Ex. 31–18–1). Their arguments rely on: (1) The lack of a statistically significant increased lung cancer risk for workers exposed below a cumulative Cr(VI) exposure of 1.0 mg/m^3-yr (*e.g.,* roughly equivalent to 20 $\mu g/m^3$ TWA for a 45 year working lifetime) and below ''a highest reported eight hour average'' Cr(VI) concentration of 52 $\mu g/m^3$ (*i.e.,* OSHA PEL); (2) the presumed existence of ''an overall reducing capacity'' within the lung for extracellular reduction of Cr(VI) to Cr(III) that must be exceeded before Cr(VI) can damage cellular DNA, and (3) a reported dose rate effect for lung tumor development in rats exposed to Cr(VI) by long-term, repeated intratracheal instillations.

The lack of a statistically significant result for a subset of the entire cohort should not be construed to imply a threshold. As pointed out in an earlier discussion (section VII.D) and by Crump *et al.,* the Luippold data set does not have the statistical power to detect small increases in risk that may be associated with the lower cumulative exposures in the cohort (Ex. 35–58). In their report, Exponent acknowledges that the non-significant increase in lung cancer deaths in the Luippold cohort below 1.25 mg $Cr(VI)/m^3-yr$ cumulative exposure is consistent with predictions from a linear relative risk model (Ex. 31–18–1, p.25).

The Chrome Coalition characterized the work of De Flora *et al.* as providing convincing support for the existence of a threshold exposure (*i.e.,* exposure below which the probability of disease is zero) for Cr(VI) carcinogenicity. De Flora *et al.* determined the amount of soluble Cr(VI) reduced to Cr(III) *in vitro* by human bronchioalveolar fluid and pulmonary alveolar macrophage fractions over a short period (Ex. 31–18–7). These specific activities were used to estimate an ''overall reducing capacity'' of 0.9–1.8 mg Cr(VI) and 136 mg Cr(VI) per day per individual for the two preparations, respectively. As discussed in Health Effects section VI.A., cell membranes are permeable to Cr(VI) but not Cr(III), so only Cr(VI) enters cells to any appreciable extent. De Flora *et al.* interpreted these data to mean that high levels of Cr(VI) would be required to ''overwhelm'' the reduction capacity before significant amounts of Cr(VI) could enter lung cells and damage DNA, thus creating a biological threshold to the exposure-response (Ex. 31–18–8).

There are several problems with the threshold interpretation of De Flora *et al.* The *in vitro* reducing capacities were determined in the absence of cell uptake. Cr(VI) uptake into lung cells happens concurrently and in parallel with its extracellular reduction, so it cannot be concluded from the De Flora data that a threshold reduction capacity must be exceeded before uptake occurs. The rate of Cr(VI) reduction to Cr(III) is critically dependant on the presence of adequate amounts of reductant, such as ascorbate or GSH (Ex. 35–65). It has not been established that sufficient amounts of these reductants are present throughout the thoracic and alveolar regions of the respiratory tract to create a biological threshold. Moreover, the *in vitro* activity of Cr(VI) reduction in epithelial lining fluid and alveolar macrophages was shown to be highly variable among individuals (Ex. 31–18–7, p. 533). It is possible that Cr(VI) is not rapidly reduced to Cr(III) in some workers or some areas of the lung. Finally, even if there was an exposure threshold created by extracellular reduction, the De Flora data do not establish the dose range in which the putative threshold would occur. It has already been shown that a physiological concentration of ascorbate substantially reduces, but may not eliminate, the uptake in cells treated with low M concentrations of Cr(VI) for 24 hours (Ex. 35–68). OSHA does not believe that there is sufficient scientific evidence to support the Chrome Coalition conclusion that the De Flora data ''suggest a linear, non-threshold model to predict cancer risk at low exposure levels [at least, those being considered by OSHA] is overly conservative and inappropriate'' (Ex. 31–18–1, p.2).

The Chrome Coalition has stated that the intratracheal instillation study in rats by Steinhoff *et al.* ''suggests that there is likely a threshold exposure level below which there is no increase in lung cancer risk, and that the threshold is compound-specific.'' (Ex. 31–18–1, p. 2). The Steinhoff study is discussed in detail in section VI.B. on carcinogenic effects. Briefly, the study showed that rats intratracheally administered 1.25 mg/kg of soluble sodium dichromate or slightly soluble calcium chromate once a week for 30 months developed significant increases (about 17 percent incidence) in lung tumors (Ex. 11–7). The same total dose administered more frequently (*e.g.*, five times weekly) at a five-fold lower dose level did not increase lung tumor incidence in the sodium dichromate-treated rats and significantly increased lung tumor incidence (about 7.5 percent) in the calcium chromate-treated rats by only about half as much as rats that received the greater dose level.

OSHA does not believe that the accelerated tumor development at the high Cr(VI) dose levels in the Steinhoff *et al.* study ''clearly support that there is a threshold for Cr(VI) exposures'' or indicate that ''peak exposures high enough to overload the reductive capacity of the lung may be a better

predictor of lung cancer risk than lifetime cumulative exposure'' as stated by Chrome Coalition (Ex. 31–18–1, p. 31). Rather, OSHA believes these findings should be interpreted to suggest that Cr(VI)-induced carcinogenesis is influenced not only by the total Cr(VI) dose retained in the respiratory tract but also by the rate at which the dose is administered. For example, the highest dose level (i.e., 1.25 mg/kg) in the study was reported to cause moderate to severe lung damage, including inflammation and hyperplasia. It is likely that these effects caused a proliferative stimulus that accelerated the neoplastic transformation and expansion of initiated (i.e., genetically altered) cells. The Steinhoff *et al.* study also suggests that lung damage is not an absolute requirement for Cr(VI)-induced tumorigenesis. This is illustrated by the significant, but smaller, increased tumor incidence in the animals receiving a lower dose level (i.e., 0.25 mg/kg) of Cr(VI), as calcium chromate, that caused relatively minor non-neoplastic changes in the lungs.

OSHA believes that the existence of dose rate effects is supported by the available scientific evidence and may introduce uncertainty when projecting lung cancer risk based on workers exposed to higher Cr(VI) concentrations for shorter durations to workers exposed to the same cumulative exposure but at substantially lower Cr(VI) concentrations for substantially longer periods. However, the Steinhoff *et al.* study instilled the Cr(VI) compounds directly on the trachea rather than introduce the test compound by inhalation and was only able to characterize a significant dose rate effect at one cumulative dose level (*e.g.*, 1.25 mg/kg). For these reasons, OSHA considers the data inadequate to reliably determine the human exposures where a dose rate effect might occur and to confidently predict its magnitude.

OSHA solicits comment on the whether the linear relative risk model is the most appropriate approach on which to estimate risk associated with occupational exposure to Cr(VI). OSHA is particularly interested in whether there is convincing scientific evidence of a non-linear exposure-response relationship and, if so, whether there are sufficient data to develop a non-linear model that would provide more reliable risk estimates than the linear approach being used in the preliminary assessment.

3. Influence of Smoking, Race, and the Healthy Worker Survivor Effect

A common confounder in estimating lung cancer risk to workers from exposure to a specific agent such as Cr(VI) is the impact of cigarette smoking. First, cigarette smoking is known to cause lung cancer. Ideally, lung cancer risk attributable to smoking among the Cr(VI)-exposed cohorts should be controlled or adjusted for in characterizing exposure-response. Secondly, cigarette smoking may interact with the agent (i.e., Cr(VI)) or its biological target (i.e., susceptible lung cells) in a manner that enhances or even reduces the risk of developing Cr(VI)-induced lung cancer from occupational exposures, yet is not accounted for in the risk model.

OSHA believes its risk estimates have adequately accounted for the potential confounding effects of cigarette smoking in the underlying exposure-lung cancer response data, particularly for the Gibb cohort. One of the key issues in this regard is whether or not the reference population utilized to derive the expected number of lung cancers appropriately reflects the smoking behavior of the cohort members. The risk analyses of the Gibb cohort by NIOSH and Environ indicate that cigarette smoking was properly controlled for in the exposure-response modeling. NIOSH applied a smoking-specific correction factor that included a cumulative smoking term for individual cohort members (Ex.33–13). Environ applied a generic correction factor and used lung cancer mortality rates from Baltimore City as a reference population that was most similar to the cohort members with respect to smoking behavior and other factors that might affect lung cancer rates (Ex. 33–12). Environ also used internally standardized models that did not require use of a reference population and included a smoking-specific (yes/no) variable. All these models predicted very similar estimates of risk over a wide range of Cr(VI) exposures. There was less information about smoking status for the Luippold cohort. However, regression modeling that controlled for smoking indicated that it was not a significant confounding factor when relating Cr(VI) exposure to the lung cancer mortality (Ex. 35–58).

Smoking has been shown to interact in a synergistic manner (i.e., combined effect of two agents are greater than the sum of either agent alone) with some lung carcinogens, most notably asbestos (Ex. 35–114). NIOSH reported a slightly negative but nonsignificant interaction between cumulative Cr(VI) exposure and smoking in a model that had separate linear terms for both variables (Ex. 33–13). This means that, at any age, the smoking and Cr(VI) contributions to the lung cancer risk appeared to be additive, rather than synergistic, given the limited smoking information in the Gibb cohort along with the cumulative smoking assumptions of the analysis. In their final linear relative risk model, NIOSH included smoking as a multiplicative term in the background rate in order to estimate lifetime lung cancer risks attributable to Cr(VI) independent of smoking. Although this linear relative risk model makes no explicit assumptions with regard to an interaction between smoking and Cr(VI) exposure, the model does assume a multiplicative relationship between the background rate of lung cancer in the reference population and Cr(VI) exposure. Therefore, to the extent that smoking is a predominant influence on the background lung cancer risk, the linear relative risk model implicitly assumes a multiplicative (*e.g.*, greater than additive and synergistic, in most situations) relationship between cumulative Cr(VI) exposure and smoking. Since current lung cancer rates reflect a mixture of smokers and non-smokers, it is reasonable to expect that the excess lung cancer risks from Cr(VI) exposure predicted by the linear relative risk model to overestimate the risks to non-smokers to some unknown extent. By the same token, the model may underestimate the risk from Cr(VI) exposure to a heavy smoker. Because there were so few non-smokers in the study cohorts (*e.g.*, approximately 15 percent of the exposed workers and four lung cancer deaths in the Gibb cohort), it was not possible to reliably estimate risk for this subpopulation.

Although OSHA is not aware of any convincing evidence of a specific interaction between cigarette smoking and Cr(VI) exposure, prolonged cigarette smoking does have profound effects on lung structure and function that may indirectly influence lung cancer risk from Cr(VI) exposure . Cigarette smoke is known to cause chronic irritation and inflammation of the respiratory tract. This leads to decreases in airway diameter that could result in an increase in Cr(VI) particulate deposition. It also leads to increased mucous volume and decreased mucous flow, that could result in reduced Cr(VI) particulate clearance. Increased deposition and reduced clearance would mean greater residence time of Cr(VI) particulates in the respiratory tract and a potentially greater probability of developing bronchogenic cancer. Chronic cigarette

smoking also leads to lung remodeling and changes in the proliferative state of lung cells that could influence susceptibility to neoplastic transformation. While the above effects are plausible consequences of cigarette smoking on Cr(VI)-induced carcinogenesis, the likelihood and magnitude of their occurrence have not been firmly established and, thus, the impact on risk of lung cancer in workers is uncertain.

Differences in lung cancer incidence with race may also introduce uncertainty in risk estimates. Gibb *et al.* reported differing patterns for the cumulative exposure-lung cancer mortality response between whites and non-whites in their cohort of chromate production workers (Ex. 31–22–11). In the assessment of risk from the Gibb cohort, NIOSH reported a strong interaction between cumulative Cr(VI) exposure and race, such that nonwhites had a higher cumulative exposure coefficient (i.e., higher lung cancer risk) than whites based on a linear relative risk model (Ex. 33–13). If valid, this might explain the slightly lower risk estimates in the predominantly white Luippold cohort. However, Environ found that including race as an explanatory variable in the Cox proportional hazards model C1 did not significantly improve model fit (p=0.15) once cumulative Cr(VI) exposure and smoking status had been considered (Ex. 33–12).

NIOSH suggested that exposure or smoking misclassification might plausibly account for the Cr(VI) exposure-related differences in lung cancer by race seen in the Gibb cohort (Ex. 33–13, p. 15). It is possible that such misclassification might have occurred as a result of systematic differences between whites and non-whites with respect to job-specific Cr(VI) exposures at the Baltimore plant, unrecorded exposure to Cr(VI) or other lung carcinogens when not working at the plant, or in smoking behavior. Unknown racial differences in biological processes critical to Cr(VI)-induced carcinogenesis could also plausibly account for an exposure-race interaction. However, OSHA is not aware of evidence that convincingly supports any of these possible explanations.

Another source of uncertainty that may impact the risk estimates is the healthy worker survivor effect. Studies have consistently shown that short-term employed workers have higher mortality rates than workers with long-term employment status. This is possibly due to a higher proportion of ill individuals and those with a less healthy lifestyle (Ex. 35–60). As a result, exposure-response analyses based on mortality of long-term healthy workers will tend underestimate the risk to short-term workers and vice versa, even when their cumulative exposure is similar. This might partially explain the higher risk estimates from the Gibb data set relative to the Luippold data set for the same cumulative exposures using similar risk models. The Gibb cohort contained a higher proportion of workers with short duration of employment, lower cumulative Cr(VI) exposure, and is arguably more prone to mortality. On the other hand, the Luippold cohort consisted of longer-term workers at higher cumulative exposures that may be more prone to negative confounding as a result of the survivor effect. The healthy worker survivor effect is thought to be less of a factor in diseases with a multifactorial causation and long onset, such as cancer.

4. Potency Considerations of Different Cr(VI) Compounds

An issue that needs to be addressed is whether the excess lung cancer risks derived from epidemiologic data for chromate production workers are representative of the risks for other Cr(VI)-exposed workers (*e.g.*, plating, painting, welding operations). Typically, OSHA has used epidemiologic studies from one industry to estimate risk for other industries. In many cases, this approach is acceptable because it is exposure to a common agent of concern that is the primary determinant of risk and not some other factor unique to the workplace. However, in the case of Cr(VI), workers in different industries are exposed to various Cr(VI) compounds that differ in carcinogenic potency depending to a large extent on water solubility. The chromate production workers in the Gibb and Luippold cohorts were primarily exposed to certain highly water-soluble chromates. As more fully described in section VI.B. of the Cancer Effects section and summarized below, the scientific evidence indicates that the carcinogenic potency of the highly water-soluble chromates is likely lower than the potency of other less water-soluble Cr(VI) compounds. Therefore, OSHA believes that the lung cancer risk of workers in other industries exposed to equivalent levels of Cr(VI) will be of similar magnitude, or possibly even greater in the case of some workers exposed to certain Cr(VI) compounds, than the risks projected from chromate production workers in the Gibb and Luippold cohorts.

The primary operation at the plants in Painesville and Baltimore was the production of the water-soluble sodium dichromate from which other primarily water-soluble chromates such as sodium chromate, potassium dichromate, and chromic acid could be made (Exs. 7–14; 35–61). Therefore, it is likely that the Gibb and Luippold cohorts were principally exposed to water-soluble Cr(VI). The Painesville plant used a high-lime process known to form some less water-soluble Cr(VI) compounds (Ex. 35–61). Less water-soluble chromates is a designation that refers to all chromates not considered to be highly water soluble and readily captured by an aqueous impinger sampling device. These would include both slightly water-soluble chromates, such as calcium and strontium chromate and the more water-insoluble chromates, such as zinc and lead chromate. The 1953 USPHS survey confirmed that approximately 20 percent of the total Cr(VI) in the roasting residue at the Painesville plant consisted of the less water-soluble chromates (Ex. 2–14). The Painesville plant subsequently reduced and eliminated exposure to Cr(VI) roasting residue through improvements in the production process. The high-lime process was not used at the Baltimore plant and the 1953 USPHS survey detected minimal levels of less soluble Cr(VI) at this facility (Ex. 7–17). Proctor *et al.* estimated that a proportion of the Luippold cohort prior to 1950 were probably exposed to the less water-soluble Cr(VI) compounds, but that it would amount to less than 20 percent of their total Cr(VI) exposure (Ex. 35–61). A small proportion of workers in the Special Products Division of the Baltimore plant may also have been exposed to less water-soluble Cr(VI) compounds during the occasional production of these compounds over the years.

As discussed in the preamble section VI.B on carcinogenic effects, both water-soluble and insoluble forms of Cr(VI) compounds are regarded as carcinogenic to the respiratory tract as a result of inhalation. This is not only supported by epidemiologic studies of the chromate production workers above, but also by studies of chromate pigment workers exposed primarily to the insoluble zinc and lead chromates (Exs. 7–36; 7–42; 7–49). The standardized lung cancer incidence and mortality ratios reported among these pigment workers were relatively high and clearly significant. Langard and Vigander found that the lung cancer incidence among a cohort of workers exposed primarily to zinc chromate, but also lead chromate, at a pigment production plant in

Norway was 44 times what would be expected from an age- and sex-adjusted Norwegian population (Ex. 7–36). The Davies study found from 2.2-($p<0.01$) to 5.6-fold ($p<0.001$) excess lung cancer mortality for various cohorts of pigment workers exposed to both zinc and lead chromate at two British factories (Ex. 7–42). Workers in jobs judged to involve the highest Cr(VI) exposure had the highest risk of lung cancer. A cohort study of workers exposed to the highly water-soluble chromic acid during electroplating operations also reported excess lung cancer mortality (Ex. 35–62). While the lung cancer mortality was significantly elevated in pigment and electroplating cohorts, there was inadequate exposure information for risk analysis.

The slightly water-soluble Cr(VI) compounds, calcium and strontium chromate, led to significant increases in tumors when instilled in the respiratory tract of experimental animals (Exs. 11–7; 11–2). Levy *et al.* reported a bronchial carcinoma incidence of 43 percent (43/99) and 25 percent (25/100) after a single 2 mg intrabronchial instillation of strontium chromate and calcium chromate, respectively (Ex. 11–2). This compares with the non-significant bronchial carcinoma incidence of one percent (1/100) in rats instilled with 2 mg of highly water-soluble sodium dichromate in the same study. Steinhoff *et al.* reported a 7.5 percent tumor incidence (6/80, $p<0.01$) following repeated intratracheal instillations of 0.25 mg/kg slightly water-soluble calcium chromate in rats (Ex. 11–7). The same dosing of the highly water-soluble sodium dichromate produced no tumor incidence (0/80) in the same study. This and other evidence led IARC to conclude that there was sufficient evidence for carcinogenicity in experimental animals of the less water-soluble strontium chromate, calcium chromate, zinc chromates, and lead chromates but only limited evidence for carcinogencity in experimental animals of the highly water-soluble chromic acid and sodium dichromate (Ex. 18–1, p. 213). Because the above animal studies either used an inadequate number of dose levels (*e.g.*, single dose level) or employed a less appropriate route of administration (*e.g.*, tracheal instillation), it was not possible to determine a reliable quantitative estimate of risk for human workers breathing these chromates during occupational exposure. IARC drew the overall conclusion that all Cr(VI) compounds are carcinogenic to humans based on the combined results of animal studies, human epidemiological evidence and other data relevant to the carcinogenic mode of action.

Other studies reported that insoluble Cr(VI) compounds are retained in the lung for longer periods and are considered a more persistent source of locally available Cr(VI) for uptake into lung cells than water-soluble Cr(VI) compounds. Bragt and Van Dura found that water-soluble sodium chromate is more rapidly absorbed and cleared from the lung than the highly insoluble lead chromate when intratracheally instilled in rats (Ex. 35–56). On day 50 after instillation, 13.8 percent of the initial lead chromate remained in the lungs as opposed to only 3.0 percent of the initial sodium chromate. Research at George Washington University Medical Center showed that treatment of embryo cells in culture with insoluble lead chromate particulates led to cell-enhanced dissolution and uptake of Cr(VI) resulting in DNA damage and neoplastic transformation (Exs. 35–104; 35–69; 35–132). Internalization, dissolution, and uptake of lead chromate and the resulting damage to DNA were later shown to also occur in normal human lung epithelial cells (Exs. 35–66; 35–327). Elias *et al.* showed that a wide range of insoluble lead and zinc chromate pigments could morphologically transform normal mammalian cells into neoplastic cells (Ex. 12–5). These studies have led the researchers to suggest that the less water-soluble Cr(VI) compounds may be more carcinogenic in the lung than the highly water-soluble Cr(VI) since these insoluble chromate particulates provide a persistent source of high Cr(VI) concentration within the immediate microenvironment of the lung cell surface (Exs. 35–67; 35–149).

Experts have evaluated the combined epidemiologic, animal, and mechanistic evidence and concluded that the less water-soluble chromates are likely more carcinogenic than highly water-soluble Cr(VI) compounds (Exs. 17–101; 17–5B). This is reflected in the lower recommended ACGIH TLVs for insoluble Cr(VI) compounds (i.e., 10 mg/m3) and certain slightly soluble Cr(VI) compounds (*e.g.*, 1 mg/m3 for calcium chromate; 0.5 mg/m3 for strontium chromate) than the recommended TLV for the water-soluble Cr(VI) compounds (*e.g.*, 50 mg/m3). For all the reasons cited above, OSHA believes the lung cancer risk for workers exposed to equivalent levels of Cr(VI) compounds other than sodium chromate and sodium dichromate over a working lifetime is likely to be similar in magnitude to the risks projected from the chromate production workers in the Gibb and Luippold cohorts, or possibly even greater in the case of inhaled slightly water-soluble and insoluble Cr(VI) particulates.

OSHA seeks comment on whether its preliminary assessment of risk based on the exposure-response data from the two cohorts of chromate production workers is reasonably representative of the risks expected from equivalent exposures to different Cr(VI) compounds encountered in other industry sectors. Of particular interest is whether there is convincing evidence that the preliminary risk estimates from worker cohorts primarily engaged in the production of the highly water soluble sodium chromate and sodium dichromate would substantially overpredict the lung cancer risk for workers exposed at the same level and duration to airborne Cr(VI) during welding operations, chromic acid aerosol in electroplating operations, the less water soluble Cr(VI) particulates encountered during pigment production and painting operations, or Cr(VI) exposure in other important industry sectors and job categories.

H. Expert Peer Review of the OSHA Draft Preliminary Quantitative Risk Assessment

OSHA contracted an independent organization known as Toxicology Excellence for Risk Assessment (TERA) to organize an external scientific peer review of the January 21, 2004 Draft Quantitative Risk Assessment (Exs. 36–1–1; 36–1–2). TERA selected three peer reviewers based on a high level of competence in occupational epidemiology and/or risk assessment. The reviewers were screened to ensure no apparent conflict of interest or involvement in the key studies that provided the basis for the OSHA assessment. OSHA did not participate in the selection process other than to examine reviewer credentials to confirm their qualifications. The three peer reviewers selected by TERA were Dr. David Gaylor, Dr. Allan Smith, and Dr. Irva Hertz-Picciotto. Curriculum Vitae of the three reviewers have been submitted to the docket (Ex. 36–1–3).

TERA provided the peer reviewers with a review package that consisted of the draft quantitative risk assessment, copies of the key studies, and a set of instructions and questions (Ex. 36–1–1). The reviewers were asked to comment on several aspects of the draft OSHA risk assessment including the suitability of the different data sets for exposure-response analysis, the choice of exposure metric and risk models, the appropriateness of the risk estimates, and the characterization of key issues and uncertainties. The peer reviewers filed written draft reports with TERA

which then reviewed the comments for completeness before passing the reports on to OSHA (Ex. 36–1–4). OSHA requested clarification in writing on some of the reviewer responses. These were addressed by the peer reviewers in their final peer review reports or answered in an attachment (Ex. 36–1–4–3). The clarification process with the reviewers was handled by TERA.

The three peer reviewers agreed that the results from six occupational cohorts under review were adequately evaluated as to their suitability for exposure-response analysis and concurred that the Gibb and Luippold cohorts provided the strongest data sets for quantitative assessment. There was general agreement among the peer reviewers that the risk models and statistical methodologies used in the OSHA assessment were appropriately applied. Dr. Smith remarked that ''there is no question in my mind that relative risk models are superior to others when conducting quantitative cancer risk assessments on epidemiological data'' (Ex. 36–1–4–2) and commended OSHA for supporting a relatively straightforward [linear] model widely used in epidemiology (Ex. 36–1–4–2). At his suggestion, OSHA expanded on reasons for using a linear relative risk model to fit the epidemiological data. The selection of the linear relative risk model was not solely based on mathematical fit. Relative risk models inherently adjust for age-related increases in cancer incidence. The linear relative risk model has been extensively and successfully used to analyze other cancer mortality data sets and is an accepted approach in carcinogen risk assessment.

The peer reviewers were also in general agreement that cumulative exposure based on time-weighted average air concentrations by job title and employment history was a reasonable exposure metric to use. Dr. Hertz-Picciotto stated ''the use of cumulative exposure constructed in this way is currently the standard, and the use of individual job histories is the best available method at this time (Ex. 36–1–4–4).'' She pointed out that the underlying assumption that exposure patterns and dose rate differences at equivalent cumulative exposures do not influence cancer risk is an uncertainty in the assessment. This is more fully explained in section VII.G.1 on uncertainties with regard to worker exposure.

Dr. Smith raised another limitation to the cumulative exposure metric as it relates to relative risk. It has been shown, in some instances, that relative risk of chronic disease will not continue to rise at high cumulative exposure but will tend to stabilize or attenuate. In the case of a significant attenuation, the excess risk at high Cr(VI) exposures (*e.g.*, working lifetime at the current OSHA PEL) could be overestimated by a linear relative risk model. Environ examined this possibility by fitting the Gibb and Luippold data sets to a power model that requires the exposure-response to rise steeply at low exposure and level out at high exposure (Ex. 36–2). The power model did not significantly improve the fit compared to the linear relative risk model for either data set. This analysis would not support a significant attenuation in the relative risk of lung cancer with increasing cumulative Cr(VI) exposure. Therefore, OSHA does not find adequate reason to believe its linear relative risk model would overpredict the lung cancer risk at the OSHA PEL or other cumulative exposures in the range of interest. OSHA revised its preliminary quantitative risk assessment to fully address this issue in section VII.G.1.

The peer reviewers showed less enthusiasm for the highest reported average monthly Cr(VI) air concentration as an appropriate exposure metric or for an exposure threshold below which there exists no lung cancer risk. Dr. Hertz-Picciotto remarked that ''the newly published Crump *et al.* (2003) uses the monthly maximum [Cr(VI) concentration], but fails to take duration into account, and the authors note considerable variability was present in duration at the highest monthly exposure'' and ''the inadequacy of the attempt to prove a threshold is excellently presented [by OSHA]'' (Ex. 36–1–4–4). Dr. Gaylor stated ''a threshold concentration or threshold cumulative exposure to Cr(VI) below which no excess lung cancer is expected cannot be established from the available information (Ex. 36–1–4–1).'' Dr. Smith added ''the [OSHA] reasons given for dismissing Exponent's threshold inference are valid. I would add [Exponent's] assessment ignores duration of exposure. For example, it is unlikely one could detect increased lung cancer risks in smokers whose 'peak exposure' was a quarter pack per day if they only smoked for three years. This would not mean that a quarter pack per day is a threshold (Ex. 36–1–4–2).''

The peer reviewers found the range of excess lifetime risks of lung cancer presented by OSHA to be sound and reasonable. These preferred risk estimates were those bounded by the maximum likelihood estimates determined from the featured Gibb and Luippold data sets. Dr. Gaylor wrote ''the confidence limits are tighter for the Luippold study, somewhat over a factor of two for the range from the lower to the upper 95% confidence limit, compared to a range of about 3.5 for the confidence limits in the Gibb study. However, the Gibb cohort is larger than the Luippold cohort. It appears reasonable to consider the two studies jointly to provide estimates of lung cancer risk'' (Ex. 36–1–4–1). Dr. Gaylor went on to point out that the range of maximum likelihood between the featured data sets understates the [statistical] uncertainty in the risk estimates. He recommended that the uncertainty be expressed as the lower 95% confidence limit from the Luippold data set and the 95% upper confidence limit for the Gibb data set. OSHA agrees and has revised section VII.F to make clear that while the maximum likelihood range represents the most likely estimates of lung cancer risk, the 95% confidence bounds are the better representation of statistical uncertainty.

Dr. Gaylor suggested that the OSHA assessment make clear that the 45-year working lifetime exposure should be regarded as a worst case scenario and that the typical worker would be exposed to Cr(VI) for a shorter period of time. Dr. Smith also questioned the need to estimate risk from a 45-year working lifetime. He suggested that OSHA could probably make more confident estimates of risk for shorter exposure durations (*e.g.*, ten years) within the range observed in the cohort studies. This would avoid the uncertainties of an upward extrapolation. OSHA does not disagree with these comments. However, the OSH Act is clear on the agency statutory obligation to consider the risk of material impairment from regular exposure to the hazardous agent for a full working life. The risk of lung cancer from Cr(VI) exposures for less than a full working lifetime are discussed in section VIII on Significance of Risk and section IX on Benefits Analysis.

Dr. Hertz-Picciotto felt that OSHA may have overstated the consistency in lung cancer risk between the two primary studies and the four weaker supporting studies. She pointed out that two of the supporting cohorts overlapped the featured cohorts and were not truly independent data sets. She indicated that the weaker supporting studies had serious bias that rendered the discussion of overlap in confidence intervals to be relatively meaningless and, thus, prevented a definitive evaluation of consistency. OSHA agrees that the magnitude and direction of potential bias introduced by lack of exposure data, inadequate follow-up, and other limitations in the

supporting studies prevents strong statements regarding consistency among risks estimates. However, OSHA believes the finding that its risk predictions based on the Gibb and Luippold data sets are within a statistical precision that is compatible with other exposure-response data sets enhances confidence in the estimates. OSHA notes that there was no overlap in the Mancuso and Luippold cohorts, even though they worked at the same plant, due to vastly different selection criteria and exposure estimation based on different industrial hygiene surveys. The Hayes and Gibb cohort have some overlap but the cohorts primarily worked at different facilities and exposure estimates were, again, based on different monitoring surveys. In the case of both cohort pairs, statistical comparisons show that the risk estimates from one data set would not be consistent with the other data set at the 95% confidence level. OSHA believes the risks from the different cohorts can be considered independent estimates. OSHA has revised sections VII.E and VII.F to clarify the positions discussed above.

Dr. Smith suggested that OSHA consider presenting risk estimates that can be readily calculated from the source data without use of a complex mathematical model. He contends that this would allow the reader to better understand how the risks relate to measures reported in the published studies. He provided some illustrations of simple and transparent risk estimations from the Gibb *et al.* study. OSHA agrees there is merit to comparing risk estimates easily calculated from the cohort mortality data with the more precise estimates determined from the linear relative risk model as a kind of ''reality check''. OSHA has included such calculations in sections VII.C.4 for the Gibb data set and section VII.D for the Luippold data set.

OSHA does not agree with assertions by Dr. Smith that ''there is no valid basis to conclude that more complex calculations [from mathematical models], such as found in the source material and draft [OSHA] document, have any greater validity than this estimate [directly calculated from the published cohort data]'' and ''there is no gain in validity in doing a full life table analysis but there is certainly a loss in transparency (Ex. 36–1–4–2).'' OSHA believes excess risk estimated from standard, well-supported mathematical model constructs that incorporate the entire mortality data set is considerably more accurate, more robust, more stable and more statistically rigorous than a simple calculation from a single relative risk result determined from a small subset of the cohort data as applied by Dr. Smith. The life table analysis adjusts for both the increasing probability of developing lung cancer with advancing age and the competing risk of death from other causes. These age-related factors are not accounted for in a simple relative risk calculation and may lead to a less accurate risk estimate.

While the peer reviewers felt that most uncertainties in the risk assessment were adequately characterized, they suggested certain topics receive more attention. Dr. Hertz-Picciotto suggested that sensitivity analyses on plausible alternate exposure assumptions for workers in the Gibb and Luippold cohorts during the periods when there was very limited air monitoring data ''would add concrete information on the magnitude of uncertainty in the risk estimates (Ex. 36–1–4–4).'' Environ, while under contract with OSHA, had access to annual exposure estimates on individual workers in the Gibb cohort. They explored the feasibility of generating plausible alterative exposures using a forward and reverse replacement scheme for the air concentrations imputed during periods in the Gibb *et al.* study when air monitoring was unavailable (Ex. 36–2). Unfortunately, lack of job title information and job-specific monitoring data combined with apparent high job transfer and turnover among workers made this approach impracticable for estimating plausible exposures that could lead to a meaningful analysis. OSHA did not have access to individual exposure data for the Luippold cohort.

Dr. Hertz-Picciotto recommended that OSHA address the potential impact on risk of the healthy worker survivor effect. The healthy worker survivor effect refers to a common observation that long-term workers have been found to have lower mortality than short-term workers. As a result, exposure-response analyses based on mortality of long-term healthy workers will tend to underestimate the risk to short-term workers and vice versa. This healthy worker effect may partially explain the higher risk estimates for the same cumulative exposures from the Gibb cohort, which included a higher proportion of workers with short exposure duration, relative to the Luippold cohort of longer-term workers. The healthy worker survivor effect may have also influenced risks estimated from the Mancuso cohort. OSHA agrees that the healthy worker survivor effect contributes to the uncertainty in the risk estimates and has included a discussion in section VII.G.3 on issues and uncertainties and in the section VII.E.1 on the Mancuso data set.

Dr. Smith thought that some important issues surrounding smoking needed to be better addressed in the preliminary risk assessment document. He agreed that OSHA adequately discussed the confounding due to smoking but suggested that it be made clear that the linear relative risk model, in the absence of any explicit interaction term between smoking and Cr(VI), implicitly assumes a synergy (i.e., lung cancer risk from smoking and Cr(VI) together is greater than the sum of the risks from either agent alone) between the two exposures. OSHA believes Dr. Smith has a valid point. Although the linear relative risk model makes no explicit assumptions with regard to an interaction between smoking and Cr(VI) exposure, the model does assume a multiplicative relationship between the background rate of lung cancer in the reference population and Cr(VI) exposure. Therefore, to the extent that smoking is a predominant influence on the background lung cancer risk, the linear relative risk model implicitly assumes a multiplicative (*e.g.*, greater than additive and synergistic, in most situations) relationship between cumulative Cr(VI) exposure and smoking. Since the background lung cancer rate reflects a mixture of smokers and non-smokers, the expectation is that the projected OSHA risks from Cr(VI) exposure are overestimated for a non-smoker to some unknown extent. By the same token, the model may underestimate the risk from Cr(VI) exposure to a heavy smoker. A discussion of this has been included in section VII.G.3.

Finally, the peer reviewers believed that OSHA adequately presented its position that workers in the Gibb and Luippold cohorts were primarily exposed to the less carcinogenic, highly water-soluble Cr(VI) compounds and that the lung cancer risks for workers exposed to equivalent levels of other Cr(VI) compounds will be of a similar magnitude and possibly greater in the case of certain less water-soluble Cr(VI). However, the peer reviewers stated that they lacked the expertise in toxicology and experimental carcinogenesis to critically evaluate its consistency with the existing scientific data. OSHA has made it clear in section VII.G.4 that the animal studies demonstrating higher carcinogenic potency for sparingly water-soluble Cr(VI), such as calcium chromate and strontium chromates, can not provide reliable quantitative estimates of human risk. This is because the studies employed an inadequate

number of dose levels or the studies employed routes of administration (*e.g.*, intratracheal instillation) less relevant to occupational exposure.

I. Preliminary Conclusions

OSHA believes that the best quantitative estimates of excess lifetime lung cancer risks are those derived from the data sets described by Gibb *et al.* and Luippold *et al.* Both data sets show a significant positive trend in lung cancer mortality with increasing cumulative Cr(VI) exposure. The exposure assessments for these two cohorts were reconstructed from air measurements and job histories over three or four decades and were superior to those of other worker cohorts. The linear relative risk model generally provided the best fit among a variety of different models applied to the Gibb *et al.* and Luippold *et al.* data sets. It also provided an adequate fit to four other supporting data sets. Thus, OSHA believes the linear relative risk model is the most appropriate model to estimate excess lifetime risk from occupational exposure to Cr(VI). Using the Gibb *et al.* and Luippold *et al.* data sets and a linear relative risk model, OSHA preliminarily concludes that the lifetime lung cancer risk is best expressed by the three-to five-fold range of risk projections bounded by the maximum likelihood estimates from the two featured data sets. This range of projected risks is within the 95 percent confidence intervals from all six data sets.

OSHA does not believe that it is appropriate to employ a threshold dose-response approach to estimate cancer risk from a genotoxic carcinogen, such as Cr(VI). Federal Agencies, including OSHA, assume an exposure threshold for cancer risk assessments to genotoxic agents only when there is convincing evidence that such a threshold exists. In addition, OSHA does not consider absence of a statistically significant effect in an epidemiologic or animal study that lacks power to detect such effects to be convincing evidence of a threshold. OSHA also does not consider theoretical reduction capacities determined in vitro with preparations that do not fully represent physiological conditions within the respiratory tract to be convincing evidence of a threshold. Finally, as previously discussed, linear (and some non-linear) no-threshold risk models adequately fit the existing exposure-response data.

The Gibb and Luippold cohorts were predominantly exposed to water-soluble chromates, particularly sodium dichromate. The scientific evidence indicates that the water-soluble Cr(VI) compounds are generally less potent carcinogens than slightly-water soluble and water-insoluble Cr(VI) compounds. These less water-soluble Cr(VI) compounds are retained in the lung for longer periods, are more likely to concentrate at the lung cell surface, and are a more persistent source of locally available Cr(VI) for uptake into target cells than the highly water-soluble Cr(VI) compounds. Risks estimated from chromate production workers primarily exposed to water-soluble chromates in the Gibb and Luippold cohorts should adequately represent risks to workers exposed to other water-soluble Cr(VI) compounds. OSHA believes that workers exposed to equivalent levels of the potentially more carcinogenic, less water-soluble Cr(VI) compounds may even be at greater risk of lung cancer than predicted from the Gibb and Luippold cohorts.

As with any risk assessment, there is some degree of uncertainty in the projected risks that result from the data, assumptions, and methodology used in the analysis. The exposure estimates in the Gibb *et al.* and Luippold *et al.* data sets relied, to some extent, on a paucity of air measurements using less desirable sampling techniques to reconstruct Cr(VI) exposures, particularly in the 1940s and 1950s. Additional uncertainty is introduced when extrapolating from the cohort exposures to higher Cr(VI) levels for shorter periods to an equivalent cumulative exposure of lower intensity and longer duration of interest to OSHA. The study cohorts were mostly smokers but detailed information on their smoking behavior was unavailable. While the risk assessments make some adjustments for the confounding effects of smoking, it is unknown whether the assessments fully account for any interactive effects that smoking and Cr(VI) exposure may have on the carcinogenic action. In any case, OSHA does not have reason to believe the above uncertainties would introduce errors that would result in serious overprediction or underprediction of risk.

OSHA s preliminary estimate of lung cancer risk from a 45 year occupational exposure to Cr(VI) at an 8-hour TWA at the current PEL of 52 $\mu g/m^3$ is 101 to 351 excess deaths per 1000 workers. This range, which is defined by maximum likelihood estimates based on the Gibb and Luippold epidemiological cohorts, is OSHA's best estimate of excess risk; it does not account for uncertainty due to the statistical nature of the analyses, or for other potential sources of uncertainty or bias. The wider range of 62 to 493 per 1000 represents the statistical uncertainty associated with OSHA's excess risk estimate at the current PEL, based on lowest and highest 95% confidence bounds on the maximum likelihood estimates for the two featured data sets. The excess lung cancer risks at alternative 8 hour TWA PELs that were under consideration by the Agency are shown in Table VI–8, together with the uncertainty bounds for the primary and supporting studies at these exposure concentrations. The excess lung cancer risks at alternate 8 hour TWA PELs under consideration by the Agency are shown in Table VI–8. For example, OSHA s best estimate of excess risk from 45 years' exposure at 1 $\mu g/m^3$ Cr(VI) is 2.1 to 4.6 per 1000; an interval of 1.2 − 16 per 1000 represents the statistical uncertainty of OSHA s estimate. The 45-year exposure estimates satisfy the Agency s statutory obligation to consider the risk of material impairment for an employee with regular exposure to the hazardous agent for the period of his working life (29 U.S.C. 651 *et seq.*). Occupational risks from Cr(VI) exposure to less than a full working lifetime are considered in Section VIII on the Significance of Risk and in Section IX. on the Benefits Analysis.

VIII. Significance of Risk

In promulgating health standards, OSHA uses the best available information to evaluate the risk associated with occupational exposures, to determine whether this risk is severe enough to warrant regulatory action, and to determine whether a new or revised rule will substantially reduce this risk. OSHA makes these findings, jointly referred to as the ''significant risk determination'', based on the requirements of the OSH Act and the Supreme Court's interpretation of the Act in the ''benzene'' decision of 1980 (*Industrial Union Department, AFL–CIO* v. *American Petroleum Institute,* 448 U.S. 607). The OSH Act directs the Secretary of Labor 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 * * * for the period of his working life [6(b)(5)].

OSHA's authority to promulgate regulations for the cause of worker protection is limited by the requirement that standards be ''reasonably necessary and appropriate to provide safe or healthful employment'' [3(8)].

In the benzene decision, the Supreme Court's interpretation of Section 3(8)

further defined OSHA's regulatory authority. The Court stated:

> By empowering the Secretary to promulgate standards that are ''reasonably necessary or appropriate to provide safe or healthful employment and places of employment,'' the Act implies that, before promulgating any standard, the Secretary must make a finding that the workplaces in question are not safe (IUD v. API 448 U.S. at 642).

''But 'safe' is not the equivalent of 'risk-free' '', the Court maintained. ''[T]he 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'' (*IUD* v. *API* 448 U.S. at 642). It has been Agency practice to establish this finding by estimating risk to workers using quantitative risk assessment, and determining the significance of this risk based on judicial guidance, the language of the OSH Act, and Agency policy considerations.

The Agency has considerable latitude in defining significant risk and in determining the significance of any particular risk. The Court did not stipulate a means to distinguish significant from insignificant risks, but rather instructed OSHA to develop a reasonable approach to the significant risk determination. The Court stated that ''it is the Agency's responsibility to determine in the first instance what it considers to be a ''significant'' risk'', and did not express ''any opinion on the * * * difficult question of what factual determinations would warrant a conclusion that significant risks are present which make promulgation of a new standard reasonably necessary or appropriate'' (448 U.S. at 659). The Court also stated that, while OSHA's significant risk determination should be supported by substantial evidence, the Agency ''is not required to support the finding that a significant risk exists with anything approaching scientific certainty''. Furthermore, ''A reviewing court [is] to give OSHA some leeway where its findings must be made on the frontiers of scientific knowledge [and] * * * 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'', so long as such assumptions are based in ''a body of reputable scientific thought'' (448 U.S. at 655, 656).

To make the significance of risk determination for a new or proposed standard, OSHA uses the best available scientific evidence to identify material health impairments associated with potentially hazardous occupational exposures, and, when possible, to provide a quantitative assessment of exposed workers' risk of these impairments. OSHA has reviewed extensive epidemiological and experimental research pertaining to adverse health effects of occupational Cr(VI) exposure, including lung cancer, and has established preliminary quantitative estimates of the excess lung cancer risk associated with currently allowable Cr(VI) exposure concentrations and the expected impact of the proposed PEL. OSHA has preliminarily determined that long-term exposure at the current PEL causes significant risk to workers' health, and that adoption of the proposed PEL will significantly reduce this risk.

A. Material Impairment of Health

As discussed in Section VI of this preamble, inhalation exposure to Cr(VI) causes a variety of adverse health effects, including lung cancer, nasal septum damage, and asthma. OSHA considers these conditions to be material impairments of health, as they are marked by significant discomfort and long-lasting adverse effects, can have adverse occupational and social consequences, and may in some cases have permanent or potentially life-threatening consequences. Based on this finding and on the scientific evidence linking Cr(VI) inhalation to each of these effects, OSHA concludes that exposure to Cr(VI) causes ''material impairment of health or functional capacity'' within the meaning of the OSH Act.

OSHA considers lung cancer, an irreversible and frequently fatal disease, to be a clear material impairment of health. OSHA's finding that inhaled Cr(VI) causes lung cancer is based on the best available epidemiological data, reflects substantial evidence from animal and mechanistic research, and is consistent with the conclusions of other government and public health organizations, including NIOSH, EPA, ACGIH, NTP, and IARC (Exs. 35–117; 35–52; 35–158; 17–9–D; 18–3, p. 213). The Agency's primary evidence comes from two epidemiological studies that show significantly increased incidence of lung cancer among workers in the chromate production industry (Exs. 25; 33–10). The high quality of the data collected in these studies and the analyses performed on them has been confirmed by OSHA and by independent peer review. Supporting evidence of Cr(VI) carcinogenicity comes from occupational cohort studies in chromate production, chromate pigment production, and chromium plating, and by cell culture research into the processes by which Cr(VI) disrupts normal gene expression and replication. Studies demonstrating uptake, metabolism, and genotoxicity of a variety of soluble and insoluble Cr(VI) compounds support the Agency's position that all Cr(VI) compounds should be regulated as occupational carcinogens (Exs. 35–148; 35–68; 35–67; 35–66; 12–5; 35–149; 35–134).

While OSHA has relied primarily on the association between Cr(VI) inhalation and lung cancer to demonstrate the necessity of the proposed standard, the Agency has also determined that several other material health impairments can result from exposure to airborne Cr(VI). As shown in several cross-sectional and cohort studies, inhalation of Cr(VI) can cause nasal passage atrophy, ulceration, and septum perforation (Exs. 35–1; 7–3; 9–126; 35–10; 9–18; 3–84; 7–50; 31–22–12). Septum ulcerations are often accompanied by swelling and bleeding, heal slowly, and in some cases may progress to a permanent perforation that can only be repaired surgically. Inhalation of Cr(VI) can also lead to occupational asthma, a potentially life-threatening condition in which workers become allergic to Cr(VI) compounds and experience symptoms such as coughing, wheezing, and difficulty in breathing upon exposure to small amounts of airborne Cr(VI). Several case reports have documented occupational asthma from Cr(VI) exposure, confirming Cr(VI) as the sensitizing agent by bronchial challenge (Exs. 35–7; 35–12; 35–16; 35–21).

B. Risk Assessment

When possible, epidemiological or experimental data and statistical methods are used to characterize the risk of disease that workers may experience under the current PEL, as well as the expected reduction of risk that would occur with implementation of the proposed PEL. The Agency finds that the available epidemiological data are sufficient to support quantitative risk assessment for lung cancer among Cr(VI)-exposed workers. Using the best available studies, OSHA has preliminarily identified a range of expected risk from regular occupational exposure at the current PEL (101–351 excess lung cancer deaths per 1000 workers) and at the proposed PEL of 1 $\mu g/m^3$ (2.1–9.1 per 1000 workers), assuming a working lifetime of 45 years' exposure in each case. These values represent the best estimates of multiple analysts working with data on two extensively studied worker populations,

and are highly consistent across analyses using a variety of modeling techniques and assumptions. While some attempts have been made to assess the relationship between Cr(VI) exposure level and noncancer adverse health effects, the Agency does not believe that a reliable quantitative risk assessment can be performed for noncancer effects at this time, and has therefore characterized noncancer risk qualitatively.

For preliminary estimates of lung cancer risk from Cr(VI) exposure, OSHA has relied upon data from two cohorts of chromate production workers. The Gibb cohort, which originates from a chromate production facility in Baltimore, Maryland, includes 2357 workers who began work between 1950 and 1974 and were followed up through 1992 (Ex. 25). The extensive exposure documentation available for this cohort, the high statistical power afforded by the large cohort size, and the availability of information on individual workers' race and smoking status provide a particularly strong basis for risk analysis. The Luippold cohort, from a facility in Painesville, Ohio, includes 482 workers who began work between 1940 and 1972, worked for at least one year at the plant, and were followed up through 1997 (Ex. 33–10). This cohort also provides a very strong basis for risk analysis, in that it has high-quality documentation of worker Cr(VI) exposure and mortality, a long period of followup, and a large proportion of relatively long-term employees (55% > 5 years).

Risk assessments were performed on the Gibb cohort data by Environ International Corporation (Ex. 33–12), under contract with OSHA; Park *et al.*, as part of an ongoing effort by NIOSH (Ex. 33–13); and Exponent on behalf of the Chrome Coalition (Ex. 31–18–15–1). A variety of statistical models were considered, allowing OSHA to identify the most appropriate models and assess the resulting risk estimates' sensitivity to alternate modeling approaches. Models were tried with additive and relative risk assumptions; various exposure groupings and lag times; linear and nonlinear exposure-response functions; external and internal standardization; reference lung cancer rates from city-, state-, and national-level data; inclusion and exclusion of short-term workers; and a variety of ways to control for the effects of smoking. OSHA's preferred approach, a relative risk model using Baltimore lung cancer reference rates, and NIOSH's preferred approach, a relative risk model using detailed smoking information and U.S. lung cancer reference rates, are among several models that use reasonable assumptions and provide good fits to the data. As discussed in section VII, the Environ, Park *et al.*, and linear Exponent models yield similar predictions of excess risk from exposure at the current and proposed PELs (see Tables VII–3 and VII–4). OSHA's preferred model predicts about 350 excess lung cancers per 1000 workers exposed for a working lifetime of 45 years at the current PEL (MLE 351, 95% CI 181–493) when person-years of exposure are spread evenly across exposure groups (see Table VII–3). Implementation of the proposed PEL is expected to reduce this risk to about 10 excess lung cancers per 1000 workers (MLE 9.1, 95% CI 4–16).

Environ and Crump *et al.* performed risk assessments on the Luippold cohort, exploring additive and relative risk models, linear and quadratic exposure-response functions, and several exposure groupings (Exs. 35–59; 35–58). Additive and relative risk models by both analyst groups fit the data adequately with linear exposure-response. The linear models by all of the analyst groups predicted similar excess risks, from which OSHA has selected preferred estimates based on the Crump *et al.* analysis of about 100 excess lung cancer deaths per 1000 workers exposed for 45 years at the current PEL (MLE 101, 95% CI 62–147), and two excess lung cancer deaths per 1000 workers exposed for 45 years at the proposed PEL (MLE 2.1, 95% CI 1.2–3.1).

The risk assessments performed on the Luippold cohort yield somewhat lower estimates of lung cancer risk than those performed on the Gibb cohort. This discrepancy is probably not due to statistical error in the risk estimates, as the confidence intervals for the estimates do not overlap. The risk estimates based on the Gibb and Luippold cohorts are nonetheless reasonably close. OSHA believes that both cohorts support reasonable estimates of lung cancer risk, and based on their results has selected a representative range of 101–351 per 1000 for 45 years' occupational exposure at the current PEL and 2.1–9.1 per 1000 for 45 years' occupational exposure at the proposed PEL for the significant risk determination. OSHA's confidence in these risk estimates is further strengthened by the results of the independent peer review to which the risk assessment and the primary supporting studies were submitted, which generally supported the Agency's approach and results.

Although nasal damage and asthma are well-established effects of occupational exposure to airborne Cr(VI), OSHA has preliminarily determined that there are no adequate studies to support a quantitative risk assessment for these effects. The Agency has nonetheless made careful use of the best available scientific information in its evaluation of noncancer health risks from occupational Cr(VI) exposure. In lieu of a quantitative analysis linking the risk of noncancer health effects with specific occupational exposure conditions, the Agency has considered information on the extent of these effects and occupational factors affecting risk, as discussed below.

Damage to the nasal mucosa and septum can occur from inhalation of airborne Cr(VI) or transfer of Cr(VI) on workers' hands to the interior of the nose. Epidemiological studies have found varying, but substantial, prevalence of nasal damage among workers exposed to high concentrations of airborne Cr(VI). In the cohort of 2357 chromate production workers studied by Gibb *et al.*, over 60% experienced nasal septum ulcerations at some point during their employment, with half of these workers' first ulcerations occurring within 22 days from the date they were hired (Ex. 31–22–12). The authors found a statistically significant relationship between nasal ulceration and workers' contemporaneous exposures, with about half of the workers who developed ulcerations first diagnosed with ulcerations while employed in a job with average exposure concentrations greater than 20 $\mu g/m^3$. Nasal septum perforations were reported among 17% of the Gibb cohort workers, and appeared to develop over relatively long periods of exposure (median time 172 days from hire date to diagnosis).

Another important study, Lindberg and Hedenstierna's 1983 examination of nasal effects among Swedish chrome platers, characterizes the prevalence of nasal irritation, atrophy, ulceration, and perforation among workers exposed to various concentrations of Cr(VI) (Ex. 9–126). Workers' daily average exposure concentrations were measured as 8-hour averages using personal air samplers, and estimates of workers' peak exposures were derived from 6-hour average concentrations collected with stationary equipment near the chrome electroplating baths. Among 43 workers exposed almost exclusively to Cr(VI), septum ulceration and perforation were not observed among those exposed to peak exposures less than 20 $\mu g/m^3$ or those exposed to 8-hour average concentrations less than 2 $\mu g/m^3$, a result used by the EPA to identify a lowest-observed adverse effect level (LOAEL) for their inhalation reference

concentration (Ex. 35–156). Nasal septum atrophy, a condition that can progress to ulceration and perforation, was observed less frequently among workers with 8-hour mean exposure concentrations less than 2 μg/m^3 and those with peak exposures less than 20 μg/m^3 than among workers exposed to higher concentrations. It is not clear whether workers who had nasal septum atrophy at these exposure levels eventually developed ulcerations or perforations. Although Lindberg and Hedenstierna's results suggest increasing risk of nasal septum damage with increasing exposure concentrations, there are considerable uncertainties associated with the cross-sectional study design and the possible contribution of hand-to-nose transfer of Cr(VI) to the observed nasal effects.

C. Significance of Risk and Risk Reduction

The Supreme Court's benzene decision of 1980 states that ''before he can promulgate any permanent health or safety standard, the Secretary [of Labor] 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'' (*IUD* v. *API,* 448 U.S. at 642). The Court broadly describes the range of risks OSHA might determine to be significant:

> 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 percent benzene will be fatal, a reasonable person might well consider the risk significant and take the appropriate steps to decrease or eliminate it. (*IUD* v. *API*,448 U.S. at 655).

The Court further stated, ''The requirement that a 'significant' risk be identified is not a mathematical straitjacket * * *. Although the Agency has no duty to calculate the exact probability of harm, it does have an obligation to find that a significant risk is present before it can characterize a place of employment as 'unsafe' and proceed to promulgate a regulation.'' (*IUD* v. *API*,448 U.S. at 655).

Table VIII–1 presents the estimated excess risk of lung cancer associated with various levels of Cr(VI) exposure allowed under the current rule, based on OSHA's risk assessment and assuming either 20 years' or 45 years' occupational exposure to Cr(VI) as indicated. The purpose of the OSH Act, as stated in Section 6(b), is to ensure ''that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to the hazard * * * for the period of his working life.'' 29 U.S.C. 655(b)(5). Taking a 45-year working life from age 20 to age 65, as OSHA has done in significant risk determinations for previous standards, the Agency preliminarily finds an excess lung cancer risk of approximately 100 to 350 per 1000 workers exposed at the current PEL of 52 μg/m^3 Cr(VI). This risk is clearly significant, falling well above the level of risk the Supreme Court indicated a reasonable person might consider acceptable. Even assuming only a 20-year working life, the excess risk of about 50 to 200 per 1000 workers is still clearly significant. The proposed PEL of 1 μg/m^3 Cr(VI) is expected to reduce these risks substantially, to below 10 excess lung cancers per 1000 workers. However, even at the proposed PEL, the risk posed to workers with a lifetime of regular exposure is still clearly significant.

Table VIII–1.—Expected Excess Lung Cancer Deaths Per 1000 Workers

	Cr(VI) concentratin, μg/m^3	*20-year exposure*	*45-year exposure*
Current PEL	52	43–198	101–351
	20	17–83	41–164
	10	9–43	21–86
	5.0	4.3–22	10–45
	2.5	2.1–11	5.3–23
Proposed PEL	1.0	0.85–4.4	2.1–9.1
	0.5	0.43–2.2	1.1–4.6
	0.25	0.21–1.1	0.53–2.3

Workers exposed to lower concentrations of Cr(VI) and for shorter periods of time may also have significant excess cancer risk. OSHA's estimates of risk are therefore proportional to concentration for any given exposure duration; for example, workers exposed for 20 years to 10 μg/m^3 Cr(VI) have about ten times the risk of workers exposed for 20 years to 1 μg/m^3 Cr(VI). The Agency's risk estimates are also roughly proportional to duration for any given exposure concentration, but not exactly proportional due to competing mortality effects. The estimated risk to workers exposed at any fixed concentration for 10 years is about one-half the risk to workers exposed for 20 years; the risk for five years' exposure is about one-fourth the risk for 20 years. For example, about 11 to 55 out of 1000 workers exposed at the current PEL for five years are expected to die from lung cancer as a result of their exposure. Those exposed to 5 μg/m^3 Cr(VI) for 5 years have an estimated excess risk of 1–6 lung cancer deaths per 1000 workers. It is thus not only workers exposed for many years at high levels who have significant cancer risk under the current standard; even workers exposed for shorter periods at levels below the current PEL are at substantial risk, and will benefit from implementation of the proposed PEL.

To further demonstrate significant risk, OSHA compares the risk from currently permissible Cr(VI) exposures to risks found across a broad variety of occupations. The Agency has used similar occupational risk comparisons in the significant risk determination for substance-specific standards promulgated since the benzene decision. This approach is supported by evidence in the legislative record that Congress intended the Agency to regulate unacceptably severe occupational hazards, and not ''to establish a utopia free from any hazards''(116 Cong. Rec. 37614 (1970), Leg. Hist 480), or to address risks comparable to those that exist in virtually any occupation or workplace. It is also consistent with Section 6(g) of the OSH Act, which 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.''

Fatal injury rates for most U.S. industries and occupations may be obtained from data collected by the Bureau of Labor Statistics. Table VIII–2 shows average annual fatality rates per 1000 employees for several industries between 1992 and 2001, as well as projected fatalities per 1000 employees for periods of 20 and 45 years based on these annual rates (Ex. 35–305). While it is difficult to compare aggregate fatality rates meaningfully to the risks estimated in the quantitative risk assessment for Cr(VI), which target one specific hazard (inhalation exposure to Cr(VI)) and health outcome (lung cancer), these rates provide a useful frame of reference for considering risk from Cr(VI) inhalation. For example, OSHA's best estimate of excess lung cancer deaths per 1000 workers from regular occupational exposure to Cr(VI) in the range of 2.5–5 μg/m^3 is roughly comparable to the average number of fatal injuries in high-risk occupations such as mining, assuming the same duration of employment (*see* Table VIII–1). Regular exposures at higher levels, including the current PEL of 52 μg/m^3 Cr(VI), are expected to cause substantially more deaths per 1000 workers from lung cancer than result from occupational injuries in most private industry. At the proposed PEL of 1 μg/m^3 Cr(VI) the Agency's estimate of excess lung cancer mortality falls much closer to the private industry average fatal injury rate, given the same employment time, but still exceeds the rates found in lower-risk industries such as finance and health services.

Table VIII–2.—Fatal Inuries per 1000 Employees, by Industry

	Over 1 year	*Over 20 years*	*Over 45 years*
All Private Industry	0.06	1.1	2.5
Coal Mining	0.41	8.3	18.6
Mining (General)	0.27	5.5	12.3
Construction	0.19	3.9	8.7
Manufacturing	0.04	0.8	1.8
Wholesale Trade	0.04	0.8	1.7
Retail Trade	0.03	0.6	1.4
Finance, Insurance, and Real Estate	0.02	0.3	0.7
Health Services	0.01	0.2	0.4

Because there is little available information on the incidence of occupational cancer, risk from Cr(VI) exposure cannot be compared with overall risk from other workplace carcinogens. However, OSHA's previous risk assessments provide estimates of risk from exposure to certain carcinogens. These risk assessments, like the current assessment for Cr(VI), were based on animal or human data of reasonable or high quality and used the best information then available. Table VIII–3 shows the Agency's best estimates of cancer risk from 45 years' occupational exposure to several carcinogens, as published in the preambles to final rules promulgated since the benzene decision in 1980.

Table VIII–3.—Selected OSHA Risk Estimates (Excess Cancers per 1000 Workers)

Standard	*Risk at prior PEL*	*Risk at current PEL*	*Federal Register date*
Ethylene Oxide	63–109 per 1000	1.2–2.3 per 1000	June 22, 1984.
Asbestos	64 per 1000	6.7 per 1000	June 20, 1986.
Benzene	95 per 1000	10 per 1000	September 11, 1987.
Formaldehyde	0.4–6.2 per 1000	.0056 per 1000	December 4, 1987.
Formaldehyde	* .0056 per 1000	* <.0056 per 1000	May 27, 1992.
Methylenedianiline	** 6–30 per 1000	0.8 per 1000	August 10, 1992.
Cadmium	58–157 per 1000	3–15 per 1000	September 14, 1992.
1,3-Butadiene	11.2–59.4 per 1000	1.3–8.1 per 1000	November 4, 1996.
Methylene Chloride	126 per 1000	3.6 per 1000	January 10, 1997.
Chromium VI		106–351 per 1000	October 2004

* From information in December 4, 1987 **Federal Register.**
** No prior standard; reported risk is based on estimated exposures at the time of the rulemaking.

At 106–351 excess lung cancer deaths per 1000 workers, the estimated risk from lifetime occupational exposure to Cr(VI) at the current PEL is much higher than the estimated risk from permissible exposures to other workplace carcinogens for which OSHA has performed risk assessments (Table VIII–3, ''Risk at Current PEL''). The Cr(VI) risk estimate is also higher than many risks the Agency has found to be significant in previous rules (Table VIII–3, ''Risk at Prior PEL''). The estimated risk from lifetime occupational exposure to Cr(VI) at the proposed PEL is 2.2–9.1 excess lung cancer deaths per 1000 workers, a range comparable to the risks from other carcinogenic exposures remaining under recent rules (Table VIII–3, ''Risk at Current PEL'').

Based on the results of the quantitative risk assessment, the Supreme Court's guidance on acceptable risk, comparison with rates of occupational fatality in various industries, and comparison with cancer risk estimates developed in previous rules, OSHA preliminarily finds that the risk of lung cancer posed to workers under currently permissible levels of occupational Cr(VI) exposure is significant. The proposed PEL of 1 μg/m^3 is expected to significantly reduce risks to workers in Cr(VI)-exposed occupations. OSHA additionally finds that nasal septum ulceration and

perforation can occur with significant frequency and seriousness in exposure conditions allowed by the current rule. The proposed reduction of the Cr(VI) PEL from 52 μg/m³ to 1 μg/m³ is expected to substantially reduce or eliminate workers' risk of these adverse health effects.

IX. Summary of the Preliminary Economic Analysis and Initial Regulatory Flexibility Analysis

A. Introduction

OSHA's Preliminary Economic and Initial Regulatory Flexibility Analysis (PEA) addresses issues related to the costs, benefits, technological and economic feasibility, and the economic impacts (including small business impacts) of the Agency's Occupational Exposure to Hexavalent Chromium rule. The full Preliminary Economic and Regulatory Flexibility Analysis has been placed in the docket as Ex. 35–391. The analysis also evaluates regulatory alternatives to the proposed rule. This rule is an economically significant rule under 3(f)(1) of Executive Order 12866 and has been reviewed by the Office of Information and Regulatory Affairs in the Office of Management and Budget, as required by executive order.

The purpose of this Preliminary Economic and Regulatory Flexibility Analysis is to:

- Identify the establishments and industries potentially affected by the proposed rule;
- Estimate current exposures and the technologically feasible methods of controlling these exposures;
- Estimate the benefits of the rule in terms of the reduction in lung cancer and dermatoses employers will achieve by coming into compliance with the standard;
- Evaluate the costs and economic impacts that establishments in the regulated community will incur to achieve compliance with the proposed standard;
- Assess the economic feasibility of the rule for affected industries; and
- Evaluate the principal regulatory alternatives to the proposed rule that OSHA has considered.

The Full Preliminary Economic Analysis contains the following chapters:

Chapter I. Introduction
Chapter II. Industrial Profile
Chapter III.Technological Feasibility
Chapter IV. Costs of Compliance
Chapter V. Economic Impacts
Chapter VI. Benefits and Net Benefits
Chapter VII. Regulatory Flexibility Analysis
Chapter VIII. Environmental Impacts
Chapter IX. Non Regulatory Alternatives.

These chapters are summarized in sections B to G of this Preamble summary.

B. Introduction and Industrial Profile (Chapters I and II)

The proposed standard for occupational exposure to hexavalent chromium was developed by OSHA in response to evidence that occupational exposure to Cr(VI) poses a significant risk of lung cancer, nasal septum ulcerations and perforations and dermatoses. Exposure to Cr(VI) can also lead to asthma. To protect exposed workers from these effects, OSHA has set a Permissible Exposure Limit (PEL) of 1 μg/m³ measured as an 8-hour time weighted average. OSHA has also examined alternative PELs ranging from 20 μg/m³ to 0.25 μg/m³ measured as 8-hour time weighted averages.

OSHA's proposed standards for occupational exposure to Cr(VI) are similar in format and content to other OSHA health standards promulgated under Section 6(b)(5) of the Act. In addition to setting PELS, the proposal requires employers to:

- Monitor the exposure of employees (except in shipyards and construction);
- Establish regulated areas when exposures may reasonably be expected to exceed the PEL (except in shipyards and constructions);
- Implement engineering and work practice controls to reduce employee exposures to Cr(VI);
- Provide respiratory protection to supplement engineering and work practice controls where they are not feasible, where such controls are insufficient to meet the PELS, or in emergencies;
- Provide other protective clothing and equipment as necessary for dermal protection;
- Make industrial hygiene facilities (hand washing stations) available in some situations;
- Provide medical surveillance when employees are exposed above the PEL in general industry (In the shipyard and construction sectors, medical exposure is only required for signs or symptoms of Cr(VI) related disease);
- Train workers about the hazards of Cr(VI) (including elements already required by OSHA's Hazard Communication Standard); and
- Keep records related to the standard.

The contents of the standards, and the reasons for proposing the separate standards for general industry, construction and shipyard employment, are more fully discussed the Summary and Explanation Section of this Preamble.

Chapter II of the full PEA describes the uses of Cr(VI) and the industries in which such uses occur. Employee exposures are defined in terms of ''application groups,'' i.e., groups of firms where employees are exposed to Cr(VI) when performing a particular function. This methodology is appropriate to exposure to Cr(VI) where a widely used chemical like chromium may lead to exposures in many kinds of firms in many industries, but the processes used, exposures generated, and controls needed to achieve compliance may be the same. For example, because a given type of welding produces Cr(VI) exposures that are essentially the same regardless of whether the welding occurs in a ship, on a construction site, as part of a manufacturing process, or as part of a repair process, it is appropriate to analyze such processes as a group. However, OSHA's analysis of costs and economic feasibility reflect the fact that baseline controls, ease of implementing ancillary provisions, and the economic situation of the employer may differ within different industries in an application group. One complication with the use of the application group concept is that some firms may have exposures in two or more different application groups. For example, a large transportation equipment company may engage in chromium electroplating, painting with paints that use chromium pigments, and welding of metal containing chromium.

The most common reasons to encounter occupational exposure to Cr(VI), in addition to the production and use of chromium metal and chromium metal alloys, are chromium electroplating; welding of metals containing chromium, such as stainless steel or other high chromium steels, or with chromium coatings; the production and use of Cr(VI) containing compounds, particularly Cr(VI) pigments, but also Cr(VI) catalysts, chromic acid, and the production of chromium-containing pesticides.

Some industries are seeing sharp declines in chromium use. However, many of the industries that are seeing a sharp decline have either a small number of employees or have low exposure levels (*e.g.*, Wood Working, Printing Ink Manufacturers, and Printing). In the case of lead chromate in Pigment Production, OSHA's sources indicate that there is no longer domestic output containing lead chromates. Therefore, this trend has been recognized in the PEA. Painting activities in General Industry primarily

involve the application of strontium chromate coatings to aerospace parts; these exposures are likely to continue into the foreseeable future. Similarly, removal of lead chromate in Construction and Maritime is likely to present occupational risks for many years.

In application groups where exposures are particularly significant, both in terms of workforce size and exposure levels—notably in electroplating and welding—OSHA anticipates very little decline in exposures to hexavalent chromium due to the low potential for substitution in the foreseeable future.

Table IX–1 shows the application groups analyzed in OSHA's PEA, as well as the principle industries in each application group, and for each provides the number of establishments affected, the number of employees working in those establishments, the number of entities (firms or governments) fitting SBA's small business criteria for the industry, and the number of employees in those firms. (The table shows data for both establishments, and entities–defined as firms or governments. An entity may own more than one establishment.) The table also shows the revenues of affected establishment and entities. (This table provides the latest available data at the time this analysis was produced. However, since the analysis was produced, there have been changes to some of the affected industries. OSHA will continue to incorporate more recent data as it becomes available.) As shown in the table, there are a total of 38,000 to 55,000 establishments, depending on the degree of overlap between application groups in some industries, affected by the proposed standard.

Table IX-1. Characteristics of Industries and Application Groups Affected by OSHA's Proposed Standard for Hexavalent Chromium

	Industry or Application Group	NAICS	Category	SBA Small Business Classification (Limit for revenues or employment)[A]		Affected Entities[H]		Affected Establishments[H]			
						Small Business or Government Entities	Total	< 20 Employees	≥ 20 Employees	Small Businesses	Total
1	Electroplating - General Industry	All General Industry		500 employees	[B]	2,761	3,015	1,477	2,021	2,969	3,498
		331		500 employees	[C]	98	106	56	59	90	115
		332812	Metal Coating, Engraving (Except Jewelry and Silverware), and Allied Services to Manufacturers	500 employees		722	792	119	807	838	926
		332813	Electroplating, Plating, Polishing, Anodizing, and Coloring	500 employees		1,153	1,172	770	457	1,203	1,227
		332 (Other)	Fabricated Metal Product Manufacturing	500 employees	[D]	268	328	275	252	279	527
		333	Machinery Manufacturing	500 employees	[E]	138	144	94	65	140	159
		336 (except 33661)	Transportation Equipment Manufacturing	1,000 employees	[F]	213	263	72	232	223	304
		339	Miscellaneous Manufacturing	500 employees		108	109	18	100	117	118
		Other General Industry			[G]	61	101	73	49	70	122
	Total Electroplating					2,761	3,015	1,477	2,021	2,960	3,498
2A	Welding - General Industry	31-33	Manufacturing		[H]	14,566	15,016	9,112	8,132	15,274	17,244
2B	Welding - Maritime Industry	336611	Ship Building and Repairing	1,000 employees	[I]	261	279	111	196	276	307
2C	Welding - Construction Industry	233[Z], 234[AA],235[BB]	Building, Developing, and General Contracting; Heavy Construction; Special Trade Contractors	$28.5 million	[J]	2,394	2,419	2,220	277	2,410	2,497
2D	Welding - Government	999200	State	50,000 population		0	26	0	26	0	26
		999300	Local	50,000 population		231	815	0	815	231	815
	Total Welding					17,452	18,555	11,443	9,446	18,191	20,889
3A	Painting - General Industry	31-33	Manufacturing								
		332812	Metal Coating, Engraving (Except Jewelry and Silverware), and Allied Services to Manufacturers	500 employees		82	82	43	73	116	116
		336411, 336414, 336415, 336419, 336992	Transportation Equipment Manufacturing	1,000 employees	[K]	68	73	42	72	78	114
3B	Painting - Maritime Industry	33661	Ship and Boat Building	1,000 employees	[L]	781	791	555	321	808	876
3C	Painting - Construction Industry	234[AA],235[BB]	Heavy Construction, Special Trade Contractors	$28.5 million		6,343	6,440	5,524	1,055	6,482	6,579
		234[AA]	Heavy Construction	$28.5 million						944	
		235[BB]	Special Trade Contractors	$12.0 million						5,538	
3D	Painting - Government	999200	State	50,000 population		0	26	0	26	0	26
		999300	Local	50,000 population		628	1,439	0	1,439	628	1,439
	Total Painting					7,902	8,851	6,164	2,986	14,594	9,150

Table IX-1. Characteristics of Industries and Application Groups Affected by OSHA's Proposed Standard for Hexavalent Chromium

				Affected Employees[II]		Revenues ($)[JJ]			Revenues per Entity ($)		
	Industry or Application Group	NAICS	Category	Small Business or Government Entities	Total	Small Business or Government Entities	Establishments with < 20 Employees	Total	Small Business or Government Entities	Establishments with < 20 Employees	Total
1	Electroplating - General Industry	All General Industry		25,479	33,592	$21,324,663,027	$2,456,040,049	$26,502,032,675	$7,723,529	$1,662,857	$8,790,061
		331		977	1,543	$1,937,012,146	$89,629,709	$3,043,112,348	$19,765,430	$1,600,531	$28,708,607
		332812	Metal Coating, Engraving (Except Jewelry and Silverware), and Allied Services to Manufacturers	7,759	8,817	$4,471,294,171	$74,666,885	$4,991,006,612	$6,192,928	$627,453	$6,301,776
		332813	Electroplating, Plating, Polishing, Anodizing, and Coloring	10,594	11,684	$1,710,129,336	$300,217,773	$1,770,100,489	$1,483,200	$389,893	$1,510,325
		332 (Other)	Fabricated Metal Product Manufacturing	1,859	5,433	$1,340,621,777	$297,490,267	$3,762,522,743	$5,002,320	$1,081,783	$11,471,106
		333	Machinery Manufacturing	898	1,155	$1,452,885,721	$140,996,999	$1,827,894,399	$10,528,157	$1,499,968	$12,693,711
		336 (except 33661)	Transportation Equipment Manufacturing	2,135	3,215	$26,521,611,237	$232,407,315	$39,851,510,652	$124,514,607	$3,227,879	$151,526,656
		339	Miscellaneous Manufacturing	917	1,037	$2,314,796,502	$21,214,996	$2,585,030,049	$21,433,301	$1,178,611	$23,715,872
		Other General Industry		340	708	$233,265,779	$127,461,076	$485,072,072	$3,824,029	$1,746,042	$4,802,694
	Total Electroplating			25,479	33,592	$39,981,616,668	$1,284,085,021	$58,316,249,364	$14,480,846	$869,387	$19,342,040
2A	Welding - General Industry	31-33	Manufacturing	40,779	45,326	$108,500,153,472	$8,351,328,981	$120,716,959,291	$7,448,864	$916,520	$8,039,222
2B	Welding - Maritime Industry	336611	Ship Building and Repairing	4,062	4,720	$8,599,192,787	$135,596,427	$10,071,786,544	$32,947,099	$1,221,589	$36,099,593
2C	Welding - Construction Industry	233[Z], 234[AA],235[BB]	Building, Developing, and General Contracting; Heavy Construction; Special Trade Contractors	53,837	60,450	$5,692,029,178	$2,187,657,249	$6,354,044,489	$2,377,623	$985,431	$2,626,724
2D	Welding - Government	999200	State	0	128	$0	N/A	$336,858,834,000	N/A	N/A	$12,956,109,000
		999300	Local	231	815	$847,770,000	N/A	$64,736,832,720	$3,670,000	N/A	$79,431,697
	Total Welding			98,909	111,439	$123,639,145,437	$10,674,582,657	$538,738,457,044	$7,084,526	$932,848	$29,034,678
3A	Painting - General Industry	31-33	Manufacturing								
		332812	Metal Coating, Engraving (Except Jewelry and Silverware), and Allied Services to Manufacturers	6,817	6,817	$464,316,049	$32,039,306	$464,316,049	$5,662,391	$745,100	$5,662,391
		336411, 336414, 336415, 336419, 336992	Transportation Equipment Manufacturing	670	1,297	$15,348,456,496	$264,105,530	$29,629,673,691	$225,712,596	$6,288,227	$405,885,941
3B	Painting - Maritime Industry	33661	Ship and Boat Building	2,506	3,155	$14,344,823,950	$677,982,136	$16,951,028,194	$18,367,252	$1,221,589	$21,429,871
3C	Painting - Construction Industry	234[AA],235[BB]	Heavy Construction, Special Trade Contractors	29,956	32,608	$11,616,107,029	$2,674,735,826	$12,501,994,459	$1,831,327	$484,203	$1,941,303
		234[AA]	Heavy Construction			$9,027,356,999		$9,425,998,857			
		235[BB]	Special Trade Contractors			$2,063,201,175		$2,269,442,385			
3D	Painting - Government	999200	State	0	1,535	$0	N/A	$336,858,834,000	N/A	N/A	$12,956,109,000
		999300	Local	1,878	6,613	$2,304,760,000	N/A	$91,027,413,880	$3,670,000	N/A	$63,257,411
	Total Painting			41,827	52,025	$55,169,021,699	$3,648,862,796	$499,128,701,514	$6,981,653	$591,963	$56,392,351

Table IX-1. Characteristics of Industries and Application Groups Affected by OSHA's Proposed Standard for Hexavalent Chromium

	Industry or Application Group	NAICS	Category	SBA Small Business Classification (Limit for revenues or employment)[A]	Affected Entities[B]		Affected Establishments[B]			
					Small Business or Government Entities	Total	< 20 Employees	≥ 20 Employees	Small Businesses	Total
4	Chromate (Chromite Ore Production)	325188	All Other Basic Inorganic Chemical Mfg.	1,000 employees	0	1	0	2	0	2
5	Chromate Pigment Producers	325131	Inorganic Dye and Pigment Mfg.	1,000 employees	2	3	1	2	2	3
6	Chromated Copper Arsenate Producers	325320	Pesticide and Other Agricultural Chemical Mfg.	500 employees	3	3	0	3	3	3
7	Chromium Catalyst Producers	325188	All Other Basic Inorganic Chemical Mfg.	1,000 employees	3	3	0	5	5	5
8	Paint and Coatings Producers	325510	Paint and Coating Mfg.	500 employees	165	174	132	84	180	216
9	Printing Ink Producers	325910	Printing Ink Mfg.	500 employees	6	9	10	3	9	13
10	Plastic Colorant Producers and Users	325211	Plastics Material and Resin Mfg.	500 employees	96	104	45	92	100	137
		325991	Custom Compounding of Purchased Resin	500 employees						
		3261	Plastic Product Mfg.	500 employees [M]						
11	Plating Mixture Producers	325998	All Other Miscellaneous Chemical Product and Preparation Mfg.	500 employees	10	10	4	6	10	10
12	Wood Preserving	321114	Wood Preservation	500 employees	N/A	N/A	N/A	N/A	N/A	N/A
13	Chromium Metal Producers	331112	Electrometallurgical Ferroalloy Product Mfg.	750 employees	0	1	0	1	0	1
14	Steel Mills	331111	Iron and Steel Mills	1,000 employees	48	54	17	53	49	70
15	Iron and Steel foundries	3315	Iron foundries	500 employees	278	306	144	198	289	342
		331512	Steel Investment foundries	500 employees						
		331513	Steel foundries (except investment)	500 employees						
16	Chromium Dioxide Producers	325188	All Other Inorganic Chemicals, n.e.c.	1,000 employees	N/A	N/A	N/A	N/A	N/A	N/A
17	Chromium Dye Producers	3251317	Chrome Colors and Other Inorganic Pigments	1,000 employees	3	3	1	3	4	4
18	Chromium Sulfate Producers	325188	All Other Inorganic Chemicals, n.e.c.	1,000 employees	2	3	5	0	2	5

Table IX-1. Characteristics of Industries and Application Groups Affected by OSHA's Proposed Standard for Hexavalent Chromium

	Industry or Application Group	NAICS	Category	Affected Employees[II]		Revenues ($)[JJ]			Revenues per Entity ($)		
				Small Business or Government Entities	Total	Small Business or Government Entities	Establishments with < 20 Employees	Total	Small Business or Government Entities	Establishments with < 20 Employees	Total
4	Chromate (Chromite Ore Production)	325188	All Other Basic Inorganic Chemical Mfg.	0	150	$0	$0	$114,000,000	N/A	N/A	$114,000,000
5	Chromate Pigment Producers	325131	Inorganic Dye and Pigment Mfg.	50	52	$155,766,777	$2,197,133	$157,963,909	$77,883,388	$2,197,133	$52,654,636
6	Chromated Copper Arsenate Producers	325320	Pesticide and Other Agricultural Chemical Mfg.	27	27	$78,632,194	$0	$78,632,194	$26,210,731	N/A	$26,210,731
7	Chromium Catalyst Producers	325188	All Other Basic Inorganic Chemical Mfg.	313	313	$280,072,817	$0	$280,072,817	$93,357,606	N/A	$93,357,606
8	Paint and Coatings Producers	325510	Paint and Coating Mfg.	1,779	2,569	$2,795,429,904	$440,533,212	$3,899,407,613	$16,941,999	$3,337,373	$22,410,389
9	Printing Ink Producers	325910	Printing Ink Mfg.	77	112	$95,077,283	$51,943,818	$140,018,733	$15,846,214	$5,194,382	$15,557,637
10	Plastic Colorant Producers and Users	325211 325991 3261	Plastics Material and Resin Mfg. Custom Compounding of Purchased Resin Plastic Product Mfg.	303	492	$849,804,409	$46,928,948	$1,346,164,556	$8,852,129	$1,042,866	$12,943,890
11	Plating Mixture Producers	325998	All Other Miscellaneous Chemical Product and Preparation Mfg.	49	118	$312,210,037	$17,540,363	$744,613,779	$31,221,004	$4,385,091	$74,461,378
12	Wood Preserving	321114	Wood Preservation	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
13	Chromium Metal Producers	331112	Electrometallurgical Ferroalloy Product Mfg.	0	63	$0	$0	$72,676,523	N/A	N/A	$72,676,523
14	Steel Mills	331111	Iron and Steel Mills	3,162	5,205	$8,184,955,092	$35,620,837	$13,532,955,697	$170,519,898	$2,095,343	$250,610,291
15	Iron and Steel foundries	3315 331512 331513	Iron foundries Steel Investment foundries Steel foundries (except investment)	23,461	30,222	$3,751,280,407	$104,928,176	$4,922,922,329	$13,493,814	$728,668	$16,087,981
16	Chromium Dioxide Producers	325188	All Other Inorganic Chemicals, n.e.c.	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
17	Chromium Dye Producers	3251317	Chrome Colors and Other Inorganic Pigments	104	104	$235,871,033	$2,165,058	$235,871,033	$78,623,678	$2,165,058	$78,623,678
18	Chromium Sulfate Producers	325188	All Other Inorganic Chemicals, n.e.c.	4	11	$5,798,347	$14,495,866	$14,495,866	$2,899,173	$2,899,173	$4,831,955

Table IX-1. Characteristics of Industries and Application Groups Affected by OSHA's Proposed Standard for Hexavalent Chromium

					Affected Entities[H]		Affected Establishments[H]			
Industry or Application Group		NAICS	Category	SBA Small Business Classification (Limit for revenues or employment)[A]	Small Business or Government Entities	Total	< 20 Employees	≥ 20 Employees	Small Businesses	Total
19	Chemical Distributors	42269[CC]	Other Chemical and Allied Products	100 employees	1,228	1,258	1,577	209	1,568	1,786
20	Textile Dyeing	313	Textile Mills	500 employees [N]	992	1,026	759	374	1,030	1,133
		314	Textile Product Mills	500 employees [O]						
21	Colored Glass Producers	3272123	Other Pressed and Blown Glass and Glassware Mfg.	750 employees	22	23	19	6	22	25
		3272129	Other Pressed and Blown Glass and Glassware Mfg.	750 employees						
22	Printing	32311	Printing Ink Mfg.	500 employees	490	495	400	100	493	500
		323113	Commercial Screen Printing\	500 employees						
23	Leather Tanning	3161	Leather and Hide Tanning and Finishing	500 employees [P]	N/A	N/A	N/A	N/A	N/A	N/A
24	Chromium Catalyst Users	325110	Petrochemical Mfg., Including Styrene	1,000 employees	33	71	0	163	44	163
		325120	Industrial Gas Mfg., Including Hydrogen and Ammonia Gas	1,000 employees						
		325211	Plastics Materials, Synthetic Resins, and Nonvulcanizable Elastomers, Including Polyethylene	750 employees						
		325199	Industrial Inorganic Chemicals, Not Otherwise Classified, Including Butadiene and Methanol	1000 employees						
24A	Chromium Catalyst Users - Service Companies	561790	Other Services to Buildings and Dwellings, Including Catalyst handling	$6 million	5	11	4	21	6	25
	Total Chromium Catalyst Users				38	82	4	184	50	188
25	Refractory Brick Producers	327125	Nonclay Refractory Mfg.	750 employees	1	6	0	6	1	6
26A	Wood Working -General Industry	321	General Industry	500 employees	203	219	100	187	236	287
26B	Wood Working - Maritime Industry	336611	Ship Building and Repairing	1,000 employees [Q]	48	64	37	42	52	79
26C	Wood Working - Construction Industry	2332[DD], 2333[EE], 2349[FF], 23551[GG]	Construction	$28.5 million [R]	7,217	7,285	5,960	1,489	7,304	7,449
26D	Wood Working - Government	999200	State	50,000 population	0	26	0	26	0	26
		999300	Local	50,000 population	27	94	0	94	27	94
	Total Wood Working				7,495	7,688	6,097	1,838	7,619	7,935

Table IX-1. Characteristics of Industries and Application Groups Affected by OSHA's Proposed Standard for Hexavalent Chromium

				Affected Employees[II]		Revenues ($)[JJ]			Revenues per Entity ($)		
	Industry or Application Group	NAICS	Category	Small Business or Government Entities	Total	Small Business or Government Entities	Establishments with < 20 Employees	Total	Small Business or Government Entities	Establishments with < 20 Employees	Total
19	Chemical Distributors	42269[CC]	Other Chemical and Allied Products	2,917	3,572	$4,806,227,832	$2,194,757,331	$5,870,907,660	$3,913,866	$1,391,729	$4,666,858
20	Textile Dyeing	313 314	Textile Mills Textile Product Mills	19,798	25,341	$5,005,894,785	$423,916,784	$6,386,454,435	$5,046,265	$558,520	$6,224,614
21	Colored Glass Producers	3272123 3272129	Other Pressed and Blown Glass and Glassware Mfg. Other Pressed and Blown Glass and Glassware Mfg.	154	295	$190,209,506	$17,256,484	$363,162,528	$8,645,887	$908,236	$15,789,675
22	Printing	32311 323113	Printing Ink Mfg. Commercial Screen Printing\	6,289	6,600	$384,050,231	$98,720,864	$402,525,299	$783,776	$246,802	$813,182
23	Leather Tanning	3161	Leather and Hide Tanning and Finishing	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
24	Chromium Catalyst Users	325110 325120 325211 325199	Petrochemical Mfg., Including Styrene Industrial Gas Mfg., Including Hydrogen and Ammonia Gas Plastics Materials, Synthetic Resins, and Nonvulcanizable Elastomers, Including Polyethylene Industrial Inorganic Chemicals, Not Otherwise Classified, Including Butadiene and Methanol	65	243	$4,267,606,002	N/A	$15,809,540,418	$129,321,394	N/A	$222,669,583
24A	Chromium Catalyst Users - Service Companies	561790	Other Services to Buildings and Dwellings, Including Catalyst handling	121	707	$7,307,698	$3,625,078	$42,292,582	$1,461,540	$906,270	$3,844,780
	Total Chromium Catalyst Users			186	950	$4,274,913,700	$3,625,078	$15,851,833,000	$112,497,729	$906,270	$193,315,037
25	Refractory Brick Producers	327125	Nonclay Refractory Mfg.	15	90	$49,494,145	N/A	$296,964,870	$49,494,145	N/A	$49,494,145
26A	Wood Working -General Industry	321	General Industry	320	388	$1,369,573,470	$113,191,992	$1,717,809,321	$6,746,667	$1,131,920	$7,843,878
26B	Wood Working - Maritime Industry	336611	Ship Building and Repairing	261	319	$1,844,559,942	$45,198,809	$2,174,382,406	$38,428,332	$1,221,589	$33,974,725
26C	Wood Working - Construction Industry	2332[DD], 2333[EE], 2349[FF], 23551[GG]	Construction	12,947	13,952	$52,803,055,536	$10,242,955,458	$56,935,013,853	$7,316,483	$1,718,617	$7,815,376
26D	Wood Working - Government	999200	State	0	27	$0	N/A	$336,858,834,000	N/A	N/A	$12,956,109,000
		999300	Local	27	94	$99,090,000	N/A	$7,428,828,360	$3,670,000	N/A	$79,030,089
	Total Wood Working			13,555	14,780	$56,116,278,949	$10,401,346,259	$405,114,867,940	$7,487,162	N/A	$52,694,442

Table IX-1. Characteristics of Industries and Application Groups Affected by OSHA's Proposed Standard for Hexavalent Chromium

	Industry or Application Group	NAICS	Category	SBA Small Business Classification (Limit for revenues or employment)[A]	Affected Entities[II]		Affected Establishments[II]			
					Small Business or Government Entities	Total	< 20 Employees	≥ 20 Employees	Small Businesses	Total
27	Solid Waste Incineration	562213	Solid Waste Combustors and Incinerators	$10.5 million	67	97	66	55	70	121
27A	Solid Waste Incineration - govt	999300	Local	50,000 population	0	33	0	33	0	33
	Total Incineration				67	130	66	88	70	154
28	Oil and Gas Well Drilling	213111	Drilling Oil and Gas Wells	500 employees	N/A	N/A	N/A	N/A	N/A	N/A
29	Portland Cement Producers	327310	Cement Mfg.	750 employees	141	178	130	138	156	268
30	Superalloy Producers	331492	Secondary Smelting, Refining and Alloying of Nonferrous Metal	750 employees	1	11	0	18	1	18
		331528	Other Nonferrous Foundries	500 employees						
31B	Construction - Refractory Brick Restoration and Maintenance	235[BB]	Special Trade Contractors	$12.0 million	180	182	166	18	182	184
31C	Construction - Hazardous Waste Site Work	2333[EE]	Nonresidential Building Construction	$28.5 million	201	203	161	49	204	210
31CG	Hazardous Waste Site Work - Government	999200	State	50,000 population	0	1	0	1	0	1
		999300	Local	50,000 population	64	226	0	226	64	226
31D	Construction - Industrial Rehabilitation and Maintenance	23493[HH]	Industrial Nonbuilding Structure Construction	$28.5 million	231	235	221	62	240	283
31DG	Industrial Rehabilitation and Maintenance - Government	999200	State	50,000 population	0	18	0	18	0	18
		999300	Local	50,000 population	24	83	0	83	24	83
	Total Construction				700	948	548	457	714	1,005
32	Precast Concrete Products Producers	327331, 327332, 327390	Concrete Pipe, Brick, and Block Mfg.	500 employees	2,813	2,929	2,303	1,400	3,286	3,703
	Total – All Affected Entities				32,783	34,552	23,487	14,904	34,297	38,391

Note: Total affected entities, establishments, employees, and revenues were estimated by adding entities (establishments, etc.) from each industry segment calculated by the following method:

General Industry = general industry welding entities + 1/2 (remaining general industry entities)

Maritime = maritime painting entities + 1/2 (remaining maritime entities)

Construction = construction woodworking entities + 1/2 (remaining construction entities)

Government = government painting entities

Table IX-1. Characteristics of Industries and Application Groups Affected by OSHA's Proposed Standard for Hexavalent Chromium

				Affected Employees[II]		Revenues ($)[JJ]			Revenues per Entity ($)		
	Industry or Application Group	NAICS	Category	Small Business or Government Entities	Total	Small Business or Government Entities	Establishments with < 20 Employees	Total	Small Business or Government Entities	Establishments with < 20 Employees	Total
27	Solid Waste Incineration	562213	Solid Waste Combustors and Incinerators	682	2,285	$256,063,699	$112,006,353	$1,283,887,769	$3,821,846	$1,697,066	$13,235,956
27A	Solid Waste Incineration - govt	999300	Local	0	106	$0	N/A	$3,610,169,640	N/A	N/A	$109,399,080
	Total Incineration			682	2,391	$256,063,699	$112,006,353	$4,894,057,409	$3,821,846		$37,646,595
28	Oil and Gas Well Drilling	213111	Drilling Oil and Gas Wells	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
29	Portland Cement Producers	327310	Cement Mfg.	4,844	12,636	$2,348,722,985	$339,217,713	$5,953,218,570	$16,657,610	$2,609,367	$33,445,048
30	Superalloy Producers	331492	Secondary Smelting, Refining and Alloying of Nonferrous Metal	121	2,164	$26,787,791	N/A	$482,180,241	$26,787,791	N/A	$43,834,567
		331528	Other Nonferrous Foundries								
31B	Construction - Refractory Brick Restoration and Maintenance	235[BB]	Special Trade Contractors	367	392	$175,281,510	$79,407,858	$187,265,716	$973,786	$478,361	$1,040,365
31C	Construction - Hazardous Waste Site Work	2333[EE]	Nonresidential Building Construction	1,111	1,213	$928,919,198	$196,411,140	$1,013,517,325	$4,621,489	$1,219,945	$5,042,375
31CG	Hazardous Waste Site Work - Government	999200	State	0	2	$0	N/A	$12,956,109,000	N/A	N/A	$12,956,109,000
		999300	Local	192	677	$234,880,000	N/A	$17,957,530,960	$3,670,000	N/A	$79,458,102
31D	Construction - Industrial Rehabilitation and Maintenance	23493[HH]	Industrial Nonbuilding Structure Construction	1,139	1,684	$763,222,962	$135,692,924	$1,128,350,024	$3,303,996	$613,995	$4,884,632
31DG	Industrial Rehabilitation and Maintenance - Government	999200	State	0	18	$0	N/A	$233,209,962,000	N/A	N/A	$12,956,109,000
		999300	Local	24	83	$88,080,000	N/A	$6,542,625,720	$3,670,000	N/A	$78,826,816
	Total Construction			2,833	4,066	$2,190,383,669	$411,511,922	$272,995,360,745	$3,129,120	$750,934	$290,420,597
32	Precast Concrete Products Producers	327331, 327332, 327390	Concrete Pipe, Brick, and Block Mfg.	59,825	71,220	$8,431,493,928	$20,794,317,954	$9,995,322,907	$2,997,332	$885,373	$3,412,538
	Total – All Affected Entities			306,763	380,599	$242,603,809,053	$35,196,413,601	$720,482,070,076	$7,400,406	$1,498,581	$20,854,523

Note: Total affected entities, establishments, employees, and revenues were estimated by adding entities (establishments, etc.) from each industry segment calculated by the following method:
General Industry = general industry welding entities + 1/2 (remaining general industry entities)
Maritime = maritime painting entities + 1/2 (remaining maritime entities)
Construction = construction woodworking entities + 1/2 (remaining construction entities)
Government = government painting entities

Footnotes

[A] SBA size standards taken from 13 CFR Ch.1 § 121.201. January 1, 2003

[B] Includes industries in NAICS 31-33, NAICS 42, NAICS 51.

[C] Except 311221 "Wet Corn Milling", 311312 "Cane Sugar Refining", 311313 "Beet Sugar Manufacturing", and 311821 Cookie and Cracker Manufacturing, which have an SBA size standard of 750 employees, and also 311223 "Other Oilseed Processing", 311225 "Fats and Oils Refining and Blending", 311230 "Breakfast Cereal Manufacturing", 311422 "Special Canning", which have an SBA size standard of 1,000 employees.

[D] Except 332811 "Metal Heat Treating," 332991 "Ball and Roller Bearing Manufacturing," and 332998 "Enameled Iron and Metal Sanitary Ware Manufacturing," all of which have an SBA size standard of 750 employees; 332431 "Metal Can Manufacturing," 332992 "Small Arms Ammunition Manufacturing," and 332994 "Small Arms Manufacturing," all of which have an SBA size standard of 1,000 employees; and 332993 "Ammunition (except Small Arms) Manufacturing," the SBA size standard for which is 1,500 employees.

[E] Except 333120 "Construction Machinery Manufacturing," 333415 "Air-Conditioning and Warm Air Heating Equipment," and 333924 Industrial Truck, Tractor, Trailer," all of which have an SBA size standard of 750 employees; and except 333313 Office Machinery Manufacturing," 333611 "Turbine and Turbine Generator Set Unit Manufacturing," and 333618 "Other Engine Equipment Manufacturing," all of which have an SBA size standard of 1,000 employees.

[F] Except for 336212 "Truck Trailer Manufacturing," 336214 "Travel Trailer and Camper Manufacturing," 336311 "Carburetor, Piston, Piston Ring and Valve Manufacturing," 336321 "Vehicular Lighting Equipment Manufacturing," 336360 "Motor Vehicle Seating and Interior Trim Manufacturing," 336370 "Motor Vehicle Metal Stamping," 336991 Motorcycle, Bicycle and Parts Manufacturing," and 336999 "All Other Transportation Equipment Manufacturing," all of which have an SBA size standard of 500 employees; 336312 "Gasoline Engine and Engine Parts Manufacturing," 336322 "Other Motor Vehicle Electrical and Electronic Equipment Manufacturing," 336330 "Motor Vehicle Steering and Suspension Components Manufacturing (except Spring)," 336340 "Motor Vehicle Brake System Manufacturing," 336350 "Motor Vehicle Transmission and Power Train Parts Manufacturing," 336391 Motor Vehicle Air-Conditioning Manufacturing," 336399 "All Other Motor Vehicle Parts Manufacturing, all of which have an SBA size standard of 750 employees; and 336411 "Aircraft Manufacturing," which has an SBA size standard of 1,500 employees.

[G] Includes industries in NAICS 31-33, NAICS 42, NAICS 44-45, NAICS 51, NAICS 53, NAICS 54, NAICS 56, NAICS 61, NAICS 71, and NAICS 81.

[H] Includes industries in NAICS 11, NAICS 22, NAICS 31-33, NAICS 42, NAICS 44-45, NAICS 48-49, NAICS 51, NAICS 52, NAICS 53, NAICS 54, NAICS 56, NAICS 61, NAICS 62, NAICS 71, NAICS 72, and NAICS 81.

[I] Except 336612 "Boat Building," which has an SBA size standard of 500 employees.

[J] Except 2331 "Land Subdivision and Land Development," which has an SBA size standard of $6.0 million.

[K] Except 336411 "Aircraft Manufacturing"

[L] Except 336612 "Boat Building," which has an SBA size standard of 500 employees.

[M] All of NAICS CODE 3261 have an SBA size standard of 500 employees except 326192 "Resilient Floor Covering Mfg.", the size standard for which is 750 employees.

[N] All of NAICS CODE 313 have an SBA size standard of 500 employees except 313210 "Broad Woven Fabric Mills", 313320 "Broad Woven Finishing Mills", and 313320 "Fabric Coating Mills" all of which have a size standard of 1,000 employees.

[O] All of NAICS CODE 314 have an SBA size standard of 500 employees except 314992 "Tire Cord and Tire Fabric Mill", the size standard for which is 1,000 employees.

[P] All of NAICS CODE 3161 have an SBA size standard of 500 employees except 316211 "Rubber and Plastics Footwear Mfg.", the size standard for which is 1,000 employees.

[Q] Except 336612 "Boat Building," which has an SBA size standard of 500 employees.

[R] Except 23551 which has an SBA size standard of $12 million.

[Z] 1997 NAICS Code is 233, Building, Developing, and General Contracting. 2002 NAICS Code is 236, Construction of Buildings.

[AA] 1997 NAICS Code is 234, Heavy Construction. 2002 NAICS Code is 236, Heavy and Civil Engineering Construction.

[BB] 1997 NAICS Code is 235, Special Trades Contractors. 2002 NAICS Code is 236, Special Trades Contractors.

[CC] 1997 NAICS Code is 42269, Other Chemical and Allied Products. 2002 NAICS Code is 424690, Other Chemical and Allied Products Merchant Wholesalers.

[DD] 1997 NAICS Code is 2332, Residential Building Construction. 2002 NAICS Code is 23611, Residential Building Construction.

[EE] 1997 NAICS Code is 2333, Nonresidential Building Construction. 2002 NAICS Code is 2362, Nonresidential Building Construction.

[FF] 1997 NAICS Code is 2349, Other Heavy Construction. 2002 NAICS Code is 237, Heavy and Civil Engineering Construction.

[GG] 1997 NAICS Code is 23551, Carpentry. 2002 NAICS Codes are 23835, Finish Carpentry Contractors, and 23813, Framing Contractors.

[HH] 1997 NAICS Code is 23493, Industrial Non-Building Structure Construction. 2002 NAICS Code is 23621, Industrial Building Construction.

[II] "Entities" refer to business firms or governmental bodies; "establishments" refer to industrial plants. Data on affected entities, establishments, and employees are from multiple sources; see the industrial profiles in Chapter II in the PEA (Ex. 35-391) for the complete list of references.

[JJ] Industry revenues were estimated from data reported in I.R.S., ***Corporation Source Book of Statistics of Income, 2000.*** Data on revenues for State and Local Governments were taken from U.S. Census Bureau, ***Government Finances: 1999-2000***, January 2003.

Source: U.S. Dept. of Labor, OSHA, Office of Regulatory Analysis, based on IT, 2004.

Various types of welding applications account for the greatest number of establishments and number of employees affected by the proposed standard.

Table IX–2 shows the current exposures to Cr(VI) by application group. The exposure data relied on by OSHA in developing the exposure profile and evaluating technological feasibility was compiled in a database of exposures taken from OSHA compliance officers, Site visits by OSHA contractors and the National Institute for Occupational Safety and Health (NIOSH), the U.S. Navy, published literature, and interested parties.

In all sectors OSHA has used the best available information to determine baseline exposures and technological feasibility. In a few sectors this information has been difficult to obtain and OSHA has had to rely on limited data in the industry or used analogous operations from similar processes. In these cases OSHA (or its contractor) discussed issues with industry experts and used their professional judgment to determine technological feasibility. The sectors that fall into the above categories are steel mills, welding in construction, woodworking and catalyst users.

Data obtained for steel mills included several sources such as NIOSH HHEs, IMIS exposure data and a site visit from IT Corporation, an OSHA contractor. OSHA's contractor could only obtain permission to conduct a site visit at a steel mill that used the teeming and primary rolling method versus continuous casting which is now used in approximately 95% of the steel mills. OSHA acknowledges this and uses exposures from analogous operations with additional information from industry experts. OSHA requests worker exposure information from steel mills using the continuous casting process. Exposure information was also limited for welding at construction sites. OSHA could use analogous operations from welding in maritime in open spaces. This could give a more detailed distribution for the baseline exposure profile. OSHA requests comments on the use of the Maritime data as an analogous operation for welding at construction sites.

In several sectors, such as woodworking and catalyst use, OSHA anticipates that airborne exposures will be low. In these cases exposure monitoring has been performed infrequently. OSHA then used professional judgment or has calculated exposure using total dust exposure to estimate employees' exposures to Cr(VI).

OSHA's analysis of technological feasibility analyzes employee exposures at the operation or task level to the extent that such data are available. There are a total of 380,000 workers exposed to Cr(VI), of which 84,000 are exposed above the proposed PEL of 1 microgram per cubic meter.

Table IX-2. Exposure Profile by Application Group for Cr(VI)

Application Group	Total	Number of Exposed Workers (ug/m^3)							
		Below LOD	LOD to 0.25	0.25 to 0.5	0.5 to 1.0	1.0 to 5.0	5.0 to 10.0	10.0 to 20.0	> 20.0
Electroplating	33,590	20,688	675	465	1,502	3,943	3,150	1,673	1,494
		61.6%	2.0%	1.4%	4.5%	11.7%	9.4%	5.0%	4.4%
Welding General Industry	45,326	20,271	449	0	4,541	6,107	2,683	7,572	3,703
		44.7%	1.0%	0.0%	10.0%	13.5%	5.9%	16.7%	8.2%
Welding (maritime)	4,666	2,140	566	571	556	442	99	102	190
		45.9%	12.1%	12.2%	11.9%	9.5%	2.1%	2.2%	4.1%
Welding (construction)	60,450	20,855	1,360	1,360	10,881	12,846	1,514	6,348	5,286
		34.5%	2.2%	2.2%	18.0%	21.3%	2.5%	10.5%	8.7%
Welding (government)	942	325	21	21	170	199	23	100	83
		34.5%	2.2%	2.2%	18.0%	21.1%	2.4%	10.6%	8.8%
Painting General Industry	8,143	2,421	1,266	843	1,069	1,174	430	471	469
		29.7%	15.5%	10.4%	13.1%	14.4%	5.3%	5.8%	5.8%
Painting Maritime	3,154	538	353	302	312	985	271	117	276
		17.1%	11.2%	9.6%	9.9%	31.2%	8.6%	3.7%	8.8%
painting (construction)	32,282	6,522	7,989	1,141	9,130	5,217	1,141	326	815
		20.2%	24.7%	3.5%	28.3%	16.2%	3.5%	1.0%	2.5%
Painting Government	8,063	1,630	1,996	285	2,281	1,304	285	111	171
		20.2%	24.8%	3.5%	28.3%	16.2%	3.5%	1.4%	2.1%
Chromate Production	150	1	89	24	24	12	0	0	0
		0.7%	59.3%	16.0%	16.0%	8.0%	0.0%	0.0%	0.0%
Chromate pigmen Producers	52	0	0	0	1	16	5	6	24
		0.0%	0.0%	0.0%	1.9%	30.8%	9.6%	11.5%	46.2%
Chromated Copper Arsenate (CCA) Producers	27	0	12	0	5	5	5	0	0
		0.0%	44.4%	0.0%	18.5%	18.5%	18.5%	0.0%	0.0%
Chromium Catalyst Producers	313	0	127	25	31	71	11	29	19
		0.0%	40.6%	8.0%	9.9%	22.7%	3.5%	9.3%	6.1%
Paint and Coatings Producers	2,569	400	1,443	38	38	150	0	21	479
		15.6%	56.2%	1.5%	1.5%	5.8%	0.0%	0.8%	18.6%
Printing Ink Producers	113	27	4	3	17	62	0	0	0
		23.9%	3.5%	2.7%	15.0%	54.9%	0.0%	0.0%	0.0%
Plastic Colorant Producers and users	492	37	15	15	0	250	36	64	75
		7.5%	3.0%	3.0%	0.0%	50.8%	7.3%	13.0%	15.2%
Plating Mixture Producers	118	0	16	80	0	22	0	0	0
		0.0%	13.6%	67.8%	0.0%	18.6%	0.0%	0.0%	0.0%
Chromium Material Producers	47	16	8	1	13	4	5	0	0
		34.0%	17.0%	2.1%	27.7%	8.5%	10.6%	0.0%	0.0%
Steel Mills	5,205	1,634	567	1,689	289	1,026	0	0	0
		31.4%	10.9%	32.4%	5.6%	19.7%	0.0%	0.0%	0.0%
Iron and Steel Foundries	30,252	4,214	11,875	3,481	4,578	4,495	643	322	644
		13.9%	39.3%	11.5%	15.1%	14.9%	2.1%	1.1%	2.1%
Chromium Dye Producers	104	0	0	0	0	40	6	10	48

		Number of Exposed Workers (ug/m^3)							
Application Group	Total	Below LOD	LOD to 0.25	0.25 to 0.5	0.5 to 1.0	1.0 to 5.0	5.0 to 10.0	10.0 to 20.0	> 20.0
		0.0%	0.0%	0.0%	0.0%	38.5%	5.8%	9.6%	46.2%
Chromium Sulfate Producers	11	0	8	0	0	3	0	0	0
		0.0%	72.7%	0.0%	0.0%	27.3%	0.0%	0.0%	0.0%
Chemical Distributors	3,572	3,572	0	0	0	0	0	0	0
		100.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Textile Dyeing	25,341	17,992	3,386	3,963	0	0	0	0	0
		71.0%	13.4%	15.6%	0.0%	0.0%	0.0%	0.0%	0.0%
Colored Glass Producers	295	291	2	0	0	2	0	0	0
		98.6%	0.7%	0.0%	0.0%	0.7%	0.0%	0.0%	0.0%
Printing	6,600	6,600	0	0	0	0	0	0	0
		100.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Chromium Catalyst Users	949	20	141	294	172	161	161	0	0
		2.1%	14.9%	31.0%	18.1%	17.0%	17.0%	0.0%	0.0%
Refractory Brick Producer	90	21	54	3	12	0	0	0	0
		23.3%	60.0%	3.3%	13.3%	0.0%	0.0%	0.0%	0.0%
Woodworking Construction	13,952	4,651	0	4,651	0	3,100	0	1,550	0
		33.3%	0.0%	33.3%	0.0%	22.2%	0.0%	11.1%	0.0%
Woodworking Maritime	319	239	80	0	0	0	0	0	0
		74.9%	25.1%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Woodworking General Industry	388	334	0	0	0	0	0	54	0
		86.1%	0.0%	0.0%	0.0%	0.0%	0.0%	13.9%	0.0%
Woodworking Government	121	40	0	40	0	27	0	14	0
		33.1%	0.0%	33.1%	0.0%	22.3%	0.0%	11.6%	0.0%
Solid Waste Incineration	1,544	1,069	0	289	186	0	0	0	0
		69.2%	0.0%	18.7%	12.0%	0.0%	0.0%	0.0%	0.0%
Solid Waste Incineration Government	51	29	0	13	9	0	0	0	0
		56.9%	0.0%	25.5%	17.6%	0.0%	0.0%	0.0%	0.0%
Portland Cement Producers	12,636	1,314	10,690	632	0	0	0	0	0
		10.4%	84.6%	5.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Superalloy Production	2,164	1,466	588	100	0	0	0	10	0
		67.7%	27.2%	4.6%	0.0%	0.0%	0.0%	0.5%	0.0%
Construction Other—not including welding, painting and woodworking	3,289	2,594	622	73	0	0	0	0	0
		78.9%	18.9%	2.2%	0.0%	0.0%	0.0%	0.0%	0.0%
Construction Other (government)	780	610	129	41	0	0	0	0	0
		78.2%	16.5%	5.3%	0.0%	0.0%	0.0%	0.0%	0.0%
Precast Concrete Products Producers	71,220	18,448	50,920	1,852	0	0	0	0	0
		25.9%	71.5%	2.6%	0.0%	0.0%	0.0%	0.0%	0.0%
Total	380,192	141,339	95,451	22,458	36,132	41,667	10,468	18,900	13,776
		37.2%	25.1%	5.9%	9.5%	11.0%	2.8%	5.0%	3.6%

C. Technological Feasibility

In Chapter II of OSHA's PEA, OSHA also assesses the technological feasibility of the proposed standard across a range of potential PELs in all affected industry sectors.

Many employers, and some entire application groups already have nearly all exposures below the proposed PEL. However, OSHA recognizes that some employers in some application groups may not be able to achieve the proposed PEL with engineering controls and work practices for all job categories and may need to use respirators.

In general, OSHA considered the following kinds of possible controls that could reduce employee exposures to Cr(VI): Local exhaust ventilation (LEV) which could include the maintenance or upgrade of the current LEV or installation of additional LEV; process enclosures that would isolate the worker

from the exposure; process modifications that would reduce the generation of Cr(VI) dust or fume in the work place; improved housekeeping; improved work practices; and the supplemental use of respiratory protection if engineering controls are not sufficient to meet the proposed PEL. The technologies used in this analysis are commonly known, readily available and are currently used to some extent in the affected industries and processes. OSHA's assessment of feasible controls and what PELs they can achieve is based on information collected by Shaw Environmental, Inc., consultant to OSHA, on current exposure levels and associated existing controls, on the availability of additional controls needed to reduce employee exposures and on other evidence presented in the docket.

OSHA has determined that the primary controls most likely to be effective in reducing employee exposure to Cr(VI) are LEV, process enclosure and process modification, or substitution. In some cases, firms need not improve their local exhaust systems, but instead must spend more effort insuring that the exhaust system is working according to design specification throughout the process. In other cases, employers will need to upgrade or install new LEV. This includes installing duct work, a type of hood and/or a collection system. Examples of processes that would need to improve, maintain, or install LEV include hard chrome plating and welding processes that generate large volumes of fume such as shielded metal arc welding (SMAW) and gas metal arc welding (GMAW). (LEV is defined to include portable LEV systems such as fume extraction guns (FEG).) Other sectors where new or better maintained LEV may be needed are: painting and abrasive blasting, chromate production, the production of pigments, catalyst, dyes and plastic colorants.

OSHA estimates that process enclosures will be needed for difficult to control operations such as dusty operations. These enclosures would isolate the employees from high exposure processes and reduce the need for respirators. For example, the packaging of chromic acid in small bags is totally enclosed and therefore, employees only need to enter the room during product upset or planned changes. This technology could also be applied to other packaging operations involving similar sized bags in other industries such as pigment manufacturing, catalyst production and plastic colorants. Process modifications can also be effective in reducing exposures in some industries. For example, employers can significantly reduce employee exposure through the use of automation in catalyst production, the use of fume suppressants in electroplating and significant reduction of welding fume emission, by up to 80 percent, is attainable using the pulsed arc GMAW welding process as compared to the conventional short arc GMAW process.

OSHA recognizes that there are certain instances where the supplemental use of respirators may be needed because engineering and work practices are not sufficient to reduce airborne exposures below the proposed PEL. For example, this is the case for hard chrome electroplating in some circumstances. There are many factors that are involved in the generation of Cr(VI) including the size of the part and the thickness of the coating needed. In some worst case conditions, respirators will be needed to supplement engineering controls. Welding also includes many factors that contribute to Cr(VI) exposures; these include type of welding, the base metal, the consumable, as well as the environment in which the welding is being conducted. As a result, engineering controls and work practices may not be sufficient in the most severe conditions and therefore the supplemental use of respirators will be needed. Table IX–3 shows OSHA's estimate of respirator use by industry for each of the proposed PELs.

Table IX–3 identifies sectors where respirators will be needed for some workers. Even at a PEL of 1 $\mu g/m^3$, a majority of exposed workers in the chromium catalyst user application group will need respirators, but this use is largely intermittent. As a result, workers will not need to wear respirators on a daily basis.

PELs lower than 1 $\mu g/m^3$ could not be achieved by means of engineering controls and work practices alone for some types of welding (particularly GMAW and SMAW) and in hard chromium plating. Based on this finding, OSHA has preliminarily determined that a PEL of 1 $\mu g/m^3$ is the lowest technologically feasible level.

For a complete analysis of technical feasibility please see the Preliminary Economic Analysis, Chapter III, where feasibility is reviewed for each industry/process by job category.

TABLE IX-3. Respiratory Use by Industry at Various Potential PELs after Engineering and Work Practice Controls Have Been Applied

Industry	# Employees	0.25 µg/m³	0.50 µg/m³	1 µg/m³	5 µg/m³	10 µg/m³	20 µg/m³
		Number and Percentage of Employees Using Respirators					
Electroplating	33,591	7,229	3,934	710	0	0	0
		21.5%	11.7%	2.1%	0.0%	0.0%	0.0%
Welding SS General Industry	45,326	14,921	10,471	1,688	808	148	118
Welding SS Maritime	4,666	948	298	41	0	0	0
Welding SS Construction	60,450	9,199	8,912	5,930	5,563	875	788
Welding SS Government	942	143	139	92	87	14	12
Welding SS Grand Total	111,384	25,211	19,820	7,751	6,458	1,037	918
		22.6%	17.8%	7.0%	5.8%	0.9%	0.8%
Painting General Industry	8,143	1,369	157	157	0	0	0
Painting Construction	32,282	7,875	6,369	3,324	1,012	506	253
Painting Maritime	3,154	732	558	520	199	117	69
Painting Government	8,063	1,967	1,591	830	253	126	63
Grand Total Painting	51,642	11,943	8,675	4,832	1,463	749	386
		23.1%	16.8%	9.4%	2.8%	1.5%	0.7%
Producers of Chromates	150	75	**21**	**21**	0	0	0
		50.0%	14.0%	14.0%	0.0%	0.0%	0.0%
Chromate pigment producers	52	39	**12**	**12**	0	0	0
		75.0%	23.1%	23.1%	0.0%	0.0%	0.0%
CCA Producers	27	6	0	0	0	0	0
		22.2%	0.0%	0.0%	0.0%	0.0%	0.0%
Chromium Catalyst producers	313	125	91	**82**	0	0	0
		39.9%	29.1%	26.2%	0.0%	0.0%	0.0%
Paint and Coatings producers	2,569	0	0	0	0	0	0
		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Printing Ink Producers	113	3	0	0	0	0	0
		2.7%	0.0%	0.0%	0.0%	0.0%	0.0%
Plastic Colorant	492	0	0	0	0	0	0
		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Plating Mixtures	118	0	0	0	0	0	0
		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

Ferrochromium	47	5	0	0	0	0	0
		10.6%	0.0%	0.0%	0.0%	0.0%	0.0%
Steel mills	5,205	0	0	0	0	0	0
		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Iron and Steel Foundries	30,252	2,574	0	0	0	0	0
		8.5%	0.0%	0.0%	0.0%	0.0%	0.0%
Chromium Dye Producers	104	**10**	**10**	**10**	0	0	0
		9.6%	**9.6%**	**9.6%**	0.0%	0.0%	0.0%
Chromium sulfate producers	11	0	0	0	0	0	0
		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Chemical Distributors	3,572	0	0	0	0	0	0
		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Textile Dyeing	25,341	0	0	0	0	0	0
		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Producers of Colored Glass	295	0	0	0	0	0	0
		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Printing	6,600	0	0	0	0	0	0
		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Chromium Catalyst Users	949	**705**	**705**	**705**	0	0	0
		74.3%	74.3%	74.3%	0.0%	0.0%	0.0%
Producers of refractory bricks	90	0	0	0	0	0	0
		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Wood Working	14,780	0	0	0	0	0	0
		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Solid Waste Incinerations	1,595	0	0	0	0	0	0
		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Portland cement producers	12,636	0	0	0	0	0	0
		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Non-ferrous metallurgical uses of chromium	2,164	39	39	0	0	0	0
		1.8%	1.8%	0.0%	0.0%	0.0%	0.0%
Construction Other	4,069	90	0	0	0	0	0
		2.2%	0.0%	0.0%	0.0%	0.0%	0.0%
Precast Cast Concrete	71,220	0	0	0	0	0	0
		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
All Industries	380,192	48,058	33,309	14,125	7,921	1,786	1,304
		12.7%	8.8%	3.7%	2.1%	0.5%	0.3%

Bold numbers indicate intermittent use

Construction other - Welding, painting and woodworking not included

D. Costs

The costs employers are expected to incur to comply with the proposed standard are $223 million per year. In addition, OSHA estimates that employers will incur $67 million per year to comply with the personal protective equipment and hygiene requirements already present in existing generic standards. The proposed requirements to provide protective clothing and equipment and hygiene

areas are closely aligned with the requirements of OSHA's current generic PPE and Sanitation standards (*e.g.* 1910.132 and 1926.95 for PPE and 1910.142 and 1926.51 for the hygiene requirements). Therefore, OSHA estimates that the marginal cost of complying with the new PPE and sanitation requirements of the Cr(VI) standard were lower for firms currently subject to and in compliance with existing generic standards. OSHA's research on these current standards, however, uncovered some noncompliance. The baseline chosen for the Cr(VI) regulatory impact analysis reflects this non-compliance with current requirements. Although OSHA estimates that employers would need to spend an additional $67 million per year to bring themselves into compliance with the personal protective equipment and hygiene requirements already prescribed in existing generic standards, this additional expenditure is not attributable to the Cr(VI) rulemaking. However, by incurring the obligation and expense of providing PPE to their employees, employers are essentially transferring a benefit to employees $24 million per year.

All costs are measured in 2003 dollars. Any one-time costs are annualized over a ten year period, and all costs are annualized at a discount rate of 7 percent. (A sensitivity analysis using a discount rate of 3 percent is presented in the discussion of net benefits.) The derivation of these costs is presented in Chapter III of the full PEA. Table IX–4 provides the annualized costs by provision and by industry. Engineering control costs represent 45 percent of the costs of the new provisions of the proposed standard, and respiratory protection costs represent 19 percent of the costs of the new provisions of the proposed standard.

Table IX-4. Annualized Costs for All Establishments Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group and Regulatory Requirement for a PEL of 1 ug/m^3)

	Application Group	Engineering Controls	Initial Exposure Monitoring	Periodic Exposure Monitoring	Respiratory Protection
1	Electroplating	$38,179,276	$536,969	$3,238,675	$2,189,604
2A	Welding (general industry)	$31,230,424	$3,729,347	$19,082,460	$16,277,836
2B	Welding (maritime industry)	$1,294,354	$41,001	$0	$392,984
2C	Welding (construction industry)	$16,408,707	$107,472	$0	$9,897,057
2D	Welding (government)	$253,727	$35,616	$0	$157,812
3A	Painting (general industry)	$827,520	$100,567	$406,599	$2,184,738
3B	Painting (maritime industry)	$339,058	$33,949	$0	$6,992,874
3C	Painting (construction industry)	$0	$30,351	$0	$0
3D	Painting (government)	$0	$6,480	$0	$0
4	Chromate (chromite ore) production	$309,000	$2,585	$3,087	$13,937
5	Chromate Pigment Producers	$47,400	$4,288	$17,495	$39,774
6	Chromated Copper Arsenate (CCA) Producers	$0	$3,502	$14,065	$2,680
7	Chromium Catalyst Producers	$2,272,600	$13,232	$71,440	$587,133
8	Paint and Coatings Producers	$4,224,524	$99,510	$128,901	$32,797
9	Printing Ink Producers	$0	$10,909	$7,890	$198,295
10	Plastic Colorant Producers and Users	$0	$230,301	$1,143,725	$327,473
11	Plating Mixture Producers	$144,780	$7,905	$28,902	$0
12	Wood Preserving	$0	$0	$0	$0
13	Chromium Material Producers	$23,500	$5,470	$9,177	$10,197
14	Steel Mills	$455,071	$48,299	$35,763	$165,268
15	Iron and Steel Foundries	$1,984,734	$432,919	$863,111	$2,270,528
16	Chromium Dioxide Producers	$0	$0	$0	$0
17	Chromium Dye Producers	$0	$30,966	$153,686	$63,217
18	Chromium Sulfate Producers	$0	$5,297	$18,525	$0
19	Chemical Distributors	$0	$502,670	$0	$0
20	Textile Dyeing	$0	$439,585	$0	$0
21	Colored Glass Producers	$1,337	$18,619	$0	$0
22	Printing	$0	$157,113	$0	$0
23	Leather Tanning	$0	$0	$0	$0
24	Chromium Catalyst Users	$0	$88,754	$178,042	$566
24A	Chromium Catalyst Users (Service)	$0	$28,584	$136,534	$0
25	Refractory Brick Producers	$0	$17,189	$16,295	$5,529
26A	Woodworking (general industry)	$43,050	$75,375	$0	$0
26B	Woodworking (maritime industry)	$0	$9,742	$0	$0
26C	Woodworking (construction industry)	$2,703,987	$918,618	$0	$0
26D	Woodworking (government)	$43,560	$14,799	$0	$0
27	Solid Waste Incineration	$0	$258,213	$532,755	$246,691
27A	Incinerators (government)	$0	$16,337	$37,392	$11,005
28	Oil and Gas Well Drilling	$0	$0	$0	$0
29	Portland Cement Producers	$0	$95,191	$0	$0
30	Superalloy Producers	$12,000	$13,770	$9,177	$15,490
31B	Construction (Refractory Repair)	$0	$0	$0	$0
31C	Construction (Hazardous Waste Work)	$0	$23,606	$0	$0
31CG	Haz. Waste (government)	$0	$25,517	$0	$0
31D	Construction (Industrial Rehabilitation)	$0	$0	$0	$0
31DG	Industrial Rehab. (government)	$490	$16,617	$0	$0
32	Precast Concrete Products Producers	$0	$3,706,667	$0	$0
	General Industry (including Government)	$80,052,993	$10,779,163	$26,133,697	$24,800,570
	Construction	$19,112,694	$1,080,047	$0	$9,897,057
	Maritime	$1,633,412	$84,692	$0	$7,385,858
	Total	$100,799,100	$11,943,903	$26,133,697	$42,083,485

Table IX-4. Annualized Costs for All Establishments Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group and Regulatory Requirement for a PEL of 1 ug/m^3)

	Application Group	Housekeeping	Medical Surveillance	Information and Training	Recordkeeping
1	Electroplating	$9,189,100	$459,403	$500,074	$132,200
2A	Welding (general industry)	$0	$1,175,453	$1,845,145	$96,600
2B	Welding (maritime industry)	$0	$814	$75,239	$9,900
2C	Welding (construction industry)	$0	$11,633	$1,622,152	$157,600
2D	Welding (government)	$0	$197	$80,631	$10,800
3A	Painting (general industry)	$664,000	$111,462	$416,016	$40,100
3B	Painting (maritime industry)	$0	$269	$215,172	$13,400
3C	Painting (construction industry)	$0	$3,812	$2,047,572	$154,500
3D	Painting (government)	$0	$996	$824,967	$43,100
4	Chromate (chromite ore) production	$6,400	$418	$2,734	$900
5	Chromate Pigment Producers	$3,150	$2,584	$989	$300
6	Chromated Copper Arsenate (CCA) Producers	$0	$319	$460	$130
7	Chromium Catalyst Producers	$16,000	$17,866	$5,842	$1,820
8	Paint and Coatings Producers	$231,160	$20,105	$39,535	$11,120
9	Printing Ink Producers	$16,430	$0	$1,448	$1,070
10	Plastic Colorant Producers and Users	$21,320	$0	$13,958	$2,860
11	Plating Mixture Producers	$54,570	$686	$1,829	$510
12	Wood Preserving	$0	$0	$0	$0
13	Chromium Material Producers	$4,190	$452	$827	$270
14	Steel Mills	$224,500	$36,204	$63,150	$20,700
15	Iron and Steel Foundries	$720,800	$186,849	$421,191	$186,700
16	Chromium Dioxide Producers	$0	$0	$0	$0
17	Chromium Dye Producers	$5,290	$0	$2,056	$580
18	Chromium Sulfate Producers	$10,100	$457	$291	$100
19	Chemical Distributors	$4,859,700	$4	$34,858	$0
20	Textile Dyeing	$712,800	$81	$276,803	$76,300
21	Colored Glass Producers	$18,500	$91	$1,099	$200
22	Printing	$52,600	$0	$70,307	$18,700
23	Leather Tanning	$0	$0	$0	$0
24	Chromium Catalyst Users	$466,300	$2,652	$6,593	$1,080
24A	Chromium Catalyst Users (Service)	$71,510	$47,942	$10,593	$3,350
25	Refractory Brick Producers	$40,620	$12	$937	$300
26A	Woodworking (general industry)	$814,900	$1,580	$6,315	$500
26B	Woodworking (maritime industry)	$0	$95	$2,292	$400
26C	Woodworking (construction industry)	$0	$3,745	$320,994	$44,900
26D	Woodworking (government)	$0	$32	$3,736	$400
27	Solid Waste Incineration	$0	$145	$22,923	$4,820
27A	Incinerators (government)	$0	$10	$1,150	$140
28	Oil and Gas Well Drilling	$0	$0	$0	$0
29	Portland Cement Producers	$504,400	$650	$130,586	$40,300
30	Superalloy Producers	$16,580	$453	$9,325	$2,940
31B	Construction (Refractory Repair)	$0	$42	$14,028	$1,890
31C	Construction (Hazardous Waste Work)	$0	$131	$34,747	$5,620
31CG	Haz. Waste (government)	$0	$74	$22,405	$3,270
31D	Construction (Industrial Rehabilitation)	$0	$182	$50,939	$8,220
31DG	Industrial Rehab. (government)	$0	$11	$4,740	$490
32	Precast Concrete Products Producers	$9,593,400	$3,877	$870,527	$268,600
	General Industry (including Government)	$28,318,320	$2,071,066	$5,694,042	$971,250
	Construction	$0	$19,546	$4,090,431	$372,730
	Maritime	$0	$1,178	$292,704	$23,700
	Total	$28,318,320	$2,091,791	$10,077,177	$1,367,680

Table IX-4. Annualized Costs for All Establishments Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group and Regulatory Requirement for a PEL of 1 ug/m^3)

	Application Group	Total for Incremental Requirements	Current Requirements for PPE and Hygiene Areas			Total for Incremental and Current Requirements
			PPE (not supplied in baseline)	PPE (supplied in baseline)	Hygiene Areas	
1	Electroplating	$54,425,302	$0	$12,163,429	$1,688,800	$68,277,530
2A	Welding (general industry)	$73,437,266	$0	$0	$0	$73,437,266
2B	Welding (maritime industry)	$1,814,292	$0	$0	$0	$1,814,292
2C	Welding (construction industry)	$28,204,622	$0	$0	$0	$28,204,622
2D	Welding (government)	$538,783	$0	$0	$0	$538,783
3A	Painting (general industry)	$4,751,003	$10,872,247	$2,338,343	$348,400	$18,309,992
3B	Painting (maritime industry)	$7,594,722	$5,661,140	$921,241	$407,800	$14,584,903
3C	Painting (construction industry)	$2,236,235	$0	$944,546	$0	$3,180,780
3D	Painting (government)	$875,543	$0	$263,107	$0	$1,138,650
4	Chromate (chromite ore) production	$339,062	$0	$17,909	$4,400	$361,371
5	Chromate Pigment Producers	$115,980	$0	$6,089	$3,000	$125,069
6	Chromated Copper Arsenate (CCA) Producers	$21,156	$12,587	$2,086	$1,200	$37,028
7	Chromium Catalyst Producers	$2,985,933	$110,290	$26,303	$12,700	$3,135,226
8	Paint and Coatings Producers	$4,787,651	$3,777,438	$602,900	$142,300	$9,310,290
9	Printing Ink Producers	$236,043	$6,435	$851	$6,200	$249,529
10	Plastic Colorant Producers and Users	$1,739,637	$31,030	$5,180	$33,600	$1,809,448
11	Plating Mixture Producers	$239,182	$0	$100,396	$9,400	$348,978
12	Wood Preserving	$0	$0	$0	$0	$0
13	Chromium Material Producers	$54,083	$0	$0	$0	$54,083
14	Steel Mills	$1,048,954	$0	$0	$0	$1,048,954
15	Iron and Steel Foundries	$7,066,833	$0	$0	$0	$7,066,833
16	Chromium Dioxide Producers	$0	$0	$0	$0	$0
17	Chromium Dye Producers	$255,794	$21,250	$4,643	$5,800	$287,488
18	Chromium Sulfate Producers	$34,770	$18,113	$965	$2,800	$56,647
19	Chemical Distributors	$5,397,232	$0	$0	$0	$5,397,232
20	Textile Dyeing	$1,505,570	$1,236,379	$226,048	$1,383,800	$4,351,797
21	Colored Glass Producers	$39,846	$0	$0	$0	$39,846
22	Printing	$298,720	$373,708	$60,835	$171,700	$904,962
23	Leather Tanning	$0	$0	$0	$0	$0
24	Chromium Catalyst Users	$743,988	$143,158	$27,090	$39,200	$953,436
24A	Chromium Catalyst Users (Service)	$298,513	$0	$82,380	$33,900	$414,793
25	Refractory Brick Producers	$80,882	$29,900	$5,262	$5,300	$121,343
26A	Woodworking (general industry)	$941,720	$0	$0	$0	$941,720
26B	Woodworking (maritime industry)	$12,530	$0	$0	$0	$12,530
26C	Woodworking (construction industry)	$3,992,244	$4,848,041	$578,853	$2,858,900	$12,278,038
26D	Woodworking (government)	$62,527	$48,096	$20,338	$27,600	$158,561
27	Solid Waste Incineration	$1,065,547	$0	$613,804	$80,200	$1,759,552
27A	Incinerators (government)	$66,035	$0	$46,816	$19,700	$132,550
28	Oil and Gas Well Drilling	$0	$0	$0	$0	$0
29	Portland Cement Producers	$771,127	$1,051,893	$202,073	$213,800	$2,238,893
30	Superalloy Producers	$79,735	$0	$0	$0	$79,735
31B	Construction (Refractory Repair)	$15,961	$0	$0	$0	$15,961
31C	Construction (Hazardous Waste Work)	$64,105	$90,563	$262,183	$107,500	$524,350
31CG	Haz. Waste (government)	$51,266	$0	$165,417	$60,900	$277,582
31D	Construction (Industrial Rehabilitation)	$59,341	$0	$0	$0	$59,341
31DG	Industrial Rehab. (government)	$22,348	$0	$0	$0	$22,348
32	Precast Concrete Products Producers	$14,443,071	$25,688,840	$4,450,356	$4,859,400	$49,441,666
	General Industry (including Government)	$178,821,101	$43,421,364	$21,432,619	$9,154,100	$252,829,184
	Construction	$34,572,507	$4,938,603	$1,785,581	$2,966,400	$44,263,091
	Maritime	$9,421,545	$5,661,140	$921,241	$407,800	$16,411,725
	Total	$222,815,153	$54,021,107	$24,139,441	$12,528,300	$313,504,001

Source: U.S. Dept. of Labor, OSHA, Office of Regulatory Analysis, based on IT, 2004.

Costs for the new provisions for General Industry are $179 million per year, costs for constructions $35 million per year, and costs for the shipyard sector and $9 million per year. (In developing the costs for construction, OSHA assumed that all work by construction firms would be covered by the construction standard. However, in practice some work by construction firms takes the form of maintenance operations that would be covered by the

general industry standard. OSHA seeks comment on the extent to which welding, painting, and wood working done by construction firms might be covered by the general industry standard.) Table IX–4 also shows the costs by application group. The various types of welding represent the most expensive application group, accounting for 47 percent of the total costs.

OSHA also presents the distribution of compliance costs according at the time they are imposed in Table IX–5. Because firms will have the choice of whether to finance expenditures in order to spread out, for example, startup costs over several years, OSHA considers it unlikely that a firm would be impacted in an amount equal to the entire startup cost in the year that the initial requirements are imposed. On the other hand, capital markets are not perfectly liquid and particular firms may face additional lending constraints, therefore OSHA believes that identifying startup costs and the time distribution of imposed costs, in addition to the annualized costs, is relevant when exploring the question of economic feasibility and the overall impact of this rulemaking.

E. Economic Impacts

To determine whether the proposed rule's projected costs of compliance would raise issues of economic feasibility for employers in affected industries, i.e., would adversely alter the competitive structure of the industry,

Table IX-5. Estimated Total First-Year Compliance Costs Associated with the Proposed Standard for Hexavalent Chromium

Cost Category	General Industry	Government	Construction	Maritime	Total
Engineering Controls	$242,133,012	$1,559,708	$100,270,508	$10,157,887	$354,121,115
Initial Exposure Assessment	$149,391,244	$575,213	$7,585,801	$571,473	$158,123,732
Respiratory Protection	$28,642,044	$71,534	$11,104,439	$7,673,020	$47,491,038
Housekeeping	$44,183,186	$0	$0	$0	$44,183,186
Medical Surveillance	$8,871,106	$1,315	$21,199	$1,466	$8,895,086
Training and Familiarization	$15,372,902	$1,607,142	$8,555,306	$650,203	$26,185,553
Recordkeeping	$1,319,313	$56,382	$405,565	$28,194	$1,809,454
Total for Incremental Requirements	**$489,912,807**	**$3,871,296**	**$127,942,818**	**$19,082,242**	**$640,809,163**
PPE (supplied in baseline)	$24,698,382	$495,677	$1,785,581	$921,241	$27,900,881
PPE (not supplied in baseline)	$58,754,760	$48,096	$4,938,603	$5,661,140	$69,402,600
Hygiene Areas	$35,227,714	$335,882	$7,223,809	$1,432,863	$44,220,268
Total for Incremental and Current Requirements	**$608,593,662**	**$4,750,951**	**$141,890,812**	**$27,097,487**	**$782,332,912**

Source: U.S. Dept. of Labor, OSHA, Office of Regulatory Analysis, based on IT, 2004.

OSHA developed quantitative estimates of the economic impact of the proposed rule on the affected establishments. In this analysis, compliance costs are compared with industry revenues and profits.

To assess the potential economic impacts of the proposed standard, OSHA compared the anticipated costs of achieving compliance against revenues and profits of entities affected by the rule. OSHA compared the baseline financial data (from Table IX–1) with total annualized costs of compliance by computing compliance costs as a percentage of revenues. This impact assessment is presented in Table IX–6. This table is considered a screening analysis because it measures costs as a percentage of pre-tax profits and revenues but does not predict impacts on pre-tax profits and sales.

Table IX-6. Economic Impacts on All Entities Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group for a PEL of 1 ug/m^3)

	Application Group	NAICS	Category	Cost per Entity[L]			Impacts for Incremental-Requirement Costs	
				Incremental-Requirement Costs	Revenue per Entity[M]	Profit per Entity[M]	Cost/Revenue Impact	Cost/Profit Impact
1	Electroplating - General Industry	All General Industry		$19,040	$8,790,061	$337,538	0.22%	5.64%
		331		$16,825	$28,708,607	$854,339	0.06%	1.97%
		332812	Metal Coating, Engraving (Except Jewerly and Silverware), and Allied Services to Manufacturers	$24,276	$6,301,776	$399,369	0.39%	6.08%
		332813	Electroplating, Plating, Polishing, Anodizing, and Coloring	$14,058	$1,510,325	$95,715	0.93%	14.69%
		Other 332	Fabricated Metal Product Manufacturing	$24,095	$11,471,106	$698,751	0.21%	3.45%
		333	Machinery Manufacturing	$15,278	$12,693,711	$638,709	0.12%	2.39%
		336 (except 33661)	Transportation Equipment Manufacturing	$22,164	$151,526,656	$4,508,524	0.01%	0.49%
		339	Miscellaneous Manufacturing	$22,096	$23,715,872	$1,687,930	0.09%	1.31%
		Other General Industry[A]		$16,759	$4,802,694	$179,025	0.35%	9.36%
2A	Welding - General Industry	31-33[B]	Manufacturing	$4,891	$8,039,222	$368,301	0.06%	1.33%
2B	Welding - Maritime Industry	336611	Ship Building and Repairing	$6,503	$36,099,593	$1,678,090	0.02%	0.39%
2C	Welding - Construction Industry	233[C]	Building, Developing, and General Contracting; Heavy Construction; Special Trade Contractors	$11,660	$2,626,724	$109,075	0.44%	10.69%
2D	Welding - Government	999200	State	$2,537	$12,956,109,000	N/A	0.00%	N/A
		999300	Local	$580	$109,399,080	N/A	0.00%	N/A
3A	Painting - General Industry	31-33	Manufacturing					
		332812	Metal Coating, Engraving (Except Jewerly and Silverware), and Allied Services to Manufacturers	$29,185	$5,662,391	$358,848	0.52%	8.13%
		336411, 336414, 336415, 336419, 336992	Transportation Equipment Manufacturing	$32,299	$405,885,941	$13,192,511	0.01%	0.24%
3B	Painting - Maritime Industry	33661	Ship and Boat Building	$9,601	$21,429,871	$996,168	0.04%	0.96%
3C	Painting - Construction Industry	234[D], 235[E]	Heavy Construction, Special Trade Contractors	$347	$1,941,303	$47,804	0.02%	0.73%
		234[D], 235[E]	Heavy Construction	$711	$9,995,757	$355,849	0.01%	0.20%

Table IX-6. Economic Impacts on All Entities Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group for a PEL of 1 ug/m^3)

	Application Group	NAICS	Category	Cost per Entity[L]			Impacts for Incremental-Requirement Costs	
				Incremental-Requirement Costs	Revenue per Entity[M]	Profit per Entity[M]	Cost/Revenue Impact	Cost/Profit Impact
		235[E]	Special Trade Contractors	$285	$412,851	$17,933	0.07%	1.59%
3D	Painting - Government	999200	State	$3,995	$12,956,109,000	N/A	0.00%	N/A
		999300	Local	$536	$86,827,088	N/A	0.00%	N/A
4	Chromate (Chromite Ore Production)	325188	All Other Basic Inorganic Chemical Mfg.	$339,062	$114,000,000	$6,498,000	0.30%	5.22%
5	Chromate Pigment Producers	325131	Inorganic Dye and Pigment Mfg.	$38,660	$52,654,636	$2,121,982	0.07%	1.82%
6	Chromated Copper Arsenate Producers	325320	Pesticide and Other Agricultural Chemical Mfg.	$7,052	$26,210,731	$1,480,199	0.03%	0.48%
7	Chromium Catalyst Producers	325188	All Other Basic Inorganic Chemical Mfg.	$995,311	$93,357,606	$3,762,312	1.07%	26.45%
8	Paint and Coatings Producers	325510	Paint and Coating Mfg.	$27,515	$22,410,389	$1,063,776	0.12%	2.59%
9	Printing Ink Producers	325910	Printing Ink Mfg.	$26,227	$15,557,637	$786,018	0.17%	3.34%
10	Plastic Colorant Producers and Users	325211	Plastics Material and Resin Mfg.	$16,727	$12,943,890	$662,598	0.13%	2.52%
		325991	Custom Compounding of Purchased Resin					
		3261	Plastic Product Mfg.					
11	Plating mixture Producers	325998	All Other Miscellaneous Chemical Product and Preparation Mfg.	$23,918	$31,221,004	$1,577,379	0.08%	1.52%
12	Wood Preserving	321114	Wood Preservation	N/A	N/A	N/A	N/A	N/A
13	Chromium Metal Producers	331112	Electrometallurgical Ferroalloy Product Mfg.	$54,083	$72,676,523	$1,435,797	0.07%	3.77%
14	Steel Mills	331111	Iron and Steel Mills	$19,425	$250,610,291	$4,951,057	0.01%	0.39%
15	Iron and Steel foundries	3315	Iron foundries	$23,094	$16,087,981	$782,085	0.14%	2.95%
		331512	Steel Investment foundries					
		331513	Steel foundries (except investment)					
16	Chromium Dioxide Producers	325188	All Other Inorganic Chemicals, n.e.c.	N/A	N/A	N/A	N/A	N/A
17	Chromium Dye Producers	3251317	Chrome Colors and Other Inorganic Pigments	$85,265	$78,623,678	$3,168,534	0.11%	2.69%
18	Chromium Sulfate Producers	325188	All Other Inorganic Chemicals, n.e.c.	$11,590	$4,831,955	$194,728	0.24%	5.95%
19	Chemical Distributors	42269[F]	Other Chemical and Allied Products	$4,290	$4,666,858	$195,107	0.09%	2.20%
20	Textile Dyeing	313	Textile Mills	$1,467	$6,224,614	$195,272	0.02%	0.75%
		314	Textile Product Mills					
21	Colored Glass Producers	3272123	Other Pressed and Blown Glass and Glassware Mfg.	$1,732	$15,789,675	$497,154	0.01%	0.35%
		3272129	Other Pressed and Blown Glass and Glassware Mfg.					
22	Printing	32311	Printing	$603	$813,182	$34,683	0.07%	1.74%

Table IX-6. Economic Impacts on All Entities Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group for a PEL of 1 ug/m^3)

	Application Group	NAICS	Category	Cost per Entity[L]			Impacts for Incremental-Requirement Costs	
				Incremental-Requirement Costs	Revenue per Entity[M]	Profit per Entity[M]	Cost/Revenue Impact	Cost/Profit Impact
		323113	Commercial Screen Printing					
23	Leather Tanning	3161	Leather and Hide Tanning and Finishing	N/A	N/A	N/A	N/A	N/A
24	Chromium Catalyst Users	325110	Petrochemical Mfg., Including Styrene	$10,479	$222,669,583	$9,959,565	0.00%	0.11%
24A	Chromium Catalyst Users - Service Companies	561790	Other Services to Buildings and Dwellings, Including Catalyst handling	$27,138	$3,844,780	$147,632	0.71%	18.38%
25	Refractory Brick Producers	327125	Nonclay Refractory Mfg.	$13,480	$49,494,145	$2,092,266	0.03%	0.64%
26A	Wood Working -General Industry	321	General Industry	$4,300	$7,843,878	$243,160	0.05%	1.77%
26B	Wood Working - Maritime Industry	336611	Ship Building and Repairing	$196	$33,974,725	$1,579,825	0.00%	0.01%
26C	Wood Working - Construction Industry	2332[G], 2333[H], 2349[I], 23551[J]	Construction	$548	$7,815,376	$324,338	0.01%	0.17%
26D	Wood Working - Government	999200	State	$522	$12,956,109,000	N/A	0.00%	0.00%
		999300	Local	$521	$108,838,617	N/A	0.00%	0.00%
27	Solid Waste Incineration	562213	Solid Waste Combustors and Incinerators	$10,985	$13,235,956	$424,411	0.08%	2.59%
27A	Solid Waste Incineration - govt	999300	Local Governments	$2,001	$151,220,000	N/A	0.00%	0.00%
28	Oil and Gas Well Drilling	213111	Drilling Oil and Gas Wells	N/A	N/A	N/A	N/A	N/A
29	Portland Cement Producers	327310	Cement Mfg.	$4,332	$33,445,048	$2,431,957	0.01%	0.18%
30	Superalloy Producers	331492	Secondary Smelting, Refining and Alloying of Nonferrous Metal	$7,249	$43,834,567	$1,304,473	0.02%	0.56%
		331528	Other Nonferrous Foundries					
31B	Construction - Refractory Brick Restoration and Maintenance	235[E]	Special Trade Contractors	$87	$1,040,365	$45,190	0.01%	0.19%
31C	Construction - Hazardous Waste Site Work	2333[H]	Nonresidential Building Construction	$319	$5,042,375	$212,284	0.01%	0.15%
31CG	Hazardous Waste Site Work - Government	999200	State	$196	$12,956,109,000	N/A	0.00%	0.00%
		999300	Local	$226	$109,435,929	N/A	0.00%	0.00%
31D	Construction - Industrial Rehabilitation and Maintenance	23493[K]	Industrial Nonbuilding Structure Construction	$210	$4,884,632	$202,024	0.00%	0.10%
31DG	Industrial Rehabilitation and Maintenance - Government	999200	State	$217	$12,956,109,000	N/A	0.00%	0.00%
		999300	Local	$216	$108,554,940	N/A	0.00%	0.00%
32	Precast Concrete Products Producers	327331, 327332, 327390	Concrete Pipe, Brick, and Block Mfg.	$4,931	$3,412,538	$248,143	0.14%	1.99%

Footnotes

[A] Includes industries in NAICS 31-33, NAICS 42, NAICS 44-45, NAICS 51, NAICS 53, NAICS 54, NAICS 56, NAICS 61, NAICS 71, and NAICS 81.

[B] Includes industries in NAICS 11, NAICS 22, NAICS 31-33, NAICS 42, NAICS 44-45, NAICS 48-49, NAICS 51, NAICS 52, NAICS 53, NAICS 54, NAICS 56, NAICS 61, NAICS 62, NAICS 71, NAICS 72, and NAICS 81.

[C] 1997 NAICS Code is 233, Building, Developing, and General Contracting. 2002 NAICS Code is 236, Construction of Buildings.

[D] 1997 NAICS Code is 234, Heavy Construction. 2002 NAICS Code is 236, Heavy and Civil Engineering Construction.

[E] 1997 NAICS Code is 235, Special Trades Contractors. 2002 NAICS Code is 236, Special Trades Contractors.

[F] 1997 NAICS Code is 42269, Other Chemical and Allied Products. 2002 NAICS Code is 424690, Other Chemical and Allied Products Merchant Wholesalers.

[G] 1997 NAICS Code is 2332, Residential Building Construction. 2002 NAICS Code is 23611, Residential Building Construction.

[H] 1997 NAICS Code is 2333, Nonresidential Building Construction. 2002 NAICS Code is 2362, Nonresidential Building Construction.

[I] 1997 NAICS Code is 2349, Other Heavy Construction. 2002 NAICS Code is 237, Heavy and Civil Engineering Construction.

[J] 1997 NAICS Code is 23551, Carpentry. 2002 NAICS Codes are 23835, Finish Carpentry Contractors, and 23813, Framing Contractors.

[K] 1997 NAICS Code is 23493, Industrial Non-Building Structure Construction. 2002 NAICS Code is 23621, Industrial Building Construction.

[L] "Entities" refer to business firms or governmental bodies; "establishments" refer to industrial plants. Data on affected entities, establishments, and employees are from multiple sources; see the industrial profiles in Chapter II in the PEA (Ex. 35-391) for the complete list of references.

[M] Industry revenues and profits were estimated from data reported in I.R.S., ***Corporation Source Book of Statistics of Income, 2000.*** Data on revenues for State and Local Governments were taken from U.S. Census Bureau, ***Government Finances: 1999-2000***, January 2003.

Source: U.S. Dept. of Labor, OSHA, Office of Regulatory Analysis, based on IT, 2004.

This screening analysis is used to determine whether the compliance costs potentially associated with the standard would lead to significant impacts on establishments in the affected industries. The actual impact of the standard on the viability of establishments in a given industry will depend on the price elasticity of demand for the services sold by establishments in that industry.

Price elasticity refers to the relationship between the price charged for a service and the demand for that service; that is, the more elastic the relationship, the less able an establishment is to pass the costs of compliance through to its customers in the form of a price increase and the more it will have to absorb the costs of compliance from its profits. When demand is inelastic, establishments can recover most of the costs of compliance simply by raising the prices they charge for that service; under this scenario, profit rates are largely unchanged and the industry remains viable. On the other hand, when demand is elastic, establishments cannot recover all the costs simply by passing the cost increase through in the form of a price increase; instead, they must absorb some of the increase from their profits. Commonly, this will mean both reductions in the quantity of goods and services produced and in profits. In general, ''when an industry is subject to a higher cost, it does not simply swallow it, it raises its price and reduces its output, and in this way shifts a part of the cost to its consumers and a part to its suppliers,'' in the words of the court in *American Dental Association* v. *Secretary of Labor* (984 F.2d 823, 829 (Seventh Cir. 1993)).

Specifically if demand is completely inelastic (i.e., price elasticity is 0), then the impact of compliance costs that amount to 1 percent of revenues would be a 1 percent increase in the price of the product or service, with no decline in demand or in profits. Such a situation is rare but might be approximately correct in situations in which there are few, if any, substitutes for the product or service offered by the affected sector or if the products or services of the affected sector account for only a small portion of the income of its consumers. If the demand is perfectly elastic (i.e., the price elasticity is infinitely large), then no increase in price is possible, and before-tax profits would be reduced by an amount equal to the costs of compliance (minus any savings resulting from improved worker health) if the industry attempted to keep producing the same amount of goods and services as previously. Under this scenario, if the costs of compliance represent a large percentage of the sector's profits, some establishments might be forced to close. This scenario is highly unlikely to occur, however, because it can only arise when there are other goods and services that are, in the eye of the consumer, perfect substitutes for the goods and services the affected establishments produce or provide.

A common intermediate case would be a price elasticity of one. In this situation, if the costs of compliance amount to 1 percent of revenues, then production would decline by 1 percent and prices would rise by 1 percent. In this case, the industry revenues would stay the same, with somewhat lower production but similar profit rates. Consumers would, however, get less of the product or the service for their expenditures, and producers would collect lower total profits; this, as the court described in *ADA* v. *Secretary of Labor*, is the more typical case.

Table IX–6 provides costs as percentage of revenues and profits for all affected establishments. OSHA believes that this is the best way to examine its statutory responsibility to determine whether the standard affects the viability of an industry as a whole. There is only one industry where costs exceed one percent of revenues (chromium catalyst production), and none in which costs exceed 1.5 percent of revenues. In only four industries (electroplating, construction welding, chromium catalyst production and chromium catalyst service) do compliance costs exceed 10 percent of profits.

In the case of construction, such cost changes are unlikely to significantly alter the demand for construction welding services which are essential for many projects and not subject to foreign competition. Independent electroplating shops have also been subject to annual changes larger in magnitude than the

costs of hexavalent chromium. The required price increase to fully restore profits of 0.93 percent is significantly less than the average annual increase in price of electroplating services. While such an additional price change might cause some small drop in the demand for services, the historical data clearly show that such price changes can be incurred without affecting the viability of the industry. Chromium catalyst production and service companies are also unlikely to be affected by costs of the relative magnitude found here. While there may be a small long term shift from the use of chromium catalysts as a result of the regulation, most companies are locked into the use of specific catalyst without major new investments. As a result, while there may be some long term shift away from the use of chromium catalysts, a price change of one percent are unlikely to immediately prompt such a change. This also means that the market for the services of chrome catalyst services is likely to be maintained. Further, faced with a new regulation, companies are more rather than less likely to turn to a service company to handle chromium products. Based on these considerations, OSHA preliminarily determines that the proposed standard is economically feasible.

Table IX–7 shows costs as percentage of profits and revenues for firms classified as small by the Small Business Administration and Table IX–8 shows costs as a percentage of revenues and profits for establishments with less than 20 employees. These Tables show greater potential impacts, especially for small electroplating establishments. Based on these results, OSHA has prepared an Initial Regulatory Flexibility Analysis to examine the impacts on small businesses and how they can be alleviated.

Table IX-7. Economic Impacts on Small Business Entities Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group for a PEL of 1 ug/m^3)

	Application Group	NAICS	Category	Cost per Entity[L] Incremental-Requirement Costs	Revenue per Entity[M]	Profit per Entity[M]	Impacts for Incremental-Requirement Costs: Cost/Revenue Impact	Impacts for Incremental-Requirement Costs: Cost/Profit Impact
1	Electroplating - General Industry	All General Industry		$17,073	$7,723,529	$296,584	0.22%	5.76%
		331		$12,869	$19,765,430	$588,199	0.07%	2.19%
		332812	Metal Coating, Engraving (Except Jewerly and Silverware), and Allied Services to Manufacturers	$23,931	$6,192,928	$392,471	0.39%	6.10%
		332813	Electroplating, Plating, Polishing, Anodizing, and Coloring	$13,902	$1,483,200	$93,996	0.94%	14.79%
		Other 332	Fabricated Metal Product Manufacturing	$12,799	$5,002,320	$304,711	0.26%	4.20%
		333	Machinery Manufacturing	$13,414	$10,528,157	$529,745	0.13%	2.53%
		336 (except 33661)	Transportation Equipment Manufacturing	$18,961	$124,514,607	$3,704,808	0.02%	0.51%
		339	Miscellaneous Manufacturing	$20,734	$21,433,301	$1,525,472	0.10%	1.36%
		Other General Industry[A]		$13,971	$3,824,029	$142,545	0.37%	9.80%
2A	Welding - General Industry	31-33[B]	Manufacturing	$4,502	$7,448,864	$341,255	0.06%	1.32%
2B	Welding - Maritime Industry	336611	Ship Building and Repairing	$6,035	$32,947,099	$1,531,546	0.02%	0.39%
2C	Welding - Construction Industry	233[C]	Building, Developing, and General Contracting; Heavy Construction; Special Trade Contractors	$10,715	$2,377,623	$98,731	0.45%	10.85%
2D	Welding - Government	999200	State	N/A	N/A	N/A	N/A	N/A
		999300	Local	$580	$3,670,000	N/A	0.02%	0.02%
3A	Painting - General Industry	31-33	Manufacturing					
		332812	Metal Coating, Engraving (Except Jewerly and Silverware), and Allied Services to Manufacturers	$29,185	$5,662,391	$358,848	0.52%	8.13%
		336411, 336414, 336415, 336419, 336992	Transportation Equipment Manufacturing	$19,580	$225,712,596	$7,336,336	0.01%	0.27%
3B	Painting - Maritime Industry	33661	Ship and Boat Building	$8,505	$18,367,252	$853,802	0.05%	1.00%
3C	Painting - Construction Industry	234[D], 235[E]	Heavy Construction, Special Trade Contractors	$340	$1,831,327	$44,949	0.02%	0.76%
		234[D], 235[E]	Heavy Construction	$708	$9,931,086	$353,547	0.01%	0.20%
		235[E]	Special Trade Contractors	$279	$379,684	$16,492	0.07%	1.69%

Table IX-7. Economic Impacts on Small Business Entities Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group for a PEL of 1 ug/m^3)

	Application Group	NAICS	Category	Cost per Entity[L] Incremental-Requirement Costs	Revenue per Entity[M]	Profit per Entity[M]	Impacts for Incremental-Requirement Costs: Cost/Revenue Impact	Impacts for Incremental-Requirement Costs: Cost/Profit Impact
3D	Painting - Government	999200	State	N/A	N/A	N/A	N/A	N/A
		999300	Local	$536	$3,670,000	N/A	0.01%	0.02%
4	Chromate (Chromite Ore Production)	325188	All Other Basic Inorganic Chemical Mfg.	N/A	N/A	N/A	N/A	N/A
5	Chromate Pigment Producers	325131	Inorganic Dye and Pigment Mfg.	$54,112	$77,883,388	$3,138,701	0.07%	1.72%
6	Chromated Copper Arsenate Producers	325320	Pesticide and Other Agricultural Chemical Mfg.	$7,052	$26,210,731	$1,480,199	0.03%	0.48%
7	Chromium Catalyst Producers	325188	All Other Basic Inorganic Chemical Mfg.	$995,311	$93,357,606	$3,762,312	1.07%	26.45%
8	Paint and Coatings Producers	325510	Paint and Coating Mfg.	$24,564	$16,941,999	$804,203	0.14%	3.05%
9	Printing Ink Producers	325910	Printing Ink Mfg.	$26,872	$15,846,214	$800,598	0.17%	3.36%
10	Plastic Colorant Producers and Users	325211	Plastics Material and Resin Mfg.	$12,563	$8,852,129	$453,140	0.14%	2.77%
		325991	Custom Compounding of Purchased Resin					
		3261	Plastic Product Mfg.					
11	Plating mixture Producers	325998	All Other Miscellaneous Chemical Product and Preparation Mfg.	$23,918	$31,221,004	$4,675,601	0.08%	0.51%
12	Wood Preserving	321114	Wood Preservation	N/A	N/A	N/A	N/A	N/A
13	Chromium Metal Producers	331112	Electrometallurgical Ferroalloy Product Mfg.	N/A	N/A	N/A	N/A	N/A
14	Steel Mills	331111	Iron and Steel Mills	$14,413	$170,519,898	$3,368,791	0.01%	0.43%
15	Iron and Steel foundries	3315	Iron foundries	$20,120	$13,493,814	$655,975	0.15%	3.07%
		331512	Steel Investment foundries					
		331513	Steel foundries (except investment)					
16	Chromium Dioxide Producers	325188	All Other Inorganic Chemicals, n.e.c.	N/A	N/A	N/A	N/A	N/A
17	Chromium Dye Producers	3251317	Chrome Colors and Other Inorganic Pigments	$85,265	$78,623,678	$3,168,534	0.11%	2.69%
18	Chromium Sulfate Producers	325188	All Other Inorganic Chemicals, n.e.c.	$6,954	$2,899,173	$116,837	0.24%	5.95%
19	Chemical Distributors	42269[F]	Other Chemical and Allied Products	$3,859	$3,913,866	$163,627	0.10%	2.36%
20	Textile Dyeing	313	Textile Mills	$1,325	$5,046,265	$158,306	0.03%	0.84%
		314	Textile Product Mills					
21	Colored Glass Producers	3272123	Other Pressed and Blown Glass and Glassware Mfg.	$1,392	$8,645,887	$272,224	0.02%	0.51%
		3272129	Other Pressed and Blown Glass and Glassware Mfg.					
22	Printing	32311	Printing	$593	$783,776	$33,429	0.08%	1.77%
		323113	Commercial Screen Printing					

Table IX-7. Economic Impacts on Small Business Entities Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group for a PEL of 1 ug/m^3)

				Cost per Entity[L]			Impacts for Incremental-Requirement Costs	
	Application Group	NAICS	Category	Incremental-Requirement Costs	Revenue per Entity[M]	Profit per Entity[M]	Cost/Revenue Impact	Cost/Profit Impact
23	Leather Tanning	3161	Leather and Hide Tanning and Finishing	N/A	N/A	N/A	N/A	N/A
24	Chromium Catalyst Users	325110	Petrochemical Mfg., Including Styrene	$6,086	$129,321,394	$5,784,287	0.00%	0.11%
24A	Chromium Catalyst Users - Service Companies	561790	Other Services to Buildings and Dwellings, Including Catalyst handling	$11,349	$1,461,540	$56,120	0.78%	20.22%
25	Refractory Brick Producers	327125	Nonclay Refractory Mfg.	$13,480	$49,494,145	$2,092,266	0.03%	0.64%
26A	Wood Working -General Industry	321	General Industry	$3,815	$6,746,667	$209,147	0.06%	1.82%
26B	Wood Working - Maritime Industry	336611	Ship Building and Repairing	$175	$38,428,332	$1,786,917	0.00%	0.01%
26C	Wood Working - Construction Industry	2332[G], 2333[H], 2349[I], 23551[J]	Construction	$542	$7,316,483	$303,634	0.01%	0.18%
26D	Wood Working - Government	999200	State	N/A	N/A	N/A	N/A	N/A
		999300	Local	$521	$3,670,000	N/A	0.01%	0.04%
27	Solid Waste Incineration	562213	Solid Waste Combustors and Incinerators	$3,671	$3,821,846	$122,548	0.10%	3.00%
27A	Solid Waste Incineration - govt	999300	Local Governments	N/A	$3,670,000	N/A	N/A	0.11%
28	Oil and Gas Well Drilling	213111	Drilling Oil and Gas Wells	N/A	N/A	N/A	N/A	N/A
29	Portland Cement Producers	327310	Cement Mfg.	$2,940	$16,657,610	$1,211,258	0.02%	0.24%
30	Superalloy Producers	331492	Secondary Smelting, Refining and Alloying of Nonferrous Metal	$4,430	$26,787,791	$797,178	0.02%	0.56%
		331528	Other Nonferrous Foundries					
31B	Construction - Refractory Brick Restoration and Maintenance	235[E]	Special Trade Contractors	$84	$973,786	$42,298	0.01%	0.20%
31C	Construction - Hazardous Waste Site Work	2333[H]	Nonresidential Building Construction	$304	$4,621,489	$194,565	0.01%	0.16%
31CG	Hazardous Waste Site Work - Government	999200	State	N/A	N/A	N/A	N/A	N/A
		999300	Local	$226	$3,670,000	N/A	0.01%	0.03%
31D	Construction - Industrial Rehabilitation and Maintenance	23493[K]	Industrial Nonbuilding Structure Construction	$179	$3,303,996	$136,650	0.01%	0.13%
31DG	Industrial Rehabilitation and Maintenance - Government	999200	State	N/A	N/A	N/A	N/A	N/A
		999300	Local	$216	$3,670,000	N/A	0.01%	0.01%
32	Precast Concrete Products Producers	327331, 327332, 327390	Concrete Pipe, Brick, and Block Mfg.	$4,513	$2,997,332	$217,951	0.15%	2.07%

Footnotes

[A] Includes industries in NAICS 31-33, NAICS 42, NAICS 44-45, NAICS 51, NAICS 53, NAICS 54, NAICS 56, NAICS 61, NAICS 71, and NAICS 81.

[B] Includes industries in NAICS 11, NAICS 22, NAICS 31-33, NAICS 42, NAICS 44-45, NAICS 48-49, NAICS 51, NAICS 52, NAICS 53, NAICS 54, NAICS 56, NAICS 61, NAICS 62, NAICS 71, NAICS 72, and NAICS 81.

[C] 1997 NAICS Code is 233, Building, Developing, and General Contracting. 2002 NAICS Code is 236, Construction of Buildings.

[D] 1997 NAICS Code is 234, Heavy Construction. 2002 NAICS Code is 236, Heavy and Civil Engineering Construction.

[E] 1997 NAICS Code is 235, Special Trades Contractors. 2002 NAICS Code is 236, Special Trades Contractors.

[F] 1997 NAICS Code is 42269, Other Chemical and Allied Products. 2002 NAICS Code is 424690, Other Chemical and Allied Products Merchant Wholesalers.

[G] 1997 NAICS Code is 2332, Residential Building Construction. 2002 NAICS Code is 23611, Residential Building Construction.

[H] 1997 NAICS Code is 2333, Nonresidential Building Construction. 2002 NAICS Code is 2362, Nonresidential Building Construction.

[I] 1997 NAICS Code is 2349, Other Heavy Construction. 2002 NAICS Code is 237, Heavy and Civil Engineering Construction.

[J] 1997 NAICS Code is 23551, Carpentry. 2002 NAICS Codes are 23835, Finish Carpentry Contractors, and 23813, Framing Contractors.

[K] 1997 NAICS Code is 23493, Industrial Non-Building Structure Construction. 2002 NAICS Code is 23621, Industrial Building Construction.

[L] "Entities" refer to business firms or governmental bodies; "establishments" refer to industrial plants. Data on affected entities, establishments, and employees are from multiple sources; see the industrial profiles in Chapter II in the PEA (Ex. 35-391) for the complete list of references.

[M] Industry revenues and profits were estimated from data reported in I.R.S., ***Corporation Source Book of Statistics of Income, 2000.*** Data on revenues for State and Local Governments were taken from U.S. Census Bureau, ***Government Finances: 1999-2000***, January 2003.

Source: U.S. Dept. of Labor, OSHA, Office of Regulatory Analysis, based on IT, 2004.

Table IX-8. Economic Impacts on Small (<20 Employees) Establishments Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group for a PEL of 1 ug/m^3)

	Application Group	NAICS	Category	Cost per Establishment[L] Incremental-Requirement Costs	Revenue per Establishment[M]	Profit per Establishment[M]	Impacts for Incremental-Requirement Costs: Cost/Revenue Impact	Impacts for Incremental-Requirement Costs: Cost/Profit Impact
1	Electroplating - General Industry	All General Industry		$7,985	$1,662,857	$63,854	0.48%	12.51%
		331		$7,985	$1,600,531	$47,630	0.50%	16.77%
		332812	Metal Coating, Engraving (Except Jewerly and Silverware), and Allied Services to Manufacturers	$7,985	$627,453	$39,764	1.27%	20.08%
		332813	Electroplating, Plating, Polishing, Anodizing, and Coloring	$7,985	$389,893	$24,709	2.05%	32.32%
		Other 332	Fabricated Metal Product Manufacturing	$7,985	$1,081,783	$65,896	0.74%	12.12%
		333	Machinery Manufacturing	$7,985	$1,499,968	$75,474	0.53%	10.58%
		336 (except 33661)	Transportation Equipment Manufacturing	$7,985	$3,227,879	$96,042	0.25%	8.31%
		339	Miscellaneous Manufacturing	$7,985	$1,178,611	$83,885	0.68%	9.52%
		Other General Industry[A]		$7,985	$1,746,042	$65,085	0.46%	12.27%
2A	Welding - General Industry	31-33[B]	Manufacturing	$2,203	$916,520	$41,989	0.24%	5.25%
2B	Welding - Maritime Industry	336611	Ship Building and Repairing	$2,028	$1,221,589	$56,786	0.17%	3.57%
2C	Welding - Construction Industry	233[C]	Building, Developing, and General Contracting; Heavy Construction; Special Trade Contractors	$5,742	$985,431	$40,920	0.58%	14.03%
2D	Welding - Government	999200	State	N/A	N/A	N/A	N/A	N/A
		999300	Local	N/A	N/A	N/A	N/A	N/A
3A	Painting - General Industry	31-33	Manufacturing					
		332812	Metal Coating, Engraving (Except Jewerly and Silverware), and Allied Services to Manufacturers	$6,167	$745,100	$47,220	0.83%	13.06%
		336411, 336414, 336415, 336419, 336992	Transportation Equipment Manufacturing	$6,167	$6,288,227	$204,386	0.10%	3.02%
3B	Painting - Maritime Industry	33661	Ship and Boat Building	$3,577	$1,221,589	$56,786	0.29%	6.30%
3C	Painting - Construction Industry	234[D], 235[E]	Heavy Construction, Special Trade Contractors	$251	$484,203	$10,042	0.05%	2.50%
		234[D], 235[E]	Heavy Construction	$251	$673,405	$23,973	0.04%	1.05%

Table IX-8. Economic Impacts on Small (<20 Employees) Establishments Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group for a PEL of 1 ug/m^3)

	Application Group	NAICS	Category	Cost per Establishment[L] Incremental-Requirement Costs	Revenue per Establishment[M]	Profit per Establishment[M]	Impacts for Incremental-Requirement Costs Cost/Revenue Impact	Cost/Profit Impact
		235[E]	Special Trade Contractors	$251	$248,785	$10,806	0.10%	2.33%
3D	Painting - Government	999200	State	N/A	N/A	N/A	N/A	N/A
		999300	Local	N/A	N/A	N/A	N/A	N/A
4	Chromate (Chromite Ore Production)	325188	All Other Basic Inorganic Chemical Mfg.	N/A	N/A	N/A	N/A	N/A
5	Chromate Pigment Producers	325131	Inorganic Dye and Pigment Mfg.	$7,759	$2,197,133	$88,544	0.35%	8.76%
6	Chromated Copper Arsenate Producers	325320	Pesticide and Other Agricultural Chemical Mfg.	N/A	N/A	N/A	N/A	N/A
7	Chromium Catalyst Producers	325188	All Other Basic Inorganic Chemical Mfg.	N/A	N/A	N/A	N/A	N/A
8	Paint and Coatings Producers	325510	Paint and Coating Mfg.	$24,217	$3,337,373	$158,418	0.73%	15.29%
9	Printing Ink Producers	325910	Printing Ink Mfg.	$11,618	$5,194,382	$262,436	0.22%	4.43%
10	Plastic Colorant Producers and Users	325211	Plastics Material and Resin Mfg.	$8,480	$1,042,866	$53,384	0.81%	15.89%
		325991	Custom Compounding of Purchased Resin					
		3261	Plastic Product Mfg.					
11	Plating mixture Producers	325998	All Other Miscellaneous Chemical Product and Preparation Mfg.	$22,664	$4,385,091	$221,548	0.52%	10.23%
12	Wood Preserving	321114	Wood Preservation	N/A	N/A	N/A	N/A	N/A
13	Chromium Metal Producers	331112	Electrometallurgical Ferroalloy Product Mfg.	N/A	N/A	N/A	N/A	N/A
14	Steel Mills	331111	Iron and Steel Mills	$8,687	$2,095,343	$41,396	0.41%	20.99%
15	Iron and Steel foundries	3315	Iron foundries	$8,010	$728,668	$35,423	1.10%	22.61%
		331512	Steel Investment foundries					
		331513	Steel foundries (except investment)					
16	Chromium Dioxide Producers	325188	All Other Inorganic Chemicals, n.e.c.	N/A	N/A	N/A	N/A	N/A
17	Chromium Dye Producers	3251317	Chrome Colors and Other Inorganic Pigments	$20,803	$2,165,058	$87,252	0.96%	23.84%
18	Chromium Sulfate Producers	325188	All Other Inorganic Chemicals, n.e.c.	$6,954	$2,899,173	$116,837	0.24%	5.95%
19	Chemical Distributors	42269[F]	Other Chemical and Allied Products	$3,022	$1,391,729	$58,184	0.22%	5.19%
20	Textile Dyeing	313	Textile Mills	$982	$558,520	$17,521	0.18%	5.61%
		314	Textile Product Mills					
21	Colored Glass Producers	3272123	Other Pressed and Blown Glass and Glassware Mfg.	$1,127	$908,236	$28,597	0.12%	3.94%
		3272129	Other Pressed and Blown Glass and Glassware Mfg.					
22	Printing	32311	Printing	$428	$246,802	$10,526	0.17%	4.06%

Table IX-8. Economic Impacts on Small (<20 Employees) Establishments Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group for a PEL of 1 ug/m^3)

	Application Group	NAICS	Category	Cost per Establishment[L] Incremental-Requirement Costs	Revenue per Establishment[M]	Profit per Establishment[M]	Impacts for Incremental-Requirement Costs Cost/Revenue Impact	Impacts for Incremental-Requirement Costs Cost/Profit Impact
		323113	Commercial Screen Printing					
23	Leather Tanning	3161	Leather and Hide Tanning and Finishing	N/A	N/A	N/A	N/A	N/A
24	Chromium Catalyst Users	325110	Petrochemical Mfg., Including Styrene	N/A	N/A	N/A	N/A	N/A
24A	Chromium Catalyst Users - Service Companies	561790	Other Services to Buildings and Dwellings, Including Catalyst handling	$7,824	$906,270	$34,799	0.86%	22.48%
25	Refractory Brick Producers	327125	Nonclay Refractory Mfg.	$0	NA	NA	0.00%	0.00%
26A	Wood Working -General Industry	321	General Industry	$3,282	$1,131,920	$35,090	0.29%	9.35%
26B	Wood Working - Maritime Industry	336611	Ship Building and Repairing	$148	$1,221,589	$56,804	0.01%	0.26%
26C	Wood Working - Construction Industry	2332[G], 2333[H], 2349[I], 23551[J]	Construction	$524	$1,718,617	$71,323	0.03%	0.73%
26D	Wood Working - Government	999200	State	N/A	N/A	N/A	N/A	N/A
		999300	Local	N/A	N/A	N/A	N/A	N/A
27	Solid Waste Incineration	562213	Solid Waste Combustors and Incinerators	$2,021	$1,697,066	$54,416	0.12%	3.71%
27A	Solid Waste Incineration - govt	999300	Local Governments	N/A	N/A	N/A	N/A	N/A
28	Oil and Gas Well Drilling	213111	Drilling Oil and Gas Wells	N/A	N/A	N/A	N/A	N/A
29	Portland Cement Producers	327310	Cement Mfg.	$2,275	$2,609,367	$189,740	0.09%	1.20%
30	Superalloy Producers	331492	Secondary Smelting, Refining and Alloying of Nonferrous Metal	N/A	N/A	N/A	N/A	N/A
		331528	Other Nonferrous Foundries					
31B	Construction - Refractory Brick Restoration and Maintenance	235[E]	Special Trade Contractors	$52	$478,361	$20,779	0.01%	0.25%
31C	Construction - Hazardous Waste Site Work	2333[H]	Nonresidential Building Construction	$226	$1,219,945	$51,360	0.02%	0.44%
31CG	Hazardous Waste Site Work - Government	999200	State	N/A	N/A	N/A	N/A	N/A
		999300	Local	N/A	N/A	N/A	N/A	N/A
31D	Construction - Industrial Rehabilitation and Maintenance	23493[K]	Industrial Nonbuilding Structure Construction	$52	$613,995	$25,394	0.01%	0.20%
31DG	Industrial Rehabilitation and Maintenance - Government	999200	State	N/A	N/A	N/A	N/A	N/A
		999300	Local	N/A	N/A	N/A	N/A	N/A
32	Precast Concrete Products Producers	327331, 327332, 327390	Concrete Pipe, Brick, and Block Mfg.	$3,395	$885,373	$64,380	0.38%	5.27%

Footnotes

[A] Includes industries in NAICS 31-33, NAICS 42, NAICS 44-45, NAICS 51, NAICS 53, NAICS 54, NAICS 56, NAICS 61, NAICS 71, and NAICS 81.

[B] Includes industries in NAICS 11, NAICS 22, NAICS 31-33, NAICS 42, NAICS 44-45, NAICS 48-49, NAICS 51, NAICS 52, NAICS 53, NAICS 54, NAICS 56, NAICS 61, NAICS 62, NAICS 71, NAICS 72, and NAICS 81.

[C] 1997 NAICS Code is 233, Building, Developing, and General Contracting. 2002 NAICS Code is 236, Construction of Buildings.

[D] 1997 NAICS Code is 234, Heavy Construction. 2002 NAICS Code is 236, Heavy and Civil Engineering Construction.

[E] 1997 NAICS Code is 235, Special Trades Contractors. 2002 NAICS Code is 236, Special Trades Contractors.

[F] 1997 NAICS Code is 42269, Other Chemical and Allied Products. 2002 NAICS Code is 424690, Other Chemical and Allied Products Merchant Wholesalers.

[G] 1997 NAICS Code is 2332, Residential Building Construction. 2002 NAICS Code is 23611, Residential Building Construction.

[H] 1997 NAICS Code is 2333, Nonresidential Building Construction. 2002 NAICS Code is 2362, Nonresidential Building Construction.

[I] 1997 NAICS Code is 2349, Other Heavy Construction. 2002 NAICS Code is 237, Heavy and Civil Engineering Construction.

[J] 1997 NAICS Code is 23551, Carpentry. 2002 NAICS Codes are 23835, Finish Carpentry Contractors, and 23813, Framing Contractors.

[K] 1997 NAICS Code is 23493, Industrial Non-Building Structure Construction. 2002 NAICS Code is 23621, Industrial Building Construction.

[L] "Entities" refer to business firms or governmental bodies; "establishments" refer to industrial plants. Data on affected entities, establishments, and employees are from multiple sources; see the industrial profiles in Chapter II in the PEA (Ex. 35-391) for the complete list of references.

[M] Industry revenues and profits were estimated from data reported in I.R.S., ***Corporation Source Book of Statistics of Income, 2000.*** Data on revenues for State and Local Governments were taken from U.S. Census Bureau, ***Government Finances: 1999-2000***, January 2003.

Source: U.S. Dept. of Labor, OSHA, Office of Regulatory Analysis, based on IT, 2004.

F. Benefits and Net Benefits

OSHA estimated the benefits associated with alternative PELs for Cr(VI) by applying the dose-response relationship developed in the risk assessment to current exposure levels. OSHA determined current exposure levels by first developing an exposure profile for industries with Cr(VI) exposures using OSHA inspection and site visit data, and then applying this profile to the current worker population. The industry by industry exposure profile was given in Table IX–2 above.

By applying the dose-response relationship to estimates of current exposure levels across industries, it is possible to project the number of lung cancers expected to occur in the worker population given current exposures (the ''baseline''), and the number of these cases that would be avoided under alternative, lower PELs. OSHA assumed that exposures below the limit of detection (LOD) are equivalent to no exposure to Cr(VI), thus assigning no baseline or avoided lung cancers (and hence, no benefits) to these exposures. For exposures above the current PEL and for purposes of determining the benefit of reducing the PEL, OSHA assumed exposure at exactly the PEL. Consequently, the benefits computed below are attributable only to a change in the PEL. No benefits are assigned to the effect of a new standard increasing compliance with the current PEL. OSHA estimates that between 2,247 and 8,708 lung cancers attributable to Cr(VI) exposure will occur during the working lifetime of the current worker population. Table IX–9 shows the number of avoided lung cancers by PEL. At the proposed PEL of 1 $\mu g/m^3$, and estimated 1,970 to 7,500 lung cancers would be prevented over the working lifetime of the current worker population.

TABLE IX–9.—AVOIDED LUNG CANCERS ESTIMATES BY PEL

PEL ($\mu g/^3m$)	0.25	0.5	1	5	10	20
Avoided Cancers (Total)	2,147–8,270	2,078–7,968	1,970–7,500	1,440–5,233	1,052–3,649	585–1,864
Avoided Cancers (Annual)	48–184	46–177	44–167	32–116	23–81	13–41

Note that the Agency based these estimates on a worker that is employed in a Cr(VI) exposed occupation for his entire working life, from age 20 to 65. The calculation also does not allow workers to enter or exit Cr(VI) jobs, or switch to other exposure groups during their working lives. While the assumptions of 45 years of exposure and no mobility among exposure groups may seem restrictive, these assumptions actually are likely to yield somewhat conservative estimates of the number of avoided cancers, given the nature of the risk assessment model. For example, consider the case of job covered by five workers, each working nine years rather than one worker for 45 years. The former situation will likely yield a slightly higher rate of lung cancers, since more workers are exposed to the carcinogen (albeit for a shorter period of time) and that the average age of the workers exposed is likely to decrease. This is due to: (1) The linearity of the estimated dose-response relationship, and (2) once an individual accumulates a dose, the increase in relative risk persists for the remainder of his lifetime. For example, a worker exposed from age 20 to 30 will have a constant increased relative risk for about 50 or so years (from age 30 on, assuming no lag between exposure and increased risk and death at age 80), whereas a person exposed from age 40 to 50 will have only about 30 years of increased risk (again assuming no lag and death at age 80). The persistence of the increased relative risk for a lifetime follows directly from the risk assessment, and is typical of life table analysis. OSHA intends to investigate the implications of alternative exposure scenarios in the

course of further developing its economic benefits assessment.

For informational purposes only, OSHA has estimated the monetary value of the benefits associated with the draft proposed rule. These estimates are informational because OSHA cannot use benefit-cost analysis as a basis for determining the PEL for a health standard. In order to estimate monetary values for the benefits associated with the proposed rule, OSHA reviewed the approaches taken by other regulatory agencies for similar regulatory actions. OSHA found that occupational illnesses are analogous to the types of illnesses targeted by EPA regulations and has thus used them in this analysis.

OSHA is adopting EPA's approach, applying a value of $6.8 million to each premature fatality avoided. The $6.8 million value represents individuals' willingness-to-pay (WTP) to reduce the risk of premature death.

Nonfatal cases of lung cancer can be valued using a cost of illness (COI) approach, using data on associated medical costs. The EPA Cost of Illness Handbook (Ex.35–333) reports that the medical costs for a nonfatal case of lung cancer are, on average, $136,460. Updating the EPA figure to 2003 dollars yields the value of $160,030 Including values for lost productivity, the total COI which is applied to the OSHA estimate of nonfatal cases of lung cancer is $188,502.

An important limitation of the COI approach is that it does not measure individuals' WTP to avoid the risk of contracting nonfatal cancers or illnesses. As an alternative approach, nonfatal cancer benefits may be estimated by adjusting the value of lives saved estimates. In its Stage 2 Disinfection and Disinfection Byproducts water rule, EPA used studies on the WTP to avoid nonfatal lymphoma and chronic bronchitis as a basis for valuing nonfatal cancers. In sum, EPA valued nonfatal cancers at 58.3% of the value of a fatal cancer. Using WTP information would yield a higher estimate of the benefits associated with the reduction in nonfatal lung cancers, as the nonfatal cancers would be valued at $4 million rather than $188,502 per case. These values represent the upper bound values for nonfatal cases of lung cancer avoided.

Using these assumptions, and latency periods of 10, 20 and 35 years and possible increases in the value of life over time, OSHA estimated the total annual benefits of the standard at various PELS in Table IX–10, considering both the benefits from preventing fatal and non-fatal cases of lung cancer.

TABLE IX–10.—TOTAL ANNUAL LUNG CANCER BENEFITS

[Millions of 2003 Dollars]

PEL (μg/m³)	0.25	0.5	1	5	10	20
Undiscounted	$287–1,189	$278–1,145	$263–1,078	$192–753	$141–525	$78–269
Discount Rate = 3%	102–1,131	99–1,090	94–1,026	69–716	50–500	28–256
Discount Rate = 7%	27–773	26–745	25–701	18–490	14–342	8–175

Occupational exposure to Cr(VI) has also been linked to a multitude of other health effects, including irritated and perforated nasal septum, skin ulceration, asthma, and dermatitis. Current data on Cr(VI) exposure and health effects are insufficient to quantify the precise extent to which many of these ailments occur. However, it is possible to provide an upperbound estimate of the number of cases of dermatitis that occur annually and an upper estimate of the number that will be prevented by a standard. This estimate is an upperbound because it uses data on incidence of dermatitis among cement workers, where dermatitis is more common than it would be for other exposures to Cr(VI). It is important to note that if OSHA were able to quantify all Cr(VI)-related health effects, the quantified benefits would be somewhat higher than the benefits presented in this analysis.

Using National Institute for Occupational Safety and Health (NIOSH) data, Ruttenberg and Associates (Ex. XXXX) estimate that the incidence of dermatitis among concrete workers is between 0.2 and 1 percent. Applying the 0.2 percent–1 percent incidence rate indicates that there are presently 418–2,089 cases of dermatitis occurring annually. This approach represents an overestimate for cases of dermatitis in other application groups, since some dermatitis among cement workers is caused by other known factors, such as the high alkalinity of cement. If the measures in this draft proposed standard are 50 percent effective in preventing dermatitis, then there would be an estimated 209–1,045 cases of Cr(VI) dermatitis avoided annually.

To assign values to the cases of avoided dermatitis OSHA applied the COI approach. Ruttenberg and Associates computed that, on average, the medical costs associated with a case of dermatitis are $119 (in 2003 dollars) and the indirect and lost productivity costs are $1,239. These estimates were based on an analysis of BLS data on lost time associated with cases of dermatitis, updated to current dollars. Based on the Ruttenberg values, OSHA estimates that a Cr(VI) standard will yield $0.3 million to $1.4 million in annual benefits due to reduced incidence of dermatitis. (These benefits associated with dermatitis are not included in the net benefits analysis, as these benefits largely result from full compliance with existing requirements for PPE and hygiene areas.)

Occupational exposure to Cr(VI) can lead to nasal septum ulcerations and nasal septum perforations. As for cases of dermatitis, the data were insufficient to conduct a formal quantitative risk assessment to relate exposures and incidence. However, previous studies provide a basis for developing an approximate estimate of the number of nasal perforations expected under the current PEL as well as PELs of 0.25 μg/m³, 0.5 μg/m³, 1.0 μg/m³, 5.0 μg/m³, 10.0 μg/m³ and 20.0 μg/m³. Cases of nasal perforations were computed only for workers in electroplating and chrome production. The percentage of workers with nasal tissue damage is expected to be over 50 percent for those regularly exposed above approximately 20 μg/m³. Less than 25 percent of workers could reasonably be expected to experience nasal tissue damage if Cr(VI) exposure was kept below an 8-hour TWA of 5 μg/m³ and regular short-term exposures *e.g.* an hour or so) were below 10 μg/m³. Less than 10 percent of workers could reasonably be expected to experience nasal tissue damage at a TWA Cr(VI) below 2 μg/m³ [and short-term exposures below 10 μg/m³]. It appears likely that nasal damage might be avoided completely if all Cr(VI) [short-term and full shift] exposures were kept below 1 μg/m³.

OSHA estimates that 5,387 nasal perforations/ulcerations occur annually

under the current PEL. All of these are expected to be prevented under the proposed PEL of 1 μg/m³. Due to insufficient data, it was not possible to monetize the benefits. Thus, the benefits associated with a reduction in nasal perforations/ulcerations are excluded from the net benefits analysis presented below.

Finally, for informational purposes, OSHA examined the net benefits of the standard, based on the benefits and costs presented above, and the costs per case of cancer avoided as shown in Table IX–11.

TABLE IX–11.—ANNUAL NET BENEFITS AND COST PER CANCER AVOIDED BY PEL

[Millions of 2003 Dollars]

PEL (μg/m³)	0.25	0.5	1	5	10	20
Discount Rate = 3% Costs (Millions of 2003 Dollars)						
Total Annual	$524	$381	$212	$119	$91	$81
Net Benefits (Millions of 2003 Dollars)						
Minimum	−422	−282	−119	−51	−41	−53
Maximum	606	708	813	596	408	174
Midpoint	92	213	347	273	183	60
Cost Per Cancer Avoided (Millions of 2003 Dollars)						
Minimum	2.9	2.2	1.3	1.0	1.1	2.0
Maximum	11.0	8.3	4.8	3.7	3.9	6.2
Midpoint	6.9	5.2	3.1	2.4	2.5	4.1
Discount Rate = 7% Costs (Millions of 2003 Dollars)						
Total Annual	548	402	223	125	95	84
Net Benefits (Millions of 2003 Dollars)						
Minimum	−521	−376	−198	−107	−82	−77
Maximum	224	342	477	363	246	90
Midpoint	−149	−17	139	128	82	7
Cost Per Cancer Avoided (Millions of 2003 Dollars)						
Minimum	3.0	2.3	1.3	1.1	1.2	2.0
Maximum	11.5	8.7	5.1	3.9	4.1	6.5
Midpoint	7.2	5.5	3.2	2.5	2.6	4.2

In addition to examining alternative PELs, OSHA also examined alternatives to other provisions of the standard. These alternatives are discussed in the Initial Regulatory Flexibility Analysis in the next section.

As noted above, the OSH Act requires OSHA to set standards based on eliminating risk to the extent feasible. Eliminating risk to the extent feasible does not necessarily have anything to do with the results of a benefit cost analysis. Thus, these analyses of net benefits cannot be used as the basis for a decision concerning the choice of a PEL for a Cr(VI) standard.

Incremental costs and benefits are those that are associated with increasing stringency of the standard. Comparison of incremental benefits and costs provides and indication of the relative efficiency of the various PELs. OSHA cannot use this information in selecting a PEL, but it has conducted these calculations for informational purposes. Incremental costs, benefits, net benefits and cost per cancer avoided are presented in Table IX–12. Note that dermal benefits are excluded since they do not vary with the PEL and hence, do not affect the calculations.

TABLE IX–12.—INCREMENTAL BENEFITS, COSTS, NET BENEFITS AND COST PER CANCER AVOIDED

	20→10	10→5	5→1	1→0.5	0.5→0.25
Discount Rate = 3%					
Benefits	$133.0	$117.4	$167.4	$34.5	$22.3
Costs	−10.0	−28.0	−93.0	−169.0	−143.0
Net Benefits	123.0	89.4	74.4	134.5	120.7
Cost Per Cancer Avoided	1.6	0.1	−0.7	−2.3	−1.7
Discount Rate = 7%					
Benefits	86.2	76.4	109.1	22.5	14.5
Costs	−11.0	−30.0	−98.0	179.0	−146.0

TABLE IX–12.—INCREMENTAL BENEFITS, COSTS, NET BENEFITS AND COST PER CANCER AVOIDED—Continued

	20→10	10→5	5→1	1→0.5	0.5→0.25
Net Benefits	75.2	46.4	11.1	156.5	131.5
Cost Per Cancer Avoided	1.6	0.1	−0.7	−2.3	−1.7

G. Initial Regulatory Flexibility Analysis

Reasons Why Action by the Agency Is Being Considered

Several well-conducted scientific investigations have found increased lung cancer mortality among workers breathing Cr(VI) dusts and mists in the workplace. The high rate of lung cancer mortality has been documented in workers from several countries across multiple industries that use a broad spectrum of Cr(VI) compounds. Many of the studies found that the rate of lung cancer was greatest among workers in jobs where Cr(VI) exposure was highest and in workers employed in those jobs for the longest periods of time. These exposure-related trends implicate Cr(VI) as a likely causative agent and suggest that other known lung carcinogens to which the workers may be exposed, such as cigarette smoke, are unlikely to account for the increased lung cancers observed in the studies. The International Agency for Research on Cancer, the U.S. Environmental Protection Agency, and the American Conference of Governmental Industrial Hygienists have evaluated the human, animal, and other experimental evidence and concluded that Cr(VI) compounds are ''known'' or ''confirmed'' human carcinogens.

Two independent epidemiologic studies of workers from chromate production plants in Baltimore, Maryland (Gibb *et al.*, Ex. 31–22–11) and Painesville, Ohio (Luippold *et al.*, Ex. 33–10) were considered to present the strongest data sets for quantitative risk assessment. OSHA's analysis found that a linear, relative risk model provided the best fit to the data (Ex. 33–15; Ex. 33–12). The Agency preliminarily estimates that the excess lifetime lung cancer risk for workers exposed at the current Permissible Exposure Limit (PEL) of 52 μg/m^3 Cr(VI), as an eight-hour time-weighted average for a 45-year working lifetime, ranges from 106 to 351 excess lung cancers per thousand workers exposed. OSHA applied the linear relative risk model to preliminarily estimate excess lifetime lung cancer risks from 45-year exposure at alternative PELs ranging from 0.25 μg/m^3 to 20 μg/m^3 (the range considered for the draft proposed standard). The projected risks at these alternate PELs are between four- and 200-fold lower than risks estimated at the current PEL. NIOSH and the Exponent group have reported similar lung cancer risks based on the Gibb (Ex. 33–13; Ex. 31–18–15–1) and the Luippold (Ex. 31–18–3) data sets and a relative risk model. The risk estimates at the very lowest Cr(VI) exposure levels under consideration (*e.g.*, 0.25 to 2.5 μg/m^3) are considered to be somewhat more uncertain than those projected at the higher Cr(VI) levels because they involve risk model extrapolations below the range of exposures experienced by the Gibb and Luippold worker cohorts.

Exposure to airborne Cr(VI) can cause other adverse effects to the respiratory tract and the skin. Occupational surveys and medical examinations have found nasal septum ulcerations and perforations (i.e. ''chrome holes'') among chromium production workers and chrome electroplaters exposed repeatedly to relatively high levels of Cr(VI) (*e.g.*, 20 μg/m^3 to 50 μg/m^3). (Exs. 31–22–11; 9–126). Several case reports have also documented occupational asthma triggered by breathing Cr(VI) compounds in the workplace. Workers can also develop an allergic reaction of the skin known as allergic contact dermatitis as a result of repeated direct dermal contact with Cr(VI) solutions or other Cr(VI)-containing materials. Allergic contact dermatitis is most common on the hands and arms of workers who mix and use wet Cr(VI)-containing cement. Dermal contact with Cr(VI) can also cause an irritant dermatitis and ulceration of the skin called ''chrome ulcers''. This type of dermatitis is not an allergic condition and requires contact with a fairly concentrated form of Cr(VI). It has been reported primarily in chromate production plants and chrome electroplating facilities with poor industrial hygiene (work) practices.

A full discussion of the health effects and risk assessment that support the reasons why this action is being considered are given in Section VI of the Preamble, Health Effects, and Section VII, Quantitative Risk Assessment.

Objective of and Legal Basis for the Proposed Rule

The objective of the proposed rule is to reduce the numbers of fatalities and illnesses occurring among employees exposed to Cr(VI) in general industry, construction, and shipyard sectors. This objective will be achieved by requiring employers to install engineering controls where appropriate and to provide employees with the equipment, respirators, training, medical surveillance, and other protective measures to perform their jobs safely.

The legal basis for the rule is the responsibility given the U.S. Department of Labor through the Occupational Safety and Health Act of 1970 (OSH Act). The OSH Act authorizes the Secretary of Labor to promulgate occupational safety and health standards as necessary ''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.'' 29 U.S.C. 651(b). The legal authority can also be cited as 29 U.S.C. 655(b).

In addition to the statutory basis for a possible standard, the legal basis for the action also involves litigation on the need for and timetable for a Cr(VI) standard. See the Preamble Section III, for a fuller discussion.

Description and Estimate of Affected Small Entities

Table IX–1 above provides an overview of the number of small entities affected by the standard, by sector. Additional detail is provided in the Full Preliminary Economic Analysis and Initial Regulatory Flexibility Analysis (Ex. 35–391).

Summary of Reporting, Recordkeeping, and Other Compliance Requirements

Table IX–13 shows the costs of the proposed standard for entities classified as small businesses by the SBA.

IX-13. Annualized Costs for Small Businesses Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group and Regulatory Requirement for a PEL of 1 ug/m^3)

	Application Group	Engineering Controls	Initial Exposure Monitoring	Periodic Exposure Monitoring	Respiratory Protection
1	Electroplating	$31,965,164	$646,859	$3,701,220	$1,812,409
2A	Welding (general industry)	$27,886,699	$3,321,234	$16,999,831	$14,595,338
2B	Welding (maritime industry)	$1,108,624	$36,905	$0	$355,039
2C	Welding (construction industry)	$14,898,873	$103,541	$0	$8,986,310
2D	Welding (government)	$62,301	$9,790	$0	$37,223
3A	Painting (general industry)	$1,252,774	$152,248	$615,545	$3,307,451
3B	Painting (maritime industry)	$307,929	$31,651	$0	$6,096,148
3C	Painting (construction industry)	$0	$29,734	$0	$0
3D	Painting (government)	$0	$2,682	$0	$0
4	Chromate (chromite ore) production	$0	$0	$0	$0
5	Chromate Pigment Producers	$47,400	$3,570	$13,808	$39,774
6	Chromated Copper Arsenate (CCA) Producers	$0	$3,502	$14,065	$2,680
7	Chromium Catalyst Producers	$2,272,600	$13,232	$71,440	$587,133
8	Paint and Coatings Producers	$3,594,464	$82,689	$104,921	$22,646
9	Printing Ink Producers	$0	$7,502	$5,452	$135,180
10	Plastic Colorant Producers and Users	$0	$160,679	$807,622	$209,210
11	Plating Mixture Producers	$144,780	$7,905	$28,902	$0
12	Wood Preserving	$0	$0	$0	$0
13	Chromium Material Producers	$0	$0	$0	$0
14	Steel Mills	$303,642	$31,621	$23,564	$102,318
15	Iron and Steel Foundries	$1,565,262	$341,451	$685,829	$1,770,253
16	Chromium Dioxide Producers	$0	$0	$0	$0
17	Chromium Dye Producers	$0	$30,966	$153,686	$63,217
18	Chromium Sulfate Producers	$0	$2,119	$7,410	$0
19	Chemical Distributors	$0	$441,314	$0	$0
20	Textile Dyeing	$0	$381,011	$0	$0
21	Colored Glass Producers	$669	$12,558	$0	$0
22	Printing	$0	$153,903	$0	$0
23	Leather Tanning	$0	$0	$0	$0
24	Chromium Catalyst Users	$0	$23,958	$48,061	$153
24A	Chromium Catalyst Users (Service)	$0	$3,663	$27,979	$0
25	Refractory Brick Producers	$0	$2,865	$2,716	$922
26A	Woodworking (general industry)	$35,400	$61,981	$0	$0
26B	Woodworking (maritime industry)	$0	$6,413	$0	$0
26C	Woodworking (construction industry)	$2,651,352	$900,736	$0	$0
26D	Woodworking (government)	$9,801	$3,330	$0	$0
27	Solid Waste Incineration	$0	$59,882	$129,602	$49,605
27A	Incinerators (government)	$0	$0	$0	$0
28	Oil and Gas Well Drilling	$0	$0	$0	$0
29	Portland Cement Producers	$0	$52,552	$0	$0
30	Superalloy Producers	$667	$765	$510	$861
31B	Construction (Refractory Repair)	$0	$0	$0	$0
31C	Construction (Hazardous Waste Work)	$0	$22,932	$0	$0
31CG	Haz. Waste (government)	$0	$7,194	$0	$0
31D	Construction (Industrial Rehabilitation)	$0	$0	$0	$0
31DG	Industrial Rehab. (government)	$0	$3,949	$0	$0
32	Precast Concrete Products Producers	$0	$3,217,948	$0	$0
	General Industry (including Government)	$69,141,622	$9,244,919	$23,442,160	$22,736,371
	Construction	$17,550,225	$1,056,943	$0	$8,986,310
	Maritime	$1,416,553	$74,969	$0	$6,451,187
	Total	$88,108,401	$10,376,832	$23,442,160	$38,173,867

IX-13. Annualized Costs for Small Businesses Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group and Regulatory Requirement for a PEL of 1 ug/m^3)

	Application Group	Housekeeping	Medical Surveillance	Information and Training	Recordkeeping
1	Electroplating	$8,108,190	$375,787	$418,922	$110,504
2A	Welding (general industry)	$0	$1,051,260	$1,638,701	$86,403
2B	Welding (maritime industry)	$0	$694	$65,381	$8,461
2C	Welding (construction industry)	$0	$10,629	$1,510,241	$143,018
2D	Welding (government)	$0	$49	$21,677	$2,976
3A	Painting (general industry)	$1,005,222	$168,741	$629,802	$60,707
3B	Painting (maritime industry)	$0	$230	$194,731	$11,976
3C	Painting (construction industry)	$0	$3,659	$1,978,139	$147,915
3D	Painting (government)	$0	$353	$318,230	$15,493
4	Chromate (chromite ore) production	$0	$0	$0	$0
5	Chromate Pigment Producers	$0	$2,483	$909	$280
6	Chromated Copper Arsenate (CCA) Producers	$0	$319	$460	$130
7	Chromium Catalyst Producers	$16,000	$17,866	$5,842	$1,820
8	Paint and Coatings Producers	$192,529	$15,810	$31,214	$8,746
9	Printing Ink Producers	$11,375	$0	$985	$741
10	Plastic Colorant Producers and Users	$17,150	$0	$9,533	$1,846
11	Plating Mixture Producers	$54,570	$686	$1,829	$510
12	Wood Preserving	$0	$0	$0	$0
13	Chromium Material Producers	$0	$0	$0	$0
14	Steel Mills	$157,142	$22,140	$38,762	$12,657
15	Iron and Steel Foundries	$608,890	$142,534	$321,261	$157,765
16	Chromium Dioxide Producers	$0	$0	$0	$0
17	Chromium Dye Producers	$5,290	$0	$2,056	$580
18	Chromium Sulfate Producers	$4,040	$183	$116	$40
19	Chemical Distributors	$4,266,556	$3	$30,603	$0
20	Textile Dyeing	$654,931	$80	$218,381	$59,710
21	Colored Glass Producers	$16,350	$63	$841	$150
22	Printing	$51,807	$0	$67,122	$17,797
23	Leather Tanning	$0	$0	$0	$0
24	Chromium Catalyst Users	$125,872	$716	$1,780	$292
24A	Chromium Catalyst Users (Service)	$17,161	$5,492	$1,870	$581
25	Refractory Brick Producers	$6,770	$2	$156	$50
26A	Woodworking (general industry)	$670,089	$1,288	$5,207	$413
26B	Woodworking (maritime industry)	$0	$74	$1,620	$300
26C	Woodworking (construction industry)	$0	$3,572	$309,717	$42,816
26D	Woodworking (government)	$0	$7	$835	$86
27	Solid Waste Incineration	$0	$35	$5,771	$1,060
27A	Incinerators (government)	$0	$0	$0	$0
28	Oil and Gas Well Drilling	$0	$0	$0	$0
29	Portland Cement Producers	$297,693	$257	$49,184	$14,893
30	Superalloy Producers	$921	$25	$518	$163
31B	Construction (Refractory Repair)	$0	$40	$13,335	$1,769
31C	Construction (Hazardous Waste Work)	$0	$123	$32,726	$5,269
31CG	Haz. Waste (government)	$0	$21	$6,325	$923
31D	Construction (Industrial Rehabilitation)	$0	$124	$35,559	$5,579
31DG	Industrial Rehab. (government)	$0	$3	$1,126	$116
32	Precast Concrete Products Producers	$8,513,073	$3,267	$734,285	$225,842
	General Industry (including Government)	$24,801,622	$1,809,470	$4,564,304	$783,274
	Construction	$0	$18,146	$3,879,717	$346,366
	Maritime	$0	$999	$261,732	$20,738
	Total	$24,801,622	$1,828,614	$8,705,752	$1,150,378

IX-13. Annualized Costs for Small Businesses Affected by OSHA's Proposed Standard for Hexavalent Chromium (by Application Group and Regulatory Requirement for a PEL of 1 ug/m^3)

			Current Requirements for PPE and Hygiene Areas			
	Application Group	Total for Incremental Requirements	PPE (not supplied in baseline)	PPE (supplied in baseline)	Hygiene Areas	Total for Incremental and Current Requirements
1	Electroplating	**$47,139,054**	$0	$10,004,481	$1,546,751	**$58,690,286**
2A	Welding (general industry)	**$65,579,466**	$0	$0	$0	**$65,579,466**
2B	Welding (maritime industry)	**$1,575,105**	$0	$0	$0	**$1,575,105**
2C	Welding (construction industry)	**$25,652,611**	$0	$0	$0	**$25,652,611**
2D	Welding (government)	**$134,016**	$0	$0	$0	**$134,016**
3A	Painting (general industry)	**$7,192,490**	$16,459,373	$3,539,991	$527,439	**$27,719,293**
3B	Painting (maritime industry)	**$6,642,665**	$5,162,686	$811,853	$382,830	**$13,000,034**
3C	Painting (construction industry)	**$2,159,446**	$0	$904,287	$0	**$3,063,733**
3D	Painting (government)	**$336,758**	$0	$94,645	$0	**$431,403**
4	Chromate (chromite ore) production	**$0**	$0	$0	$0	**$0**
5	Chromate Pigment Producers	**$108,223**	$0	$5,731	$2,300	**$116,254**
6	Chromated Copper Arsenate (CCA) Producers	**$21,156**	$12,587	$2,086	$1,200	**$37,028**
7	Chromium Catalyst Producers	**$2,985,933**	$110,290	$26,303	$12,700	**$3,135,226**
8	Paint and Coatings Producers	**$4,053,019**	$3,123,597	$464,353	$117,891	**$7,758,861**
9	Printing Ink Producers	**$161,234**	$4,385	$576	$4,243	**$170,438**
10	Plastic Colorant Producers and Users	**$1,206,040**	$20,784	$3,319	$28,377	**$1,258,519**
11	Plating Mixture Producers	**$239,182**	$0	$100,396	$9,400	**$348,978**
12	Wood Preserving	**$0**	$0	$0	$0	**$0**
13	Chromium Material Producers	**$0**	$0	$0	$0	**$0**
14	Steel Mills	**$691,845**	$0	$0	$0	**$691,845**
15	Iron and Steel Foundries	**$5,593,244**	$0	$0	$0	**$5,593,244**
16	Chromium Dioxide Producers	**$0**	$0	$0	$0	**$0**
17	Chromium Dye Producers	**$255,794**	$21,250	$4,643	$5,800	**$287,488**
18	Chromium Sulfate Producers	**$13,908**	$7,245	$386	$1,120	**$22,659**
19	Chemical Distributors	**$4,738,477**	$0	$0	$0	**$4,738,477**
20	Textile Dyeing	**$1,314,112**	$978,517	$176,040	$1,102,876	**$3,571,544**
21	Colored Glass Producers	**$30,630**	$0	$0	$0	**$30,630**
22	Printing	**$290,629**	$357,881	$57,692	$165,906	**$872,109**
23	Leather Tanning	**$0**	$0	$0	$0	**$0**
24	Chromium Catalyst Users	**$200,831**	$38,644	$7,313	$10,582	**$257,369**
24A	Chromium Catalyst Users (Service)	**$56,746**	$0	$14,327	$11,010	**$82,082**
25	Refractory Brick Producers	**$13,480**	$4,983	$877	$883	**$20,224**
26A	Woodworking (general industry)	**$774,378**	$0	$0	$0	**$774,378**
26B	Woodworking (maritime industry)	**$8,408**	$0	$0	$0	**$8,408**
26C	Woodworking (construction industry)	**$3,908,193**	$4,785,549	$555,884	$2,822,822	**$12,072,449**
26D	Woodworking (government)	**$14,059**	$10,822	$4,576	$6,204	**$35,661**
27	Solid Waste Incineration	**$245,955**	$0	$155,579	$42,719	**$444,253**
27A	Incinerators (government)	**$0**	$0	$0	$0	**$0**
28	Oil and Gas Well Drilling	**$0**	$0	$0	$0	**$0**
29	Portland Cement Producers	**$414,579**	$430,115	$77,799	$100,876	**$1,023,369**
30	Superalloy Producers	**$4,430**	$0	$0	$0	**$4,430**
31B	Construction (Refractory Repair)	**$15,144**	$0	$0	$0	**$15,144**
31C	Construction (Hazardous Waste Work)	**$61,051**	$90,000	$244,563	$104,120	**$499,734**
31CG	Haz. Waste (government)	**$14,463**	$0	$46,706	$17,161	**$78,330**
31D	Construction (Industrial Rehabilitation)	**$41,261**	$0	$0	$0	**$41,261**
31DG	Industrial Rehab. (government)	**$5,193**	$0	$0	$0	**$5,193**
32	Precast Concrete Products Producers	**$12,694,415**	$21,660,562	$3,698,534	$4,139,874	**$42,193,386**
	General Industry (including Government)	**$156,523,742**	$43,241,035	$18,486,352	$7,855,312	**$226,106,441**
	Construction	**$31,837,707**	$4,875,549	$1,704,734	$2,926,942	**$41,344,932**
	Maritime	**$8,226,178**	$5,162,686	$811,853	$382,830	**$14,583,547**
	Total	**$196,587,626**	$53,279,270	$21,002,939	$11,165,085	**$282,034,920**

Source: U.S. Dept. of Labor, OSHA, Office of Regulatory Analysis, based on IT, 2004.

Table IX–14 shows the unit costs these estimates are based on. (For a full discussion of the engineering control costs, and of the basis for the unit costs, see Chapter 3 of the Preliminary Economic Analysis and Initial Regulatory Flexibility Analysis).

Table IX–14.—Unit Costs Applied in OSHA's Preliminary Analysis of the Proposed Standard

Cost description	Basis	Base cost	Escalation factor (October 2003 basis)	Index used for price escalation	Unit cost
Cost per hour for an outside industrial hygiene contractor.	Estimate by In-house CIH	$90.00	1	NONE	$90.00
Cost of a personal sampling pump	Gilian 3500; Sensidyne, 16333 Bayvista Drive, Clearwater, FL 33760.	680.00	1	NONE	680.00
Variable Cost per sample (*e.g.*, laboratory analysis).	Estimate by In-house CIH	60.00	1	NONE	60.00
Flat Fee For Training Course	Estimate by In-house CIH.	400.00	1	NONE	400.00
Cost of a calibration unit	GILIBRATOR–2; Sensidyne, 16333 Bayvista Drive, Clearwater, FL 33760.	1,075.00	1	NONE	1,075.00
Unit cost of OSHA-regulation warning signs with mounting materials.	July 1993 EMMED Co, Inc. Catalog	3.03	1.2702	CPI—All items	3.84
Cost of materials per qualitative fit-testing.	Banana Oil Fit Test Kit; Lab Safety Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	0.07	1	NONE	0.07
Unit cost per worker for an air-supplied respirator.	Allegro One-Worker Full Face Kit; Lab Safety Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	1,473.33	1	NONE	1,473.33
Unit cost per employee for a full-face respirator.	MSA Ultra Twin Full Face Respirator; Lab Safety Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	243.00	1	NONE	243.00
Unit cost per employee for a half-mask respirator.	MSA Comfro Classic Half-Mask Respirator; Lab Safety Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	35.30	1	NONE	35.30
Cost of replacement cartridges cartridges per mask).	MSA P100 Filter (2 Cartridge: Lab Safety Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1369.	13.74	1	NONE	13.74
Unit cost per employee for a blasting helmet air-supplied respirator.	Allegro Three Person Air Pump, Bullard 1/2″ Hose, 100′L, Bullard Helmet w/ constant air flow; Lab Safety Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	1,164.00	1	NONE	1,164.00
Cost of materials to clean one respirator.	Respirator Cleaning/Storage Kit; Lab Safety Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	1.86	1	NONE	1.86
Cost of PE coated Tyvek coveralls	KAPPLER Poly-Coat Coveralls; Lab Safety Supply Catalog 2003, PO Box 1368, Janesville, WI 53547– 1368.	6.60	1	NONE	6.60
Cost of Saranex coveralls	Tychem QC Coveralls; Lab Safety Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	32.85	1	NONE	32.85
Cost of Tyvek coveralls	Tyvek Protective Wear Coveralls; Lab Safety Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	4.50	1	NONE	4.50
Cost of bib aprons	Polypropylene Bib Apron; Lab Safety Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	0.58	1	NONE	0.58
Cost of laundering uniforms for one employee per week.	Aramark Cincinnati Representative	5.50	1	NONE	5.50
Cost of laundering uniforms for one employee per week.	Aramark Cincinnati Representative	3.75	1	NONE	3.75
Cost of clear indirect vent goggles	Lab Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	6.00	1	NONE	6.00
Cost of clear lens safety glasses	Lab Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	5.00	1	NONE	5.00
Cost of grey lens safety glasses	Lab Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	5.00	1	NONE	5.00
Cost of lined nitrile gloves	Ansell Sol-Vex Flock Lined Nitrile Gloves; Lab Safety Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	2.50	1	NONE	2.50
Cost of powder surgical nitrile gloves	N-Dex 4-mil powdered disposable Nitrile Lab Gloves; Lab Safety Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	0.24	1	NONE	0.24

Table IX–14.—Unit Costs Applied in OSHA's Preliminary Analysis of the Proposed Standard—Continued

Cost description	Basis	Base cost	Escalation factor (October 2003 basis)	Index used for price escalation	Unit cost
Cost of rough PVC gloves	BEST Super Flex PVC-gloves Coated Gloves; Lab Safety Supply Catalog 2003, PO Box 1368, Janesville, WI 53547–1368.	4.10	1	NONE	4.10
Unit cost of change rooms per employee.	Based upon Means Square Foot Costs, 1989.	856.00	1.4742	CPI—All items	1,261.92
Cost per shower head	Based upon Means Square Foot Costs, 1989.	3,590.00	1.4742	CPI—All items	5,292.39
Cost per hand washing facility	Glacier Bay 4 in Chrome Two Handle Bar Faucet, 40 in x 24In. White Double Bowl Utility Tub, 505 E. Kemper Rd., Cincinnati, OH 45246—Estimated Installation Cost.	500.00	1	NONE	500.00
Variable cost per shower (soap, clean towel, water, etc.).	Estimate	0.50	1	NONE	0.50
Variable cost per hand washing facility (roll paper towels, liquid soap, water).	Kimberly-Clark OnePak Dispenser, WINDSOFT Bleached White Paper Roll Towels; The Betty Mills Company, 60 East 3rd Ave, Ste 201, San Mateo, CA 94401 (2003).	0.06	1	NONE	0.06
Unit cost of HEPA vacuums	CONSAD (1993) base price is 1991	1,580.00	1.4742	CPI—All items	2,329.24
Unit cost of HEPA vacuum replacement filters.	CONSAD (1993) base price is 1991	212.00	1.4742	CPI—All items	312.53
Unit cost of garbage bags and disposal	Estimate—Including RCRA disposal	500.00	1	NONE	500.00
Full cost of a comprehensive medical exam.	1994 Quote from two hospitals. Bethesda Care, Cincinnati, OH and Abington Memorial Hospital, Willow Grove, PA.	282.00	1.4211	CPI—Medical Care Services.	400.76
Full cost of a limited medical exam	2003 cost of physical exams in Maryland (as directed by OSHA)..	125.00	1	NONE	125.00
Cost of additional medical testing after exam results are abnormal.	Estimated to be equal to cost of limited medical exam.	150.00	1.4211	CPI—Medical Care Services.	213.17
Cost of a partial comprehensive medical exam.	1994 Quote from two hospitals. Bethesda Care, Cincinnati, OH and Abington Memorial Hospital, Willow Grove, PA—Estimated half of comprehensive and/or limited exam cost.	141.00	1.4211	CPI—Medical Care Services.	200.38
Cost of a partial medical exam	1994 Quote from two hospitals. Bethesda Care, Cincinnati, OH and Abington Memorial Hospital, Willow Grove, PA—Estimated half of comprehensive and/or limited exam cost.	75.00	1.4211	CPI—Medical Care Services.	106.59
Cost per employee for training aids and materials.	Estimate	2.00	1	NONE	2.00
Cost per employee for computer file space.	Estimate	1.00	1	NONE	1.00
Cost of Medical History Questionnaire	OSHA. Preliminary Regulatory Impact and Regulatory Flexibility Analysis of the Proposed Respiratory Protection Standard, 1994.	25	1.4211	CPI—Medical Care Services.	35.53
Cost of Medical Exam for Respirator Use.	OSHA. Preliminary Regulatory Impact and Regulatory Flexibility Analysis of the Proposed Respiratory Protection Standard. 1994.	75	1.4211	CPI—Medical Care Services.	106.58
Cost of Mop and Bucket	The Home Depot. Contico, 35qt Mop Bucket and Wringer. Wilen, 16oz Cotton Cut-End Mop.	62.92	1	NONE	62.92
Cost of Mop	The Home Depot. Wilen, 16oz Cotton Cut-End Mop.	62.92	1	NONE	62.92
Cost of Mobile Shower Unit (construction).	Ameri-can Engineering. Basic 828 Decontamination Trailer. 2003. 15886 Michigan Road. Argos, IN 46501.	42,960	1	NONE	42,960
Cost of Change Area per employee (construction).	Estimate	720	1	NONE	300

Source: U.S. Dept. of Labor, OSHA, Office of Regulatory Analysis, based on IT, 2004, Ex. 35–390.

Federal Rules That May Duplicate, Overlap, or Conflict With the Proposed Rules

OSHA's SBREFA panel for this rule suggested that OSHA address a number of possible overlapping or conflicting rules: EPA's Maximum Achievable Control Technology (MACT) standard for chromium electroplaters; EPA's standards under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) for Chromium Copper Arsenate (CCA) applicators; and state use of OSHA PELs for setting fenceline air quality standards. The Panel was also concerned that, in some cases other OSHA standards might overlap and be sufficient to assure that a new proposed standard would not be needed, or that some of the proposed standard's provisions might not be needed.

OSHA has discussed EPA's MACT standard with EPA. The standards are not duplicative or conflicting. The rules are not duplicative because they have different goals—environmental protection and protection against occupation exposure. It is quite possible, as many electroplaters are now doing, to achieve environmental protection goals without achieving occupational protection goals. The regulations are not conflicting because there exist controls that can achieve both goals without interfering with one another. However, it is possible that meeting the proposed OSHA standard would cause someone to incur additional costs for the MACT standard. If an employer has to make major changes to install LEV, this could result in significant expenses to meet EPA requirements not accounted for in OSHA's cost analysis. OSHA believes that chromium electroplaters can generally meet a PEL of 1 $\mu g/m^3$ without such major changes, and has not included costs. This issue is discussed in detail in Chapter 2 of the full PEA. However, OSHA welcomes comment on this issue.

OSHA examined the potential problem of overlapping jurisdiction for CCA applicators, and found that there would indeed be overlapping jurisdiction. For this proposed rule, OSHA had excluded CCA applicators from the scope of the coverage of the proposed rule. OSHA has been unable to find a case where a state, as a matter of law, bases fenceline standards on OSHA PELs. OSHA notes that the OSHA PEL is designed to addresses the risks associated with life long occupational exposure only. OSHA welcomes comment on this issue.

OSHA has also examined other OSHA standards, and where standards are overlapping, referred to them by reference in the proposed standard. Existing OSHA standards that may duplicate the proposed provisions in some respect include the standards addressing respiratory protection (29 CFR 1910.134); hazard communication (29 CFR 1910.1200); access to medical and exposure records (29 CFR 1910.1020); general requirements for personal protective equipment in general industry (29 CFR 1910.132), construction (29 CFR 1926.95), and shipyards (29 CFR 1915.152); and sanitation in general industry (29 CFR 1910.141), construction (29 CFR 1926.51), and shipyards (29 CFR 1915.97).

Regulatory Alternatives

This section discusses various alternatives to the proposed standard that OSHA is considering, with an emphasis on the those suggested by the SBREFA Panel as potentially alleviating impacts on small firms. (A discussion on the costs of some if these alternatives to OSHA's proposed regulatory requirements for the hexavalent chromium standard can be found in **Section III.2 Costs of Regulatory Alternatives** in the final report by OSHA's contractor, IT (IT, 2004). In the IT report, Tables III.42–III.51, costs are analyzed by regulatory alternative and major industry sector at discount rates of 7 percent and 3 percent).

Scope: The proposed standard covers exposure to all types of Cr(VI) compounds in general industry, construction, and shipyard. Cement work in construction is excluded.

OSHA considered the Panel recommendation that sectors where there is little or no known exposure to Cr(VI) be excluded from the scope of the standard. OSHA has preliminarily decided against this option. The costs for such sectors are relatively small—probably even smaller than OSHA has estimated because OSHA did not assume that any industry would use objective data to demonstrate that initial assessment was not needed. However, it is possible that changes in technology and production processes could change the exposure of employees in what are currently low exposure industries. If this happens, OSHA would need to issue a new standard to address the situation. As a result, OSHA is reluctant to exempt industries from the scope of the standard.

As stated above, the proposed standard does not cover cement work in construction. OSHA's preliminary assessment of the data indicates that the primary exposure to cement workers is dermal contact that can lead to irritant or contact allergic dermatitis. Current information indicates that the exposures in wet cement work in construction are well below 0.25 $\mu g/^3$. Moreover, unlike other exposures in construction, general industry or shipyards, exposures from cement work are most likely to be solely from dermal contact. There is little potential for airborne exposures and unlikely to be any in the future, as Cr(VI) appears in wet cement in only minute quantities naturally. Cement work also is found in the general industry setting, however the data there indicate that, because of the volume of cement involved and the nature of the work, airborne exposures are likely to be slightly higher, with 3–5% of the exposures being greater than 0.25 $\mu g/m^3$. Given these factors, the proposed standard excludes cement work in construction. OSHA has made a preliminary determination that addressing the dermal hazards from these exposures to Cr(VI) through guidance materials and enforcement of existing personal protective equipment and hygiene standards may be a more effective approach. Such guidance materials would include recommendations for specific work practices and personal protective equipment for cement work in construction.

OSHA's analysis suggests that there are 2,093 to 10,463 cases of dermatitis among cement workers annually. Using a cost of illness (COI) approach, avoiding 95 percent of these dermatoses would be valued at $2.5 to $12.6 million annually, and avoiding 50 percent of these dermatoses would be valued $1.3 million to $6.6 million annually.

The costs of including wet cement would depend on what requirements were applied to wet cement workers. OSHA estimates that adding wet cement to the scope of the standard would have costs of $33 million per year. The cost of addressing the problem through existing standards could range from $80 to $300 million per year. OSHA considered the SBREFA Panel recommendation that sectors where there is little or no known exposure to Cr(VI) be excluded from the scope of the standard. OSHA has preliminarily decided against this option. The costs for such sectors are relatively small—probably even smaller than OSHA has estimated because OSHA did not assume that any industry would use objective data to demonstrate that initial assessment was not needed. Beyond the initial exposure assessment (required only in general industry), very little would be required in workplaces where Cr(VI) exposures are below the PEL and no hazard is present from skin or eye

contact with Cr(VI). Additional requirements would generally be limited to housekeeping (in general industry) and hazard communication (warning labels on containers of Cr(VI)-contaminated materials that are consigned for disposal, training regarding the Cr(VI) standard). Where exposures in general industry exceed the Action Level, periodic monitoring would also be required. However, it is possible that changes in technology and production processes could change the exposure of employees in what are currently low exposure industries. If this happens, OSHA would need to issue a new standard to address the situation. As a result, OSHA is reluctant to exempt industries from the scope of the standard.

PELS: Section F of this preamble summary presented data on the costs and benefits of alternative PELS for all industries. The full PEA contains detailed data on the impacts of small firms at each level of PEL.

The SBREFA Panel also suggested alternatives to a uniform PEL across all industries and exposures. The Panel recommended that OSHA consider alternative approaches to industries that are intermittent users of Cr(VI). OSHA has preliminarily adopted the concept of permitting employers with intermittent exposures to meet the requirements of the standard using respirators rather than engineering controls. This approach has been used in other standards and does not require workers to routinely wear respirators.

The SBREFA Panel also recommended considering Separate Engineering Control Airborne Limits (SECALs). OSHA has preliminarily not adopted this approach because OSHA does not believe it would serve workers or small businesses well. If an approach which requires a significant number of workers to wear respirators on a regular basis were to be adopted, that approach would result in many workers wearing respirators with the associated risks, and in setting a lower PEL in accord with the QRA's estimate that there is significant risk at PELS lower than one.

The SBREFA Panel also suggested that OSHA consider different PELs for different Cr(VI) compounds leading to exposure to Cr(VI). This issue is fully discussed in the QRA. Here, it will only be noted that this would suggest lower PELs than OSHA is setting in at least some industries, and thus potentially increase impacts on small businesses.

Special Approaches to the Shipyard and Construction Industries: The SBREFA Panel was concerned that changing work conditions in the shipyard and construction industry would make it difficult to apply some of the provisions that OSHA suggested at the time of the Panel. OSHA has preliminarily decided to change its approach in these sectors. OSHA is proposing 3 separate standards, one for general industry, one for construction, and one for shipyards. In shipyard and construction, OSHA will not require exposure monitoring of any kind; will not have an action level; will require medical surveillance only for persons with signs and symptoms; and will not require regulated areas. However, employers must still meet the PEL with engineering controls and work practices where feasible.

This approach reduces the specification oriented aspects of the standard in these sectors, but may make it difficult for employers to determine how to comply with the standard. OSHA is considering a more specification oriented approach, similar to that used in the asbestos in construction standard, and in ''control banding'' approaches used abroad. Such an approach would require OSHA to specify what controls would need to be used in various circumstances, and employers using such controls would be considered to be in compliance with the standard. OSHA does not have the information at this time to develop or cost such an approach. OSHA welcomes comments on how it might develop such an approach.

Timing of the Standard: The SBREFA Panel also recommended considering a multi-year phase in of the standard. OSHA is examining and soliciting comment on this issue. Such a phase-in would have several advantages from a viewpoint of impacts on small businesses. First, it would reduce the one time initial costs of the standard by spreading them out over time. This would be particularly useful for small businesses that have trouble borrowing large amounts of capital in a single year. A phase-in would also be useful in the electroplating sector by allowing employers to coordinate their environmental and occupational safety and health control strategies to minimize potential costs. A differential phase-in for smaller firms would also aid very small firms by allowing them to gain from the control experience of larger firms. However a phase-in would also postpone the benefits of the standard.

SBREFA Panel

Table IX–15 lists all of the SBREFA Panel recommendations and notes OSHA responses to these recommendations.

TABLE IX–15.—SBREFA PANEL RECOMMENDATIONS AND OSHA RESPONSES

SBREFA panel recommendation	OSHA response
The Panel recommends that, as time permits, OSHA revise its economic and regulatory flexibility analyses as appropriate to reflect the SERs' comments on underestimation of costs and that the Agency compare the OSHA revised estimates to alternative estimates provided and methodologies suggested by the SERs. For those SER estimates and methodological suggestions that OSHA does not adopt, the Panel recommends that OSHA explain its reasons for preferring an alternative estimate and solicit comment on the issue.	OSHA has extensively reviewed its costs estimates, and changed many of them in response to SER comments and solicits comments on these revised cost estimates. A few examples of OSHA's cost changes are given in the responses to specific issues, below (*e.g.*, medical exams, training and familiarization).
The Panel recommends that, to the extent time permits, OSHA should carefully consider the ability of each potentially affected industry to meet any proposed PEL for CR(VI) and solicit comment on the costs and technological feasibility of the PEL.	The PEA reflects OSHA's judgment on technological feasibility and includes responses to specific issues raised by the Panel and SERs. OSHA will solicit comment on the accuracy and reasonableness of these judgments.
The Panel recommends that OSHA carefully review the basis for its estimated medical surveillance compliance costs, consider these concerns raised by the SERs, and ensure that its estimates are revised, as appropriate and time permits, to fully reflect the costs likely to be incurred by potentially affected establishments.	OSHA has increased the estimated time for a limited medical exam from 1.5 hours to 3 hours and solicits comment on all other cost projections for medical surveillance. See **Chapter IV OF THE PEA; COSTS OF COMPLIANCE, COSTS BY PROVISION**—*Medical Surveillance*, for details of OSHA's unit costs for medical surveillance.

TABLE IX–15.—SBREFA PANEL RECOMMENDATIONS AND OSHA RESPONSES—Continued

SBREFA panel recommendation	OSHA response
The Panel recommends that, as time permits, OSHA consider alternatives that would alleviate the need for extensive monitoring on construction sites, and solicit comment on this issue. If OSHA does not adopt such alternatives, then OSHA should consider increasing the estimated costs of such monitoring in construction, and solicit comment on the costs of monitoring.	OSHA revised the standard to relieve Construction and Shipyards from requirements for exposure assessment; for General Industry, OSHA believes that its unit cost estimates are realistic but will raise that as an issue. See **CHAPTER IV OF THE PEA: COSTS OF COMPLIANCE, COSTS BY PROVISION**—*Exposure Monitoring (Initial and Periodic*), for details of OSHA's unit costs for exposure monitoring in general industry.
The Panel recommends that OSHA carefully review the basis for its estimated hygiene compliance costs, consider the concerns raised by the SERs, and, to the extent time permits, ensure that its estimates are revised, as appropriate, to fully reflect the costs likely to be incurred by potentially affected establishments.	OSHA's proposed standard will permit hand washing as a hygiene option; OSHA's analysis will also reflect, where data confirm, any cost premium related to handling contaminated waste water or laundry, or where uncertainty exists, the issue will be raised.
The Panel recommends that OSHA examine and solicit comment on this issue [possible understates in the costs of regulated areas].	OSHA has recognized costs for training and familiarization to cover a better understanding of the costs of regulated areas, and solicit comment on the issue. See **CHAPTER IV OF THE PEA; COSTS OF COMPLIANCE, COSTS BY PROVISION**—*Communication of Hazards to Employees—Training and Familiarization*, for details of OSHA's unit costs for this provision.
The Panel recommends that OSHA examine and solicit comment on these issues [costs of laundering PPE].	OSHA has examined and solicits comment on this issue and the cost OSHA has estimated. See **CHAPTER IV OF THE PEA; COSTS OF COMPLIANCE, COSTS BY PROVISION**—*Housekeeping, Protective Work Clothing and Equipments*, and Table IV–8 for details of OSHA's unit costs for laundering PPE and other related costs.
The Panel recommends that OSHA examine whether its cost estimates reflect the full costs of complying with the hazard communication standard.	OSHA's analysis assumes that employers will need time for familiarization with the standard, training on the standard, and increased initial supervision.
The Panel recommends that OSHA thoroughly review the economic impacts of compliance with a proposed Cr(VI) standard and develop more detailed feasibility analyses where appropriate. The Panel also recommends that OSHA, to the extent permitted by time and the availability of economic data, reexamine its estimates of profits and revenues in light of SER comments, and update economic data to better reflect recent changes in the economic status of the affected industries, consistent with its statutory mandate. The Panel also recommends that OSHA examine, to the extent feasible with the time available, the possibility that users will substitute non-Cr(VI) products for Cr(VI) products. The Panel recommends that OSHA solicit comment on the extent to which foreign competition may or may not impact what is feasible for the industries affected by this rule.	OSHA has reviewed and revised many of its revenue and profit estimates in the light of specific SER comments. Examples of application groups with revised revenue and profit estimates include Group 4, Chromate Production; Group 5, Chromate Pigment Producers; and Group 17, Chromium Dye Producers. However, OSHA has not updated revenue and profit impacts across the board—OSHA estimates of costs, revenues, and profits require consistent data sets which are not yet available for more recent years. OSHA's continues to examine, and will solicit comment on this issue.
The Panel recommends that OSHA consider and solicit comments on selective exemption of some industries from the proposed standard, especially those industries whose inclusion is not supported by the industry-specific data or in which inhalation exposure to Cr(VI) is minimal.	OSHA is reluctant to exempt industries where exposures are minimal because changes in technology could change exposures in the future. However, OSHA is seeking comment on the issue of the scope of the standard and data that would support not covering certain sectors.
The Panel recommends that OSHA exempt applicators of CCA given that they are already regulated by EPA as pesticide applicators under FIFRA. In addition, OSHA should clarify and seek comment as to why users of CCA-treated wood should be covered under the Cr(VI) proposal given that the use of CCA-treated wood was previously excluded by OSHA in its standard for inorganic arsenic.	OSHA has decided to exempt applicators of CCA in this proposal.
The Panel recommends that OSHA clearly explain the way that Cr(VI) exposure and risk for the worker cohort studies used in the quantitative risk assessment were calculated, and should consider and seek comment as to whether the major assumptions used in these calculations are reasonable.	The Quantitative Risk Assessment section of the Preamble addresses this issue in detail, and OSHA is seeking comments on this issue.
The Panel recommends that OSHA consider the available information on reduction of inhaled Cr(VI) to Cr(III) in the body, to determine whether exposures below a threshold concentration can be shown not to cause the genetic alterations that are believed to cause cancer. In addition, OSHA should review epidemiological analyses relevant to the question of threshold dose, to determine whether such a dose is identifiable from the available human data. OSHA should further consider and seek comment on these findings in relation to the risk assessment and the proposed PEL, allowing for a higher PEL than those presented in the draft standard if the risk assessment so indicates.	The Quantitative Risk Assessment of this Preamble addresses the issue of possible threshold effects and OSHA is seeking comments on the issue.

TABLE IX–15.—SBREFA PANEL RECOMMENDATIONS AND OSHA RESPONSES—Continued

SBREFA panel recommendation	OSHA response
The Panel recommends that OSHA should clarify the meaning of the projected lung cancer risk estimates used to support the proposed standard. In particular, OSHA should explain these estimates, which are based on a working lifetime of 45 years' exposure at the highest allowable Cr(VI) concentration, and, where appropriate, note projected excess cancers that may result from shorter periods of occupational Cr(VI) exposure.	OSHA is required by law to set health standards so that they avoid significant risk over a working lifetime. Both in the QRA and in the Benefits Chapter of the PEA, OSHA has examined alternative exposure scenarios. See **VII. Preliminary Quantitative Risk Assessment** in the Preamble and **CHAPTER VI of the PEA; BENEFITS and NET BENEFITS,** *Lung Cancers Avoided* in this PEA.
The Panel recommends that OSHA solicit information to better characterize the exposure patterns and Cr(VI) compounds encountered in the maritime environment, and should encourage input from marine chemists at appropriate points in the rulemaking.	OSHA has added information provided by firms in the shipyard industry since the Panel meeting. (See **Chapter II** of the PEA; **PROFILE OF AFFECTED INDUSTRIES, PROCESSES, AND APPLICATIONS GROUPS, AFFECTED INDUSTRIES— *Welding*** and ***Painting*** and **Chapter III: Technological Feasibility, *Welding*** and ***Painting)***. OSHA is soliciting comment on shipyard issues and from maritime chemists.
The Panel recommends that OSHA consider the appropriateness of separate PELs for specific Cr(VI) compounds, with attention to the weight and extent of the best available scientific evidence regarding their relative carcinogenic potency.	OSHA considered this possibility and preliminarily decided against it, in part, because it would require lower PELs with many persons in respirators. OSHA is soliciting comment on this issue.
The Panel recommends that OSHA solicit information to better define construction activities likely to be above and below the PEL (for initial exposure monitoring purposes) to minimize the amount of respiratory protection that would need to be used for compliance.	OSHA has eliminated the requirement for monitoring in the construction industry. OSHA has considered a control banding approach to construction, but lacks the data to fully implement this approach, and solicits comment on the issue.
The Panel recommends that OSHA provide a better explanation of how to implement an exposure assessment program for construction activities. Also, OSHA should provide further explanation on monitoring-related topics like the selection of sampling and analytical methods, the selection of plus-or-minus 25% as a confidence interval, and the use of objective data in lieu of monitoring.	OSHA has removed the requirement for exposure monitoring in construction and shipyards. The monitoring-related topics are further discussed in the Preamble, XVII. **Summary and Explanation of the Standard.**
The Panel recommends that OSHA consider less frequent monitoring for exposures above the PEL, especially in situations where the employer has already engineered down to the lowest feasible level and is not able to maintain levels below the PEL.	OSHA has preliminarily left the monitoring frequency unchanged, but has solicited comment on the issue.
The Panel recommends that OSHA review the technologies used to reduce Cr(VI) exposure to ensure to ensure that they are available or reasonably anticipated to be available in the future.	OSHA has reviewed its technological feasibility analysis and solicited comment on it.
The Panel recommends that OSHA clarify the purpose of the prohibition on the use of employee rotation to meet the PEL and take into account the needs expressed by the SERs on the issue.	The Summary and Explanation of the Preamble explains further the prohibition on employee rotation and the methods of compliance.
The Panel recommends that OSHA clarify the methods of compliance section.	
The Panel recommends that OSHA clarify how to implement the use of regulated areas particularly for construction activities. OSHA should better explain how employers would delineate boundaries for regulated areas and should better clarify the use of respiratory protection, personal protective clothing and equipment, and hygiene facilities and practices in regulated areas.	OSHA has eliminated the requirement for regulated areas in construction and shipyards. The Summary and Explanation section of the Preamble explains the regulated area requirements in General Industry.
The Panel recommends that OSHA provide a clearer explanation of why it is necessary to remove Cr(VI)-contaminated protective clothing and wash hands prior to entering non-Cr(VI) work areas and eating, drinking or smoking and take into account lost time and costs associated with conducting such activities.	These issues are addressed in the Summary and Explanation Section of the Preamble.
The Panel recommends that OSHA clarify its definition of contaminated clothing or waste, provide evidence supporting the view that ''contaminated'' clothing presents a hazard, and better explain the special treatment of such items and why the treatment is necessary.	
The Panel recommends that OSHA clarify its definition of reasonably anticipated skin and eye contact.	OSHA has changed the rule from SBREFA draft in order to clarify when PPE is required and to assure that it is not required except where a dermal hazard exists.
The Panel recommends that OSHA clarify the circumstances under which the proposed rule would require the use of personal protective equipment to prevent dermal exposures to solutions containing Cr(VI). In particular, OSHA should reconsider the requirements for the use of dermal protection when the PEL is exceeded; consider alternatives that are more clearly risk based; and determine whether the use of very dilute Cr(VI) solutions, as used in some laboratories, requires the use of personal protective equipment..	

TABLE IX–15.—SBREFA PANEL RECOMMENDATIONS AND OSHA RESPONSES—Continued

SBREFA panel recommendation	OSHA response
The Panel recommends that OSHA provide a clearer explanation of the benefits and the need for its proposed medical surveillance provisions.	OSHA has preliminarily dropped routine medical surveillance in the shipyard and construction industries. The Preamble Summary and Explanation clarify what is required of medical surveillance, and the extent to which the same medical examination can be used to meet the requirements of different standards.
The Panel recommends that OSHA provide a clearer guidance as to which employees are intended to be covered under the medical surveillance provisions and, in particular, how the standard is intended to cover employees who work for several different employers during the course of a year.	
The Panel recommends that OSHA clarify the qualifications necessary to provide a medical examination (including what knowledge of Cr(VI) is necessary) and what the elements of such a medical examination should be.	
The Panel recommends that OSHA design the medical surveillance provisions to be consistent with existing OSHA standards (*e.g.*, lead and arsenic) wherever possible, in order to minimize the need for duplicative medical examinations. The Panel also recommends that OSHA clarify that differences in medical surveillance requirements that may be unavoidable across OSHA standards nevertheless often will not require completely separate medical examinations.	
With respect to the EPA electroplating standards, the Panel recommends that OSHA examine whether important costs have been omitted, seek to develop alternatives that minimize these costs, and seek comment on the issue.	OSHA discusses the impact of EPA's electroplating standard in the PEA, (See **Chapter II: Technological Feasibility, Electroplating** and **Chapter VIII: Environmental Impacts)** and seeks comments on this issue.
With respect to possible dual jurisdiction with FIFRA, the Panel recommends that OSHA consider dropping CCA applicators from the scope of the rule, and seek comment on this issue.	OSHA preliminarily has decided to exclude CCA applicators from the scope of the standard.
With respect to the issue of using OSHA PELs as a basis for fenceline standards, the Panel recommends that OSHA make clear the purpose of its PELs, and explain that they are not developed or examined in terms of their validity as a basis for air quality standards.	OSHA solicits comment on the ''fence line'' standard issue.
The Panel recommends that OSHA examine whether existing standards are adequate to cover occupational exposure to Cr(VI), and, if not, develop the Cr(VI) standard in such a way as to eliminate duplicative and overlapping efforts on the part of employers.	OSHA has preliminarily determined that, except for CCA applicators and wet cement workers, other standards cannot provide the worker protection needed, but has sought to avoid duplication of effort between standards.
The Panel recommends that OSHA consider the scientific evidence in favor of a higher PEL, analyze the costs and economic impacts of a PEL of 20 or greater, and solicit comment on this option.	OSHA has included an analysis of the costs and benefits of a PEL of 20 in this Preamble summary, and has a full analysis of this option in the PEA.
The Panel recommends that OSHA carefully examine the entire issue of intermittent exposures, consider options that can alleviate the burden on such firms while meeting the requirements of the OSH Act, and solicit comment on such options.	OSHA preliminarily determined that intermittent users need not use engineering controls to assure compliance with the PEL.
Some SERs argued that some Cr(VI) compounds offer lesser risks of cancer than others, and should be subject to different PELs. The Panel recommends that OSHA consider these arguments and seek comment on the issue.	OSHA has preliminarily determined that all Cr(VI) compounds should have the same PEL, but seeks comment on the issue.
The Panel recommends that OSHA continue to exempt wet cement from the scope of the standard, and that if OSHA seeks comment on this option, OSHA should note the Panel's recommendation and the reasons for the recommendation. The Panel also recommends that OSHA seek ways of adapting the standard better to the dynamic working conditions of the construction industry, examine the extent to which Cr(VI) exposures are already covered by other standards, and seek comment on these issues. The Panel also recommends that OSHA consider the alternative of developing a construction standard in a separate rulemaking.	OSHA has preliminarily determined to exempt wet cement from the scope of the standard, but has sought comment on the issue. OSHA has made a number of changes to the construction standard in this proposal, including eliminating the exposure assessment requirements, the regulated area requirement, and the action level. OSHA seeks comment on its new approach.
The Panel recommends that OSHA consider, and solicit comment on, approaches to their special problems; that OSHA consider the possibility of making the maritime proposed standard more similar to the construction draft standard, or consider the alternative of developing a maritime standard in a separate rulemaking.	OSHA has made a number of changes to the shipyard standard in this proposal, including eliminating the exposure assessment requirements, the regulated area requirement, and the action level. OSHA has sought comment on its new approach.
The Panel recommends that OSHA consider and seek comment on multi-year phase-in alternatives.	This option is discussed in the regulatory alternatives section of the PEA, and OSHA is seeking comments on this alternative.
The Panel recommends that OSHA better explain the action level, including its role in ensuring workers are protected.	OSHA has eliminated the action level in the construction and shipyard standards, and explains its role in the General Industry in the Summary and Explanation of the Preamble.
The Panel recommends that OSHA consider the use of SECALs and solicit comment on whether and in what industries they are appropriate using the Cadmium standard as a model.	OSHA has preliminarily determined not to use SECALs, but solicits comments on this issue.

X. OMB Review Under the Paperwork Reduction Act of 1995

The proposed standard for chromium (VI) contains collections of information (paperwork) that are subject to review by the Office of Management and Budget (OMB) under the Paperwork Reduction Act of 1995 (PRA95), 44 U.S.C. 3501 et seq, and its regulation at 5 CFR Part 1320. PRA 95 defines collection of information to mean, ''the obtaining, causing to be obtained, soliciting, or requiring the disclosure to third parties or the public of facts or opinions by or for an agency regardless of form or format'' [44 U.S.C. § 3502(3)(A)].

The title, description of the need for and proposed use of the information, summary of the collections of information, description of respondents, and frequency of response of the information collection are described below with an estimate of the annual cost and reporting burden has required by § 1320.5(a) (1)(iv) and § 1320.8(d)(2). The reporting burden includes the time for reviewing instructions, gathering and maintaining the data needed, and completing and reviewing the collection of information.

OSHA invites comments on whether each proposed collection of information:

(1) Ensures that the collection of information is necessary for the proper performance of the functions of the agency, including whether the information will have practical utility;

(2) Estimates the projected burden accurately, including the validity of the methodology and assumptions used;

(3) Enhances the quality, utility, and clarity of the information to be collected; and

(4) Minimizes the burden of the collection of information on those who are to respond, including through the use of appropriate automated, electronic, mechanical, or other technological collection techniques or other forms of information technology, *e.g.*, permitting electronic submissions of responses.

Title: Chromium (VI) Standard for General Industry (§ 1910.1026), Shipyards (§ 1915.1026); and Construction (§ 1926.1126)

Description: The proposed Cr(VI) standard is an occupational safety and health standard's information collection requirements are essential components that will assist both employers and their employees in identifying exposures as well as identifying means to take to reduce or eliminate Cr(VI) overexposures.

Summary of the Collections of Information:

• **1910.1026(d)—Exposure Assessment**

Paragraph (d)(5) of this section requires the employer to notify employees of their exposure monitoring results within 15 working days after the receipt for the exposure monitoring performed in this section (§ 1910.1026(d)(2) Initial Exposure Monitoring, § 1910.1026(d)(3) Periodic Monitoring, and § 1910.1026 (d)(4) Additional Monitoring).

Employers may notify each affected employee individually in writing of the results or by posting the exposure-monitoring results in an appropriate location that is accessible to all affected employees. If the exposure monitoring results indicate that employee exposure is above the PEL, the employer must include in the written notification the corrective action being taken to reduce employee exposure to or below the PEL.

• **1910.1026(g), 1915.1026(e), 1926.1126(e)—Respiratory Protection**

Paragraph (g)(2) in the general industry section, and paragraph (e)(2) in the shipyards and construction sections require the employer to institute a respiratory protection program in accordance with 29 CFR 1910.134. The Respiratory Protection Standard's (§ 1910.134) information collection requirements require employers to: Develop a written respirator program; conduct employee medical evaluations and provide follow-up medical evaluations to determine the employee's ability to use a respirator; provide the physician or other licensed health care professional with information about the employee's respirator and the conditions under which the employee will use the respirator; and administer fit-tests for employees who will use negative or positive-pressure, tight-fitting facepieces.

• **1910.1026(h), 1915.1026(f), 1926.1126(f)—Protective Work Clothing and Equipment**

Paragraph (h)(3)(iii) in the general industry section and (f)(3)(iii) in the shipyards and construction sections require the employer to inform any person who launders or cleans protective clothing or equipment contaminated with chromium (VI) of the potentially harmful effects of exposure to chromium (VI) and that the clothing and equipment should be laundered or cleaned in a manner that minimizes skin or eye contact with chromium (VI) and effectively prevents the release of airborne chromium (VI) in excess of the PEL.

• **1910.1026(k), 1915.1026(h), and 1926.1126(h)—Medical Surveillance**

Paragraphs (k)(4) in the general industry section and (h)(4) in the shipyards and construction sections require the employer to provide the examining PLHCP with a copy of the standard. In addition, for each employee receiving a medical examination, the employer must provide the following information:

1. A description of the affected employee's former, current, and anticipated duties as they relate to the employee's occupational exposure to chromium (VI);

2. The employee's former, current and anticipated levels of occupational exposure to chromium;

3. A description of any personal protective equipment used or to be used by the employee, including when and for how long the employee has used that equipment; and,

4. Information from records of employment-related medical examinations previously provided to the affected employee currently within the control of the employer.

Paragraphs (k)(5) in the general industry section, and (h)(5) in shipyards and construction sections require the employer to obtain a written medical opinion from the PLHCP, within 30 days for each medical examination performed on each employee. The employer must provide the employee with a copy the PLHCPs written medical opinion within two weeks of receipt. This written opinion must contain the following information:

1. The PLHCP's opinion as to whether the employee has any detected medical condition(s) that would place the employee at increased risk of material impairment to health from further exposure to chromium (VI);

2. Any recommended limitations upon the employee's exposure to chromium (VI) or upon the use of personal protective equipment such as respirators;

3. A statement that the PLHCP has explained to the employee the results of the medical examination, including any medical conditions related to chromium (VI) exposure that require further evaluation or treatment, and any special provisions for use of protective clothing or equipment.

• **1910.1026(l), 1915.1026(i), and 1926.1126(i)—Communication of Chromium (VI) Hazards to Employees**

Paragraph (l)(4) of the general industry section, and (i)(3) of the shipyards and construction sections require that the employer provide

training for all employees who are exposed to airborne chromium (VI), or who have skin or eye contact with chromium (VI). Employers must maintain a record of the training provided. Also employers must provide initial training prior to or at the time of initial assignment to a job involving potential exposure to chromium (VI). However, employers do not need to provide training to a new employee, if they can demonstrate that a new employee has received training within the last 12 months that addresses the elements specified in the paragraph and that the employee can demonstrate knowledge of those elements. Employers must provide training that is understandable to the employee and must ensure that each employee can demonstrate knowledge of at least the following:

1. The health hazards associated with chromium (VI) exposure;

2. The location, manner of use, and release of chromium (VI) in the workplace and the specific nature of operations that could result in exposure to chromium (VI), especially above the PEL;

3. The engineering controls and work practices associated with the employee's job assignment;

4. The purpose, proper selection, fitting, proper use, and limitations of respirators and protective clothing;

5. Emergency procedures;

6. Measures employees can take to protect themselves from exposure to chromium (VI), including modification of personal hygiene and habits such as smoking;

7. The purpose and a description of the medical surveillance program required by paragraph (k) of the general industry section and paragraph (h) of shipyards and construction sections;

8. The contents of the standard; and

9. The employee s rights of access to records under 29 CFR 1910.1020(g).

• **1910.1026(m), 1915.1026(j), and 1926.1126(j)—Recordkeeping**

Paragraph (m)(1) of the general industry section requires that employers maintain an accurate record of all employee exposure-monitoring records required in paragraph (d) of this section. The record must include at least the following information:

1. The date of measurement for each sample taken;

2. The operation involving exposure to chromium (VI) that is being monitored;

3. Sampling and analytical methods used and evidence of their accuracy;

4. Number, duration, and the results of samples taken;

5. Type of personal protective equipment, such as respirators worn; and,

6. The name, social security number, and job classification of all employees represented by the monitoring, indicating which employees were actually monitored.

Employers must maintain and make available employee exposure monitoring records in accordance with 29 CFR 1910.1020.

Paragraph (m)(2) of the general industry section requires employers who rely on historical monitoring data to maintain a record of historical data. The record must include information that reflects the following conditions:

1. The data were collected using methods that meet the accuracy requirements of paragraph (d)(6) of the general industry section;

2. The processes and work practices that were in use when the historical monitoring data were obtained are essentially the same as those to be used during the job for which initial monitoring will not be performed;

3. The characteristics of the chromium (IV) containing material being handled when the historical monitoring data were obtained are the same as those on the job for which initial monitoring will not be performed;

4. Environmental conditions prevailing when the historical monitoring data were obtained are the same as those on the job for which initial monitoring will not be performed; and

5. Other data relevant to the operations, materials, processing, or employee exposures covered by the exception.

This record must be maintained and must be made available in accordance with 29 CFR 1910.1020.

Paragraph (m)(3) of the general industry section requires employers who rely on objective data to satisfy initial monitoring requirements to establish and maintain an accurate record of the objective data relied upon. The record must include at least the following information:

1. The chromium (VI)-containing material in question;

2. The source of the objective data;

3. The testing protocol and results of testing, or analysis of the material for the release of chromium (VI);

4. A description of the operation exempted from initial monitoring and how the data support the exemption; and

5. Other data relevant to the operations, materials, processing or employee exposures covered by the exemption.

Employers must maintain this record for the duration of the employer's reliance upon such objective data and must make such records available in accordance with 29 CFR 1910.1020.

Paragraph (m)(4) of the general industry section, and paragraph (j)(1) of the shipyard and construction sections, require employers to establish and maintain an accurate record for each employee covered by medical surveillance under paragraph (k) of the general industry section, or paragraph (h) of the shipyard and construction sections. This record must include the following information about the employee:

1. Name and social security number;

2. A copy of the PLHCP's written opinions as required by paragraph (k)(5) of the general industry section, or paragraph (h)(5) for the shipyard and construction sections;

3. A copy of the information provided to the PLHCP as required by paragraph (k)(4) of the general industry section, or (h)(4) in the shipyards and construction sections; Employers must ensure that medical records are maintained and made available in accordance with 29 CFR 1910.1020.

Paragraph (m)(5) of the general industry section and paragraph (j)(2) of the shipyards and construction sections require employers to prepare a record at the completion of training that indicates the identity of the individuals trained and the date the training was completed. This record must be maintained for three years after the completion of training. The employer must provide to the Assistant Secretary or the Director, upon request, all materials relating to employee information and training.

Respondents: Employers in general industry, shipyards or construction whose employees work in jobs where there is a potential for chromium (VI) exposure (38,391 businesses).

Frequency of Response: Frequency of response varies depending on the specific collection of information.

Average Time Per Response: Varies from 5 minutes (.08 hour) for the employer to provide a copy of the written physician's opinion to the employee, to 12 hours to conduct exposure monitoring.

Total burden hours: 696,659.

Costs: (purchase of capital/startup costs): $30,793,697.

The Agency has submitted a copy of the information collection request to OMB for its review and approval. Interested persons may submit comments regarding the burden

estimates or other aspects of the information collection request to the OSHA Docket Office, Docket No. H054A, Occupational Safety and Health Administration, Room N–2625, 200 Constitution Avenue, NW., Washington, DC 20210, and to the Office of Information and Regulatory Affairs, Office of Management and Budget, New Executive Office Building, Room 10235, 725 17th Street, NW., Washington, DC 20503 (Attn: OSHA Desk Officer (RIN 1218–AB45)). Comments submitted in response to this notice will be summarized and/or included in the request for OMB approval of the final information collection request, and they will also become a matter of public record.

Copies of the referenced information collection request are available for inspection and copying in the OSHA Docket Office and will be provided to persons who request copies by telephoning Todd Owen at (202) 693–1941. For electronic copies of the chromium (VI) information collection request, contact the OSHA Web page on the Internet at *http://www.osha.gov/.*

XI. Federalism

The Agency reviewed the proposed Cr(VI) standard according to the most recent Executive Order on Federalism (Executive Order 13132, 64 FR 43225, August 10, 1999). This Executive Order requires that federal agencies, to the extent possible, refrain from limiting state policy options, consult with states before taking actions that restrict their policy options, and take such actions only when clear constitutional authority exists and the problem is of national scope. The Executive Order allows federal agencies to preempt state law only with the expressed consent of Congress; in such cases, federal agencies must limit preemption of state law to the extent possible. Under section 18 of the Occupational Safety and Health Act (the ''Act'' or ''OSH Act''), Congress expressly provides that OSHA preempt state occupational safety and health standards to the extent that the Agency promulgates a federal standard under section 6 of the Act. Accordingly, under section 18 of the Act OSHA preempts state promulgation and enforcement of requirements dealing with occupational safety and health issues covered by OSHA standards unless the state has an OSHA-approved occupational safety and health plan (i.e., is a state-plan state) [*see Gade* v. *National Solid Wastes Management Association,* 112 S. Ct. 2374 (1992)]. Therefore, with respect to states that do not have OSHA-approved plans, the Agency concludes that this proposal falls under the preemption provisions of the Act. Additionally, section 18 of the Act prohibits states without approved plans from issuing citations for violations of OSHA standards; the Agency finds that this proposed rulemaking does not expand this limitation. OSHA has authority under Executive Order 13132 to propose a Cr(VI) standard because the problems addressed by these requirements are national in scope.

As explained in section VIII of this preamble, employees face a significant risk from exposure to Cr(VI) in the workplace. These employees are exposed to Cr(VI) in general industry, construction, and shipyards. Accordingly, the proposal would establish requirements for employers in every state to protect their employees from the risks of exposure to Cr(VI). However, section 18(c)(2) of the Act permits state-plan states to develop their own requirements to deal with any special workplace problems or conditions, provided these requirements are at least as effective as the final requirements that result from this proposal.

XII. State Plans

The 26 states and territories with their own OSHA-approved occupational safety and health plans must adopt comparable provisions within six months after the Agency publishes the final hexavalent chromium standard. These states and territories are: Alaska, Arizona, California, Hawaii, Indiana, Iowa, Kentucky, Maryland, Michigan, Minnesota, Nevada, New Mexico, North Carolina, Oregon, Puerto Rico, South Carolina, Tennessee, Utah, Vermont, Virginia, Virgin Islands, Washington, and Wyoming. Connecticut, New Jersey and New York have OSHA-approved State Plans that apply to state and local government employees only. Until a state-plan state promulgates its own comparable provisions, Federal OSHA will provide the state with interim enforcement assistance, as appropriate.

XIII. Unfunded Mandates

The Agency reviewed the proposed Cr(VI) standard according to the Unfunded Mandates Reform Act of 1995 (UMRA)(2 U.S.C. 1501 *et seq.*) and Executive Order 12875. As discussed in section IX of this preamble, OSHA estimates that compliance with this proposal would require private-sector employers to expend about $223 each year. However, while this proposal establishes a federal mandate in the private sector, it is not a significant regulatory action within the meaning of section 202 of the UMRA (2 U.S.C. 1532). OSHA standards do not apply to state and local governments, except in states that have voluntarily elected to adopt an OSHA-approved state occupational safety and health plan. Consequently, the proposed provisions do not meet the definition of a ''Federal intergovernmental mandate'' [*see* section 421(5) of the UMRA (2 U.S.C. 658(5)]. Therefore, based on a review of the rulemaking record to date, the Agency believes that few, if any, of the employers affected by the proposal are state, local, and tribal governments. Therefore, the proposed Cr(VI) requirements do not impose unfunded mandates on state, local, and tribal governments.

XIV. Protecting Children From Environmental Health and Safety Risks

Executive Order 13045 requires that Federal agencies submitting covered regulatory actions to OMB's Office of Information and Regulatory Affairs (OIRA) for review pursuant to Executive Order 12866 must provide OIRA with (1) an evaluation of the environmental health or safety effects that the planned regulation may have on children, and (2) an explanation of why the planned regulation is preferable to other potentially effective and reasonably feasible alternatives considered by the agency. Executive Order 13045 defines ''covered regulatory actions'' as rules that may (1) be economically significant under Executive Order 12866 (i.e., a rulemaking that has an annual effect on the economy of $100 million or more, or would adversely effect in a material way the economy, a sector of the economy, productivity, competition, jobs, the environment, public health or safety, or state, local, or tribal governments or communities), and (2) concern an environmental health risk or safety risk that an agency has reason to believe may disproportionately affect children. In this context, the term ''environmental health risks and safety risks'' means risks to health or safety that are attributable to products or substances that children are likely to come in contact with or ingest (*e.g.*, through air, food, water, soil, product use). The proposed Cr(VI) standard is economically significant under Executive Order 12866 (*see* section IX of this preamble). However, after reviewing the proposed Cr(VI) standard, OSHA has determined that the standard would not impose environmental health or safety risks to children as set forth in Executive Order 13045. The proposed standard would require employers to limit employee exposure to Cr(VI) and take other precautions to protect employees from adverse health effects associated with exposure to Cr(VI). To

the best of OSHA's knowledge, no employees under 18 years of age work under conditions that involve exposure to Cr(VI). However, if such conditions exist, children who are exposed to Cr(VI) in the workplace would be better protected from exposure to Cr(VI) under the proposed rule than they are currently. Based on this preliminary determination, OSHA believes that the proposed Cr(VI) standard does not constitute a covered regulatory action as defined by Executive Order 13045.

XV. Environmental Impacts

The Agency reviewed the proposed Cr(VI) standard according to the National Environmental Policy Act (NEPA) of 1969 (42 U.S.C. 4321 *et seq.*), the regulations of the Council on Environmental Quality (40 CFR part 1500), and the Department of Labor's NEPA procedures (29 CFR part 11).

As a result of this review, OSHA has made a preliminary determination that the proposed Cr(VI) standard will have no impact on air, water, or soil quality; plant or animal life; the use of land or aspects of the external environment. Therefore, OSHA concludes that the proposed Cr(VI) standard would have no significant environmental impacts.

XVI. Public Participation—Notice of Hearing

OSHA encourages members of the public to participate in this rulemaking by submitting comments on the proposal, and by providing oral testimony and documentary evidence at the informal public hearing that the Agency will convene after the comment period ends. The Agency invites interested persons having knowledge of, or experience with, occupational exposure to Cr(VI) to participate in this process, and welcomes any pertinent data and cost information that will provide it with the best available evidence on which to develop the final regulatory requirements. This section describes the procedures the public must use to submit their comments to the docket in a timely manner, and to schedule an opportunity to deliver oral testimony and provide documentary evidence at informal public hearings on the proposal. Comments, notices of intention to appear, hearing testimony, and documentary evidence will be available for inspection and copying at the OSHA Docket Office. You also should read the sections above titled **DATES** and **ADDRESSES** for additional information on submitting comments, documents, and requests to the Agency for consideration in this rulemaking.

Written Comments. OSHA invites interested persons to submit written data, views, and arguments concerning this proposal. In particular, OSHA encourages interested persons to comment on the issues raised in section II of this preamble. When submitting comments, parties must follow the procedures specified above in the sections titled **DATES** and **ADDRESSES.** The comments must clearly identify the provision of the proposal you are addressing, the position taken with respect to each issue, and the basis for that position. Comments, along with supporting data and references, received by the end of the specified comment period will become part of the record, and will be available for public inspection and copying at the OSHA Docket Office.

Informal Public Hearing. Pursuant to section 6(b)(3) of the Act, members of the public will have an opportunity to provide oral testimony concerning the issues raised in this proposal at informal public hearings. The hearings will commence at 9:30 a.m. on February 1, 2005. At that time, the presiding administrative law judge (ALJ) will resolve any procedural matters relating to the proceeding. The legislative history of section 6 of the OSH Act, as well as OSHA's regulation governing public hearings (29 CFR 1911.15), establish the purpose and procedures of informal public hearings.

Although the presiding officer at such hearings is an ALJ, and questioning by interested persons is allowed on crucial issues, the proceeding is informal and legislative in purpose. Therefore, the hearing provides interested persons with an opportunity to make effective and expeditious oral presentations in the absence of procedural restraints or rigid procedures that could impede or protract the rulemaking process. The hearing is an informal administrative proceeding, rather than adjudicative one in which the technical rules of evidence would apply; its primary purpose is to gather and clarify information. The regulations that govern public hearings, and the pre-hearing guidelines issued for this hearing, will ensure participants fairness and due process, and also will facilitate the development of a clear, accurate, and complete record. Accordingly, application of these rules and guidelines will be such that questions of relevance, procedure, and participation generally will favor development of the record. Conduct of the hearing will conform to the provisions of 29 CFR part 1911, ''Rules of Procedure for Promulgating, Modifying, or Revoking Occupational Safety and Health Standards.''

Although the ALJs who preside over these hearings make no decision or recommendation on the merits of OSHA's proposal, they do have the responsibility and authority to ensure that the hearing progresses at a reasonable pace and in an orderly manner. To ensure that interested persons receive a full and fair informal hearing as specified by 29 CFR part 1911, the ALJ has the authority and power to: Regulate the course of the proceedings; dispose of procedural requests, objections, and comparable matters; confine the presentations to matters pertinent to the issues raised; use appropriate means to regulate the conduct of the parties who are present at the hearing; question witnesses, and permit others to question witnesses; and limit the time for such questioning.

At the close of the hearing, the ALJ will establish a post-hearing comment period for parties who participated in the hearing. During the first part of this period, the participants may submit additional data and information to OSHA, while during the second part of this period, they may submit briefs, arguments, and summations.

Notice of Intention to Appear to Provide Testimony at the Informal Public Hearing. Interested persons who intend to provide oral testimony at the informal public hearing must file a notice of intention to appear by using the procedures specified above in the sections titled **DATES** and **ADDRESSES.** This notice must provide the: Name, address, and telephone number of each individual who will provide testimony; capacity (*e.g.*, name of the organization the individual is representing; the individual's title and position) in which each individual will testify; approximate amount of time required for each individual's testimony; specific issues each individual will address, including a brief statement of the position that the individual will take with respect to each of these issues; and any documentary evidence the individual will present, including a brief summary of the evidence. The hearings are open to the public, and all interested persons are welcome to attend. However, only a person who files a proper notice of intention to appear may ask questions and participate fully in the proceedings. While a person who did not file a notice of intention to appear may be allowed to testify at the hearing if time permits, this determination is at the discretion of the presiding ALJ.

Hearing Testimony and Documentary Evidence. Any person requesting more than 10 minutes to testify at the informal public hearing, or who intends to submit documentary evidence at the hearing, must provide the complete text

of the testimony and the documentary evidence as specified above in the **DATES** and **ADDRESSES** sections. The Agency will review each submission and determine if the information it contains warrants the amount of time requested. If OSHA believes the requested time is excessive, it will allocate an appropriate amount of time to the presentation, and will notify the participant of this action, and the reasons for the action, prior to the hearing. The Agency may limit to 10 minutes the presentation of any participant who fails to comply substantially with these procedural requirements; in such instances, OSHA may request that the participant return for questioning at a later time.

Certification of the Record and Final Determination After the Informal Public Hearing. Following the close of the hearing and post-hearing comment period, the presiding ALJ will certify the record to the Assistant Secretary of Labor for Occupational Safety and Health; the record will consist of all of the written comments, oral testimony, and documentary evidence received during the proceeding. OSHA will review the proposed Cr(VI) standard in light of all the evidence received as part of the record, and will make its decisions based on substantial evidence in the record as a whole.

XVII. Summary and Explanation of the Standards

OSHA believes that, based on currently available information, the proposed requirements set forth in this notice are necessary and appropriate to provide adequate protection to employees exposed to Cr(VI). OSHA has considered responses to the RFI as well as numerous reference works, journal articles, and other data obtained by the Agency in the development of this proposed standard.

The language of the standards and the order of the various provisions are generally consistent with drafting in other recent OSHA health standards, such as the methylene chloride, formaldehyde, and cadmium standards. OSHA believes that a similar style should be followed from standard to standard when possible in order to facilitate uniformity of interpretation of similar provisions. This approach is also consistent with Section 6(b)(5) of the OSH Act, which states that health standards shall consider ''experience gained under this and other health and safety laws.''

(a) Scope and Application

OSHA is proposing to issue separate standards addressing hexavalent chromium exposure in general industry, construction, and shipyards. The standard for shipyards would also apply to marine terminals and longshoring. The standards are intended to provide equivalent protection for all workers, while accounting for the different work activities, anticipated exposures, and other conditions in these sectors. The proposed standards for construction and shipyards are very similar to each other, but differ in some respects from the proposed standard for general industry. This summary and explanation will describe the proposed standard for general industry and will note differences between it and the proposed standards for construction and shipyards.

Based on the record developed to date, OSHA believes that certain activities in construction and shipyards are different enough to warrant requirements that are somewhat modified from those proposed for general industry. This preliminary determination is consistent with the recommendation of the Maritime Advisory Committee on Occupational Safety and Health (MACOSH), which has recommended that a separate standard be developed for maritime. The proposed standards do not cover the agricultural sector. OSHA is not aware of significant exposures to Cr(VI) in agriculture. The Agency is interested in any evidence indicating that significant exposures to Cr(VI) occur in sectors not covered under the proposed standards. Accordingly, the subject has been raised in the ''Issues'' section of this proposal.

The proposed standard applies to occupational exposures to hexavalent chromium (also referred to as chromium (VI) or Cr(VI)), that is, any chromium species with a valence of positive six, regardless of form or compound. Examples of Cr(VI) compounds include chromium oxide (CrO_2), ammonium dichromate ($(NH_4)_2Cr_2O_7$), calcium chromate ($CaCrO_4$), chromium trioxide (CrO_3), lead chromate ($PbCrO_4$), potassium chromate (K_2CrO_4), potassium dichromate ($K_2Cr_2O_7$), sodium chromate (Na_2CrO_4), strontium chromate ($SrCrO_4$), and zinc chromate ($ZnCrO_4$).

Some stakeholders have argued that specific Cr(VI) compounds should be excluded from this rulemaking and addressed in a separate standard. Notably, after OSHA was initially petitioned to issue a Cr(VI) standard, the Color Pigments Manufacturers Association (CPMA) submitted a cross-petition calling for a separate standard for lead chromate pigments (Ex. 2). CPMA argued that differences in the bioavailability and toxicity of lead chromate when compared to other Cr(VI) compounds warranted a separate standard (Ex. 2, p. 5). CPMA stated:

> Simply put, there are no studies which show a link between lead chromate pigments in a finished form and cancer caused by exposure to Chromium VI. To the contrary, studies of lead chromate workers in the manufacture of lead chromate pigments alone do not show any increased risk of cancer (Ex. 2, p. 5).

Because CPMA deemed that lead chromate pigments posed little threat to employee health, and because of concern about adverse economic impacts associated with regulation, the Association considered that ''* * * no good purpose would be served by additional restrictions on lead chromate pigments'' (Ex. 2, p. 6). This position was reiterated in CPMA's response to the RFI (Ex. 31–15, p. 6).

In its response to the RFI, the Boeing Company also expressed the view that OSHA should consider the bioavailability of different Cr(VI) compounds (Ex. 31–16, p. 8). Boeing indicated that exposures to strontium chromate and zinc chromate used in aerospace manufacturing are not equivalent to Cr(VI) exposures in other industries. The findings of two epidemiological studies of Cr(VI)-exposed aerospace workers were said to support this conclusion.

OSHA has proposed a rule that covers all Cr(VI) compounds because the Agency believes the evidence supports this approach. As discussed in Section VI.A of this preamble, absorption of Cr(VI) from the lung into the bloodstream is greatly dependent on the solubility of the Cr(VI) compound. Insoluble chromates are poorly absorbed and as a result remain in the lungs for a longer period of time (Ex. 35–87). While in the lungs, insoluble Cr(VI) particulates can come into contact with the epithelial cell surface, resulting in uptake into cells (Exs. 35–68; 35–67). Cellular uptake leads to DNA damage, apoptosis, and neoplastic transformation (Ex. 35–119). Less water-soluble chromates (*e.g.*, lead chromate) appear to be more potent carcinogens than more soluble chromates (*e.g.*, sodium chromate). (For a detailed discussion, see Section VI.B.8 of this preamble.)

Experimental studies involving Syrian hamster embryo cells support the belief that cytotoxicity and neoplastic transformation occur when exposures involve lead chromate pigments (Ex. 12–5). Evidence indicates that even chromates that are encapsulated in a paint matrix may be released in the lungs (Ex. 31–15, p. 2). OSHA therefore sees no reason to exempt these

compounds from the current Cr(VI) rulemaking.

OSHA believes this view is consistent with the epidemiological studies involving chromate pigment production workers and aerospace workers. While co-exposures to other Cr(VI) compounds do not allow for specific findings related to lead chromate exposure, OSHA has found that epidemiological studies of workers in the chromate pigment production industry have consistently shown excess risks for lung cancer (see Section VI.B.2 of this preamble). The studies of aerospace workers did not find an increased risk of lung cancer. However, this is not convincing evidence that aerospace workers are not at risk from Cr(VI) exposure. The small cohort size, lack of smoking data, relatively young age of the population, and number of members lost to follow-up in the study reported by Alexander *et al.* (Ex. 31–16–3) and the lack of exposure information in the report of Boice *et al.* (Ex. 31–16–4) do not allow for any broad conclusions regarding aerospace workers to be reached on the basis of these two studies. OSHA's preliminary conclusion that Cr(VI) compounds should be addressed collectively under a single standard is consistent with the findings of IARC, NTP, and NIOSH. These organizations have each found Cr(VI) compounds to be carcinogenic, without exception. Although ACGIH has issued different TLVs for soluble and insoluble Cr(VI) compounds, and for certain specific compounds, the TLV for insoluble Cr(VI) compounds is five-fold lower than the TLV for soluble Cr(VI) compounds. This is consistent with OSHA's preliminary finding that less soluble Cr(VI) compounds, to the extent that they differ from more soluble Cr(VI) compounds, are more potent carcinogens and pose a greater risk to the health of workers.

The proposed standard applies to occupational exposure in which Cr(VI), in any quantity, is present in an occupationally related context. Exposure of employees to the ambient environment, which may contain small concentrations of Cr(VI) unrelated to the job, is not subject to this standard.

The proposed standard for construction does not cover exposure to Cr(VI) in portland cement. Cement ingredients (clay, gypsum, and chalk), chrome steel grinders used to crush ingredients, refractory bricks lining the cement kiln, and ash may serve as sources of chromium that may be converted to Cr(VI) during kiln heating, leaving trace amounts of Cr(VI) in the finished product (Ex. 35–317, p. 148).

The amount of Cr(VI) in American cement is generally less than 20 μg/g (Ex. 9–57). While the Cr(VI) in cement may represent a dermal hazard, the evidence obtained by OSHA thus far indicates that the Cr(VI) concentration is generally so low that the proposed PEL could not be reached without exceeding OSHA's current PEL for Particulates Not Otherwise Regulated (PNOR). The PEL for PNOR (15 μg/m^3 for total dust) thus is at least as protective as the proposed Cr(VI) PEL in limiting the Cr(VI) inhalation exposure of cement workers. OSHA's preliminary exposure profile indicates that no employees are exposed to levels of Cr(VI) above 0.25 μg/m^3 as an 8-hour TWA during cement work in construction. Because airborne exposures to Cr(VI) during cement work in construction are expected to be minimal, and because of the economic burden of applying the ancillary provisions of the proposed standard to workers exposed to portland cement in the construction environment, OSHA has preliminarily concluded that exposures to Cr(VI) from portland cement are best addressed by providing guidance to employers rather than including portland cement in the construction rule.

OSHA has proposed to cover exposures to Cr(VI) in portland cement in general industry. The Agency's preliminary exposure profile indicates that some employees in general industry are exposed to airborne Cr(VI) levels associated with a significant risk of lung cancer as a result of work with portland cement. OSHA's preliminary findings show that nearly 2500 workers in general industry are exposed to Cr(VI) levels between 0.25 μg/m^3 and 0.5 μg/m^3 as an 8-hour TWA. Because of the evidence of higher airborne Cr(VI) exposures in general industry than in construction, and because lower burdens are anticipated in the more stable work environments found in general industry, the Agency believes it is appropriate to cover Cr(VI) exposures from portland cement under the general industry proposed standard. OSHA is interested in comments and information regarding this preliminary determination, and has included this topic in the ''Issues'' section of this preamble.

This proposal does not cover exposures to Cr(VI) that occur in the application of pesticides. Some Cr(VI)-containing chemicals, such as chromated copper arsenate (CCA) and acid copper chromate (ACC), are used for wood treatment and are regulated by EPA as pesticides. Section 4(b)(1) of the OSH Act precludes OSHA from regulating working conditions of employees where other Federal agencies exercise statutory authority to prescribe or enforce standards or regulations affecting occupational safety or health. Therefore, OSHA proposes to specifically exclude those exposures regulated by EPA from coverage under the standard.

The manufacture of pesticides containing Cr(VI) is not considered pesticide application, and is covered under this proposed standard. The use of wood treated with pesticides containing Cr(VI) is also covered. In this respect, the proposed Cr(VI) standard differs from OSHA's Inorganic Arsenic standard (29 CFR 1910.1018). The Inorganic Arsenic standard explicitly exempts the use of wood treated with arsenic. When the Inorganic Arsenic standard was issued in 1978, OSHA found that the evidence in the record indicated ''the arsenic in the preserved wood is bound tightly to the wood sugars, exhibits substantial chemical differences from other pentavalent arsenicals after reaction, and appears not to leach out in substantial amounts'' (43 FR 19584, 19613 (5/5/78)). Based on the record in that rulemaking, OSHA did not consider it appropriate to regulate the use of preserved wood. The record in this rulemaking indicates that work with wood treated with pesticides containing Cr(VI) can involve significant Cr(VI) exposures. OSHA's exposure profile for woodworking indicates that over 30% of current employee Cr(VI) exposures exceed the proposed PEL. OSHA therefore believes it appropriate to include these activities under the scope of the proposed standard.

(b) Definitions

''Action level'' is defined as an airborne concentration of Cr(VI) of 0.5 micrograms per cubic meter of air (0.5 μg/m^3) calculated as an eight-hour time-weighted average (TWA). The action level triggers requirements for exposure monitoring and medical surveillance in general industry workplaces. In this proposal, as in other standards, the action level has been set at one-half of the PEL.

Because of the variable nature of employee exposures to airborne concentrations of Cr(VI), maintaining exposures below the action level provides reasonable assurance that employees will not be exposed to Cr(VI) at levels above the PEL on days when no exposure measurements are made. Even when all measurements on a given day may fall below the PEL (but are above the action level), there is some chance that on another day, when exposures are not measured, the employee's actual exposure may exceed

the PEL. When exposure measurements are above the action level, the employer cannot be reasonably confident that employees may not be exposed to Cr(VI) concentrations in excess of the PEL during at least some part of the work week. Therefore, requiring periodic exposure measurements when the action level is exceeded provides the employer with a reasonable degree of confidence in the results of the exposure monitoring.

The action level is also intended to encourage employers to lower exposure levels in order to avoid the costs associated with the exposure monitoring and medical surveillance provisions. Some employers would be able to reduce exposures below the action level in all work areas, and other employers in some work areas. As exposures are lowered, the risk of adverse health effects among workers decreases.

OSHA s preliminary risk assessment indicates that significant risk remains at the proposed PEL of 1.0 μg/m^3. Where there is continuing significant risk, the decision in the Asbestos case (Building and Construction Trades Department, *AFL–CIO* v. *Brock,* 838 F. 2d 1258, (D.C. Cir 1988)) indicated that OSHA should use its legal authority to impose additional requirements on employers to further reduce risk when those requirements will result in a greater than de minimus incremental benefit to workers' health. OSHA's preliminary conclusion is that the action level will result in a very real and necessary, but non-quantifiable, further reduction in risk beyond that provided by the PEL alone. OSHA's choice of proposing an action level of one-half of the PEL is based on the Agency's successful experience with other standards, including those for inorganic arsenic (29 CFR 1910.1018), ethylene oxide (29 CFR 1910.1047), benzene (29 CFR 1910.1028), and methylene chloride (29 CFR 1910.1052).

As discussed under the requirements for exposure monitoring, OSHA has not proposed an action level for construction and shipyards. This definition is therefore not included in the proposed standards for construction and shipyards.

''Chromium (VI) [hexavalent chromium or Cr(VI)]'' means chromium with a valence of positive six, in any form or chemical compound in which it occurs. This term includes Cr(VI) in all states of matter, in any solution or other mixture, even if encapsulated by another or several other substances. The term also includes Cr(VI) when created by an industrial process, such as when welding of stainless steel generates Cr(VI) fume.

For regulatory purposes, OSHA is treating Cr(VI) generically, instead of addressing specific compounds individually. This is based on OSHA's preliminary determination that the toxicological effect on the human body is similar from Cr(VI) in any of the substances covered under the scope of this standard, regardless of the form or compound in which it occurs. As discussed in Section VI of this preamble, some variation in potency may result due to differences in the solubility of compounds. Other factors, such as encapsulation, may have some effect on the bioavailability of Cr(VI). However, OSHA believes that these factors do not result in differences that merit separate provisions for different Cr(VI) compounds. OSHA considers it appropriate to apply the requirements of the proposed standard uniformly to all Cr(VI) compounds.

''Emergency'' means any occurrence that results, or is likely to result, in an uncontrolled release of Cr(VI), such as, but not limited to, equipment failure, rupture of containers, or failure of control equipment. Every spill or leak is not necessarily an emergency. The exposure to Cr(VI) must be unexpected and significant.

If an incidental release of Cr(VI) may be safely cleaned up by employees at the time of release, it is not considered to be an emergency situation for the purposes of this section. The particular circumstances of the release itself, such as the quantity involved, confined space considerations, and the adequacy of ventilation will have an impact on employee safety. In addition, factors such as the knowledge of employees in the immediate work area, the personal protective equipment available, pre-established standard operating procedures for responding to releases, and engineering controls that employees can activate to assist them in controlling and stopping the release are all factors that must be considered in determining whether a release is incidental or an emergency. Those instances that constitute an emergency trigger certain requirements in this proposed standard (*e.g.*, medical surveillance) that are discussed later in this section.

''Employee exposure'' means exposure to airborne Cr(VI) that would occur if the employee were not using a respirator. This definition is included to clarify the fact that employee exposure is measured outside any respiratory protection worn. It is consistent with OSHA's previous use of the term in other standards.

''Physician or other licensed health care professional (PLHCP)'' refers to an individual who is legally permitted to provide some or all of the health care services required by this section. This definition is included because the proposed standard requires that all medical examinations and procedures be performed by or under the supervision of a PLHCP.

Any professional may perform the medical examinations and procedures provided under the standard when they are licensed by state law to do so. The Agency recognizes that this means that the personnel qualified to provide the required medical examinations and procedures may vary from state to state, depending on state licensing laws. This provision grants the employer the flexibility to retain the services of a variety of qualified licensed health care professionals, provided that these individuals are licensed to perform the specified service. OSHA believes that this flexibility will reduce cost and compliance burdens for employers and increase convenience for employees. The approach taken in this proposed standard is consistent with the approach OSHA has taken in other recent standards, such as those for methylene chloride (29 CFR 1910.1052), bloodborne pathogens (29 CFR 1910.1030), and respiratory protection (29 CFR 1910.134).

''Regulated area'' means an area, demarcated by the employer, where an employee's exposure to airborne concentrations of Cr(VI) exceeds, or can reasonably be expected to exceed the PEL. This definition is consistent with the use of the term in other standards, including those for cadmium (29 CFR 1910.1027), butadiene (29 CFR 1910.1051), and methylene chloride (29 CFR 1910.1052).

OSHA has not proposed a requirement for regulated areas in construction and shipyards. This definition is therefore not included in the proposed standards for construction and shipyards.

The definitions for ''Assistant Secretary'', ''Director'', ''High-efficiency particulate air [HEPA] filter'', and ''This section'' are consistent with OSHA's previous use of these terms found in other health standards.

(c) Permissible Exposure Limit (PEL)

OSHA proposes to set an 8-hour time-weighted average (TWA) exposure limit of 1 microgram of Cr(VI) per cubic meter of air (1 μg/m^3). This limit means that over the course of any 8-hour work shift, the average exposure to Cr(VI) cannot exceed 1 μg/m^3. The proposed limit applies to Cr(VI), as opposed to the current PEL which is measured as CrO_3. The current PEL of 1 milligram per 10 cubic meters of air (1 μg/10 m^3, or 100

μg/m³) reported as CrO_3 is equivalent to a limit of 52 μg/m³ as Cr(VI). The current PEL is enforced as a TWA in construction and as a ceiling (a level not to be exceeded at any time) in general industry.

OSHA proposes a new PEL of 1 μg/m³ because the Agency has preliminarily determined that occupational exposure to Cr(VI) at the current PEL results in a significant risk of lung cancer among exposed workers, and that compliance with the proposed standard will substantially reduce that risk. OSHA's preliminary risk assessment, presented in Section VII of this preamble, indicates that the most reliable lifetime estimate of risk from a 45 year exposure to Cr(VI) at the current PEL is 101 to 351 excess deaths from lung cancer per 1000 workers. As discussed in Section VIII of this preamble, this clearly represents a risk of material impairment of health that is significant within the context of the Benzene decision. OSHA believes that lowering the PEL to 1 μg/m³ would reduce the lifetime excess risk of death from lung cancer to between 2.1 and 9.1 per 1000 workers.

OSHA considers the level of risk remaining at the proposed PEL to be significant. However, as discussed in Section IX of this preamble, the proposed PEL is set at the lowest level that the Agency believes to be feasible in all affected industry sectors. As guided by the 1988 Asbestos decision, OSHA is proposing additional requirements to further reduce the remaining risk. OSHA anticipates that the ancillary provisions in the proposed standard will further reduce the risk beyond the reduction that would be achieved by the proposed PEL alone.

OSHA believes that it is appropriate to establish a single PEL that applies to all Cr(VI) compounds. OSHA's preferred estimates of risk supporting the proposed PEL are derived from worker cohorts that were predominantly exposed to soluble sodium chromate. The evidence reviewed by OSHA indicates that similar doses of less soluble chromates result in higher numbers of lung tumors when compared to more soluble compounds such as sodium chromate (see Section VI of this preamble). Thus, any variation in toxicological effect due to solubility is expected to result in a higher level of risk than is indicated by OSHA's preliminary risk estimates. OSHA consequently believes that the Agency's findings regarding significance of risk are valid regardless of the solubility of the Cr(VI) compound. However, the available evidence is not sufficient to make quantitative estimates of risk for each individual Cr(VI) compound. OSHA is therefore proposing a single PEL for all Cr(VI) compounds. The Agency seeks comment on whether different PELs for different Cr(VI) compounds should be set and how such determinations should be made, and has included this topic in the ''Issues'' section of the preamble.

(d) Exposure Monitoring

The proposed general industry standard imposes monitoring requirements pursuant to Section 6(b)(7) of the OSH Act (29 U.S.C. 655) which mandates that any standard promulgated under section 6(b) shall, where appropriate, ''provide for monitoring or measuring of employee exposure at such locations and intervals, and in such manner as may be necessary for the protection of employees.''

The purpose of requiring assessment of employee exposures to Cr(VI) include: determination of the extent and degree of exposure at the worksite; identification and prevention of employee overexposure; identification of the sources of exposure to Cr(VI); collection of exposure data so that the employer can select the proper control methods to be used; and evaluation of the effectiveness of those selected methods. Assessment enables employers to meet their legal obligation to ensure that their employees are not exposed to Cr(VI) in excess of the permissible exposure level and to notify employees of their exposure levels, as required by section 8(c)(3) of the Act. In addition, the availability of exposure data enables the PLHCP performing medical examinations to be informed of the extent of occupational exposures.

Paragraph (d)(1) contains proposed general requirements for exposure monitoring. Monitoring to determine employee exposures must represent the employee's time-weighted average exposure to airborne Cr(VI) over an eight-hour workday. Samples must be taken within the employee's breathing zone (i.e., ''personal breathing zone samples'' or ''personal samples''), and must represent the employee's exposure without regard to the use of respiratory protection.

Employers must accurately characterize the exposure of each employee to Cr(VI). In some cases, this will entail monitoring all exposed employees. In other cases, monitoring of ''representative'' employees is sufficient. Representative exposure sampling is permitted when a number of employees perform essentially the same job under the same conditions. For such situations, it may be sufficient to monitor a fraction of these employees in order to obtain data that are ''representative'' of the remaining employees. Representative personal sampling for employees engaged in similar work with Cr(VI) exposure of similar duration and magnitude can be achieved by monitoring the employee(s) reasonably expected to have the highest Cr(VI) exposures. For example, this may involve monitoring the Cr(VI) exposure of the employee closest to an exposure source. This exposure result may then be attributed to the remaining employees in the group.

Exposure monitoring should include, at a minimum, one full-shift sample taken for each job function in each job classification, in each work area, for each shift. These samples must consist of at least one sample characteristic of the entire shift or consecutive representative samples taken over the length of the shift. Where employees are not performing the same job under the same conditions, representative sampling will not adequately characterize actual exposures, and individual monitoring is necessary.

OSHA proposes that employers who have workplaces covered by the general industry standard determine if any of their employees are exposed to Cr(VI) at or above the action level. Further obligations under the standard would be based on the results of this assessment. These may include obligations for periodic monitoring, establishment of regulated areas, implementation of control measures, and provision of medical surveillance.

Initial monitoring need not be conducted under two circumstances. First, where the employer has previously monitored for Cr(VI) in the past 12 months and the data were obtained during work operations conducted under workplace conditions closely resembling the processes, types of material, control methods, work practices, and environmental conditions used and prevailing in the employer's current operations, and where that monitoring satisfies all other requirements of this section, including the accuracy and confidence requirements, the employer may rely on such earlier monitoring results to satisfy the initial monitoring requirements of this section. This provision is designed to make it clear that OSHA does not intend to require employers who have recently performed appropriate employee monitoring to conduct ''initial'' monitoring. OSHA anticipates that this provision will reduce the compliance burden on employers, since monitoring for tasks that involve essentially the same exposures would

not be required. The Agency believes allowing the use of 12 month old data is appropriate; samples taken earlier than 12 months previously may not adequately represent current workplace conditions. The 12 month limit is consistent with the Methylene Chloride standard (29 CFR 1910.1052).

Second, where the employer has objective data demonstrating that a particular product or material containing Cr(VI) or a specific process, operation, or activity involving Cr(VI) cannot release dust, fumes, or mist in concentrations at or above the action level under any expected conditions of use, the employer may rely upon such data to satisfy initial monitoring requirements. The data must reflect workplace conditions closely resembling the processes, types of material, control methods, work practices, and environmental conditions in the employers' current operations.

Objective data demonstrate that the work operation or the product may not reasonably be foreseen to release Cr(VI) in airborne concentrations at or above the action level under the expected conditions of use that will cause the greatest possible release, or in any plausible accident. The objective data may include monitoring data, or mathematical modeling or calculations based on the chemical and physical properties of a material. For example, data collected by a trade association from its members that meet the definition of objective data may be used. When using the term ''objective data'', OSHA is referring to employers' reliance on manufacturers' worst case studies, laboratory studies, and other research that demonstrates, usually by means of exposure data, that meaningful exposures cannot occur. OSHA has allowed employers to use objective data in other standards such as those for formaldehyde (29 CFR 1910.1048) and asbestos (29 CFR 1910.1001) in lieu of initial monitoring and hence, from most of the provisions of these standards.

Paragraph (d)(3) contains requirements for periodic monitoring. The requirement for continued monitoring depends on the results of initial monitoring. If the initial monitoring indicates that employee exposures are below the action level, no further monitoring would be required unless changes in the workplace result in new or additional exposures. If the initial determination reveals employee exposures to be at or above the action level but below the PEL, the employer must perform periodic monitoring at least every six months. If the initial monitoring reveals employee exposures to be above the PEL, the employer must repeat monitoring at least every three months.

The proposed rule also includes provisions to adjust the frequency of periodic monitoring based on monitoring results. If periodic monitoring results indicate that employee exposures have fallen below the action level, and those results are confirmed by consecutive measurements taken at least seven days later, the employer may discontinue monitoring for those employees whose exposures are represented by such monitoring. Similarly, if periodic monitoring measurements indicate that exposures are below the PEL but above the action level, and those results are confirmed by consecutive measurements taken at least seven days later, the employer may reduce the frequency of the monitoring to at least every six months.

OSHA recognizes that exposures in the workplace may fluctuate. Periodic monitoring provides the employer with assurance that employees are not experiencing higher exposures that may require the use of additional control measures. In addition, periodic monitoring reminds employees and employers of the continued need to protect against the hazards associated with exposure to Cr(VI).

Because of the fluctuation in exposures, OSHA believes that when initial monitoring results exceed the action level but are below the PEL, employers should continue to monitor employees to ensure that exposures remain below the PEL. Likewise, when initial monitoring results exceed the PEL, periodic monitoring allows the employer to maintain an accurate profile of employee exposures. If the employer installs or upgrades controls, periodic monitoring will demonstrate whether or not controls are working properly. Selection of appropriate respiratory protection also depends on adequate knowledge of employee exposures.

In general, the more frequently periodic monitoring is performed, the more accurate the employee exposure profile. Selecting an appropriate interval between measurements is a matter of judgment. OSHA believes that the proposed frequency of six months for subsequent periodic monitoring for exposures above the action level but below the PEL, and three months for exposures above the PEL, provides intervals that are both practical for employers and protective for employees. This belief is supported by OSHA's experience with comparable monitoring intervals in other standards, including those for cadmium (29 CFR 1910.1027), methylenedianiline (29 CFR 1910.1050), methylene chloride (29 CFR 1910.1052), and formaldehyde (29 CFR 1910.1048). The proposed requirement for periodic monitoring is also consistent with OSHA's Standards Improvement Project (SIPs) proposal for monitoring frequency (67 FR 66494, 66504 (8/31/02)).

OSHA recognizes that monitoring can be a time-consuming, expensive endeavor and therefore offers employers the incentive of discontinuing monitoring for employees whose sampling results indicate exposures are below the action level. The Agency does not believe that periodic monitoring is generally necessary when monitoring results show that exposures are below the action level because there is a low probability that the results of future samples would exceed the PEL. The Agency intends for this provision to encourage employers to control their employees' exposures to Cr(VI) below the action level, thus maximizing the protection of employees' health.

Under paragraph (d)(4), employers are to perform additional monitoring when there is a change in production process, raw materials, equipment, personnel, work practices, or control methods, that may result in new or additional exposures to Cr(VI). In addition, there may be other situations which can result in new or additional exposures to Cr(VI) which are unique to an employer's work situation. In order to cover those special situations, OSHA requires the employer to perform additional monitoring whenever the employer has any reason to believe that a change has occurred which may result in new or additional exposures. This additional monitoring is necessary to ensure that monitoring results accurately represent existing exposure conditions. This is necessary so that the employer can take appropriate action to protect exposed employees, such as instituting additional engineering controls or providing appropriate respiratory protection.

Under paragraph (d)(5) of the general industry standard, employers are to notify each affected employee of their monitoring results within 15 working days after the receipt of the results. The employer shall either notify each affected employee in writing or post the monitoring results in an appropriate location accessible to all affected employees. In addition, whenever the PEL has been exceeded, the written notification must contain a description of the corrective action(s) being taken by the employer to reduce the employee's exposure to or below the PEL. The requirement to inform employees of the

corrective actions the employer is taking to reduce the exposure level to or below the PEL is necessary to assure employees that the employer is making efforts to furnish them with a safe and healthful work environment, and is required under section 8(c)(3) of the Act.

The proposal would require that all affected employees be notified of the monitoring results. When using the term ''affected employees'' in this context, OSHA is not referring only to the employee(s) actually subject to personal monitoring. Affected employees include all employees represented by the employee(s) sampled.

Individual notification in writing or posting would be acceptable under the proposed rule. This is consistent with other OSHA standards such as those for methylenedianiline (29 CFR 1910.1050), butadiene (29 CFR 1910.1051), and methylene chloride (29 CFR 1910.1052). In addition, the SIPs proposal (67 FR 66494, 66508 (10/31/02)) allows for employer choice of notification method. The Cr(VI) proposal is also consistent with SIPs in that SIPs specifies 15 working days after the receipt of monitoring results as the appropriate time to notify employees in general industry (67 FR 66494, 66508 (10/31/03)).

Under paragraph (d)(6), the employer would be required to use monitoring and analytical methods that can measure airborne levels of Cr(VI) to within an accuracy of plus or minus 25% (+/-25%) and can produce accurate measurements to within a statistical confidence level of 95% percent for airborne concentrations at or above the action level. Many laboratories presently have methods to measure Cr(VI) at the proposed action level with at least the required degree of accuracy. One example of an acceptable method of monitoring and analysis is OSHA method ID215. Rather than specifying a particular method that must be used, OSHA proposes to take a performance approach and instead allows the employer to use any method as long as the chosen method meets the accuracy specifications.

Paragraph (d)(7) requires the employer to provide affected employees or their designated representatives an opportunity to observe any monitoring of employee exposure to Cr(VI). When observation of monitoring requires entry into an area where the use of protective clothing or equipment is required, the employer must provide the observer with that protective clothing or equipment, and assure that the observer uses such clothing or equipment and complies with all other applicable safety and health procedures.

The requirement for employers to provide employees or their representatives the opportunity to observe monitoring is consistent with the OSH Act. Section 8(c)(3) of the OSH Act mandates that regulations developed under Section 6 provide employees or their representatives with the opportunity to observe monitoring or measurements. Also, Section 6(b)(7) of the OSH Act states that where appropriate, OSHA standards are to prescribe suitable protective equipment to be used in dealing with hazards. The provision for observation of monitoring and protection of the observers is also consistent with OSHA's other substance-specific health standards such as those for cadmium (29 CFR 1910.1027) and methylene chloride (29 CFR 1910.1052).

The proposed construction and shipyard standards for Cr(VI) do not include provisions for exposure monitoring. OSHA recognizes that in these sectors in many instances the results of exposure monitoring required under this proposed standard would not be available until after operations involving Cr(VI) exposure have been completed. For example, a welding task may be finished in a single day. If air monitoring is performed, the task would be completed before the employer is informed of the monitoring results. Therefore, the employer would not be in a position to make use of the monitoring results to determine appropriate control measures for that task. In other cases, the workplace conditions in construction and shipyard worksites may vary to such a great extent that it may be difficult to accurately characterize employee exposure from one day to the next. For example, a stainless steel welder may work outdoors on a windy day one day and in an enclosed environment the next day. Personal monitoring for Cr(VI) exposure on a given day may not accurately reflect these changing conditions. OSHA has therefore proposed a performance-oriented requirement for construction and shipyard employers. Rather than include specific requirements for exposure monitoring for these employers, OSHA proposes to allow construction and shipyard employers the flexibility to assess Cr(VI) exposures in any manner they choose. Thus, construction and shipyard employers could use historical data, objective data, or employee monitoring to determine employee exposures. Because the obligation to comply with the PEL would remain, whatever method the employer chooses would have to be sufficient to ensure that no employee is exposed to an airborne concentration of Cr(VI) in excess of the PEL.

In some cases, the employer may choose not to perform any monitoring. For example, certain tasks (*e.g.*, abrasive blasting of materials coated with Cr(VI); welding, cutting, or torch burning of stainless steel or of materials coated with Cr(VI); or spray application of Cr(VI) containing paints or coatings) frequently entail exposures to Cr(VI) above the proposed PEL. OSHA estimates that approximately 43% of the exposures in construction welding and 17.9% of the exposures in shipyard welding are greater than the proposed PEL of 1 $\mu g/m^3$. A construction or shipyard employer has the option of assuming the employee is exposed above the PEL and providing appropriate protective measures as prescribed by the standard.

Similarly, an employer may not find it necessary to perform exposure monitoring where exposures are well below the PEL. For example, there are several construction application groups (*e.g.*, industrial rehabilitation and maintenance, hazardous waste site work, and refractory restoration and maintenance) where a large percentage of exposures are either below 0.25 $\mu g/m^3$ or below the limit of detection for Cr(VI). In these situations, employers may be relatively assured that employees' exposure are well below the PEL and would therefore not need to conduct exposure monitoring.

This approach is consistent with OSHA's standard for air contaminants (29 CFR 1910.1000), which establishes PELs for over 400 substances, but does not include specific requirements for exposure monitoring. The Agency seeks comment as to whether this performance-oriented approach to exposure monitoring is appropriate in construction and shipyard workplaces, and has included this topic in the ''Issues'' section of this preamble.

(e) Regulated Areas

Under paragraph (e), general industry employers must establish regulated areas wherever an employee's exposure to airborne concentrations of Cr(VI) is, or can reasonably be expected to be, in excess of the PEL. Regulated areas are to be demarcated from the rest of the workplace in a manner that adequately establishes and alerts employees to the boundaries of these areas, and would be required to include the warning signs specified in paragraph (l)(2) of the proposed standard. Access to regulated areas is limited to persons authorized by the employer and required by work

duties to be present in the regulated area; any person entering the regulated area to observe monitoring procedures; or any person authorized by the OSH Act or regulations issued under it to be in a regulated area.

The purpose of a regulated area is to ensure that the employer makes employees aware of the presence of Cr(VI) at levels above the PEL, and to limit Cr(VI) exposure to as few employees as possible. The establishment of a regulated area is an effective means of limiting the risk of exposure to substances known to have carcinogenic effects. Because of the potentially serious results of exposure and the need for persons entering the area to be properly protected, the number of persons given access to the area should be limited to those employees needed to perform the job. Limiting access to regulated areas also has the benefit of reducing the employer's obligation to implement provisions of this proposal to as few employees as possible.

In keeping with the performance orientation of this proposed standard, OSHA has not specified how employers are to demarcate regulated areas. The demarcation should effectively warn employees not to enter the area unless they are authorized, and then only if they are using the proper personal protective equipment. The demarcation must include display of warning signs at all approaches to the regulated areas, consistent with the requirements of paragraph (l)(2) of this proposed standard. In many cases these warning signs alone will be sufficient to identify the boundaries of the regulated area.

Access to the regulated area is restricted to ''authorized persons''. For the purposes of this proposed standard, these are persons required by their job duties to be present in the area, as authorized by the employer. In addition, persons exercising the right to observe monitoring procedures are also allowed to enter regulated areas. Employees in some workplaces may designate a union representative to observe monitoring; this person would be allowed to enter the regulated area. Persons authorized under the OSH Act, such as OSHA compliance officers, are also allowed access to regulated areas.

OSHA has not included a requirement for regulated areas in construction and shipyard workplaces, due to the expected difficulties in establishing regulated areas in construction and shipyard workplaces. For example, several small entity representatives (SERs) from the construction and shipyard industries who participated in the SBREFA review noted that in their work settings regulated areas would be particularly problematic and might require that the entire worksite be designated as a regulated area. They also noted that due to the changing nature of the work site (namely construction sites) the demarcation of the regulated area would have to be changed each day as the work progressed (*e.g.,* Exs. 34–6, 34–14). The same rationale applies to shipyards. The Agency seeks comment as to whether a requirement for the establishment of regulated areas would be appropriate for construction or shipyard workplaces and how such areas could be established, and has included this topic in the ''Issues'' section of this preamble.

(f) Methods of Compliance

The proposed standard requires employers to institute effective engineering and work practice controls as the primary means to reduce and maintain employee exposures to Cr(VI) to levels that are at or below the PEL, unless the employer can demonstrate that such controls are not feasible, or if employees are not exposed above the PEL for 30 or more days per year. Employers would be required to institute engineering controls and work practices to reduce exposure to the lowest feasible level even if these measures alone would not reduce the concentration of airborne Cr(VI) to or below the PEL. The employer would then be required to supplement these controls with respirators to ensure that employees are not exposed to Cr(VI) above the PEL.

Primary reliance on engineering controls and work practices is consistent with good industrial hygiene practice and with OSHA's traditional adherence to a hierarchy of preferred controls. Engineering controls are reliable, provide consistent levels of protection to a large number of workers, can be monitored continually and inexpensively, allow for predictable performance levels, and can efficiently remove toxic substances from the workplace. Once removed, the toxic substance no longer poses a threat to employees. The effectiveness of engineering controls does not generally depend to any substantial degree on human behavior, and the operation of equipment is not as vulnerable to human error as is personal protective equipment. For these reasons, engineering controls are preferred by OSHA.

Engineering controls can be grouped into three main categories: (1) Substitution; (2) isolation; and (3) ventilation, both general and localized. Quite often a combination of these controls can be applied to an industrial hygiene control problem to achieve satisfactory air quality. It may not be necessary to apply all these measures to any specific potential hazard.

Substitution can be an ideal control measure. One of the best ways to prevent workers from being exposed to a toxic substance is to stop using it entirely. Although substitution is not always possible, replacement of a toxic material with a less hazardous alternative should always be considered.

In those cases where substitution of a less toxic material is not possible, substituting one type of process for another process may provide effective control of an air contaminant. For example, process changes from batch operations to continuous operations will usually reduce exposures. This is true primarily because the frequency and duration of workers' potential contact with process materials is reduced in continuous operations. Similarly, automation of a process can further reduce the potential hazard.

In addition to substitution, isolation should be considered as an option for controlling employee exposures to Cr(VI). Isolation can involve containment of the source of a hazard, thereby separating it from most workers. Workers can be isolated from Cr(VI) by working in a clean room or booth, or by placing some other type of barrier between the source of exposure and the employee. Employees can also be protected by being placed at a greater distance from the source of Cr(VI) emissions.

Frequently, isolation enhances the benefits of other control methods. For example, Cr(VI) compounds may be used in the formulation of certain paints. If the mixing operation is conducted in a small, enclosed room the airborne Cr(VI) potentially generated by the operation could be confined to a small area. By ensuring containment, local exhaust ventilation is more effective.

Ventilation is a method of controlling airborne concentrations of a contaminant by supplying or exhausting air. A local exhaust system is used to remove an air contaminant by capturing the contaminant at or near its source before it spreads throughout the workplace. General ventilation (dilution ventilation), on the other hand, allows the contaminant to spread throughout the work area but dilutes it by circulating large quantities of air into and out of the area. A local exhaust system is generally preferred to dilution ventilation because it provides a cleaner and healthier work environment.

Work practices controls involve adjustments in the way a task is performed. In many cases, work practice controls complement engineering controls in providing worker protection. For example, periodic inspection and maintenance of process equipment and control equipment such as ventilation systems is an important work practice control. Frequently, equipment which is in disrepair or near failure will not perform normally. Regular inspections can detect abnormal conditions so that timely maintenance can then be performed. If equipment is routinely inspected, maintained, and repaired or replaced before failure is likely, there is less chance that hazardous exposures will occur.

Workers must know the proper way to perform their job tasks in order to minimize their exposure to Cr(VI) and to maximize the effectiveness of control measures. For example, if an exhaust hood is designed to provide local ventilation and a worker performs a task that generates a contaminant away from the exhaust hood, the control measure will be of no use. Workers can be informed of proper operating procedures through information and training. Good supervision provides further support for ensuring that proper work practices are carried out by workers. By persuading a worker to follow proper procedures, such as positioning the exhaust hood in the correct location to capture the contaminant, a supervisor can do much to minimize unnecessary exposure.

Employees' exposures can also be controlled by scheduling operations with the highest exposures at a time when the fewest employees are present. For example, routine clean-up operations that involve Cr(VI) releases might be performed at night or at times when the usual production staff is not present.

OSHA has traditionally relied less on respiratory protection in the hierarchy of controls because the use and efficacy of respirators depends to a great extent on human behavior. Often work is strenuous, and the increased breathing resistance of the respirator reduces its acceptability to employees. Respirators can limit an employee's vision and ability to communicate. In some difficult and dangerous jobs, effective vision or communication is vital to a safe, efficient operation. Voice communication when using a respirator can be difficult and fatiguing. In any event, movement of the jaw in speaking can cause a temporary breaking of the face-to-facepiece seal, thereby reducing the efficiency of the respirator and decreasing the employee's protection. Skin irritation can result from wearing a respirator in hot, humid conditions. Such irritation can cause considerable distress to workers and may disrupt work schedules. To be used effectively, respirators must be individually selected; fitted and periodically refitted; conscientiously and properly worn; regularly maintained, including filter changes; and replaced as necessary. In some workplaces, these preconditions for effective respirator use can be difficult to achieve. It is more difficult to assure that each employee is wearing a respirator correctly than to ascertain that engineering controls are operational. Thus, OSHA has concluded that reliance upon respirators should be minimized when engineering and work practice controls are found to be effective.

OSHA has proposed an exception to the general requirement for primary reliance on engineering and work practice controls for those employers who do not have employee exposures above the PEL for 30 or more days per year (12 consecutive months) from a particular process or task. Thus, if an employee is exposed to Cr(VI) on only 29 days during any 12 consecutive months from a particular process or task, even if the exposure is above the PEL on all of these days, the employer would not be required by this proposed standard to implement engineering and work practice controls to control exposures to the PEL. The burden would be on the employer to show that exposures do not exceed the PEL on 30 or more days per year. OSHA believes this provision would provide needed flexibility to employers, while still protecting workers.

Under the proposed exception, the employer's obligation to implement engineering and work practice controls to comply with the PEL would not be triggered until an employee in a process or task is exposed above the PEL on 30 or more working days during a year. Where the exposure is for fewer than 30 working days, the employer could use any combination of controls to prevent employees from being exposed above the PEL, including respirators alone. The employer may use this exception if he or she has a reasonable basis for believing that employees in a process or task will not be exposed above the PEL for 30 or more days per year (12 consecutive months). OSHA intends for this exception to be process- or task-based, *i.e.,* it is specific to a process where engineering controls might be implemented to reduce exposures below the PEL. For example, an employer might have two processes, A and B, where A involved an ongoing process in the facility with exposures above the PEL for more than 30 days and another process, B, only resulted in exposures above the PEL between 10 and 29 days. The fact that the employer had employees exposed above the PEL for more than 30 days in process A would not be used to determine that engineering and work practice controls had to be used for process B. OSHA intends this exception to be similarly applied by process or task in the construction and shipyard environments where employees may move from one work site to another.

OSHA has proposed this exception because the Agency realizes that in some industries (*e.g.,* color pigment manufacturing), exposure to Cr(VI) is typically infrequent (*i.e.,* fewer than 30 days, over 12 consecutive months). For example, certain Cr(VI) processes may occur only several days a year when production of a particular product is needed. Under such conditions of exposure, it may not be economically feasible or cost effective to invest the monies needed to install engineering controls or to institute work practices to control Cr(VI) to the PEL. Without such an exception, employers would be required to implement feasible engineering controls or work practice controls wherever employees are exposed to Cr(VI) above the PEL, even if they are only exposed on one or several days a year. OSHA believes that the expense of implementing engineering and work practice controls in such circumstances may not be justified. Consequently, incorporating an exception is a reasonable way to lessen the burden on employers while still protecting employees. OSHA's proposed exception for fewer than 30 working days per year is consistent with the standards for lead (29 CFR 1910.1025) and cadmium (29 CFR 1910.1027), both of which incorporate similar provisions.

In proposing this exception, OSHA intends to provide relief exclusively to employers whose employees are exposed to Cr(VI) only for short periods (in terms of days and weeks) and otherwise are not exposed to Cr(VI) above the PEL. Where the employee has other exposures above the PEL, the employer would be obligated to achieve the PEL by means of engineering and work practice controls. The Agency believes the proposed 30-working-day exclusion would make the standard more flexible in workplaces where exposure days are extremely limited.

In order for this exception to apply, the proposed standard states that the employer must have a ''reasonable basis for believing that no employees in a

process or task will be exposed above the PEL for 30 or more days''. Historical data, objective data, or exposure monitoring data may all provide a reasonable basis for believing that employees will not be exposed above the PEL for 30 or more days per year. Other information, such as production orders showing that processes involving Cr(VI) exposures are conducted on fewer than 30 days per year, may also serve as a reasonable basis for believing that employees will not be exposed above the PEL for 30 or more days per year.

In order to take advantage of the proposed exception, the employer would have the burden to demonstrate that his or her employees in a process or task will not be exposed above the PEL for more than 30 days per year. The burden of proof is placed on the employer because the employer has access to needed information about employee exposure levels and processes and tasks at the worksite. Where existing information is inadequate, the employer is also in the best position to develop the necessary information. The obligation to demonstrate that a reasonable basis exists for believing that employees in a process or task will not be exposed above the PEL for more than 30 days per year is the same for general industry, construction, and shipyard employers.

Paragraph (f)(2) of the proposed rule (paragraph (d)(2) of the construction and shipyard proposals) would prohibit the employer from using employee rotation as a means of compliance with the PEL. Worker rotation reduces the exposures to individual employees, but increases the number of employees exposed. Since OSHA has made a preliminary determination that Cr(VI) is carcinogenic, the Agency considers it inappropriate to place more workers at risk. Since no threshold has been established for the carcinogenic effects of Cr(VI), it is prudent to limit the number of workers exposed at any concentration. This provision does not, however, prohibit worker rotation when it is conducted for reasons other than compliance with the PEL. For example, an employer may rotate workers in order to provide cross-training on different tasks, or to allow workers to alternate physically demanding tasks with less strenuous activities. OSHA does not intend for this provision to be interpreted as a general prohibition on employee rotation where workers are exposed to Cr(VI). This proposed provision is consistent with other OSHA standards such as those for butadiene (29 CFR 1910.1051), methylene chloride (29 CFR 1910.1052), and cadmium (29 CFR 1910.1027).

(g) Respiratory Protection

When engineering controls and work practices cannot reduce employee exposure to Cr(VI) to within the PEL, OSHA proposes that the employer must protect employees' health through the use of respirators. Specifically, respirators would be required as supplementary protection to reduce employee exposure during the installation and implementation of engineering and work practice controls; during work operations where engineering and work practice controls are not feasible; when all feasible engineering and work practice controls have been implemented, but are not sufficient to reduce exposure to or below the PEL; during work operations where employees are exposed above the PEL for fewer than 30 days per year, and the employer has elected not to implement engineering and work practice controls to achieve the PEL; and during emergencies.

These limitations on the required use of respirators are generally consistent with other OSHA health standards, such as those for butadiene (29 CFR 1910.1051) and methylene chloride (29 CFR 1910.1052). They reflect the Agency's determination, discussed in the section on methods of compliance, that respirators are inherently less reliable than engineering and work practice controls. OSHA has therefore proposed to allow reliance on respirators only in certain designated situations.

In those circumstances where engineering and work practice controls cannot be used to achieve the PEL (*e.g.,* in emergencies, or during periods when equipment is being installed), or where engineering controls may not be reasonably necessary (*e.g.,* where employees are exposed above the PEL for fewer than 30 days per year), OSHA recognizes that respirators may be essential to reduce worker exposure, and provision is made for their use as primary controls. In other circumstances, where feasible work practices and engineering controls alone cannot reduce exposure levels to the PEL, respirators also may be used for supplemental protection. In these situations, the burden of proof is placed on the employer to demonstrate that engineering and work practice controls are not feasible.

OSHA anticipates that engineering and work practice controls will be in place by the effective dates specified in paragraph (n) of this proposal (paragraph (k) for construction and shipyards). The Agency realizes that in some cases employers may commence operations that involve employee Cr(VI) exposures after that date, may install new or modified equipment, or make other workplace changes that result in new or additional exposures to Cr(VI). In these cases, a reasonable amount of time may be needed before appropriate engineering controls can be installed and proper work practices implemented. When employee exposures exceed the PEL in these situations, employers are expected to provide respiratory protection to protect workers.

Respiratory protection is also required during work operations where engineering and work practice controls are not feasible. OSHA anticipates that there will be very few situations where no engineering and work practice controls are feasible to limit employee exposure to Cr(VI). In other cases, some engineering and work practice controls may be feasible, but these controls may not be capable of lowering employee exposures to or below the PEL. For example, tasks such as stainless steel welding or abrasive blasting may present certain difficulties when performed in confined spaces. In these cases, the employer would be required to provide respiratory protection. In any event, the employer must always install engineering controls and implement work practice controls when such controls are feasible to reduce exposures, even if these controls cannot reduce exposures below the PEL.

The requirement to provide respiratory protection when feasible engineering controls are not sufficient to reduce exposures to within the PEL would also apply in instances where effective engineering controls have been installed and are being maintained or repaired. In these situations, controls may not be effective while maintenance or repair is underway. Where exposures exceed the PEL, the employer would be required to provide respirators.

As discussed earlier with regard to methods of compliance, OSHA is proposing an exemption from the general requirement for use of engineering and work practice controls where employee exposures do not exceed the PEL on 30 or more days per year. Where this exception applies, the employer would then be required to provide respiratory protection to achieve the PEL. OSHA also believes that emergencies are situations where respirators must be used to protect employees. Since an emergency, by definition, involves or is likely to involve an uncontrolled release of Cr(VI), it is important to protect

employees from the significant exposures that may occur.

Whenever respirators are used to comply with the requirements of the standard, OSHA proposes that the employer implement a comprehensive respiratory protection program in accordance with the Agency's Respiratory Protection standard (29 CFR 1910.134). The respiratory protection program is designed to ensure that respirators are properly used in the workplace, and are effective in protecting workers. The program must include procedures for selecting respirators for use in the workplace; medical evaluation of employees required to use respirators; fit testing procedures for tight-fitting respirators; procedures for proper use of respirators in routine and reasonably foreseeable emergency situations; procedures and schedules for maintaining respirators; procedures to ensure adequate quality, quantity, and flow of breathing air for atmosphere-supplying respirators; training of employees in the proper use of respirators; and procedures for evaluating the effectiveness of the program. In addition, this provision will serve as a reminder to employers covered by the Cr(VI) rule that they must also comply with the Respiratory Protection standard when respirators are provided to employees.

OSHA has proposed to revise the Respiratory Protection standard to include assigned protection factors (68 FR 34036 (6/6/03)). The proposed revision includes a table which indicates the level of respiratory protection that a given respirator or class of respirators is expected to provide, and will apply to employers whose employees use respirators for protection against Cr(VI) when it becomes a final rule (68 FR 34036, 34115 (6/6/03)).

(h) Protective Work Clothing and Equipment

The proposed standard would require that the employer provide protective clothing and equipment at no cost to employees where a hazard is present or is likely to be present from skin or eye contact with Cr(VI). The employer would also be required to ensure that employees use the clothing and equipment provided. The intent of this provision is to prevent the adverse health effects associated with dermal exposure to Cr(VI) (described in Section VI.D of this preamble) and the potential for inhalation of Cr(VI) that may be deposited on employees' street clothing. The proposed requirements for protective clothing and equipment are similar to those in other OSHA health standards such as those for cadmium (29 CFR 1910.1027) and methylenedianiline (29 CFR 1910.1050), and are based upon widely accepted principles and conventional practices of industrial hygiene. The proposed requirements are also consistent with Section 6(b)(7) of the OSH Act which states that, where appropriate, standards shall prescribe suitable protective equipment to be used in connection with hazards.

OSHA has proposed a standard that will cover payment for personal protective equipment in all workplaces (64 FR 15401 (3/31/99)). The Agency is incorporating the record of that rulemaking into the Cr(VI) rulemaking and will give due consideration to all relevant comments.

Criteria for determining when a hazard is present or is likely to be present from skin or eye contact with Cr(VI) are not specified. When evaluating the potential for hazardous eye or skin contact with Cr(VI), OSHA anticipates that the employer will assess the workplace in a manner consistent with the current requirements of the Agency's standards for use of personal protective equipment in general industry (29 CFR 1910.132) and shipyards (29 CFR 1915.152). These standards require the employer to assess the workplace to determine if hazards (including hazards associated with eye and skin contact with chemicals) are present, or are likely to be present.

As described in the non-mandatory appendices providing guidance on hazard assessment for these standards (29 CFR 1910 Subpart I Appendix B; 29 CFR 1915 Subpart I Appendix A), the employer should ''exercise common sense and appropriate expertise'' in assessing hazards. The recommended approach involves a walk-through survey to identify sources of hazards to workers. Review of injury/accident data is also recommended. Information obtained during this process provides a basis for the evaluation of potential hazards.

Based on the results of this assessment, the employer must determine what clothing and equipment is necessary to protect employees from Cr(VI) hazards. The proposed requirement is performance-oriented, and is designed to allow the employer flexibility in selecting the clothing and equipment most suitable for his or her particular workplace. The type of protective clothing and equipment needed to protect employees from Cr(VI) hazards will depend on the potential for exposure and the conditions of use in the workplace. Examples of protective clothing and equipment include, but are not limited to gloves, aprons, coveralls, foot coverings, and goggles. Ordinary street clothing and work uniforms or other accessories that do not serve to protect workers from Cr(VI) hazards are not considered protective clothing and equipment under this proposed standard.

The employer must exercise reasonable judgment in selecting the appropriate clothing and equipment to protect employees from Cr(VI) hazards. This provision is consistent with OSHA's current standards for provision of personal protective equipment (*e.g.*, 29 CFR 1910.132, 29 CFR 1915.152, 29 CFR 1926.95). For example, a worker who is constructing a home foundation using wood treated with chromated copper arsenate, leather gloves may be all that is necessary to prevent hazardous Cr(VI) exposure. In other situations, such as when a worker is performing abrasive blasting on a structure covered with Cr(VI)-containing paint, more extensive measures such as coveralls, head coverings, and goggles may be needed. Where exposures to Cr(VI) are minute, no protective clothing or equipment may be necessary. Many Cr(VI) compounds are acidic or alkaline (*e.g.*, chromic acid, portland cement), and these characteristics may also influence the choice of protective clothing and equipment. For example, a chrome plater may require an apron, gloves, and goggles to protect against possible splashes of chromic acid that could result in both Cr(VI) exposure and chemical burns.

OSHA has not proposed a threshold concentration of Cr(VI) for determining when a substance would be covered under the rule. In some OSHA standards an exemption from certain requirements based on percentage composition has been included. For example, the standard for formaldehyde requires that the employer prevent eye and skin contact with liquids containing one percent or more formaldehyde (29 CFR 1910.1048(h)(1)(i)). Contact with liquids containing less than one percent formaldehyde is exempt from this provision. Such exemptions have been included so that coverage would not be extended to trivial exposures that were not associated with adverse health effects.

A similar exemption has not been included in this proposed standard because adverse health effects have been shown to occur as a result of dermal contact to relatively low concentrations of Cr(VI). For example, exposures to portland cement have been associated with allergic contact dermatitis, even though Cr(VI) concentrations in the cement were reported to be below 10 μg/

g (i.e., 0.001%) (Ex. 35–326). OSHA is not aware of any evidence that would allow establishment of a threshold concentration of Cr(VI) below which adverse dermal effects would not occur.

Paragraph (h)(2) (paragraph (f)(2) of the proposals for construction and shipyards) contains proposed requirements for removal and storage of protective clothing and equipment. The employer must ensure that all protective clothing and equipment contaminated with Cr(VI) is removed at the completion of the work shift or at the completion of tasks involving Cr(VI) exposure. Where employees must change their clothes (i.e., take off their street clothes), removal of protective clothing and equipment must occur in change rooms provided in accordance with paragraph (i) of this section (paragraph (g) of the construction and shipyard proposals). This provision is intended to reduce Cr(VI) contamination of the workplace, and limit Cr(VI) exposures outside the workplace. Wearing contaminated clothing outside the work area could lengthen the duration of exposure, and could carry Cr(VI) from regulated areas to other areas of the workplace. In addition, contamination of personal clothing could result in Cr(VI) being carried to employees' cars and homes, increasing the worker's exposure as well as exposing other individuals to Cr(VI) hazards.

Contaminated protective clothing and equipment must be removed at the end of the work shift or at the completion of tasks involving Cr(VI) exposure, whichever comes first. This language is intended to convey that protective clothing contaminated with Cr(VI) must generally not be worn when tasks involving Cr(VI) exposure have been completed for the day. For example, if employees perform work tasks involving Cr(VI) exposure for the first two hours of a work shift, and then perform tasks that do not involve Cr(VI) exposure, they must remove their protective clothing after the exposure period to avoid the possibility of increasing the duration of exposure and contamination of the work area from Cr(VI) residues on the protective clothing. If, however, employees are performing tasks involving Cr(VI) exposure intermittently throughout the day, or if employees are exposed to other contaminants where their protective clothing and equipment is needed, this provision does not prevent them from wearing the clothing and equipment until the completion of their shift.

To limit exposures outside the workplace, OSHA proposes that the employer ensure that Cr(VI)-contaminated protective clothing and equipment be removed from the workplace only by those employees whose job it is to launder, clean, maintain, or dispose of such clothing or equipment. Furthermore, the proposed standard would require that clothing and equipment that is to be laundered, cleaned, maintained, or disposed of be placed in closed, impermeable containers. This provision is intended to assure that contamination of the change room is minimized and that employees who later handle these items are protected. Those cleaning the Cr(VI)-contaminated clothing and equipment will be further protected by the requirement that warning labels be placed on containers to inform them of the potential hazards of exposure to Cr(VI).

The proposed standard requires that the employer clean, launder, repair and replace protective clothing as needed to ensure that the effectiveness of the clothing and equipment is maintained. This provision is necessary to ensure that clothing and equipment continue to serve their intended purpose of protecting workers. This would also prevent unnecessary exposures outside the workplace from employees taking contaminated clothing and equipment home for cleaning.

In keeping with the performance-orientation of the proposed rule, OSHA does not specify how often clothing and equipment should be cleaned, repaired or replaced. The Agency believes that appropriate time intervals may vary widely based on the types of clothing and equipment used, Cr(VI) exposures, and other circumstances in the workplace. The obligation of the employer, as always, is to keep the clothing and equipment in the condition necessary to perform its protective functions.

Removal of Cr(VI) from protective clothing and equipment by blowing, shaking, or any other means which disperses Cr(VI) in the air would be prohibited. Such actions would result in unnecessary exposure to airborne Cr(VI) as well as possible dermal contact.

The proposal would require that the employer inform any person who launders or cleans protective clothing or equipment contaminated with Cr(VI) of the potentially harmful effects of exposure to Cr(VI), and the need to launder or clean contaminated clothing and equipment in a manner that effectively prevents skin or eye contact with Cr(VI) or the release of airborne Cr(VI) in excess of the PEL. This provision is intended to ensure that persons who clean or launder Cr(VI)-contaminated items are aware of the associated hazards, and can then take appropriate protective measures.

The proposed standard would require employers to provide protective clothing and equipment at no cost to employees. The Agency believes that the employer is generally in the best position to select and obtain the proper type of protective clothing and equipment. OSHA also believes that by providing and owning protective clothing and equipment, the employer will be in a better position to maintain control over the inventory of protective clothing and equipment, conduct periodic inspections, and, when necessary, repair or replace it to maintain its effectiveness. The protective clothing and equipment at issue is designed and intended for work use. As discussed above, employees must remove contaminated clothing and equipment at the end of the work shift or the completion of tasks involving Cr(VI) exposure, whichever comes first. Employees may not remove contaminated clothing and equipment from the worksite, except for the employees whose job it is to launder, clean, maintain, or dispose of such clothing or equipment. The employer is responsible for cleaning or disposing of the protective clothing and equipment and retains complete control over it. The Agency is seeking comment on the proposed provision, and has included this topic in the ''Issues'' section of this preamble.

(i) Hygiene Areas and Practices

The proposed standard would require employers to provide hygiene facilities and to assure employee compliance with basic hygiene practices that serve to minimize exposure to Cr(VI). The proposal includes requirements for change rooms and washing facilities, ensuring that Cr(VI) exposure in eating and drinking areas is minimized, and a prohibition on certain practices that may contribute to Cr(VI) exposure. OSHA believes that strict compliance with these provisions would substantially reduce employee exposure to Cr(VI).

Several of these provisions are presently required under other OSHA standards. For example, OSHA's current standard addressing sanitation in general industry (29 CFR 1910.141) requires that whenever employees are required by a particular standard to wear protective clothing because of the possibility of contamination with toxic materials, change rooms equipped with storage facilities for street clothes and separate storage facilities for protective clothing shall be provided. The sanitation standard also includes

provisions for washing facilities, and prohibits storage or consumption of food or beverages in any area exposed to a toxic material. Similar provisions are in place for construction (29 CFR 1926.51). The hygiene provisions of this paragraph are intended to augment the requirements established under other standards with additional provisions applicable specifically to Cr(VI) exposure.

In workplaces where employees must change their clothes to use protective clothing and equipment, OSHA believes it is essential to have change rooms with separate storage facilities for street and work clothing to prevent contamination of employees' street clothes. This provision will minimize employee exposure to Cr(VI) after the work shift ends, because it reduces the duration of time they may be exposed to contaminated work clothes. Potential exposure resulting from contamination of the homes or cars of employees is also avoided. Change rooms also provide employees with privacy while changing their clothes. OSHA intends the proposed requirement for change rooms to apply to all covered workplaces where employees must change their clothes (i.e., take off their street clothes) to use protective clothing and equipment. In those situations where removal of street clothes would not be necessary (*e.g.*, in a workplace where only gloves are used as protective clothing), change rooms would not be required.

Paragraph (i)(3) (paragraph (g)(3) of the proposals for construction and shipyards) contains proposed requirements for washing facilities. The employer is to provide readily accessible washing facilities capable of removing Cr(VI) from the skin and is to ensure that affected employees use these facilities when necessary. Also, the employer is to ensure that employees who have skin contact with Cr(VI) wash their hands and faces at the end of the work shift and prior to eating, drinking, smoking, chewing tobacco or gum, applying cosmetics, or using the toilet.

Washing reduces exposure by diminishing the period of time that Cr(VI) is in contact with the skin. Although engineering and work practice controls and protective clothing and equipment are designed to prevent hazardous skin and eye contact from occurring, OSHA realizes that in some circumstances these exposures will occur. For example, a worker who wears gloves to protect against hand contact with Cr(VI) may inadvertently touch his face with the contaminated glove during the course of the day. The intent of this provision is to have employees wash in order to mitigate the adverse effects when skin and eye contact does occur. At a minimum, employees are to wash their hands and faces at the end of the shift because washing is needed to remove any residual Cr(VI) contamination. Likewise, washing prior to eating, drinking, smoking, chewing tobacco or gum, applying cosmetics or using the toilet also protects against further Cr(VI) exposure.

OSHA has made a preliminary determination that washing facilities would be sufficient to allow employees to remove significant levels of Cr(VI) contamination that may occur under the proposed standard. A requirement for provision and use of showers has not been included in the proposal. Some other health standards, such as the standards for cadmium (29 CFR 1910.1027) and lead (29 CFR 1910.1025), have included requirements for showers. OSHA requests information and comment as to whether provisions for showers should be included in a final Cr(VI) standard, and has included this topic in the ''Issues'' section of this preamble.

To minimize the possibility of food contamination and to reduce the likelihood of additional exposure to Cr(VI) through inhalation or ingestion, OSHA believes it is imperative that employees have a clean place to eat. Where the employer chooses to allow employees to eat at the facility, the proposal would require the employer to ensure that eating and drinking areas and surfaces are maintained as free as practicable of Cr(VI). Employers would also be required to assure that employees do not enter eating or drinking areas wearing protective clothing, unless properly cleaned beforehand. This is to further minimize the possibility of contamination and reduce the likelihood of additional Cr(VI) exposure from contaminated food or beverages. Employers are given discretion to choose any method for removing surface Cr(VI) from clothing and equipment that does not disperse the dust into the air or onto the employee's body. For example, if a worker is wearing coveralls for protection against Cr(VI) exposure, thorough HEPA vacuuming of the coveralls could be performed prior to entry into a lunchroom.

The employer is not required to provide eating and drinking facilities to employees. Employees may consume food or beverages off the worksite. However, where the employer chooses to allow employees to consume food or beverages at a worksite where Cr(VI) is present, OSHA intends to ensure that employees are protected from Cr(VI) exposures in these areas.

Proposed paragraph (i)(5) (paragraph (g)(5) in the construction and shipyard proposals) specifies certain activities that would be prohibited. These activities would include eating, drinking, smoking, chewing tobacco or gum, or applying cosmetics in regulated areas, or in areas where skin or eye contact occurs. Products associated with these activities, such as food and beverages, could not be carried or stored in these areas. This provision is intended to protect employees from additional sources of exposure to Cr(VI). Because the construction and shipyard proposals do not include requirements for regulated areas, reference to regulated areas is omitted in the proposed regulatory text for these standards.

(j) Housekeeping

The proposed standard includes housekeeping provisions that would require the employer to maintain surfaces as free as practicable of Cr(VI), promptly clean Cr(VI) spills and leaks, use appropriate cleaning methods, and properly dispose of Cr(VI)-contaminated waste. These provisions are exceptionally important because they minimize additional sources of exposure that engineering controls generally are not designed to address. Good housekeeping is a cost effective way to control employee exposures by removing accumulated Cr(VI) that can become entrained by physical disturbances or air currents and carried into an employee's breathing zone, thereby increasing employee exposure. Contact with contaminated surfaces may also result in dermal exposure to Cr(VI). The proposed provisions are consistent with housekeeping requirements in other OSHA standards, such as those for cadmium (29 CFR 1910.1027) and lead (29 CFR 1910.1025).

Cr(VI) deposited on ledges, equipment, floors, and other surfaces should be removed as soon as practicable, to prevent it from becoming airborne and to minimize the likelihood that skin contact will occur. When Cr(VI) is released into the workplace as a result of a leak or spill, the proposal would require the employer to promptly clean up the spill. Measures for clean-up of liquids should provide for the rapid containment of the leak or spill to minimize potential exposures. Clean-up procedures for dusts must not disperse the dust into the workplace air. These work practices aid in minimizing the number of employees exposed, as well as the extent of any potential Cr(VI) exposure.

The proposed standard would require that, where possible, surfaces contaminated with Cr(VI) be cleaned by vacuuming or other methods that minimize the likelihood of Cr(VI) exposure. OSHA believes vacuuming to be the most reliable method of cleaning surfaces on which dust accumulates, but equally effective methods may be used. Shoveling, dry or wet sweeping, and brushing would be permitted only if the employer shows that vacuuming or other methods that are usually as efficient as vacuuming are not effective under the particular circumstances found in the workplace. The proposal would also require that vacuum cleaners be equipped with HEPA filters to prevent the dispersal of Cr(VI) into the workplace. The use of compressed air for cleaning would only be allowed when used in conjunction with a ventilation system designed to capture the dust cloud created by the compressed air. This provision is also intended to prevent the dispersal of Cr(VI) into the workplace.

Cleaning equipment is to be handled in a manner that minimizes the reentry of Cr(VI) into the workplace. For example, cleaning and maintenance of HEPA-filtered vacuum equipment should be done carefully to avoid exposures to Cr(VI). Filters need to be changed and the contents of bags disposed of properly to avoid unnecessary Cr(VI) exposures.

The proposal would also require that items contaminated with Cr(VI) and consigned for disposal be collected and disposed of in sealed impermeable bags or other closed impermeable containers. These containers would include warning labels to inform individuals who handle these items of the potential hazards. By alerting employers and employees who are involved in disposal to the potential hazards of Cr(VI) exposure, they will be better able to implement protective measures.

No housekeeping provision has been included in the proposals covering construction or shipyards. OSHA has made a preliminary determination that a specific housekeeping provision is not appropriate because of the difficulties of performing housekeeping related to Cr(VI) exposure in the construction and shipyard environments. For example, in shipyard and particularly in construction work environments the generally dusty nature of outdoor work settings is likely to make it difficult to distinguish Cr(VI)—contaminated dusts from other dirt and dusts commonly found at the work site. The same control measures that apply to general industry are likely to be more difficult to implement and burdensome in these environments.

This preliminary determination differs from OSHA's determination in the standards for lead in construction (29 CFR 1926.62) and cadmium in construction (29 CFR 1926.1127), where the Agency included housekeeping provisions. In these rulemakings, OSHA did not find housekeeping provisions to present the difficulties anticipated with Cr(VI). The Agency believes that Cr(VI)-contaminated dusts will not generally be as easily identified as lead- or cadmium-contaminated dusts. Welding, in particular, could result in deposition of minute quantities of Cr(VI) that would be difficult for a construction or shipyard employer to identify. OSHA seeks comment on this preliminary finding, and has included this topic in the ''Issues'' section of this preamble.

Construction and shipyard employers would still need to comply with the general housekeeping requirements found at 29 CFR 1926.25 (for construction) or 29 CFR 1915.91 (for shipyards). These standards include general provisions for keeping workplaces clear of debris, but do not contain the more specific requirements found in the proposed Cr(VI) standard for general industry (such as those addressing cleaning methods) that are designed to limit Cr(VI) contamination of the workplace.

(k) Medical Surveillance

OSHA proposes to require that each employer covered by this rule make medical surveillance available at no cost, and at a reasonable time and place, for all employees who are experiencing signs or symptoms of the adverse health effects associated with Cr(VI) exposure, or who are exposed in an emergency. In addition, general industry employers would be required to provide medical surveillance for all employees exposed to Cr(VI) at or above the PEL for 30 or more days a year. The required medical surveillance must be performed by or under the supervision of a physician or other licensed health care professional.

The purpose of medical surveillance for Cr(VI) is, where reasonably possible, to determine if an individual can be exposed to the Cr(VI) present in his or her workplace without experiencing adverse health effects; to identify Cr(VI)-related adverse health effects so that appropriate intervention measures can be taken; and to determine the employee's fitness to use personal protective equipment such as respirators. The proposal is consistent with Section 6(b)(7) of the OSH Act which requires that, where appropriate, medical surveillance programs be included in OSHA health standards to aid in determining whether the health of workers is adversely affected by exposure to toxic substances. Other OSHA health standards have also included medical surveillance requirements.

The proposed standard is intended to encourage participation by requiring that medical examinations be provided by the employer without cost to employees (also required by section 6(b)(7) of the Act), and at a reasonable time and place. If participation requires travel away from the worksite, the employer would be required to bear the cost. Employees would have to be paid for time spent taking medical examinations, including travel time. OSHA is proposing that medical surveillance be provided to employees in general industry exposed at or above the PEL for 30 or more days a year in order to focus on those workers at greatest risk. Employees exposed below the PEL, or exposed for only a few days in a year, will be at lower risk of developing Cr(VI)-related disease. OSHA believes that these cutoffs, based both on exposure level and on the number of days an employee is exposed to Cr(VI), are a reasonable and administratively convenient basis for providing medical surveillance benefits to Cr(VI)-exposed workers. In past health standards, OSHA has used 30 days above the action level for triggering medical surveillance. Because of the large reduction in the PEL down to 1 μg/m^3 OSHA believes that 30 days above the PEL may be more reasonable since exposures above the PEL are more likely to result in adverse health effects that might benefit from medical surveillance. OSHA is seeking comment on the appropriateness of this trigger for medical surveillance, and whether the Agency should consider a trigger at the action level or an alternative trigger.

OSHA has not included exposure above the PEL for 30 or more days per year as a trigger for medical surveillance in the construction or shipyard Cr(VI) proposals. As discussed earlier, OSHA has not proposed to require exposure monitoring for construction or shipyard employment because of the difficulties in conducting such monitoring in these work settings. While OSHA assumes that some monitoring will be conducted in order for employers to know when or if they are above the PEL, OSHA also assumes that certain employers will not conduct exposure monitoring and may choose to presume that certain work processes or practices are above the PEL or rely on historical or objective data to show exposure levels. However, if medical surveillance for individual

employees is triggered by exposures above the PEL for 30 days or more, these employers would be forced to do monitoring in order to determine which employees are exposed above the PEL for 30 days or more. This would have the effect of re-introducing an exposure monitoring burden that the Agency is attempting to relieve.

Some employees may exhibit signs and symptoms of the adverse health effects associated with Cr(VI) exposure even when not exposed above the PEL for 30 or more days per year. These employees could be especially sensitive, may have been unknowingly exposed, or may have been exposed to greater amounts than the exposure assessment suggests. OSHA has therefore proposed that employees who experience signs or symptoms of the adverse health effects associated with Cr(VI) exposure be subject to medical surveillance. Signs and symptoms that may warrant surveillance include dermatitis, chrome holes, and nasal septum ulcers or perforations. Thus, the proposal would protect all employees exposed to Cr(VI) in unusual circumstances even if they fall outside the criteria for routine medical surveillance.

Appropriate surveillance would be required to be made available for employees exposed in an emergency regardless of the airborne concentrations of Cr(VI) normally found in the workplace. Emergency situations involve uncontrolled releases of Cr(VI), and the significant exposures that occur in these situations justify a requirement for medical surveillance. The proposed requirement for medical examinations after exposure in an emergency is consistent with the provisions of several other OSHA health standards, including the standards for methylenedianiline (29 CFR 1910.1050), butadiene (29 CFR 1910.1051), and methylene chloride (29 CFR 1910.1052).

OSHA has made a preliminary determination not to include eye or skin contact as a basis for medical surveillance. OSHA believes that compliance with the proposed provisions for protective work clothing and equipment, hygiene areas and practices, and other protective measures will minimize the potential for adverse eye and skin effects. When such health effects occur, OSHA believes that trained employees will be able to detect these conditions, report them to their employer, and obtain medical assistance. In such situations, affected employees would be provided medical surveillance on the basis that they are experiencing signs or symptoms of Cr(VI)-related health effects.

OSHA has proposed that the medical examinations provided under the rule be performed by or under the supervision of a physician or other licensed health care professional (PLHCP). The Agency considers it appropriate to allow any professional to perform medical examinations and procedures provided under the standard when they are licensed by state law to do so. This provision provides flexibility to the employer, and would reduce cost and compliance burdens. The proposed requirement is consistent with the approach of other recent OSHA standards, such as those for methylene chloride (29 CFR 1910.1052), bloodborne pathogens (29 CFR 1910.1030), and respiratory protection (29 CFR 1910.134).

The proposed standard also specifies how frequently medical examinations are to be offered to those employees covered by the medical surveillance program. Employers would be required to provide all covered employees with medical examinations whenever an employee shows signs or symptoms of Cr(VI) exposure; within 30 days after an emergency resulting in an uncontrolled release of Cr(VI); and within 30 days after a PLHCP's written medical opinion recommends an additional examination. In addition, employers in general industry would be required to provide covered employees with examinations within 30 days after initial assignment unless the employee has received a medical examination provided in accordance with the standard within the past 12 months; annually; and at the termination of employment, unless an examination has been given less than six months prior to the date of termination.

Signs or symptoms may indicate that adverse health effects attributable to Cr(VI) exposure are occurring. In such situations OSHA believes it would be appropriate to evaluate the employee's condition to determine if exposure to Cr(VI) is the cause of the condition, and to determine if protective measures are necessary. Emergency situations may involve high or unknown exposures, and OSHA believes that a medical examination is necessary to evaluate the possible adverse effects of these exposures.

In addition to medical evaluations after exposures in an emergency or when signs or symptoms occur, OSHA is proposing that additional examinations be offered following a PLHCP's recommendation that additional exams are necessary. A PLHCP may recommend additional evaluations in order to follow developments in a worker's condition, or to allow for specialized evaluation. For example, if nasal ulceration is identified in a Cr(VI)-exposed worker, a PLHCP may recommend follow-up examinations to ensure that treatment and workplace interventions are successful in addressing the condition, or a worker who exhibits dermatitis may be referred to a dermatologist for testing to determine if they are sensitized to Cr(VI).

The proposed requirement for general industry that a medical examination be offered at the time of initial assignment is intended to achieve the objective of determining if an individual will be able to work in the job involving Cr(VI) exposure without adverse effects. It also serves the useful function of establishing a health baseline for future reference. Where an examination that complies with the requirements of the standard has been provided in the past 12 months, that previous examination would serve these purposes, and an additional examination would not be needed.

OSHA believes that the provision of medical surveillance on an annual basis in general industry is an appropriate frequency for screening employees for Cr(VI)-related diseases. The main goal of periodic medical surveillance for workers is to detect adverse health effects at an early and potentially reversible stage. The proposed requirement for annual examinations is consistent with other OSHA health standards, including those for cadmium (29 CFR 1910.1027), formaldehyde (29 CFR 1910.1048), and methylene chloride (29 CFR 1910.1052). Based on the Agency's experience, OSHA believes that annual surveillance would strike a reasonable balance between the need to diagnose health effects at an early stage, and the limited number of cases likely to be identified through surveillance. The proposed requirement for general industry that the employer offer a medical examination at the termination of employment is intended to assure that no employee terminates employment while carrying an active, but undiagnosed, disease.

The examination to be provided by the PLHCP is to consist of a medical and work history; a physical examination of the skin and respiratory tract; and any additional tests considered appropriate by the PLHCP. Special emphasis is placed on the portions of the medical and work history focusing on Cr(VI) exposure, health effects associated with Cr(VI) exposure, and smoking. The physical exam focuses on organs and systems known to be susceptible to Cr(VI) toxicity. The information obtained will allow the PLHCP to assess

the employee's health status, identify adverse health effects related to Cr(VI) exposures, and determine if limitations should be placed on the employee's exposure to Cr(VI).

The proposal does not indicate specific tests that must be included in the medical examination. OSHA does not believe that any particular tests are generally applicable to all employees covered by the medical surveillance requirements, and the Agency proposes to give the examining PLHCP the flexibility to determine any appropriate tests to be selected for a given employee. For example, tests for dermal sensitization exist, but they are not recommended as a screening tool because they are capable of sensitizing persons who had not been affected previously. These tests should be considered by the PLHCP if a medical history indicating probable sensitization exists or if the employee experiences signs or symptoms indicative of sensitization. Radiological examinations and pulmonary function tests may also be useful in evaluating possible effects of Cr(VI). OSHA believes that the PLHCP is in the best position to decide which medical tests are necessary for each individual examined. Where specific tests are deemed appropriate by the PLHCP, the proposed standard would require that they be provided.

OSHA is aware that certain methods are available for evaluating Cr(VI) exposures based on analysis of chromium in urine or blood. However, the Agency is not aware of evidence indicating that these methods adequately characterize Cr(VI) exposures in most occupational environments. OSHA has also found no medical justification for routine urine or blood analysis for the detection of Cr(VI)-related health effects. Therefore, no requirement for such analysis is proposed.

The proposed standard would require the employer to ensure the PLHCP has a copy of the standard, and to provide the following information: a description of the affected employee's former and current duties as they relate to Cr(VI) exposure; the employee's former, current, and anticipated exposure level; a description of any personal protective equipment used or to be used by the employee, including when and for how long the employee has used that equipment; and information from records of employment-related medical examinations previously provided to the affected employee, currently within the control of the employer. Making this information available to the PLHCP will aid in the evaluation of the employee's health in relation to assigned duties and fitness to use personal protective equipment, when necessary.

The results of exposure monitoring are part of the information that would be supplied to the PLHCP responsible for medical surveillance. These results contribute valuable information to assist the PLHCP in determining if an employee is likely to be at risk of harmful effects from Cr(VI) exposure. A well-documented exposure history would also assist the PLHCP in determining if a condition (*e.g.*, dermatitis) may be related to Cr(VI) exposure.

The proposed rule would require employers to obtain from the examining PLHCP a written opinion containing the results of the medical examination with regard to Cr(VI) exposure, the PLHCP's opinion as to whether the employee would be placed at increased risk of material health impairment as a result of exposure to Cr(VI), and any recommended limitations on the employee's exposure or use of personal protective equipment. The PLHCP would also need to state in the written opinion that these findings were explained to the employee. The purpose of requiring the PLHCP to supply a written opinion to the employer is to provide the employer with a medical basis to aid in the determination of placement of employees and to assess the employee's ability to use protective clothing and equipment. The employer must obtain the written opinion within 30 days of the examination; OSHA believes this will provide the PLHCP sufficient time to receive and consider the results of any tests included in the examination, and allow the employer to take any necessary protective measures in a timely manner. The proposed requirement that the opinion be in written form is intended to ensure that employers and employees have the benefit of this information.

The PLHCP would not be allowed to include findings or diagnoses which are unrelated to Cr(VI) exposure in the written opinion provided to the employer. OSHA has proposed this provision to reassure employees participating in medical surveillance that they will not be penalized or embarrassed by the employer's obtaining information about them not directly pertinent to Cr(VI) exposure. The employee would be informed directly by the PLHCP of all results of his or her medical examination, including conditions of non-occupational origin. The employer would also be required to provide a copy of the PLHCP's written opinion to the employee within two weeks after receiving it, to ensure that the employee has been informed of the result of the examination in a timely manner.

In some OSHA health standards, a provision for medical removal protection (MRP) has been included. MRP typically requires that the employer temporarily remove an employee from exposure when such an action is recommended in a written medical opinion. During the time of removal, the employer is required to maintain the total normal earnings, as well as all other employee rights and benefits. However, MRP is not intended to serve as a worker's compensation system. The primary reason MRP has been included in these previous standards has been to encourage employee participation in medical surveillance. By protecting employees who are removed on a temporary basis from economic loss, this potential disincentive to participating in medical surveillance is alleviated.

The proposed rule does not include a provision for MRP, because OSHA has made a preliminary determination that MRP is not reasonably necessary or appropriate for Cr(VI)-related health effects. The Agency believes that Cr(VI)-related health effects generally fall into one of two categories: Either they are chronic conditions that temporary removal from exposure will not remedy (*e.g.*, lung cancer, respiratory or dermal sensitization), or they are conditions that can be addressed through proper application of control measures and do not require removal from exposure (*e.g.*, irritant dermatitis). Since situations where temporary removal would be appropriate are not anticipated to occur, OSHA does not believe that MRP is necessary. The Agency seeks comment on this preliminary determination, and has included this topic in the ''Issues'' section of this preamble.

(1) Communication of Hazards to Employees

The proposed standard includes requirements intended to ensure that the dangers of Cr(VI) exposure are communicated to employees by means of signs, labels, and employee information and training. These proposed requirements would parallel the existing requirements of OSHA's Hazard Communication standard (29 CFR 1910.1200). The hazard communication requirements of the proposed rule are designed to be substantively as consistent as possible with the Hazard Communication standard, while including additional specific requirements needed to protect employees exposed to Cr(VI).

The proposed standard would require that all approaches to regulated areas be

posted with legible and readily visible warning signs stating: Danger; Chromium (VI); Cancer Hazard; Can Damage Skin, Eyes, Nasal Passages, and Lungs; Authorized Personnel Only; Respirators Required in this Area. Such warning signs would be required wherever a regulated area exists, that is, wherever the PEL is exceeded in general industry. Because the construction and shipyard proposals do not include requirements for regulated areas, no provision is included for warning signs in the proposed regulatory text for the construction and shipyard standards.

The signs are intended to serve as a warning to employees who otherwise may not be aware that they are entering a regulated area, and to remind employees of the hazards of Cr(VI) so that they take necessary protective steps before entering the area. These signs are intended to supplement the training that employees receive regarding the hazards of Cr(VI), since even trained employees need to be reminded of the locations of regulated areas and of the precautions necessary before entering these dangerous areas.

In some instances, regulated areas are permanent, because the employer is unable to reduce Cr(VI) exposures in that area below the PEL with engineering controls. In those cases, the signs serve to warn employees not to enter the area unless they are authorized and are wearing respirators. In other cases, such as emergency situations and maintenance operations, regulated areas may be established temporarily. The use of warning signs is particularly important in these situations to make employees who are regularly scheduled to work at these sites aware of the hazards. Access is limited to authorized personnel to ensure that those entering the area are adequately trained and equipped, and to limit exposure to only those whose presence is absolutely necessary.

The proposed standard specifies the wording of the warning signs for regulated areas in order to ensure that the proper warning is given to employees. OSHA believes that the use of the word ''Danger'' is appropriate, based on the evidence of the toxicity and carcinogenicity of Cr(VI). ''Danger'' is used to attract the attention of workers in order to alert them to the fact that they are entering an area where the PEL may be exceeded and to emphasize the importance of the message that follows. The use of the word ''Danger'' is also consistent with other OSHA health standards dealing with carcinogens such as cadmium (29 CFR 1910.1027), methylenedianiline (29 CFR 1910.1050), asbestos (29 CFR 1910.1001), and benzene (29 CFR 1910.1028).

The proposed standard would also require that the sign indicate that respirators are required in the area. Regulated areas are areas demarcated by the employer where the employee's exposure to airborne concentrations of chromium (VI) exceeds, or can reasonably be expected to exceed the PEL (definition of a regulated area). The employer has made the determination that such areas are regulated on the basis of his/her own exposure assessments of the employees in the area. Since the employer has determined that such areas are not able to be reduced below the PEL, respirators are required as a means of control to protect the employees in those areas. The sign also serves as a means to warn other employees not in the regulated area not to enter, or if those other employees enter the area, they need to protect themselves in situations where excessive exposures can occur.

The proposal would require that warning labels be affixed to all bags or containers of contaminated clothing and equipment that are to be removed from the workplace for laundering, cleaning, or maintenance. Containers of waste, scrap, debris, and any other materials contaminated with Cr(VI) that are consigned for disposal would also need to be labeled. The labels must state: Danger; Contains Chromium (VI); Cancer Hazard; Can Damage Skin, Eyes, Nasal Passages, and Lungs. The purpose of this requirement is to ensure that all affected employees, not only those of a particular employer, are apprised of the hazardous nature of Cr(VI) exposure. These proposed requirements are consistent with the mandate of Section (6)(b)(7) of the OSH Act, which requires that OSHA health standards prescribe the use of labels or other appropriate forms of warning to apprise employees of the hazards to which they are exposed. Because the construction and shipyard proposals do not include disposal requirements, no provision is included in the construction and shipyard proposals for placing warning labels on containers of waste, scrap, debris, and other materials contaminated with Cr(VI).

Information and training is essential to inform employees of the hazards to which they are exposed and to provide employees with the necessary understanding of the degree to which they themselves can minimize potential health hazards. As part of an overall hazard communication program, training serves to explain and reinforce the information presented on labels and in material safety data sheets. These written forms of communication will be successful and relevant only when employees understand the information presented and are aware of the actions to be taken to avoid or minimize exposures, thereby reducing the possibility of experiencing adverse health effects.

OSHA proposes that employers provide training for all employees who are exposed to airborne Cr(VI) or who have skin or eye contact with Cr(VI), ensure that employees participate in the training, and maintain a record of the training provided. Training would be provided to all employees exposed to Cr(VI), and would not be limited to only those exposed above the PEL or action level. This proposed requirement is consistent with the Hazard Communication standard (29 CFR 1910.1200), which requires training for all employees exposed to hazardous chemicals and defines this to include potential (*e.g.*, accidental or possible) exposure. This training would allow employees to make efforts to avoid exposures altogether or mitigate those exposures that do occur.

The employer is to provide initial training prior to or at the time of initial assignment to a job involving potential exposure to Cr(VI). An employer who is able to demonstrate that a new employee has received training within the last 12 months is allowed to use that training for purposes of initial training required by the standard, provided the previous training has addressed the elements specified in the training provisions of the proposal, and the employee is able to demonstrate knowledge of those elements. In cases where understanding of some elements is lacking or inadequate, the employer would be required to provide training only in those elements. This allowance for prior training is intended to ensure that employees receive sufficient training, without requiring unnecessary repetition of that training.

The training requirements in this standard are performance-oriented. The proposed standard lists the subjects that must be addressed in training, but not the specific ways that this is to be accomplished. Hands-on training, videotapes, slide presentations, classroom instruction, informal discussions during safety meetings, written materials, or any combination of these methods may be appropriate. Such performance-oriented requirements are intended to encourage employers to tailor training to the needs of their workplaces, thereby resulting in the most effective training program in each specific workplace.

OSHA believes that the employer is in the best position to determine how the training can most effectively be accomplished. The Agency has therefore laid out the objectives to be met to ensure that employees are made aware of the hazards associated with Cr(VI) in their workplace and how they can help to protect themselves. The specifics regarding how this is to be achieved are left up to the employer.

In order for the training to be effective, the employer must ensure that it is provided in a manner that the employee is able to understand. Employees have varying educational levels, literacy, and language skills, and the training must be presented in a language and at a level of understanding that accounts for these differences in order to meet the proposed requirement that individuals being trained understand the specified elements. This may mean, for example, providing materials, instruction, or assistance in Spanish rather than English if the workers being trained are Spanish-speaking and do not understand English. The employer would not be required to provide training in the employee's preferred language if the employee understood both languages; as long as the employee is able to understand the language used, the intent of the proposed standard would be met.

In order to ensure that employees comprehend the material presented during training, it is critical that trainees have the opportunity to ask questions and receive answers if they do not fully understand the material that is presented to them. When videotape presentations or computer-based programs are used, this requirement may be met by having a qualified trainer available to address questions after the presentation, or providing a telephone hotline so that trainees will have direct access to a qualified trainer.

Under the proposal, the employer would be required to ensure that each employee can demonstrate knowledge of the specified elements. This could be determined through methods such as discussion of the required training subjects, written tests, or oral quizzes.

The frequency of training under the proposed standard would be determined by the needs of the workplace. Individuals would need to be trained sufficiently to understand the specified elements. Additional training is needed periodically to refresh and reinforce the memories of individuals who have previously been trained, and to ensure that these individuals are informed of new developments in the workplace that may result in new or additional exposures to Cr(VI). For example, training after new control measures are implemented would generally be necessary in order to ensure that employees are able to properly use the new controls that are introduced. Employees would likely be unfamiliar with new work practices undertaken, with the operation of new engineering controls, or the use of new personal protective equipment; training would rectify this lack of understanding. Additional training would ensure that employees are able to actively participate in protecting themselves under the conditions found in the workplace, even if those conditions change.

(m) Recordkeeping

The proposed standard for general industry would require employers to maintain exposure monitoring, medical surveillance, and training records. Because the proposed construction and shipyard standards do not include requirements for exposure monitoring, no provision for retention of exposure monitoring records is included in the proposed regulatory texts for construction and shipyards. However, the record retention requirements of OSHA's standard on access to medical and exposure records (29 CFR 1910.1020) apply to any exposure records that construction and shipyard employers produce.

The recordkeeping requirements are proposed in accordance with section 8(c) of the OSH Act, which authorizes OSHA to require employers to keep and make available records as necessary or appropriate for the enforcement of the Act or for developing information regarding the causes and prevention of occupational injuries and illnesses. The proposed recordkeeping provisions are also consistent with the OSHA's standard addressing access to employee exposure and medical records (29 CFR 1910.1020).

The proposal would require that records be kept of environmental monitoring results that identify the monitored employee and accurately reflect the employee's exposure. The employer would be required to keep records for each exposure measurement taken. Specifically, records must include the following information: The date of measurement for each sample taken; the operation involving exposure to Cr(VI) that was monitored; sampling and analytical methods used and evidence of their accuracy; the number, duration, and results of samples taken; the type of personal protective equipment used; and the name, social security number, and job classification of all employees represented by the monitoring, indicating which employees were actually monitored.

Most of OSHA's substance-specific standards require that exposure monitoring and medical surveillance records include the employee's social security number. OSHA has included this requirement in the past because social security numbers are particularly useful in identifying employees, since each number is unique to an individual for a lifetime and does not change when an employee changes employers. When employees have identical or similar names, identifying employees solely by name makes it difficult to determine to which employee a particular record pertains. However, based on privacy concerns, OSHA is examining alternatives to requiring social security numbers for employee identification. In its Standards Improvement Project proposal, the Agency requested public comment on the necessity, usefulness, and effectiveness of social security numbers as a means of identifying employee records, and any privacy concerns or issues raised by this requirement, as well as the availability of other effective methods of identifying employees for OSHA recordkeeping purposes (67 FR 66493 (19/31/02)). OSHA intends for the requirements of the Cr(VI) standard to conform with any final determination made through the Standards Improvement Project.

The proposal would allow the employer to rely on Cr(VI) monitoring results obtained in the past 12 months when the data were obtained during operations conducted under workplace conditions closely resembling the employer's current operations. Where historical monitoring data are used, the proposal would require that records of these data be maintained. The records of historical data must demonstrate that exposures on a particular job will be below the action level by showing that the work being performed, Cr(VI)-containing material being handled, and environmental conditions at the time the historical data were obtained are the same as those on the job for which monitoring was not performed. The records must also demonstrate that the data were obtained using a method sufficiently accurate to be allowed under the standard. Other data relevant to operations, materials, processing, or employee exposures must also be included in records.

A provision allowing the use of objective data in place of initial monitoring is included in this proposed standard. Objective data are information demonstrating that a particular product or material cannot release Cr(VI) in

concentrations at or above the action level under any expected conditions of use, even under conditions of worst-case release. Where objective data are used to satisfy initial monitoring requirements, the proposal would require employers to establish and maintain accurate records of the objective data relied upon. Since the use of objective data exempts the employer from requirements for conducting periodic monitoring and certain other provisions of the proposal due to the low level of potential exposure, it is critical that this determination be carefully documented. The record would be required to include identification of the Cr(VI)-containing material in question; the source of the objective data; the testing protocol and results of testing, or analysis of the material for the release of Cr(VI); a description of the operation exempted from initial monitoring and how the data support the exemption; and any other data relevant to the operations, materials, processing or employee exposures covered by the exemption.

Compliance with the requirement to maintain a record of objective data protects the employer at later dates from the contention that initial monitoring was not conducted in an appropriate manner. The record would also be available to employees so that they can examine the determination made by the employer. The employer would be required to maintain the record for the duration of the employer's reliance upon the objective data.

In addition to records relating to employee exposures to Cr(VI), the proposal would require the employer to establish and maintain an accurate medical surveillance record for each employee subject to the medical surveillance requirements of the standard. OSHA believes that medical records, like exposure records, are necessary and appropriate both to the enforcement of the standard and to the development of information regarding the causes and prevention of occupational illnesses. Good medical records, including the record of the examination at termination of employment itself, can be useful to the Agency and others in enumerating illnesses and deaths attributable to Cr(VI), in evaluating compliance programs, and in assessing the accuracy of the Agency's risk estimates. Furthermore, medical records are necessary for the proper evaluation of the employee's health.

The medical surveillance records would be required to include the following information: The name, social security number, and job classification of the employee; a copy of the PLHCP's written opinions; and a copy of the information provided to the PLHCP. This information includes the employee's duties as they relate to Cr(VI) exposure, Cr(VI) exposure levels, and descriptions of personal protective equipment used by the employee.

The employer would be required under the proposal to maintain records of employees' Cr(VI)-related training. At the completion of training, the employer would be required to prepare a record that indicates the identity of the individuals trained and the date the training was completed. The record would need to be maintained for three years after the completion of training. In addition, the employer would need to provide materials relating to employee information and training to OSHA or NIOSH, if requested.

OSHA believes that a three year retention period for training records is reasonable. Since OSHA is not proposing specific intervals for periodic retraining, but is making retraining contingent upon the need to maintain employee understanding of safe use and handling of Cr(VI) and workplace changes which result in significant increases in employee exposures to Cr(VI), it is appropriate to have records of training to allow employers to determine when and how employees have been trained. The proposed requirement to provide training materials upon request is necessary to allow for evaluation of training programs, and is consistent with the other OSHA standards such as those for bloodborne pathogens (29 CFR 1910.1030) methylene chloride (29 CFR 1910.1052), butadiene (29 CFR 1910.1051), and methylenedianiline (29 CFR 1910.1050).

All medical and exposure records developed under the Cr(VI) rule would be made available to employees and their designated representatives in accordance with OSHA's standard on access to records (29 CFR 1910.1020). The medical and exposure records standard requires that exposure records be kept for at least 30 years and that medical records be kept for the duration of employment plus thirty years. It is necessary to keep these records for extended periods because of the long latency period commonly associated with cancer. Cancer often cannot be detected until 20 or more years after first exposure. The extended record retention period is therefore needed because diagnosis of disease in employees is assisted by, and in some cases can only be made by, having present and past exposure data as well as the results of present and past medical examinations.

(n) Dates

OSHA proposes that the final Cr(VI) rule become effective 60 days after its publication in the **Federal Register**. This period is intended to allow affected employers the opportunity to familiarize themselves with the standard. Employer obligations to comply with most requirements of the final rule would begin 90 days after the effective date (150 days after publication of the final rule). This is designed to allow employers sufficient time to complete initial exposure assessments, establish regulated areas, obtain appropriate work clothing and equipment, and comply with other provisions of the rule.

Additional time would be allowed for the employer to establish change rooms and to implement engineering controls. Change rooms would be required no later than one year after the effective date of the standard, and engineering controls would need to be in place within two years after the effective date. This is to allow affected employers sufficient time to design and construct change rooms (where necessary), and to design, obtain, and install the necessary control equipment. OSHA solicits comment on the adequacy of these proposed start-up dates. In particular, the Agency is aware that in some cases employers may be required to reevaluate modified ventilation systems for compliance with regulations governing discharges of Cr(VI) to the environment. OSHA would like to ensure that employers are provided sufficient time to complete this process, and has included this topic in the ''Issues'' section of this preamble.

XVIII. Authority and Signature

This document was prepared under the direction of John L. Henshaw, Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, 200 Constitution Avenue, NW., Washington, DC 20210.

The Agency issues the proposed sections under the following authorities: Sections 4, 6(b), 8(c), and 8(g) of the Occupational Safety and Health Act of 1970 (29 U.S.C. 653, 655, 657); section 107 of the Contract Work Hours and Safety Standards Act (the Construction Safety Act) (40 U.S.C. 333); section 41, the Longshore and Harbor Worker's Compensation Act (33 U.S.C. 941); Secretary of Labor's Order No. 5–2002 (67 FR 65008); and 29 CFR Part 1911.

List of Subjects in 29 CFR Parts 1910, 1915, 1917, 1918, and 1926

Cancer, Chemicals, Hazardous substances, Health, Occupational safety and health, Reporting and recordkeeping requirements.

Signed at Washington, DC, this 21st day of September, 2004.

John L. Henshaw,

Assistant Secretary of Labor.

XIX. Proposed Standards

Chapter XVII of Title 29 of the Code of Federal Regulation is proposed to be amended as follows:

PART 1910—[AMENDED]

Subpart Z—[Amended]

1. The authority citation for Subpart Z of Part 1910 is revised to read as follows:

Authority: Secs. 4, 6, 8 of the Occupational Safety and Health Act of 1970 (29 U.S.C. 653, 655, 657: Secretary of Labor's Order No. 12–71 (36 FR 8754), 8–76 (41 FR 25059), 9–83 (48 FR 35736), 1–90 (55 FR 9033), 6–96 (62 FR 111), 3–2000 (65 FR 50017), or 5–2002 (67 FR 65008), as applicable; and 29 CFR part 1911.

All of subpart Z issued under section 6(b) of the Occupational Safety and Health Act, —except those substances that have exposure limits listed in Tables Z–1, Z–2, and Z–3 of 29 CFR 1910.1000. The latter were issued under Sec. 6(a) (29 U.S.C. 655(a)).

Section 1910.1000, Tables Z–1, Z–2 and Z–3 also issued under 5 U.S.C. 553, Section 1910.1000 Tables Z–1, Z–2, and Z–3 not issued under 29 CFR part 1911 except for the arsenic (organic compounds), benzene, and cotton dust listings.

Section 1910.1001 also issued under Sec. 107 of the Contract Work Hours and Safety Standards Act (40 U.S.C. 3704) and 5 U.S.C. 553.

Section 1910.1002 also issued under 5 U.S.C. 553 but not under 29 U.S.C. 655 or 29 CFR part 1911.

Sections 1910.1018, 1910.1029 and 1910.1200 also issued under 29 U.S.C. 653.

Section 1910.1030 also issued under Pub. L. 106–430, 114 Stat. 1901.

§ 1910.1000 [Amended]

2. In § 1910.1000, Table Z–2, the entry for Chromic acid and chromates 1.0 mg/10 m^3 is removed and the following entry added in its place:

§ 1910.1000 Air contaminants.

* * * * *

TABLE Z–2

Substance	8-hour time weighted average	Acceptable ceiling concentration	Acceptable maximum peak above the acceptable ceiling average concentration for an 8-hr shift	
			Concentration	Maximum duration
*	*	*	*	*	*	*
Chromium (VI) compounds (as Cr); see 1910.1026.				
*	*	*	*	*	*	*

* * * * *

3. A new § 1910.1026 is added to read as follows:

§ 1910.1026 Chromium (VI).

(a) *Scope.* This standard applies to occupational exposures to chromium (VI) in all forms and compounds in general industry, except exposures that occur in the application of pesticides (*e.g.*, the treatment of wood with preservatives).

(b) *Definitions.* For the purposes of this section the following definitions apply:

Action level means a concentration of airborne chromium (VI) of 0.5 microgram per cubic meter of air (0.5 $\mu g/m^3$) calculated as an 8-hour time-weighted average (TWA).

Assistant Secretary means the Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, or designee.

Chromium (VI) [hexavalent chromium or Cr(VI)] means chromium with a valence of positive six, in any form and in any compound.

Director means the Director of the National Institute for Occupational Safety and Health (NIOSH), U.S. Department of Health and Human Services, or designee.

Emergency means any occurrence that results, or is likely to result, in an uncontrolled release of chromium (VI). If an incidental release of chromium (VI) can be controlled at the time of release by employees in the immediate release area, or by maintenance personnel, it is not an emergency.

Employee exposure means the exposure to airborne chromium (VI) that would occur if the employee were not using a respirator.

High-efficiency particulate air [HEPA] filter means a filter that is at least 99.97 percent efficient in removing mono-dispersed particles of 0.3 micrometers in diameter or larger.

Physician or other licensed health care professional [PLHCP] is an individual whose legally permitted scope of practice (i.e., license, registration, or certification) allows him or her to independently provide or be delegated the responsibility to provide some or all of the particular health care services required by paragraph (k) of this section.

Regulated area means an area, demarcated by the employer, where an employee's exposure to airborne concentrations of chromium (VI) exceeds, or can reasonably be expected to exceed, the PEL.

This section means this chromium (VI) standard.

(c) *Permissible exposure limit (PEL).* The employer shall ensure that no employee is exposed to an airborne concentration of chromium (VI) in excess of 1 microgram per cubic meter of air (1 $\mu g/m^3$), calculated as an 8-hour time-weighted average (TWA).

(d) *Exposure assessment.* (1) *General.* The employer shall determine the 8-hour TWA exposure for each employee on the basis of a sufficient number of personal breathing zone air samples to accurately characterize full shift exposure on each shift, for each job classification, in each work area. Where an employer does representative sampling instead of sampling all employees in order to meet this requirement, the employer shall sample the employee(s) expected to have the highest chromium (VI) exposures.

(2) *Initial exposure monitoring.* (i) Except as provided for in paragraphs (d)(2)(ii) and (d)(2)(iii) of this section, each employer who has a workplace or work operation covered by this section shall determine if any employee may be

exposed to chromium (VI) at or above the action level.

(ii) Where the employer has monitored for chromium (VI) in the past 12 months, and the data were obtained during work operations conducted under workplace conditions closely resembling the processes, types of material, control methods, work practices, and environmental conditions used and prevailing in the employer's current operations, and where that monitoring satisfies all other requirements of this section, including the accuracy and confidence levels of paragraph (d)(6) of this section, the employer may rely on such earlier monitoring results to satisfy the requirements for initial monitoring.

(iii) Where the employer has objective data demonstrating that a material containing chromium (VI) or a specific process, operation, or activity involving chromium (VI) cannot release dust, fumes, or mist of chromium (VI) in concentrations at or above the action level under any expected conditions of use, the employer may rely upon such data to satisfy initial monitoring requirements. The data must reflect workplace conditions closely resembling the processes, types of material, control methods, work practices, and environmental conditions in the employer's current operations.

(3) *Periodic monitoring.* (i) If initial monitoring or periodic monitoring indicates that employee exposures are below the action level, the employer may discontinue monitoring for those employees whose exposures are represented by such monitoring.

(ii) If initial monitoring or periodic monitoring reveals employee exposures to be at or above the action level, the employer shall perform periodic monitoring at least every six months.

(iii) If initial monitoring reveals employee exposures to be at or above the PEL, the employer shall perform periodic monitoring at least every three months.

(iv) If periodic monitoring indicates that employee exposures are below the action level, and the result is confirmed by the result of another monitoring taken at least seven days later, the employer may discontinue the monitoring for those employees whose exposures are represented by such monitoring.

(4) *Additional monitoring.* The employer shall perform additional monitoring when there has been any change in the production process, raw materials, equipment, personnel, work practices, or control methods that may result in new or additional exposures to chromium (VI), or when the employer has any reason to believe that new or additional exposures have occurred.

(5) *Employee notification of monitoring results.* (i) Within 15 working days after the receipt of the results of any monitoring performed under this section, the employer shall either notify each affected employee individually in writing of the results or shall post the results of the exposure monitoring in an appropriate location that is accessible to all affected employees.

(ii) Whenever monitoring results indicate that employee exposure is above the PEL, the employer shall describe in the written notification the corrective action being taken to reduce employee exposure to or below the PEL.

(6) *Accuracy of measurement.* The employer shall use a method of monitoring and analysis that can measure chromium (VI) to within an accuracy of plus or minus 25 percent (+/− 25%) and can produce accurate measurements to within a statistical confidence level of 95 percent for airborne concentrations at or above the action level.

(7) *Observation of monitoring.* (i) The employer shall provide affected employees or their designated representatives an opportunity to observe any monitoring of employee exposure to chromium (VI).

(ii) When observation of monitoring requires entry into an area where the use of protective clothing or equipment is required, the employer shall provide the observer with clothing and equipment and shall assure that the observer uses such clothing and equipment and complies with all other applicable safety and health procedures.

(e) *Regulated areas.* (1) *Establishment.* The employer shall establish a regulated area wherever an employee's exposure to airborne concentrations of chromium (VI) is, or can reasonably be expected to be, in excess of the PEL.

(2) *Demarcation.* The employer shall ensure that regulated areas are demarcated from the rest of the workplace in a manner that adequately establishes and alerts employees of the boundaries of the regulated area, and shall include the warning signs required under paragraph (l)(2) of this section.

(3) *Access.* The employer shall limit access to regulated areas to:

(i) Persons authorized by the employer and required by work duties to be present in the regulated area;

(ii) Any person entering such an area as a designated representative of employees for the purpose of exercising the right to observe monitoring procedures under paragraph (d) of this section; or

(iii) Any person authorized by the Occupational Safety and Health Act or regulations issued under it to be in a regulated area.

(f) *Methods of compliance.* (1) *Engineering and work practice controls.* (i) Except as permitted in paragraph (f)(1)(ii) of this section, the employer shall use engineering and work practice controls to reduce and maintain employee exposure to chromium (VI) to or below the PEL unless the employer can demonstrate that such controls are not feasible. Wherever feasible engineering and work practice controls are not sufficient to reduce employee exposure to or below the PEL, the employer shall use them to reduce employee exposure to the lowest levels achievable, and shall supplement them by the use of respiratory protection that complies with the requirements of paragraph (g) of this section.

(ii) Where the employer has a reasonable basis for believing that no employee in a process or task will be exposed above the PEL for 30 or more days per year (12 consecutive months), the requirement to implement engineering and work practice controls to achieve the PEL does not apply to that process or task.

(2) *Prohibition of rotation.* The employer shall not rotate employees to different jobs to achieve compliance with the PEL.

(g) *Respiratory protection.* (1) *General.* The employer shall provide respiratory protection for employees during:

(i) Periods necessary to install or implement feasible engineering and work practice controls;

(ii) Work operations, such as maintenance and repair activities, for which engineering and work practice controls are not feasible;

(iii) Work operations for which an employer has implemented all feasible engineering and work practice controls and such controls are not sufficient to reduce exposures to or below the PEL;

(iv) Work operations where employees are exposed above the PEL for fewer than 30 days per year, and the employer has elected not to implement engineering and work practice controls to achieve the PEL; or

(v) Emergencies.

(2) *Respiratory protection program.* Where respirator use is required by this section, the employer shall institute a respiratory protection program in accordance with 29 CFR 1910.134.

(h) *Protective work clothing and equipment.* (1) *Provision and use.* Where a hazard is present or is likely to be present from skin or eye contact with chromium (VI), the employer shall provide appropriate personal protective

clothing and equipment at no cost to employees, and shall ensure that employees use such clothing and equipment.

(2) *Removal and storage.* (i) The employer shall ensure that employees remove all protective clothing and equipment contaminated with chromium (VI) at the end of the work shift or at the completion of their tasks involving chromium (VI) exposure, whichever comes first.

(ii) The employer shall ensure that no employee removes chromium (VI)-contaminated protective clothing or equipment from the workplace, except for those employees whose job it is to launder, clean, maintain, or dispose of such clothing or equipment.

(iii) When contaminated protective clothing or equipment is removed for laundering, cleaning, maintenance, or disposal, the employer shall ensure that it is stored and transported in sealed, impermeable bags or other closed, impermeable containers.

(iv) Bags or containers of contaminated protective clothing or equipment that are removed from change rooms for laundering, cleaning, maintenance, or disposal shall be labeled in accordance with paragraph (l) of this section.

(3) *Cleaning and replacement.* (i) The employer shall clean, launder, repair and replace all protective clothing and equipment required by this section as needed to maintain its effectiveness.

(ii) The employer shall prohibit the removal of chromium (VI) from protective clothing and equipment by blowing, shaking, or any other means that disperses chromium (VI) into the air or onto an employee's body.

(iii) The employer shall inform any person who launders or cleans protective clothing or equipment contaminated with chromium (VI) of the potentially harmful effects of exposure to chromium (VI) and that the clothing and equipment should be laundered or cleaned in a manner that minimizes skin or eye contact with chromium (VI) and effectively prevents the release of airborne chromium (VI) in excess of the PEL.

(i) *Hygiene areas and practices.* (1) *General.* Where protective clothing and equipment is required, the employer shall provide change rooms in conformance with 29 CFR 1910.141. Where skin contact with chromium (VI) occurs, the employer shall provide washing facilities in conformance with 29 CFR 1910.141. Eating and drinking areas provided by the employer shall also be in conformance with § 1910.141.

(2) *Change rooms.* The employer shall assure that change rooms are equipped with separate storage facilities for protective clothing and equipment and for street clothes, and that these facilities prevent cross-contamination.

(3) *Washing facilities.* (i) The employer shall provide readily accessible washing facilities capable of removing chromium (VI) from the skin, and shall ensure that affected employees use these facilities when necessary.

(ii) The employer shall ensure that employees who have skin contact with chromium (VI) wash their hands and faces at the end of the work shift and prior to eating, drinking, smoking, chewing tobacco or gum, applying cosmetics, or using the toilet.

(4) *Eating and drinking areas.* (i) Whenever the employer allows employees to consume food or beverages at a worksite where chromium (VI) is present, the employer shall ensure that eating and drinking areas and surfaces are maintained as free as practicable of chromium (VI).

(ii) The employer shall ensure that employees do not enter eating and drinking areas with protective work clothing or equipment unless surface chromium (VI) has been removed from the clothing and equipment by methods that do not disperse chromium (VI) into the air or onto an employee's body.

(5) *Prohibited activities.* The employer shall ensure that employees do not eat, drink, smoke, chew tobacco or gum, or apply cosmetics in regulated areas, or in areas where skin or eye contact with chromium (VI) occurs; or carry the products associated with these activities, or store such products in these areas.

(j) *Housekeeping.* (1) *General.* The employer shall ensure that:

(i) All surfaces are maintained as free as practicable of accumulations of chromium (VI).

(ii) All spills and releases of chromium (VI) containing material are cleaned up promptly.

(2) *Cleaning methods.* (i) The employer shall ensure that surfaces contaminated with chromium (VI) are cleaned by HEPA-filter vacuuming or other methods that minimize the likelihood of exposure to chromium (VI).

(ii) Shoveling, sweeping, and brushing may be used only where HEPA-filtered vacuuming or other methods that minimize the likelihood of exposure to chromium (VI) have been tried and found not to be effective.

(iii) The employer shall not allow compressed air to be used to remove chromium (VI) from any surface unless the compressed air is used in conjunction with a ventilation system designed to capture the dust cloud created by the compressed air.

(iv) The employer shall ensure that cleaning equipment is handled in a manner that minimizes the reentry of chromium (VI) into the workplace.

(3) *Disposal.* The employer shall ensure that:

(i) Waste, scrap, debris, and any other materials contaminated with chromium (VI) and consigned for disposal are collected and disposed of in sealed, impermeable bags or other closed, impermeable containers.

(ii) Bags or containers of waste, scrap, debris, and any other materials contaminated with chromium (VI) that are consigned for disposal are labeled in accordance with paragraph (l) of this section.

(k) *Medical surveillance.* (1) *General.* (i) The employer shall make medical surveillance available at no cost to the employee, and at a reasonable time and place, for all employees:

(A) Who are or may be occupationally exposed to chromium (VI) above the PEL for 30 or more days a year;

(B) Experiencing signs or symptoms of the adverse health effects associated with chromium (VI) exposure; or

(C) Exposed in an emergency.

(ii) The employer shall assure that all medical examinations and procedures required by this section are performed by or under the supervision of a PLHCP.

(2) *Frequency.* The employer shall provide a medical examination:

(i) Within 30 days after initial assignment, unless the employee has received a chromium (VI) related medical examination, provided in accordance with this standard, within the last twelve months;

(ii) Annually;

(iii) Within 30 days after a PLHCP's written medical opinion recommends an additional examination;

(iv) Whenever an employee shows signs or symptoms of the adverse health effects associated with chromium (VI) exposure;

(v) Within 30 days after exposure during an emergency which results in an uncontrolled release of chromium (VI); or

(vi) At the termination of employment, unless the last examination that satisfied the requirements of paragraph (k) of this section was less than six months prior to the date of termination.

(3) *Contents of examination.* A medical examination consists of:

(i) A medical and work history, with emphasis on: past, present, and anticipated future exposure to chromium (VI); any history of respiratory system dysfunction; any

history of asthma, dermatitis, skin ulceration, or nasal septum perforation; and smoking status and history;

(ii) A physical examination of the skin and respiratory tract; and

(iii) Any additional tests deemed appropriate by the examining PLHCP.

(4) *Information provided to the PLHCP.* The employer shall ensure that the examining PLHCP has a copy of this standard, and shall provide the following information:

(i) A description of the affected employee's former, current, and anticipated duties as they relate to the employee's occupational exposure to chromium (VI);

(ii) The employee's former, current, and anticipated levels of occupational exposure to chromium (VI);

(iii) A description of any personal protective equipment used or to be used by the employee, including when and for how long the employee has used that equipment; and

(iv) Information from records of employment-related medical examinations previously provided to the affected employee, currently within the control of the employer.

(5) *PLHCP's written medical opinion.* (i) The employer shall obtain a written medical opinion from the PLHCP, within 30 days for each medical examination performed on each employee, which contains:

(A) The PLHCP's opinion as to whether the employee has any detected medical condition(s) that would place the employee at increased risk of material impairment to health from further exposure to chromium (VI);

(B) Any recommended limitations upon the employee's exposure to chromium (VI) or upon the use of personal protective equipment such as respirators;

(C) A statement that the PLHCP has explained to the employee the results of the medical examination, including any medical conditions related to chromium (VI) exposure that require further evaluation or treatment, and any special provisions for use of protective clothing or equipment.

(ii) The PLHCP shall not reveal to the employer specific findings or diagnoses unrelated to occupational exposure to chromium (VI).

(iii) The employer shall provide a copy of the PLHCP's written medical opinion to the examined employee within two weeks after receiving it.

(l) *Communication of chromium (VI) hazards to employees.*

(1) *General.* In addition to the requirements of the Hazard Communication Standard, 29 CFR 1910.1200, for labels, material safety data sheets, and training, employers shall comply with the following requirements.

(2) *Warning signs.* (i) The employer shall ensure that legible and readily visible warning signs are displayed at all approaches to regulated areas so that an employee may read the signs and take necessary protective steps before entering the area.

(ii) Warning signs required by paragraph (l)(2)(i) of this section shall include at least the following information:

DANGER
CHROMIUM (VI)
CANCER HAZARD
CAN DAMAGE SKIN, EYES, NASAL PASSAGES, AND LUNGS
AUTHORIZED PERSONNEL ONLY
RESPIRATORS REQUIRED IN THIS AREA

(3) *Warning labels.* The employer shall ensure that bags or containers of contaminated clothing and equipment to be removed for laundering, cleaning, or maintenance, and containers of waste, scrap, debris, and any other materials contaminated with chromium (VI) that are consigned for disposal, bear appropriate warning labels that include at least the following information:

DANGER
CONTAINS CHROMIUM (VI)
CANCER HAZARD
CAN DAMAGE SKIN, EYES, NASAL PASSAGES, AND LUNGS

(4) *Employee information and training.* (i) For all employees who are exposed to airborne chromium (VI), or who have skin or eye contact with chromium (VI), the employer shall provide training, ensure employee participation in training, and maintain a record of training provided.

(ii) The employer shall provide initial training prior to or at the time of initial assignment to a job involving potential exposure to chromium (VI). An employer who is able to demonstrate that a new employee has received training within the last 12 months that addresses the elements specified in paragraph (l)(4)(iii) of this section is not required to repeat such training provided that the employee can demonstrate knowledge of those elements.

(iii) The employer shall provide training that is understandable to the employee and shall ensure that each employee can demonstrate knowledge of at least the following:

(A) The health hazards associated with chromium (VI) exposure;

(B) The location, manner of use, and release of chromium (VI) in the workplace and the specific nature of operations that could result in exposure to chromium (VI), especially exposure above the PEL;

(C) The engineering controls and work practices associated with the employee's job assignment;

(D) The purpose, proper selection, fitting, proper use, and limitations of respirators and protective clothing;

(E) Emergency procedures;

(F) Measures employees can take to protect themselves from exposure to chromium (VI), including modification of personal hygiene and habits such as smoking;

(G) The purpose and a description of the medical surveillance program required by paragraph (k) of this section;

(H) The contents of this section; and

(I) The employee's rights of access to records under 29 CFR 1910.1020(g).

(iv) The employer shall provide additional training when:

(A) Training is necessary to ensure that each employee maintains an understanding of the safe use and handling of chromium (VI) in the workplace.

(B) Workplace changes (such as modification of equipment, tasks, or procedures) result in an increase in employee exposures to chromium (VI), and those exposures exceed or can reasonably be expected to exceed the action level or result in a hazard from skin or eye contact with chromium (VI).

(v) The employer shall make a copy of this section and its appendices readily available without cost to all affected employees.

(m) *Recordkeeping.* (1) *Exposure measurements.* (i) The employer shall maintain an accurate record of all measurements taken to monitor employee exposure to chromium (VI) as prescribed in paragraph (d) of this section.

(ii) This record shall include at least the following information:

(A) The date of measurement for each sample taken;

(B) The operation involving exposure to chromium (VI) that is being monitored;

(C) Sampling and analytical methods used and evidence of their accuracy;

(D) Number, duration, and the results of samples taken;

(E) Type of personal protective equipment, such as respirators worn; and

(F) Name, social security number, and job classification of all employees represented by the monitoring, indicating which employees were actually monitored.

(iii) The employer shall ensure that exposure records are maintained and made available in accordance with 29 CFR 1910.1020.

(2) *Historical monitoring data.* (i) Where the employer has monitored for chromium (VI) in the past 12 months, and has relied on this historical monitoring data to demonstrate that exposures on a particular job will be below the action level, the employer shall establish and maintain an accurate record of the historical monitoring data relied upon.

(ii) The record shall include information that reflects the following conditions:

(A) The data were collected using methods that meet the accuracy requirements of paragraph (d)(6) of this section;

(B) The processes and work practices that were in use when the historical monitoring data were obtained are essentially the same as those to be used during the job for which initial monitoring will not be performed;

(C) The characteristics of the chromium (VI) containing material being handled when the historical monitoring data were obtained are the same as those on the job for which initial monitoring will not be performed;

(D) Environmental conditions prevailing when the historical monitoring data were obtained are the same as those on the job for which initial monitoring will not be performed; and

(E) Other data relevant to the operations, materials, processing, or employee exposures covered by the exception.

(iii) The employer shall ensure that historical exposure records are maintained and made available in accordance with 29 CFR 1910.1020.

(3) *Objective data.* (i) Where an employer uses objective data to satisfy initial monitoring requirements, the employer shall establish and maintain an accurate record of the objective data relied upon.

(ii) This record shall include at least the following information:

(A) The chromium (VI)-containing material in question;

(B) The source of the objective data;

(C) The testing protocol and results of testing, or analysis of the material for the release of chromium (VI);

(D) A description of the operation exempted from initial monitoring and how the data support the exemption; and

(E) Other data relevant to the operations, materials, processing or employee exposures covered by the exemption.

(iii) The employer shall maintain this record for the duration of the employer's reliance upon such objective data and shall make such records available in accordance with 29 CFR 1910.1020.

(4) *Medical surveillance.* (i) The employer shall establish and maintain an accurate record for each employee covered by medical surveillance under paragraph (k) of this section.

(ii) The record shall include the following information about the employee:

(A) Name and social security number;

(B) A copy of the PLHCP's written opinions;

(C) A copy of the information provided to the PLHCP as required by paragraph (k)(4) of this section.

(iii) The employer shall ensure that medical records are maintained and made available in accordance with 29 CFR 1910.1020.

(5) *Training.* (i) At the completion of training, the employer shall prepare a record that indicates the identity of the individuals trained and the date the training was completed. This record shall be maintained for three years after the completion of training.

(ii) The employer shall provide to the Assistant Secretary or the Director, upon request, all materials relating to employee information and training.

(n) *Dates.* (1) *Effective date.* This section shall become effective [60 days after publication of the final rule in the **Federal Register**].

(2) *Start-up dates.* All obligations of this section commence 90 days after the effective date except as follows:

(i) Change rooms required by paragraph (i) of this section shall be provided no later than one year after the effective date.

(ii) Engineering controls required by paragraph (f) of this section shall be implemented no later than two years after the effective date.

PART 1915—[AMENDED]

4. The authority citation for 29 CFR part 1915 is revised to read as follows:

Authority: Sec. 41, Longshore and Harbor Workers' Compensation Act (33 U.S.C. 941); secs. 4, 6, 8, Occupational Safety and Health Act of 1970 (29 U.S.C. 653, 655, 657); Secretary of Labor's Order No. 12–71 (36 FR 8754), 8–76 (41 FR 25059), 9–83 (48 FR 35736), 1–90 (55 FR 9033), 6–96 (62 FR 111), 3–2000 (65 FR 50017) or 5–2002 (67 FR 65008), as applicable.

Sections 1915.120, 1915.152 and 1915.1026 also issued under 29 CFR part 1911.

5. In § 1915.1000, Table Z, the entry for ''Chromic acid and chromates (as CrO(3)) 0.1'' is removed and the following entry added in its place:

§ 1915.1000 Air contaminants.

* * * * *

TABLE Z—SHIPYARDS

Substance	CAS No.[d]	ppm [a] *	mg/m [3] [b] *	Skin designation
*	*	*	*	*
Chromium (VI) compounds (as Cr); see 1915.1026.			
*	*	*	*	*

* * * * * * *

[3] Use Asbestos Limit § 1915.1001.

* * * * * * *

* The PELS are 8-hour TWAs unless otherwise noted; a (C) designation denotes a ceiling limit. They are to be 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] Milligrams of substance per cubic meter of air. When entry is in this column only, the value is exact; when listed with a ppm entry, it is approximate.

* * * * * * *

[d] The CAS number is for information only. Enforcement is based on the substance name. For an entry covering more than one metal compound, measured as the metal, the CAS number for the metal is given—not CAS numbers for the individual compounds.

* * * * *

6. A new § 1915.1026 is added, to read as follows:

§ 1915.1026 Chromium (VI).

(a) *Scope.* This standard applies to occupational exposures to chromium (VI) in all forms and compounds in shipyards, marine terminals, and longshoring.

(b) *Definitions.* For the purposes of this section the following definitions apply:

Assistant Secretary means the Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, or designee.

Chromium (VI) [hexavalent chromium or Cr(VI)] means chromium with a valence of positive six, in any form and in any compound.

Director means the Director of the National Institute for Occupational Safety and Health (NIOSH), U.S. Department of Health and Human Services, or designee.

Emergency means any occurrence that results, or is likely to result, in an uncontrolled release of chromium (VI). If an incidental release of chromium (VI) can be controlled at the time of release by employees in the immediate release area, or by maintenance personnel, it is not an emergency.

Employee exposure means the exposure to airborne chromium (VI) that would occur if the employee were not using a respirator.

High-efficiency particulate air [HEPA] filter means a filter that is at least 99.97 percent efficient in removing mono-dispersed particles of 0.3 micrometers in diameter or larger.

Physician or other licensed health care professional [PLHCP] is an individual whose legally permitted scope of practice (i.e., license, registration, or certification) allows him or her to independently provide or be delegated the responsibility to provide some or all of the particular health care services required by paragraph (h) of this section.

This section means this chromium (VI) standard.

(c) *Permissible exposure limit (PEL).* The employer shall ensure that no employee is exposed to an airborne concentration of chromium (VI) in excess of 1 microgram per cubic meter of air (1 $\mu g/m^3$), calculated as an 8-hour time-weighted average (TWA).

(d) *Methods of compliance.* (1) *Engineering and work practice controls.* (i) Except as permitted in paragraph (d)(1)(ii) of this section, the employer shall use engineering and work practice controls to reduce and maintain employee exposure to chromium (VI) to or below the PEL unless the employer can demonstrate that such controls are not feasible. Wherever feasible engineering and work practice controls are not sufficient to reduce employee exposure to or below the PEL, the employer shall use them to reduce employee exposure to the lowest levels achievable, and shall supplement them by the use of respiratory protection that complies with the requirements of paragraph (e) of this section.

(ii) Where the employer has a reasonable basis for believing that no employee in a process or task will be exposed above the PEL for 30 or more days per year (12 consecutive months), the requirement to implement engineering and work practice controls to achieve the PEL does not apply to that process or task.

(2) *Prohibition of rotation.* The employer shall not rotate employees to different jobs to achieve compliance with the PEL.

(e) *Respiratory protection.* (1) *General.* The employer shall provide respiratory protection for employees during:

(i) Periods necessary to install or implement feasible engineering and work practice controls;

(ii) Work operations, such as maintenance and repair activities, for which engineering and work practice controls are not feasible;

(iii) Work operations for which an employer has implemented all feasible engineering and work practice controls and such controls are not sufficient to reduce exposures to or below the PEL;

(iv) Work operations where employees are exposed above the PEL for fewer than 30 days per year, and the employer has elected not to implement engineering and work practice controls to achieve the PEL; or

(v) Emergencies.

(2) *Respiratory protection program.* Where respirator use is required by this section, the employer shall institute a respiratory protection program in accordance with 29 CFR 1910.134.

(f) *Protective work clothing and equipment.* (1) *Provision and use.* Where a hazard is present or is likely to be present from skin or eye contact with chromium (VI), the employer shall provide appropriate personal protective clothing and equipment at no cost to employees, and shall ensure that employees use such clothing and equipment.

(2) *Removal and storage.* (i) The employer shall ensure that employees remove all protective clothing and equipment contaminated with chromium (VI) at the end of the work shift or at the completion of their tasks involving chromium (VI) exposure, whichever comes first.

(ii) The employer shall ensure that no employee removes chromium (VI)-contaminated protective clothing or equipment from the workplace, except for those employees whose job it is to launder, clean, maintain, or dispose of such clothing or equipment.

(iii) When contaminated protective clothing or equipment is removed for laundering, cleaning, maintenance, or disposal, the employer shall ensure that it is stored and transported in sealed, impermeable bags or other closed, impermeable containers.

(iv) Bags or containers of contaminated protective clothing or equipment that are removed from change rooms for laundering, cleaning, maintenance, or disposal shall be labeled in accordance with paragraph (i) of this section.

(3) *Cleaning and replacement.* (i) The employer shall clean, launder, repair and replace all protective clothing and equipment required by this section as needed to maintain its effectiveness.

(ii) The employer shall prohibit the removal of chromium (VI) from protective clothing and equipment by blowing, shaking, or any other means that disperses chromium (VI) into the air or onto an employee's body.

(iii) The employer shall inform any person who launders or cleans protective clothing or equipment contaminated with chromium (VI) of the potentially harmful effects of exposure to chromium (VI) and that the clothing and equipment should be laundered or cleaned in a manner that minimizes skin or eye contact with chromium (VI) and effectively prevents the release of airborne chromium (VI) in excess of the PEL.

(g) *Hygiene areas and practices.* (1) *General.* Where protective clothing and equipment is required, the employer shall provide change rooms in conformance with 29 CFR 1910.141. Where skin contact with chromium (VI) occurs, the employer shall provide washing facilities in conformance with 29 CFR 1915.97. Eating and drinking areas provided by the employer shall also be in conformance with § 1915.97.

(2) *Change rooms.* The employer shall assure that change rooms are equipped with separate storage facilities for protective clothing and equipment and for street clothes, and that these facilities prevent cross-contamination.

(3) *Washing facilities.* (i) The employer shall provide readily accessible washing facilities capable of removing chromium (VI) from the skin, and shall ensure that affected employees use these facilities when necessary.

(ii) The employer shall ensure that employees who have skin contact with chromium (VI) wash their hands and faces at the end of the work shift and prior to eating, drinking, smoking, chewing tobacco or gum, applying cosmetics, or using the toilet.

(4) *Eating and drinking areas.* (i) Whenever the employer allows employees to consume food or beverages at a worksite where chromium (VI) is present, the employer shall ensure that eating and drinking areas and surfaces are maintained as free as practicable of chromium (VI).

(ii) The employer shall ensure that employees do not enter eating and drinking areas with protective work clothing or equipment unless surface chromium (VI) has been removed from the clothing and equipment by methods that do not disperse chromium (VI) into the air or onto an employee's body.

(5) *Prohibited activities.* The employer shall ensure that employees do not eat, drink, smoke, chew tobacco or gum, or apply cosmetics in areas where skin or eye contact with chromium (VI) occurs; or carry the products associated with these activities, or store such products in these areas.

(h) *Medical surveillance.* (1) *General.* (i) The employer shall make medical surveillance available at no cost to the employee, and at a reasonable time and place, for all employees:

(A) Experiencing signs or symptoms of the adverse health effects associated with chromium (VI) exposure; or

(B) Exposed in an emergency.

(ii) The employer shall assure that all medical examinations and procedures required by this section are performed by or under the supervision of a PLHCP.

(2) *Frequency.* The employer shall provide a medical examination:

(i) Whenever an employee shows signs or symptoms of the adverse health effects associated with chromium (VI) exposure;

(ii) Within 30 days after exposure during an emergency which results in an uncontrolled release of chromium (VI); or

(iii) Within 30 days after a PLHCP's written medical opinion recommends an additional examination.

(3) *Contents of examination.* A medical examination consists of:

(i) A medical and work history, with emphasis on: Past, present, and anticipated future exposure to chromium (VI); any history of respiratory system dysfunction; any history of asthma, dermatitis, skin ulceration, or nasal septum perforation; and smoking status and history;

(ii) A physical examination of the skin and respiratory tract; and

(iii) Any additional tests deemed appropriate by the examining PLHCP.

(4) *Information provided to the PLHCP.* The employer shall ensure that the examining PLHCP has a copy of this standard, and shall provide the following information:

(i) A description of the affected employee's former, current, and anticipated duties as they relate to the employee's occupational exposure to chromium (VI);

(ii) The employee's former, current, and anticipated levels of occupational exposure to chromium (VI);

(iii) A description of any personal protective equipment used or to be used by the employee, including when and for how long the employee has used that equipment; and

(iv) Information from records of employment-related medical examinations previously provided to the affected employee, currently within the control of the employer.

(5) *PLHCP's written medical opinion.* (i) The employer shall obtain a written medical opinion from the PLHCP, within 30 days for each medical examination performed on each employee, which contains:

(A) The PLHCP's opinion as to whether the employee has any detected medical condition(s) that would place the employee at increased risk of material impairment to health from further exposure to chromium (VI);

(B) Any recommended limitations upon the employee's exposure to chromium (VI) or upon the use of personal protective equipment such as respirators;

(C) A statement that the PLHCP has explained to the employee the results of the medical examination, including any medical conditions related to chromium (VI) exposure that require further evaluation or treatment, and any special provisions for use of protective clothing or equipment.

(ii) The PLHCP shall not reveal to the employer specific findings or diagnoses unrelated to occupational exposure to chromium (VI).

(iii) The employer shall provide a copy of the PLHCP's written medical opinion to the examined employee within two weeks after receiving it.

(i) *Communication of chromium (VI) hazards to employees.*

(1) *General.* In addition to the requirements of the Hazard Communication Standard, 29 CFR 1910.1200, for labels, material safety data sheets, and training, employers shall comply with the following requirements.

(2) *Warning labels.* The employer shall ensure that bags or containers of contaminated clothing and equipment to be removed for laundering, cleaning, or maintenance, bear appropriate warning labels that include at least the following information:

DANGER
CONTAINS CHROMIUM (VI)
CANCER HAZARD
CAN DAMAGE SKIN, EYES, NASAL PASSAGES, AND LUNGS

(3) *Employee information and training.* (i) The employer shall provide training for all employees who are potentially exposed to chromium (VI), ensure employee participation in training, and maintain a record of training provided.

(ii) The employer shall provide initial training prior to or at the time of initial assignment to a job involving potential exposure to chromium (VI). An employer who is able to demonstrate that a new employee has received training within the last 12 months that addresses the elements specified in paragraph (l)(4)(iii) of this section is not required to repeat such training provided that the employee can demonstrate knowledge of those elements.

(iii) The employer shall provide training that is understandable to the employee and shall ensure that each employee can demonstrate knowledge of at least the following:

(A) The health hazards associated with chromium (VI) exposure;

(B) The location, manner of use, and release of chromium (VI) in the workplace and the specific nature of operations that could result in exposure to chromium (VI), especially exposure above the PEL;

(C) The engineering controls and work practices associated with the employee's job assignment;

(D) The purpose, proper selection, fitting, proper use, and limitations of respirators and protective clothing;

(E) Emergency procedures;

(F) Measures employees can take to protect themselves from exposure to chromium (VI), including modification of personal hygiene and habits such as smoking;

(G) The purpose and a description of the medical surveillance program required by paragraph (h) of this section;

(H) The contents of this section; and

(I) The employee's rights of access to records under 29 CFR 1910.1020(g).

(iv) The employer shall provide additional training when:

(A) Training is necessary to ensure that each employee maintains an understanding of the safe use and handling of chromium (VI) in the workplace.

(B) Workplace changes (such as modification of equipment, tasks, or procedures) result in an increase in employee exposures to chromium (VI), and those exposures exceed or can reasonably be expected to exceed the PEL or result in a hazard from skin or eye contact with chromium (VI).

(v) The employer shall make a copy of this section and its appendices readily available without cost to all affected employees.

(j) *Recordkeeping.* (1) *Medical surveillance.* (i) The employer shall establish and maintain an accurate record for each employee covered by medical surveillance under paragraph (h) of this section.

(ii) The record shall include the following information about the employee:

(A) Name and social security number;

(B) A copy of the PLHCP's written opinions;

(C) A copy of the information provided to the PLHCP as required by paragraph (h)(4) of this section.

(iii) The employer shall ensure that medical records are maintained and made available in accordance with §1910.1020.

(2) *Training.* (i) At the completion of training, the employer shall prepare a record that indicates the identity of the individuals trained and the date the training was completed. This record shall be maintained for three years after the completion of training.

(ii) The employer shall provide to the Assistant Secretary or the Director, upon request, all materials relating to employee information and training.

(k) *Dates.* (1) *Effective date.* This section shall become effective [60 days after publication of the final rule in the **Federal Register**].

(2) *Start-up dates.* All obligations of this section commence 90 days after the effective date except as follows:

(i) Change rooms required by paragraph (g) of this section shall be provided no later than one year after the effective date.

(ii) Engineering controls required by paragraph (d) of this section shall be implemented no later than two years after the effective date.

PART 1917—[AMENDED]

7. The authority citation for 29 CFR Part 1917 is revised to read as follows:

Authority: Sec. 41, Longshore and Harbor Workers' Compensation Act (33 U.S.C. 941); secs. 4, 6, 8, Occupational Safety and Health Act of 1970 (29 U.S.C. 653, 655, 657); Secretary of Labor's Order Nos. 12–71 (36 FR 8754), 8–76 (41 FR 25059), 9–83 (48 FR 35736), 6–96 (62 FR 111), or 5–2002 (67 FR 65008), as applicable; 29 CFR part 1911.

Section 1917.28 also issued under 5 U.S.C. 553.

8. New paragraphs (a)(2)(xiii)(E) and (b) are added to § 1917.1, to read as follows:

§ 1917.1 Scope and applicability.

* * * * *

(a) * * *

(2) * * *

(xiii) * * *

(E) Hexavalent chromium § 1910.1026 (See § 1915.1026)

* * * * *

(b) Section 1915.1026 applies to any occupational exposures to hexavalent chromium in workplaces covered by this part.

PART 1918—[AMENDED]

9. The authority citation for 29 CFR Part 1918 is revised to read as follows:

Authority: Secs. 4, 6, 8, Occupational Safety and Health Act of 1970, 29 U.S.C. 653, 655, 657; Walsh-Healey Act, 41 U.S.C. 35 *et seq.*; Service Contract Act of 1965, 41 U.S.C. 351 et seq,; Sec. 107, Contract Work Hours and Safety Standards Act (Construction Safety Act), 40 U.S.C. 333; Sec. 41, Longshore and Harbor Workers' Compensation Act, 33 U.S.C. 941; National Foundation of Arts and Humanities Act, 20 U.S.C. 951 *et seq.*; Secretary of Labor's Order Nos. 6–96 (62 FR 111) or 5–2002 (67 FR 65008), as applicable; and 29 CFR part 1911.

10. New paragraphs (b)(9)(v) and (c) are added to § 1918.1 to read as follows:

§ 1918.1 Scope and application.

* * * * *

(b) * * *

(9) * * *

(v) Hexavalent chromium § 1910.1026 (See § 1915.1026)

* * * * *

(c) Section 1915.1026 applies to any occupational exposures to hexavalent chromium in workplaces covered by this part.

PART 1926—[AMENDED]

Subpart D—[Amended]

11. The authority citation for subpart D of 29 CFR Part 1926 is revised to read as follows:

Authority: Sec. 107, Contract Work Hours and Safety Standards Act (40 U.S.C. 333); secs. 4, 6, 8, Occupational Safety and Health Act of 1970 (29 U.S.C. 653, 655, 657); Secretary of Labor's Order Nos. 12–71 (36 FR 8754), 8–76 (41 FR 25059), 9–83 (48 FR 35736), 6–96 (62 FR 111), or 5–2002 (67 FR 65008), as applicable; and 29 CFR part 1911.

§ 1926.55 [Amended]

12. In Appendix A to § 1926.55, the entry for ''Chromic acid and chromates (as CrO_3) 0.1'' is removed and the following entry added in its place:

§ 1926.55 Gases, vapors, fumes, dusts, and mists.

* * * * *

THRESHOLD LIMIT VALUES OF AIRBORNE CONTAMINANTS FOR CONSTRUCTION

Substance	CAS No.[d]	ppm [a]	mg/m [3] [b]	Skin Designation
*	*	*	*	*
Chromium (VI) compounds (as Cr); see 1926.1126.				
*	*	*	*	*

[3] Use Asbestos Limit§ 1915.1001

* * * * * * *

[a] Parts of vapor or gas per million parts of contaminated air by volume at 25 ° C and 760 torr.

[3b] Milligrams of substance per cubic meter of air. When entry is in this column only, the value is exact; when listed with a ppm entry, it is approxiate

* * * * * * *

[d] The CAS number is for information only. Enforcement is based on the substance name. For an entry covering more than one metal compound, measured as the metal, the CAS number for the metal is given—not CAS numbers for the individual compounds.

* * * * *

Subpart Z—[Amended]

13. The authority citation for subpart Z of 29 CFR Part 1926 is revised to read as follows:

Authority: Sec. 107, Contract Work Hours and Safety Standards Act (40 U.S.C. 333); secs. 4, 6, 8, Occupational Safety and Health Act of 1970 (29 U.S.C. 653, 655, 657); Secretary of Labor's Order Nos. 12–71 (36 FR 8754), 8–76 (41 FR 25059), 9–83 (48 FR 35736), 1–90 (55 FR 9033), 6–96 (62 FR 111), or 5–2002 (67 FR 65008), as applicable; and 29 CFR part 1911.

Section 1926.1102 not issued under 29 U.S.C. 655 or 29 CFR part 1911; also issued under 5 U.S.C. 553.

14. A new § 1926.1126 is added to subpart Z of 29 CFR Part 1926 to read as follows:

§ 1926.1126 Chromium (VI).

(a) *Scope.* This standard applies to occupational exposures to chromium (VI) in all forms and compounds in construction, except for exposures to portland cement.

(b) *Definitions.* For the purposes of this section the following definitions apply:

Assistant Secretary means the Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, or designee.

Chromium (VI) [hexavalent chromium or Cr(VI)] means chromium with a valence of positive six, in any form and in any compound.

Director means the Director of the National Institute for Occupational Safety and Health (NIOSH), U.S. Department of Health and Human Services, or designee.

Emergency means any occurrence that results, or is likely to result, in an uncontrolled release of chromium (VI). If an incidental release of chromium (VI) can be controlled at the time of release by employees in the immediate release area, or by maintenance personnel, it is not an emergency.

Employee exposure means the exposure to airborne chromium (VI) that would occur if the employee were not using a respirator.

High-efficiency particulate air [HEPA] filter means a filter that is at least 99.97 percent efficient in removing mono-dispersed particles of 0.3 micrometers in diameter or larger.

Physician or other licensed health care professional [PLHCP] is an individual whose legally permitted scope of practice (i.e., license, registration, or certification) allows him or her to independently provide or be delegated the responsibility to provide some or all of the particular health care services required by paragraph (h) of this section.

This section means this chromium (VI) standard.

(c) *Permissible exposure limit (PEL).* The employer shall ensure that no employee is exposed to an airborne concentration of chromium (VI) in excess of 1 microgram per cubic meter of air (1 $\mu g/m^3$), calculated as an 8-hour time-weighted average (TWA).

(d) *Methods of compliance.* (1) *Engineering and work practice controls.* (i) Except as permitted in paragraph (d)(1)(ii) of this section, the employer shall use engineering and work practice controls to reduce and maintain employee exposure to chromium (VI) to or below the PEL unless the employer can demonstrate that such controls are not feasible. Wherever feasible engineering and work practice controls are not sufficient to reduce employee exposure to or below the PEL, the employer shall use them to reduce employee exposure to the lowest levels achievable, and shall supplement them by the use of respiratory protection that complies with the requirements of paragraph (e) of this section.

(ii) Where the employer has a reasonable basis for believing that no employee in a process or task will be exposed above the PEL for 30 or more days per year (12 consecutive months), the requirement to implement engineering and work practice controls to achieve the PEL does not apply to that process or task.

(2) *Prohibition of Rotation.* The employer shall not rotate employees to different jobs to achieve compliance with the PEL.

(e) *Respiratory Protection.* (1) *General.* The employer shall provide respiratory protection for employees during:

(i) Periods necessary to install or implement feasible engineering and work practice controls;

(ii) Work operations, such as maintenance and repair activities, for which engineering and work practice controls are not feasible;

(iii) Work operations for which an employer has implemented all feasible engineering and work practice controls and such controls are not sufficient to reduce exposures to or below the PEL;

(iv) Work operations where employees are exposed above the PEL for fewer than 30 days per year, and the employer has elected not to implement engineering and work practice controls to achieve the PEL; or

(v) Emergencies.

(2) *Respiratory protection program.* Where respirator use is required by this section, the employer shall institute a respiratory protection program in accordance with 29 CFR 1910.134.

(f) *Protective work clothing and equipment.* (1) *Provision and use.* Where a hazard is present or is likely to be present from skin or eye contact with chromium (VI), the employer shall provide appropriate personal protective clothing and equipment at no cost to employees, and shall ensure that employees use such clothing and equipment.

(2) *Removal and storage.* (i) The employer shall ensure that employees remove all protective clothing and equipment contaminated with chromium (VI) at the end of the work shift or at the completion of their tasks involving chromium (VI) exposure, whichever comes first.

(ii) The employer shall ensure that no employee removes chromium (VI)-contaminated protective clothing or equipment from the workplace, except for those employees whose job it is to launder, clean, maintain, or dispose of such clothing or equipment.

(iii) When contaminated protective clothing or equipment is removed for laundering, cleaning, maintenance, or disposal, the employer shall ensure that it is stored and transported in sealed, impermeable bags or other closed, impermeable containers.

(iv) Bags or containers of contaminated protective clothing or equipment that are removed from change rooms for laundering, cleaning, maintenance, or disposal shall be labeled in accordance with paragraph (i) of this section.

(3) *Cleaning and replacement.* (i) The employer shall clean, launder, repair and replace all protective clothing and equipment required by this section as needed to maintain its effectiveness.

(ii) The employer shall prohibit the removal of chromium (VI) from protective clothing and equipment by blowing, shaking, or any other means that disperses chromium (VI) into the air or onto an employee's body.

(iii) The employer shall inform any person who launders or cleans protective clothing or equipment contaminated with chromium (VI) of the potentially harmful effects of exposure to chromium (VI) and that the clothing and equipment should be laundered or cleaned in a manner that minimizes skin or eye contact with chromium (VI) and effectively prevents the release of airborne chromium (VI) in excess of the PEL.

(g) *Hygiene areas and practices.* (1) *General.* Where protective clothing and equipment is required, the employer shall provide change rooms in conformance with 29 CFR 1926.51.

Where skin contact with chromium (VI) occurs, the employer shall provide washing facilities in conformance with 29 CFR 1926.51. Eating and drinking areas provided by the employer shall also be in conformance with § 1926.51.

(2) *Change rooms.* The employer shall assure that change rooms are equipped with separate storage facilities for protective clothing and equipment and for street clothes, and that these facilities prevent cross-contamination.

(3) *Washing facilities.* (i) The employer shall provide readily accessible washing facilities capable of removing chromium (VI) from the skin, and shall ensure that affected employees use these facilities when necessary.

(ii) The employer shall ensure that employees who have skin contact with chromium (VI) wash their hands and faces at the end of the work shift and prior to eating, drinking, smoking, chewing tobacco or gum, applying cosmetics, or using the toilet.

(4) *Eating and drinking areas.* (i) Whenever the employer allows employees to consume food or beverages at a worksite where chromium (VI) is present, the employer shall ensure that eating and drinking areas and surfaces are maintained as free as practicable of chromium (VI).

(ii) The employer shall ensure that employees do not enter eating and drinking areas with protective work clothing or equipment unless surface chromium (VI) has been removed from the clothing and equipment by methods that do not disperse chromium (VI) into the air or onto an employee's body.

(5) *Prohibited activities.* The employer shall ensure that employees do not eat, drink, smoke, chew tobacco or gum, or apply cosmetics in areas where skin or eye contact with chromium (VI) occurs; or carry the products associated with these activities, or store such products in these areas.

(h) *Medical Surveillance.* (1) *General.* (i) The employer shall make medical surveillance available at no cost to the employee, and at a reasonable time and place, for all employees:

(A) Experiencing signs or symptoms of the adverse health effects associated with chromium (VI) exposure; or

(B) Exposed in an emergency.

(ii) The employer shall assure that all medical examinations and procedures required by this section are performed by or under the supervision of a PLHCP.

(2) *Frequency.* The employer shall provide a medical examination:

(i) Whenever an employee shows signs or symptoms of the adverse health effects associated with chromium (VI) exposure;

(ii) Within 30 days after exposure during an emergency which results in an uncontrolled release of chromium (VI); or

(iii) Within 30 days after a PLHCP's written medical opinion recommends an additional examination.

(3) *Contents of examination.* A medical examination consists of:

(i) A medical and work history, with emphasis on: Past, present, and anticipated future exposure to chromium (VI); any history of respiratory system dysfunction; any history of asthma, dermatitis, skin ulceration, or nasal septum perforation; and smoking status and history;

(ii) A physical examination of the skin and respiratory tract; and

(iii) Any additional tests deemed appropriate by the examining PLHCP.

(4) *Information provided to the PLHCP.* The employer shall ensure that the examining PLHCP has a copy of this standard, and shall provide the following information:

(i) A description of the affected employee's former, current, and anticipated duties as they relate to the employee's occupational exposure to chromium (VI);

(ii) The employee's former, current, and anticipated levels of occupational exposure to chromium (VI);

(iii) A description of any personal protective equipment used or to be used by the employee, including when and for how long the employee has used that equipment; and

(iv) Information from records of employment-related medical examinations previously provided to the affected employee, currently within the control of the employer.

(5) *PLHCP's Written Medical Opinion.* (i) The employer shall obtain a written medical opinion from the PLHCP, within 30 days for each medical examination performed on each employee, which contains:

(A) The PLHCP's opinion as to whether the employee has any detected medical condition(s) that would place the employee at increased risk of material impairment to health from further exposure to chromium (VI);

(B) Any recommended limitations upon the employee's exposure to chromium (VI) or upon the use of personal protective equipment such as respirators;

(C) A statement that the PLHCP has explained to the employee the results of the medical examination, including any medical conditions related to chromium (VI) exposure that require further evaluation or treatment, and any special provisions for use of protective clothing or equipment.

(ii) The PLHCP shall not reveal to the employer specific findings or diagnoses unrelated to occupational exposure to chromium (VI).

(iii) The employer shall provide a copy of the PLHCP's written medical opinion to the examined employee within two weeks after receiving it.

(i) *Communication of chromium (VI) hazards to employees.* (1) *General.* In addition to the requirements of the Hazard Communication Standard, 29 CFR 1910.1200, for labels, material safety data sheets, and training, employers shall comply with the following requirements.

(2) *Warning labels.* The employer shall ensure that bags or containers of contaminated clothing and equipment to be removed for laundering, cleaning, or maintenance, bear appropriate warning labels that include at least the following information:

DANGER
CONTAINS CHROMIUM (VI)
CANCER HAZARD
CAN DAMAGE SKIN, EYES, NASAL PASSAGES, AND LUNGS

(3) *Employee information and training.* (i) The employer shall provide training for all employees who are potentially exposed to chromium (VI), ensure employee participation in training, and maintain a record of training provided.

(ii) The employer shall provide initial training prior to or at the time of initial assignment to a job involving potential exposure to chromium (VI). An employer who is able to demonstrate that a new employee has received training within the last 12 months that addresses the elements specified in paragraph (l)(4)(iii) of this section is not required to repeat such training provided that the employee can demonstrate knowledge of those elements.

(iii) The employer shall provide training that is understandable to the employee and shall ensure that each employee can demonstrate knowledge of at least the following:

(A) The health hazards associated with chromium (VI) exposure;

(B) The location, manner of use, and release of chromium (VI) in the workplace and the specific nature of operations that could result in exposure to chromium (VI), especially exposure above the PEL;

(C) The engineering controls and work practices associated with the employee's job assignment;

(D) The purpose, proper selection, fitting, proper use, and limitations of respirators and protective clothing;

(E) Emergency procedures;

(F) Measures employees can take to protect themselves from exposure to chromium (VI), including modification of personal hygiene and habits such as smoking;

(G) The purpose and a description of the medical surveillance program required by paragraph (h) of this section;

(H) The contents of this section; and

(I) The employee's rights of access to records under 29 CFR 1910.1020(g).

(iv) The employer shall provide additional training when:

(A) Training is necessary to ensure that each employee maintains an understanding of the safe use and handling of chromium (VI) in the workplace.

(B) Workplace changes (such as modification of equipment, tasks, or procedures) result in an increase in employee exposures to chromium (VI), and those exposures exceed or can reasonably be expected to exceed the PEL or result in a hazard from skin or eye contact with chromium (VI).

(v) The employer shall make a copy of this section and its appendices readily available without cost to all affected employees.

(j) *Recordkeeping.* (1) *Medical surveillance.* (i) The employer shall establish and maintain an accurate record for each employee covered by medical surveillance under paragraph (h) of this section.

(ii) The record shall include the following information about the employee:

(A) Name and social security number;

(B) A copy of the PLHCP's written opinions;

(C) A copy of the information provided to the PLHCP as required by paragraph (h)(4) of this section.

(iii) The employer shall ensure that medical records are maintained and made available in accordance with §1910.1020.

(2) *Training.* (i) At the completion of training, the employer shall prepare a record that indicates the identity of the individuals trained and the date the training was completed. This record shall be maintained for three years after the completion of training.

(ii) The employer shall provide to the Assistant Secretary or the Director, upon request, all materials relating to employee information and training.

(k) *Dates.* (1) *Effective date.* This section shall become effective [60 days after publication of the final rule in the **Federal Register**].

(2) *Start-up dates.* All obligations of this section commence 90 days after the effective date except as follows:

(i) Change rooms required by paragraph (g) of this section shall be provided no later than one year after the effective date.

(ii) Engineering controls required by paragraph (d) of this section shall be implemented no later than two years after the effective date.

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