

**Responses to Major Comments on
Technical Support Document**

**Public Health Goal
For
Chromium
In Drinking Water**

Prepared by

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INTRODUCTION

The following are responses to major comments received by the Office of Environmental Health Hazard Assessment (OEHHA) on the proposed public health goal (PHG) technical support document for chromium as discussed at the PHG workshop held on October 6, 1998, or as revised following the workshop. Some commenters provided comments on both the first and second drafts. For the sake of brevity, we have selected the more important or representative comments for responses. Comments appear in quotation marks where they are directly quoted from the submission; paraphrased comments are in italics.

These comments and responses are provided in the spirit of the open dialogue among scientists that is part of the process under Health and Safety Code Section 57003. For further information about the PHG process or to obtain copies of PHG documents, visit the OEHHA web site at www.oehha.org. OEHHA may also be contacted at:

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RESPONSES TO MAJOR COMMENTS RECEIVED

Comments from Castaic Lake Water Agency

Comment 1: “There are very few analytical procedures....”

Response 1: PHGs are based on health effects only, and do not take into account technical feasibility matters, such as analytical methods and detection limits.

Comment 2: “Chromium VI is reduced by gastric juice....”

Response 2: While it is true that Cr VI may be reduced by gastric juice, we do not have quantitative data to show whether all Cr VI would be reduced to Cr III in the human stomach following ingestion of Cr VI in drinking water. Individuals in the population vary in the acidity of their stomach contents, and therefore they would be expected to vary in the degree to which Cr VI would be reduced to Cr III. Because of this variability, we cannot be sure that reduction would be complete before chromium ions are absorbed into the bloodstream.

Comment 3: “The authors assume that all chromium VI compounds are equally carcinogenic.”

Response 3: The PHG is based on the carcinogenicity of the chromium VI ion, and does not deal with different salts. If chromium VI is detected in drinking water, it is not possible to know which salt it came from, and this is irrelevant once it is dissolved in water.

Comment 4: “Human occupational epidemiological literature is supportive of the hypothesis that only water insoluble chromates are carcinogenic.”

Response 4: The distinction between soluble and insoluble salts does not apply to the ingestion route. Whereas inhalation exposure is to solid particles of chromium salts that deposit directly onto lung tissue and then may or may not dissolve, and may or may not be taken up by mechanisms such as phagocytosis or pinocytosis, exposure via ingestion involves exposure of gastrointestinal tissues to ions in solution. The question of solubility of the salts is not relevant to the ingestion route because the mode of exposure to the target tissues is completely different from inhalation exposure, and because ions in solution are by definition soluble. The cancer potency was calculated based on ingestion exposure to a soluble form of chromium. Since tumors were produced in the experiment, the soluble chromium must have been carcinogenic.

Comment 5: Chromates are only carcinogenic to the lungs.

Response 5: The PHG is based on stomach tumors produced in animals by chromate exposure.

Comment 6: “The most powerful reducing conditions occur in the human stomach. At a pH of 1 and large amounts of organic material such as food and mucous, chromium VI is rapidly reduced in the stomach.” The commenter goes on to cite reports in the literature that observe that chromium VI is reduced to chromium III in the human stomach.

Response 6: It is probably true that much ingested chromium VI is reduced to chromium III in the human stomach, however we do not have data to tell us how rapidly and completely this happens. We have based our risk assessment on mouse data that shows that ingested chromium VI caused an increase in benign and malignant stomach tumors in mice. The human stomach is probably more acidic and a better reducing environment than the stomachs of the mice in the Borneff experiment, but we do not have data that would allow us to make a direct comparison. In the absence of such data, we assume that chromium VI could cause tumors in humans.

Comment 7: “Throughout the PHG document the authors assume that all chromates are equally carcinogenic. However, the animal toxicology literature offers strong evidence that only certain species of WATER INSOLUBLE chromates are carcinogenic in the most cancer sensitive organ, the lungs.”

Response 7: The comment refers to inhalation experiments in animals (mostly rats) in which microscopic particles of these salts are being deposited directly onto lung tissue. Rats appear to be uniquely sensitive to particulates as such, therefore not much weight can be placed on the results of rat inhalation experiments in terms of distinguishing between different forms of chromium.

In animal inhalation experiments the lung tissue is exposed to a salt as such (both ions are present). In the case of chromium VI ions in drinking water the exposure situation is quite different. In this case the target tissues are exposed to chromium VI ions, and whatever other ions may be present in the drinking water. They are not exposed to chromium salts as such. In the drinking water situation we cannot assume that only certain salts would be carcinogenic.

Comment 8: “Similarly, the human occupational epidemiological literature is also supportive of the hypothesis that only water insoluble chromates are carcinogenic.”

Response 8: As discussed in the previous response, the mode of exposure of the tissues to chromium ions is very different in the case of ingestion of chromium ions in drinking water compared to inhalation of chromium salts in particulates. For this reason it would not be appropriate to apply any generalizations derived from epidemiological studies of workers exposed to chromium salts by inhalation to the question of potential carcinogenicity of chromium ions by the ingestion route.

Comment 9: “70% of all cancers occur in the skin and so it is by far the most sensitive organ for cancer. Dermatitis has historically been the first observed health effect associated with chromium (VI) exposure... Despite this, chromates have not been indicated as skin carcinogens.”

Response 9: The commenters premise appears to be that skin is the most sensitive site, so if cancers are not seen on the skin, they are not likely to be induced in other (presumably less sensitive) tissues. Skin cancers are very common because the skin is exposed to UV light from the sun and some other carcinogens. This does not mean that skin is the most sensitive tissue for all carcinogens. In fact different cancers may arise from different kinds of genetic changes, so one cannot predict in advance which tissue will be the most sensitive for a particular carcinogen. The absence of skin cancers in workers exposed to chromium VI does not prove that chromium VI cannot be carcinogenic to other tissues.

Comment 10: “In the concluding section of the PHG document the authors make the following arguments.” This is followed by six arguments, and a rebuttal for each one.

Response 10: No one of these arguments is conclusive in itself, but the totality of them argue in favor of considering chromium a carcinogen by the oral route. Specific arguments are responded to in the table below:

| Argument from PHG document | Comment from Kimbrough | Response from OEHHA |
|--|---|---|
| Chromium is a known human carcinogen by the inhalation route. | Only water insoluble chromates (not chromium) are known human carcinogens by the inhalation route. | The carcinogenicity of chromium by the inhalation route appears to be limited to certain salts, but this limitation may not apply to chromium ion in drinking water. |
| Non-respiratory cancers have been found in workers exposed to chromium VI by inhalation. | Most epidemiological studies do not show an elevated risk of non-respiratory cancer among chromate exposed workers. | The fact that a few studies show elevated risk for non-respiratory cancers casts doubt on the hypothesis that chromium carcinogenicity is limited only to lung tissue. |
| Inhaled chromium VI causes respiratory tumors in rats. | The most definitive rat studies support the conclusion that only water insoluble chromates are carcinogenic to the rat and human lung. | Chromium has been shown to be carcinogenic in more than one species. |
| Chromium VI causes contact site tumors in laboratory animals. | There is no textual support for this statement in the PHG document. | This was a deficiency in the PHG document that has been corrected. An example of a study that showed injection site tumors is Hueper (1955)*. |
| Ingested chromium VI has been associated with stomach tumors in mice. | As the authors note, only one mouse study out of a much larger number of animal studies showed a slight non-significant increase in malignant tumors. | The total tumors (malignant and non-malignant) were statistically significant (p=0.003)**. The non-malignant tumors appeared to be related to the malignant tumors. One cannot ignore a positive study. |
| Chromium VI has been positive in a number of assays for genotoxicity. | [no comment] | [no response required] |

*Hueper, WC (1955). Experimental studies in metal carcinogenesis. VII. Tissue reactions to parenterally introduced powdered metallic chromium and chromite ore. J. National Cancer Institute 16, 447-462.

**Siegel, DM to Standards/Criteria Workgroup Members (1990). Carcinogenicity of Chromium via ingestion.

Comment 12: “Even if water soluble chromates were carcinogenic to the stomach, the reductive capacity of the stomach acids and foods and beverages would prevent any risk of stomach cancer.”

Response 12: The question of the reduction of chromium VI to chromium III in the stomach is discussed above and in responses to other commenters. OEHHA’s concern about the potential carcinogenicity of chromium VI is not necessarily limited to the stomach as target organ.

Comment from Metropolitan Water District of Southern California

Comment 1: OEHHA is proposing to set a PHG for hexavalent chromium when there is: a. no existing or proposed drinking water standard in California or nationally, b. Water suppliers are not required to analyze and speciate for various valences of chromium, and c. There are no laboratory methods to detect hexavalent chromium at such extremely low levels.

Response 1: OEHHA has revised the PHG document and developed a PHG for total chromium rather than separate PHGs for chromium III and chromium VI. The health protective levels for the separate valence states are still in the document as a matter of information, and as an intermediate step in calculating the PHG for total chromium, but there is not a PHG for hexavalent chromium. PHGs are based solely on public health considerations, without regard to detection limits or methods of analysis. Newer methodology may be developed in the future that will allow detection of smaller amounts than can be detected with current methods. Improvements in analytical technology will not require changes in the PHGs.

Comments from ATSDR

Comment 1: “ATSDR has recently produced an updated Toxicological Profile for Chromium, ...This 1998 Draft should be consulted for updated information and cited as appropriate in place of the 1993 version.”

Response 1: These changes have been made in the text.

Comment 2: “The ATSDR Toxicological Profile is inappropriately cited as an authoritative source for the cancer classification of chromium.”

Response 2: This has been changed in the text as suggested.

Comment 3: “The Draft PHG states that ATSDR has concluded that chromium VI is unlikely to be carcinogenic to humans by the oral route, and attributes to ATSDR (1993) a statement that there is some controversy as to whether chromium VI should be considered a carcinogen by the oral route.”

Response 3: The statements in the text have been changed as requested.

Comment 4: Under “Specific Comments” the commenter has made numerous suggestions for detailed changes in the text.

Response 4: All of the suggested changes have been considered, and changes have been made in the text as requested.

Comments from a resident of Santa Monica

Comment 1: “The presence of a redox system in water can certainly reduce Cr VI to Cr III, and a sufficiently acidic pH can solubilize insoluble forms of chromium present in water.”

Response 1: The first statement is widely supported in the literature. We would be interested in seeing some support for the second statement. In developing a PHG for chromium, OEHHA needs to know how much Cr VI and Cr III are likely to be present in drinking water. The chemistry of chromium in water is complex, and influenced by many factors. It is unlikely that we can estimate the amount of chromium VI or III on theoretical grounds. Therefore, we need monitoring data to tell us what percentage of the chromium found in water is in each of the valence states. Unfortunately, little such data exists.

Comment 2: “Clearly, unreduced Cr VI can and does leave the stomach in sufficient quantities to be distributed and found throughout the body. The presence of Cr VI in the kidneys and spleen indicates that not all of the Cr VI reaching the liver is converted to Cr III.

Response 2: The report cited (Maruyama, 1982) measured chromium in the target organs following administration of Cr VI or Cr III, and compared the distribution of chromium to different target organs. However, we cannot tell from this report whether the chromium administered as Cr VI reached the target organs still in the form of Cr VI, or whether it was reduced to Cr III somewhere along the way. The commenter is correct in pointing out that this difference in distribution shows that Cr VI does not behave exactly the same as Cr III in terms of absorption and distribution.

Comment 3: “No specific peer-reviewed documentation accompanies this statement [that Cr VI is reduced to Cr III in the human stomach]. Are there specific reducing agents other than ascorbic acid available to reduce Cr VI to Cr III? Are there specific redox systems that will convert Cr VI to Cr III? In which organs are they located?”

Response 3: It is well documented that Cr VI is reduced to Cr III in the stomach. See Kerger et al. (1997). The text has been amended to include this reference. [Kerger, BD, Finley, BL, Corbett, GC, Dodge, DG, Paustenbach, DJ (1997). Ingestion of chromium (VI) in drinking water by human volunteers: absorption, distribution, and excretion of single and repeated doses. *Journal of Toxicology and Environmental Health* 50, 67-95.]

Comment 4: “Unreduced Cr VI moves through the entire digestive tract and is eliminated from the body. That fraction of the Cr VI that is absorbed is converted by the redox systems available in the liver to Cr III. But this conversion is obviously not complete, since Cr VI is found in the kidneys and in other organs.”

Response 4: All of these points are well taken, except perhaps the last. As noted above, we do not have direct evidence that Cr VI reaches the kidneys as Cr VI.

Comment 5: “The cellular penetrability of Cr VI exceeds that of Cr III.”

Response 5: Yes, this is stated in the document.

Comment 6: Cr VI is able to cross the placental barrier and cause severe fetal damage. It is also able to reduce female fertility and cause damage to testicular structures and functions.

Response 6: These potential reproductive and developmental effects are discussed in the text. The PHG that has been developed based on carcinogenicity is low enough to protect against these effects as well.

Comment 7: “Moreover, Cr VI causes increased immunotoxic effects and neurotoxic effects ... in animals by inhalation.”

Response 7: These effects are also discussed in the document. The PHG is low enough to be protective against these effects.

Comment 8: “The results of the [Borneff] study ... leave no doubt about the potential of Cr VI to induce carcinogenic growths.”

Response 8: OEHHA used this study as evidence for potential carcinogenicity of Cr VI by the oral route, and to calculate a cancer potency. However, there is room for some doubt, as the number of malignant tumors was not statistically significant.

Comment 9: “What is the source of electrons in gastric juice and its contents needed to convert Cr VI to Cr III?”

Response 9: Organic acids in food may provide the reducing potential. In any case it is clear that Cr VI is reduced to Cr III in the human stomach as discussed above. However, we lack evidence to tell us how quickly or thoroughly Cr VI is reduced in the human stomach.

Comment 10: “Considering the importance of the presence of hexavalent chromium in drinking water... I believe that using the mean value for the percentage of Cr VI in drinking water samples from two sources in North Carolina is incomprehensible.”

Response 10: OEHHA would like to have more data, but these were the only data found in the published literature after a very extensive search.

Comment 11: “As a scientist, I believe that many more water samples must be taken and analyzed to provide a reliable and meaningful value for the percentage of the total chromium present in drinking water as Cr VI.”

Response 11: We agree. More data are badly needed, especially for California drinking water sources. When more data become available, the PHG can be revised accordingly, as provided for in the law.

Comment 12: “The importance of these considerations lies in the fact that the geometric mean, 7.1 percent, is used with the calculated ToxRisk value of 0.18 ppb for the carcinogenicity of Cr VI to obtain the PHG for total chromium, 2.5 ppb.”

Response 12: We agree. More reliable data on the percentage of Cr VI in total chromium in California drinking water would be extremely helpful.

Comments from Upper Los Angeles River Area Watermaster

Comment 1: “There is ample evidence to support the carcinogenicity of chromium VI by inhalation; however, in reviewing the draft PHG for chromium, it appears the potential for chromium VI to be carcinogenic by oral ingestion was not scientifically determined.” The commenter goes on to criticize the fact that the determination of oral carcinogenicity was based on a single mouse study, and to point out that cancer mortality studies of individuals exposed to as high as 20 mg/L of chromium VI were negative.

Response 1: It is true that there was only one positive animal study, and this fact was acknowledged in the document. It is also true that the malignant tumors were not statistically significant, but the combined tumors were statistically significant. This was the only data set that could be used to calculate a cancer potency for chromium VI.

The epidemiological study referred to is that of Zhang and Li (1997). It is true that this study showed no statistically significant increase in cancer incidence in villages with high chromium VI levels in drinking water compared to other provinces in China. However, the study looked at the exposed populations only 13 years after their exposure. This period of time may not be sufficient to allow the appearance of cancers caused by the exposure. The study should be repeated at a later date. Another problem with the Zhang and Li (1997) study is that it does not describe the two unexposed populations that were used as controls. We cannot tell whether or not these control populations had high exposures to other carcinogens beside chromium VI. These deficiencies in the study make it impossible to draw any firm conclusions from it regarding the potential carcinogenicity of chromium VI in drinking water to human populations. Additional discussion of the Zhang and Li (1997) study has been added to the document in response to this comment.

The decision to regard chromium VI as a carcinogen was based on a weight of evidence approach. The PHG support document acknowledges that while there is some evidence that weighs against considering chromium VI an oral carcinogen, there is stronger evidence arguing in favor of its carcinogenicity. Individual epidemiological studies can yield misleading results for a number of reasons (confounding, lack of statistical power). A single epidemiological study cannot outweigh other positive evidence.

Comment 2: “The actual percentage of chromium VI contained in total chromium can vary greatly depending on location, season, and water source (i.e. surface water or groundwater).”

Response 2: The data used in the PHG document was the only data on speciation of chromium in water that OEHHA was able to locate. OEHHA has not been able to locate any data on speciation of chromium in California water supplies.

Comment 3: “However, the available studies seem to suggest that chromium VI may not be an oral carcinogen.”

Response 3: No studies are cited by the commenter, so it is not clear which “available studies” are being referred to. The reasons for regarding chromium VI as an oral carcinogen are given in the PHG document. The document acknowledges that there is controversy about whether chromium VI is a carcinogen by the oral route, but OEHHA has chosen to make the health protective assumption that it is based on a number of lines of evidence.

Comments from Merck Manufacturing Division

Comment 1: The commenter points out that the carcinogen potency for chromium VI was calculated based on a single study with a single dose group. He further points out that ATSDR concluded based on the Borneff study that chromium VI is unlikely to be a carcinogen by the oral route.

Response 1: It is true that OEHHA calculated cancer potency based on a single experiment with a single dose group. This was the only study available that could be used to calculate a cancer potency for chromium VI. OEHHA’s interpretation is independent of ATSDR’s.

Comment 2: “The Borneff study used potassium chromate to dose animals and Merck questions the relevance of these data to the determination of hazard and the estimation of risk for other chromium species.”

Response 2: The PHG is for total chromium, based on carcinogenicity of chromium VI ion in solution, not for particular chromium salts. The only data available on which to base a calculation of cancer potency for chromium VI ions was the Borneff study. There are no data on which to base a supposition that other chromium VI salts in drinking water would be more or less carcinogenic than potassium chromate.

Comment 3: “In deriving the cancer slope factor used to develop the PHG, the slope of a dose response curve with only two points, 0 mg/L and 500 mg/L was used. We feel this approach is inconsistent with accepted risk assessment methodology.” The comment goes on to state that such a calculation is statistically unreliable.

Response 3: OEHHA acknowledges that a study with multiple data points would be a better basis for a cancer potency calculation, however the Borneff study can still be used in the absence of better data.

Comment 4: “The proposed PHG of 0.2 ppb is far below all current regulatory standards for Cr VI in drinking water.” The comment goes on to discuss other chromium standards.

Response 4: OEHHA has developed a PHG of 2.5 ppb for total chromium in its final document. The “current regulatory standards” referred to are for total chromium, not for “Cr VI in drinking water.” PHGs are based solely on public health considerations and may be below regulatory levels which take into account other factors such as detection limits, purification methods, etc.

Comment 5: “The proposed PHG is well below the background concentrations for chromium in water.” The comment goes on to discuss levels of chromium found in some drinking water supplies, and concludes that “much of the drinking water in the State of California will be unnecessarily non-compliant.”

Response 5: PHGs are based solely on public health considerations, and do not take into account the frequency of occurrence of a contaminant in drinking water supplies.

Comment 6: “Merck and Co. Inc., requests your Office consider not adopting the proposed PHG for drinking water of 0.2 ppb for Cr VI.”

Response 6: OEHHA is not developing a PHG for Cr VI. OEHHA has adopted a PHG of 2.5 ppb for total chromium in its final document.

Comments from ACWA

Comment 1: “OEHHA has developed a PHG for chromium VI of 0.2 ppb and chromium III of 2,000,000 ppb.” The commenter goes on to suggest that OEHHA should consider sampling techniques, analytical methods and other practical considerations in reaching the proposed PHG.

Response 1: OEHHA is proposing only one PHG, for total chromium of 2.5 ppb. PHGs are intended to consider only public health concerns, and do not take into account sampling and analytical technology. These matters are considered by CDHS in setting the MCL.

Comment 2: “ACWA questions the departure from total chromium species.”

Response 2: OEHHA is not developing separate PHGs for the two significant species of chromium. OEHHA has developed only one PHG for total chromium in the final document.

Comment 3: The commenter urges OEHHA to consider the comments from Castaic Lake Water Agency to the effect that chromium VI is not a carcinogen by the oral route of exposure.

Response 3: OEHHA has considered those comments and responded to them.

Comments from Courtauds Aerospace

Comment 1: “We find that the OEHHA conclusions about the carcinogenicity of hexavalent chromium by the oral route are unfounded and inconsistent with other OEHHA actions and U.S. EPA information.” The comment goes on to quote from U.S. EPA to the effect that “the carcinogenicity of hexavalent chromium by the oral route of exposure cannot be determined.”

Response 1: The basis of the decision by OEHHA to regard hexavalent chromium as a carcinogen by the oral route is set forth in the PHG document. The OEHHA “action” referred to is dealt with below under comment number 4. OEHHA’s interpretation of the data is independent of U.S. EPA’s.

Comment 2: The commenter points out that the incidence of malignant tumors in the Borneff study was not statistically significant.

Response 2: OEHHA acknowledge that this incidence was not statistically significant. OEHHA decided to calculate the cancer potency based on combined data for malignant and non-malignant tumors. The combined incidence was statistically significant.

Comment 3: The comment refers to the Zhang and Li (1997) study reviewed in the PHG document, and points out that there was “no statistical increase in cancer mortality in the three most-exposed villages” which had chromium levels as high as 20 mg/L.

Response 3: OEHHA reviewed this study as part of the PHG analysis and acknowledges that the results of this study do not support the conclusion that chromium is an oral carcinogen in humans. However, this is a single ecological epidemiological study. There are no other epidemiological studies that compare cancer rates in areas with high chromium VI in drinking water versus areas without chromium VI. Single epidemiological studies sometimes yield misleading results either by chance or because of confounding or other problems. When numerous epidemiological studies are compared, often there are both positive and negative studies for the same chemicals. For this reason, negative results in a single epidemiological study cannot outweigh a strong set of positive evidence, including a positive result in an animal study (Borneff et al., 1968), and positive genotoxicity information.

Comment 4: “In the Proposition 65 regulations at Title 22 California Code of Regulations, 12707, Routes of Exposure, reads as follows: (b) the following chemicals present no significant risk of cancer by the route of ingestion: (4) Chromium (hexavalent compounds).”

Response 4: The Proposition 65 program has criteria for significant risk that are separate from the criteria used for the PHG program. The decision cited above represents a regulatory decision that does not necessarily imply that chromium VI is not a carcinogen by the oral route. The requirement for PHGs is to identify a level without significant risk. The proposition 65 level is the level with a significant risk. A cancer level associated with insignificant risk is generally considered to be below 1×10^{-6} in OEHHA. The Proposition 65 significant risk level is normally based on a 1×10^{-5} cancer risk level.

Comment 5: “The Borneff study was not mentioned in the U.S. EPA Toxicology Review of Hexavalent Chromium document.” The commenter speculates that “U.S. EPA did not consider the result significant.”

Response 5: It is difficult to draw a conclusion based on the absence of this study from U.S. EPA’s review. Even a study that is not statistically significant should be included in a complete review of the literature. OEHHA did review the study, and decided that it presents data that are useful in considering the potential of chromium VI to cause cancer in humans.

Comment 6: The commenter quotes NIOSH (1979) as saying that “certain forms of chromium VI are noncarcinogenic” including potassium chromate, the salt used in the Borneff study.

Response 6: This statement is related to carcinogenicity of chromium VI compounds by the inhalation route in humans. These conclusions do not necessarily apply to chromium ions in solution in drinking water. OEHHA assumes that all chromium VI salts are carcinogenic by the inhalation route. This determination was made after the NIOSH report was published. The Borneff study demonstrates that potassium chromate is carcinogenic to mice by the oral route of exposure.

Comment 7: The commenter quotes OEHHA’s seven arguments supporting the identification of chromium VI as an oral carcinogen, and comments on each one.

Response 7: These are responded to in the table below:

| OEHHA argument from PHG document | Comment from Mel Young, Courtaulds Aerospace | Response from OEHHA |
|---|--|--|
| 1. Chromium VI is a known human carcinogen by the inhalation route. | “This is not a valid reason for considering chromium VI to be carcinogenic by ingestion.” Commenter goes on to argue that inhalation involves direct deposition of particles in the lung, which would be very different from “dilute water solutions.” | The fact that chromium VI is a human carcinogen by the inhalation route demonstrates that this chemical has the ability to cause neoplastic transformation of human cells. This indicates that the cancer causing potential of Cr VI in humans is not in question. This is meant to address hazard identification, not dose/response assessment. This level of evidence is much greater than that for many other substances. |

| OEHHA argument from PHG document | Comment from Mel Young, Courtaulds Aerospace | Response from OEHHA |
|---|---|---|
| 2. Non-respiratory cancers have been found in workers exposed to chromium VI by inhalation. | “This data is not adequate to establish a relationship between ingested chromium VI contaminated water and stomach cancer.” | The non-respiratory cancers in workers do not establish that chromium VI would be carcinogenic in drinking water, but it does show that the carcinogenicity is not limited to the inhalation route. |
| 3. Inhaled chromium VI causes respiratory tumors in rats. | “This data is not relevant.” | The data are relevant in the sense that a chemical which is carcinogenic by one route may be carcinogenic by other routes. |
| 4. Chromium VI causes contact site tumors in laboratory animals. | There is no evidence to establish a relationship between direct skin contact with chromium VI in high concentration and oral ingestion of dilute solutions. | The contact site tumors include injection site tumors, so they are not limited to skin. These data contribute to the weight of evidence that chromium VI can be carcinogenic by several routes of exposure. |
| 5. Ingested chromium VI has been associated with stomach tumors in mice. | “The only report of this is the Borneff study, which was not statistically significant.” | It is true that there is only one such study. The study is statistically significant in terms of combined malignant and non-malignant stomach tumors. |
| 6. Chromium VI has been positive in a number of assays for genotoxicity. | “The lack of any other supporting data makes this unimportant.” | Genotoxicity is important evidence supporting a possible mechanism by which the chemical may act as a carcinogen. |
| 7. For the protection of public health, it is safer to assume that a substance which is carcinogenic by one route may also be carcinogenic by other routes. | “This is not a valid assumption. Many chemical substances have been found to be carcinogenic by a single route of exposure.” | In the case of chromium VI, a consideration of all available information does not lead to a compelling case that it is carcinogenic by only one route of exposure. The commenter did not provide evidence to support the statement that “many substances have been found to be carcinogenic by a single route of exposure.” |

Comment 8: “It is our conclusion that OEHHA should not establish a Public Health Goal for carcinogenicity for Chromium VI by the oral route of exposure.

Response 8: Having reviewed all the available evidence, OEHHA has concluded that a prudent public health decision is to regard chromium VI as a carcinogen by the oral route. OEHHA has therefore based its PHG for total chromium on the potential carcinogenicity of chromium VI by the oral route.

Comment 9: “Although it may be technically true that ‘PHGs established by OEHHA exert no regulatory burden’ it will establish a ceiling, above which it will be extremely difficult for regulatory agencies to set regulatory limits. Therefore, the recommended PHG must be evaluated for its regulatory impact.”

Response 9: In the past regulatory agencies have set MCLs and other regulatory limits which are very different from the recommended levels similarly derived for public health goals. The California Safe Drinking Water Act specifically requires OEHHA to consider only public health considerations (not “regulatory impact”) in setting PHGs.

Comment 10: “The Introduction section contains a summary of California water monitoring data from 1984 to 1996. The median value for total chromium was 17 mg/L [sic]. If we use the assumption of 7.1% chromium VI...more than half the drinking water sources in California would fail to meet the standard.”

Response 10: The median of 17 µg/L was for those water sources in which chromium was detected (detection limit of 10 µg/L). The document states that chromium was only detected in 9% of the water sources surveyed.

Comment 11: “This would also quite likely affect cleanup standards.”

Response 11: PHGs are not intended to be used for cleanup standards, however a cancer risk number based on the Borneff study has in the past been used as the basis for cleanup levels, so the cancer risk estimate in the PHG document would not introduce something totally new to the cleanup standards (David Siegel, personal communication).

Comment 12: “These problems, of course, would be acceptable as necessary if the Public Health Goal of 0.02 mg/L were based on sound science.”

Response 12: The PHG is 2.5 µg/L, or 0.0025 mg/L. OEHHA believes this level is based on sound science, together with a prudent consideration of public health in the face of scientific uncertainty. The PHG can be revised in the future if new data are produced which warrant a reconsideration of the question of carcinogenicity of chromium VI.

Comments from Coachella Valley Water District

Comment 1: Chromium VI is reduced to chromium III in the stomach. “Too stringent an MCL enacted for chromium VI could deprive someone with a diet otherwise deficient in chromium from receiving an essential dietary level of chromium III.”

Response 1: It is true that some individuals may benefit from ingesting chromium VI that is reduced to chromium III and contributes to their dietary requirement. However, this benefit must be weighed against the risk posed by chromium VI as a potential carcinogen. It would be much safer to ingest chromium III directly in the diet. Most drinking water sources in California contain no detectable chromium at all, so drinking water cannot be relied on as a source of dietary chromium. OEHHA has developed a PHG for total chromium. The MCL can be set at a higher level than the PHG based on other considerations.

Comment 2: “We believe inadequate data exists to justify a total chromium PHG of 2.5 parts per billion. OEHHA’s finding that chromium is a carcinogen by ingestion is unsupported and contradicts EPA’s finding that there is no evidence that chromium in drinking water has the potential to cause cancer....”

Response 2: In the PHG document OEHHA gives seven arguments for considering chromium VI as a carcinogen by the oral route (see response 7 to Mel Young, above). OEHHA’s document is independent of U.S. EPA’s decision-making process.

Comment 3: “Poor science exists throughout this document. OEHHA readily incorporates clearly unrepresentative data into the chromium PHG calculation while not accounting for what should be a significant component of this same calculation... the reduction of ingested chromium VI to chromium III in the human stomach.”

Response 3: OEHHA used data from two lakes in North Carolina to estimate the percentage of chromium VI in total chromium in potential drinking water supplies. No better data were found. Any new data representative of California water supplies will be used in the next consideration of chromium in drinking water. Likewise it would be useful to have data comparing the rate of reduction of chromium VI to chromium III in the human stomach with the rate in the rodent stomach. If such data were available to OEHHA, we could use them as part of the extrapolation from rodent to human cancer risk. Better data could result in a better basis for deriving a PHG, but OEHHA is required to develop a PHG for chromium with the data available.

Comment 4: “The conclusions used by OEHHA in developing the chromium PHG lack good, peer-reviewed scientific evidence setting a bad precedent for future risk assessment and risk management.”

Response 4: The chromium PHG document was peer-reviewed both within OEHHA and externally. The document cannot set any precedent for risk management, since it does not include any discussion of risk management.

Comments from Superfund Division, U.S. EPA

Comment 1: “In light of the insufficient data on this issue, we are concerned that OEHHA has assumed oral carcinogenicity of chromium (VI).”

Response 1: OEHHA made this decision based on the seven arguments set forth in the PHG support document, and discussed in response to comment 7 from Mel Young. OEHHA believes this decision is protective of public health in the face of scientific uncertainty.

Comment 2: “ We believe that further scientific review of the data for chromium is necessary before a new PHG for chromium exposure, via the oral route, is determined. Given our shared interest in chromium, we request the opportunity to participate with you in a scientific peer review.”

Response 2: During the formal review process, OEHHA sent the draft PHG document to three U.S. EPA programs for their review. The law requires OEHHA to review its PHGs at least every five years. During the review of the chromium PHG, OEHHA will be happy to receive and consider any input that U.S EPA wishes to contribute. We would be appreciative of any data which documents the absence of chromium VI carcinogenicity by the oral route.