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ATTN: PHG Project

Dear Mr. Baes:

These comments are submitted on behalf of NRDC (Natural Resources Defense Council), a nonprofit environmental policy organization with 1.2 million members and activists, more than 250,000 of whom are Californians. We are writing to comment on and support the Draft Public Health Goal (PHG) for Hexavalent Chromium in Drinking Water released in December 2010 (“OEHHA PHG Draft”). The proposed PHG is appropriate based on the best scientific information available, and it is correctly designed to protect vulnerable populations as required by statute. However, the California Legislature required that a primary drinking water standard for hexavalent chromium be established on or before January 1, 2004; therefore the PHG is at least seven years late, and it must be established immediately under the law.

Requirement to Establish a Drinking Water Standard

Under SB 351 (Ortiz), legislation that was signed into law on October 7, 2001, the State of California must have in place a primary drinking water standard for hexavalent chromium by January 1, 2004. The statute reads:

116365.5. (a) The Department of Health Services shall commence the process for adopting a primary drinking water standard for hexavalent chromium that complies with the criteria established under Section 116365.

(b) The department shall report to the Legislature on its progress in developing a primary drinking standard for hexavalent chromium by January 1, 2003.

(c) The department shall establish a primary drinking water standard for hexavalent chromium on or before January 1, 2004.

[Section 116365.5 Health and Safety Code]

No primary drinking water standard has been set for hexavalent chromium, and we have recently passed the seven year mark since the legislative deadline. The California Department of Public Health (DPH) is unable to set a primary drinking water standard under the law until OEHHA has finalized a Public Health Goal. Cal. Health and Safety

Code § 116365.5(a) presumes the existence of a PHG before a MCL is set; the primary drinking water standard (also known as a maximum contaminant limit or “MCL”) must be “set at a level that is as close as feasible to the corresponding public health goal.” Id. § 116365(a). In fact, for a newly regulated contaminant, the statute requires OEHHA to publish a PHG at the same time DPH proposes the adoption of a primary drinking water standard. Health & Saf. Code § 116365(e)(2). Therefore OEHHA must finalize the PHG immediately in order to allow the promulgation of a primary drinking water standard for this chemical.

Appropriate and Necessary to Regulate Hexavalent Chromium

It is necessary and appropriate to promulgate a PHG and an MCL for hexavalent chromium. The existing California standard for total chromium of 50 ppb is inappropriate and insufficient to protect public health. It is inappropriate to only set a regulatory standard for total chromium because the trivalent form of the metal is an essential nutrient at low doses and is relatively non-toxic, whereas the hexavalent form is highly toxic and carcinogenic. A standard based on total chromium conflates these two very different forms of the metal and is therefore scientifically inappropriate.

Furthermore it is necessary to regulate hexavalent chromium in drinking water. As of February 2009, 2,208 California water sources – about one-third of California water sources - contained hexavalent chromium at levels above 1 ppb. (OEHHA PHG Draft p. 3). Approximately ten percent of these have levels over 5 ppb. This information is alarming in light of the toxicity and cancer potency of hexavalent chromium, and it is urgent to reduce public exposures to this chemical as quickly as possible.

Ingestion of Hexavalent Chromium is Carcinogenic

Hexavalent chromium (CrVI) is a known carcinogen in humans. Multiple epidemiologic studies have shown cancers of the lung and respiratory tract in workers in the electroplating industry and other chromium-exposed worker populations. The hypothesis that CrVI may be carcinogenic only when inhaled has been disproven in both animals and humans. A large and determinative study by the National Toxicology Program (NTP) published in 2008, found statistically significant and dose-related tumors of the small intestine in mice and in the oral cavity in rats. The one epidemiologic study that has been done on ingestion of CrVI identified a statistically significant increase in mortality related to stomach cancers in a population exposed to the chemical via drinking water. This latter study was subject to a highly controversial and ethically-suspect reanalysis by an industry consulting firm that purported to find no significant increase in cancers. It was therefore appropriate for OEHHA to conduct an independent statistical analysis of the data to sort out the conflicting findings.

Appropriate to Use a Linear Model

Some commenters have advanced the hypothesis that there may be a threshold below which CrVI is not carcinogenic in humans. There is very little basis for such a

hypothesis, and the available data at this time indicate that a threshold is highly unlikely, and even if such a threshold exists, it is unlikely to be relevant at a population level. Mechanistic data suggest the absence of a threshold; CrVI is a genotoxic and mutagenic carcinogen as evidenced by its behavior in numerous bioassays. CrVI causes elevated levels of sister chromatid exchange, chromosomal aberrations, DNA-protein crosslinks, and mutations in multiple *in vitro* and *in vivo* systems. Mutagenic carcinogens are assumed not to have a threshold, as even low levels can interact with DNA and cause genetic mutations that increase the risk of cancer.

Another argument for a threshold posits that although at a cellular level there may be no threshold, in the acidic gastric environment, most ingested CrVI is converted to non-toxic CrIII and is therefore inactivated, theoretically resulting in no exposure to the cells of the stomach or small intestine (ie. the site of the cancers). This argument presumes that everyone in the population has a gastric environment that is capable of rapidly reducing CrVI to CrIII prior to any contact with cells. Such an assumption is highly suspect for three reasons: (1) CrVI is rapidly transported into cells via the anion transport system, so even brief contact with the gastric cells can result in rapid uptake and resulting DNA damage; (2) the process of transformation of CrVI to CrIII generates free radicals and reactive intermediates that themselves can cause cell and DNA damage; and (3) the reduction of CrVI is reliant on a gastric environment with a very low Ph (<4), which is not present in a large proportion of the population. OEHHA correctly points out that: “Infants’ stomachs are near neutral pH during the first days to weeks after birth, and stomach pH levels generally remain higher than adults during the first three months of life.” (OEHHA Draft PHG p. 70). Furthermore, millions of people take over-the-counter antacid medications, or prescription medications to treat gastritis, ulcers, and gastrointestinal reflux disease. These medications, especially the Proton Pump Inhibitors, are designed to increase the pH of the stomach environment to over 4. In such a gastric environment, efficient reduction of CrVI would not occur, and the half-life of this carcinogenic compound would therefore be significantly longer resulting in much higher risk. Therefore the presence of a threshold would be highly unlikely when sensitive members of the population are included in the distribution.

Need to Adjust to Protect Vulnerable Populations

We commend OEHHA for significantly improving on the 1999 draft PHG by following its own guidelines for assessing early-life susceptibility to carcinogens. OEHHA was directed by the California legislature in 2000 to “to assess methodologies used in addressing early-in-life risk, compile animal data to evaluate those methods, and develop methods to adequately address carcinogenic exposures to the fetus, infants, and children” [AB 2872 (Shelley), in Health & Saf. Code § 901 [a-e]]. OEHHA completed this analysis and published it in a May 2009 document entitled: “In Utero and Early Life Susceptibility to Carcinogens: The Derivation of Age-at-Exposure Sensitivity Measures” (OEHHA 1999), [http://oehha.ca.gov/air/hot_spots/2009/AppendixJEarly.pdf]. As outlined in OEHHA 1999, cancer potency is adjusted by an Age Sensitivity Factor that accounts for the scientific data that show children can be more susceptible to carcinogens. These well-

researched adjustment factors apply appropriately to CrVI. In fact, it is reasonable to assume that infants, whose gastric pH is essentially neutral, are at especially high risk from a contaminant such as CrVI, and it is even possible that an adjustment factor of 10-fold for that age group may not be sufficient.

In summary, the OEHHA draft PHG for CrVI is well-researched, scientifically sound, and is reasonably health-protective. We commend OEHHA for completing this draft, and we urge that it be finalized quickly so that an enforceable drinking water standard can be set as required by law.

Thank you for the opportunity to comment.

Sincerely,



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Senior Scientist



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