MEMORANDUM

TO: Joan E. Denton, Ph.D.
    Director

VIA: George V. Alexeeff, Ph.D.
    Deputy Director for Scientific Affairs

VIA: Anna M. Fan, Ph.D., Chief
    Pesticide and Environmental Toxicology Branch

VIA: Robert A. Howd, Ph.D., Chief
    Water Toxicology Section
    Pesticide and Environmental Toxicology Branch

FROM: Richard Sedman, Ph.D.
    Staff Toxicologist
    Pesticide and Environmental Toxicology Branch

DATE: December 19, 2006

SUBJECT: UPDATE OF THE PUBLIC HEALTH GOAL FOR CADMIUM

Under the Calderon-Sher California Safe Drinking Water Act of 1996, the Office of Environmental Health Hazard Assessment (OEHHA) develops public health goals (PHGs) for regulated chemicals in drinking water and reviews and updates the risk assessments every five years (Health and Safety Code Section 116365(e)(1)). This memorandum announces an update of the literature review and re-evaluation of the existing PHG for cadmium. Our re-evaluation supports a lowering of the previous PHG, derived in 1999, from 0.07 parts per billion (ppb) to 0.04 ppb.

Summary of Review

Cadmium is a heavy-metal trace element which is used in a number of industrial processes, and has been associated with both occupational and non-occupational toxicity. Environmental levels of cadmium have been increasing from smelting, cement production, and combustion of fossil fuels. Drinking water does not appear to be a significant source of cadmium exposure.
The current federal and California Maximum Contaminant Level (MCL) for cadmium is 5 ppb; the detection limit for the purpose of reporting (DLR) is 1 ppb. According to the California Department of Health Services monitoring report (CDHS, 2006), cadmium was detected in drinking water sources 11 times over the past four years (2002-2005); none of these detections exceeded the MCL.

The cadmium PHG of 0.04 ppb is based on kidney toxicity. Kidney is a sensitive organ because of the gradual accumulation of cadmium in this tissue over the first several decades of life. Changes in the level of certain urinary proteins, urinary biomarker that are very sensitive indicators of the onset of renal toxicity, were used to identify an exposure level that would not result in renal toxicity. Studies in humans showed that when urinary cadmium levels exceed 2 to 5 µg/g creatinine, proteinuria indicative of renal damage begins to occur. When urinary cadmium levels remain at or below 1 µg/g creatinine, proteinuria and other more severe signs of renal toxicity are not detected.

Toxicokinetic studies in human males were employed to estimate the long-term daily oral cadmium intake associated with urinary cadmium levels of 1 µg/g creatinine after 50 years of exposure. This represents an update of the methods used to estimate the PHG level in the 1999 PHG document. A daily oral intake of cadmium of 19 µg/day is estimated to yield a urinary cadmium level of 1 µg/g creatinine after 50 years of exposure, which is considered by OEHHA to be equivalent to a no-observed-adverse-effect level (NOAEL) for cadmium in humans. A relative lack of data on the toxicokinetics and critical effect level of cadmium in women, who appear to be more sensitive to many cadmium effects, led us to apply an uncertainty factor of 5 to this level to protect this sensitive population. The increased sensitivity is based on the observation of a higher body burden of cadmium in women which appears to be due to a higher level of oral absorption but other factors may be involved; occurrence of Itai Itai disease, a disease caused by cadmium, principally in women; and significant increases in mortality observed in women but not men exposed to cadmium in rice in Japan.

OEHHA (2005), IARC (1993), and U.S. EPA (2006) have determined that there is sufficient evidence that cadmium is carcinogenic to humans. No oral studies in humans or animals were identified that were judged suitable for developing an oral cancer potency. Available information that would allow an extrapolation of the inhalation potency to the oral route was determined by OEHHA to be inadequate and therefore extrapolation of the inhalation potency to the oral route was judged to be inappropriate. To address cancer risk due to oral exposure to cadmium, an additional 10-fold uncertainty factor was employed in the derivation of the PHG. A relative source contribution of 20 percent was selected for calculation of the PHG because most exposure is attributable to sources other than drinking water.

The NOAEL of 19 µg/day, an overall uncertainty factor of 50, and a relative source contribution of 20 percent, results in the PHG of 0.04 µg/L for cadmium. Full documentation of the risk assessment is available on the OEHHA Web site at www.oehha.ca.gov/water.html.
References:


