PUBLIC NOTICE

Initiation of Process to Update Public Health Goals in Drinking Water and Request for Relevant Information: Carbofuran, Diquat, Endrin, Radium-226, Radium-228, Strontium-90, Thiobencarb, Toxaphene, and Tritium

Office of Environmental Health Hazard Assessment
California Environmental Protection Agency

October 17, 2014

Public health goals (PHGs) are concentrations of drinking water contaminants that pose no significant acute or chronic health risks. The Office of Environmental Health Hazard Assessment (OEHHA) establishes PHGs, which are used as the health basis for the development of California's primary drinking water standards. OEHHA also reviews and updates existing PHGs. This public notice announces the initiation of update reviews for the following nine chemicals:

- Carbofuran
- Diquat
- Endrin
- Radium-226
- Radium-228
- Strontium-90
- Thiobencarb
- Toxaphene
- Tritium

Updates of existing PHGs for these chemicals are being initiated based on the availability of new data, methodology updates, environmental exposures, and/or potential significance. A brief description of these chemicals is provided below. OEHHA is requesting information on these contaminants that could assist in updating the risk assessment and potentially calculating a revised PHG.

All information submitted to OEHHA in response to this request is considered public. Please do not submit proprietary information. In order to be considered during the update process, **OEHHA must receive information by 5:00 p.m. on Monday, November 17, 2014. We encourage you to submit information in electronic form, rather than in paper form. Information transmitted by e-mail should be addressed to PHG.Program@oehha.ca.gov with the chemical name in the subject line.** Information submitted in paper form may be mailed or delivered in person to the address below:

Hermelinda Jimenez
PHG Program
Pesticide and Environmental Toxicology Branch
Office of Environmental Health Hazard Assessment
1515 Clay St., 16th floor
Oakland, California 94612

If you have any questions, please contact Ms. Hermelinda Jimenez at (510) 622-3173 or email PHG.Program@oehha.ca.gov.

All relevant information will be considered in the development of the PHGs for these chemicals. The State Water Resources Control Board (SWRCB) will use the final risk assessments in considering potential revisions to the existing regulatory Maximum Contaminant Levels (MCLs) for these chemicals. For more information on this process, go to the SWRCB web site at http://www.waterboards.ca.gov/drinkingwater/.

Background on PHG Program

The Calderon-Sher California Safe Drinking Water Act of 1996 (Health and Safety Code section 116365), hereafter referred to as the Act, requires OEHHA to post a notice on its web site when it initiates work to develop new or update existing PHGs. PHGs are concentrations of chemicals in drinking water that are not anticipated to produce adverse health effects. OEHHA is required to consider potential adverse effects on members of sensitive subgroups of the population, including infants, children, pregnant women, the elderly, and individuals with a history of serious illness.

PHGs are non-regulatory in nature but are used as the health basis to update the state's primary drinking water standards (MCLs) established by SWRCB. The Act requires OEHHA to develop PHGs for approximately 90 chemicals for which state or federal MCLs are provided. The Act says that OEHHA shall review these PHGs every five years and update them as appropriate. SWRCB may also ask OEHHA to develop a PHG for a contaminant that it wishes to regulate through adoption of an MCL.

At the initial posting of a draft document, a 45-day public comment period is provided. A public workshop will be held to hear public comment on the draft. An external scientific peer review will then be conducted as required by Health and Safety Code section 57004(b). All relevant comments received are considered in the preparation of the next draft, which is posted for a 30-day public comment period. After consideration of any additional comments, the PHG is finalized and published on the OEHHA web site for public reference and for use by SWRCB in developing California MCLs.

OEHHA has published PHGs for 89 chemicals, and re-evaluations of the original PHG have been completed for 29 of these chemicals. All PHGs and supporting documents are available at http://www.oehha.ca.gov/water/phg/index.html.

PHGs in progress

PHG documents are currently in progress for several chemicals for which initiation of review has previously been announced. The draft trihalomethanes PHG is undergoing revision following the second comment period. An update of the 2004 perchlorate PHG is nearing completion. Listed below are other chemicals for which PHGs are being updated and that have been previously noticed.

- Alachlor
- Antimony
- Atrazine/Simazine
- Cyanide
- Diethylhexylphthalate
- 1,1-Dichloroethane
- 1,2-Dibromo-3-chloropropane
- 1,4-Dichlorobenzene
- 1,1-Dichloroethylene
- 1,2-Dichloropropane
- Ethylbenzene
- Fluoride
- Haloacetic acids
- Nitrate/Nitrite
- Picloram
- 1,2,4-Trichlorobenzene
- Trichloroethylene
- Xylene

Descriptions of chemicals or substances subject to this notice

CARBOFURAN

Carbofuran (2,3-dihydro-2,2-dimethyl-7-benzofuranyl methylcarbamate; Chemical Abstracts Service (CAS) Registry Number: 1563-66-2) is a highly toxic insecticide and nematocide that was banned for use in the U.S. in 2011. It belongs to the carbamate class of pesticides and causes neurotoxicity in pest species via a rapid, although reversible, inhibition of acetylcholinesterase. Prior to its cancellation, there were a number of products registered in California under the trade name Furadan® with carbofuran as the active ingredient (DPR, 2014a). Use of carbofuran in California has declined over the last decade; four pounds were applied in 2010 and one pound in 2011 (DPR, 2014b). In the last three years, carbofuran has not been detected in California drinking water supply wells at levels above its detection limit of 5 parts per billion (ppb) for purposes of reporting.[1]

Based on reproductive toxicity in male rats, a PHG of 1.7 ppb for carbofuran was published in 2000. The update of the risk assessment for carbofuran will consider the more recent toxicology literature since the publication of the PHG in 2000, and will incorporate the application of updated risk assessment methodologies.

[1] Data accessed with GeoTracker GAMA: http://geotracker.waterboards.ca.gov/gama/

Selected References

Aziz N, Shah SW, Aziz RN (2008). Histological changes in male rat reproductive organs post-treated with insecticide carbofuran (Furadan). *Ann Microsc* 8: 83-89.

Baligar PN and Kaliwal BB (2002). Reproductive toxicity of carbofuran to the female mice: Effects on estrous cycle and follicles. *Ind Health* 40: 345-352.

Brkić DV, Vitorović SL, Gašić SM, Nešković NK (2008). Carbofuran in water: Subchronic toxicity in rats. *Environ Toxicol Pharmacol* 25: 334-341.

Chauhan LKS, Pant N, Gupta SK, Srivastava SP (2000). Induction of chromosome aberrations, micronucleus formation and sperm abnormalities in mouse following carbofuran exposure. *Mutat Res* 465: 123-129.

DPR (2014a). Output reporting for carbofuran, all products. Query retrieved on 10 June 2014, from http://apps.cdpr.ca.gov/cgi-bin/label/labq.pl?p_chem=106&activeonly=off.

DPR (2014b). Summary of Pesticide Use Report Data 2012, Indexed by Chemical. Department of Pesticide Regulation, California Environmental Protection Agency, Sacramento, CA.

Elayan OEA, Karyono S, Sujuti H (2013). The effect of carbofuran on testosterone serum concentrations and histological change of leydig cell in mice. *J Pharm Biol Sci* 7: 1-4.

Gallegos-Avila G, Ancer-Rodríguez J, Niderhauser-García A, Ortega-Martínez M, Jaramillo-Rangel G (2010). Multinucleation of spermatozoa and spermatids in infertile men chronically exposed to carbofuran. *Reprod Toxicol* 29:458-460.

OEHHA (2000). Public Health Goals for Chemicals in Drinking Water: Carbofuran. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA.

Satar S, Satar S, Sebe A, Yesilagac H (2005). Carbofuran poisoning among farm workers. *Mt Sinai J Med* 72: 389-392.

DIQUAT

Diquat, 1,1'-ethylene-2,2'-dipyridylium, is a charged quaternary ammonium compound used in commerce as a dibromide salt (CAS Registry Number: 85-00-7). Diquat dibromide is used as a pre-harvest desiccant in alfalfa seed production and as a nonselective herbicide to control broadleaf weeds. Total usage of diquat dibromide in California in 2012 was 88,834 pounds (DPR, 2014). In the past three years, there have

been four detections of diquat in California public water supply wells, with concentrations ranging from 0.44 to 2.8 ppb,[2] considerably below the California MCL of 20 ppb and PHG of 15 ppb.

The primary health effect of diquat exposure observed in animal studies is the formation of cataracts, which was the basis of the PHG published in 2000. Neurotoxic effects have been observed in humans after exposure to high doses of diquat (OEHHA, 2000). However, limitations of case reports have precluded their use in developing health-protective levels for diquat.

The update of the risk assessment for diquat will consider the more recent toxicology literature since the publication of the PHG in 2000, and incorporate the application of updated risk assessment methodologies.

Selected References

DPR (2014). Summary of Pesticide Use Report Data 2012, Indexed by Chemical. Department of Pesticide Registration, California Environmental Protection Agency, Sacramento, CA.

Dimitrov BD, Gadeva PG, Benova DK, Bineva MV (2006). Comparative genotoxicity of the herbicides Roundup, Stomp and Reglone in plant and mammalian test systems. *Mutagenesis* 21: 375-382.

Greenlee AR, Ellis TM, Berg RL (2004). Low-dose agrochemicals and lawn-care pesticides induce developmental toxicity in murine preimplantation embryos. *Environ Health Perspect* 112: 703-709.

Higuchi M, Yoshikawa Y, Orino K, Watanabe K (2011). Effect of diquat-induced oxidative stress on iron metabolism in male Fischer-344 rats. *Biometals* 24: 1123-1131.

Karuppagounder SS, Ahuja M, Buabeid M, Parameshwaran K, Abdel-Rehman E, Suppiramaniam V, Dhanasekaran M (2012). Investigate the chronic neurotoxic effects of diquat. *Neurochem Res* 37: 1102-1111.

OEHHA (2000). Public Health Goals for Chemicals in Drinking Water: Diquat. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA.

[2] Data accessed with GeoTracker GAMA: http://geotracker.waterboards.ca.gov/gama/

ENDRIN

Endrin (CAS Registry Number: 72-20-8) is an organochlorine pesticide used to control insects, rodents, and birds. In the United States, all permitted uses of endrin were discontinued by 1991 (OEHHA, 1999). Due to its persistence, endrin residues could potentially be found in agricultural soils and hazardous waste sites, and releases from these sites can continue to be sources of endrin exposure to humans. However, endrin has not been detected at levels above 0.1 ppb, its detection limit for purposes of reporting, in California public water supplies in the last three years.[3]

Adverse health effects of endrin include seizures and pathological changes in the liver observed in dogs. These effects formed the basis of the PHG of 1.8 ppb published in 1999 and updated in 2008. Endrin is also listed under Proposition 65 as a chemical known by the State of California to cause reproductive toxicity (developmental endpoint).

The update of the risk assessment for endrin will consider the more recent toxicology literature since the update of the PHG in 2008, and incorporate the application of updated risk assessment methodologies.

Selected References

Allen EMG, Florang VR, Davenport LL, Jinsmaa Y, Doorn JA (2013). Cellular localization of dieldrin and structure-activity relationship of dieldrin analogues in dopaminergic cells. *Chem Res Toxicol* 26: 1043-1054.

Bedi JS, Gill JPS, Aulakh RS, Kaur P, Sharma A, Pooni PA (2013). Pesticide residues in human breast milk: risk assessment for infants from Punjab, India. *Sci Total Environ* 463-464: 720-726.

CDC (2013). Center for Disease Control and Prevention (CDC) National Biomonitoring Program Biomonitoring Summary, Organochlorine Pesticides Overview: Endrin CAS No. 72-20-8. In: The Fourth National Report on Human Exposure to Environmental Chemicals, Updated Tables, September. http://www.cdc.gov/biomonitoring/Endrin_BiomonitoringSummary.html.

Chung SWC, Chen BLS (2011). Determination of organochlorine pesticide residues in fatty foods: a critical review on the analytical methods and their testing capabilities. *J Chromatogr A* 1218: 5555-5567.

Freire C, Lopez-Espinosa MJ, Fernandez, M, Molina-Molina JM, Prada R, Olea N (2011). Prenatal exposure to organochlorine pesticides and TSH status in newborns from Southern Spain. *Sci Total Environ* 409: 3281-3287.

[3] Data accessed with GeoTracker GAMA: http://geotracker.waterboards.ca.gov/gama/

OEHHA (1999). Public Health Goal for Endrin in Drinking Water. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA.

RADIUM-226/228

Radium-226 and -228 (CAS Registry Number: 7440-14-4) are naturally-occurring radionuclides formed from the decay of the primordial radionuclides uranium-238 and thorium-232, respectively, in the earth's crust. Thus, there is a small amount of radium-226 and -228 in most environmental media, including drinking water. In the past three years, radium-226 and -228 have been detected in California public drinking water supplies at concentrations ranging from 0.01 to 96 picocuries per liter (pCi/L) and 0.001to 12.4 pCi/L, respectively.[4] California's MCL is 5 pCi/L for both radium-226 and -228.

The 2006 PHGs of 0.050 and 0.019 pCi/L for radium-226 and -228, respectively, were based on carcinogenic effects and were developed using the cancer risk coefficients presented in the U.S. Environmental Protection Agency's (U.S. EPA) *Cancer Risk Coefficients for Environmental Exposure to Radionuclides: Federal Guidance Report No. 13* (FGR 13). A health-protective concentration of 200 pCi/L for both radium-226 and -228 was also calculated for the non-carcinogenic endpoint of jaw necrosis (OEHHA, 2006).

Since the publication of the radium-226 and -228 PHGs in 2006, two epidemiological studies on radium have been published. The update of the radium-226 and -228 PHGs will consider these new studies, as well as incorporate updated risk assessment methodologies where appropriate.

Selected References

Cech I, Burau KD, Walston J (2007). Spatial distribution of orofacial cleft defect births in Harris County, Texas, 1990 to 1994, and historical evidence for the presence of low-level radioactivity in tap water. *South Med J* 100: 560-569.

Hirunwatthanakul P, Sriplung H, Geater A (2006). Radium-contaminated water: a risk factor for cancer of the upper digestive tract. *Asian Pac J Cancer Prev* 7: 295-298.

OEHHA (2006). Public Health Goals for Chemicals in Drinking Water: Radium-226 and -228. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA.

[4] Data accessed with GeoTracker GAMA: http://geotracker.waterboards.ca.gov/gama/

Szabo Z, DePaul VT, Fischer JM, Kraemer TF, and Jacobson E (2012). Occurrence and geochemistry of radium in water from principle drinking-water aquifer systems of the United States. *App Geochem* 27: 729-752.

U.S. EPA (1999). Cancer Risk Coefficients for Environmental Exposures to Radionuclides. Federal Guidance Report No. 13. United States Environmental Protection Agency, Washington, D.C. September 1999. EPA 402-R-99-001.

STRONTIUM-90

Strontium-90 (CAS Registry Number: 10098-97-2) is a radioactive isotope released as fallout from atmospheric testing of nuclear weapons and incidental events at nuclear processing facilities. Strontium-90 has a half-life of approximately 29 years and decays to yttrium-90 (half-life of 64.4 hours). Strontium-90 is currently in use as a radiation source to treat cancer and as an environmental tracer. In the last three years, it has been infrequently detected in California public drinking water supply wells at levels below the MCL (5 pCi/L), ranging from 0.507-1.59 pCi/L.[5]

The PHG of 0.35 pCi/L, published in 2006, was calculated based on known carcinogenic effects of radiation. The U.S. EPA's FGR 13 (U.S. EPA, 1999) served as the basis for this calculation. A health-protective concentration of 600 pCi/L was also calculated utilizing a non-carcinogenic endpoint of reduced life expectancy in beagle dogs (White et al., 1993).

Since the publication of the PHG in 2006, several studies on the biokinetics of strontium-90 have been published. Additionally, a case-control study examining the correlation between the amount of strontium-90 in deciduous teeth and cancer was published. These studies will be reviewed and the PHG for strontium-90 will incorporate updated risk assessment methodologies where appropriate.

Selected References

Li WB, Hollriegl V, Roth P, and Oeh U (2008). Influence of human biokinetics of strontium on internal ingestion dose of 90Sr and absorbed dose of 89Sr to organs and metasteses. *Radiat Environ Biophys* 47: 225-239.

Mangano J, and Sherman JD (2011). Elevated *In vivo* strontium-90 from Nuclear weapons test fallout among cancer decedents: a case-control study of deciduous teeth. *Internat J Health Services* 41: 137-158.

OEHHA (2006). Public Health Goals for Chemicals in Drinking Water: Strontium-90. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA.

[5] Data accessed with GeoTracker GAMA: http://geotracker.waterboards.ca.gov/gama/

Synhaeve N, Stefani J, Tourlonias E, Dublineau I, and Bertho JM (2011). Biokinetics of 90Sr after chronic ingestion in a juvenile and adult mouse model. *Radiat Environ Biophys* 50: 501-511.

U.S. EPA (1999). Cancer Risk Coefficients for Environmental Exposures to Radionuclides. Federal Guidance Report No. 13. United States Environmental Protection Agency, Washington, D.C. September 1999. EPA 402-R-99-001.

THIOBENCARB

Thiobencarb (CAS Registry Number: 28249-77-6) is a pre-emergent and early post-emergent systemic thiocarbamate herbicide used to control many broadleaf weeds, grasses, and sedges in food crops such as rice. This product is currently registered for use under the trade name Bolero®, and 278,647 pounds of thiobencarb were used in California in 2012. Thiobencarb was previously associated with a bitter taste in the drinking water, primarily due to the runoff from treated rice fields into the Sacramento River. Because of this, a secondary MCL of 1 ppb was set for thiobencarb, to prevent the off-taste of drinking water. In the past three years, thiobencarb has been detected at concentrations ranging from 0.5 to 1.5 ppb in California public water supply wells.[6] California's primary MCL for thiobencarb is 70 ppb. There is no federal MCL for thiobencarb.

The PHG of 70 ppb for thiobencarb, published in 2000, was based on decreased body weight gain observed in a chronic feeding study in rats (OEHHA, 2000). Recently, the European Food Safety Authority (EFSA) considered a proposal to raise the maximum residue level for thiobencarb in or on food from 0.1 milligrams per kilogram (mg/kg) to 0.2 mg/kg based on the Evaluating Member State's (EMS) opinion that the decreased body weight gain in rats was not treatment-related. The EMS further suggested that the acceptable daily intake should be based on liver histopathological changes observed in a mouse chronic toxicity study (EFSA, 2013).

The update of the thiobencarb PHG will consider the findings and information in the EFSA report (EFSA, 2013), as well as other recent toxicology literature since the publication of the PHG in 2000, and incorporate the application of updated risk assessment methodologies.

Selected References

EFSA (2013). Reasoned opinion on the setting of a new MRL for thiobencarb in rice. Environmental Food Safety Authority. *ESFA Journal* 11: 3427.

[6] Data accessed with GeoTracker GAMA: http://geotracker.waterboards.ca.gov/gama/

Kaya B, Marcos R, Yanikoğlu A, Creus A (2004). Evaluation of the genotoxicity of four herbicides in the wing spot test of *Drosophila melanogaster* using two different strains. *Mutat Res* 557: 53-62.

OEHHA (2000). Public Health Goals for Chemicals in Drinking Water: Thiobencarb. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA.

Sancho E, Fernández-Vega C, Ferrando MD, Andreu-Moliner E (2003). Eel ATPase activity as biomarker of thiobencarb exposure. *Ecotoxicol Environ Saf* 56: 434-441.

TOXAPHENE

Toxaphene (CAS Registry Number: 8001-35-2) is a persistent organochlorine pesticide consisting of a complex mixture of chlorinated compounds, primarily bornanes and camphenes, and a wide range of molecular weights. Toxaphene has not been used in California for over 20 years, but it was used heavily in 1975 and 1976 as a substitute for DDT (1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane), which was banned in the U.S. in 1972. Toxaphene is persistent in soils and sediments, with a half-life ranging from at least a year to longer than 14 years. However, it has not been detected at levels above 1 ppb, its detection limit for purposes of reporting, in California public water supply wells in the last three years.[7]

Toxaphene is listed as a toxic air contaminant by California's Air Resources Board and is on the Proposition 65 list as a carcinogen. The 2003 PHG of 0.03 ppb for toxaphene was based on carcinogenicity observed in mice (OEHHA, 2003). A health-protective concentration of 10 ppb was developed for the non-carcinogenic effects of toxaphene based on histological changes in the liver, kidney and thyroid of rats. The California and federal MCL for toxaphene is 3 ppb.

The update of the risk assessment for toxaphene will consider the more recent toxicology literature since the publication of the PHG in 2003, and incorporate the application of updated risk assessment methodologies.

Selected References

ATSDR (2010). Toxicological Profile for Toxaphene, CAS# 8001-35-2, Draft for Public Comment. Agency for Toxic Substances and Disease Registry (ATSDR), Centers for Disease Control and Prevention (CDC), Public Health Service (PHS), U.S. Department of Health and Human Services (DHHS), Atlanta, GA.
http://www.atsdr.cdc.gov/toxprofiles/tp94.pdf

[7] Data accessed with GeoTracker GAMA: http://geotracker.waterboards.ca.gov/gama/

Bartos, T., Skarek, M., Cupr, P., Kosubova, P., Holoubek, I. (2005). Genotoxic activity of a technical toxaphene mixture and its photodegradation products in SOS genotoxicity tests. *Mutat Res* 565(2):113-120.

NTP (2011). Toxaphene. In: Report on Carcinogens, 12th edition, pp. 418-420. National Toxicogy Program (NTP), National Institute of Environmental Health Sciences (NIEHS), NIH, DHHS, Research Triangle Park, NC. http://ntp.niehs.nih.gov/ntp/roc/twelfth/profiles/toxaphene.pdf

OEHHA (2003). Public Health Goal for Chemicals in Drinking Water: Toxaphene. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA.

TRITIUM

Tritium (CAS Registry Number: 10028-17-8) is a low energy, beta-emitter with a half-life of approximately 12 years. Low levels of tritium exist in the natural environment, but anthropogenic use of nuclear technology is the most common source of elevated levels. Tritium is chemically similar to hydrogen and is generally present in the environment as either organically-bound tritium (OBT) or incorporated in a water molecule (HTO). Over the past three years, tritium has been detected at levels ranging from 0.4 to 714 pCi/L in California public water supply wells.[8] California's MCL for tritium is 20,000 pCi/L.

The PHG of 400 pCi/L, published in 2006 and based on carcinogenic effects, was developed using a cancer risk coefficient presented in U.S. EPA's FGR 13. Health-protective concentrations of 6×10^7 pCi/L for children and 2.1×10^8 pCi/L for adults were also calculated for non-carcinogenic hematopoietic effects (OEHHA, 2006).

A renewed interest in tritium has emerged over the last five years due to concerns of beta radiation being more damaging than originally suspected (Kocher and Hoffman 2011). Furthermore, U.S. EPA recently released a revision of its methodology for estimating cancer risks from radiation exposure, *EPA Radiogenic Cancer Risk Models and Projections for the U.S. Population* (U.S. EPA, 2011). The estimates of radiogenic risk in the U.S. EPA report are based, for the most part, on models recommended in the National Academy of Sciences' *Health Risks from Exposure to Low Levels of Ionizing Radiation*, *BEIR VII Phase 2* (NAS, 2006).

An update to the PHG for tritium will include revised risk assessment methodologies as well as consider the most recent developments from the aforementioned documents and scientific literature.

[8] Data accessed with GeoTracker GAMA: http://geotracker.waterboards.ca.gov/gama/

Selected References

Kocher DC and Hoffman FO (2011). Drinking water standard for tritium-what's the risk? *Health Phys* 101: 274-285.

NAS (2006). Health Risks from Exposure to Low Levels of Ionizing Radiation. BEIR VII, Phase 2. National Academy of Sciences, Washington, D.C.

OEHHA (2006). Public Health Goals for Chemicals in Drinking Water: Tritium. Office of Envrionmental Health Hazard Assessment, California Environmental Protection Agency, Sacramento, CA.

U.S. EPA (2011). EPA Radiogenic Cancer Risk Models and Projections for the U.S. Population. Office of Radiation and Indoor Air, United States Environmental Protection Agency, Washington, D.C.