Appendix D

Maximum Contaminant Levels and Health Advisory Levels

General Information Regarding Health Standards

To understand the significance of exceeding health-based levels for noncarcinogens, one must first understand the method used to calculate these values. The lifetime health advisory, which is also known as the maximum contaminant level goal (MCLG), is derived through a three step process (EPA 1993). The critical step is the calculation of the oral reference dose (RfD), which is the dose below which no adverse noncarcinogenic health effects should result from a lifetime of exposure. This value is typically based on a subchronic or chronic animal study in which the dose at which no adverse effects occurred is identified. Sometimes, however, a study is selected where all doses administered to the animals resulted in adverse health effects. In this case, the lowest dose is selected to derive the RfD. When the study and dose level have been identified, the RfD is calculated by dividing the dose by an uncertainty factor that considers inadequacies of the study, animal-to-human extrapolation, sensitive subpopulations, and inadequacies of the database. This adjustment factors in a margin of safety into the calculation of the RfD.

The second step in derivation of the lifetime health advisory is the calculation of the drinking water equivalent level (DWEL). In this step, the RfD is converted to a concentration in drinking water by assuming a consumption of 2 L/day of drinking water by a 70-kg adult. The lifetime health advisory is finally determined by adjusting the DWEL by the relative source contribution, which is the level of exposure believed to result from drinking water compared to other sources (e.g., air). One important note on the calculation of the lifetime health advisory is that a safety factor is included for chemicals identified as possible human carcinogens (Group C). Although the lifetime health advisory is ultimately based on the RfD (i.e., a dose below which no adverse noncarcinogenic health effects should result over a lifetime of exposure), exceeding this value does not mean that a specific set of adverse health effects will occur in humans ingesting the water. The risk of adverse health effects, however, increases.

The lifetime health advisory (i.e., MCLG) is used as the basis for determining the maximum contaminant level (MCL), which is the enforceable drinking water standard. Although the MCL is set as close to the lifetime health advisory as possible, this standard also considers "analytical methodology, treatment technology and costs, economic impact, and regulatory impact" (EPA 1992). Similar to the lifetime health advisory, exceeding this standard does not mean that a specific set of adverse health effects will occur in humans consuming the water.

[Lifetime health advisories are generally not calculated for known (Group A) or probable (Group B) human carcinogens (i.e., the MCLG is set at zero). For these chemicals, available cancer data are used in conjunction with a mathematical model to predict theoretical upper-bound lifetime cancer risks. The MCLs are then set at concentrations that theoretically would result in a 10^{-4} to 10^{-6} excess cancer risk (EPA 1992).]

References

U.S. Environmental Protection Agency (EPA). 1993. Health Advisories for Drinking Water Contaminants. Office of Water Health Advisories. Lewis Publishers, Ann Arbor.

U.S. Environmental Protection Agency (EPA). 1992. Integrated Risk Information System (IRIS) Background Document 4: U.S. EPA Regulatory Action Summaries. Cincinnati, OH: Office of Research and Development. January.

Primary Drinking Water Standards and Health Advisories

Chemicals	Primary Standards		Health Advisories For 70-kg Adult			Cancer Group
	Status Reg.	MCL (mg/l)	Status HA	Noncancer Lifetime (mg/l)	mg/l at 10^{-4} Cancer Risk	
ORGANICS						
Acenaphthene	-	-	-	-	-	-
Acifluorfen	T	-	F	-	0.1	B2
Acrylamide	F	TT	F	-	0.001	B2
Acrylonitrile	T	-	D	-	0.006	B1*
Adipate (diethylhexyl)	F	0.4	-	0.4	3	C
Alachlor	F	0.002	F	-	0.04	B2
Aldicarb**	D	0.007	D	0.007	-	D
Aldicarb sulfone**	D	0.007	D	0.007	-	D
Aldicarb sulfoxide**	D	0.007	D	0.007	-	D
Aldrin	-	-	D	-	0.0002	B2
Ametryn	-	-	F	0.06	-	D
Ammonium sulfamate	-	-	F	2	-	D
Anthracene (PAH)***	-	-	-	-	-	D
Atrazine	F	0.003	F	0.003*	-	C
Baygon	-	-	F	0.003	-	C
Bentazon	T	-	F	***0.2***$^{++}$	-	D
Benz(a)anthracene (PAH)	-	-	-	-	-	B2
Benzene	F	0.005	F	-	0.1	A
Benzo(a)pyrene (PAH)	F	0.0002	-	-	0.0002^{+}	B2*
Benzo(b)fluoranthene (PAH)	-	-	-	-	-$^{+}$	B2
Benzo(g,h,i)perylene (PAH)	-	-	-	-	-	D
Benzo(k)fluoranthene (PAH)	-	-	-	-	-$^{+}$	B2
bis-2-Chloroisopropyl ether	-	-	F	0.3	-	D
Bromacil	L	-	F	0.09	-	C
Bromobenzene	L	-	D	-	-	-
Bromochloroacetonitrile	***T***	-	D	-	-	-
Bromochloromethane	-	-	F	0.01	-	-
Bromodichloromethane (THM)	P	0.1*/0.08^{+}	D	-	0.06	B2

Adapted from drinking water standards tables found at: http://www.epa.gov/OST/Tools/dwstds.html
* Under review.
**NOTE: The HA value or the MCLG/MCL value for any two or more of these three chemicals should remain at 0.007 mg/L because of similar mode of action.
***PAH = Polyaromatic hydrocarbon
$^{+}$See 40CFR Parts 141 and 142
$^{++}$Revised value based on change in RfD
NOTE: Anthracene and Benzo(g,h,i)perylene — not proposed in Phase V.
NOTE: Recent changes are noted in Italic and Bold Face print.

Primary Drinking Water Standards and Health Advisories

Chemicals	Primary Standards		Health Advisories For 70-kg Adult			Cancer Group
	Status Reg.	MCL (mg/l)	Status HA	Noncancer Lifetime (mg/l)	mg/l at 10^{-4} Cancer Risk	
Bromoform (THM)	P	0.1*/0.08[+]	D	-	0.4	B2
Bromomethane	T	-	***D***	0.01	-	D
Butyl benzyl phthalate (PAE)[+++]	**-**	**-**	-	-	-	C
Butylate	-	-	F	0.35	-	D
Butylbenzene n-	-	-	D	-	-	-
Butylbenzene sec-	-	-	D	-	-	-
Butylbenzene tert-	-	-	D	-	-	-
Carbaryl	-	-	F	0.7	-	D
Carbofuran	F	0.04	F	0.04	-	E
Carbon tetrachloride	F	0.005	F	-	0.03	B2
Carboxin	-	-	F	0.7	-	D
Chloral hydrate	P	0.06[++]	D	0.06	-	C
Chloramben	-	-	F	0.1	-	D
Chlordane	F	0.002	F	-	0.003	B2
Chlorodibromomethane (THM)	P	0.1*/0.08[+]	D	0.06	-	C
Chloroethane	L	-	D	-	-	B
Chloroform (THM)	P	0.1*/0.08[+]	D	-	0.6	B2
Chloromethane	L	-	F	0.003	-	C
Chlorophenol (2-)	-	-	D	0.04	-	D
p-Chlorophenyl methyl sulfide/sulfone/sulfoxide	-	-	**	-	-	D
Chloropicrin	L	-	-	-	-	-
Chlorothalonil	-	-	F	-	0.15	B2
Chlorotoluene o-	L	-	F	0.1	-	D
Chlorotoluene p-	L	-	F	0.1	-	D
Chlorpyrifos	-	-	F	0.02	-	D
Chrysene (PAH)	-	-	-	-	-	B2
Cyanazine[++++]	T	-	D	0.001[++++]	-	C

* Current MCL

[**]A HA will not be developed due to insufficient data; a "Database Deficiency Report has been published.

[+] 1994 Proposed rule for Disinfectants and Disinfection By-products: Total for all THMs combined cannot exceed the 0.08 level.

[++]Total for all haloacetic acids cannot exceed 0.06 level.

[+++]PAE = phthalate acid ester

[++++]Draft HA updated for the Phase VIB regulation, which has been postponed. It includes the change of the cancer classification from D to C, thus justifying the use of an additional 10-fold safety factor for the lifetime HA.

Primary Drinking Water Standards and Health Advisories

Chemicals	Primary Standards		Health Advisories For 70-kg Adult			Cancer Group
	Status Reg.	MCL (mg/l)	Status HA	Noncancer Lifetime (mg/l)	mg/l at 10^{-4} Cancer Risk	
Cyanogen chloride	***T***	-	-	-	-	-
Cymene p-	-	-	D	-	-	-
2,4-D	F	0.07	F	0.07	-	D
DCPA (Dacthal)	L	-	F	-	-	D
Dalapon	F	0.2	F	0.2	-	D
Di[2-ethylhexyl]adipate	F	0.4	-	0.4	3	C
Diazinon	-	-	F	0.0006	-	E
Dibromoacetonitrile	L	-	D	0.02	-	C
Dibromochloropropane (DBCP)	F	0.0002	F	-	0.003	B2
Dibromomethane	L	-	-	-	-	D
Dibutyl phthalate (PAE)+++	-	-	-	-	-	D
Dicamba	L	-	F	0.2	-	D
Dichloroacetaldehyde	L	-	D	-	-	-
Dichloroacetic acid	P	0.06[++]	D	-	-**	B2
Dichloroacetonitrile	L	-	D	0.006	-	C
Dichlorobenzene o-	F	0.6	F	0.6	-	D
Dichlorobenzene m- *	-	-	F	0.6	-	D
Dichlorobenzene p-	F	0.075	F	0.075	-	C
Dichlorodifluoromethane	L	-	F	1	-	D
Dichloroethane (1,2-)	F	0.005	F	-	0.04	B2
Dichloroethylene (1,1-)	F	0.007	F	0.007	-	C
Dichloroethylene (cis-1,2-)	F	0.07	F	0.07	-	D
Dichloroethylene (trans-1,2-)	F	0.1	F	0.1	-	D
Dichloromethane	F	0.005	F	-	0.5	B2
Dichlorophenol (2,4-)	-	-	D	0.02	-	D
Dichloropropane (1,1-)	-	-	D	-	-	-
Dichloropropane (1,2-)	F	0.005	F	-	0.06	B2
Dichloropropane (1,3-)	L	-	D	-	-	-

* The values for m-dichlorobenzene are based on data for o-dichlorobenzene.

** A quantitative risk estimate has not been determined.

[++] Total for all haloacetic acids cannot exceed 0.06 level.

Primary Drinking Water Standards and Health Advisories

Chemicals	Primary Standards		Health Advisories For 70-kg Adult			Cancer Group
	Status Reg.	MCL (mg/l)	Status HA	Noncancer Lifetime (mg/l)	mg/l at 10^{-4} Cancer Risk	
Dichloropropane (2,2-)	L	-	D	-	-	-
Dichloropropene (1,1-)	L	-	D	-	-	-
Dichloropropene (1,3-)	T	-	F	-	0.02	B2
Dieldrin	-	-	F	-	0.0002	B2
Diethyl phthalate (PAE)	-	-	D	5	-	D
Diethylene glycol dinitrate	-	-	*	-	-	-
Di(2-ethylhexyl)phthalate (PAE)	F	0.006	D	-	0.3	B2
Diisopropyl methylphosphonate	-	-	F	0.6	-	D
Dimethrin	-	-	F	2	-	D
Dimethyl methylphosphonate	-	-	F	0.1	0.7	C
Dimethyl phthalate (PAE)	-	-	-	-	-	D
1,3-Dinitrobenzene	-	-	F	0.001	-	D
Dinitrotoluene (2,4-)	L	-	F	-	0.005	B2
Dinitrotoluene (2,6-)	L	-	F	-	0.005	B2
tg 2,6 & 2,4 dinitrotoluene **	-	-	-	-	0.005	B2
Dinoseb	F	0.007	F	0.007	-	D
Dioxane p-	-	-	F	-	0.7	B2
Diphenamid	-	-	F	0.2	-	D
Diphenylamine	-	-	F	0.2	-	D
Diquat	F	0.02	-	0.02	-	D
Disulfoton	-	-	F	0.0003	-	E
Dithiane (1,4-)	-	-	F	0.08	-	D
Diuron	-	-	F	0.01	-	D
Endothall	F	0.1	F	0.1	-	D
Endrin	F	0.002	F	0.002	-	D
Epichlorohydrin	F	TT	F	-	0.4	B2
Ethylbenzene	F	0.7	F	0.7	-	D
Ethylene dibromide (EDB)	F	0.00005	F	-	0.00004	B2
Ethylene glycol	-	-	F	7	-	D
ETU	L	-	F	-	0.03	B2
Fenamiphos	-	-	F	0.002	-	D

* An HA will not be developed due to insufficient data; a "Database Deficiency Report" has been published.
** tg = technical grade

Primary Drinking Water Standards and Health Advisories

Chemicals	Primary Standards		Health Advisories For 70-kg Adult			Cancer Group
	Status Reg.	MCL (mg/l)	Status HA	Noncancer Lifetime (mg/l)	mg/l at 10^{-4} Cancer Risk	
Fluometron	-	-	F	0.09		D
Fluorene (PAH)	-	-	-	-	-	D
Fluorotrichloromethane	L	-	F	2	-	D
Fog Oil	-	-	D	-	-	-
Fonofos	-	-	F	0.01	-	D
Formaldehyde	D	-	D	1	-	B1**
Gasoline, unleaded (benzene)	-	-	D	0.005	-	-
Glyphosate	F	0.7	F	0.7	-	E
Heptachlor	F	0.0004	F	-	0.0008	B2
Heptachlor epoxide	F	0.0002	F	-	0.0004	B2
Hexachlorobenzene	F	0.001	F	-	0.002	B2
Hexachlorobutadiene	T	-	F	0.001	-	C
Hexachlorocyclopentadiene	F	0.05	-	-	-	D
Hexachloroethane	L	-	F	0.001	-	C
Hexane (n-)	-	-	F	-	-	D
Hexazinone	-	-	F	0.2*	-	D
HMX	-	-	F	0.4	-	D
Indeno(1,2,3,-c,d)pyrene (PAH)	-	-	D	-	-***	B2
Isophorone	L	-	F	0.1	4	C
Isopropyl methylphosphonate	-	-	D	0.7	-	D
Isopropylbenzene	-	-	D	-	-	-
Lindane	F	0.0002	F	0.0002	-	C
Malathion	-	-	F	0.2	-	D
Maleic hydrazide	-	-	F	4	-	D
MCPA	-	-	F	0.01	-	E
Methomyl	L	-	F	0.2	-	D
Methoxychlor	F	0.04	F	0.04	-	D
Methyl ethyl ketone*	-	-	F	-	-	D
Methyl parathion	-	-	F	0.002	-	D

* Under review.

** Carcinogenicity based on inhalation exposure.

***See 40CFR Parts 141 and 142

Primary Drinking Water Standards and Health Advisories

Chemicals	Primary Standards		Health Advisories For 70-kg Adult			Cancer Group
	Status Reg.	MCL (mg/l)	Status HA	Noncancer Lifetime (mg/l)	mg/l at 10^{-4} Cancer Risk	
Methyl tert butyl ether	L	-	D	0.02-0.2[+]		C***
Metolachlor	L	-	F	0.07	-	C
Metribuzin	L	-	F	0.1	-	D
Monochloroacetic acid	L	-	D	-	-	-
Monochlorobenzene	F	0.1	F	0.1	-	D
Naphthalene	-	-	F	0.02	-	D
Nitrocellulose (non-toxic)	-	-	F	-	-	-
Nitroguanidine	-	-	F	0.7	-	D
Nitrophenol p-	-	-	F	0.06	-	D
Oxamyl (Vydate)	F	0.2	F	0.2	-	E
Paraquat	-	-	F	0.03	-	E
Pentachloroethane	-	-	D	-	-	-
Pentachlorophenol	F	0.001	F	-	0.03	B2
Phenanthrene (PAH)	-	-	-	-	-	-
Phenol	-	-	D	4	-	D
Picloram	F	0.5	F	0.5	-	D
Polychlorinated biphenyls (PCBs)	F	0.0005	P	-	0.0005	B2
Prometon	L	-	F	0.1*	-	D
Pronamide	-	-	F	0.05	-	C
Propachlor	-	-	F	0.09	-	D
Propazine	-	-	F	0.01	-	C
Propham	-	-	F	0.1	-	D
Propylbenzene n-	-	-	D	-	-	-
Pyrene (PAH)	-	-	-	-	-	D
RDX	-	-	F	0.002	0.03	C
Simazine	F	0.004	F	0.004	-	C
Styrene	F	0.1	F	0.1	-	C
2,4,5-T	L	-	F	0.07	-	D
2,3,7,8-TCDD (Dioxin)	F	3E-08	F	-	2E-08	B2

* Under review. NOTE: Phenanthrene — not proposed.

*** Tentative.

[+] If the cancer classification C is accepted, the Lifetime HA is ***0.02***; otherwise it is 0.200 mg/L

Primary Drinking Water Standards and Health Advisories

Chemicals	Primary Standards		Health Advisories For 70-kg Adult			Cancer Group
	Status Reg.	MCL (mg/l)	Status HA	Noncancer Lifetime (mg/l)	mg/l at 10^{-4} Cancer Risk	
Tebuthiuron	-	-	F	0.5	-	D
Terbacil	-	-	F	0.09	-	E
Terbufos	-	-	F	0.0009	-	D
Tetrachloroethane (1,1,1,2-)	L	-	F	0.07	0.1	C
Tetrachloroethane (1,1,2,2-)	L	-	D	-	-	-
Tetrachloroethylene	F	0.005	F	-	0.07	-
Tetranitromethane	-	-	**	-	-	-
Toluene	F	1	F	1	-	D
Toxaphene	F	0.003	F	-	0.003	B2
2,4,5-TP	F	0.05	F	0.05	-	D
1,1,2-Trichloro-1,2,2-trifluoroethane	-	-	-	-	-	-
Trichloroacetic acid	P	0.06[++]	D	0.3	-	C
Trichloroacetonitrile	L	-	D	-	-	-
Trichlorobenzene (1,2,4-)	F	0.07	F	0.07	-	D
Trichlorobenzene (1,3,5-)	-	-	F	0.04	-	D
Trichloroethane (1,1,1-)	F	0.2	F	0.2	-	D
Trichloroethane (1,1,2-)	F	0.005	F	0.003	-	C
Trichloroethanol (2,2,2-)	L	-	-	-	-	-
Trichloroethylene	F	0.005	F	-	0.3	B2
Trichlorophenol (2,4,6-)	L	-	D	-	0.3	B2
Trichloropropane (1,1,1-)	-	-	D	-	-	-
Trichloropropane (1,2,3-)	L	-	F	0.04	0.5	B2
Trifluralin	L	-	F	0.005	0.5	C
Trimethylbenzene (1,2,4-)	-	-	D	-	-	-
Trimethylbenzene (1,3,5-)	-	-	D	-	-	-
Trinitroglycerol	-	-	F	0.005	-	-
Trinitrotoluene	-	-	F	0.002	0.1	C
Vinyl chloride	F	0.002	F	-	0.0015	A
Xylenes	F	10	F	10	-	D

** A HA will not be developed due to insufficient data; a "Database Deficiency Report" has been published.

[++] Total for all haloacetic acids cannot exceed 0.06 mg/l level.

Primary Drinking Water Standards and Health Advisories

Chemicals	Primary Standards		Health Advisories For 70-kg Adult			Cancer Group
	Status Reg.	MCL (mg/l)	Status HA	Noncancer Lifetime (mg/l)	mg/l at 10^{-4} Cancer Risk	
INORGANICS						
Aluminum	L	-	D	-	-	-
Ammonia	-	-	D	30	-	D
Antimony	F	0.006	F	0.003	-	D
Arsenic	*	0.05	D	-	0.002	A
Asbestos (fibers/l >10µm length)	F	7 MFL	-	-	700 MFL	A
Barium	F	2	F	2	-	D
Beryllium	F	0.004	D	-	0.0008	B2
Boron	L	-	D	0.6	*	D
Bromate	L	0.01	-	-	-	-
Cadmium	F	0.005	F	0.005	-	D
Chloramine	P	4	D	3/4***	-	-
Chlorate	L	-	D	-	-	-
Chlorine	P	4	D	-	-	D
Chlorine dioxide	T	0.8	D	0.3	-	D
Chlorite	L	1	D	0.08	-	D
Chromium (total)	F	0.1	F	0.1	-	D
Copper (at tap)	F	TT**	-	-	-	D
Cyanide	F	0.2	F	0.2	-	D
Fluoride*	F	4	-	-	-	-
Hypochlorite	P	-	-	-	-	-
Hypochlorous acid	P	-	-	-	-	-
Lead (at tap)	F	TT**	-	-	-	B2
Manganese	L	-	-	-	-	-
Mercury (inorganic)	F	0.002	F	0.002	-	D
Molybdenum	L	-	D	0.04	-	D
Nickel	F	0.1[1]	F	0.1	-	D
Nitrate (as N)	F	10	F	-	-	*

* Under review.
** Copper — action level 1.3 mg/L, Lead — action level 0.015 mg/L
*** Measured as free chlorine.
[1] Being remanded

Primary Drinking Water Standards and Health Advisories

Chemicals	Primary Standards		Health Advisories For 70-kg Adult			Cancer Group
	Status Reg.	MCL (mg/l)	Status HA	Noncancer Lifetime (mg/l)	mg/l at 10^{-4} Cancer Risk	
Nitrite (as N)	F	1	F	-	-	*
Nitrate + Nitrite (both as N)	F	10	F	-	-	*
Selenium	F	0.05	-	-	-	-
Silver	-	-	D	0.1	-	D
Sodium	-	-	D	-	-	-
Strontium	L	-	D	17	-	D
Sulfate	P	500	D	-	-	-
Thallium	F	0.002	F	0.0005	-	-
Vanadium	***T***	-	D	-	-	D
White phosphorous	-	-	F	0.0001		D
Zinc	L	-	***D***	2	-	D
Zinc chloride (measured as Zinc)	L	-	F	2	-	D
MICROBIOLOGY						
Cryptosporidium	L	-[1]				
Giardia lamblia	F	TT[1]				
Legionella	F[+++]	TT[1]				
Standard Plate Count	F[+++]	TT[1]				
Total Coliforms	F	**[1]				
Turbidity	F	PS[1]				
Viruses	F[+++]	TT[1]				
RADIONUCLIDES						
Beta particle and photon activity (formerly man-made radionuclides)	***F***	4 mrem	-	-	4 mrem/y	A
Gross alpha particle activity	***F***	15 pCi/L	-	-	15 pCi/L	A
Combined Radium 226 & 228	***F***	5 pCi/L	-	-	20 pCi/L	A
Radon[+]	P	300 pCi/L	-	-	150 pCi/L	A
Uranium[+]	P	20 µg/L	-	-	*	A

* Under review. ** Guidance.

+ 1991 Proposed National Primary Drinking Water Rule for Radionuclides

++ No final MCLG, but zero proposed in 1991.

PS, TT, F, defined in attached Legend.

[+++] Final for systems using surface water; also being considered for regulation under groundwater disinfection rule.

[1] Microbiology Standards are defined in atttached legend.

Secondary Maximum Contaminant Levels

Chemicals	Status	SMCLs (mg/L)
Aluminum	F	0.05 to 0.2
Chloride	F	250
Color	F	15 color units
Copper	F	1.0
Corrosivity	F	non-corrosive
Fluoride*	F	2.0
Foaming agents	F	0.5
Iron	F	0.3
Manganese	F	0.05
Odor	F	3 threshold odor numbers
pH	F	6.5 — 8.5
Silver	F	0.1
Sulfate	F	250
Total dissolved solids (TDS)	F	500
Zinc	F	5

Adapted from drinking water standards tables found at:
http://www.epa.gov/OST/Tools/dwstds.html Status Codes: P — proposed, F — final,
* Under review.

Secondary Drinking Water Standards are unenforceable federal guidelines regarding taste, odor, color and certain other non-aesthetic effects of drinking water. EPA recommends them to the States as reasonable goals, but federal law does not require water systems to comply with them. States may, however, adopt their own enforceable regulations governing these concerns. To be safe, check your State's drinking water rules.

Legend

Abbreviations column descriptions are:

MCLG: Maximum Contaminant Level Goal. A non-enforceable concentration of a drinking water contaminant that is protective of adverse human health effects and allows an adequate margin of safety.

MCL: Maximum Contaminant Level. Maximum permissible level of a contaminant in water which is delivered to any user of a public water system.

RfD: Reference Dose. An estimate of a daily exposure to the human population that is likely to be without appreciable risk of deleterious effects over a lifetime.

DWEL: Drinking Water Equivalent Level. A lifetime exposure concentration protective of adverse, non-cancer health effects, that assumes all of the exposure to a contaminant is from a drinking water source.

The codes for the Status Reg and Status HA columns are as follows:

F final

D draft

L listed for regulation

P proposed

T tentative (not officially proposed)

Other codes found in the table include the following:

NA not applicable

PS performance standard 0.5 NTU-1.0 NTU

TT treatment technique

Large discrepancies between Lifetime and Longer-term HA values may occur because of the Agency's conservative policies, especially with regard to carcinogenicity, relative source contribution, and less-than-lifetime exposures in chronic toxicity testing. These factors can result in a cumulative UF (uncertainty factor) of up to 5 to 5000 when calculating a Lifetime HA.

The scheme for categorizing chemicals according to their carcinogenic potential is as follows:*

Group A: **Human carcinogen**	Sufficient evidence in epidemiologic studies to support causal association between exposure and cancer
Group B: **Probable human carcinogen**	Limited evidence in epidemiologic studies (Group B1) and/or sufficient evidence from animal studies (Group B2)

Group C: Possible human carcinogen	Limited evidence from animal studies and inadequate or no data in humans
Group D: Not classifiable	Inadequate or no human and animal evidence of carcinogenicity
Group E: No evidence of carcinogenicity for humans	No evidence of carcinogenicity in at least two adequate animal tests in different species *or* in adequate epidemiologic and animal studies

Drinking Water Health Advisories (HAs) are defined as follows:

Non Cancer Lifetime HA:	The concentration of a chemical in drinking water that is not expected to cause any adverse noncarcinogenic effects over a lifetime of exposure, with a margin of safety.
10^{-4} Cancer Risk:	The concentration of a Group A or B (and in a few cases, Group C) carcinogen that, if consumed over a lifetime, would result in a 1 in 10,000 chance of getting cancer.

*EPA is in the process of revising the Cancer Guidelines.

Microbiology Primary Standards are defined as follows:

Cryptosporidium	A protozoan parasite that can cause a gastroenteric disease called Cryptosporidiosis.
Giardia lamblia	A bacteria that can cause a gastroenteric disease called Giardiasis. 99.99% of all bacterial killed/inactivated.
Legionella	A bacteria that can cause Legionnaire's Disease, commonly known as pneumonia. Found naturally in water; multiplies in heating systems. No limit, but if Giardia and viruses are inactivated, Legionella will most likely also be controlled.
Standard plate count	Indicate how effective treatment is at controlling microorganisms. No more that 500 bacterial colonies per milliliter
Total Coliforms	Used as an indicator that other potentially harmful bacteria may be present. No more than 5.0% samples of total coliform-positive in a month. For water systems that collect fewer that 40 routine samples per month, no more that one sample can be total coliform-positive. Every sample that has total coliforms must be analyzed for fecal coliforms. There cannot be any fecal coliforms.

Turbidity	Turbidity has no health effects but can interfere with disinfection and provide a medium for microbial growth. It may indicate the presence of microbes. At no time can turbidity (cloudiness of water) go above 5 nephelometric turbidity units (NTU); systems that filter must ensure that turbidity go no higher than 1 NTU (0.5 NTU for conventional or direct filtration) in at least 95% of the daily samples for any two consecutive months.
Viruses (enteric)	Viruses that can cause gastroenteric diseases. Found in human and animal fecal waste. 99.99% of all viruses killed/inactivated.

The following paragraphs describe the critical or adverse effects noted in the study(ies) that served as the basis for derivation of the drinking water standard(s) and/or guidelines for the contaminant(s) of concern. Adverse health effects that have been observed in humans following ingestion of the contaminant(s) are also noted.

Primary MCLs/HALs -- Inorganics

Aluminum

Summary

EPA is in the process of drafting health advisory levels for aluminum (EPA 1996). Low-level exposure to aluminum from food, air, water, or contact with skin is not thought to harm human health (ATSDR 1999). However, humans exposed to high levels of aluminum dust in air may have respiratory problems including coughing and asthma. Aluminum has been linked to Alzheimer's disease because patients suffering from the disease have high levels of aluminum in their brains (ATSDR 1999). However, scientists do not know whether aluminum causes the disease or whether the buildup of aluminum happens to people who already have the disease. Infants and adults who received large doses of aluminum as a medical treatment developed bone diseases, suggesting that aluminum may cause skeletal problems (ATSDR 1999).

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicity Frequently Asked Questions (ToxFAQs) Fact Sheets. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Division of Toxicology. http://www.atsdr.cdc.gov/tfacts.html. April.

U.S. Environmental Protection Agency (EPA). 1996. Drinking Water Regulations and Health Advisories. Office of Water. EPA 822-B-96-002.

Ammonia

Summary

EPA is in the process of drafting health advisory levels for ammonia (USEPA, 1996). No data were located indicating adverse health effects from exposure to ammonia in water, and there is no RfD for ammonia (USEPA, 1999a). The RfC for ammonia was developed based on information that showed no effect based on a pulmonary function test but respiratory symptoms in workers who inhaled ammonia (USEPA, 1999a).

References

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

U.S. Environmental Protection Agency (EPA). 1996. Drinking Water Regulations and Health Advisories. Office of Water. EPA 822-B-96-002.

Antimony

Summary

The RfD for antimony is based on a study in which rats were administered antimony in the drinking water from weaning until death (EPA 1993). The most toxic of the common antimony compounds (i.e., potassium antimony tartrate) was used in this study. The critical effects consisted of decreased longevity and altered levels of blood glucose and serum cholesterol in both male and female rats.

Gastrointestinal effects have been noted in humans accidentally ingesting antimony. Specifically, persons who ingested lemonade contaminated with antimony at a concentration of 130 mg/L (i.e., orders of magnitude above the MCL) exhibited stomach pains, colic, nausea, and vomiting (EPA 1999). Reports of accidental poisonings have also noted gastrointestinal effects, such as vomiting and diarrhea (EPA 1993).

Finally, myocardial effects have been observed in humans receiving antimony as a treatment for parasites. One study conducted on patients receiving oral doses of antimony as sodium stibogluconate to treat leishmaniasis revealed abnormalities in the electrocardiograms that increased with dose and duration of treatment (EPA 1993). The doses administered to these patients, however, were at least an order of magnitude greater than the dose used to derive the RfD.

References

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

U.S. Environmental Protection Agency (EPA). 1993. Health Advisories for Drinking Water Contaminants. Office of Water Health Advisories. Lewis Publishers, Ann Arbor.

Arsenic

Summary

Arsenic is classified as a human carcinogen (EPA 1999). Occupational epidemiology studies indicate that inhalation exposure is associated with an increased risk of lung cancer. Epidemiology studies of the general population indicate that ingestion exposure is associated with an increased prevalence of skin cancer and possibly increased risk of bladder, kidney, lung, and liver cancer. Noncarcinogenic effects resulting from exposure to high levels of arsenic in the

drinking water include skin damage (i.e., hyperpigmentation and keratosis) and possible circulatory system problems (i.e., blackfoot disease).

Reference

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

Barium

Summary

A weight-of-evidence approach was used to derive the currently accepted RfD for barium (EPA 1999). Hypertension has been observed in humans who ingested high doses of barium compounds and in workers who inhaled dust containing barium. Studies in which animals have been administered barium intravenously or in drinking water have also supported the observation that hypertension may result from exposure to barium. The dose level used to calculate the current RfD was taken from a study in which human volunteers drank water containing barium. The dose administered did not cause hypertension in the volunteers. Therefore, this dose level was taken as the no adverse effect level and was used to derive the current RfD. Although animal studies have indicated that exposure to barium may also affect the kidney, the current RfD was derived using a dose level that would protect against this type of effect.

Reference

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

Beryllium

Summary

Beryllium was reclassified in 1998 as a probable human carcinogen (EPA 1999). Limited human data from occupational studies suggest an association between inhalation exposure and an increased risk of lung cancer. Animal studies, however, provide strong support for this association. Injection studies with animals have also suggested a relationship between beryllium exposure and bone tumors (i.e., osteosarcomas). Regarding ingestion of beryllium, no adequate assessments have been conducted to determine the human carcinogenic potential for this route of exposure. Noncarcinogenic health effects observed in experimental animals ingesting beryllium include gastrointestinal tract lesions.

Reference

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

Boron

Summary

The RfD for boron is based on two studies in which dogs were administered boron in the diet for either 38 weeks or 2 years (EPA 1993). At the highest dose tested, severe testicular atrophy and spermatogenic arrest occurred. Other animals studies have shown similar effects at higher dose levels than those used in the dog studies and appear to indicate that the dog is the most sensitive species for this effect. Human exposure data are limited and are primarily restricted to accidental poisonings or ingestion in children.

Reference

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

Cadmium

Summary

The current RfDs for cadmium (i.e., one for ingestion of water and the other for the ingestion of food) are based on a large database of human and animal toxicity studies (EPA 1999). A toxicokinetic model has been developed and was used to determine the doses required to yield the cadmium concentration in kidney tissue that has not been associated with kidney damage (i.e., significant

proteinuria, the critical effect). Studies in which animals have been orally administered cadmium compounds have not provided any evidence of carcinogenicity; however, occupational studies and animal studies in which cadmium compounds were administered by intramuscular or subcutaneous injection or by inhalation exposure have provided sufficient evidence to classify cadmium as a probable human carcinogen. The current MCL is based on the noncarcinogenic effect (i.e., kidney damage).

Reference

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

Chromium (total)

Summary

EPA has determined that acute dermal exposures to chromium in drinking water at levels above the MCL may potentially cause skin irritation or ulceration effects (EPA 1995). Dermal exposure to chromium has been observed to produce allergic contact dermatitis, which is characterized by symptoms of redness, swelling, papules, small vesicles, dryness, scaling, and fissuring (EPA 1999). One study suggests that humans ingesting hexavalent chromium at levels of 20 mg/L in drinking water (200 times the MCL) may experience gastrointestinal effects such as diarrhea, stomach ache, indigestion, and vomiting (EPA 1999). Long-term exposures to chromium at levels above the MCL have the potential to cause dermatitis and damage to the liver, kidney, circulatory, and nerve tissues (EPA 1995). According to EPA (1995 and 1999), there is no evidence that chromium in drinking water has the potential to cause cancer from lifetime exposures in drinking water.

References

U.S. Environmental Protection Agency (EPA). 1995. National Primary Drinking Water Regulations Contaminant Specific Fact Sheets Inorganic Chemicals - Technical Version. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. EPA 811-95-002-T. http://www.epa.gov/OGWDW/dwh/t-ioc.html. October.

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

Copper

Summary

In humans, trace dietary levels of copper are essential, but high doses of copper can lead to liver or kidney damage, anemia, or gastrointestinal distress (EPA 1998). The MCL for copper is dictated by treatment techniques, including optimization of corrosion control, with an action level of 1.3 mg/L as determined from tap water samples.

References

United States Environmental Protection Agency (EPA). 1998. National primary drinking water regulations. 40 CFR §141.32.

Cyanide

Summary

The RfD for free cyanide is based on a study in which rats exhibited no toxic effects following exposure for two years in their diet (EPA 1999a). In another study with rats, subchronic or chronic oral exposure resulted in weight loss, decreased thyroxin levels (a thyroid hormone), and degeneration of the myelin coating on their nerves.

EPA has found cyanide compounds to potentially cause rapid breathing, tremors, and other neurological effects from acute exposures at levels above the MCL (EPA 1999b). Cyanide compounds have the potential to cause weight loss, thyroid effects, and nerve damage from long-term exposures at levels above the MCL.

EPA had determined that there is not enough data to classify free cyanide as to its human carcinogenicity (EPA 1999a). Therefore, there is inadequate evidence to state whether cyanide compounds have the potential to cause cancer from lifetime exposures in drinking water (EPA 1999b).

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. April.

Fluoride

Summary

The RfD for fluoride is based on a study in which children consuming fluoride in their drinking water exhibited moderate to severe dental fluorosis (EPA 1999). Dental fluorosis results from excessive exposure to fluoride during the age of calcification of the teeth (up to about 8 years of age for the anterior teeth). In its mild form, dental fluorosis is characterized by white opaque areas covering 50% of the tooth. Brown to black stains and pitting can occur in more severe cases. EPA considers moderate to severe dental fluorosis to be a cosmetic effect rather than an adverse health effect. Nevertheless, the RfD is protective for crippling skeletal fluorosis, which can occur after long-term exposure to higher levels of fluoride.

Reference

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

Hypochlorite

Summary

In pure water, chlorine can form hypochlorous acid, which dissociates to hypochlorite as pH increases (EPA 1999). Therefore, EPA has proposed to regulate hypochlorite in drinking water as chlorine (EPA 1996). The RfD for chlorine is based on a study in which rats no observable adverse effects were seen in rats exposed to chlorine in drinking water for two years (EPA 1999). In another study, rats exposed to sodium hypochlorite for two years in drinking water died. Animal studies have demonstrated no evidence of reproductive or developmental effects.

References

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

U.S. Environmental Protection Agency (EPA). 1996. Drinking Water Regulations and Health Advisories. Office of Water. EPA 822-B-96-002.

Lead

Summary

Lead has been shown to cause developmental delays (physical and/or mental) in children, and also kidney problems and slight increases in blood pressure in adults (EPA 1998). Based on the potential health effects, the MCLG for lead has been established at 0. The standard for lead is a treatment technique, including optimization of corrosion control, with an action level (as determined from tap water samples) of 0.015 mg/L.

Reference

United States Environmental Protection Agency (EPA). 1998. National primary drinking water regulations. 40 CFR §141.32.

Mercury (inorganic)

Summary

Short-term and long-term exposures to inorganic mercury in drinking water at levels above the MCL may potentially cause kidney damage (EPA 1995 and 1999). The most sensitive adverse effect found for mercuric chloride (HgCl2) has been identified as the formation of mercuric-mercury-induced autoimmune glomerulonephritis, a disease of the kidney (EPA 1999). Exposure to high levels of inorganic mercury salts can also cause nervous system effects, nausea, and diarrhea (ATSDR 1999). There is inadequate evidence to state whether or not inorganic mercury or elemental mercury has the potential to cause cancer from lifetime exposures in drinking water (EPA 1995 and 1999); however, EPA has classified mercuric chloride as a possible human carcinogen.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicity Frequently Asked Questions (ToxFAQs) Fact Sheets. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Division of Toxicology. http://www.atsdr.cdc.gov/tfacts.html. March.

U.S. Environmental Protection Agency (EPA). 1995. National Primary Drinking Water Regulations Contaminant Specific Fact Sheets Inorganic Chemicals - Technical Version. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. EPA 811-95-002-T. http://www.epa.gov/OGWDW/dwh/t-ioc.html. October.

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

Molybdenum

Summary

The RfD is based on a 6-year to lifetime human dietary exposure study in which the critical effect was identified to be increased uric acid levels in the blood (EPA 1999). This effect was associated with other symptoms of gout, which include pain, swelling, inflammation, and deformities of the joints. High levels of ingested molybdenum may also be associated with potential mineral imbalance, observed as increased serum ceruloplasmin and urinary excretion of copper (EPA 1999). EPA has not yet evaluated the carcinogenicity of molybdenum.

References

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

Nickel

Summary

The RfD for nickel is based on a study in which rats exhibited decreased body and organ weights after receiving nickel in the diet for two years (EPA 1999a). Other studies in rats and dogs noted similar effects (EPA 1999a). Several studies also have demonstrated increased neonatal mortality in offspring of rats exposed to nickel in drinking water (EPA 1999a).

Nickel has the potential to cause decreased body weight, heart and liver damage, and dermatitis from long-term exposure at levels above the MCL (EPA 1999b). The most common health effect of nickel in humans is an allergic reaction characterized by a skin rash and, less commonly, asthma (ATSDR 1999). Initial sensitization to nickel is believed to result from dermal contact; however, people who are sensitive to nickel can react when they drink it in water (ATSDR 1999, EPA 1999a).

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicity Frequently Asked Questions (ToxFAQs) Fact Sheets. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Division of Toxicology. http://www.atsdr.cdc.gov/tfacts.html. March

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. March.

Nitrate

Summary

Acute nitrate toxicity is due primarily to its conversion to nitrite, which reacts with hemoglobin in the blood and makes a compound called methemoglobin (EPA 1999a, 1999b). Methemoglobin does not bind oxygen, and as more and more hemoglobin is converted to methemoglobin, the amount of oxygen transported from the lungs to the body tissues is reduced (EPA 1999b). This condition is known as methemoglobinemia, and symptoms include shortness of breath and blueness of the skin (EPA 1999a, 1999b). Conversion of nitrate to nitrite in the gastrointestinal system is largely done by bacteria (EPA 1999a). Infants are more susceptible to nitrate-induced methemoglobinemia because their gastrointestinal systems normally have a low acidity (high pH), which favors the bacteria that convert nitrate to nitrite (EPA 1999a).

The RfD for nitrate is based on two separate epidemiological evaluations of infants who had methemoglobinemia (EPA 1999a). These studies provided evidence that methemoglobinemia could occur in infants exposed to nitrate in drinking water.

Effects of chronic exposure to high levels of nitrate include diuresis and increased starchy deposits and hemorrhaging of the spleen (EPA 1999b).

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. March.

Nitrite

Summary

Nitrite, which is a conversion product of nitrate, reacts with hemoglobin in the blood and makes a compound called methemoglobin (EPA 1999a, 1999b). Methemoglobin does not bind oxygen, and as more and more hemoglobin is converted to methemoglobin, the amount of oxygen transported from the lungs to the body tissues is reduced (EPA 1999b). This condition is known as methemoglobinemia,

and symptoms include shortness of breath and blueness of the skin (EPA 1999a, 1999b). Conversion of nitrate to nitrite in the gastrointestinal system is largely done by bacteria (EPA 1999a). Infants are more susceptible to nitrate-induced methemoglobinemia because their gastrointestinal systems normally have a low acidity (high pH), which favors the bacteria that convert nitrate to nitrite (EPA 1999a).

The RfD for nitrite is based on an epidemiological study in which methemoglobinemia in infants was associated with nitrate-contaminated water (EPA 1999a). Several more recent studies also support these findings.

Effects of chronic exposure to high levels of nitrite include diuresis and increased starchy deposits and hemorrhaging of the spleen (EPA 1999b).

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. March.

Nitrate/Nitrite

Summary

Nitrite, which is a conversion product of nitrate, reacts with hemoglobin in the blood and makes a compound called methemoglobin (EPA 1999a, 1999b). Methemoglobin does not bind oxygen, and as more and more hemoglobin is converted to methemoglobin, the amount of oxygen transported from the lungs to the body tissues is reduced (EPA 1999b). This condition is known as methemoglobinemia, and symptoms include shortness of breath and blueness of the skin (EPA 1999a, 1999b). Conversion of nitrate to nitrite in the gastrointestinal system is largely done by bacteria (EPA 1999a). Infants are more susceptible to nitrate-induced methemoglobinemia because their gastrointestinal systems normally have a low acidity (high pH), which favors the bacteria that convert nitrate to nitrite (EPA 1999a).

The RfD for nitrate is based on two separate epidemiological evaluations of infants who had methemoglobinemia (EPA 1999a). These studies provided evidence that methemoglobinemia could occur in infants exposed to nitrate in drinking water. The RfD for nitrite is based on an epidemiological study in which methemoglobinemia in infants was associated with nitrate-contaminated water (EPA 1999a). Several more recent studies also support these findings.

Effects of chronic exposure to high levels of nitrate/nitrite include diuresis and increased starchy deposits and hemorrhaging of the spleen (EPA 1999b).

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. March.

Radium (226 & 228)

Summary

The MCL for radium (226 & 228) is based on carcinogenicity (EPA 1996); however, EPA has withdrawn its formal carcinogenicity assessment of radium (226 & 228) (EPA 1999b). Studies indicate that long-term oral exposure to radium causes lung, bone, brain, and nasal passage tumors in humans (EPA 1999a).

References

U.S. Environmental Protection Agency (EPA). 1999a. Health Effects Notebook for Hazardous Air Pollutants. Office of Air Quality and Planning Standards. http://www.epa.gov/ttn/uatw/hapindex.html. April.

U.S. Environmental Protection Agency (EPA). 1999b. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/subst/0295.html#II. April.

U.S. Environmental Protection Agency (EPA). 1996. Drinking Water Regulations and Health Advisories. Office of Water. EPA 822-B-96-002.

Selenium

Summary

The RfD for selenium is based on an epidemiological study of people living in an area of China with unusually high environmental concentrations of selenium (EPA 1999a). Symptoms observed in some of these people that were attributed to selenium exposure included thickened and brittle nails, hair and nail loss, lowered hemoglobin levels, mottled teeth, skin lesions, and numbness and pain in the extremities.

EPA has found selenium to potentially cause the following health effects from acute exposures at levels above the MCL: hair and fingernail changes, damage to the peripheral nervous system, and fatigue and irritability (EPA 1999b). Selenium has the potential to cause the following health effects from long-term exposure at levels above the MCL: hair and fingernail loss and damage to kidney and liver tissue and the nervous and circulatory systems (EPA 1999b).

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. March.

Silver

Summary

Silver compounds have been used for centuries as therapeutic agents for variety of illnesses (EPA 1999). Patients being treated with these compounds have exhibited argyria, which is a bluish-gray discoloration of the skin on exposure to light. No other adverse health effects have been associated with exposure to these silver compounds or deposition of silver in the skin. Accordingly, the RfD for silver is based on this non-adverse health effect. The dose used to derive the RfD was the minimal effect level in a study in which humans were intravenously administered silver arsphenamine.

Reference

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

Strontium

Summary

The RfD for strontium is based on a study in which young and adult rats were given strontium in the diet for 20 days (EPA 1999). Effects on bone growth and mineralization occurred in young and adult rats; however, young rats were affected more severely at lower dietary strontium levels than were adult rats. Other studies noted similar effects (EPA 1999).

In humans, excessive strontium intakes can alter bone mineralization, such as inhibiting the incorporation of calcium or replacing calcium, and cause bone deformities (EPA 1999). The adequacy of calcium nutrition is a critical factor regarding strontium toxicity; strontium effects on bones can be exacerbated by inadequate calcium levels. Young animals are more sensitive than adult animals to excessive strontium intakes because their bones are actively growing.

Reference

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

Sulfate

Summary

EPA is in the process of drafting health advisory levels for sulfate (EPA 1996). Health concerns regarding sulfate in drinking water have been raised due to reports that diarrhea may be associated with ingestion of water containing high levels of sulfate (EPA 1999). People may be at greater risk when they abruptly change from drinking water with low sulfate concentrations to drinking water with high sulfate concentrations. The 1996 Safe Drinking Water Act Amendments directed EPA and the Centers for Disease Control and Prevention to jointly conduct a study to establish a reliable dose-response relationship for the adverse human health effects from exposure to sulfate in drinking water. The sulfate study was completed in January 1999 and is currently undergoing peer review.

References

U.S. Environmental Protection Agency (EPA). 1999. Sulfate in Drinking Water. Office of Groung Water and Drinking Water. http://www.epa.gov/OGWDW/sulfate.html. April.

U.S. Environmental Protection Agency (EPA). 1996. Drinking Water Regulations and Health Advisories. Office of Water. EPA 822-B-96-002.

Thallium

Summary

The RfDs for thallium compounds are based on a study in which rats were administered thallium sulfate by feeding tube for 90 days (EPA 1999). Potential treatment-related effects included an increased incidence of alopecia (i.e., hair loss) and changes in several blood chemistry parameters (i.e., increased levels of certain serum enzymes, increased levels of sodium, and decreased blood sugar levels). The highest dose was used as the no adverse effect level because the observed changes were not supported by gross or microscopic alterations in organs or tissues.

Other oral exposure studies conducted at higher doses have indicated similar effects (e.g., alopecia), but also have indicated other potential health effects (i.e., damage to testicular tissue, reduced sperm motility, damage to peripheral nerves) (EPA 1999). One animal study in which rats were administered thallium acetate by subcutaneous injection indicated that thallium exposure may cause damage to the kidneys, liver, and brain at much higher doses than the one used to derive the RfD (EPA 1993).

The health effects observed in humans following thallium ingestion are restricted to intentional or accidental poisoning at doses at least an order of magnitude above the one used to derive the RfD (EPA 1993). In these cases, a variety of symptoms were observed including alopecia, peripheral neuropathy, and gastrointestinal effects. These symptoms have also been observed in women using a depilatory ointment containing thallous acetate.

References

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

U.S. Environmental Protection Agency (EPA). 1993. Health Advisories for Drinking Water Contaminants. Office of Water Health Advisories. Lewis Publishers, Ann Arbor.

Vanadium

Summary

The RfD for vanadium pentoxide is based on a study in which chronic exposure (no duration specified) in the diet of rats resulted in a decrease in the amount of cystine in their hair (EPA 1999). In a separate study, rats fed vanadium pentoxide in their diet for 68 days exhibited decreased cystine in their hair as well as decreased levels of hemoglobin and red cells in their blood. There is no information about health effects in humans who ingested vanadium. Lung irritation, coughing, wheezing, chest pain, runny nose, and sore throat have been noted in workers who inhaled vanadium (ATSDR 1999). These effects ceased soon after the workers stopped breathing the contaminated air.

EPA has not evaluated or classified vanadium as to its human carcinogenicity (EPA 1999). No studies are available on the carcinogenicity of vanadium in humans. In a long-term study in which animals were exposed to vanadium in their drinking water, no increase in tumors was noted (ATSDR 1999).

References

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. July.

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicity Frequently Asked Questions (ToxFAQs) Fact Sheets. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Division of Toxicology. http://www.atsdr.cdc.gov/tfacts.html. July.

White Phosphorus

Summary

The RfD for white phosphorus is based on a study in which rats were given oral doses prior to mating continuing through weaning of two complete reproductive cycles (EPA 1999). Effects that were attributed to treatment with white phosphorus were loss of hair on the forelimbs and death while giving birth. Other studies with animals reveal effects on weight gain and skeletal development following oral exposure. Chronic exposure to white phosphorus in humans has been associated with a progressive disease of the jaw bones known as "phossy jaw." This disease has been documented among workers in the production of phosphorus matches (white phosphorus is no longer used for this purpose), fire crackers, and white phosphorus.

Reference

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

Zinc

Summary

The RfD for zinc is based on a study that investigated the effects of zinc supplements on copper and iron balance in adult females (EPA 1999). A decrease in a copper-containing enzyme (erythrocyte superoxide dismutase) occurred after 10 weeks of zinc exposure. In addition, several studies have found that zinc supplements decrease high-density lipoprotein (HDL) cholesterol (the "good" form of cholesterol) levels of adult males (EPA 1999).

Reference

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

Primary MCLs/HALs -- Organics

Alachlor

Summary

The RfD for alachlor is based on a study in which administration of alachlor to dogs in the diet for one year resulted in hemosiderosis (excessive deposition of iron) in the liver, kidney, and spleen as well as anemia due to red blood cell destruction (EPA 1999a). In another study, alachlor was administered to rats in the diet for two years; an increase in mortality occurred in the females and liver damage was observed in the males.

EPA has found alachlor to potentially cause slight skin and eye irritation from acute exposures at levels above the MCL (EPA 1999b). Alachlor has the potential to cause damage to the liver, kidney, spleen, nasal mucosa, and eye from long-term exposure at levels above the MCL. There is some evidence that alachlor may have the potential to cause cancer from a lifetime exposure at levels above the MCL (EPA 1999b); however, EPA has not formally assessed the carcinogenic potential of alachlor (EPA 1999a).

*Reference*s

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. March.

Aldicarb

Summary

The RfD for aldicarb is based on two studies with human subjects in which a single oral dose of aldicarb resulted in clinical signs of acetylcholinesterase (an enzyme critical to the transmission of nerve impulses) inhibition, including sweating, pinpoint pupils, and leg weakness (EPA 1999a). Additional

studies in humans, rats, and dogs demonstrated acetylcholinesterase inhibition as well. Decreased weight and viability occurred in offspring of rats administered aldicarb in the diet prior to mating.

EPA has found aldicarb to potentially cause nausea, diarrhea, and relatively minor neurological symptoms from acute exposures at levels above the MCL (EPA 1999b). However, these effects appear to be rapidly and completely reversible after exposure. Aldicarb has the potential to cause neurological effects associated with the inhibition of acetylcholinesterase such as sweating, pupillary constriction, and leg weakness from chronic exposures at levels above the MCL. There is inadequate evidence to state whether or not aldicarb has the potential to cause cancer from lifetime exposures in drinking water (EPA 1999a, 1999b).

*Reference*s

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. March.

Aldrin

Summary

The RfD for aldrin is based on a study in which rats that were exposed to aldrin in their diet for two years exhibited liver and kidney damage (EPA 1999). Other studies with rats and dogs provide supportive evidence that exposure to aldrin in the diet can result in liver damage. In addition, EPA has classified aldrin as a probable human carcinogen. Aldrin has been shown to cause cancer in laboratory animals such as rats and mice when the animals were exposed to high levels over their lifetimes.

Aldrin also may affect the central nervous system (ATSDR 1999). Accidental or intentional ingestion of high levels of aldrin (thousands of times higher than average exposure) can result in convulsions and death. Because aldrin levels can build up in the body, however, moderate levels of aldrin ingested over a long period of time also can cause convulsions. In addition, studies in animals suggest that aldrin may reduce the body's ability to resist infection.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicity Frequently Asked Questions (ToxFAQs) Fact Sheets. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Division of Toxicology. http://www.atsdr.cdc.gov/tfacts.html. April.

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

Atrazine

Summary

The RfD for atrazine is based on a study in which rats exhibited decreased body weight gain after receiving atrazine in the diet for two years (EPA 1999a). Other studies with animals have demonstrated effects on the cardiovascular system in dogs and developmental effects in offspring of rats orally exposed to atrazine (EPA 1999a).

EPA has found atrazine to potentially cause a variety of health effects from acute exposure at levels above the MCL, including congestion of the heart, lungs and kidneys, hypotension, antidiuresis, muscle spasms, weight loss, and adrenal degeneration (EPA 1999b). Atrazine has the potential to cause weight loss, cardiovascular damage, retinal and some muscle degeneration, and mammary tumors from a lifetime exposure at levels above the MCL (EPA 1999b).

*Reference*s

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. March.

Benzene

Summary

The MCL for benzene is based on carcinogenicity (EPA 1996). EPA has classified benzene as a known human carcinogen (EPA 1999a). Benzene has been associated with significantly increased risks of leukemia among certain industrial workers who were exposed to relatively large amounts of benzene during their working careers (EPA 1998). These human data are supported by animal studies. Animal data also suggest that exposure to benzene increases the risk of other types of cancer besides leukemia (EPA 1999a). Acute exposure to benzene at levels above the MCL can result in mild to severe central nervous system effects as well as immune system depression and bone marrow toxicity leading to aplastic anemia (EPA 1999b).

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. April.

United States Environmental Protection Agency (EPA). 1998. National primary drinking water regulations. 40 CFR §141.32.

U.S. Environmental Protection Agency (EPA). 1996. Drinking Water Regulations and Health Advisories. Office of Water. EPA 822-B-96-002.

Benzo(a)pyrene

Summary

EPA has found polycyclic aromatic hydrocarbons similar to benzo(a)pyrene to potentially cause red blood cell damage, leading to anemia, and suppressed immune system from acute exposures at levels above the MCL (EPA 1999b). Long-term exposures to benzo(a)pyrene at levels above the MCL may result in developmental and reproductive effects.

EPA has classified benzo(a)pyrene as a probable human carcinogen (EPA 1999a). Data specifically linking benzo(a)pyrene to a carcinogenic effect in humans are lacking; however, multiple studies in many animal species using numerous exposure routes have demonstrated the carcinogenic potential of benzo(a)pyrene. Therefore, benzo(a)pyrene has the potential to cause cancer in humans from a lifetime exposure to levels above the MCL (EPA 1999b).

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. August.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. August.

Carbofuran

Summary

The RfD for carbofuran is based on a study in which administration of carbofuran to dogs in the diet for one year resulted in acetylcholinesterase (an enzyme critical to the transmission of nerve impulses) inhibition and testicular and uterine effects (EPA 1999a).

EPA has found carbofuran to potentially cause a variety of nervous system effects from acute exposures, including headaches, sweating, nausea, diarrhea, chest pains, blurred vision, anxiety, and general muscular weakness (EPA 1999b). These effects, which are the result of carbofuran's ability to rapidly inhibit acetylcholinesterase activity, are generally reversible once exposure ceases. Available data on chronic toxic effects from oral exposures to carbofuran indicate that low doses have little or no adverse health effects, whereas higher doses have the potential to cause damage to the nervous and reproductive systems. There is no evidence that carbofuran has the potential to cause cancer from lifetime exposures in drinking water.

*Reference*s

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. March.

Chlordane

Summary

The RfD for chlordane is based on a study in which mice exhibited liver damage (hepatic necrosis) following exposure to chlordane in their diet for two years (EPA 1999a). Other studies confirm that the liver is the most sensitive target organ in rodents following chronic oral exposure to chlordane. However, available occupational studies give no indication that the liver is a target organ in humans as a consequence of chronic exposure to low levels of chlordane. EPA has found chlordane to potentially cause central nervous system effects (including irritability, excess salivation, labored breathing, tremors, convulsions, and deep depression) and blood system effects from acute exposures at levels above the MCL (EPA 1999b).

EPA has classified chlordane as a probable human carcinogen (EPA 1999a). Data specifically linking chlordane to a carcinogenic effect in humans are lacking; however, chlordane treatment has

induced benign or malignant liver tumors in mice. Therefore, chlordane has the potential to cause cancer in humans from a lifetime exposure to levels above the MCL (EPA 1999b).

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. August.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. August.

Chloroform

Summary

The RfD for chloroform is based on a study in which liver damage occurred following oral administration to dogs for 7.5 years (EPA 1999). In addition. EPA has classified chloroform as a probable human carcinogen. Chloroform has been shown to cause cancer in laboratory animals such as rats and mice when the animals were exposed to high levels over their lifetimes.

Reference

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

Cyanazine

Summary

EPA has developed a tentative (not officially proposed) MCL for cyanazine (EPA 1996); however, the RfD for cyanazine has been withdrawn and is being revised by the Agency (EPA 1999). Animal studies indicate that oral exposure to cyanazine during pregnancy may cause birth defects in offspring, including decreased brain and kidney weights, incomplete bone development, and cleft palate (EXTOXNET 1999).

References

Extension Toxicology Network (EXTOXNET). 1999. Pesticide Information Profiles. Oregon State University. http://ace.orst.edu/info/extoxnet/pips/ghindex.html. April.

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. March.

U.S. Environmental Protection Agency (EPA). 1996. Drinking Water Regulations and Health Advisories. Office of Water. EPA 822-B-96-002.

Dibromochloropropene

Summary

The MCL for dibromochloropropane (DBCP) is based on carcinogenicity (EPA 1996, 1998); however, the Agency has not completed its formal assessment of the carcinogenic potential of DBCP (EPA 1999a). Nevertheless, DBCP has been shown to cause cancer in laboratory animals such as rats and mice when the animals were exposed to high levels over their lifetimes (EPA 1998). In addition, EPA has found DBCP to potentially cause kidney and liver damage and atrophy of the testes following acute exposure (EPA 1999b). DBCP has the potential to cause kidney damage and antifertility effects from long-term exposure to levels above the MCL (EPA 1999b).

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. April.

United States Environmental Protection Agency (EPA). 1998. National primary drinking water regulations. 40 CFR §141.32.

U.S. Environmental Protection Agency (EPA). 1996. Drinking Water Regulations and Health Advisories. Office of Water. EPA 822-B-96-002.

1,2-Dichloropropane

Summary

The MCL for 1,2-dichloropropane is based on carcinogenicity (EPA 1996, 1998); however, the Agency has not conducted a formal assessment of the carcinogenic potential of 1,2-dichloropropane (EPA 1999a). Nevertheless, 1,2-dichloropropane has been shown to cause cancer in laboratory animals such as rats and mice when the animals were exposed to high levels over their

lifetimes (EPA 1998). In addition, EPA has found that short-term and long-term exposures to 1,2-dichloropropane at levels above the MCL to potentially affect the liver, kidneys, bladder, gastrointestinal tract, and respiratory tract (EPA 1999b).

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. April.

United States Environmental Protection Agency (EPA). 1998. National primary drinking water regulations. 40 CFR §141.32.

U.S. Environmental Protection Agency (EPA). 1996. Drinking Water Regulations and Health Advisories. Office of Water. EPA 822-B-96-002.

Di(2-ethylhexyl)phthalate

Summary

The RfD for di(2-ethylhexyl)phthalate (DEHP) is based on a study in which guinea pigs exhibited increased liver weight following exposure to DEHP for one year in their diet (EPA 1999a). DEHP also was a reproductive toxicant to mice following dietary exposure; it significantly decreased fertility and the proportion of live pups and damaged the male reproductive organs. In addition. EPA has classified DEHP as a probable human carcinogen. DEHP has been shown to cause cancer in laboratory animals such as rats and mice when the animals were exposed to high levels over their lifetimes.

EPA has found DEHP to potentially cause mild gastrointestinal disturbances, nausea, and vertigo following short-term exposure at levels above the MCL (EPA 1999b). Long-term exposure at levels above the MCL could result in damage to the liver and testes and reproductive effects.

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. April.

Endrin

Summary

The RfD for endrin is based on a study in which dogs exhibited liver damage and convulsions following exposure to endrin in their diets for two years (EPA 1999a). In addition, EPA has determined that there is inadequate evidence to state whether or not endrin has the potential to cause cancer in humans.

EPA has found endrin to potentially cause tremors, labored breathing, mental confusion, and convulsions following short-term exposure at levels above the MCL (EPA 1999b). Long-term exposure at levels above the MCL could result in convulsions and liver damage.

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. April.

Ethylene Glycol

Summary

The RfD for ethylene glycol is based on a study in which rats that were exposed to ethylene glycol in their diet for two years exhibited increased mortality, neutrophil count, water intake, kidney hemoglobin and hematocrit, and chronic nephritis (EPA 1999). Additional studies with rats indicated that exposure to high levels of ethylene glycol in the diet can resulted in increased mortality, decreased growth, increased water consumption, and other kidney effects (i.e., proteinurea and renal calculi).

In humans, drinking large amounts can result in nausea, convulsions, slurred speech, disorientation, and heart problems (ATSDR 1999). In addition, ethylene glycol affects the body's chemistry by increasing the amount of acid, resulting in metabolic problems. At this writing, EPA has not yet developed a complete evaluation of the human carcinogenic potential of ethylene glycol.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1997. Toxicity Frequently Asked Questions (ToxFAQs) Fact Sheets. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Division of Toxicology. http://www.atsdr.cdc.gov/tfacts.html. September.

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. July.

Formaldehyde

Summary

EPA is in the process of drafting health advisory levels for formaldehyde (EPA 1996). The RfD for formaldehyde is based on a study in which rats exhibited decreased weight gain as well as gastrointestinal tract and kidney damage after receiving formaldehyde in drinking water for two years (EPA 1999). In addition, EPA has classified formaldehyde as a probable human carcinogen. Formaldehyde has been shown to cause cancer in laboratory animals such as rats and mice when the animals were exposed to high levels over their lifetimes.

References

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

U.S. Environmental Protection Agency (EPA). 1996. Drinking Water Regulations and Health Advisories. Office of Water. EPA 822-B-96-002.

Heptachlor Epoxide

Summary

The RfD for heptachlor expoxide in which dogs exhibited increased liver weight following exposure to heptachlor epoxide in their diet for over a year (EPA 1999a). In addition. EPA has classified heptachlor epoxide as a probable human carcinogen. Heptachlor epoxide has been shown to cause cancer in laboratory animals such as rats and mice when the animals were exposed to high levels over their lifetimes.

EPA has found heptachlor expoxide to potentially cause liver and central nervous system damage from short-term exposure at levels above the MCL (EPA 1999b). Heptachlor epoxide has the potential to cause extensive liver damage from long-term exposure at levels above the MCL.

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. April.

Methylene Chloride (Dichloromethane)

Summary

The RfD for methylene chloride is based on a study in which rats were exposed to the substance in their drinking water for two years (EPA 1999a). The critical effect was liver damage. In addition, EPA has classified methylene chloride as a probable human carcinogen. Methylene chloride has been shown to cause cancer in laboratory animals such as rats and mice when the animals were exposed to high levels over their lifetimes.

EPA has found methylene chloride to potentially cause adverse neurological effects and blood cell damage from acute exposures to levels above the MCL (EPA 1999b). Methylene chloride has the potential to cause liver damage following long-term exposure to levels above the MCL.

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. April.

Naphthalene

Summary

The RFD for naphthalene is based on a study in which male rats that were administered naphthalene in oil for 13 weeks had decreased body weight at study termination (EPA 1999). EPA has classified naphthalene as a possible human carcinogen (Group C) (EPA 1999).

Humans who have ingested large amounts of naphthalene have experience anemia, liver toxicity (elevated levels of hepatic enzymes), kidney toxicity (elevation of creatinine and blood urea nitrogen, presence of protein and hemoglobin in the urine), and central nervous system effects (e.g., confusion, lethargy, vertigo) (ATSDR 1999).

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1995. Toxicological profile for naphthalene, 1-methylnaphthalene, and 2-methylnaphthalene (Update). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services.

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. July.

Pentachlorophenol

Summary

The RfD for pentachlorophenol is based on a study in which rats were exposed to the substance in their diet water for two years (EPA 1999a). The critical effects were liver and kidney damage. In addition, EPA has classified pentachlorophenol as a probable human carcinogen. Pentachlorophenol has been shown to cause cancer in laboratory animals such as rats and mice when the animals were exposed to high levels over their lifetimes.

EPA has found pentachlorophenol to potentially cause nervous system effects from acute exposures to levels above the MCL (EPA 1999b). Pentachlorophenol has the potential to cause reproductive effects and liver and kidney damage following long-term exposure to levels above the MCL.

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. April.

Phenol

Summary

The RfD for phenol is based on a study in which the offspring of rats orally exposed to phenol during pregnancy exhibited reduced body weight (EPA 1999). Other developmental toxicity studies with animals demonstrated similar effects. In one study the offspring of rats orally exposed to phenol during pregnancy also exhibited birth defects. EPA has determined that there is inadequate evidence to state whether or not phenol has the potential to cause cancer in humans.

In humans, repeated exposure to low levels of phenol in drinking water has been associated with diarrhea and mouth sores (ATSDR 1999). Muscle tremors and loss of coordination occurred in animals exposed to high levels of phenol in their drinking water.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Public Health Statements. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Division of Toxicology. http://www.atsdr.cdc.gov/ToxProfiles. April.

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

Tetrachloroethene (Tetrachloroethylene)

Summary

The MCL for tetrachloroethene is based on carcinogenicity (EPA 1996, 1998); however, the Agency has not completed its formal assessment of the carcinogenic potential of tetrachloroethene (EPA 1999a). Nevertheless, tetrachloroethene has been shown to cause cancer in laboratory animals such as rats and mice when the animals were exposed to high levels over their lifetimes (EPA 1998).

The RfD for tetrachloroethene is based on a study in which mice exhibited liver damage following oral exposure for six weeks (EPA 1999a). In another study, administration of tetrachloroethene in drinking water to rats resulted in depressed body weights. In addition, EPA has found tetrachloroethene to potentially cause detrimental effects to the liver, kidneys, and central nervous system from short-term or long-term exposures to levels above the MCL (EPA 1999b).

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. April.

United States Environmental Protection Agency (EPA). 1998. National primary drinking water regulations. 40 CFR §141.32.

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Toluene

Summary

The RfD for toluene is based on a study in which rats were exposed orally for 13 weeks (EPA 1999a). The critical effects was increased liver and kidney weights; however, female rats also exhibited increased heart weight. Liver, kidney, brain, and urinary bladder damage also were noted in some rats. In another 13-week study, mice exposed orally to toluene exhibited central nervous system effects such as convulsions, prostration, and ataxia. EPA has determined that there is inadequate evidence to state whether or not endrin has the potential to cause cancer in humans.

EPA has found toluene to potentially cause fatigue, nausea, weakness, and confusion following short-term exposure at levels above the MCL (EPA 1999b). Long-term exposure to levels above the MCL could result in: spasms, tremors, and imbalance; impaired speech, hearing, vision, memory, and coordination; liver and kidney damage.

References

U.S. Environmental Protection Agency (EPA). 1999a. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

U.S. Environmental Protection Agency (EPA). 1999b. National Primary Drinking Water Regulations Technical Fact Sheets. Washington, D.C.: Office of Water, Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/hfacts.html. April.

Total Trihalomethanes

Summary

There are four trihalomethanes: bromodichloromethane, bromoform, chlorodibromomethane, and chloroform. EPA has classified all four trihalomethanes as probable human carcinogens (EPA 1999). The non-cancer effects of trihalomethanes may include liver, kidney, and thyroid damage. The RfD for bromodichloromethane is based on a study in which oral exposure to mice and rats for two years resulted in liver and kidney damage (rats and male mice) as well thyroid damage (mice). In another study, oral exposure in rats and mice for 90 days resulted in liver damage (rats and female mice) and kidney damage (male rats and male mice). The RfD for bromoform is based on a study in which oral doses to mice and rats for 90 days resulted in liver damage (males only). The RfD for chlorodibromomethane is based on a study in which rats and mice (males only) exhibited liver damage following oral exposure for 90 days. In another study, oral exposure in rats and mice for two years resulted in liver damage (rats and mice), kidney damage (female rats), and thyroid damage (female mice). The RfD for chloroform is based on a study in which liver damage occurred following oral administration to dogs for 7.5 years.

Reference

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

Primary MCLs/HALs -- Microbiology

Total Coliforms

Summary

Coliforms are ubiquitous in the environment and generally are not considered harmful, but their presence in treated drinking water supplies points to deficiencies in either water treatment or in the water distribution system. The presence of coliforms is an indication that other more harmful microorganisms also may be present. The disease symptoms caused by pathogenic microorganisms that may be present when coliforms are detected include diarrhea, abdominal cramps, nausea, and possibly jaundice. The MCL for total coliforms is based on sampling results, rather than a specific density or other quantification, and allows no more than 5.0 percent of samples collected during a month to be positive for coliforms (EPA 1998). (Smaller water systems that collect fewer than 40 samples per month can have no more than one total coliform-positive sample.)

The presence of fecal coliforms (including *E. coli*) are used as an indicator of contamination of the treated drinking water supply with sewage or animal wastes. Although these coliform bacteria are not considered harmful, their presence in treated drinking water indicates that there may also be

contamination by other more pathogenic (*i.e.*, disease-causing) organisms. The disease symptoms caused by pathogenic microorganisms that may be present when fecal coliforms are detected include diarrhea, abdominal cramps, nausea, and possibly jaundice. The primary drinking water regulations require that all drinking water samples must be free of fecal coliforms (EPA 1998).

Reference

United States Environmental Protection Agency (EPA). 1998. National primary drinking water regulations. 40 CFR §141.32.

Turbidity

Summary

Turbidity in drinking water is not directly associated with adverse health effects in humans, but it may either indicate the presence of bacteria, or encourage the growth of bacteria, or interfere with the disinfection of drinking water (removal or killing of microorganisms). The EPA has established an MCL for turbidity in drinking water based on a performance standard: the cloudiness or turbidity of water cannot exceed 5 nephelometric turbidity units, or NTUs. For water systems using filters, the turbidity cannot exceed 1 NTU (0.5 NTU for conventional or direct filtration) in at least 95% of the daily samples for any two consecutive months (EPA 1998).

Secondary MCLs

Secondary MCLs are not based on potential health effects but instead are primarily established to prevent objectionable aesthetic qualities in drinking water, most commonly taste. Although secondary MCLs do not have to be met to protect human health, water suppliers must treat their water to these levels because consumers will not drink the water if it tastes or smells bad.

Aluminum

Summary

Exceeding the secondary MCL for aluminum (0.2 mg/L) may cause adverse aesthetic effects (primarily on taste) in drinking water (EPA 1998).

Reference

United States Environmental Protection Agency (EPA). 1998. National secondary drinking water regulations. 40 CFR §143.

Chloride

Summary

Exceeding the secondary MCL for chloride (250 mg/L) may cause adverse aesthetic effects (primarily on taste) in drinking water (EPA 1984, 1998).

References

United States Environmental Protection Agency (EPA). 1998. National secondary drinking water regulations. 40 CFR §143.

United States Environmental Protection Agency (EPA). 1984. National secondary drinking water regulations. Publication No. EPA 570/9-76-000.

Copper

Summary

Exceeding the secondary MCL for copper (1.0 mg/L) may cause adverse aesthetic effects on the taste of drinking water (EPA 1984, 1998).

References

United States Environmental Protection Agency (EPA). 1998. National secondary drinking water regulations. 40 CFR §143.

United States Environmental Protection Agency (EPA). 1984. National secondary drinking water regulations. Publication No. EPA 570/9-76-000.

Iron

Summary

Exceeding the secondary MCL for iron (0.3 mg/L) may cause adverse aesthetic effects (primarily on taste) in drinking water and also may cause objectionable staining of plumbing fixtures and laundry (EPA 1984, 1998).

References

United States Environmental Protection Agency (EPA). 1998. National secondary drinking water regulations. 40 CFR §143.

United States Environmental Protection Agency (EPA). 1984. National secondary drinking water regulations. Publication No. EPA 570/9-76-000.

Manganese

Summary

Exceeding the secondary MCL for manganese (0.05 mg/L) may cause adverse aesthetic effects including brown blotches in laundry items and black precipitates (EPA 1984, 1998).

References

United States Environmental Protection Agency (EPA). 1998. National secondary drinking water regulations. 40 CFR §143.

United States Environmental Protection Agency (EPA). 1984. National secondary drinking water regulations. Publication No. EPA 570/9-76-000.

pH

Summary

The designated pH range was determined to minimize corrosive properties (and the subsequent release of metals from water delivery pipes); to optimize the amount of chlorine required; and to minimize the formation of trihalomethanes (increased halogen reactions occur in water above the designated pH range).

References

United States Environmental Protection Agency (EPA). 1998. National secondary drinking water regulations. 40 CFR §143.

United States Environmental Protection Agency (EPA). 1984. National secondary drinking water regulations. Publication No. EPA 570/9-76-000.

Sulfate

Summary

Exceeding the secondary MCL sulfates (250 mg/L) may cause adverse aesthetic effects (primarily on taste) in drinking water (EPA 1998). The EPA has also determined that when sulfate levels in water are below the secondary MCL that the potential laxative effects of sulfates will be prevented (EPA 1984).

References

United States Environmental Protection Agency (EPA). 1998. National secondary drinking water regulations. 40 CFR §143.

United States Environmental Protection Agency (EPA). 1984. National secondary drinking water regulations. Publication No. EPA 570/9-76-000.

Total Dissolved Solids

Summary

Exceeding the secondary MCL for total dissolved solids (500 mg/L) may cause adverse aesthetic effects (primarily on taste) in drinking water (EPA 1984, 1998). Highly mineralized water may also contribute to deterioration of plumbing and appliances (EPA 1984).

References

United States Environmental Protection Agency (EPA). 1998. National secondary drinking water regulations. 40 CFR §143.

United States Environmental Protection Agency (EPA). 1984. National secondary drinking water regulations. Publication No. EPA 570/9-76-000.

Others

Hydrogen Sulfide

Summary

The RfD for hydrogen sulfide is based on a study in which adult pigs exhibited digestive disorders after receiving hydrogen sulfide in their diet (EPA 1999). No other information about health effects resulting from the ingestion of hydrogen sulfide were located. When inhaled, hydrogen sulfide is acutely toxic to humans and can be fatal. Some industrial workers who were exposed to low concentrations for several hours had eye injury, headaches, nausea, and insomnia.

Reference

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

Manganese

Summary

Adverse effects in humans have been associated with both deficiency and excess intake of manganese (EPA 1999). In humans, there are many reports of toxicity (specifically central nervous system effects) following inhalation exposure; however, much less is known about toxicity resulting from exposure to manganese in food or water. Because humans generally exert an efficient homeostatic control over manganese concentrations in their bodies, manganese is generally not considered to be very toxic when ingested in the diet. In fact, there are no quantitative data available to indicate toxic levels of manganese in the diet of humans. In addition, rodent studies are not considered appropriate for evaluating the toxic effects of manganese because the dietary requirements of rodents are much higher than humans. Therefore, the RfD for manganese is based on a review of several large scale investigations of dietary intake levels of manganese in humans in which no adverse effects were observed.

Reference

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. April.

Organic Wastes with High Biochemical Oxygen Demand

Summary

Biochemical oxygen demand (BOD) is a measure of the amount of oxygen consumed in the biological processes that break down organic matter in water. The greater the BOD, the greater the degree of organic pollution (EPA 1988). Organic waste injectates that have high BOD levels could negatively impact the taste, odor, and turbidity of groundwater sources of drinking water. The potential for these impacts would depend on the efficacy of treatment and disinfection measures, such as the application of hypochlorite.

Ground water contaminated with organic wastes might reach surface water bodies and adversely affect the ecology of the receiving water body. Organic wastes provide large amounts of food for aquatic decomposer organisms, and increased food supplies can lead to an excessive abundance of these organisms. The respiratory activity of the increased populations of decomposers can result in a depletion of dissolved oxygen concentrations, which can lead to significant ecological effects, such as stress or death of species, loss of populations, and changes in aquatic communities (EPA 1995).

References

U.S. Environmental Protection Agency (EPA). 1988. *Glossary of Environmental Terms and Acronym List.* Washington, DC: Office of Public Affairs. OPA 87-017.

U.S. Environmental Protection Agency (EPA). 1995. *Ecological Restoration: A Tool To Manage Stream Quality.* Washington, DC: Office of Water. EPA 841/F-95-007. http://www.epa.gov/OWOW/NPS/Ecology/

Total Phosphorus (Phosphates)

Summary

There are no EPA drinking water standards for phosphorous. Phosphorus, in the form of phosphate, is a mineral essential for life. It has been suggested, but not confirmed, that a diet high in phosphates may contribute to the development of osteoporosis in humans (NRC 1980). Rats fed a diet containing 5 percent phosphorus as dihydrogen phosphate (NaH_2PO_4) for 20 to 30 days were observed to develop renal damage (NRC 1980). Sodium orthophosphate (Na_3PO_4), is poorly absorbed and relatively nontoxic (NRC 1980).

High concentrations of phosphates in the groundwater can impact surface water ecosystems if the contaminated groundwater eventually leaches to surface water bodies. Elevated concentrations of phosphates in surface water (about 20 µg/L) in combination with elevated levels of inorganic nitrogen are associated with excessive growths of algae and other aquatic plants (EPA 1995). These algal growths impart undesirable tastes and odors to water, interfere with water treatment, make the water body aesthetically unpleasant, and alter the chemistry of the water supply (EPA 1986). The latter effect is the most significant in terms of ecological impact. The high metabolic demands by dense algal growths and the decay of dead algae can decrease oxygen concentrations in the affected surface water. These demands can lead to depletion of dissolved oxygen concentrations, which, in turn, can lead to severe stress or death of many species, loss of aquatic populations, and substantial shifts and simplification of aquatic communities (EPA 1995). To prevent algal growths, EPA recommends that total phosphates as phosphorus concentrations in water should not exceed 25 µg/L within a lake or reservoir (EPA 1986).

References

National Research Council (NRC). 1980. *Drinking Water and Health. Volume 3*. Washington, DC: National Academy Press. 3: 278-279.

U.S. Environmental Protection Agency (EPA). 1986. *Quality Criteria for Water.* Washington, DC: Office of Water. EPA 440/5-86-001.

U.S. Environmental Protection Agency (EPA). 1995. *Ecological Restoration: A Tool To Manage Stream Quality.* Washington, DC: Office of Water. EPA 841/F-95-007. http://www.epa.gov/OWOW/NPS/Ecology/

Uranium

Summary

The RfD for soluble salts of uranium is based on a study which rabbits exhibited kidney toxicity following exposure to uranyl nitrate hexahydrate (a uranium salt) in their diet for 30 days (EPA 1999). Similar, but less severe, effects occurred in rats and dogs exposed for up to two years. Uranium is known to cause kidney toxicity in humans as well. Additional animal studies have demonstrated effects of dietary uranium exposure on reproduction (ATSDR 1999, EPA 1999).

Exposure to any radioactive material may increase a person's risk of developing cancer; however, natural uranium has very low levels of radioactivity (ATSDR 1999). There is no conclusive evidence that natural uranium causes cancer in humans or animals. EPA currently is reviewing the carcinogenicity potential of natural uranium (EPA 1999).

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Public Health Statements. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Division of Toxicology. http://www.atsdr.cdc.gov/ToxProfiles. July.

U.S. Environmental Protection Agency (EPA). 1999. Integrated Risk Information System (IRIS). Cincinnati, OH: Office of Research and Development, National Center for Environmental Assessment. http://www.epa.gov/ngispgm3/iris/index.html. July.