

exposed to chromium can develop allergic contact dermatitis. "There is sufficient evidence for increased incidence of lung cancer among workers in the chromate-producing industry and, possibly, also among chromium platers and chromium alloy workers. There is also a suggestion of increased incidence of cancers at other sites. However, the chromium compounds responsible cannot be specified."³

Mutagenicity:

Chromates have been found to cause mutations and cell transformations in various bioassays. Chromium compounds have been reported to induce morphologic changes in tertiary cultures of mouse fetal cells and chromosome aberrations in bone marrow cells of rats.

Carcinogenicity:

Sufficient evidence exists for the carcinogenicity of chromium and certain chromium compounds both in humans and experimental animals. "Calcium chromate is carcinogenic in rats when given by several routes at the sites of administration. Lead chromate, sintered calcium chromate, zinc chromate, sintered chromium trioxide, lead chromate oxide, and cobalt-chromium alloy produce sarcomas at the site of their cutaneous, intramuscular and/or intrapleural administration in rats; lead chromate also produced renal carcinomas following its intramuscular administration in rats."³

Teratogenicity/Reproductive Effects:

Little evidence of fetal toxicity from chromium exposure is available. Embryonic abnormalities have been seen in chicks exposed to some chromium compounds.

Environmental Fate:

There appears to be little bioconcentration of chromium in aquatic animals.

Risk Assessment:

The toxicity of chromium has been reviewed by several scientific panels, including the EPA. Some chromium compounds are mutagenic and carcinogenic. The hexavalent compounds are the ones which most consistently produce carcinogenic responses. Some human occupational exposures to chromium compounds have been associated with an increased incidence of lung cancer. These exposures are by inhalation. No carcinogenicity has been established in animals or man via an ingestion route.

The EPA has established the following numbers for the more toxic hexavalent chromium.

NOEL: , 2.5 mg/kg/day
Safety factor: 1,000

Recommendations and Conclusions:

During the public comment period, DNR and DHSS received from the USEPA a current staff review updating the existing chromium Interim MCL which is being used in the preparation of EPAs proposed RMCLs. Unlike the 1976 Interim

Primary Drinking Water Regulations, the current review recognized the carcinogenicity of chromium. The proposed RMCLs discussed in the USEPA document are closer to the existing MCL than to the carcinogen risk assessment presented by EPA in the 1980 Ambient Water Criteria Document and upon which DHSS based its Recommended Enforcement Standard. Should the EPA final RMCL for chromium differ substantially from the existing MCL which DHSS now proposes utilizing as the most appropriate federal number for recommended enforcement standard, DHSS will review the EPA RMCL and evaluate whether to accept this new number as its groundwater standard.

Since the USEPA will shortly complete its review and development of the proposed RMCL for chromium, DHSS now feels that it is most appropriate to utilize the 1976 MCL of .05 mg/l as the federal number upon which Wisconsin groundwater standards should be based.

Because hexavalent chromium is a carcinogen in animals and humans, and ss. 160 does not stipulate that classification as a carcinogen only pertains to carcinogenicity via the water ingestion route, DHSS recommends that the PAL factor remain at 10%.

Revised Recommended Enforcement Standard: 50 µg/liter (50 ppb)
Recommended Preventive Action Limit factor: 10%

References:

1. National Research Council, 1980. Drinking Water and Health. National Academy Press. Washington, D.C. Vol. 3, p. 29-37, 366
2. DNR Personal Communication. December, 1984
3. USDHHS, 1983. Third Annual Report on Carcinogens - Summary. Public Health Service. Springfield, VA. p. 41-42

General: USEPA, 1980. Ambient Water Quality Criteria for Chromium. Office of Water Regulations and Standards, Criteria and Standards Division. Springfield, VA

Federal Register, 40, 1975 (December 24) page 59570

PUBLIC COMMENTS FROM NR 140 HEARINGS AND AGENCY RESPONSES:

COMMENT: The proposed 2 micrograms/liter enforcement standard for arsenic and 30 micrograms/liter for cadmium are unreasonable and impractical. Setting the proposed enforcement standard for chromium at half the current EPA standard for chromium is unwarranted. (Source: WAMC; Merlin Horn, PW&L)

RESPONSE: During the public comment period for ch. NR 140, DNR and DHSS received from the USEPA a current staff review updating the existing interim maximum contaminant level (MCL) for chromium, which is being used in the preparation of EPAs proposed RMCLs. Unlike the 1976 Interim

Primary Drinking Water Regulations, the current review recognizes the carcinogenicity of chromium.

The proposed RMCL discussed in the USEPA document for chromium is closer to the existing MCL than to the carcinogen risk assessment presented by EPA in 1980 Ambient Water Criteria Document and upon which DHSS based its initial recommended enforcement standard. Should the EPA final RMCL for chromium differ substantially from the existing MCL which DHSS now proposes utilizing as the most appropriate federal number for the recommended enforcement standards, DHSS will review the EPA RMCL and evaluate whether to adopt this new number as its groundwater standard. Since the EPA will shortly complete its review and development of the proposed RMCLs for chromium, DHSS feels that it is appropriate to utilize the 1976 MCLs of .05 mg/l for chromium as the enforcement standards. Because hexavalent chromium is a carcinogen in animals and humans, DHSS recommends that the PAL factor be 10% for chromium.

MERCURY

Introduction:

Mercury is one of the least abundant elements in the crust of the earth, with trace amounts of the metal found in at least 30 ores. Only one ore, cinnabar, contains enough mercury to justify commercial extraction. Mercury's largest use is as a cathode in the electrolytic preparation of chlorine and caustic soda with lesser uses in electrical apparatus, industrial and control instruments, control of fungal diseases, dental amalgams, catalysts, pulp and paper manufacture, pharmaceuticals, and metallurgy and mining.

A 1970 survey of U.S. waters found mercury concentrations greater than 10 µg/liter in only 4% of surface water samples. Highest concentrations of mercury were found in samples from small streams. The mercury content of lakes and reservoirs was reported between 0.1 and 1.8 µg/liter. Of 261 community, recreational, and federal installation water supplies surveyed, 95.5% showed either no detectable levels or less than 1.0 µg/liter of mercury in the raw and finished water.¹ No mercury has ever been detected in Wisconsin community water systems.²

Human Exposure Routes:

Exposure to metallic mercury other than by inhalation is infrequent. Ingestion of methylmercury in fish and shellfish has been reported to cause acute toxicity and death, as in the Minamata Bay episode in Japan. No reports have been found linking the consumption of mercury-contaminated water with adverse human health effects.¹

Acute and Chronic Toxicity:

Limited animal data suggests that mercury poisoning in animals is similar to that in humans. Animals exposed to mercury develop neurological damage, kidney damage, and peripheral nervous system damage.

Human Health Effects:

Acute poisoning due to mercury vapor inhalation can occur due to accidental contamination of poorly ventilated areas, such as tanks, during the extraction of mercury from its ore, or during the heating of mercury-based alloys. Symptoms of acute toxicity include pulmonary irritation (chemical pneumonia), which can lead to acute pulmonary edema. Renal involvement is possible in these situations. Acute poisoning is more often the result of accidental or voluntary ingestion of a mercury salt, which can cause severe inflammation of the gastrointestinal tract, followed by renal insufficiency due to necrosis of the proximal convoluted tubules. Metallic mercury can cause allergic contact eczema, and its salts irritate skin.³

Early detection of chronic mercury poisoning is difficult to achieve. Chronic mercury poisoning is manifested through digestive and nervous system abnormalities. Early symptoms of chronic poisoning include anorexia, intermittent tremor, and neurotic disorders. If exposure to mercury is terminated as soon as initial symptoms are diagnosed, the patient may recover.

Should exposure continue and the intoxication become firmly established, no more than an alleviation of symptoms can be expected to occur in the majority of cases. Commonly observed digestive system disorders include gingivitis, ulceromembraneous stomatitis, and non-specific pharyngitis. Nervous system involvement may follow two lines: fine-intention tremor, reminiscent of that found in persons suffering from multiple sclerosis; or parkinsonism, with tremor at rest and reduced motor function. Chronic exposure to mercury can cause "mercurialentis" in the eyes, characterized by discoloration of anterior capsule of the crystalline lens. Chronic intoxication is accompanied by blood disorders such as mild anemia which may be preceded by polycythaemia.³

Mutagenicity:

No mutagenic effects in human populations from exposure to methylmercury and other short chain alkyl mercurials have been reported. One report notes that alkylmercury compounds may damage gametes prior to fertilization in test rats, but similar experiments in mice failed to demonstrate statistically significant effects. Methylmercury has been shown to block mitosis in plant cells, human leukocytes treated in vivo and human cells in tissue culture, to cause chromosome breakage in plant cells, and to cause point mutations in Drosophila. No data exists on the mutagenic effects of mercury vapor and liquid metallic mercury in humans, animals, or in vitro tests. No data has been published on the mutagenicity of mercury salts in humans.

Carcinogenicity:

Peritoneal injection of metallic mercury into rats caused sarcomas only in those tissues that had direct contact with the material.

Teratogenicity/Reproductive Effects:

Animals exposed to methylmercury exhibited embryotoxic and teratogenic effects including brain damage, increased frequency of cleft palate, reduced birth weight, and non-lethal anatomical malformations. Rats exposed to mercury vapor have died within 6 days after birth.

Environmental Fate:

Several forms of mercury, ranging from elemental to dissolved inorganic and organic species, are found in the environment and are considered a serious pollutant of aquatic ecosystems. Once methylation of mercurial compounds in the environment occurs, uptake by aquatic life is extremely rapid and depuration is slow, due to methylmercury binding to sulfhydryl groups in muscle tissue.

Risk Assessment:

The toxicologic effects of mercury have been reviewed by several scientific panels, including the EPA. Mercury compounds have not demonstrated mutagenic or carcinogenic activity, but have been found to be teratogenic. Although the largest source of mercury is in the diet (primarily fish and shellfish), the EPA established an MCL for mercury with drinking water as the main source. The National Interim Primary Drinking Water Regulations establish an MCL of .002 mg/liter (2 ppb) for mercury.

Recommendations and Conclusions:

The Department of Health and Social Services recommends adopting the EPA MCL of .002 mg/l as the groundwater enforcement standard.

Recommended Enforcement Standard: 2 µg/liter (2 ppb)
Recommended Preventive Action Limit factor: 10% (teratogen)

References:

1. National Research Council, 1977. Drinking Water and Health. National Academy Press. Washington, D.C. Vol. 1, p. 270-279
 2. DNR Personal Communication. December, 1984
 3. International Labour Office, 1983. Encyclopedia of Occupational Health and Safety. International Labour Organization, Geneva
- General: USEPA, 1980. Ambient Water Quality Criteria for Mercury. Office of Water Regulations and Standards, Criteria and Standards Division. Springfield, VA (EPA 440/5-80-058)
- Federal Register, 40, 1975 (December 24) page 59570

LEAD

Introduction:

Lead ores are found throughout the world. The richest ore is galena (lead sulphide), which is used for commercial purposes. Metallic lead is used in the form of sheeting or pipes where pliability and resistance to corrosion are required, for cable sheathing, as an ingredient in solder, and as a filler in the automobile industry. Lead, its alloys, and compounds are used in shielding material for ionizing radiations, as protective coatings, in the manufacture of storage batteries, as a heat treatment bath in wire drawing, as compounding agents in rubber manufacture, as paint ingredients, as constituents of glass, enamels and glazes, and as an antiknock ingredient in gasoline.¹

The solubility of lead compounds ranges from 10,000,000 µg/liter at pH 5.5 to 1 µg/liter at pH 9.0. The natural content of lead in lakes and river water worldwide is estimated at 1-10 µg/liter. Rural upstate New York streams had natural background levels of soluble lead at 0.12 µg/liter, lead in suspended particulate matter at 484 ppm, and lead in soil at 7.0 ppm. Lead has been detected in 115 Wisconsin community water system samples in concentrations of 4.0-43.0 ppb during the period from 1979 to 1984.² In a survey of the 100 largest cities in the U.S., lead was found in finished water at concentrations of 0.0 to 62 µg/liter (mean=23 µg/liter). Another study, during the period 1962-1967, found from 1-139 µg/liter (mean=33.9 µg/liter) of lead in finished water from around the U.S; and the corresponding values for raw water ranged from 2-140 µg/liter (mean=23 µg/liter). The increment in mean residue values from raw to finished water suggests that lead is entering finished drinking water from the plumbing system. Water samples collected at the tap from 969 water systems throughout the U.S. had an average lead concentration of 13.1 µg/liter. Available data indicates that addition of lead to drinking water occurs chiefly in the distribution system, including household plumbing. Such additions are most likely to occur in areas with soft water.³

Human Exposure Routes:

The most important source of lead ingestion by humans is from food constituents, with lesser amounts coming from water and air. Dermal exposure is a problem only in occupational settings.

Acute Toxicity:

The following values have been reported in the literature:

*Intraperitoneal LD₅₀ (rats): 150 mg/kg (lead acetate)
Oral doses of 300 mg/kg have been reported lethal to dogs.⁴

Chronic Toxicity:

Pre- and perinatal exposure to lead may alter neurological development, behavior, and learning ability in laboratory animals.

Human Health Effects:

Acute lead poisoning in humans is rare, but subchronic or chronic lead poisoning is a common occurrence, especially among urban children. Infants and young children are more susceptible than adult females and adult males to the effects of lead poisoning. Excessive lead intake results in adverse effects to the heme-hemoprotein system, the kidneys, and the nervous system, especially the developing nervous system. Of major concern is the reported subtle effect of lead on behavior, especially in infants and young children. Occupational and environmental exposure to lead has been associated with premature births, miscarriages, sperm abnormalities, and other reproductive system abnormalities.⁴

Mutagenicity:

Lead acetate was reported to be mutagenic in the sperm abnormality assay in mice but not in the micronucleus or Salmonella tests.⁴

Carcinogenicity:

Several studies show that lead can cause renal tumors in rats. Some evidence links lead to induction of brain tumors in rats, renal tumors in mice, and lung tumors in hamsters. All of these tests used very high doses of lead. There is no evidence of lead-induced cancer in humans.⁴

Teratogenicity/Reproductive Effects:

There are no conclusive data indicating that lead is teratogenic in humans. Lead has been shown repeatedly in animal tests to be teratogenic. Teratogenic effects in animals appear to be preceded by embryotoxicity.

Environmental Fate:

Lead has been shown to bioaccumulate in aquatic organisms with bioaccumulation factors ranging from 42-1,700.

Risk Assessment:

Lead is a well-known environmental toxicant. Human epidemiologic and clinical poisoning experience is extensive. Lead remains under scientific scrutiny because of a continuing concern over possible subtle effects on neurobehavioral and growth parameters at low levels of chronic exposures. Lead has displayed mutagenic, carcinogenic and teratogenic responses in some test systems. Multiple scientific groups continue to monitor advances in the understanding of lead toxicity. The EPA established .050 mg/l as a MCL under the National Interim Drinking Water Regulations.⁵ Other reviews have maintained that this level is adequate. As new assessments are made concerning acceptable total daily lead absorption, the acceptable contribution from water may be revised.

Recommendations and Conclusions:

Until additional information becomes available, the Department of Health and Social Services recommends adopting the EPA MCL of .050 mg/l (50 ppb) as the groundwater enforcement standard.

Recommended Enforcement Standard: 50 µg/liter (50 ppb)
Recommended Preventive Action Limit factor: 10% (mutagen,
carcinogen, teratogen)

References:

1. International Labour Office, 1983. Encyclopedia of Occupational Health and Safety. International Labour Organization. Geneva
 2. DNR Personal Communication. December, 1984
 3. National Research Council, 1977. Drinking Water and Health. National Academy Press. Washington, D.C. Vol. 1, p. 210-221, 254-261, 302-304, 309-311
 4. National Research Council, 1982. Drinking Water and Health. National Academy Press. Washington, D.C. Vol. 4, p. 179-183
 5. USEPA, 1976. National Interim Primary Drinking Water Regulations. p. 69-72
- General: USEPA, 1980. Ambient Water Quality Criteria for Lead. Office of Water Regulations and Standards, Criteria and Standards Division. (EPA 440/5-80-057)

Federal Register, 40, 1975 (December 24) page 59570

SELENIUM

Introduction:

Selenium is a naturally-occurring element, usually found as a sulfide ore of the heavy metals. Selenium is used in photocopying, manufacture of glass, electronic devices, pigments, dyes, insecticides, veterinary medicines and anti-dandruff shampoos. Weathering rocks and soils provide the major sources of selenium to the environment, with human activities contributing an additional 3,500 metric tons per year. Inorganic selenium may be converted to the organic form by biological action. Selenium solubility varies from greater than 40% by weight for the sodium selenates to 16,000 to 33,000 µg/liter for the silver selenates.

Only 1 of 418 drinking water samples in one study exceeded 10 µg/liter. A study of home tap water samples from geographically dispersed locations found only 9.96% of the samples with concentrations greater than the detection limit of 1 µg/liter. Water from some springs and shallow wells was shown to contain selenium residues at more than 100 µg/liter. Selenium has been detected in one Wisconsin community water system sample at a concentration of 9.0 ppb during the time period 1980-1984.¹ Water from some Wyoming wells found in seleniferous areas contains selenium concentrations sufficient to poison man and animals. Available reports indicate that humans face little danger from selenium in finished water, but wells drilled through seleniferous strata containing soluble selenium may yield water with selenium concentrations great enough to cause toxic effects.

Human Exposure Routes:

Human exposure to selenium occurs by ingestion of contaminated water or food, by inhalation, or by dermal contact. Selenium concentrations in food plants depend on selenium levels in farm soils. Foodstuffs contain an average selenium content ranging from 0.006 to 0.532 µg/gm net weight. Most urban regions have atmospheric particulate selenium concentrations of 0.1 to 10 mg/m³. Toxicity from dermal exposure occurs only in occupational settings.

Acute Toxicity:

The following values have been reported in the literature:

- *Intravenous LD₅₀ (laboratory animals): 3 mg/kg b.w. (selenite and selenate)
- *Intraperitoneal LD₅₀ (mice): 1.3 mg/kg (dimethyl selenide)

Toxic concentrations of selenium caused toxic effects in laboratory animals including poor growth in weanling rats, growth depression, enlargement of the pancreas, reduction of hemoglobin content, increased serum bilirubin, general visceral congestion, and cirrhosis of the liver²

Chronic Toxicity:

Concentrations of selenium causing chronic toxicity depend on the compound tested. Dogs and rats display symptoms of chronic selenium toxicity at levels

of about 5 to 10 mg/kg selenium in the diet. Adverse health effects caused by chronic exposure include liver damage in the form of atrophy, cirrhosis, hemorrhage, marked and progressive anemia, and changes in the ovaries, pituitary, and adrenal glands.

Human Health Effects:

While elemental selenium is relatively nontoxic, some compounds such as soluble salts of selenium dioxide, selenium trioxide, and some halogen compounds are highly toxic to humans. Hydrogen selenide is one of the most toxic and irritating selenium compounds. Human intake of selenium dust in occupational settings can cause irritation of the eyes and mucous membranes, sneezing, coughing, dizziness, dyspnea, dermatitis, headaches, pulmonary edema, nausea, and garlic breath odor. Prolonged exposure can result in death. Chronic exposures from ingestion or dust inhalation can produce depression, nervousness, occasional dermatitis, gastrointestinal disturbance, giddiness, garlic breath and sweat.²

Mutagenicity:

Selenium affects the genetic process in barley and in Drosophila melanogaster. Barley treated prior to meiosis with sodium selenite exhibited structural alterations in the meiotic chromatin.

Carcinogenicity:

Selenium sulfide administered by gavage to rats and mice induced hepatocellular carcinomas in male and female rats and female mice, and alveolar/bronchiolar carcinomas and adenomas in female mice, but was not carcinogenic to male mice. These results provide sufficient evidence of selenium carcinogenicity in experimental animals.³

Teratogenicity/Reproductive Effects:

Chick embryos are highly sensitive to selenium, and exposures can result in poor hatchability of eggs and deformed eggs. Normal development of mammalian embryos exposed to selenium has been reported. It has been suggested that selenium may be a teratogen in humans.

Environmental Fate:

Selenium does not appear to readily bioaccumulate in aquatic organisms.

Risk Assessment:

Selenium is important in human nutrition. The NAS Food and Nutrition Board has established that a safe and adequate range of intakes for selenium in adults is 50 - 200 µg per day. Although some reviewers consider selenium to be an animal carcinogen, its role in human carcinogenesis has not been fully evaluated. Mutagenicity and teratogenicity have not been shown conclusively.

The EPA established an MCL of 10 µg/l for selenium as part of the National Interim Drinking Water Regulations. A 1980 review by EPA reconfirmed the adequacy of that concentration.

Recommendations and Conclusions:

Until more definitive information becomes available, the Department of Health and Social Services recommends that the EPA MCL of .01 mg/l be adopted as the groundwater enforcement standard.

Recommended Enforcement Standard: 10 µg/liter (10 ppb)
Recommended Preventive Action Limit factor: 10% (carcinogen)

References:

1. DNR, Personal Communication, December, 1984
2. National Research Council, 1977. Drinking Water and Health. National Academy Press. Washington, D.C. Vol. 1, p. 344-369, 430-433
3. USDHHS, 1983. Third Annual Report on Carcinogens - Summary. Public Health Service. Springfield, VA. p. 117-119

General: USEPA, 1980. Ambient Water Quality Criteria for Selenium. Office of Water Regulations and Standards, Criteria and Standards Division. Springfield, VA. (EPA 440/5-80-070)

Federal Register, 40, 1975 (December 24) page 59570

PUBLIC COMMENTS FROM NR 140 HEARINGS AND AGENCY RESPONSES:

COMMENT: Identification of selenium as a human carcinogen by DHSS is technically unfounded. (Source: Merlin Horn, WP&L)

RESPONSE: In reaching a conclusion that selenium is a carcinogen, DHSS utilized and concurred with toxicological data from various sources, including the EPA, NAS and IARC, that indicate selenium is carcinogenic in animals.

URANIUM

Introduction:

Uranium is a silvery-white metal which occurs with ubiquitous distribution in the earth's crust in three isotopic forms, U-238, U-235, and U-234, in the relative abundance of 99.27%, 0.72% and 0.006%, respectively. All isotopes produce alpha particles, but on a weight basis the activity of U-234 is 17,000 fold, and that of U-235 6-fold, greater than that of U-238. U-235 is used in atomic and hydrogen bombs, while U-234 and U-235 are used as fuel for nuclear power reactors.¹ The relationship between mass and radioactivity is: 1 μg =0.67 pCi. Concentrations of uranium in drinking water range from 0.02 to 200 μg /liter in fresh waters. U-238 has been detected in tap water at less than 0.03 pCi/liter.

Human Exposure Routes:

Minimal amounts of uranium may be ingested from drinking water. It is estimated that drinking water rarely contributes more than 2-5% of the total uranium ingested daily. Other routes of exposure include inhalation, diet, and occupational exposure.

Acute Toxicity:

The following values have been reported in the literature:

- *Oral LD₅₀ (rats): 1.12 mg/kg b.w.
- *Oral LD₅₀ (rabbits): 0.55 mg/kg b.w.³

Rabbits given intravenous doses of uranium showed decreases in weight, hemoglobin and erythrocytes, increases in nonprotein nitrogen and urea, and histological examinations revealed nephropathology and hepatotoxicity.

Chronic Toxicity:

Rats fed 2.0 mg/kg and rabbits 60.0 mg/kg uranium displayed altered metabolism of nucleic acids in the kidney and the liver.

Human Health Effects:

Few recent data are available on uranium toxicity to man. Epidemiological studies of two towns in Russia having 0.04-0.05 mg/liter (Town A) and 0.002-0.004 mg/liter (Town B) of uranium in their drinking water showed no differences in health. The greatest amounts of uranium were found in the kidneys and bones of deceased residents in both towns. Additional experiments found a difference in the ratio of serum albumin to globulins. Inhabitants of Town A had a decrease in albumin but an increase in globulins compared to inhabitants of Town B.

Mutagenicity:

No data were available for review.

Carcinogenicity:

Carcinogenicity studies in uranium mine workers are of limited value since other compounds may have caused the noted malignancies. Rats given uranium suspensions in their femurs developed tumors (sarcoma) in the tissues surrounding the injection site. Tumors underwent metastasis to inguinal, lung and lymph node sites. Injection of uranium into the pleural cavity of rats produced tumors at the site of injection.

Teratogenicity/Reproductive Effects:

No data were available for review.

Environmental Fate:

No data were available for review.

Risk Assessment:

Uranium is a naturally-occurring element which can be found in groundwater. It is a carcinogen but has not been adequately evaluated for mutagenic and teratogenic activity. Both the chemical and carcinogenic risks have been evaluated by the EPA-ODW, and a health-effects guidance level of 10 pCi/liter was recommended, which represents a 30×10^{-6} lifetime cancer risk.

Recommendations and Conclusions:

Until more data becomes available, the Department of Health and Social Services recommends that the EPA-ODW health-effects guidance level of 10 pCi/liter be adopted as the groundwater enforcement standard.

Recommended Enforcement Standard: 10 pCi/liter
Recommended Preventive Action Limit factor: 10% (carcinogen)

References:

1. National Research Council, 1977. Drinking Water and Health. National Academy Press. Washington, D.C. Vol. 1, p. 173, 859-860
 2. International Labour Office, 1972. Encyclopedia of Occupational Health and Safety. McGraw-Hill. New York. p. 1188-1190
- General: National Research Council, 1980. Drinking Water and Health. National Academy Press. Washington, D.C. Vol. 3, p. 173-177