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 trioxidem, 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 procued 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. Encylopedia 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)

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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 $LD_{50}$ (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 preceeded 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 contaminiated 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/m$^3$. Toxicity from dermal exposure occurs only in occupational settings.

## Acute Toxicity:

The following values have been reported in the literature:

*Intravenous $LD_{50}$ (laboratory animals): 3 mg/kg b.w. (selenite and selenate)
*Intraperitoneal $LD_{50}$ (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 liver[2]

## 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 carcinogenicity 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 $LD_{50}$ (rats): 1.12 mg/kg b.w.
*Oral $LD_{50}$ (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 globulin. 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 $30 \times 10^{-6}$ 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

# SECTION 2: ORGANIC CHEMICAL STANDARDS AND EVALUATIONS

The organic parameters reviewed for water quality standards reflect the four organic chemicals (benzene, ethyl benzene, toluene, and xylene) reported in: "Preliminary Analyses of Ohio Oilfield Produced Brines for Selected Heavy Metals and Aromatic Hydrocarbons," (1986) by D.R. Christ and G. Hudak of the Ohio Department of Natural Resources, Division of Oil and Gas, Underground Injection Control Section. In addition, reported drinking water standards for polynuclear aromatic hydrocarbons and phenolic compounds are included, although these compounds were not analyzed in Ohio brines. These chemical classes are likely to occur since they are reported in: "Analysis for Aromatic Hydrocarbons in Oil Field Brines: A Preliminary Report." (1984) by the Michigan Department of Natural Resources, Geological Survey Division, Oil Field Brine Sampling Committee.

Table 2 summarizes the existing health and drinking water quality standards on selected organic contaminants of brines. Two reference sources were utilized for reporting on quality standards and risk assessment: the Wisconsin Department of Health and Social Services Groundwater Standards for 1985 (benzene, toluene, and xylene) and 1986 (ethylbenzene); and the Installation Restoration Program Toxicology Guide.

The Wisconsin Department of Health and Social Services under that state's groundwater protection law has been charged with the task of establishing water quality standards for many existing and potential groundwater contaminants. For benzene, ethylbenzene, toluene and xylene, the appended, abbreviated risk assessment summarizes the following information:

1. general chemical information;
2. human exposure routes;
3. acute toxicity;
4. chronic toxicity;
5. human health effects;
6. mutagenicity;
7. carcinogenicity;
8. teratogenicity/reproductive effects;
9. environmental fate;
10. risk assessment;
11. recommendations and conclusions;
12. references.

Background information on the Wisconsin approach to these standards are included prior to the individual inorganic chemical reports in Section 1. A table of all existing federal drinking water standards and reference to their full documentation are also included prior to the individual inorganic chemical reports.

The U.S. Department of Defense has identified several facilities across the nation in which past uses of chemicals has resulted in environmental contamination problems similar to those observed at so-called "Superfund" hazardous waste sites. In response the Air Force Toxic Hazards Division has developed a thorough toxicology guidance document to evaluate the environmental impacts of selected chemicals. Such data is directed towards the potential for soil and groundwater contamination, and therefore it has high relevancy towards the brine disposal problem.

Comprehensive summary reports are appended for the four measured organic contaminants (benzene, ethylbenzene, toluene, and xylene) in Ohio brines. Each report contains the following information:

1. Physicochemical Data;
2. Persistance in Soil-Water Systems;
3. Pathways of exposure;
4. Health Hazards Data;
5. Handling Precautions;
6. Emergency First Aid Treatment;
7. Environmental and Occupational Standards;
8. Chemical Usage;
9. Detailed Environmental Fate and Exposure Pathways;
10. Human Health Considerations; and,
11. Sampling and Analysis Considerations.

In addition to the four identified organics, reports on the polynuclear aromatic hydrocarbon, naphthalene, and the phenolic compound, 2.4-dimethylphenol, are included. The Michigan Department of Natural Resources had identified these classes of chemicals as present within selected brine samples.

Table 2. Summary of Health and Water Quality Standards on Selected Organic Chemicals.

a. Identified in Ohio Brines.

| Chemical | Standard | Source |
|---|---|---|
| Benzene | 0.67 ug/l | WI (1985); U.S.EPA |
| Ethylbenzene | 1360 ug/l | WI (1986); U.S.EPA |
| Toluene | 343 ug/l | WI (1985); U.S.EPA |
| Xylene | 620 ug/l | WI (1985); U.S.EPA |

b. Likely to Occur in Ohio Brine.

| Chemical Class | Standard | Source |
|---|---|---|
| Polynuclear Aromatic Hydrocarbons | 0.2 ug/l | EEC Directive (IRP Toxicology Guide) |
| Phenolic Compounds | 0.5 ug/l | EEC Directive (IRP Toxicology Guide) |

Standards reported as micrograms per liter (ug/l).

Sources: WI (1985); U.S.EPA: Standard established by the Wisconsin Department of Health and Social Services during 1985 and including an evaluation of current and proposed U.S.EPA Drinking Water Standards either as National Interim Primary Drinking Water Standards or as Maximum Contaminant Levels.

WI (1986); U.S.EPA: Similar Standards established by the Wisconsin Department of Health and Solcial Services during 1986.

EEC Directive (IRP Toxicology Guide): Standard established by the European Economic Community as maximum admissible concentrations relating to quality of water intended for human consumption. Reported in "The Installation Restoration Program Toxicology Guide." Vol. 1 (1985) prepared by Arthur D. Little, Inc. for the Harry G. Armstrong Medical Research Laboratory, Wright-Patterson AFB, OH 45433-6573.

BENZENE

Introduction:

Benzene is produced by petroleum refining, coal tar distillation, coal processing, and coal coking. It is primarily used as a chemical intermediate in the manufacture of styrene, cyclohexane, detergents, pesticides, and, in more modern times, as a starting substance for the manufacture of plastics. Total estimated production in 1984 exceeds 11 billion gallons per year.[1] Benzene is soluble in water to 0.82 g/liter. USEPA sampling of community wells found benzene contamination at 0.4 and 0.95 µg/liter. In another EPA survey, 10 water supplies had levels of benzene ranging from 0.1-0.3 µg/liter with the highest concentration found at 10 µg/liter. Benzene has been detected at concentrations of 1.3-9.5 ppb in 10 community wells in Wisconsin.[2]

Human Exposure Routes:

An estimated 3 million workers are potentially exposed to benzene during the production or use of substances containing the chemical as an ingredient or contaminant. Human exposure occurs via inhalation of the vapor and through skin absorption and also, potentially, through ingestion of contaminated water.

Acute Toxicity:

The following values have been reported in the literature:

*Oral $LD_{50}$ (newborn rats): <0.9 g/kg b.w.
*Oral $LD_{50}$ (14-day old rats): 3.0 (1.8-5.0) g/kg b.w.
*Oral $LD_{50}$ (young adult rats): 3.3 (2.6-4.2) g/kg b.w.
*Oral $LD_{50}$ (old adult rats): 4.9 (3.5-6.2) g/kg b.w.

Rabbits absorbed benzene through the skin and underwent anesthesia at 35,000-45,000 ppm. Lesions of lipoprotein membranes was seen in mice which inhaled benzene at 60 mg/liter in air (18,750 ppm).[1]

Chronic Toxicity:

Leukopenia is the most commonly observed toxic effect caused by benzene exposure. Decreases in circulating leukocytes were seen in rats exposed to benzene at air concentrations of as little as 61 ppm of benzene for 5 hour/day, 4 days/week for 6 or more weeks. Rats administered benzene subcutaneously developed leukopenia and weight loss. Oral administration of benzene to rats resulted in development of leukopenia and erythrocytopenia at the lowest minimal effect level of 50 mg/kg.

Human Health Effects:

Chronic benzene exposure in humans causes myelocytic anemia, thrombocytopenia, or leukopenia, and leukemia (particularly acute myelogenous and monocytic leukemia). Workers exposed to benzene in a rubber sheeting factory from 1940-1949 at less than the 100 ppm standard showed a significant excess of leukemia with a 10-fold increase in risk of death from myeloid and monocytic leukemia. The data suggests that benzene is a leukemogen in humans. Effects

of benzene on bone marrow proliferation in humans includes an increased incidence of chromosomal aberrations with aneuploidy and breakage.

Mutagenicity:

The toxic effects of benzene on bone marrow cells in laboratory animals, and the changes in chromosome number and chromosome breakage resemble those found in humans. Numerous mutagenicity bioassays have not shown benzene to be a mutagen. EPA-CAG (1980) suggests that somatic mutations may occur at benzene concentrations as low as 1 ppm in air.

Carcinogenicity:

There is limited evidence that benzene is carcinogenic in experimental animals. Rats administered benzene by stomach tubes exhibited an increased incidence of Zymbal gland carcinomas. An increased incidence of lymphoid tumors was reported in male mice exposed to benzene by inhalation. Sufficient evidence exists to call benzene a carcinogen in man. Case reports and epidemiological evidence links benzene exposure with leukemia. Two cohort studies reported an increased incidence of acute nonlymphocytic leukemias in workers exposed to benzene. Large numbers of leukemia cases, primarily acute nonlymphocytic, have been reported in a group of workers exposed to benzene.

Teratogenicity/Reproductive Effects:

Pregnant mice, given subcutaneous injections of acute toxic doses of benzene (3 ml/kg b.w.) on days 11 to 15 of gestation, produced malformations in some fetuses including cleft palate, agnathia, or micrognathia.

Environmental Fate:

No data was available to assess environmental fate parameters.

Risk Assessment:

Benzene is mutagenic, carcinogenic, and teratogenic. EPA (1980) determined that there is substantial epidemiological evidence that benzene is a human leukemogen. EPA-CAG estimated that the number of cases of leukemia per year in the general population due to ambient atmospheric benzene was about 90 (the 95% confidence interval is from 34 to 235).

EPA (1984)[3] has recently completed a carcinogen risk assessment. The projected concentration for the upper limit $10^{-6}$ lifetime cancer risk for benzene is .00067 mg/l (.67 µg/l).

Recommendations and Conclusions:

The Department of Health and Social Services recommends adopting the EPA's $10^{-6}$ lifetime risk determination as Wisconsin's groundwater standard for benzene, .00067 mg/l.

Recommended Enforcement Standard: .67 µg/l (.67 ppb)
Recommended Preventive Action Limit factor: 10% (mutagen, carcinogen, teratogen)

References:

1. National Research Council, 1977. Drinking Water and Health. National Academy Press. Washington, D.C. Vol. 1, p. 688-691

2. DNR press release, November 1, 1984

3. USEPA, 1984. Federal Register, 49 (June 12) p. 24340

General: National Research Council, 1980. Drinking Water and Health. National Academy Press. Washington, D.C. Vol. 3, p. 81-84

PUBLIC COMMENTS FROM NR 140 HEARINGS AND AGENCY RESPONSES:

COMMENT: The groundwater standard that DHSS has proposed for benzene serves no practical purpose regarding the protection of public health because daily intake due to water ingestion represents only a small percentage of the total daily exposure to benzene from all sources. (Source: WAMC)

RESPONSE: The recommendations for benzene were developed to protect the groundwater resource through the use of health effects data in accordance with the procedures contained in ch. 160, Stats. The groundwater standards are based on acceptable risk levels in water and are not intended to control other routes of exposures.

BENZENE

| COMMON SYNONYMS: | CAS REG. NO.: | FORMULA: | AIR W/V CONVERSION FACTORS at 25°C (12) |
|---|---|---|---|
| Benzol<br>Benzole<br>Carbon oil<br>Coal naphtha<br>Phenylhydride<br>Pyrobenzol | 71-43-2<br>NIOSH NO.:<br>CY1400000 | $C_6H_6$ | 3.19 mg/m³ ≅ 1 ppm<br>0.313 ppm ≅ 1 mg/m³ |
| | STRUCTURE:  | | MOLECULAR WEIGHT:<br>78.11 |

| | |
|---|---|
| REACTIVITY | Benzene may generate heat, react vigorously, and possibly ignite or explode in contact with oxidizing mineral acids or other strong oxidizing agents (507,511,505). |

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA | • Physical State (at 20°C): liquid | (23) |
| | • Color: colorless to light yellow | (23) |
| | • Odor: aromatic | (23) |
| | • Odor Threshold: 4.68 ppm (15 mg/m³) | (263) |
| | • Liquid Density (g/ml at 20°C): 0.8765 | (68) |
| | • Freezing/Melting Point (°C): 5.5 | (14) |
| | • Boiling Point (°C): 80.1 | (23) |
| | • Flash Point (°C): -11 (closed cup) | (23) |
| | • Flammable Limits in Air, % by Volume: 1.3-7.9 | (60,504,506) |
| | • Autoignition Temperature (°C): 560-592 | (60,504,510) |
| | • Vapor Pressure (mm Hg at 20°C): 76 | (67) |
| | • Saturated Concentration in Air (mg/m³ at 20°C): 319,000 | (67) |
| | • Solubility in Water (mg/L at 20°C): 1780 | (67) |
| | • Viscosity (cp at 20°C): 0.6468 | (21) |
| | • Surface Tension (dyne/cm at 20°C): 29 | (23) |
| | • Log (Octanol-Water Partition Coefficient), log $K_{ow}$: 2.13 | (29) |
| | • Soil Adsorption Coefficient, $K_{oc}$: 65 | (652) |
| | • Henry's Law Constant (atm·m³/mol at 25°C): $5.43 \times 10^{-3}$ | (74) |
| | • Bioconcentration Factor: 6.5 (estim) | (659) |

| | |
|---|---|
| PERSISTENCE IN THE SOIL-WATER SYSTEM | Benzene is expected to be fairly mobile in the soil/ground-water system. Transport with infiltration water is expected particularly in sandy soils and soils of low organic content. Volatilization of material near the surface or in the soil-air compartment may be important. Transformation processes such as hydrolysis and biodegradation are not expected to be significant in natural soils; however, biodegradation by acclimated populations has been reported. |

| | |
|---|---|
| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of benzene to ground-water drinking water supplies. Migration has commonly occurred in the past. Inhalation resulting from volatilization from surface soils may also be important. |

| | |
|---|---|
| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure (45,12): The primary effects of inhalation and ingestion are on the central nervous system. Symptoms include headache, dizziness, drowsiness and nausea which may progress to convulsions, respiratory paralysis and death with high vapor concentrations. Benzene causes irritation of the eyes and skin. |
| | Toxicity Based on Animal Studies: $LD_{50}$ (mg/kg) oral [rat] 3800 (59) skin -- no data; $LC_{50}$ (ppm) inhalation [rat] (59) 10,000·7 hr |
| | Long-Term Effects: Pancytopenia, leukemia |
| | Pregnancy/Neonate Data: Negative |
| | Mutation Data: Mixed results |
| | Carcinogenicity Classification: IARC - category 1; NTP - clear evidence |

| | |
|---|---|
| HANDLING PRECAUTIONS (54,52) | Handle chemical only with adequate ventilation • Vapor concentrations of 10-50 ppm: supplied-air respirator or self-contained breathing apparatus • 50-1000 ppm: supplied-air respirator or self-contained breathing apparatus with full facepiece • 1000-2000 ppm: supplied-air respirator operated in pressure-demand, positive-pressure or continuous flow mode • Butyl, natural rubber, neoprene, nitrile, viton, PE, PVC or other protective clothing to prevent prolonged or repeated skin contact with the liquid • Chemical goggles if there is possibility of eye contact. |

| | |
|---|---|
| EMERGENCY FIRST AID TREATMENT (45,53) | Caution: Do not administer stimulants • Ingestion: Do not induce vomiting. Seek medical attention • Inhalation: Move victim to fresh air; give artificial respiration if necessary. Get medical attention • Skin: Remove contaminated clothing; wash skin with soap and water. If irritation persists after washing, get medical attention • Eye: Irrigate copiously with water. Get medical attention. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards

- OSHA PEL (8-hr TWA): 10 ppm; CL: 25 ppm; PEAK: 50 ppm (10 min)
- Emergency temporary AFOSH PEL (8-hr TWA): 1 ppm; CL: 5 ppm/15 min

Criteria

- NIOSH IDLH (30-min): 2000 ppm
- ACGIH TLV® (8-hr TWA): 10 ppm (A2, suspected human carcinogen)
- ACGIH STEL (15-min): deleted

WATER EXPOSURE LIMITS:

Drinking Water Standards - None established

EPA Health Advisories

In the absence of formal drinking water standards, the EPA (383) has developed the following Health Advisories (formerly termed SNARLs) for noncarcinogenic risk for short and long-term exposure to benzene in drinking water:

- 1 day: none
- 10 days: 0.23 mg/L
- long-term: none

EPA Ambient Water Quality Criteria (355)

- Human Health
  - Based on ingestion of contaminated water and aquatic organisms ($10^{-5}$, $10^{-6}$, $10^{-7}$ cancer risk), 6.6 µg/L, 0.66 µg/L, 0.066 µg/L.
  - Based on ingestion of contaminated aquatic organisms only ($10^{-5}$, $10^{-6}$, $10^{-7}$ cancer risk), 400 µg/L, 40.0 µg/L, 4.0 µg/L.

- Aquatic Life
  - Freshwater species

    acute toxicity: no criterion, but lowest effect level occurs at 5300 µg/L.

    chronic toxicity: no criterion established due to insufficient data.

  - Saltwater species

    acute toxicity: no criterion, but lowest effect level occurs at 5100 µg/L.

    chronic toxicity: none, but adverse effects occur at concentrations as low as 700 µg/L with a fish species exposed for 168 days.

WHO Drinking Water Guideline (666):

A health-based guideline for drinking water of 10 µg/L is recommended for benzene. A daily per capita consumption of two liters was assumed.

REGULATORY STATUS (as of October 1, 1985)

Promulgated Regulations

- Federal Programs

Clean Water Act (CWA)
Benzene is designated a hazardous substance. It has a reportable quantity (RQ) limit of 454 kg (347,556). It is also listed as a toxic pollutant (351). Water quality criteria have been set. Guidelines exist for benzene effluent in the iron and steel manufacturing industry (354).

Safe Drinking Water Act (SDWA)
In states with an approved Underground Injection Control program, a permit is required for the injection of benzene-containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)
Benzene is identified as a toxic hazardous waste (U019) and listed as a hazardous waste constituent (328,329). A non-specific source of benzene-containing waste is the production of chlorinated aliphatic hydrocarbons (325). Waste streams from some organic chemicals industries (production of chlorobenzenes, nitrobenzenes and aniline) contain benzene and are listed as specific sources of hazardous waste (326,327).

Comprehensive Environmental Response Compensation and Liability Act (CERCLA)
Benzene is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 454 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing benzene but these depend upon the concentration of the chemicals present in the waste stream (556).

Clean Air Act (CAA)
Benzene is a hazardous air pollutant and is subject to national emission standards for fugitive emission sources (372).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Occupational Safety and Health Act (OSHA)
Employee exposure to benzene shall not exceed an 8-hour time-weighted-average (TWA) of 10 ppm. A ceiling level of 25 ppm shall not be exceeded at any time during an 8-hour work-shift except for a duration of 10 minutes when it may reach a ceiling level of 50 ppm (298).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated benzene as a hazardous material which is subject to requirements for packaging, labeling and transportation (306).

Food, Drug and Cosmetic Act (FDCA)
Benzene is approved for use as an indirect food additive (adhesive use) (362).

- State Water Programs
  California has an action level of 0.7 μg/L in drinking water (731).
  Connecticut has an action level of 1 μg/L for drinking water (731).
  Florida has set a criterion of 0.02 μg/L for ground and drinking water (731).
  Iowa has unpublished internal criteria for benzene: 6.6 μg/L in raw water; 22 μg/L in cold water and 198 μg/L in warm water (731).
  Louisiana has a criterion of 6.6 μg/L for the public water supply (731).
  New York quality standards for ground water require that benzene not be detectable (731).

  Other states follow EPA Ambient Water Quality Criteria.

Proposed Regulations

- Federal Programs
  Clean Water Act (CWA)
  Effluent guidelines for benzene have been proposed in the pesticide chemicals category (359) and in the organic chemicals plastics and synthetic fibers category (357).

  Safe Drinking Water Act (SDWA)
  EPA has proposed a recommended maximum contaminant level (RMCL) of zero for benzene as part of the National Primary Drinking Water Regulations (64).

  Resource Conservation and Recovery Act (RCRA)
  EPA has proposed listing spent filters, spent desiccants, light ends and filter aids from chlorinated aliphatic hydrocarbon production as a non-specific source of benzene-containing hazardous waste (330).

  EPA has proposed listing solvent use (or recovery) activities as non-specific sources of benzene-containing hazardous waste (781).

  Clean Air Act (CAA)
  A standard has been proposed for benzene emissions from coke by-product recovery plants (380).

- State Water Programs
  No proposed regulations are pending.

EEC Directives

Directive on Ground Water (538)

Direct discharge into ground water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (Solvents) (544)

Benzene is listed as a Class I/a toxic substance and is subject to packaging and labeling regulations.

Directive on the Discharge of Dangerous Substances (535)

Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground water.

Directive on Marketing and Use of Dangerous Substances (541)

Benzene is not permitted in toys or parts of toys as placed on the market where the concentration of benzene in the free state is in excess of 5 mg/kg of the weight of the toy or part of toy.

Directive on Toxic and Dangerous Wastes (542)

Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto-pharmaceutical substances; ethers and aromatic polycylic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)

Benzene is classified as a flammable, toxic substance and is subject to packaging and labeling regulations.

## 18.1 MAJOR USES

In the past, benzene was widely used as a solvent. As its adverse health effects became known, usage declined to the point where it is now minimal. At present, most benzene is consumed in the chemical industry where it is used as a starting material for the synthesis of other organic compounds (2,518). Prior to World War II, the major use for benzene was as an octane-raising additive in gasoline. Presently, its use in this area is minor. Gasoline used in the U.S. contains from 0.8 to 2.0% benzene. When benzene is used in this manner, it is not added to the gasoline pool as pure benzene but rather as a mixture of benzene, toluene and xylene (21,43,518).

## 18.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 18.2.1 Transport in Soil/Ground-water Systems

#### 18.2.1.1 Overview

Benzene may move through the soil/ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical). In general, transport pathways of low soil concentrations can be assessed by equilibrium partitioning, as shown in Table 18-1. These calculations predict the partitioning of benzene among soil particles, soil water and soil air. The portions of benzene associated with the water and air phases of the soil are more mobile than the adsorbed portion.

The estimates for the unsaturated topsoil model indicate that most of the benzene (88%) is expected to be sorbed to the soil. A much smaller (yet significant) amount (7%) will be present in the soil water phase and can thus migrate by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion. For the portion of benzene in the gaseous phase of the soil (5%), diffusion through the soil-air pores up to the ground surface, and subsequent removal by wind, will be a significant loss pathway. There is no significant difference in the partitioning calculated for 25°C and 10°C.

In saturated, deep soils (containing no soil air and negligible soil organic carbon), a much higher fraction of the benzene (79%) is likely to be present in the soil-water phase (Table 18-1) and transported with flowing ground water.

TABLE 18-1

EQUILIBRIUM PARTITIONING CALCULATIONS FOR BENZENE IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil[b,c] | | | |
| at 25°C | 88.1 | 7.1 | 4.8 |
| at 10°C | 89.7 | 7.2 | 3.1 |
| Saturated deep soil[d] | 21.4 | 78.6 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34,35,36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.

b) Utilized estimated soil sorption coefficient: $K_{oc} = 65$. (Estimated by Arthur D. Little, Inc.)

c) Henry's law constant taken as 0.00543 atm·m$^3$/mol at 25°C (74), and 0.00338 atm·m$^3$/mol at 10°C (latter calculated using 25°C/10°C ratio of H values from Brown and Wasik (521).

d) Used sorption coefficient ($K_p$) calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

In a field study on the removal of river-borne organics by dune-infiltration (using water from the Rhine River), Piet et al. (226) actually found increases in the benzene concentration after infiltration. While the reason for the increase is not known, and may have been due to some artifact of the study, the results do indicate that benzene is easily transported by infiltrating water.

18.2.1.2 Sorption on Soils

The mobility of benzene in the soil/ground-water system (and its eventual migration into aquifers) is strongly affected by the extent of

its sorption on soil particles. In general, sorption on soils is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water; and
- decrease moderately with increasing dissolved organic matter content of the soil water.

Based upon its octanol-water partition coefficient of 135, the soil sorption coefficient ($K_{oc}$) is estimated to be 65. This is a relatively low number indicative of weak sorption to soils.

18.2.1.3 Volatilization from Soils

Transport of benzene vapors through the air-filled pores of unsaturated soils is an important transport mechanism for near-surface soils. In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31).

There are no data from laboratory or field tests showing actual soil volatilization rates. Sorption of the benzene vapors on the soil may slow the vapor phase transport; Politzki et al. (516) have shown, for example, that the vapor pressure of benzene in the presence of (thus partially sorbed to) silica gel was decreased by a factor of $10^4$ from the pure compound value.

The Henry's law constant (H), which provides an indication of a chemical's tendency to volatilize from solution, increases significantly with increasing temperature (28). Moderate increases in H are also observed with increasing salinity due to a decrease in benzene's solubility (517).

18.2.2 Transformation Processes in Soil/Ground-water Systems

The persistence of benzene in soil/ground-water systems is not well documented. In most cases, it should be assumed that the chemical will persist for months to years (or more). Benzene that has been released into the air will eventually undergo photochemical oxidation; tropospheric lifetimes on the order of a few hours to a few days have been estimated (10).

Benzene under normal environmental conditions is not expected to undergo hydrolysis (10,33). Further, benzene is not expected to be susceptible to oxidation or reduction reactions in the soil/ground-water environment.

Available data on the biodegradability of benzene are somewhat contradictory (10,55,519,520). Certain pure and mixed cultures can apparently degrade benzene under environmental conditions, but the chemical must be considered fairly resistant to biodegradation. Biodegradation in acclimated wastewater treatment plants (e.g., activated sludge) would be expected to be relatively easy based upon the data of Tabak et al. (55). However, in most soil/ground-water systems such aerobic degradation would be of minimal importance because of the low concentration of microorganisms (at depth) and the low dissolved oxygen (anaerobic) conditions. No data are available on the possibility of anaerobic biodegradation.

### 18.2.3 Primary Routes of Exposure From Soil/Ground-water Systems

The above discussion of fate pathways suggests that benzene is highly volatile, weakly adsorbed by soil and has a limited potential for bioaccumulation. This compound may volatilize from soil surfaces, but that portion not subject to volatilization is likely to be mobile in ground water. These fate characteristics suggest several potential exposure pathways.

Volatilization of benzene from a disposal site, particularly during drilling or restoration activities, could result in inhalation exposures. The potential for ground water contamination is high, particularly in sandy soils. Mitre (83) reported that benzene has been found at 94 of the 546 National Priority List (NPL) sites. It was detected at 72 sites in ground water, 31 sites in surface water, and 17 sites in air. The USEPA (64) reported that in state occurrence data with 646 total number of samples, 4 were positive with a range between trace-17 μg/L. The National Organic Monitoring Survey (NOMS) (90) found that out of 113 samples, 7 were positive with a mean of positives at 0.4 μg/L.

The results of the USEPA (531) Groundwater Supply Survey (GWSS) are as shown below:

| Sample Type | Occurrences* No. | % | Median of Positives (μg/L) | Maximum (μg/L) |
|---|---|---|---|---|
| Random | | | | |
| Supplies serving <10,000 people (280 samples) | 1 | 0.4 | 0.61 | 0.61 |
| Supplies serving >10,000 people (186 samples) | 2 | 1.1 | 9.0 | 15.0 |
| Non-Random | | | | |
| Supplies serving <10,000 people (321 samples) | 5 | 1.6 | 1.6 | 12.0 |
| Supplies serving >10,000 people (158 samples) | 3 | 1.9 | 2.7 | 12.0 |

*Samples having levels over quantification limit of 0.5 μg/L.

The state data include only ground-water sources and were compiled from various state reports on local contamination problems. The state data are not considered to be statistically representative of national occurrence. The National Organic Monitoring Survey (NOMS) included data from both ground and surface water supplies. The 1982 Groundwater Supply Survey (GWSS) is the most recent study (531). This study sampled a total of almost 1000 drinking water systems using ground - water, 466 selected at random, and about 500 selected by the state as potentially contaminated. The USEPA (64) estimates that 1.5% of the nation's ground-water supplies are contaminated with benzene (>10.5 μg/L).

These results indicate that benzene has the potential for movement in soil/ground-water systems. This compound may eventually reach surface waters by this mechanism, suggesting several other exposure pathways:

- Surface waters may be used as drinking water supplies, resulting in direct ingestion exposure;
- Aquatic organisms residing in these waters may be consumed, also resulting in ingestion exposure through bioaccumulation;
- Recreational use of these waters may result in dermal exposure;
- Domestic animals may consume or be dermally exposed to contaminated ground or surface waters; the consumption of meats and poultry could then result in ingestion exposures.

In general, exposures associated with surface water contamination can be expected to be lower then exposures from drinking contaminated ground water. The Henry's law constant for benzene indicates that it will volatilize upon reaching surface waters. Secondly, the bioconcentration factor for this compound is expected to be low, suggesting limited bioaccumulation in aquatic organisms or domestic animals.

### 18.2.4 Other Sources of Exposure

Benzene is a widely used chemical, in the synthesis of other organic compounds (ethylbenzene, cumene, cyclohexane, and other benzene derivatives), as a solvent and as a pesticide among other uses. It is also an important constituent of gasoline. As a result of emissions during production, use, and disposal and because of its high volatility, benzene has become pervasive in the environment.

Coniglio et al. (223), in a summary of data from SRI, NOMS and NORS, reported that benzene was found at a frequency of 21.6% in finished surface water.

Although benzene is readily photoxidized in the atmosphere, its volatility suggests that it may be found in air as well. Brodzinsky and Singh (84) compiled all available atmospheric monitoring data for a number of volatile organics. For benzene, they had data for 2789 locations. In rural and remote locations, the median concentration was 4.5 $\mu g/m^3$. In urban and suburban locations, the median concentration was 8.9 $\mu g/m^3$, and in source-dominated areas, the median concentration was 9.6 $\mu g/m^3$. These results suggest that inhalation exposure is very important for people living in urban, suburban and source-dominated areas, and for people who live in rural and remote areas as well.

## 18.3 HUMAN HEALTH EFFECTS

### 18.3.1 Animal Studies

#### 18.3.1.1 Carcinogenicity

Although laboratory animals were once considered to be poor models for the potential carcinogenic activity of benzene to humans, recent studies indicate that benzene is carcinogenic in animals. Administration of benzene (99.7% pure) by gavage to groups of 50 F344/N rats and 50 B6C3F1 mice of each sex and for each dose, 5 days per week for 103 weeks, produced clear evidence of carcinogenicity in both species. Doses of 0, 25, 50 or 100 mg/kg bw benzene (in corn oil) were administered to male and female mice and female rats; male rats were given doses of 0, 50, 100 or 200 mg/kg bw. Dose-related lymphocytopenia was observed in treated mice and rats. An increased incidence of Zymbal gland carcinomas was seen in both sexes for both species and both male and female rats exhibited elevated incidences of squamous-cell carcinomas of the oral cavity. Male rats also displayed an increased incidence of squamous-cell carcinomas of the skin. Both sexes of B6 mice had elevated incidences of malignant lymphomas and alveolar/bronchiolar carcinomas. For female mice, benzene treatment also induced increased incidences of ovarian granulosa cell tumors and carcinomas and carcinosarcomas of the mammary gland (801).

Snyder et al. (197) observed an increased incidence of thymic lymphoma in C57BL/ J6 mice which were exposed to vapor concentrations of 300 ppm, 6 hours daily, 5 days per week for life. In contrast, AKR/J mice that were exposed to 100 ppm on the same dosing schedule experienced no change in the induction of lymphoma. Poor survival of this strain at 300 ppm necessitated the lower exposure level. It

should be noted that both of these strains carry a virus which can result in a high incidence of lymphoma following exposure to radiation, carcinogens or immunosuppressive agents (203).

Maltoni et al. (201) recently completed a series of studies which show that benzene is carcinogenic in rats by both the oral and inhalation routes. In the oral studies, Sprague-Dawley rats were administered benzene (purity 99.93%) in olive oil at doses of 50 or 250 mg/kg/day, 4-5 days weekly for 52 weeks and then kept under observation until death. Mortality was higher in the benzene-treated groups and was dose-correlated. There was also a dose-related increase in the incidence of hemolymphoreticular neoplasias ("leukemias") and of mammary carcinomas. The incidence of "leukemias" was 7.7% in the high-dose group, 3.4% in the low-dose group and 1.7% in controls. The incidence of mammary carcinomas was 10.8% and 6.9% in the high and low-dose groups, respectively, and 5.2% in the controls. There was also a 12.3% incidence of Zymbal-gland carcinomas, 3.1% incidence of carcinoma of the oral cavity and a 1.5% incidence of both angiosarcomas and hepatomas, all in the high-dose group.

In the inhalation studies, Sprague-Dawley rats were exposed to vapor concentrations of 200-300 ppm, 4-7 hours daily for 104 weeks. The investigators found a 2.3% incidence of hepatomas and a 26.6% and 1.4% incidence of mammary carcinoma and leukemia, respectively (201).

IARC (202) believes there is sufficient evidence that benzene is carcinogenic to man. A discussion of human data can be found in Section 18.3.2.2, Chronic Toxicologic Effects.

18.3.1.2 Mutagenicity

Benzene is not mutagenic in bacterial systems. Studies conducted with Salmonella typhimurium, Bacillus subtilis, Saccharomyces cerevisiae, and Escherichia coli have all proven negative (202). Chromosomal abnormalities in bone marrow cells as a result of benzene exposure have been reported in various species of animals including rats, rabbits and mice (203). The animals were treated with single or multiple daily doses of benzene ranging from 0.2 to 2.0 ml/kg/day given either subcutaneously or intraperitoneally. Most of the induced abnormalities were chromatid breaks or deletions. In addition, rats exposed to benzene vapor at concentrations of 1, 10, 100 or 1000 ppm for 6 hours showed a significant increase in chromosomal abnormalities at the 2 higher exposure levels (192). A recent report noted that benzene induced statistically significant cytogenetic effects in blood lymphocytes and micronuclei in bone marrow polychromatic erythrocytes of both rats and mice after 6 hours inhalation of benezene at 1 ppm (710). Micronuclei are chromosomes or small fragments of chromosomes

that are not incorporated into daughter nuclei during cell division. They may be induced by agents that break chromosomes and/or that affect the spindle apparatus.

18.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

Chatburn et al. (193), in evaluating the benzene teratogenicity studies, have concluded that there is no evidence that benzene constitutes a teratogenic hazard. In the studies that were conducted, benzene was administered in concentrations up to 220 ppm by inhalation and 4 mL/kg subcutaneously. The feature common to all the studies was a decrease in maternal weight gain, accompanied by retardation in fetal development.

18.3.1.4 Other Toxicologic Effects

18.3.1.4.1 Short-term Toxicity

Benzene causes central nervous system depression, narcosis and death in various species of animals. An $LC_{50}$ value of 10,000 ppm·7 hr was recorded for the rat (59). Rabbits exposed to vapor concentrations ranging from 35,000 to 45,000 ppm underwent slight anesthesia after 4 minutes. They experienced other CNS effects such as excitation, tremors and loss of pupil reflexes. Death occurred within 22 to 71 minutes (628). Forty percent of rats exposed to 40,000 ppm for five 20-25 minute periods died within 24 hours (202). Benzene does not appear to cause immediate injury at the cellular level; however, oral $LD_{50}$ values in the rat varied from 3.4 to 5.6 mL/kg depending on the age and strain (12). Oral $LD_{50}$ values of 4700 mg/kg and 3800 mg/kg have also been reported for the mouse and rat, respectively (59).

The local effects of benzene liquid or vapor on the eye are slight. In the rabbit eye, it is a moderate irritant, causing conjunctival irritation and transient corneal injury. Fifty percent of rats exposed to vapor concentrations of 50 ppm developed cataracts after more than 600 hours of exposure (19). Application of benzene to the skin of laboratory animals indicates that it can be absorbed to a moderate degree. Absorption through monkey skin after a single application has been estimated to be 0.17% (191).

18.3.1.4.2 Chronic Toxicity

Leukopenia (i.e. reduction of white blood cells) is the most commonly observed effect of chronic benzene administration to laboratory animals. Leukopenia was observed in rats given 132 daily oral doses ranging from 10 to 100 mg/kg bw. The no-effect level for blood changes was determined to be 1 mg/kg bw (210). After inhalation of 17.5 ppm