

135793

# CHROMIUM

## Summary

Chromium is a heavy metal that generally exists in either a trivalent or hexavalent oxidation state. Hexavalent chromium (Cr VI) is rather soluble and is quite mobile in groundwater and surface water. However, in the presence of reducing agents it is rapidly converted to trivalent chromium (Cr III), which is strongly adsorbed to soil components and consequently is much less mobile. A number of salts of hexavalent chromium are carcinogenic in rats. In addition, an increased incidence of lung cancer was seen in workers occupationally exposed to chromium VI. Hexavalent chromium also causes kidney damage in animals and humans. Trivalent chromium is less toxic than hexavalent chromium; its main effect is contact dermatitis in sensitive individuals.

CAS Number: 7440-47-3

Chemical Formula: Cr

IUPAC Name: Chromium

## Chemical and Physical Properties (Metal)

Atomic Weight: 51.996

Boiling Point: 2672°C

Melting Point: 1857 ± 20°C

Specific Gravity: 7.20 at 28°C

Solubility in Water: Insoluble; some compounds are soluble

## Transport and Fate

Hexavalent Cr is quite soluble, existing in solution as a component of a complex anion. It is not sorbed to any significant degree by clays or hydrous metal oxides. The anionic form varies according to pH and may be a chromate, hydrochromate, or dichromate. Because all anionic forms are so soluble, they are quite mobile in the aquatic environment. Cr VI is efficiently

removed by activated carbon and thus may have some affinity for organic materials in natural water. Cr VI is a moderately strong oxidizing agent and reacts with reducing materials to form trivalent chromium. Most Cr III in the aquatic environment is hydrolyzed and precipitates as chromium hydroxide. Sorption to sediments and bioaccumulation will remove much of the remaining Cr III from solution. Cr III is adsorbed only weakly to inorganic materials. Cr III and Cr VI are readily interconvertible in nature depending on microenvironmental conditions such as pH, hardness, and the types of other compounds present. Soluble forms of chromium accumulate if ambient conditions favor Cr VI. Conditions favorable for conversion to Cr III lead to precipitation and adsorption of chromium in sediments.

In air, chromium is associated almost entirely with particulate matter. Sources of chromium in air include windblown soil and particulate emissions from industrial processes. Little information is available concerning the relative amounts of Cr III and Cr VI in various aerosols. Relatively small particles can form stable aerosols and can be transported many miles before settling out.

Cr III tends to be adsorbed strongly onto clay particles and organic particulate matter, but can be mobilized if it is complexed with organic molecules. Cr III present in minerals is mobilized to different extents depending on the weatherability and solubility of the mineral in which it is contained. Hexavalent compounds are not strongly adsorbed by soil components and Cr VI is mobile in groundwater. Cr VI is quickly reduced to Cr III in poorly drained soils having a high content of organic matter. Cr VI of natural origin is rarely found in soils.

### Health Effects

The hexavalent form of chromium is of major toxicological importance in higher organisms. A variety of chromate (Cr VI) salts are carcinogenic in rats and an excess of lung cancer has been observed among workers in the chromate-producing industry. Cr VI compounds can cause DNA and chromosome damage in animals and humans, and Cr (VI) trioxide is teratogenic in the hamster. Inhalation of hexavalent chromium salts causes irritation and inflammation of the nasal mucosa, and ulceration and perforation of the nasal septum. Cr VI also produces kidney damage in animals and humans. The liver is also sensitive to the toxic effects of hexavalent Cr, but apparently less so than the kidneys or respiratory system. Cr III is less toxic than Cr VI; its main effect in humans is a form of contact dermatitis in sensitive individuals.

## Toxicity to Wildlife and Domestic Animals

Chromium is an essential nutrient and is accumulated in a variety of aquatic and marine biota, especially benthic organisms, to levels much higher than in ambient water. Levels in biota, however, usually are lower than levels in the sediments. Passage of chromium through the food chain can be demonstrated. The food chain appears to be a more efficient pathway for chromium uptake than direct uptake from seawater.

Water hardness, temperature, dissolved oxygen, species, and age of the test organism all modify the toxic effects of chromium on aquatic life. Cr III appears to be more acutely toxic to fish than Cr VI; the reverse is true in long term chronic exposure studies.

None of the plants normally used as food or animal feed are chromium accumulators. Chromium absorbed by plants tends to remain primarily in the roots and is poorly translocated to the leaves. There is little tendency for chromium to accumulate along food chains in the trivalent inorganic form. Organic chromium compounds, about which little is known, can have significantly different bioaccumulation tendencies. Little information concerning the toxic effects of chromium on mammalian wildlife and domestic animal species is available.

## Regulations and Standards

### Ambient Water Quality Criteria (USEPA):

Cr VI:

#### Aquatic Life (Proposed Criteria)

##### Freshwater

Acute toxicity: 11 µg/liter  
Chronic toxicity: 7.2 µg/liter

##### Saltwater

Acute toxicity: 1,200 µg/liter  
Chronic toxicity: 54 µg/liter

#### Human Health

Criterion: 50 µg/liter

Cr III:

Aquatic Life (Proposed Criteria)

Freshwater

Acute toxicity:  $e^{(0.819[\ln(\text{hardness})]+3.568)}$   $\mu\text{g/liter}$

Chronic toxicity:  $e^{(0.819 [\ln(\text{hardness})]+0.537)}$   $\mu\text{g/liter}$

Saltwater

The available data are not adequate for establishing criteria.

Human Health

Criterion: 170 mg/liter

CAG Unit Risk for inhalation exposure to CR VI (USEPA):  
41 (mg/kg/day)<sup>1</sup>

National Interim Primary Drinking Water Standard: 50  $\mu\text{g/liter}$

NIOSH Recommended Standards for CR VI: 1  $\mu\text{g/m}^3$  carcinogenic  
25  $\mu\text{g/m}^3$  noncarcinogenic TWA  
50  $\mu\text{g/m}^3$  noncarcinogenic  
(15-min sample)

OSHA Standards: OSHA air standards have been set for several chromium compounds. Most recognized or suspected carcinogenic chromium compounds have ceiling limits of 100  $\mu\text{g/m}^3$ .

ACGIH Threshold Limit Values: Several chromium compounds have TWAs ranging from 0.05 to 0.5  $\text{mg/m}^3$ . Chromite ore processing (chromate), certain water insoluble Cr VI compounds, and chromates of lead and zinc are recognized or suspected human carcinogens and have 0.05  $\text{mg/m}^3$  TWAs.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1980. IARC Monograph on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 23: Some Metals and Metallic Compounds. World Health Organization, Lyon, France

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).  
1975. Criteria for a Recommended Standard--Occupational  
Exposure to Chromium (VI). Washington, D.C. DHEW Publi-  
cation No. (NIOSH) 76-129

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).  
1983. Registry of Toxic Effects of Chemical Substances.  
Data Base. Washington, D.C. October 1983

NATIONAL RESEARCH COUNCIL OF CANADA. 1976. Effects of Chromium  
in the Canadian Environment. Subcommittee on Heavy Metals  
and Certain Other Compounds, Ottawa, Canada. Environmental  
Secretariat Publication No. NRCC 15017

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-  
Related Fate of 129 Priority Pollutants. Washington,  
D.C. December 1979. EPA 440/4-79-029

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient  
Water Quality Criteria for Chromium. Office of Water  
Regulations and Standards, Criteria and Standards Division,  
Washington, D.C. October 1980. EPA 440/5-80-035

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Water  
quality criteria: Request for comments. (Proposed Criteria)  
Fed. Reg. 49:4551-4553

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health  
Effects Assessment for Trivalent Chromium. Environmental  
Criteria and Assessment Office, Cincinnati, Ohio. September  
1984. ECAO-CIN-H035 (Final Draft)

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health  
Effects Assessment for Hexavalent Chromium. Environmental  
Criteria and Assessment Office, Cincinnati, Ohio. September  
1984. ECAO-CIN-H019 (Final Draft)

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Health  
Assessment Document for Dichloromethane (Methylene Chloride).  
Office of Health and Environmental Assessment. Washington,  
D.C. February 1985. EPA 600/8-82/004F

WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics.  
62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

# MERCURY

## Summary

Both organic and inorganic forms of mercury are reported to be teratogenic and embryotoxic in experimental animals. In humans, prenatal exposure to methylmercury has been associated with brain damage. Other major target organs for organic mercury compounds in humans are the central and peripheral nervous system and the kidney. In animals, toxic effects also occur in the liver, heart, gonads, pancreas, and gastrointestinal tract. Inorganic mercury is generally less acutely toxic than organic mercury compounds, but it does affect the central nervous system adversely.

## Background Information

Several forms of mercury, including insoluble elemental mercury, inorganic species, and organic species, can exist in the environment. In general, the mercurous (+1) salts are much less soluble than the more commonly found mercuric (+2) salts. Mercury also forms many stable organic complexes that are generally much more soluble in organic liquids than in water. The nature and solubility of the chemical species that occur in an environmental system depend on the redox potential and the pH of the environment.

CAS Number: 7439-97-6

Chemical Formula: Hg

IUPAC Name: Mercury

## Chemical and Physical Properties (Metal)

Atomic Weight: 200.59

Boiling Point: 356.58°C

Melting Point: -38.87°C

Specific Gravity: 13.5939 at 20°C

Solubility in Water: 81.3 µg/liter at 30°C; some salts and organic compounds are soluble

Solubility in Organics: Depends on chemical species

Vapor Pressure: 0.0012 mm Hg at 20°C

### Transport and Fate

Mercury and certain of its compounds, including several inorganic species and dimethyl mercury, can volatilize to the atmosphere from aquatic and terrestrial sources. Volatilization is reduced by conversion of metallic mercury to complexed species and by deposition of HgS in reducing sediments, but even so atmospheric transport is the major environmental distribution pathway for mercury. Precipitation is the primary mechanism for removal of mercury from the atmosphere. Photolysis is important in the breakdown of airborne mercurials and may be important in some aquatic systems. Adsorption onto suspended and bed sediments is probably the most important process determining the fate of mercury in the aquatic environment. Sorption is strongest into organic materials. Mercury in soils is generally complexed to organic compounds.

Virtually any mercury compound can be remobilized in aquatic systems by microbial conversion to methyl and dimethyl forms. Conditions reported to enhance biomethylation include large amounts of available mercury, large numbers of bacteria, the absence of strong complexing agents, near neutral pH, high temperatures, and moderately aerobic environments. Mercury is strongly bioaccumulated by numerous mechanisms. Methylmercury is the most readily accumulated and retained form of mercury in aquatic biota, and once it enters a biological system it is very difficult to eliminate.

### Health Effects

When administered by intraperitoneal injection, metallic mercury produces implantation site sarcomas in rats. No other studies were found connecting mercury exposure with carcinogenic effects in animals or humans. Several mercury compounds exhibit a variety of genotoxic effects in eukaryotes. In general, organic mercury compounds are more toxic than inorganic compounds. Although brain damage due to prenatal exposure to methylmercury has occurred in human populations, no conclusive evidence is available to suggest that mercury causes anatomical defects in humans. Embryotoxicity and teratogenicity of methylmercury has been reported for a variety of experimental animals. Mercuric chloride is reported to be teratogenic in experimental animals. No conclusive results concerning the teratogenic effects of mercury vapor are available.

In humans, alkyl mercury compounds pass through the blood brain barrier and the placenta very rapidly, in contrast to inorganic mercury compounds. Major target organs are the central and peripheral nervous systems, and the kidney. Methylmercury is particularly hazardous because of the difficulty of eliminating it from the body. In experimental animals, organic mercury compounds can produce toxic effects in the gastrointestinal tract, pancreas, liver, heart, and gonads, with involvement of the endocrine, immunocompetent, and central nervous systems.

Elemental mercury is not highly toxic as an acute poison. However, inhalation of high concentrations of mercury vapor can cause pneumonitis, bronchitis, chest pains, dyspnea, coughing, stomatitis, gingivitis, salivation, and diarrhea. Soluble mercuric salts are highly poisonous on ingestion, with oral LD<sub>50</sub> values of 20 to 60 mg/kg reported. Mercurous compounds are less toxic when administered orally. Acute exposure to mercury compounds at high concentrations causes a variety of gastrointestinal symptoms and severe anuria with uremia. Signs and symptoms associated with chronic exposure involve the central nervous system and include behavioral and neurological disturbances.

#### Toxicity to Wildlife and Domestic Animals

The toxicity of mercury compounds has been tested in a wide variety of aquatic organisms. Although methylmercury appears to be more toxic than inorganic mercuric salts, few acute or chronic toxicity tests have been conducted with it. Among freshwater species, the 96-hour LC<sub>50</sub> values for inorganic mercuric salts range from 0.02 µg/liter for crayfish to 2,000 µg/liter for caddisfly larvae. Acute values for methylmercuric compounds and other mercury compounds are only available for fishes. In rainbow trout, methylmercuric chloride is about ten times more toxic to rainbow trout than mercuric chloride, which is acutely toxic at about 300 µg/liter at 10°C. Methylmercury is the most chronically toxic of the tested compounds, with chronic values for Daphnia magna and brook trout of 1.00 and 0.52 µg/liter, respectively. The acute-chronic ratio for Daphnia magna is 3.2.

Mean acute values for saltwater species range from 3.5 to 1,680 µg/liter. In general, molluscs and crustaceans are more sensitive than fish to the acute toxic effects of mercury. A life-cycle experiment with the mysid shrimp showed that inorganic mercury at a concentration of 1.6 µg/liter significantly influences time of appearance of first brood, time of first spawn, and productivity. The acute-chronic ratio for the mysid shrimp is 2.9.

Chronic dietary exposure of chickens to mercuric chloride at growth inhibitory levels causes immune suppression, with a differential reduction effect on specific immunoglobulins.

### Regulations and Standards

#### Ambient Water Quality Criteria (USEPA):

##### Aquatic Life (Proposed Criteria)

###### Freshwater

Acute toxicity: 1.1 µg/liter  
Chronic toxicity: 0.20 µg/liter

###### Saltwater

Acute toxicity: 1.9 µg/liter  
Chronic toxicity: 0.10 µg/liter

##### Human Health

Criterion: 144 ng/liter

Primary Drinking Water Standard: 0.002 mg/liter

NIOSH Recommended Standard: 0.05 mg/m<sup>3</sup> TWA (inorganic mercury)

OSHA Standard: 0.1 mg/m<sup>3</sup> Ceiling Level

#### ACGIH Threshold Limit Values:

0.01 mg/m<sup>3</sup> TWA (alkyl compounds)  
0.03 mg/m<sup>3</sup> STEL (alkyl compounds)  
0.05 mg/m<sup>3</sup> TWA (vapor)  
0.1 mg/m<sup>3</sup> TWA (aryl and inorganic compounds)

### REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages

BRIDGER, M.A., and THAXTON, J.P. 1983. Humoral immunity in the chicken as affected by mercury. Arch. Environ. Contam. Toxicol. 12:45-49

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).  
1983. Registry of Toxic Effects of Chemical Substances  
Data Base. Washington, D.C. October 1983

SHEPARD, T.H. 1980. Catalog of Teratogenic Agents. 3rd ed.  
Johns Hopkins University Press, Baltimore. 410 pages

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-  
Related Environmental Fate of 129 Priority Pollutants.  
Washington, D.C. December 1979. EPA 440/4-79-029

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient  
Water Quality Criteria for Mercury. Office of Water Regu-  
lations and Standards, Criteria and Standards Division,  
Washington, D.C. October 1980. EPA 440/5-80-058

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Water  
quality criteria; Request for comments. Fed. Reg. 49:  
4551-4553

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health  
Effects Assessment for Mercury. Environmental Criteria  
and Assessment Office, Cincinnati, Ohio. September 1984.  
ECAO-CIN-HO42 (Final Draft)

WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics.  
62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

WORLD HEALTH ORGANIZATION (WHO). 1976. Environmental Health  
Criteria: 1. Mercury. World Health Organization, Geneva.  
131 pages

## CADMIUM

### Summary

Cadmium is a metal that can be present in a variety of chemical forms in wastes or in the environment. Some forms are insoluble in water, but cadmium is relatively mobile in the aquatic environment. Cadmium is carcinogenic in animals exposed by inhalation and may also be in humans. It is uncertain whether it is carcinogenic in animals or humans exposed via ingestion. Cadmium is a known animal teratogen and reproductive toxin. It has chronic effects on the kidney, and background levels of human exposure are thought to provide only a relatively small margin of safety for these effects.

### Background Information

Cadmium is a soft, bluish white metal that is obtained as a by-product from the treatment of the ores of copper, lead, and iron. Cadmium has a valence of +2 and has properties similar to those of zinc. Cadmium forms both organic and inorganic compounds. Cadmium sulfate is the most common salt.

CAS Number: 7440-43-9

Chemical Formula: Cd

IUPAC Name: Cadmium

### Chemical and Physical Properties

Atomic Weight: 112.41

Boiling Point: 765°C

Melting Point: 321°C

Specific Gravity: 8.642

Solubility in Water: Salts are water soluble; metal is insoluble

Solubility in Organics: Variable, based on compound

Vapor Pressure: 1 mm Hg at 394°C

## Transport and Fate

Cadmium is relatively mobile in the aquatic environment compared to other heavy metals (USEPA 1979). It is removed from aqueous media by complexing with organic materials and subsequently being adsorbed to the sediment. It appears that cadmium moves slowly through soil, but only limited information on soil transport is available. Cadmium uptake by plants is not a significant mechanism for depletion of soil accumulations but may be significant for human exposure.

## Health Effects

There is suggestive evidence linking cadmium with cancer of the prostate in humans (USEPA 1980). In animal studies, exposure to cadmium by inhalation caused lung tumors in rats, and exposure by injection produced injection-site sarcomas and/or Leydig-cell tumors (Takenaka 1983, USEPA 1981). An increased incidence of tumors has not been seen in animals exposed to cadmium orally, but four of the five available studies were inadequate by current standards (Clement 1983).

The evidence from a large number of studies on the mutagenicity of cadmium is equivocal, and it has been hypothesized that cadmium is not directly mutagenic but impedes repair (Clement 1983). Cadmium is a known animal teratogen and reproductive toxin. It has been shown to cause renal dysfunction in both humans and animals. Other toxic effects attributed to cadmium include immunosuppression (in animals), anemia (in humans), pulmonary disease (in humans), possible effects on the endocrine system, defects in sensory function, and bone damage. The oral LD<sub>50</sub> in the rat was 225 mg/kg (NIOSH 1983).

## Toxicity to Wildlife and Domestic Animals

Laboratory experiments suggest that cadmium may have adverse effects on reproduction in fish at levels present in lightly to moderately polluted waters.

The acute LC<sub>50</sub> for freshwater fish and invertebrates generally ranged from 100 to 1,000 µg/liter; salmonids are much more sensitive than other organisms (USEPA 1980). Saltwater species were in general 10-fold more tolerant to the acute effects of cadmium. Chronic tests have been performed and show that cadmium has cumulative toxicity and acute-chronic ratios that range of from 66 to 431. Bioconcentration factors were generally less than 1,000 but were as high as 10,000 for some freshwater fish species.

No adverse effects on domestic or wild animals were reported in the studies reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life (Proposed 1984)

Freshwater

Acute toxicity:  $e^{(1.30[\ln(\text{hardness})] - 3.92)}$   $\mu\text{g/liter}$

Chronic toxicity:  $e^{(0.87[\ln(\text{hardness})] - 4.38)}$   $\mu\text{g/liter}$

Saltwater

Acute toxicity: 38  $\mu\text{g/liter}$

Chronic toxicity: 12  $\mu\text{g/liter}$

Human Health

Criterion: 10  $\mu\text{g/liter}$

CAG Unit Risk for inhalation exposure (USEPA): 6.1 (mg/kg/day)<sup>-1</sup>

Interim Primary Drinking Water Standard (USEPA): 10  $\mu\text{g/liter}$

NIOSH Recommended Standards: 40  $\mu\text{g/m}^3$  TWA  
200  $\mu\text{g/m}^3$  /15 min Ceiling Level

OSHA Standards: 200  $\mu\text{g/m}^3$  TWA  
600  $\mu\text{g/m}^3$  Ceiling Level

ACGIH Threshold Limit Values: 50  $\mu\text{g/m}^3$  TWA

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages

CLEMENT ASSOCIATES, INC. 1983. Assessment of the Weight of Evidence for Risk Assessment for Four Selected Toxic Air Pollutants. Report Prepared for the Air Economic Branch, OPRM, U.S. Environmental Protection Agency. May 1983.

FLEISCHER, M., SAROFIM, A.F., FASSETT, D.W., HAMMOND, P., SCHAKKETTE, H.T., NISBET, I.C.T., and EPSTEIN, S. 1974. Environmental impact of cadmium: A review by the panel on hazardous trace substances. Environ. Health Perspect. 7:253-323

- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).  
1983. Registry of Toxic Effects of Chemical Substances.  
Data Base. Washington, D.C. October 1983
- TAKENAKA, S., OLDIGES, H., KONIG, H., HOCHRAINER, D., and  
OBERDORSTER, G. 1983. Carcinogenicity of cadmium chloride  
aerosols in W rats. JNCI 70:367-371
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-  
Related Environmental Fate of 129 Priority Pollutants.  
Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient  
Water Quality Criteria for Cadmium. Office of Water Regu-  
lations and Standards, Criteria and Standards Division,  
Washington, D.C. October 1980. EPA 440/5-80-025
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1981. Health  
Assessment Document for Cadmium. Environment Criteria  
and Assessment Office. Research Triangle Park, North  
Carolina. October 1981. EPA 600/8-81-023
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health  
Effects Assessment for Cadmium. Environmental Criteria  
and Assessment Office, Cincinnati, Ohio. September 1984.  
ECAO-CIN-H038 (Final Draft)
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Health  
Assessment Document for Chloroform. Office of Health  
and Environmental Assessment, Washington, D.C. September  
1985. EPA 600/8-84/004F

# ARSENIC

## Summary

Arsenic is a metal that is present in the environment as a constituent of organic and inorganic compounds; it also occurs in a number of valence states. Arsenic is generally rather mobile in the natural environment, with the degree of mobility dependent on its chemical form and the properties of the surrounding medium. Arsenic is a human carcinogen; it causes skin tumors when it is ingested and lung tumors when it is inhaled. Arsenic compounds are teratogenic and have adverse reproductive effects in animals. Chronic exposure to arsenic is associated with polyneuropathy and skin lesions. It is acutely toxic to some early life stages of aquatic organisms at levels as low as 40 µg/liter.

## Background Information

Arsenic can be found in the environment in any of four valence states (-3, 0, +3, and +5) depending on the pH, Eh, and other factors. It can exist as either inorganic or organic compounds and often will change forms as it moves through the various media. The chemical and physical properties depend on the state of the metalloid. Only the properties of metallic arsenic have been listed; properties of other arsenic compounds are often quite different.

CAS Number: 7440-38-2

Chemical Formula: As

IUPAC Name: Arsenic

## Chemical and Physical Properties

Atomic Weight: 74.91

Boiling Point: 613°C

Melting Point: 817°C

Specific Gravity: 5.72 at 20°C

102017

Solubility in Water: Insoluble; some salts are soluble

### Transport and Fate

In the natural environment, arsenic has four different oxidation states, and chemical speciation is important in determining arsenic's distribution and mobility. Interconversions of the +3 and +5 states as well as organic complexation, are the most important. Arsenic is generally quite mobile in the environment. In the aquatic environment, volatilization is important when biological activity or highly reducing conditions produce arsine or methylarsenics. Sorption by the sediment is an important fate for the chemical. Arsenic is metabolized to organic arsenicals by a number of organisms; this increases arsenic's mobility in the environment. Because of its general mobility, arsenic tends to cycle through the environment. Its ultimate fate is probably the deep ocean, but it may pass through numerous stages before finally reaching the sea.

### Health Effects

Arsenic has been implicated in the production of skin cancer in humans. There is also extensive evidence that inhalation of arsenic compounds causes lung cancer in workers. Arsenic compounds cause chromosome damage in animals, and humans exposed to arsenic compounds have been reported to have an elevated incidence of chromosome aberrations. Arsenic compounds have been reported to be teratogenic, fetotoxic, and embryotoxic in several animal species, and an increased incidence of multiple malformations among children born to women occupationally exposed to arsenic has been reported. Arsenic compounds also cause noncancerous, possibly precancerous, skin changes in exposed individuals. Several cases of progressive polyneuropathy involving motor and sensory nerves and particularly affecting the extremities and myelinated long-axon neurons have been reported in individuals occupationally exposed to inorganic arsenic. Polyneuropathies have also been reported after the ingestion of arsenic-contaminated foods.

### Toxicity to Wildlife and Domestic Animals

Various inorganic forms of arsenic appear to have similar levels of toxicity; they all seem to be much more toxic than organic forms. Acute toxicity to adult freshwater animals occurs at levels of arsenic trioxide as low as 812 µg/liter and at levels as low as 40 µg/liter in early life stages of aquatic organisms. Acute toxicity to saltwater fish occurs at levels around 15 mg/liter, while some invertebrates are affected at much lower levels (508 µg/liter). Arsenic toxicity

does not appear to increase greatly with chronic exposure, and it does not seem that arsenic is bioconcentrated to a great degree.

Arsenic poisoning is a rare but not uncommon toxic syndrome among domestic animals. Arsenic causes hyperemia and edema of the gastrointestinal tract, hemorrhage of the cardiac serosal surfaces and peritoneum, and pulmonary congestion and edema; and it may cause liver necrosis. Information on arsenic toxicity to terrestrial wildlife was not reported in the literature reviewed.

### Regulations and Standards

#### Ambient Water Quality Criteria (USEPA):

##### Aquatic Life

###### Freshwater

Acute toxicity: 440 µg/liter  
Chronic toxicity: No available data

###### Saltwater

Acute toxicity: 508 µg/liter  
Chronic toxicity: No available data

##### Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of arsenic in water are:

<u>Risk</u>	<u>Concentration</u>
10 <sup>-5</sup>	22 ng/liter
10 <sup>-6</sup>	2.2 ng/liter
10 <sup>-7</sup>	0.22 ng/liter

CAG Unit Risk (USEPA): 15 (mg/kg/day)<sup>-1</sup>

National Interim Primary Drinking Water Standard (USEPA):  
50 µg/liter

NIOSH Recommended Standard (air): 2 µg/m<sup>3</sup> Ceiling Level

OSHA Standard (air): 500 µg/m<sup>3</sup> TWA

ACGIH Threshold Limit Value: 200 µg/m<sup>3</sup> (soluble compounds, as As)

## REFERENCES

- AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages
- INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1980. An evaluation of chemicals and industrial processes associated with cancer in humans based on human and animal data. IARC Monographs Volumes 1 to 20. Cancer Res. 40:1-12
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. October 1983
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient Water Quality Criteria for Arsenic. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-021
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health Effects Assessment for Arsenic. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-HO20 (Final Draft)
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Health Assessment Document for Dichloromethane (Methylene Chloride). Office of Health and Environmental Assessment. Washington, D.C. February 1985. EPA 600/8-82/004F
- WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

## POLYCHLORINATED BIPHENYLS

### Summary

Polychlorinated biphenyls (PCBs) are very persistent in the natural environment and are readily bioaccumulated. In humans, exposure to PCBs has been associated with chloracne, impairment of liver function, a variety of neurobehavioral symptoms, menstrual disorders, minor birth abnormalities, and an increased incidence of cancer. Experimental animals exposed to PCBs experienced an increased incidence of cancer; reproductive problems; neurobehavioral degradation; pathological changes in the liver, stomach, skin, and other organs; and suppression of immunological function. PCBs are often contaminated, and these contaminants may be much more toxic than the PCBs themselves.

### Background Information

Polychlorinated biphenyls (PCBs) are complex mixtures of chemicals composed of two connected benzene rings with 1 to 10 chlorine atoms attached. The chemical, physical, and biological properties of these materials depend to a large degree on the amount and location of the chlorine atoms on the two benzene rings of each specific PCB and on the particular mixture of individual chlorobiphenyls that comprise the mixture.

CAS Number: 1336-36-3

Chemical Formula:  $C_6H_5Cl_x C_6H_5Cl_x$

IUPAC Name: Specific for each polychlorinated biphenyl

Important Synonyms and Trade Names: PCBs, chlorinated biphenyls, polychlorobiphenyls, Aroclor, Kanechlor, Clophen

### Chemical and Physical Properties

Molecular Weight: 189-399\*

Boiling Point: 267°C and up\*

Melting Point: 54-310°C\*

---

\*Increases with increasing chlorination.

102021

Polychlorinated biphenyls

Page 1  
October 1985

Specific Gravity: 1.3 to 1.5 at 20°C\*

Solubility in Water: 0.003-0.6 mg/liter

Solubility in Organics: Soluble in most common organic solvents

Log Octanol/Water Partition Coefficient: 4-6\*

Vapor Pressure:  $10^{-3}$ - $10^{-5}$  mm Hg at 20°C\*\*

Henry's Law Constant:  $10^{-3}$  to  $10^{-5}$  atm m<sup>3</sup>/mole

### Transport and Fate

The transport and fate of polychlorinated biphenyls has been studied extensively, and although individual chemicals vary in the rates at which processes occur, some generalizations can be made about PCBs as a class. PCBs are relatively inert, and therefore persistent, compounds, with low vapor pressures, low water solubility, and high log octanol/water partition coefficients. Despite their low vapor pressures, they have a high activity coefficient in water, which causes a higher rate of volatilization than might normally be expected. Volatilization and persistence account for the ubiquitous nature of PCBs in the environment. Adsorption to the organic material in soil or sediments is probably the major fate of at least the more heavily chlorinated PCBs. Once bound, the PCBs may persist for years with slow desorption providing continuous, low-level exposure to the surrounding locality. Bioaccumulation of PCBs also occurs, with most of the compound stored in the adipose tissue of the body. PCBs are degraded primarily by two routes. Less heavily chlorinated PCBs (mainly the mono-, di-, and trichlorinated PCBs) can be biodegraded by some soil microorganisms. PCBs with five or more chlorines are not measurably biodegraded. These heavier PCBs can be photolyzed by ultraviolet light. This process is extremely slow, but it may be the most important degradation process for these very persistent compounds.

Assessing the toxicity of PCBs is complicated by the fact that several different mixtures have been produced and distributed commercially and by the presence of highly toxic contaminants in some commercial mixtures. Some of these contaminants can be formed by combustion of PCBs or even by high-temperature treatment in service, so that used materials may be more toxic than the commercial mixtures whose toxicity has been studied.

---

\*Increases with increasing chlorination.

\*\*Decreases with increasing chlorination.

## Health Effects

In humans exposed to PCBs (in the workplace or via accidental contamination of food), reported adverse effects include chloracne (a long-lasting, disfiguring skin disease), impairment of liver function, a variety of neurobehavioral and affective symptoms, menstrual disorders, minor birth abnormalities, and probably increased incidence of cancer. Animals experimentally exposed to PCBs have shown most of the same symptoms, as well as impaired reproduction; pathological changes in the liver, stomach, skin, and other organs; and suppression of immunological functions. PCBs are carcinogenic in rats and mice and, in appropriate circumstances, enhance the effects of other carcinogens. Reproductive and neurobiological effects of PCBs have been reported in rhesus monkeys at the lowest dose level tested, 11 µg/kg body weight/day over a period of several months.

## Toxicity to Wildlife and Domestic Animals

Polychlorinated biphenyls are bioaccumulated and can be biomagnified. Therefore, their toxicity increases with length of exposure and position of the exposed species on the food chain. The toxicity of the various PCB mixtures is also dependent on their composition. Because of the complexity of PCB toxicity, only general effects will be discussed here.

The 96-hour  $LC_{50}$  values for rainbow trout, bluegills, and channel catfish were around 20 mg/liter. The same species exposed for 10 to 20 days had  $LC_{50}$  values of about 0.1 mg/liter. Invertebrate species were also adversely affected, with some species having 7-day  $LC_{50}$  values as low as 1 µg/liter. In general, juvenile organisms appeared more susceptible to the effects of PCBs than either eggs or adults.

Three primary ways in which PCBs can affect terrestrial wildlife are outright mortality, adversely affecting reproduction, and changing behavior. PCB doses greater than 200 ppm in the diet or 10 mg/kg body weight (bw) caused some mortality in sensitive bird species exposed for several days. Doses around 1,500 ppm (diet) or about 100 mg/kg (bw) caused extensive mortality in these sensitive species. They generally caused some mortality in all species, with the level being dependent on the length of exposure and the particular PCB mixture. Some mammalian species are especially susceptible to PCBs. For example, mink died when fed as little as 5 ppm in the diet (equivalent to less than 1 mg/kg bw/day). PCBs caused lower egg production; deformities; decreased hatchability, growth, and survival; and some eggshell thinning in reproductive studies on chickens fed doses of 20 ppm in the diet (1 mg/kg bw). Mink fed 1 ppm in the diet (0.2 mg/kg bw) had lower reproductive success, and there are indications that an increased incidence

of premature births in some marine mammals was linked to PCB exposure. Behavioral effects on wildlife include increased activity, decreased avoidance response, and decreased nesting, all of which could significantly influence survival in the wild.

No toxic effects on domestic animals other than chickens were reported in the sources reviewed, but susceptible species would probably be affected in a similar manner to laboratory animals and wildlife.

### Regulations and Standards

#### Ambient Water Quality Criteria (USEPA):

##### Aquatic Life

###### Freshwater

Acute toxicity: 2 µg/liter  
Chronic toxicity: 0.014 µg/liter

###### Saltwater

Acute toxicity: 10 µg/liter  
Chronic toxicity: 0.030 µg/liter

##### Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of PCBs in water are:

<u>Risk</u>	<u>Concentration</u>
10 <sup>-5</sup>	0.79 ng/liter
10 <sup>-6</sup>	0.079 ng/liter
10 <sup>-7</sup>	0.0079 ng/liter

CAG Unit Risk (USEPA): 4.34 (mg/kg/day)<sup>-1</sup>

NIOSH Recommended Standard: 1.0 µg/m<sup>3</sup> TWA

ACGIH Threshold Limit Value: 0.5 mg/m<sup>3</sup> TWA

### REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH).  
1980. Documentation of the Threshold Limit Values. 4th  
ed. Cincinnati, Ohio. 488 pages

- INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC).** -1978.  
IARC Monographs on the Evaluation of Carcinogenic Risk  
of Chemicals to Humans. Vol. 18: Polychlorinated Biphenyls  
and Polybrominated Biphenyls. World Health Organization,  
Lyon, France. Pp. 43-103
- NATIONAL ACADEMY OF SCIENCES (NAS).** 1977. Drinking Water  
and Health. Safe Drinking Water Committee, Washington, D.C.  
939 pages
- ROBERTS, J.R., RODGERS, D.W., BAILEY, J.R., and RORKE, M.A.**  
1978. Polychlorinated Biphenyls: Biological Criteria  
for an Assessment of their Effects on Environmental Quality.  
National Research Council of Canada, Ottawa, Canada.  
NRCC No. 16077
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA).** 1976. National  
Conference on Polychlorinated Biphenyls (November 19-21,  
1975, Chicago, Illinois). Office of Toxic Substances,  
Washington, D.C. March 1976. EPA 560/6-75-004
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA).** 1979. Water-  
Related Environmental Fate of 129 Priority Pollutants.  
Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA).** 1980. Ambient  
Water Quality Criteria for Polychlorinated Biphenyls (PCBs).  
Office of Water Regulations and Standards, Criteria and  
Standards Division, Washington, D.C. October 1980. EPA  
440/5-80-054
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA).** 1984. Health  
Effects Assessment for Polychlorinated Biphenyls. Environmental  
Criteria and Assessment Office, Cincinnati, Ohio. September  
1984. ECAO-CIN-H004 (Final Draft)
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA).** 1985. Health  
Assessment Document for Dichloromethane (Methylene Chloride).  
Office of Health and Environmental Assessment. Washington,  
D.C. February 1985. EPA 600/8-82/004F

# ZINC

## Summary

Ingestion of excessive amounts of zinc can cause fever, vomiting, and stomach cramps. Zinc oxide fumes can cause metal fume fever. Inhalation of mists or fumes may irritate the respiratory tract, and contact with zinc chloride may irritate the eyes and skin. High levels of zinc in the diet have been shown to retard growth and produce defective mineralization of bone.

## Background Information

Zinc generally exists in nature as a salt with a valence of +2, although it is also found in four other stable valences.

CAS Number: 7440-66-6

Chemical Formula: Zn

IUPAC Name: Zinc

## Chemical and Physical Properties

Atomic Weight: 65.38

Boiling Point: 907°C

Melting Point: 419.58°C

Specific Gravity: 7.133 at 25°C

Solubility in Water: Insoluble; some salts are soluble

Solubility in Organics: Soluble in acid and alkali

Vapor Pressure: 1 mm Hg at 487°C

## Transport and Fate

Zinc can occur in both suspended and dissolved forms. Dissolved zinc may occur as the free (hydrated) zinc ion or as dissolved complexes and compounds with varying degrees of stability and toxicity. Suspended (undissolved) zinc may be dissolved following minor changes in water chemistry or may be sorbed to suspended matter. The predominant fate of zinc

in aerobic aquatic systems is sorption of the divalent cation by hydrous iron and manganese oxides, clay minerals, and organic material. The efficiency of these materials in removing zinc from solution varies according to their compositions and concentrations; the pH and salinity of the water; the concentrations of complexing ligands; and the concentration of zinc. Concentrations of zinc in suspended and bed sediments always exceed concentrations in ambient water. In reducing environments, precipitation of zinc sulfide limits the mobility of zinc. However, under aerobic conditions, precipitation of zinc compounds is probably important only where zinc is present in high concentrations. Zinc tends to be more readily sorbed at higher pH than lower pH and tends to be desorbed from sediments as salinity increases. Compounds of zinc with the common ligands of surface waters are soluble in most neutral and acidic solutions, so that zinc is readily transported in most unpolluted, relatively organic-free waters.

The relative mobility of zinc in soil is determined by the same factors affecting its transport in aquatic systems. Atmospheric transport of zinc is also possible. However, except near sources such as smelters, zinc concentrations in air are relatively low and fairly constant.

Since it is an essential nutrient, zinc is strongly bioaccumulated even in the absence of abnormally high ambient concentrations. Zinc does not appear to be biomagnified. Although zinc is actively bioaccumulated in aquatic systems, the biota appear to represent a relatively minor sink compared to the sediments. Zinc is one of the most important metals in biological systems. Since it is actively bioaccumulated, the environmental concentrations of zinc probably exhibit seasonal fluctuations.

### Health Effects

Testicular tumors have been produced in rats and chickens when zinc salts are injected intratesticularly, but not when other routes of administration are used. Zinc may be indirectly important with regard to cancer since its presence seems to be necessary for the growth of tumors. Laboratory studies suggest that although zinc-deficient animals may be more susceptible to chemical induction of cancer, tumor growth is slower in these animals. There is no evidence that zinc deficiency has any etiological role in human cancer. There are no data available to suggest that zinc is mutagenic or teratogenic in animals or humans.

Zinc is an essential trace element that is involved in enzyme functions, protein synthesis, and carbohydrate metabolism. Ingestion of excessive amounts of zinc may cause fever, vomiting,

stomach cramps, and diarrhea. Fumes of freshly formed zinc oxide can penetrate deep into the alveoli and cause metal fume fever. Zinc oxide dust does not produce this disorder. Contact with zinc chloride can cause skin and eye irritation. Inhalation of mists or fumes may irritate the respiratory and gastrointestinal tracts. Zinc in excess of 0.25% in the diet of rats causes growth retardation, hypochromic anemia, and defective mineralization of bone. No zinc toxicity is observed at dietary levels below 0.25%.

Studies with animals and humans indicate that metabolic changes may occur due to the interaction of zinc and other metals in the diet. Exposure to cadmium can cause changes in the distribution of zinc, with increases in the liver and kidneys, organs where cadmium also accumulates. Excessive intake of zinc may cause copper deficiencies and result in anemia. Interaction of zinc with iron or lead may also lead to changes that are not produced when the metals are ingested individually.

#### Toxicity to Wildlife and Domestic Animals

Zinc produces acute toxicity in freshwater organisms over a range of concentrations from 90 to 58,100 µg/liter and appears to be less toxic in harder water. Acute toxicity is similar for freshwater fish and invertebrates. Chronic toxicity values range from 47 to 852 µg/liter and appear to be relatively unaffected by hardness. A final acute-chronic ratio for freshwater species of 3.0 has been reported. Although most freshwater plants appear to be insensitive to zinc, one species, the alga Selenastrum capricornutum, exhibited toxic effects at concentrations from 30 to 700 µg/liter. Reported acute toxicity values range from 2,730 to 83,000 µg/liter for saltwater fish and from 166 to 55,000 µg/liter for invertebrate saltwater species. Zinc produces chronic toxicity in the mysid shrimp at 166 µg/liter. The final acute-chronic ratio for saltwater species is 3.0. Toxic effects are observed in saltwater plant species at zinc concentrations of 50 to 25,000 µg/liter. Bio-concentration factors of edible portions of aquatic organisms range from 43 for the soft-shell clam to 16,700 for the oyster.

Zinc poisoning has occurred in cattle. In one outbreak, poisoning was caused by food accidentally contaminated with zinc at a concentration of 20 g/kg. An estimated intake of 140 g of zinc per cow per day for about 2 days was reported. The exposed cows exhibited severe enteritis, and some died or had to be slaughtered. Postmortem findings showed severe pulmonary emphysema with changes in the myocardium, kidneys, and liver. Zinc concentrations in the liver were extremely high. Based on relatively limited data, some researchers have speculated that exposure to excessive amounts of zinc may

constitute a hazard to horses. Laboratory studies and findings in foals living near lead-zinc smelters suggest that excessive exposure to zinc may produce bone changes, joint afflictions, and lameness. In pigs given dietary zinc at concentrations greater than 1,000 mg/kg, decreased food intake and weight gain were observed. At dietary levels greater than 2,000 mg/kg, deaths occurred as soon as 2 weeks after exposure. Severe gastrointestinal changes and brain damage, both of which were accompanied by hemorrhages, were observed, as well as changes in the joints. High concentrations of zinc were found in the liver.

### Regulations and Standards

#### Ambient Water Quality Criteria (USEPA):

*'87 FR  
update*

##### Aquatic Life

###### Freshwater

Acute toxicity:  $e^{(0.83[\ln(\text{hardness})] + 1.95)}$   $\mu\text{g/liter}$   
Chronic toxicity: 47  $\mu\text{g/liter}$

###### Saltwater

Acute toxicity: 170  $\mu\text{g/liter}$   
Chronic toxicity: 58  $\mu\text{g/liter}$

##### Human Health

Organoleptic criterion: 5 mg/liter

Secondary Drinking Water Standard: 5 mg/liter

NIOSH Recommended Standard: 5  $\text{mg/m}^3$  (zinc oxide)

OSHA Standard: 5  $\text{mg/m}^3$  TWA (zinc oxide)

#### ACGIH Threshold Limit Values:

Zinc chloride fume:	1 $\text{mg/m}^3$ TWA
	2 $\text{mg/m}^3$ STEL
Zinc oxide fume:	5 $\text{mg/m}^3$ TWA
	10 $\text{mg/m}^3$ STEL
Zinc oxide dust:	10 $\text{mg/m}^3$ TWA (nuisance particulate)
Zinc stearate:	10 $\text{mg/m}^3$ TWA (nuisance particulate)
	20 $\text{mg/m}^3$ STEL

## REFERENCES

- AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages
- CASARETT, L.J., and DOULL, J., eds. 1975. Toxicology: The Basic Science of Poisons. Macmillan Publishing Co., New York. 768 pages
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. October 1983
- SAX, N.I. 1975. Dangerous Properties of Industrial Materials. 4th ed. Van Nostrand Reinhold Co., New York. 1,258 pages
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient Water Quality Criteria for Zinc. Office of Water Quality and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-079
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health Effects Assessment for Zinc. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-HO48
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Health Assessment Document for Dichloromethane (Methylene Chloride). Office of Health and Environmental Assessment. Washington, D.C. February 1985. EPA 600/8-82/004F
- WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

102030

# PHENOL

## Summary

When applied to the skin of mice, phenol appears to have some tumor-promoting effects and may be a weak carcinogen. There is equivocal evidence that phenol is mutagenic. Subchronic exposure to phenol caused liver, kidney, lung, and heart damage in experimental animals. In humans, phenol has been shown to irritate the eyes, nose, and throat.

CAS Number: 108-95-2

Chemical Formula:  $C_6H_5OH$

IUPAC Name: Phenol

## Chemical and Physical Properties

Molecular Weight: 94.11

Boiling Point: 181.75°C

Melting Point: 43°C

Specific Gravity: 1.0576 at 20°C

Solubility in Water: 93,000 mg/liter at 25°C

Solubility in Organics: Soluble in alcohol, chloroform, and carbon disulfide; very soluble in ether; miscible with carbon tetrachloride and hot benzene

Log Octanol/Water Partition Coefficient: 1.46

Vapor Pressure: 0.3513 mm Hg at 25°C

Vapor Density: 3.24

pKa: 10.02

Flash Point: 85°C (closed cup)

## Transport and Fate

Photooxidation may be an important degradative process, especially in aerated, clear, surface waters. Phenol may also be nonphotolytically oxidized in highly aerated waters that contain iron and copper in solution or as part of the suspended particulates. The relatively low log octanol/water partition coefficient of phenol, as well as the available experimental evidence, suggest that sorption and bioaccumulation are not important environmental fate processes. Biodegradation can be a significant fate pathway in aquatic systems and soil when significant concentrations of microorganisms are present. In addition to microorganisms, at least one species of fish is reported to be able to biotransform phenol.

The dominance of photooxidation, metal-catalyzed oxidation, or biodegradation as destructive pathways depends on the particular environmental conditions, but the degradation products are similar for all fate pathways. The first step usually involves further hydroxylation of the aromatic ring, followed by oxidation to benzoquinone and cleavage of the ring structure. There is a possibility that phenol present in surface waters can volatilize into the atmosphere. However, since this phenol would be rapidly photooxidized in the troposphere, any significant atmospheric transport is unlikely.

## Health Effects

Phenol appears to have tumor-promoting activity in many strains of mice when repeatedly applied to the shaved skin after initiation with known carcinogens. Although there is equivocal evidence that phenol may be weakly carcinogenic when applied to the skin of one sensitive strain of mice, it does not appear to be carcinogenic when applied to the skin of standard strains of mice. NCI reported that phenol was not carcinogenic when administered in drinking water to rats and mice. There is equivocal evidence that phenol may have mutagenic effects, although further evaluation is needed. There are no reports of teratogenic effects caused by exposure to phenol.

Subchronic inhalation exposure to phenol is reported to cause liver, kidney, lung, and heart damage in guinea pigs. Slight liver and kidney damage was seen in rats exposed by gavage to 100 mg/kg/day for 20 days. The oral and skin LD<sub>50</sub>s for the rat are 414 and 669 mg/kg, respectively, and the inhalation LC<sub>50</sub> is 316 mg/m<sup>3</sup>. Phenol is an eye, nose, and throat irritant and can cause systemic damage to the nervous system in humans following dermal, oral, or inhalation exposure.

## Toxicity to Wildlife and Domestic Animals

The acute toxicity of phenol to freshwater species is expressed over a range of 2 to 3 orders of magnitude. Acute values for fish species range from 5,020 µg/liter for juvenile rainbow trout to 67,500 µg/liter for the fathead minnow. The acute value for the rainbow trout, and a value of 5,000 µg/liter for Daphnia magna are the lowest acute values observed. An early life stage test on the fathead minnow resulted in a chronic value of 2,560 µg/liter, with an acute-chronic ratio of 14. Median effect concentrations for oyster and clam embryos are approximately 55,000 µg/liter. For the grass shrimp and the mountain bass, LC<sub>50</sub> values of 5,800 and 11,000 µg/liter, respectively, are reported. No chronic effects are available for saltwater species. Reported bioconcentration factors of 1.2 to 2.3 for goldfish suggest that no residue problem should occur from exposure to phenol. No appropriate data concerning effects of phenol on other wildlife or domestic animals are available.

## Regulations and Standards

### Ambient Water Quality Criteria (USEPA):

#### Aquatic Life

The available data are not adequate for establishing criteria. However, the lowest concentrations of phenol known to cause toxic effects in aquatic organisms are:

#### Freshwater

Acute toxicity: 10,200 µg/liter  
Chronic toxicity: 2,560 µg/liter

#### Saltwater

Acute toxicity: 5,800 µg/liter  
Chronic toxicity: No available data

#### Human Health

Health criterion: 3.5 mg/liter  
Organoleptic criterion: 0.3 mg/liter

NIOSH Recommended Standards: 20 mg/m<sup>3</sup> TWA  
60 mg/m<sup>3</sup>/15 min Ceiling Level

OSHA Standard: 19 mg/m<sup>3</sup>

ACGIH Threshold Limit Values: 19 mg/m<sup>3</sup> TWA  
38 mg/m<sup>3</sup> STEL

Department of Transportation: Poison

#### REFERENCES

- AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages
- NATIONAL CANCER INSTITUTE (NCI). 1980. Bioassay of Phenol for Possible Carcinogenicity. CAS No. 108-95-2. NCI Carcinogenesis Technical Report Series No. 203. Washington, D.C. USDHHS Publication No. (NTP) 80-15
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. October 1983
- SAX, N.I. 1975. Dangerous Properties of Industrial Materials. 4th ed. Van Nostrand Reinhold Co., New York. 1,258 pages
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient Water Quality Criteria for Phenol. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-066
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health Effects Assessment for Phenol. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-H007 (Final Draft)
- WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages