

## Sources of Exposure

## Toxicokinetics and Normal Human Levels

## Biomarkers/Environmental Levels

### General Populations

- The general population is not likely to be exposed to large amounts of hexachlorobenzene, but some exposure is likely in small amounts from low-level contamination of food.
- Populations that live near an industrial site where hexachlorobenzene is produced as a by-product or who live near a hazardous waste site where hexachlorobenzene has been discarded may be exposed.
- Contact with contaminated soil or dust particles or inhaling contaminated air are other potential sources of exposure.

### Occupational Populations

- Hexachlorobenzene is not currently manufactured as a commercial product in the United States. However, occupational exposure is possible for workers involved in the production of chlorinated hydrocarbons, which releases hexachlorobenzene as a by-product.
- Farmers may also be more susceptible to hexachlorobenzene exposure because of its past use as a pesticide.

### Toxicokinetics

- Hexachlorobenzene is poorly absorbed through the inhalation route in humans, but is moderately absorbed when animals are orally exposed, though gastrointestinal absorption is variable.
- Orally exposed hexachlorobenzene is distributed widely in mammalian tissues and organs, especially in adipose tissues.
- Hexachlorobenzene is slowly metabolized in mammals. Reductive dechlorination appears to be an important metabolic pathway forming metabolites including pentachlorophenol and pentachlorobenzene (in humans)
- Ingested hexachlorobenzene is excreted mostly in the feces as unchanged hexachlorobenzene, and to a lesser extent in urine as metabolites.

### Normal Human Levels

- NHANES II found the median level in blood from the general population (collected from 1976–1980) to be 1.7 ppb.

### Biomarkers

- Hexachlorobenzene levels in serum have been correlated with fecal levels, as well as length of breast-feeding and infant serum levels; however, there are insufficient data to correlate either biomarker with exposure levels.
- Porphyria is the primary biomarker of effect from human exposure to hexachlorobenzene, although it is not specific to hexachlorobenzene exposure.

### Environmental Levels

#### *Air*

- Hexachlorobenzene is usually not detected in ambient air samples except at very low concentrations.

#### *Sediment and Soil*

- No recent data are available on current concentrations in soil; however, the occurrence of hexachlorobenzene residues in agricultural soils was associated with hexachlorobenzene's registered pesticide uses rather than general environmental contamination.

#### *Water*

- No recent data are available on current concentrations in water.

### Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. Toxicological Profile for Hexachlorobenzene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

# ToxGuide™ for Hexachlorobenzene



CAS# 118-74-1

September 2002

U.S. Department of Health and  
Human Services  
Public Health Service  
Agency for Toxic Substances  
and Disease Registry  
[www.atsdr.cdc.gov](http://www.atsdr.cdc.gov)

**Contact Information:**  
Division of Toxicology  
and Environmental Medicine  
Applied Toxicology Branch

1600 Clifton Road NE, F-62  
Atlanta, GA 30333  
1-800-CDC-INFO  
1-800-232-4636



## Chemical and Physical Information

## Routes of Exposure

## Relevance to Public Health (Health Effects)

### Hexachlorobenzene is a Crystalline Solid

- Hexachlorobenzene is a crystalline solid that is practically insoluble in water.
- Hexachlorobenzene is a chlorinated hydrocarbon industrial chemical that is no longer manufactured, but is formed as a waste product when producing several chlorinated hydrocarbons.
- When heated to decomposition, it emits toxic fumes of chlorides.

- Inhalation (breathing) – Minor route of exposure for general population. Predominant route of occupational exposure.
- Oral – Predominant route of exposure via ingestion of low-level contaminated food.
- Dermal (skin) – Minor route of exposure for the general population.

### Hexachlorobenzene in the Environment

- Hexachlorobenzene is very persistent in the environment due to its chemical stability and resistance to biodegradation.
- In soil, the half life is 3–6 years, in surface water the half life is 2.7–5.7 years, and in groundwater the half life is 5.3–11.4 years.
- Because of its low solubility in water, hexachlorobenzene settles in particles on the bottom of lakes and rivers.
- Evaporation into the air is not significant under normal ambient conditions.

Health effects are determined by the dose (how much), the duration (how long), and the route of exposure.

### Minimal Risk Levels (MRLs)

#### *Inhalation*

- No MRLs were derived for acute, intermediate, or chronic-duration inhalation exposure.

#### *Oral*

- An MRL of 0.008 mg/kg/day has been derived for acute-duration oral exposure ( $\leq 14$  days).
- An MRL of 0.0001 mg/kg/day has been derived for intermediate-duration oral exposure ( $\leq 15$ –364 days).
- An MRL of 0.00005 mg/kg/day has been derived for chronic-duration oral exposure ( $\geq 1$  year).

### Health Effects

- The primary effects associated with exposure to hexachlorobenzene hepatic, reproductive, and developmental toxicity and carcinogenesis.

### Health Effects (continued)

- The most common hepatic exposure related effect is porphyria.
- Reproductive performance of rats has been affected at low doses, with the ovaries being identified as a target organ.
- Exposure-related developmental effects include impaired neurological development, cleft palate, renal agenesis and minor skeletal abnormalities.
- DHHS, IARC, and EPA consider hexachlorobenzene to be a probable human carcinogen.

### Children's Health

- Maternal hexachlorobenzene can be transferred to the fetus through the placenta, and nursing children may be exposed through breast milk where hexachlorobenzene can be concentrated.
- Infants and young children are at increased risk from exposure to hexachlorobenzene compared to adults. Orally exposed infants had a high rate of mortality associated with dermal lesions. Adolescents exhibited exposure-related effects more frequently than adults. Exposure-related neurological effects were also seen in children but not in adults.