APPENDIX W

AGENCY FOR TOXIC SUBSTANCES & DISEASE REGISTRY (ATSDR) TOXGUIDES

INTRODUCTION

Where available, the Agency for Toxic Substances and Disease Registry (ASTDR) ToxGuides™ are provided for the COCs and COPCs identified in the Shannon Road/El Camino del Cerro Final Remedial Investigation Report. These documents were downloaded from the ASTDR website (http://www.atsdr.cdc.gov/).

The ATSDR ToxGuides™ are quick reference guides providing information such as chemical and physical properties, sources of exposure, routes of exposure, minimal risk levels, children's health, and health effects. The ToxGuides™ also discuss how the substance might interact in the environment. The ToxGuides™ were developed by the ATSDR Division of Toxicology and and Human Health Sciences. Information is excerpted from the corresponding toxicological profiles.

The ToxGuide™ is developed to be used as a pocket guide. Tear off at perforation and fold along lines.

Sources of Exposure

General Populations

- Contaminated air and water are the most important sources of exposure to tetrachloroethylene.
- Tetrachloroethylene in contaminated soil can migrate to indoor air through subsurface air spaces, including cracks in foundations.
- Dermal exposure can occur from contact with contaminated water such as during showering.
- Tetrachloroethylene readily volatilizes from shower and bath water and can be inhaled.
- Exposure to tetrachloroethylene may result from living in proximity to sites where it is produced or waste sites comtaminated with the chemical.
- Using tetrachloroethylene-containing products such as printing ink, glues, sealants, polishes, lubricants, and silicones can result in exposure.
- Close proximity to clothing that was dry cleaned using tetrachloroethylene may lead to exposure of adults and children.

Occupational Populations

- Workers involved in the manufacture of tetrachloroethylene.
- Workers using degreasers and other products containing tetrachloroethylene.
- Workers in the dry cleaning industry.

Toxicokinetics and Normal Human Levels

Toxicokinetics

- Tetrachloroethylene is readily absorbed from the lung, gastrointestinal tract, and skin.
- Tetrachloroethylene is widely distributed throughout the body via the blood, regardless of the route of exposure; relatively high concentrations are found in fat, liver, and kidney.
- Metabolism of absorbed tetrachloroethylene is thought to occur mainly in the liver, lung, and kidney. Metabolism results in variety of metabolites, some of which may be toxic.
- Most absorbed tetrachloroethylene is excreted unchanged in the exhaled air regardless of route of exposure. Tetrachloroethylene metabolites (primarily trichloroacetic acid in humans) are mainly excreted in the urine.

Normal Human Levels

- Tetrachloroethylene levels in blood of the general population generally range from below the instrumentation detection limit to as much as 0.23 parts per billion; blood levels in people living in rural areas are lower than those in people living in urban and industrial areas.

Biomarkers/Environmental Levels

Biomarkers

- Tetrachloroethylene can be measured in exhaled air, blood, or urine. However, tetrachloroethylene is rapidly metabolized to other substances which are not specific to tetrachloroethylene exposure.

Environmental Levels

Air

- Tetrachloroethylene concentrations across the United States are generally less than 0.15 ppb, but may be higher near tetrachloroethylene-contaminated sites.

Water

- Tetrachloroethylene concentrations in drinking water in the United States are generally less than 5 ppb, but average levels of 17-28 ppb were measured in some California drinking water sources.

Food

- Tetrachloroethylene can migrate from air to food and concentrate to relatively high levels, particularly in areas where tetrachloroethylene air levels are high.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2014. Toxicological Profile for Tetrachloroethylene (Draft for Public Comment). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

C_2Cl_4

ToxGuide™ for Tetrachloroethylene

C_2Cl_4

CAS# 127-18-4

October 2014

U.S. Department of Health and Human Services
Public Health Service
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Chemical and Physical Information

Tetrachloroethylene is a Liquid

- Tetrachloroethylene is a colorless liquid with an ethereal odor.
- Tetrachloroethylene is used commercially as a dry cleaning agent, metal degreaser, and as a chemical intermediate.
- Tetrachloroethylene is an excellent extraction solvent for greases, oils, fats, and waxes.
- Tetrachloroethylene is used to scour fabrics during production, and as a solvent in waterless drying and finishing operations.

Routes of Exposure

- Inhalation – Significant route of exposure for workers in the dry cleaning industry; indoor and outdoor air exposure occurs among the general population.
- Oral – Important route of exposure for the general population through ingestion of contaminated drinking water.
- Dermal – Potential route of exposure, particularly among workers who handle substances with tetrachloroethylene.

Tetrachloroethylene in the Environment

- In air, tetrachloroethylene breaks down slowly and can travel long distances. Levels in air depend on location; and they are usually higher in industrial and populated areas.
- Tetrachloroethylene readily partitions from surface soil and water to air. When it leaches deeper into subsurface soil, it is not readily degraded.
- Tetrachloroethylene can be slowly broken down in air by photochemical reactions.
- In water, tetrachloroethylene volatilizes to air more readily than it undergoes photooxidation or hydrolysis.
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- Tetrachloroethylene can migrate from surface soil to indoor air through cracks in foundations, etc.
- Tetrachloroethylene has a low tendency to bioaccumulate.

Relevance to Public Health (Health Effects)

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

Minimal Risk Levels (MRLs)

Inhalation

- An MRL of 0.006 ppm has been derived for acute-duration (≤14 days) inhalation exposure to tetrachloroethylene.
- An MRL of 0.006 ppm has been derived for intermediate-duration (15-364 days) inhalation exposure to tetrachloroethylene.
- An MRL of 0.006 ppm has been derived for chronic-duration (≥365 days) inhalation exposure to tetrachloroethylene.

Oral

- An MRL of 0.008 mg/kg/day has been derived for acute-duration (≤14 days) oral exposure to tetrachloroethylene.
- An MRL of 0.008 mg/kg/day has been derived for intermediate-duration (15-364 days) oral exposure to tetrachloroethylene.
- An MRL of 0.008 mg/kg/day has been derived for chronic-duration (≥365 days) oral exposure to tetrachloroethylene.

Health Effects

- The main targets of tetrachloroethylene toxicity include the central nervous system, kidney, liver, reproductive system, and developing fetus.
- Available human data provide suggestive evidence for tetrachloroethylene-induced bladder cancer, multiple myeloma, and non-Hodgkin's lymphoma.
- U.S. Environmental Protection Agency (EPA) concluded that tetrachloroethylene is likely to be carcinogenic to humans by all routes of exposure based on sufficient evidence in animals and suggestive evidence in humans. The International Agency for Research on Cancer (IARC) concluded that tetrachloroethylene is probably carcinogenic to humans based on sufficient evidence in animals and limited evidence in humans.

Children's Health

- Children exposed to tetrachloroethylene are expected to experience effects similar to those seen in poisoned adults.
- Tetrachloroethylene has been detected in goat's milk, which indicates that it can be transferred by nursing.
- Results of some human studies suggest possible associations between *in utero* and early postnatal exposure and selected developmental abnormalities. Exposure of pregnant animals resulted in developmental abnormalities in the offspring.

The ToxGuide™ is developed to be used as a pocket guide. Tear off at perforation and fold along lines.

Sources of Exposure

General Populations

- Contaminated air and water are the most important sources of exposure to trichloroethylene.
- Indoor air may contain trichloroethylene that has migrated from contaminated soil indoors through cracks in foundations, etc.
- Trichloroethylene readily enters the air from water, including contaminated bath and shower water.
- Living in proximity to sites where trichloroethylene is produced or waste sites containing the chemical.
- Using trichloroethylene-containing products such as stains and varnishes, adhesives, typewriter correction fluids, paint removers, and cleaners.

Occupational Populations

- Workers involved in the manufacture of trichloroethylene.
- Workers using degreasers that contain trichloroethylene.
- Workers in the dry cleaning industry.

Toxicokinetics and Normal Human Levels

Toxicokinetics

- Trichloroethylene is readily absorbed from the lung, gastrointestinal tract, and skin.
- Trichloroethylene is widely distributed throughout the body via the blood, regardless of the route of exposure; relatively high concentrations are found in fat, liver, kidney and lung.
- Metabolism of absorbed trichloroethylene occurs mainly in the liver and results in a variety of breakdown products, some of which may be toxic.
- Some trichloroethylene is excreted unchanged in the exhaled air, particularly following inhalation or dermal exposure. Trichloroethylene metabolites (primarily trichloroethanol, trichloroethanol glucuronide, and trichloroacetic acid in humans) are mainly excreted in the urine.

Normal Human Levels

- Trichloroethylene levels in blood of the general population are generally below the detection limit of 0.012 parts per billion (ppb).

Biomarkers/Environmental Levels

Biomarkers

- A reliable biomarker is trichloroethylene in exhaled air, blood, or urine. However, trichloroethylene is rapidly metabolized to other substances which are not specific to trichloroethylene exposure.

Environmental Levels

Air

- Average trichloroethylene concentrations across the United States are generally between 0.01 and 0.03 ppb.

Water

- Trichloroethylene concentrations in drinking water in the United States are generally less than 30 ppb.

Food

- Trichloroethylene levels in food are generally in the low ppb range, but were as high as 140 ppb in some samples.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2014. Toxicological Profile for Trichloroethylene (Draft for Public Comment). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

C_2HCl_3

ToxGuide™ for Trichloroethylene

C_2HCl_3

CAS# 79-01-6

October 2014

U.S. Department of Health and Human Services
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Chemical and Physical Information

Trichloroethylene is a Liquid

- Trichloroethylene is a clear colorless liquid with an ethereal, chloroform-like sweet odor.
- Trichloroethylene has been used as an intermediate in the production of the refrigerant HFC-134a, pharmaceuticals, polychlorinated aliphatics, flame retardant chemicals, and insecticides.
- Trichloroethylene is an excellent extraction solvent for greases, oils, fats, waxes, and tars.
- Trichloroethylene has been used by the textile industry to scour cotton, wool, and other fabrics, and as a solvent in waterless drying and finishing operations.
- Trichloroethylene has been used as a solvent in adhesives, lubricants, paints, varnishes, paint strippers, pesticides, and cold metal cleaners.

Routes of Exposure

- Inhalation – Significant potential route of exposure for workers in degreasing operations where trichloroethylene is present; exposure route of concern for general population bathing or showering with trichloroethylene-contaminated tap water.
- Oral – Predominant route of exposure for general population through ingestion of contaminated drinking water.
- Dermal – Potential route of exposure particularly among workers who handle trichloroethylene-containing substances.

Trichloroethylene in the Environment

- Trichloroethylene does not travel long distances by air. Levels in air depend on location; they are usually higher in industrial and populated areas.
- Trichloroethylene readily partitions from soil and water to air.
- Trichloroethylene can be broken down in air by photochemical reactions.
- In water, trichloroethylene volatilizes to air more readily than it undergoes photooxidation or hydrolysis.
- Trichloroethylene on soil surfaces tends to volatilize into the air. When it leaches into soil, it is not readily degraded.
- Trichloroethylene has a low to moderate tendency to bioaccumulate.

Relevance to Public Health (Health Effects)

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

Minimal Risk Levels (MRLs)

Inhalation

- No acute-duration (≤14 days) inhalation MRL was derived for trichloroethylene.
- An intermediate-duration (15-364 days) inhalation MRL of 0.0004 ppm was adopted from the chronic-duration inhalation MRL for trichloroethylene.
- The EPA RfC of 0.0004 ppm for trichloroethylene was adopted as the chronic-duration (≥365 days) inhalation MRL.

Oral

- No acute-duration (≤14 days) oral MRL was derived for trichloroethylene.
- An intermediate-duration (15-364 days) oral MRL of 0.0005 mg/kg/day was adopted from the chronic-duration oral MRL for trichloroethylene.
- The EPA RfD of 0.0005 mg/kg/day for trichloroethylene was adopted as the chronic-duration (≥365 days) oral MRL.

Health Effects

- Main targets of trichloroethylene toxicity include the central nervous system, kidney, liver, immune system, male reproductive system, and developing fetus.
- Available human data provide strong support for trichloroethylene-induced kidney cancer and lesser support for liver cancer and malignant lymphoma in humans.
- Trichloroethylene is listed in the 13th Report on Carcinogens (RoC) as reasonably anticipated to be a human carcinogen. Since the report was released in October 2014, the National Toxicology Program (NTP) has completed its reevaluation of trichloroethylene for a possible change in its listing status in the RoC. The NTP recommends that trichloroethylene be listed in the 14th RoC as known to be a human carcinogen based on sufficient evidence from studies in humans. "EPA concluded that trichloroethylene is "carcinogenic to humans by all routes of exposure". The International Agency for Research on Cancer (IARC) concluded that trichloroethylene is "probably carcinogenic to humans".

Children's Health

- Children exposed to trichloroethylene experienced similar effects to those seen in poisoned adults.
- Trichloroethylene has been detected in human breast milk; therefore, it can be transferred to babies by nursing.
- Exposure of pregnant animals resulted in developmental abnormalities in the offspring. This often occurred with doses that were also toxic to the mothers.

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Sources of Exposure

General Populations

- The primary route of exposure for the general population is inhalation of contaminated air, especially near source areas.
- Ingestion of contaminated drinking water is another potential route of exposure.
- Exposure also can occur through the use of consumer products such as paint removers, which may contain this compound.
- Ingestion of food sources contaminated with 1,1-dichloroethane is not an important exposure pathway.

Occupational Populations

- Exposure of workers can occur in the rubber and plastic, chemical and allied products, electrical equipment and supply, medical and other health services, miscellaneous business services, and oil and gas extraction industries.

Toxicokinetics and Normal Human Levels

Toxicokinetics

- Absorption of 1,1-dichloroethane occurs following exposure via all routes, but skin absorption is minimal due to the high volatility of the chemical.
- Although no direct evidence is available, its high solubility in lipids suggests that the chemical will distribute to tissues according to their lipid content.
- 1,1-Dichloroethane is biotransformed primarily in the liver by cytochrome P450 enzymes.
- 1,1-Dichloroethane is eliminated from the body in the breath or is broken down into metabolites that are excreted in the breath or in the urine.

Normal Human Levels

- A national survey of 1,367 participants conducted in 2003–2004 concluded that the mean levels of 1,1-dichloroethane in the blood could not be determined with certainty because the portion of the data below the limit of detection (0.01 nanogram per liter [ng/L]) was too high.

Biomarkers/Environmental Levels

Biomarkers

- 1,1-Dichloroethane in blood, urine, breath, and body tissues is a biomarker for exposure to the chemical.

Environmental Levels

Air

- Concentrations ranging from a few parts per trillion (ppt) to a few parts per million (ppm) have been measured depending upon the location.

Sediment and Soil

- No quantitative data are available regarding levels of 1,1-dichloroethane in soils.

Water

- In an analysis of 13,347 groundwater sources in California in 2003, 1,1-dichloroethane was detected in 68 samples concentrations ranging from 0.51 to 30 parts per billion (ppb).

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2013. Toxicological Profile for 1,1-Dichloroethane (Draft for Public Comment). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

$C_2H_4Cl_2$

ToxGuide™ for 1,1-Dichloroethane

$C_2H_4Cl_2$

CAS# 75-34-3

June 2013

U.S. Department of Health and Human Services
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Chemical and Physical Information

1,1-Dichloroethane is a Liquid

- 1,1-Dichloroethane is a colorless, oily liquid with a sweet odor that does not occur naturally in the environment.
- It dissolves poorly in water.
- It evaporates easily at room temperature and burns easily.
- In the past it was used as surgical anesthetic.
- Currently, it is used mostly as an intermediate in the manufacture of 1,1,1-trichloroethane, and to a lesser extent vinyl chloride and high vacuum rubber.
- Other uses include fabric spreading, varnish and finish removers, organic synthesis, ore flotation, and as a fumigant and insecticide spray.

Routes of Exposure

- Inhalation – Generally considered the main route of human exposure by breathing contaminated ambient or workplace air.
- Oral – Potentially important route of exposure via drinking water for those living near industrial facilities and waste sites.
- Dermal – Potentially significant route of occupational exposure, but skin absorption is minimal.

1,1-Dichloroethane in the Environment

- 1,1-Dichloroethane can be released into the air, water, and soil.
- It breaks down slowly in air and can be transported long distances in air.
- It breaks down slowly in water. It can evaporate from the water into the air.
- 1,1-Dichloroethane does not bind strongly to soil particles.
- Small amounts of 1,1-dichloroethane released to soil can evaporate into the air or move into groundwater.
- It is not expected to accumulate in the body.

Relevance to Public Health (Health Effects)

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

Minimal Risk Levels (MRLs)

Inhalation

- No acute-, intermediate-, or chronic-duration inhalation MRLs were derived for 1,1-dichloroethane.

Oral

- No acute-, intermediate-, or chronic-duration oral MRLs were derived for 1,1-dichloroethane.

Health Effects

- High levels of 1,1-dichloroethane that cause anesthesia can cause irregular heartbeats.
- No other information is available in humans on the health effects associated with exposure to 1,1-dichlorethane.
- Kidney effects have been observed in cats exposed to 1,1-dichloroethane in air for long periods. However, kidney effects have not been observed in other animal species following long-term inhalation or oral exposure.
- The Department of Health and Human Services (DHHS) and the International Agency for Research on Cancer (IARC) have not evaluated the carcinogenic potential of 1,1-dichloroethane. The EPA has determined that 1,1-dichloroethane is a possible human carcinogen.

Children's Health

- 1,1-Dichloroethane is expected to affect children in the same manner as adults.
- It is not known whether 1,1-dichloroethane can produce birth defects in humans.
- Minor skeletal problems were observed in the fetuses of rats breathing 1,1-dichloroethane; decreases in body weight were also observed in the mothers.

The ToxGuide™ is developed to be used as a pocket guide. Tear off at perforation and fold along lines.

Sources of Exposure

General Populations

- Exposure may occur by inhaling contaminated air or smoke from small cigars or cigarettes.
- Minute amounts, considered safe, may be found in food items stored in materials containing PVC (polyvinyl chloride).
- Exposure via the drinking water is essentially zero.
- Individuals living near hazardous waste sites and landfills may be exposed to higher amounts in air and drinking water.

Occupational Populations

- Occupational exposure occurs in individuals working in facilities where vinyl chloride is produced or used.
- The main use of vinyl chloride is in the manufacture of PVC, a polymer used to make a variety of plastic products including pipes, wire and cable coatings, and packaging materials.

Toxicokinetics and Normal Human Levels

Toxicokinetics

- Inhalation absorption of vinyl chloride in humans is rapid. Volunteers exposed to low concentrations retained about 42% of the inhaled amount.
- There are no data regarding oral or dermal absorption in humans.
- There are no data regarding distribution of vinyl chloride in humans.
- In animals, vinyl chloride metabolites were found in the liver, kidney, spleen, and brain.
- Vinyl chloride metabolism in humans is attributed to the P-450 monooxygenases in the liver. Metabolism of vinyl chloride is saturable.
- Intermediates are detoxified primarily via glutathione conjugation and excreted in the urine as cysteine derivatives.
- Excretion of metabolites occurs mainly in the urine at low exposures. At high doses, where metabolic saturation occurs, vinyl chloride is exhaled as the parent compound.
- Vinyl chloride does not accumulate in the body.

Normal Human Levels

- No data available.

Biomarkers/Environmental Levels

Biomarkers

- Vinyl chloride in exhaled air can be used as biomarker of recent exposure, but is of limited utility for low-level exposures.
- Urinary levels of thiodiglycolic acid, a major metabolite of vinyl chloride, have been used to monitor occupational exposure to vinyl chloride, but it is not specific for exposure to vinyl chloride.

Environmental Levels

Air

- Mean <1 ppb in urban air, data from 1999. <1–34 ppb near manufacturing facilities. Up to 400 ppb near hazardous waste sites

Sediment and Soil

- No data are available for levels of vinyl chloride in soil.

Water

- <10 ppb in less than 1% of groundwater supplies tested in the U.S. in 1982.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2006. Toxicological Profile for Vinyl Chloride (Update). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services.

ToxGuide™ for Vinyl Chloride $CH_2=CH-Cl$

CAS# 75-01-4

July 2006

U.S. Department of Health and Human Services
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Chemical and Physical Information

Vinyl Chloride is a gas

- Vinyl chloride is a manufactured substance, but it can be formed in the environment when other chlorinated substances are degraded by microorganisms.
- It is a gas at room temperature with a mild sweet odor.
- It can exist as liquid under pressure or at low temperatures.
- Burns easily and is unstable at high temperatures.
- Vinyl chloride is only slightly soluble in water, but it is soluble in most common organic solvents.

Routes of Exposure

- Inhalation – The primary route of exposure for the general population and workers.
- Oral – Small, still safe, amounts of vinyl chloride may be found in foods stored in materials containing PVC. No significant vinyl chloride exposure is expected from ingestion of drinking water.
- Dermal – Absorption of vinyl chloride gas through the skin is negligible.

Vinyl Chloride in the Environment

- Vinyl chloride can be released into the environment (mainly the air) during its production or use.
- In the air, it is degraded by reaction with photochemically-generated hydroxyl radicals; its half-life is about 18 hours.
- Liquid vinyl chloride evaporates easily.
- Vinyl chloride in water or soil evaporates rapidly if it is near the surface.
- Vinyl chloride can migrate to groundwater. In anaerobic groundwater, degradation occurs slowly.
- Vinyl chloride is also mobile in soil and susceptible to leaching.
- Vinyl chloride does not accumulate in plants or in animals.

Relevance to Public Health (Health Effects)

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

Minimal Risk Levels (MRLs)

Inhalation

- An MRL of 0.5 ppm has been derived for acute-duration inhalation exposure (≤14 days).
- An MRL of 0.03 ppm has been derived for intermediate-duration exposure (15–364 days).
- A chronic-duration inhalation MRL was not derived for vinyl chloride.

Oral

- No acute- or intermediate-duration oral MRLs were derived for vinyl chloride.
- An MRL of 0.003 mg/kg/day has been derived for chronic-duration exposure (≥1 year).

Health Effects

- Acute high-level exposure to vinyl chloride can produce headache, dizziness, drowsiness, and loss of consciousness. Extremely high-levels can be lethal.
- Exposure of workers to high levels of vinyl chloride has resulted in altered blood flow in the hands.
- Long-term exposure of workers has resulted in alterations in the liver ranging from hypertrophy and hyperplasia to hepatocellular degeneration.
- Studies of workers who breathed vinyl chloride over many years have shown an increased incidence of liver cancer, but other cancers have also been associated with occupational exposure to vinyl chloride.
- The EPA considers vinyl chloride to be a known human carcinogen. NTP has determined that vinyl chloride is a known carcinogen.

Children's Health

- The main source of exposure to vinyl chloride for children is inhalation of contaminated air, the same as in adults.
- Children may be exposed to insignificant levels of vinyl chloride in PVC-based toys.
- Vinyl chloride has not been detected in breast milk.
- Animal studies suggest that infants and young children might be more susceptible than adults to vinyl-chloride induced cancer.

The ToxGuide™ is developed to be used as a pocket guide. Tear off at perforation and fold along lines.

Sources of Exposure

General Populations

- Exposure occurs principally by inhalation of low levels in air or ingestion of very low levels in water. These levels may be higher for people living near hazardous waste sites.
- Very small amounts of 1,1,1-trichloroethane have been found in some food items.
- People who still have at home consumer products such as glues, cleaners, and aerosol sprays that have 1,1,1-trichloroethane may be exposed to it by breathing vapors or by skin contact with the liquid.
- Intentional exposure can occur via sniffing household chemicals in an attempt to get "high".

Occupational Populations

- Exposure can occur during the manufacture of 1,1,1-trichloroethane for export or as a chemical intermediate in the manufacture of hydrofluorocarbons.

Toxicokinetics and Normal Human Levels

Toxicokinetics

- 1,1,1-Trichloroethane is rapidly and efficiently absorbed by the inhalation route.
- Pulmonary absorption of 1,1,1-trichloroethane is saturable.
- Animal data indicate a rapid and complete absorption through the gastrointestinal tract.
- Liquid 1,1,1-trichlorethane is absorbed to a lesser extent through the skin.
- Data in animals suggest that 1,1,1-trichloroethane would probably distribute preferentially to fatty tissues.
- 1,1,1-Trichloroethane is poorly metabolized.
- <10% of the absorbed dose is metabolized by P-450 enzymes to trichloroethanol and trichloroacetic acid, which are excreted in the urine.
- Most of the absorbed 1,1,1-trichloroethane is rapidly excreted as parent compound in expired air.
- 1,1,1-Trichloroethane does not accumulate in the body.

Normal Human Levels

- Mean of 0.34 ppb (µg/L) in blood of non-occupationally exposed subjects in a national survey in 1994.

Biomarkers/Environmental Levels

Biomarkers

- 1,1,1-Trichlorethane in blood, breath, and urine is a biomarker of exposure to this chemical.
- Trichloroethanol and trichloroacetic acid in urine are non-specific biomarkers of exposure to 1,1,1-trichlorethane, unless exposure to other chemicals can be ruled out.
- Assay for the parent compound must be done soon after exposure; assays of the metabolites are more useful for a longer period after exposure.

Environmental Levels

Air

- Typically 0.1–1.0 ppb in city air in the U.S.; <0.1 ppb in rural air.

Sediment and Soil

- No representative values available due probably to rapid volatilization.

Water

- Usually <1 ppb in surface waters; 0.01–3.5 ppb in drinking water from surface or groundwater sources.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2006. Toxicological Profile for 1,1,1-Trichloroethane (Update). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services.

ToxGuide™ for 1,1,1-Trichloroethane

$C_2H_3Cl_3$

CAS# 71-55-6

July 2006

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Chemical and Physical Information

1,1,1-Trichloroethane is a liquid

- 1,1,1-Trichloroethane is a manufactured organic solvent.
- 1,1,1-Trichloroethane is a colorless liquid that burns easily when it contacts a spark or flame.
- 1,1,1-Trichloroethane is volatile and has a sweet, sharp odor.
- It dissolves slightly in water and is miscible with most common organic solvents.
- In the past, it was used in industry as a solvent and as a cleaner for metal parts.
- At home, it was an ingredient of cleaners, glues, and aerosol sprays.
- Currently, limited amounts of 1,1,1-trichloroethane are produced for export.

Routes of Exposure

- Inhalation – Primary route of exposure for the general population, people near waste sites, and workers involved in the manufacture or use of 1,1,1-trichloroethane.
- Oral – Route of exposure at or near waste sites via ingestion of contaminated media. Certain foods also contain small amounts of 1,1,1-trichloroethane.
- Dermal – Route of exposure for workers involved in the manufacture and use of 1,1,1-trichloroethane and for people near waste sites via skin contact with contaminated media.

1,1,1-Trichloroethane in the Environment

- Most 1,1,1-trichloroethane released into the environment moves to the air, where it can last for about 6 years.
- 1,1,1-Trichloroethane in air can travel to the ozone layer where it undergoes photolysis.
- Much of 1,1,1-trichloroethane released to surface water or soil evaporates rapidly to the air; the remaining portions can be broken down by microorganisms.
- 1,1,1-Trichloroethane is expected to be mobile in soil and readily leach into groundwater.
- 1,1,1-Trichloroethane does not accumulate in animals or in plants.

Relevance to Public Health (Health Effects)

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

Minimal Risk Levels (MRLs)

Inhalation

- An MRL of 2 ppm has been derived for acute-duration inhalation exposure (≤14 days).
- An MRL of 0.7 ppm has been derived for intermediate-duration inhalation exposure (15–364 days).
- No chronic-duration inhalation MRL was derived for 1,1,1-trichloroethane.

Oral

- An MRL of 20 mg/kg/day has been derived for intermediate-duration oral exposure (15–364 days).
- No acute- or chronic-duration oral MRLs were derived for 1,1,1-trichloroethane.

Health Effects

- High levels of 1,1,1-trichloroethane in the air can cause dizziness, lightheadedness, and loss of coordination.
- Very high concentrations of 1,1,1-trichloroethane can lower blood pressure and damage the myocardium.
- Skin contact with the liquid can produce effects ranging from mild irritation to chemical burns, as exposure duration increases.
- High levels of 1,1,1-trichloroethane in the air can cause eye irritation.
- Based on no data in humans and inadequate data in animals, the EPA has determined that 1,1,1-trichloroethane is not classifiable as to human carcinogenicity.
- NTP has not classified 1,1,1-trichloroethane for human carcinogenicity.

Children's Health

- Children exposed to 1,1,1-trichloroethane probably would experience the same effects as adults.
- It is not known whether children are more susceptible to 1,1,1-trichloroethane poisoning than adults.

Sources of Exposure

General Populations

- The general population may be exposed to 1,4-dioxane in contaminated air, water, food, and consumer products.
- Inhalation exposure to 1,4-dioxane in tap water may also occur during activities such as showering, bathing, and laundering. Exposure during these activities may be higher than exposure via ingestion of tap water.
- Dermal exposure may occur through bathing or showering in contaminated tap water or through the use of cosmetics, detergents, shampoos, and bubble baths containing 1,4-dioxane. Although FDA monitors 1,4-dioxane in raw materials used in the manufacture of cosmetic products, not all raw material producers are effectively controlling the levels.

Occupational Populations

- Facilities that produce or use solvents containing 1,4-dioxane.
- 1,4-Dioxane is also used as a laboratory reagent in chromatography and in plastic, rubber, insecticides, and herbicides.

Toxicokinetics and Normal Human Levels

Toxicokinetics

- 1,4-Dioxane is readily absorbed through the lungs and gastrointestinal system and poorly absorbed through the skin.
- At lower doses, 1,4-dioxane is rapidly metabolized to β-hydroxyethoxy acetic acid (HEAA).
- At higher doses, the metabolic process may become saturated resulting in 1,4-dioxane being excreted in exhaled air and urine.
- 1,4-Dioxane is rapidly eliminated from the body and does not accumulate.
- 1,4-Dioxane is primarily excreted as the metabolite HEAA in urine.

Normal Human Levels

- A 2007–2008 study from a U.S. population (≥12 years old) found no detectable concentration of 1,4-dioxane in 2,053 blood samples analyzed.

Biomarkers/Environmental Levels

Biomarkers

- 1,4-Dioxane and HEAA in plasma and urine can be used as biomarkers of recent exposure in workers.

Environmental Levels

Air

- Levels in ambient air ranged from 0.028–0.11 ppb; mean concentration in indoor air was 1.03 ppb. These values are from the mid 1980s, more recent data are not available.

Sediment and Soil

- No data are available on actual measurements of 1,4-dioxane in soil.

Water

- Levels in municipal water were approximately 1 ppb in the 1970s; more recent data are not available.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2012. Toxicological Profile for 1,4-Dioxane. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services.

ToxGuide™ for 1,4-Dioxane

$C_4H_8O_2$

CAS# 123-91-1

April 2012

U.S. Department of Health and Human Services
Public Health Service
Agency for Toxic Substances and Disease Registry
www.atsdr.cdc.gov

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Chemical and Physical Information

1,4-Dioxane is a Liquid

- 1,4-Dioxane is a clear liquid that dissolves in water.
- It is primarily used as a solvent for chemical processing (e.g., adhesives, cleaning and detergent preparations, cosmetics, deodorant fumigants, emulsions and polishing compositions, fat, lacquers, pulping of wood, varnishes, waxes).
- It is unintentionally formed as a contaminant during the manufacture of alkyl ether sulfates and other ethoxylated surfactants, which are used in consumer products such as cosmetics, detergents, and shampoos. Currently, manufactures reduce 1,4-dioxane from ethoxylated surfactants to trace levels before these chemicals are made into consumer products.

Routes of Exposure and 1,4-Dioxane in the Environment

Route of Exposure

- Inhalation – Predominant route of exposure for the general population and workers. Inhalation exposure also occurs from 1,4-dioxane released from tap water during bathing and laundering.
- Oral – Predominant route of exposure for the general population ingesting contaminated drinking water and from food.
- Dermal – Use of contaminated consumer products such as cosmetics or shampoos.

1,4-Dioxane in the Environment

- 1,4-Dioxane can be released into the environment during its production, the processing of other chemicals, its use, and with its unintentional formation during the manufacture of ethoxylated surfactants.
- 1,4-Dioxane is expected to volatilize from the surfaces of water and soil at a moderate rate. In air, it is subject to photooxidation with an estimated half-life of 1–3 days.
- 1,4-Dioxane biodegrades very slowly in water and soils and is considered recalcitrant. It adsorbs weakly to soil and will move quickly into groundwater.
- Bioconcentration, bioaccumulation, and biomagnification are not considered important environmental fate processes for 1,4-dioxane.

Relevance to Public Health (Health Effects)

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

Minimal Risk Levels (MRLs)

Inhalation

- An MRL of 2 ppm has been derived for acute-duration inhalation exposure (≤14 days).
- An MRL of 0.2 ppm has been derived for intermediate-duration inhalation exposure (15–364 days).
- An MRL of 0.03 ppm has been derived for chronic-duration inhalation exposure (≥1 year).

Oral

- An MRL of 5 mg/kg/day has been derived for acute-duration oral exposure (≤14 days).
- An MRL of 0.5 mg/kg/day has been derived for intermediate-duration oral exposure (15–364 days).
- An MRL of 0.1 mg/kg/day has been derived for chronic-duration oral exposure (≥1 year).

Health Effects

- The primary targets of 1,4-dioxane toxicity are the liver, kidneys, and nasal cavity (following inhalation exposure).
- Acute exposures to airborne 1,4-dioxane can also result in eye and nose irritation in humans.
- 1,4-Dioxane is likely to be carcinogenic to humans. Liver tumors have been observed in rats and mice following chronic drinking water exposure. Nasal tumors were also observed in rats following chronic inhalation or drinking water exposure.

Children's Health

- It is not known if children are more susceptible to 1,4-dioxane poisoning than adults.

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Sources of Exposure

General Populations

- The most likely source of exposure is ingestion of contaminated food and drinking water. Exposure can also occur via inadvertent ingestion of contaminated soil/dust or lead-based paint.
- Lead can leach into drinking water from lead-soldered joints or leaded pipes in water distribution systems or individual houses. Lead may also enter foods if they are put into improperly glazed pottery or ceramic dishes.
- Some non-Western folk remedies may contain substantial amounts of lead. Some types of hair dyes and cosmetics may contain lead compounds.
- Other potential sources of exposure are hobbies that use lead: casting ammunition and m fishing weights; soldering with lead solder; making stained glass; using firing ranges. Leaded gasoline is still used in some race cars, airplanes, and off-road vehicles.

Occupational Populations

- Potentially high levels of lead may occur in the following industries: lead smelting and refining industries, battery manufacturing plants, steel welding or cutting operations, construction, rubber products and plastics industries, printing industries, firing ranges, radiator repair shops and other industries requiring flame soldering of lead solder.

Toxicokinetics and Normal Human Levels

Toxicokinetics

- Approximately 95% of deposited inorganic lead that is inhaled is absorbed.
- The extent and rate of gastrointestinal absorption of inorganic lead are influenced by the physiological state of the exposed individual and the species of the lead compound.
- Gastrointestinal absorption of lead is higher in children (40–50%) than in adults (3–10%). The presence of food in the gastrointestinal tract decreases absorption.
- Absorption of lead from soil is less than that of dissolved lead, but is similarly depressed by meals (26% fasted; 2.5% when ingested with a meal).
- In adults, about 94% of the total amount of lead in the body is contained in the bones and teeth versus about 73% in children.
- The elimination half-lives for inorganic lead in blood and bone are approximately 30 days and 27 years, respectively.
- Independent of the route of exposure, absorbed lead is excreted primarily in urine and feces.

Normal Human Levels

- Lead levels in blood (geometric mean, 1999-2002):
  1.9 µg/dL for children 1-5 years
  1.5 µg/dL for adults 20–59 years
- Lead levels in urine (geometric mean, 2001-2002):
  0.677 µg/L for ≥6 years of age

Biomarkers/Environmental Levels

Biomarkers

- Analysis of lead in whole blood is the most common and accurate method of assessing lead exposure. Erythrocyte protoporphyrin (EP) tests can also be used, but are not as sensitive at low blood lead levels (<20 µg/dL). Lead in blood reflects recent exposure.
- Bone lead measurements are an indicator of cumulative exposure.
- Measurements of urinary lead levels and hair have been used to assess lead exposure; however, they are not as reliable.

Environmental Levels

Air

- The concentration of lead in air samples (2002) is <0.05 µg/m^3.

Sediment and Soil

- The natural lead content of soil typically ranges from <10 to 30 µg/g. However, lead levels in the top layers of soil vary widely due to deposition and accumulation of atmospheric particulates from anthropogenic sources.

Water

- Levels of lead in surface water and groundwater in the U.S. range between 5 and 30 µg/L.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological Profile for Lead. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services.

ToxGuide™ for Lead

Pb

CAS# 7439-92-1

October 2007

U.S. Department of Health and Human Services
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Chemical and Physical Information

Lead is a metal

- Lead is a naturally-occurring bluish-gray metal that is rarely found in its elemental form, but occurs in the Earth's crust primarily as the mineral galena (PbS), and to a lesser extent as anglesite ($PbSO_4$) and cerussite ($PbCO_3$).
- Lead is not a particularly abundant element, but its ore deposits are readily accessible and widely distributed throughout the world. Its properties, such as corrosion resistance, density, and low melting point, make it a familiar metal in pipes, solder, weights, and storage batteries.
- Natural lead is a mixture of four stable isotopes, ^{208}Pb (51–53%), ^{206}Pb (23.5–27%), ^{207}Pb (20.5–23%), and ^{204}Pb (1.35–1.5%). Lead isotopes are the stable decay product of three naturally radioactive elements: ^{205}Pb from uranium, ^{207}Pb from actinium, and ^{208}Pb from thorium.

Routes of Exposure

- Inhalation – Primary route for occupational exposure. Larger particles (>2.5 µm) that are deposited in the ciliated airways (nasopharyngeal and tracheobronchial regions) can be transferred by mucociliary transport into the esophagus and swallowed.
- Oral – Primary route of exposure for the general population.
- Dermal – Studies in animals have shown that organic lead is well absorbed through the skin.

Lead in the Environment

- Lead is dispersed throughout the environment primarily as the result of anthropogenic activities. In the air, lead is in the form of particles and is removed by rain or gravitational settling.
- The fate of lead in soil is affected by the adsorption at mineral interfaces, which are dependent upon physical and chemical characteristics of the soil (e.g., pH, soil type, particle size, organic matter content).
- Sources of lead in dust and soil can include lead from weathering and chipping of lead-based paint from buildings, bridges, and other structures.
- The solubility of lead compounds in water is a function of pH, hardness, salinity, and the presence of humic material. Solubility is highest in soft, acidic water.

Relevance to Public Health (Health Effects)

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

Minimal Risk Levels (MRLs)

- MRLs were not derived for lead because a clear threshold for some of the more sensitive effects in humans has not been identified.
- In lieu of MRLs, ATSDR has developed a framework to guide decisions at lead sites. This approach utilizes site-specific exposure data to estimate internal doses as measured by blood lead levels (PbBs) (see Appendix D in the Toxicological Profile).

Health Effects

Hematological

- Decreased activity of several heme biosynthesis enzymes at PbB <10 µg/dL.

Gastrointestinal

- Colic in children – PbB 60–100 µg/dL.

Cardiovascular

- Elevated blood pressure – PbB<10 µg/dL.

Renal

- Decreased glomerular filtration rate at mean PbB <20 µg/dL.

Neurological

- Encephalopathy – PbB100–120 µg/dL (adults) 70–100 µg/dL (children).
- Peripheral neuropathy – PbB40 µg/dL.
- Neurobehavioral and neuropsychological effects in adults – PbB40–80 µg/dL.
- Cognitive and neurobehavioral effects in children at PbB <10 µg/dL.

Reproductive

- Reduced fertility – PbB>40 µg/dL.

Children's Health

- Children are more vulnerable to the effects of lead than adults.
- The most common source of lead exposure for children is lead-based paint.
- Lead exposures during infancy or childhood may result in anemia, neurological impairment, renal alterations, colic, and impaired metabolism of vitamin D.
- Lead exposures either *in utero*, during infancy, or during childhood may result in delays or impairment of neurological development, neurobehavioral deficits including IQ deficits, low birth weight, and low gestational age, growth retardation, and delayed sexual maturation in girls.
- Ensuring a diet that is nutritionally adequate in calcium and iron may decrease the absorbed dose of lead.

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Sources of Exposure

General Populations

- The general population may be exposed to chromium daily through food, drinking water, and air.
- The predominant route of exposure for the general population is ingestion of chromium in the diet.
- Exposure may also occur through inhalation of contaminated air and consumption of contaminated water.
- Chromium(III) is an essential nutrient required for normal energy metabolism.
- The Institute of Medicine of the National Academy of Sciences determined an adequate intake of chromium(III) of 20–45 µg/day for adolescents and adults.

Occupational Populations

- Workers in approximately 80 industries may be exposed to chromium(VI).
- Occupational exposure to chromium primarily occurs from chromate production, stainless steel production and welding, chromium plating, ferrochrome alloys, and chrome pigment production. Workers in the tanning industries are also potentially exposed to chromium.

Toxicokinetics and Normal Human Levels

Toxicokinetics

- The toxicokinetics of a given chromium compound depend on the valence state of the chromium atom and its solubility.
- Chromium is absorbed through the lungs; less-water soluble compounds have a longer retention time in the lung than more soluble forms.
- Less than 10% of an ingested chromium dose is absorbed from the gastrointestinal tract. More soluble compounds have higher absorption fractions.
- Chromium can penetrate human skin to some extent, especially if the skin is damaged.
- Absorbed chromium is distributed to nearly all tissues, with the highest concentrations found in kidneys and liver. Bone is also a major depot and may contribute to long-term retention.
- Chromium(VI) is reduced to chromium(III) via the intermediate forms of chromium(V) and chromium(IV).
- Absorbed chromium is predominantly excreted in the urine.

Normal Human Levels

- In the general population, the mean levels of chromium in serum and urine are 0.10–0.16 and 0.22 µg/L, respectively.

Biomarkers/Environmental Levels

Biomarkers

- Chromium(III) is an essential element and is normally present in blood and urine.
- Exposure to higher than normal levels of chromium may result in increased chromium levels in blood, urine, expired air, hair, and nails.
- Elevations in chromium in blood and urine are considered the most reliable biomarkers of exposure.

Environmental Levels

Air

- Median concentrations in ambient air are <20 ng/m^3.
- Indoor air in areas with cigarette smoking can be 10–400 times higher than outdoor air.

Sediment and Soil

- The mean concentration of chromium in soil is 37.0 mg/kg.

Water

- Most drinking water supplies in the United States contain <5 µg/L of chromium.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2012. Toxicological Profile for Chromium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services.

ToxGuide™ for Chromium

Cr

CAS# 7440-47-3

October 2012

U.S. Department of Health and Human Services
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Agency for Toxic Substances and Disease Registry
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Chemical and Physical Information

Chromium is a Metal

- Chromium is a naturally occurring element found in rocks, animals, plants, and soil, where it exists in combination with other elements to form various compounds.
- The main forms of chromium are: chromium(0), chromium(III), and chromium(VI).
- Chromium is widely used in manufacturing processes to make various metal alloys such as stainless steel.
- Chromium can be found in many consumer products such as: wood treated with copper dichromate, leather tanned with chromic sulfate, stainless steel cookware, and metal-on-metal hip replacements.

Routes of Exposure

- Inhalation – Predominant route of exposure for occupational populations.
- Oral – Predominant route of exposure for the general population.
- Dermal – Minor route of exposure for the general population.

Chromium in the Environment

- Chromium is released into the atmosphere via industrial, commercial, and residential fuel combustion of natural gas, oil, and coal and from emissions from metal industries such as chrome plating and steel production.
- Approximately 1/3 of atmospheric releases are believed to be in the form of chromium(VI).
- Electroplating, leather tanning, and textile industries release large amounts of chromium to surface water.
- Chromium is primarily removed from the atmosphere by fallout and precipitation, the residence time is expected to be <10 days.
- Most of the chromium released in water will be deposited in the sediments.
- Chromium is not believed to biomagnify in the food chain.

Relevance to Public Health (Health Effects)

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

Minimal Risk Levels (MRLs)

Inhalation

- No acute-duration inhalation MRLs (≤14 days) were derived for Cr(III) or Cr(VI).
- Intermediate-duration inhalation MRLs (15–364 days) were derived:
  - $5x10^{-6}$ mg Cr/m^3 for Cr(VI) aerosols and mists
  - $3x10^{-4}$ mg Cr/m^3 for Cr(VI) particulates
  - $5x10^{-3}$ mg Cr/m^3 for insoluble Cr(III) particulates
  - $1x10^{-4}$ mg Cr/m^3 for soluble Cr(III) particulates
- An MRL of $5x10^{-6}$ mg Cr/m^3 has been derived for chronic-duration inhalation exposure to Cr(VI) aerosols and mists of (≥1 year).

Oral

- No acute-duration oral MRL(≤14 days) was derived for Cr(VI).
- An MRL of $5x10^{-3}$ mg Cr/kg/day has been derived for intermediate-duration oral exposure to Cr(VI) (15–364 days).
- An MRL of $9x10^{-4}$ mg Cr/kg/day has been derived for chronic-duration oral exposure to Cr(VI) (≥1 year).
- No acute-, intermediate-, or chronic duration oral MRLs were derived for Cr(III).

Health Effects

- In general, chromium(VI) compounds are more toxic than chromium(III) compounds.
- The most sensitive targets of chromium(VI) are the respiratory (nasal and lung irritation and altered pulmonary function following inhalation exposure), gastrointestinal (irritation, ulceration, and stomach and small intestine lesions following oral exposure), hematological (microcytic, hypochromic anemia), and reproductive (decreased sperm count and epididymal damage) systems.
- The primary targets of chromium(III) compounds are the respiratory (following inhalation exposure) and immunological systems. Chromium allergic dermatitis is typically elicited by dermal contact in sensitized individuals.
- DHHS, IARC, and EPA have classified chromium(VI) as a human carcinogen.
- IARC has classified chromium(III) and metallic chromium as not classifiable as to their carcinogenicity to humans.

Children's Health

- It is unknown whether children are more sensitive to chromium poisoning than adults.
- In laboratory animals, chromium(VI) causes miscarriages, low birth weight, and changes in development of skeleton and reproductive system. These developmental effects may be related, in part, to maternal chromium toxicity.

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Sources of Exposure

General Populations

- The major sources of benzene exposure are tobacco smoke, automobile service stations, exhaust from motor vehicles, and industrial emissions.
- About 50% of the entire nationwide exposure to benzene results from smoking tobacco or from exposure to tobacco smoke.
- Vapors (or gases) from products that contain benzene, such as glues, paints, furniture wax, and detergents, can also be a source of exposure.

Occupational Populations

- Individuals employed in industries that make or use benzene may be exposed. These industries include benzene production (petrochemicals, petroleum refining, and coke and coal chemical manufacturing), rubber tire manufacturing, and storage or transport of benzene and petroleum products containing benzene.
- Other workers who may be exposed to benzene because of their occupations include steel workers, printers, rubber workers, shoe makers, laboratory technicians, firefighters, and gas station employees.

Toxicokinetics and Normal Human Levels

Toxicokinetics

- Benzene is rapidly absorbed through the lungs; approximately 50% of the benzene in air is absorbed.
- Over 90% of ingested benzene is absorbed through the gastrointestinal tract.
- Absorbed benzene is rapidly distributed throughout the body and tends to accumulate in fatty tissues.
- The liver serves an important function in benzene metabolism, which results in the production of several reactive metabolites.
- At low exposure levels, benzene is rapidly metabolized and excreted predominantly as conjugated urinary metabolites.
- At higher exposure levels, metabolic pathways appear to become saturated and a large portion of an absorbed dose of benzene is excreted as parent compound in exhaled air.

Normal Human Levels

- Median level in blood is 0.06 µg/L for non-occupationally exposed individuals and 0.05 µg/L in a subset of non-smokers.

Biomarkers/Environmental Levels

Biomarkers

- Urinary benzene level is the most sensitive biomarker of exposure to low concentrations.
- Urinary levels of several benzene metabolites including muconic acid and S-phenyl mercapturic acid are also sensitive biomarkers of exposure.

Environmental Levels

Air

- The concentration of benzene in air samples from metropolitan areas was 0.58 ppb.

Sediment and Soil

- Benzene was detected in less than 10% of sediment samples with a median level of <5 ppb.

Water

- Benzene was detected in approximately 40% of surface water samples with levels ranging for non-detectable to 100 µg/L.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological Profile for Benzene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services.

ToxGuide™ for Benzene

C_6H_6

CAS# 71-43-2

October 2007

U.S. Department of Health and Human Services
Public Health Service
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Chemical and Physical Information

Benzene is a colorless liquid

- Benzene, also known as benzol, has a sweet odor.
- Benzene is highly flammable.
- Benzene is made mostly from petroleum sources. Various industries use benzene to make other chemicals, such as styrene (for Styrofoam® and other plastics), cumene (for various resins), and cyclohexane (for nylon and synthetic fibers).
- Benzene is also used for the manufacturing of some types of rubbers, lubricants, dyes, detergents, drugs, and pesticides.
- Benzene is also a natural component of crude oil, gasoline and cigarette smoke.

Routes of Exposure

- Inhalation – Primary route of exposure for general and occupational populations.
- Oral – Minor route of exposure.
- Dermal – Minor route of exposure.

Benzene in the Environment

- Benzene enters the air, water, and soil as a result of industrial processes, emissions from burning coal and oil, tobacco smoke, gasoline exhaust and gasoline leaks, and from natural sources including volcanoes and forest fires.
- Benzene in the atmosphere chemically degrades in only a few days.
- Benzene released to soil or waterways is subject to volatilization, photooxidation, and biodegradation.

Relevance to Public Health (Health Effects)

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

Minimal Risk Levels (MRLs)

Inhalation

- An MRL of 0.009 ppm has been derived for acute-duration inhalation exposure (≤14 days).
- An MRL of 0.006 ppm has been derived for intermediate-duration inhalation exposure (15–364 days).
- An MRL of 0.003 ppm has been derived for chronic-duration inhalation exposure (≥1 year).

Oral

- No acute- or intermediate-duration oral MRLs were derived for benzene.
- An MRL of 0.0005 mg/kg/day has been derived for chronic-duration oral exposure (≥1 year).

Health Effects

- The primary target organs for acute exposure are the hematopoietic system, nervous system, and immune system.
- The primary target for adverse systemic effects of benzene following low-level chronic exposure is the hematological system.
- Benzene is a known human carcinogen and is associated with leukemia, especially acute myelogenic leukemia.
- Benzene exposure may also be associated with reproductive and developmental effects based on animal studies.

Children's Health

- It is not known if children are more susceptible to benzene poisoning than adults.

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Sources of Exposure

General Populations

- Exposure to ethylbenzene is possible through contact with gasoline, automobile emissions, solvents, printing inks, varnishes and paints, and other consumer products.
- Cigarette smoke is also a source of ethylbenzene exposure.
- Ground water from sources near manufacturing and processing facilities, petroleum refineries, and leaking underground storage tanks may be contaminated with ethylbenzene.

Occupational Populations

- Occupational exposures are expected within the petroleum industry, industries using solvents, paints, and coatings, and during the manufacture and handling of ethylbenzene and styrene.

Toxicokinetics and Normal Human Levels

Toxicokinetics

- Ethylbenzene is well absorbed from the lungs, gastrointestinal tract, and through the skin.
- Absorbed ethylbenzene is rapidly eliminated by metabolism and excretion of metabolites; the half-time in blood is less than 1 hour.
- The major metabolic pathways are side-chain and ring hydroxylation with subsequent formation of glucuronide and sulfate conjugates.
- Ethylbenzene metabolites, primarily conjugates, mandelic acid, and phenylglyoxylic acid, are excreted in the urine.

Normal Human Levels

- No data available.

Biomarkers/Environmental Levels

Biomarkers

- Ethylbenzene can be measured in blood, subcutaneous fat, and in expired air.
- Expired air concentrations have been correlated with levels of ethylbenzene in ambient air.
- Urinary levels of mandelic acid and/or phenylglyoxylic acid. However, these are also elevated following exposure to styrene.

Environmental Levels

Air

- Median ethylbenzene concentrations of 0.6 ppb in urban and suburban air and 0.01 ppb in rural air have been reported.

Sediment and Soil

- Ethylbenzene is rarely detected in soil.

Water

- Ethylbenzene is rarely detected in drinking water.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2010. Toxicological Profile for Ethylbenzene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services.

ToxGuide™ for Ethylbenzene

C_8H_{10}

CAS# 100-41-4

September 2011

U.S. Department of Health and Human Services
Public Health Service
Agency for Toxic Substances and Disease Registry
www.atsdr.cdc.gov

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ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

Chemical and Physical Information

Ethylbenzene is a Liquid

- Ethylbenzene is a colorless liquid with an aromatic odor, and is flammable and combustible. Ethylbenzene is naturally found in crude petroleum and is widely distributed in the environment.
- It is a high production volume chemical primarily used for the production of styrene. Ethylbenzene is also used as a solvent and in the manufacture of several organic compounds other than styrene.
- Consumer products containing ethylbenzene include gasoline, paints and varnishes, inks, pesticides, carpet glues, automotive products, and tobacco products.

Routes of Exposure

- Inhalation – Predominant route of exposure for general population and workers.
- Oral – Minor route of exposure via ingestion of contaminated water.
- Dermal –Skin contact may occur during showering or bathing with contaminated water or from contact with contaminated soil or products containing ethylbenzene.

Ethylbenzene in the Environment

- Ethylbenzene partitions primarily to air and removal via photochemically generated hydroxyl radicals is an important degradation mechanism. The half-life in air is approximately 1–2 days. Ethylbenzene is ubiquitous in ambient air, mainly as a result of automobile emissions.
- In surface water, most of the ethylbenzene will evaporate. The remaining ethylbenzene is broken down through photooxidation and biodegradation.
- Ethylbenzene is moderately mobile and biodegrades in soil.
- Ethylbenzene does not appear to bioconcentrate in aquatic food chains.

Relevance to Public Health (Health Effects)

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

Minimal Risk Levels (MRLs)

Inhalation

- An MRL of 5 ppm has been derived for acute-duration inhalation exposure (≤14 days).
- An MRL of 2 ppm has been derived for intermediate-duration inhalation exposure (15–364 days).
- An MRL of 0.06 ppm has been derived for chronic-duration inhalation exposure (≥1 year).

Oral

- No acute-duration oral MRL was derived for ethylbenzene.
- An MRL of 0.4 mg/kg/day has been derived for intermediate-duration oral exposure (15–364 days).
- No chronic-duration oral MRL was derived for ethylbenzene.

Health Effects

- Exposure to high levels of ethylbenzene can cause eye and throat irritation, vertigo, and dizziness.
- In animals, the most sensitive target of ethylbenzene toxicity appears to be the auditory system; a potentially irreversible damage to cochlear hair cells and hearing loss have been observed in rats following acute and intermediate-duration inhalation exposure and acute oral exposure.
- Animal studies indicate that intermediate-duration oral exposure can be hepatotoxic.
- Direct contact with liquid ethylbenzene caused eye and skin irritation in animals.
- Developmental effects (decreases in growth and increased skeletal variations) have been observed in animals following inhalation exposure to high levels of ethylbenzene.
- IARC has classified ethylbenzene as possibly carcinogenic to humans (Group 2B).

Children's Health

- Children are expected to be affected by ethylbenzene poisoning in the same manner as adults.

The ToxGuide™ is developed to be used as a pocket guide. Tear off at perforation and fold along lines.

Sources of Exposure

General Populations

- The highest exposure levels for the general population result from contact with consumer products containing xylenes.
- Very small amounts of xylenes are also present in cigarette smoke.
- Exposure may rise from ingestion of contaminated drinking water, painting, pumping gasoline, scale model building, using cleaning solvents, lacquers, and paint thinners/removers.
- People who work or live near industrial settings may receive a higher exposure to xylenes.
- Small amounts of xylene are commonly found in indoor air.

Occupational Populations

- Painters (or paint industry workers), and laboratory workers appear to be most frequently affected.
- Workers involved in distillation and purification of xylene, employed in industries using xylene as a raw material, or employed in the petroleum industry, may be at higher risk of exposure.
- Increased exposures have been observed for wood processing plant workers, painters, gas station employees, metal workers, and furniture refinishers.

Toxicokinetics and Normal Human Levels

Toxicokinetics

- Xylenes, because of their lipophilic properties, are rapidly absorbed by all routes of exposure, rapidly distributed throughout the body, and, if not metabolized, quickly eliminated in exhaled air.
- In humans, absorption has been estimated as >50% through the lungs following inhalation exposure and <50% through the gastrointestinal tract.
- The major pathway for metabolism involves mixed function oxidases in the liver, resulting mainly in the formation of isomers of methylhippuric acid that are eliminated in the urine.

Normal Human Levels

- There are no data on background concentrations of xylene in blood or urine.

Biomarkers/Environmental Levels

Biomarkers

- Measurement of blood levels of xylene is limited by the rapid metabolism of xylene.
- Detection of methylhippuric acid in the urine is the most widely used indicator of xylene exposure, but measures of this metabolite are only valid soon after exposures.

Environmental Levels

Air

- Typical concentrations of xylene in indoor air range from 1 to 10 ppb.
- Typical concentrations in outdoor air range from 1 to 30 ppb.

Sediment and Soil

- No data are available on levels of xylene in soil.

Water

- Xylene has been detected in <5% of groundwater samples. Median xylene concentrations of ≤2 ppb have been reported in urban and rural drinking water wells or monitoring wells in the United States.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological Profile for Xylenes. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services.

ToxGuide™ for Xylenes

C_8H_{10}

CAS# 1330-20-7

October 2007

U.S. Department of Health and Human Services
Public Health Service
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Chemical and Physical Information

Xylene is a colorless liquid

- Xylene is a colorless, flammable liquid with a somewhat sweet odor.
- Xylene evaporates and burns easily.
- Xylenes (mixtures of *ortho*-, *meta*-, and *para*-isomers) are used as industrial solvents, synthetic intermediates, and solvents in commercial products such as paints, coatings, adhesive removers, and paint thinners; they are also a component of gasoline.
- Xylene occurs naturally in petroleum and coal tar and is formed during forest fires.

Routes of Exposure

- Inhalation – Primary route of exposure for general and occupational populations.
- Oral – Minor route of exposure.
- Dermal – Minor route of exposure.

Xylenes in the Environment

- Xylene released to the atmosphere is quickly transformed by photooxidation with a half-life of approximately 8–14 hours.
- When released to soil or surface water, xylene volatilizes into the atmosphere, where it is quickly degraded.
- Xylene that does not volatilize quickly may undergo biodegradation in the soil or water.
- Xylene may also leach into groundwater, where degradation by microbes becomes the primary removal process.

Relevance to Public Health (Health Effects)

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

Minimal Risk Levels (MRLs)

Inhalation

- An MRL of 2 ppm has been derived for acute-duration inhalation exposure (≤14 days).
- An MRL of 0.6 ppm has been derived for intermediate-duration inhalation exposure (15–364 days).
- An MRL of 0.05 ppm has been derived for chronic-duration inhalation exposure (≥1 year).

Oral

- An MRL of 1 mg/kg/day has been derived for acute-duration oral exposure (≤14 days).
- An MRL of 0.4 mg/kg/day has been derived for intermediate-duration oral exposure (15–364 days).
- An MRL of 0.2 mg/kg/day has been derived for chronic-duration oral exposure (≥1 year).

Health Effects

- The primary effects of xylene exposure involve the nervous system by all routes of exposure, the respiratory tract by inhalation exposure, and, at higher oral exposure levels, hepatic, renal, and body weight effects.
- The nervous system effects include subjective symptoms of intoxication at higher concentrations and impaired performance on tests of short-term memory, reaction time, and equilibrium at lower concentrations.
- Humans have reported signs of nose, eye, and throat irritation during exposure to xylene vapors.
- Dermal exposure of humans to xylene causes skin irritation, dryness and scaling of the skin, and vasodilation.
- Exposures to high concentrations of xylene may also be associated with developmental effects based on animal studies.

Children's Health

- No data are available regarding the effects of exposure to xylenes in children, but it is expected that children would experience the same effects as exposed adults.
- Children exposed to xylenes by inhalation may be more sensitive to respiratory impairment than exposed adults because of their narrower airways.