

Trichloroethylene

79-01-6

Hazard Summary

Most of the trichloroethylene used in the United States is released into the atmosphere from industrial degreasing operations. Acute (short-term) and chronic (long-term) inhalation exposure to trichloroethylene can affect the human central nervous system (CNS), with symptoms such as dizziness, headaches, confusion, euphoria, facial numbness, and weakness. Liver, kidney, immunological, endocrine, and developmental effects have also been reported in humans. A recent analysis of available epidemiological studies reports trichloroethylene exposure to be associated with several types of cancers in humans, especially kidney, liver, cervix, and lymphatic system. Animal studies have reported increases in lung, liver, kidney, and testicular tumors and lymphoma. The Agency is currently reassessing the cancer classification of trichloroethylene.

Please Note: The main source of information for this fact sheet is the Agency for Toxic Substances and Disease Registry's (ATSDR's) Toxicological Profile for Trichloroethylene. (1) Another secondary source used is EPA's Health Assessment Document for Trichloroethylene. (4)

Uses

- The main use of trichloroethylene is in the vapor degreasing of metal parts. (1)
- Trichloroethylene is also used as an extraction solvent for greases, oils, fats, waxes, and tars, a chemical intermediate in the production of other chemicals, and as a refrigerant. (1)
- Trichloroethylene is used in consumer products such as typewriter correction fluids, paint removers/strippers, adhesives, spot removers, and rug-cleaning fluids. (1)
- Trichloroethylene was used in the past as a general anesthetic. (1)

Sources and Potential Exposure

- Trichloroethylene has been detected in ambient air at levels less than 1 part per billion (ppb). Ambient air measurement data from the Aerometric Information Retrieval System (which has 1,200 measurements from 25 states from 1985–1995) give a range of ambient air values from 0.01 to 3.9 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$). (1,13)
- Because of its moderate water solubility, trichloroethylene in soil has the potential to migrate into groundwater. The relatively frequent detection of trichloroethylene in groundwater confirms its mobility in soils. (13)
- Drinking water supplies relying on contaminated groundwater sources may contain trichloroethylene. ATSDR reports that trichloroethylene is the most frequently reported organic contaminant in groundwater. It estimates between 9 and 34 percent of drinking water supply sources have some trichloroethylene contamination but that most municipal water supplies are in compliance with the maximum contaminant level of 5 $\mu\text{g}/\text{L}$. (1,13)
- Workers may be exposed to trichloroethylene in the factories where it is manufactured or used. In addition, persons breathing air around these factories may be exposed to trichloroethylene. (1)
- Persons may also be exposed to trichloroethylene through the use of products containing the chemical and from evaporation and leaching from waste disposal sites. (1)

Assessing Personal Exposure

- Trichloroethylene can be measured in the breath, and breakdown products of trichloroethylene can be measured in urine or blood. (1)

Health Hazard Information

Acute Effects:

- Central nervous system effects are the primary effects noted from acute inhalation exposure to trichloroethylene in humans, with symptoms including sleepiness, fatigue, headache, confusion, and feelings of euphoria. Effects on the liver, kidneys, gastrointestinal system, and skin have also been noted. (1)
- Neurological, lung, kidney, and heart effects have been reported in animals acutely exposed to trichloroethylene. (1)
- Tests involving acute exposure of rats and mice have shown trichloroethylene to have **low** toxicity from inhalation exposure and **moderate** toxicity from oral exposure. (1,2)

Chronic Effects (Noncancer) :

- As with acute exposure, chronic exposure to trichloroethylene by inhalation also affects the human central nervous system. Case reports of intermediate and chronic occupational exposures included effects such as dizziness, headache, sleepiness, nausea, confusion, blurred vision, facial numbness, and weakness. (1)
- Effects to the liver, kidneys, and immune and endocrine systems have also been seen in humans exposed to trichloroethylene occupationally or from contaminated drinking water. (13)
- Studies have shown that simultaneous alcohol consumption and trichloroethylene inhalation increases the toxicity of trichloroethylene in humans. (1)
- Neurological, liver, and kidney effects were reported in chronically-exposed animals. (1)
- EPA is in the process of calculating a Reference Concentration (RfC) and Reference Dose (RfD) for trichloroethylene.
- ATSDR has calculated an intermediate-duration inhalation minimal risk level (MRL) of 0.1 parts per million (ppm) (0.5 milligrams per cubic meter, mg/m^3) for trichloroethylene based on neurological effects in rats. The MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure. Exposure to a level above the MRL does not mean that adverse health effects will occur. The MRL is intended to serve as a screening tool. (1)
- The California Environmental Protection Agency (CalEPA) has calculated a chronic inhalation reference exposure level of $0.6 \text{ mg}/\text{m}^3$ based on neurological effects in humans. The CalEPA reference exposure level is a concentration at or below which adverse health effects are not likely to occur. (5)

Reproductive/Developmental Effects:

- A study of nurses occupationally exposed by inhalation to trichloroethylene along with other chemicals in operating rooms, and another epidemiological study of women exposed occupationally or nonoccupationally to trichloroethylene and other solvents, have reported increases in the incidence of miscarriages. The presence of other chemicals, however, limits the ability to draw conclusions specific to trichloroethylene. (1)
- An epidemiological study of 2,000 male and female workers exposed to trichloroethylene via inhalation found no increase in malformations in babies born following exposure. (1)
- Several studies have evaluated and not found an association between adverse reproductive effects in humans and exposure to trichloroethylene in contaminated drinking water. An association was found between the occurrence of congenital heart disease in children and a drinking water supply contaminated

with trichloroethylene and other similar chemicals; however, no causal relationship with trichloroethylene could be concluded. (1)

- Animal studies have reported developmental effects from exposure to trichloroethylene and its metabolites (trichloroacetic acid [TCA] and dichloroacetic acid [DCA]). (1,4,13)

Cancer Risk:

- The cancer epidemiology for trichloroethylene has grown in recent years with several large, well-designed studies being published. A recent analysis of available epidemiological studies reports trichloroethylene exposure to be associated with several types of cancers in humans, especially kidney, liver, cervix, and lymphatic system. Consistency across epidemiological studies is strongest for an association between trichloroethylene exposure and kidney cancer. These results are supported by recent molecular epidemiology studies showing specific renal cell mutations found primarily in renal cell carcinoma patients exposed to trichloroethylene. (13)
- Animal studies have reported increases in lung, liver, kidney, and testicular tumors and lymphoma from inhalation and oral exposures in rats and mice. (1,4,13)
- EPA does not currently have a consensus classification for the carcinogenicity of trichloroethylene. However, the Agency is currently reassessing its potential carcinogenicity, and new data suggest that trichloroethylene is a likely human carcinogen. (11,13)
- EPA uses mathematical models, based on animal studies, to estimate the probability of a person developing cancer from continuously breathing air containing a specified concentration of a chemical. EPA has calculated a provisional inhalation unit risk estimate of $1.7 \times 10^{-6} (\mu\text{g}/\text{m}^3)^{-1}$. A provisional value is one which has not received Agency-wide review. EPA is currently reassessing the inhalation unit risk estimate. (10)
- EPA has also calculated a provisional oral cancer slope factor of $0.011 (\text{mg}/\text{kg}/\text{d})^{-1}$. EPA is currently reassessing the oral cancer slope factor. (10)

Physical Properties

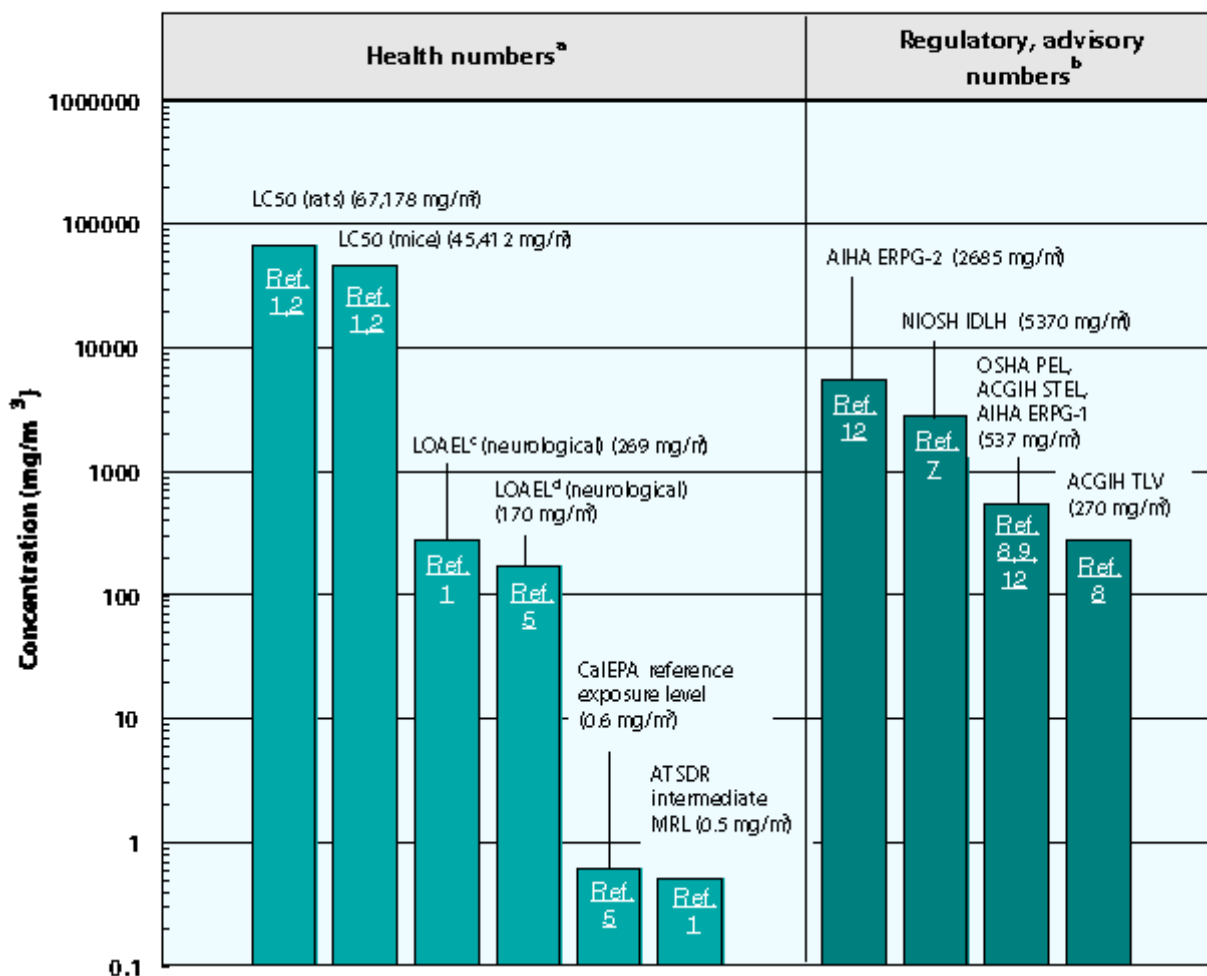
- Trichloroethylene is a nonflammable colorless liquid with a sweet odor similar to ether or chloroform. (1)
- The odor threshold for trichloroethylene is 28 ppm. (6)
- The chemical formula for trichloroethylene is C_2HCl_3 , and the molecular weight is 131.40 g/mol. (1) The vapor pressure for trichloroethylene is 74 mm Hg at 25 °C, and it has a log octanol/water partition coefficient ($\log K_{ow}$) of 2.42. (1)
- Trichloroethylene is not a persistent chemical in the atmosphere; its half-life in air is about 7 days. (1)

Conversion Factors:

To convert concentrations in air (at 25°C) from ppm to mg/m^3 : $\text{mg}/\text{m}^3 = (\text{ppm}) \times (\text{molecular weight of the compound}) / (24.45)$. For trichloroethylene: $1 \text{ ppm} = 5.37 \text{ mg}/\text{m}^3$. To convert concentrations in air from $\mu\text{g}/\text{m}^3$ to mg/m^3 : $\text{mg}/\text{m}^3 = (\mu\text{g}/\text{m}^3) \times (1 \text{ mg}/1,000 \mu\text{g})$.

Health Data from Inhalation Exposure

Trichloroethylene



ACGIH STEL--American Conference of Governmental and Industrial Hygienists' short-term exposure limit; 15-min time-weighted-average exposure that should not be exceeded at any time during a workday even if the 8-h time-weighted-average is within the threshold limit value.

ACGIH TLV--ACGIH's threshold limit value expressed as a time-weighted average; the concentration of a substance to which most workers can be exposed without adverse effects.

AIHA ERPG--American Industrial Hygiene Association's emergency response planning guidelines. ERPG 1 is the maximum airborne concentration below which it is believed nearly all individuals could be exposed up to one hour without experiencing other than mild transient adverse health effects or perceiving a clearly defined objectionable odor; ERPG 2 is the maximum airborne concentration below which it is believed nearly all individuals could be exposed up to one hour without experiencing or developing irreversible or other serious health effects that could impair their abilities to take protective action.

LC₅₀ (Lethal Concentration₅₀)--A calculated concentration of a chemical in air to which exposure for a specific length of time is expected to cause death in 50% of a defined experimental animal population.

NIOSH IDLH--National Institute of Occupational Safety and Health's immediately dangerous to life or health concentration; NIOSH recommended exposure limit to ensure that a worker can escape from an exposure condition that is likely to cause death or immediate or delayed permanent adverse health effects or prevent escape from the environment.

OSHA PEL--Occupational Safety and Health Administration's permissible exposure limit expressed as a time-weighted average; the concentration of a substance to which most workers can be exposed without adverse effect averaged over a normal 8-h workday or a 40-h workweek.

The health and regulatory values cited in this factsheet were obtained in December 1999.

^a Health numbers are toxicological numbers from animal testing or risk assessment values developed by EPA.

^bRegulatory numbers are values that have been incorporated in Government regulations, while advisory numbers are nonregulatory values provided by the Government or other groups as advice. OSHA numbers are regulatory, whereas NIOSH, ACGIH, and AIHA numbers are advisory.

^cThe LOAEL is from the critical study used as the basis for the ATSDR intermediate MRL.

^dThe LOAEL is from the critical study used as the basis for the CalEPA chronic reference exposure level.

Summary created in April 1992, updated in January 2000

References

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