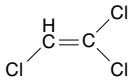
Trichloroethylene

CAS No. 79-01-6

Reasonably anticipated to be a human carcinogen

First listed in the *Ninth Report on Carcinogens* (2000)

Also known as 1,1,2-trichloroethene or TCE



Carcinogenicity

Trichloroethylene is reasonably anticipated to be a human carcinogen based on limited evidence of carcinogenicity from studies in humans, sufficient evidence of carcinogenicity from studies in experimental animals, and information from studies on mechanisms of carcinogenesis.

Cancer Studies in Humans

Evidence for the carcinogenicity of trichloroethylene in humans comes from seven cohort studies with specific trichloroethylene exposure well characterized for individual study subjects. A meta-analysis of these cohort studies found that occupational exposure to trichloroethylene was associated with excess incidences of liver cancer, kidney cancer, non-Hodgkin's lymphoma, prostate cancer, and multiple myeloma, with the strongest evidence for the first three types of cancer. Elevated risks of death from Hodgkin's disease, multiple myeloma, cervical cancer, and liver cancer also were observed. Nevertheless, these studies are based on a relatively small number of exposed workers and were confounded by exposure to other solvents and other cancer risk factors. Findings from other cohort studies, with less accurate assessment of trichloroethylene exposure, were more variable. In case-control studies, exposure to trichloroethylene was assessed less accurately; in many studies, it was estimated from exposure to solvents in general. These studies typically reported higher cancer rates for tissue sites similar to those observed in the cohort studies. Elevated risks were most consistently observed for kidney cancer, liver cancer, Hodgkin's disease, non-Hodgkin's lymphoma, and cervical cancer (Wartenberg *et al.* 2000).

Cancer Studies in Experimental Animals

Trichloroethylene caused tumors in mice and rats at several different tissue sites by two different routes of exposure. In mice, exposure to trichloroethylene by inhalation or by stomach tube caused benign and malignant liver tumors (hepatocellular adenoma and carcinoma) in both sexes (NCI 1976, Maltoni *et al*. 1988, NTP 1990, IARC 1995); inhalation exposure also caused lung tumors in both sexes and lymphoma in females (Henschler *et al*. 1980, IARC 1995). In rats, exposure to trichloroethylene by inhalation or by stomach tube caused kidney cancer (tubular adenocarcinoma) and testicular tumors (interstitial-cell tumors) in males (Maltoni *et al.* 1988, NTP 1988, 1990); leukemia also was observed in males whose survival was reduced after exposure by stomach tube (Maltoni *et al.* 1988).

Studies on Mechanisms of Carcinogenesis

Trichloroethylene is rapidly absorbed from the stomach, intestines, and lungs (IARC 1976, 1979, 1995, NTP 2000). It is distributed throughout the body and concentrates in fatty tissues, such as the liver, brain, and body fat. Trichloroethylene is metabolized primarily through oxidation by cytochrome P450 and conjugation with glutathione. Trichloroethylene metabolism in mice, rats, and humans is qualitatively similar, producing the same primary metabolites, which include chloral hydrate, dichloroacetic acid, and trichloroacetic acid. These primary toxic metabolites, produced by the P450 pathway, are associated with liver and lung toxicity in rats and mice. Another metabolite produced by the glutathione pathway, dichlorovinylcysteine, is associated with kidney toxicity. Dichlorovinylcysteine can undergo bioactivation in the kidney to chemical species that can react with cellular proteins; however, the National Research Council reported that no studies were available on its carcinogenic potential (NRC 2006).

Kidney tumors (renal-cell carcinoma) from workers occupationally exposed to high levels of trichloroethylene exhibited somatic mutations of the von Hippel-Lindau tumor-suppressor gene (*VHL*), which has been associated with renal-cell carcinoma (Brauch *et al.* 1999). Mutations in *VHL* were found in 75% of renal-cell carcinomas from 44 trichloroethylene-exposed workers. DNA sequencing analysis showed that 39% of these tumors had a specific mutation, a C to T transition at nucleotide 454, resulting in a change from proline to serine at codon 81. In four patients, the nucleotide 454 mutation was also found in the nearby noncancerous kidney tissue. Moreover, this mutation was specific to trichloroethylene exposure, because it was not found in renal-cell tumors from patients not exposed to trichloroethylene, and it was related to the disease, because it was not found in germline DNA from either diseased or nondiseased individuals.

It is reasonable from a biological perspective that the kidney tumors observed in humans are related to trichloroethylene exposure because (1) the site and histopathological characteristics of the tumors in humans and experimental animals are similar (Vamvakas *et al.* 1993, NRC 2006), (2) a portion of the molecular mechanism of this type of cancer (nephrocarcinogenicity) has been discovered (Dekant *et al.* 1986, IARC 1995, Bernauer *et al.* 1996), (3) the metabolites derived from trichloroethylene (the likely ultimate electrophilic intermediates of its bioactivation) are identical in humans and experimental animals (Birner *et al.* 1993, Clewell *et al.* 1995, IARC 1995), and (4) taking the key urinary metabolites (mercapturic acids) as an indicator of the bioactivation of trichloroethylene (Birner *et al.* 1993), humans appear to be more sensitive than rats in developing the primary biochemical lesion that precedes kidney cancer.

In general, trichloroethylene and most of its major metabolites (chloral hydrate, dichloroacetic acid, and trichloroacetic acid) are not potent genotoxicants in a broad range of bacterial, lower eukaryotic, and *in vitro* and *in vivo* mammalian test systems (NTP 2000). In mammalian cell-culture studies, trichloroethylene did not cause chromosomal aberrations in Chinese hamster ovary (CHO) cells, unscheduled DNA synthesis in rat hepatocytes, or gene mutations in human lymphoblastoid cells, but it did cause sister chromatid exchange in CHO cells, gene mutations in mouse lymphoma cells, and morphological transformation of rat embryo cells. In rodent *in vivo* studies, trichloroethylene did not induce unscheduled DNA synthesis, sister chromatid exchange, dominant lethal mutations, or chromosomal aberrations. Trichloroethylene gave mixed results for DNA single-strand breaks or alkali-labile sites in mouse liver and for micronucleus formation in mice. Studies of chromosomal aberrations, aneuploidy, and sister chromatid exchange in peripheral lymphocytes of workers exposed to trichloroethylene were inconclusive. The trichloroethylene metabolite dichlorovinylcysteine was mutagenic in *Salmonella typhimurium* and may cause DNA damage in mammalian cells *in vitro* and *in vivo*.

Properties

Trichloroethylene is a halogenated alkene that exists at room temperature as a clear, colorless, or blue mobile liquid with an ethereal odor. It is slightly soluble in water, soluble in ethanol, acetone, diethyl ether, and chloroform, and miscible in oil. It is relatively stable, but oxidizes

slowly when exposed to sunlight in air (IARC 1976). Upon combustion, trichloroethylene produces irritants and toxic gases, which may include hydrogen chloride. In the presence of moisture and light, it breaks down into hydrochloric acid (HSDB 2009). Physical and chemical properties of trichloroethylene are listed in the following table.

Property	Information
Molecular weight	131.4
Specific gravity	1.4642 at 20°C/4°C
Melting point	–84.7°C
Boiling point	87.2°C
Log K_{ow}	2.61
Water solubility	1.28 g/L at 25°C
Vapor pressure	69 mm Hg at 25°C
Vapor density relative to air	4.53

Source: HSDB 2009.

Use

Trichloroethylene is used mainly as an intermediate for hydrofluorocarbon production (67%) and as a degreaser for metal parts (30%) (CMR 2005). The remaining 3% is used primarily as a modifier for polyvinyl chloride polymerization. Five main industrial groups use trichloroethylene in vapor or cold degreasing operations: furniture and fixtures production, fabricated metal products, electrical and electronic equipment, transport equipment, and miscellaneous manufacturing industries. Trichloroethylene is used as a solvent in the rubber industry, adhesive formulations, dyeing and finishing operations, printing inks, paints, lacquers, varnishes, adhesives, and paint strippers (IARC 1995). Trichloroethylene has also been used as an extraction solvent for natural fats and oils, as a solvent in extracting spices, hops, and decaffeinated coffee, and as an anesthetic and analgesic in obstetrics and for minor surgical procedures.

Production

Two U.S. companies had a combined annual trichloroethylene production capacities of 160,000 tons (320 million pounds) in 1992 (IARC 1995) and 330 million pounds in 2005 (CMR 2005). In 2009, trichloroethylene was produced by 22 manufacturers worldwide, including two in the United States (SRI 2009), and was available from 103 suppliers, including 39 U.S. suppliers (ChemSources 2009). Reports filed under the U.S. Environmental Protection Agency's Toxic Substances Control Act Inventory Update Rule indicated that U.S. production plus imports of trichloroethylene totaled between 100 million and 500 million pounds betwen 1986 and 2006 except in 1994, when the quantity was between 50 million and 100 million pounds (EPA 2004, 2009). U.S. imports of trichloroethylene have generally increased since 1989, with a low of 329,000 kg (705,000 lb) in 1992 and a high of 27.2 million kilograms (60 million pounds) in 2007. During this period, U.S. exports of trichloroethylene ranged from a low of 16.6 million kilograms (36.7 million pounds) in 2005 to a high of 48.7 million kilograms (107.4 million pounds) in 1992. Exports in 2007 were 25 million kilograms (55 million pounds).

Trichloroethylene is reported to occur naturally in some algae in temperate to tropical climates and in one red macroalga (IARC 1995).

Exposure

Inhalation is the main route of potential exposure to trichloroethylene. Trichloroethylene is a major ingredient in numerous consumer products. For example it is listed as a major ingredient of 12 household aerosol products, constituting 80% to 100% of three products and 90% to 99% of two other products used as cleaners or degreasers and intended for use primarily in hobbies, crafts, and home maintenance (HPD 2009). Trichloroethylene has also been present in typewriter correction fluids, paint removers and strippers, adhesives, spot removers, and rug-cleaning fluids (Gist and Burg 1995). Trichloroethylene is no longer used as an extraction solvent for cosmetics or drug products or as a drycleaning agent (IARC 1995).

Trichloroethylene was identified in 72 food items in the U.S. Food and Drug Administration's Total Diet Study, including fruits, beverages, and many foods prepared with oils and fats. The highest mean concentration was found in samples of raw avocado (FDA 2006). Other studies also have found trichloroethylene in a variety of foods, with the highest levels in meats and margarine. Trichloroethylene was at one time used extensively as a solvent for extraction of natural fats and oils, spices, hops, and caffeine (from coffee), but the FDA imposed limitations on these uses in 1977. The trichloroethylene found in foods is believed to come from the use of contaminated water in food processing or from food-processing equipment cleaned with trichloroethylene (ATSDR 1997).

According to EPA's Toxics Release Inventory, environmental releases of trichloroethylene have declined steadily since 1988, when over 57 million pounds was released. In 2008, 306 facilities released a total of 3.6 million pounds of trichloroethylene. Of these facilities, 256 reported point- or area-source releases to air ranging from 0.22 to nearly 264,000 lb, and 15 facilities released a total of 435 lb to water (TRI 2010). Mean background levels of trichloroethylene in air were reported to range from 0.03 ppb (0.16 μg/m^3) in rural areas to 0.46 ppb (2.5 μg/m^3) in urban and suburban areas. In areas near emission sources, trichloroethylene was measured in air at concentrations of up to 1.2 ppb (6.4 μg/m^3) (ATSDR 1997). From 1985 to 1998, EPA's Aerometric Information Retrieval System obtained 1,200 ambient-air measurements of trichloroethylene from various state and local environmental agencies in 25 states. In 1998, 115 monitors in 14 states detected trichloroethylene at a mean concentration of 0.88 μg/m^3 (range = 0.01 to 3.9 μg/m^3). Based on this average concentration and a daily inhalation rate of 20 m^3 of air, the daily inhalation exposure to trichloroethylene was estimated at 18 μg (Wu and Schaum 2000).

Trichloroethylene concentrations were measured during EPA's large-scale Total Exposure Assessment Methodology studies conducted in Maryland, New Jersey, and California from 1981 through 1987 (Wallace *et al.* 1996). Trichloroethylene exposure concentrations were measured with personal air monitors carried by 750 individuals for 24 hours. The median personal air concentrations were 0.3 to 3.0 μg/m^3. Expired-breath samples taken from the same subjects in the evenings after several hours at home (from 50 to 350 individuals in two New Jersey cities in 1981 to 1983 and 75 individuals in two California towns in 1984) contained trichloroethylene at concentrations of 0.1 to 0.9 μg/m^3 (the median personal air concentrations ranged from 1.7 to 3.0 μg/m^3). However, trichloroethylene was not detected in the breath of 140 individuals in Los Angeles, California, in 1984 or 1987 (with trichloroethylene personal air levels ranging from 0.3 to 1.2 μg/m^3) or in the breath of 75 individuals in Baltimore, Maryland, in 1987 (with trichloroethylene personal air levels averaging 1.1 μg/m^3). As part of the Minnesota Children's Pesticide Exposure Study, personal, indoor-air, and outdoor-air trichloroethylene concentrations were measured in 284 households with children. The median values for indoor, outdoor, and personal sampling all were between 0.5 and 1 μg/m^3 (Adgate *et al.* 2004).

Trichloroethylene is a common groundwater and drinking-water contaminant (Gist and Burg 1995, IARC 1995, ATSDR 1997, Wu and Schaum 2000). Industrial wastewater is a source of trichloroethylene released into surface-water systems. Trichloroethylene background levels in 1995 were 0.001 ppb (μg/L) in the Gulf of Mexico, 0.007 ppb in the northeastern Atlantic Ocean, and 0.0008 to 0.039

ppb in rainwater and snow (Gist and Burg 1995). In EPA's Contract Laboratory Program Statistical Database, trichloroethylene occurred in about 3% of surface-water samples and 19% of groundwater samples (IARC 1995). Trichloroethylene readily volatilizes from tap water, and exposure from inhalation of volatilized trichloroethylene from contaminated tap water may equal or exceed the exposure from ingestion of contaminated drinking water. One study estimated that inhalation exposure from a 10-minute shower in trichloroethylene-contaminated water would equal the exposure expected from drinking the contaminated water. Bathing in contaminated water would also contribute a significant fraction of an individual's total dermal exposure. Based on a trichloroethylene concentration of 3.0 µg/L (the median concentration in a large California water survey) and daily water consumption of 2 L, average daily trichloroethylene exposure through ingestion of drinking water was estimated as 6 µg (Wu and Schaum 2000). This estimate is consistent with ATSDR's (1997) estimate of 2 to 20 µg for the general population.

In the Third National Health and Nutrition Examination Survey (conducted from 1988 to 1994), trichloroethylene concentrations were measured in whole-blood samples from 677 people of all ages, races, genders, and geographical regions who were not occupationally exposed to trichloroethylene. The results suggested that 10% of the U.S. population had detectable levels of trichloroethylene in their blood (detection limit = 0.01 µg/L) (Wu and Schaum 2000). Assuming that people in whose blood TCE was not detected had blood concentrations of half the detection limit, the mean concentration of trichloroethylene in blood was 0.017 µg/L.

The National Occupational Exposure Survey (conducted from 1981 to 1983) estimated that 401,373 workers at 23,225 facilities potentially were exposed to trichloroethylene (NIOSH 1990). During health hazard evaluations by the National Institute for Occupational Safety and Health, mean occupational exposure concentrations of trichloroethylene in air ranged from 1.3 mg/m^3 to 1,084 mg/m^3 (for short-term exposure); the highest mean concentration was for a degreasing operation (IARC 1995).

Regulations

Coast Guard, Department of Homeland Security

Minimum requirements have been established for safe transport of trichloroethylene on ships and barges.

Department of Transportation (DOT)

Trichloroethylene is considered a hazardous material, and special requirements have been set for marking, labeling, and transporting this material.

Environmental Protection Agency (EPA)

Clean Air Act

National Emissions Standards for Hazardous Air Pollutants: Listed as a hazardous air pollutant.
New Source Performance Standards: Manufacture of trichloroethylene is subject to certain provisions for the control of volatile organic compound emissions.
Urban Air Toxics Strategy: Identified as one of 33 hazardous air pollutants that present the greatest threat to public health in urban areas.

Clean Water Act

Designated a hazardous substance.
Effluent Guidelines: Listed as a toxic pollutant.
Water Quality Criteria: Based on fish or shellfish and water consumption = 2.5 µg/L; based on fish or shellfish consumption only = 30 µg/L.

Comprehensive Environmental Response, Compensation, and Liability Act

Reportable quantity (RQ) = 100 lb.

Emergency Planning and Community Right-To-Know Act

Toxics Release Inventory: Listed substance subject to reporting requirements.

Resource Conservation and Recovery Act

Characteristic Hazardous Waste: Toxicity characteristic leaching procedure (TCLP) threshold = 0.5 mg/L.
Listed Hazardous Waste: Waste codes for which the listing is based wholly or partly on the presence of trichloroethylene = U228, F001, F002, F024, F025, K018, K019, K020.
Listed as a hazardous constituent of waste.

Safe Drinking Water Act

Maximum contaminant level (MCL) = 0.005 mg/L.

Food and Drug Administration (FDA)

Maximum permissible level in bottled water = 0.005 mg/L.
Trichloroethylene may be used as a solvent in the manufacture of modified hop extract provided the residue does not exceed 150 ppm.
Trichloroethylene may be used as a solvent in the manufacture of specified foods with maximum residue levels ranging from 10 to 30 ppm.

Occupational Safety and Health Administration (OSHA)

While this section accurately identifies OSHA's legally enforceable PELs for this substance in 2010, specific PELs may not reflect the more current studies and may not adequately protect workers.
Permissible exposure limit (PEL) = 100 ppm.
Ceiling concentration = 200 ppm.
Acceptable peak exposure = 300 ppm (5 min in any 2 h).

Guidelines

American Conference of Governmental Industrial Hygienists (ACGIH)

Threshold limit value – time-weighted average (TLV-TWA) = 10 ppm.
Threshold limit value – short-term exposure limit (TLV-STEL) = 25 ppm.

National Institute for Occupational Safety and Health (NIOSH)

Recommended exposure limit (REL) = 25 ppm (10-h TWA).
Ceiling recommended exposure limit = 2 ppm (60-min ceiling) during use as an anesthetic agent.
Immediately dangerous to life and health (IDLH) limit = 1,000 ppm.
Listed as a potential occupational carcinogen.

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