Trichloroethylene

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Trichloroethylene (Trichloroethene, 1,1,2-Trichloroethylene, CAS no. 79-01-6) is a colorless volatile liquid. It is nonflammable, has a sweet odor of chloroform and evaporates quickly.

Usage and Exposure

Trichloroethylene is mainly used as a solvent for adhesives and for cleaning and degreasing metal parts, and for synthesis in the chemical industry [SCOEL].

Trichloroethylene was previously used as an anesthetic gas for dental and surgical procedures and in veterinary medicine. It was also used in the food and pharmaceutical industry as an extraction solvent for natural fats and palm, coconut and soybean oil as well as for spices, hops, and decaffeination of coffee. In the 1970s the United States Food and Drug Administration banned these uses of trichloroethylene because of its toxicity. It was also used in the dry cleaning industry until the 1950s when it was replaced by perchloroethylene, but it is still used as a spot remover [IARC].

Concern arose during the 1970s regarding environmental, health and safety implications of chlorinated solvents. This brought about regulations and controls that reduced the use and production of trichloroethylene. However, this declining trend was reversed in 1995 with the enactment of the Montreal Protocol on Substances that Deplete the Ozone Layer. Trichloroethylene was then required in increasing amounts in place of substances such as such as 1,1,1-trichloroethane (genklene) [IARC].

Industrial emissions have been responsible for distributing trichloroethylene in the environment. Potential environmental exposure to trichloroethylene through the air, drinking water and food has been reviewed [ATSDR].

Degreasing is the main source of occupational exposure to trichloroethylene.

Routes of exposure

Trichloroethylene is easily absorbed through inhalation, skin contact and ingestion.

In occupational settings the inhalation of vapors is the main rout of exposure, but exposure frequently occurs when the skin is in contact with the liquid or vapors, or with contaminated clothing.

The substances may be ingested accidentally in occupational settings or in instances of substance abuse.

Target Organs

There is strong evidence that trichloroethylene is toxic to the central nervous system, immune system, reproductive and developmental systems, and to the kidneys.

Metabolism

Trichloroethylene is metabolized with subsequent production of numerous toxicologically active compounds. These include chloral hydrate, trichloroacetic acid, dichloroacetic acid, and dichlorovinyl glutathione. This complex assortment of metabolic compounds is transported across multiple tissues [Chiu].

Health hazards

Acute Effects

Inhalation can cause irritation of the respiratory tract. Dermal exposure to trichloroethylene causes irritation with erythema. Local effects following ingestion of trichloroethylene include dyspepsia, gastritis and diarrhea [PHE].

Reported neurological effects that have been associated with substantial exposure to trichloroethylene include euphoria, giddiness, lethargy, confusion, subjective symptoms of vestibular impairment (dizziness, headache, nausea), difficulty swallowing, facial effects that indicate possible trigeminal nerve damage (including sensation deficits, jaw weakness, increased blink reflex latency), dysfunction of cranial nerves other than the trigeminal nerve, memory deficits, impaired hearing, impaired visual function, mood swings, muscle weakness, tremors, decreased psychomotor function, psychotic behavior, impaired cognitive function, and loss of consciousness [ATSDR].

Case studies have described cardiac arrhythmias that in some instances led to death following occupational exposure, poisoning, or anesthesia.

Chronic Effects

There is some evidence for trichloroethylene-induced hepatic effects in occupationally-exposed humans; however, limitations generally include lack of quantifiable exposure data and concomitant exposure to other chemicals.

Some studies reported changes in blood and urine indices of liver function and enlarged livers in persons occupationally exposed to trichloroethylene. Liver effects were observed only in cases where exposure levels were likely to be higher than present-day occupational exposure limits.

Renal toxicity indicated by changes in urinary proteins and N-acetyl-β-d-glucosaminidase (NAG) was noted in workers exposed to trichloroethylene and other chemicals in the workplace. Changes in urinary proteins were also observed in renal cancer patients who were reported to have been exposed to trichloroethylene [ATSDR].

Dermal effects in persons occupationally exposed to trichloroethylene may be sensitivity reactions (termed Stevens-Johnson syndrome) in many cases and may include effects on mucous membranes. Other immunological effects observed in occupational settings include decreased numbers of total lymphocytes and selected lymphocyte subsets in blood samples from workers exposed to trichloroethylene that was used for cleaning a variety of materials and products, altered serum inflammatory cytokine levels, and decreases in serum IgG and IgM.

There is some evidence for association between occupational exposure to trichloroethylene and the occurrence of scleroderma (systemic sclerosis, a chronic autoimmune disease primarily of the skin).

Suggestive evidence of an association between exposure to trichloroethylene and adverse female reproductive outcomes includes reports of reduced fecundability and menstrual cycle disturbances (including amenorrhea). Evidence of trichloroethylene-induced effects in occupationally-exposed men includes reports of decreased potency, altered sex drive or function, decreased sperm quality, and decreased serum levels of reproductive hormones.

Some human studies indicate that trichloroethylene may cause developmental effects such as spontaneous abortion, congenital heart defects, central nervous system defects, and low birth weight [ATSDR].

Human data provide inconclusive evidence for the genotoxicity of trichloroethylene. Results of testing in mammalian and nonmammalian test systems indicate a potential for trichloroethylene to induce chromosomal damage. The weight of evidence suggests that trichloroethylene does not act directly as a mutagenic agent, but that the observed mutagenic responses are likely due to production of mutagenic metabolites such as chloral/chloral hydrate, dichlorovinyl cysteine, and dichlorovinyl glutathione [ATSDR].

Carcinogenicity

The International Agency for Research on Cancer (IARC) concluded that there is sufficient evidence in humans for the carcinogenicity of trichloroethylene and that it is carcinogenic to humans. Trichloroethylene causes cancer of the kidneys. A positive association has been observed between exposure to trichloroethylene and non-Hodgkin lymphoma and liver cancer.

Trichloroethylene is carcinogenic to humans (Group 1) [IARC].

References:

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