

Pollution Control and Safety

Environmental aspects :

Carbon disulfide in the atmosphere is mainly of biogenic and industrial origin. Tropospheric concentrations have been reported to be around 3×10^{-5} ppm in remote areas and $7-30 \times 10^{-5}$ in urban and industrial locations. Natural release includes diffuse emission from soil and volcanic eruptions. Industrial sources consist mainly of release from gasification and pyrolysis of carbonaceous fuel in the presence of sulfur compounds, from sulfur recovery (Claus) plant tail gas, and from industrial users of carbon disulfide.

Investigations on the fate of carbon disulfide in the atmosphere show that the principal oxidation products are sulfur dioxide, carbonyl sulfide and carbon monoxide. Eventually these compounds degrade to carbon dioxides and sulfates.

Carbon disulfide may be removed from gas streams by oil absorption or adsorption with activated carbon. A common method used in the treatment of carbon disulfide in sulfur recovery plant tail gas involves hydrogenation and hydrolysis of these compounds to carbon dioxide and hydrogen sulfide. The hydrogen sulfide is then recovered by absorption in alkanolamine solutions.

Storage and Transportation :

Due to the high degree of flammability and flammability of carbon disulfide, strict precautions must be adhered to its handling; for instance, the liquid will flash when brought into contact with a hot steam pipe. Since carbon disulfide is heavier than water, it is only slightly soluble in water, it is often stored and transported under a layer of water. Strict ICC regulations have been setup regarding loading, handling, transporting, and labeling of containers. Shipment is usually made in steel drums or tank cars. Carbon disulfide is very toxic and is harmful to the human system by inhalation of the vapor, by prolonged or repeated contact of the liquid with the skin, or by ingestion. Acute poison may result by inhalation of high concentrations of carbon disulfide vapor.

Exposure to concentrations of about 1200 ppm may produce head ache, nausea, dizziness and if prolonged, circulatory collapse, and unconsciousness may result. A concentration of 4000ppm may prove fatal after a period of 1/2 to 1 hr. carbon disulfide poisoning generally occurs as a result of continued exposure to a relatively small concentration of the vapor, and the signs and symptoms are the result of injury to the nervous system. Symptoms of poisoning include nervousness, headache indigestion, fatigue shortness of breath etc. signs of chronic poisoning by carbon disulfide vapor are a waxy pallor, low blood pressure, defective memory, signs of injury to the nervous system and in extreme cases, a mask like face and loss of control of normal bodily functions. About 15-20 ppm of carbon disulfide in air is considered the maximum allowable concentration for periods of exposure not exceeding 8 hrs. carbon disulfide in the atmosphere is mainly of biogenic and industrial origin. Tropospheric concentrations have been reported to be around 3×10^{-5} ppm in remote areas and $7-30 \times 10^{-5}$ in industrial locations.

Toxicology and Occupational Health :

➤ Mutagenicity and Genotoxicity :

The mutagenicity and genotoxicity potential of Carbon disulfide has been evaluated in vitro and invivo. Carbon disulfide does not exhibit mutagenic activity in bacteria (*S.typhimurium* and *E.coli*) with or without the presence of activation system. Additional in vitro tests, including host-mediated assay, unscheduled DNA synthesis in human fibroblasts and primary cultures of human leucocytes, are inconclusive. However the significance of these tests cannot be properly evaluated because of methodological problems including the lack of proper positive controls. Therefore insufficient data are currently available to evaluate the mutagenic and genotoxic potential of carbon disulfide.

➤ **Human Health Effects :**

Carbon disulfide toxicity in humans is well documented in literature. It was first recognized as a workplace hazard by the French in the 1850s. Subsequent studies demonstrated

effects on the central and peripheral nervous systems and cardiovascular system. Carbon disulfide can induce polyneuropathy and encephalopathy. Poisoning may be acute or chronic.

Symptoms of polyneuropathy include paresthesia, cramps, muscle weakness, pain, distal sensory loss and neurophysiological impairment. Exposure to high levels of carbon disulfide for prolonged periods is necessary to produce the polyneuropathy. However exposures at levels of 65-170 ppm for less than 1 year are reported to result in many of the symptoms described. Symptoms of encephalopathy may include headache, sleep disturbances, general fatigue, loss of libido, impotence, organic dementia and parkinsonism. Onset of symptoms range from several months to several years. The early stages of encephalopathy is characterized by depression with sudden attacks of hallucinations. Visual disturbances that may result from high levels of carbon disulfide exposure may result from high levels of carbon disulfide exposure include abnormal color vision, loss of visual acuity, loss of accommodation and retinopathy.

➤ **Disposition :**

The primary route of human carbon disulfide exposure is inhalation. Uptake in the blood is rapid and carbon disulfide is transported to the tissues. Carbon disulfide possesses significant lipophilicity while also binding to proteins and amino acids. In humans, most (>70%) of a systemically absorbed dose is rapidly converted to metabolites, followed by urinary excretion. Less than 30% of carbon disulfide is exhaled and very small quantities are excreted unchanged.

➤ **Biochemical Mechanisms :**

Understanding the mechanism of action of carbon disulfide toxicity may lead to early clinical intervention in cases of suspected poisoning. Although a number of hypotheses have been proposed, the biochemical mechanism(s) remain unknown. A metabolite of carbon disulfide may be responsible for its hepatotoxic effects. Sulfur is liberated during conversion of carbon disulfide to carbon dioxide. The liberated sulfur may bind to cellular components or critical enzymes, giving rise to toxicity.