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Toxicology Update

Note: The Toxicology Update represents a brief review of an often extensive literature base. Only some of the directly related references may be included.

VINYL CHLORIDE

Synonyms: Chloroethene, chloroethylene, vinyl chloride monomer, VCM, vinyl C monomer, ethylene monochloride, monochloroethylene.
CAS no: 75-01-4.
Boiling point: −13.37°C.
Color: Colorless liquid or gas.
Conversion factor: 1 ppm = 2.60 mg m^{-3}.
DOT designation: Flammable gas.
Flammability limits:
Autoignition, 472°C;
LEL, 4%;
UEL, 22%.
Henry's Law constant: 1.2 atm.m^3 mol^{-1} @10°C.
Melting point: −153.8°C.
Molecular formula: C_2H_3Cl.
Molecular weight: 62.50.
Odor: Threshold, 3.4 ppm (air) and 3000 ppm (water);
Characteristics, faintly sweet odor.
Solubility: In water, 1100–2763 mg l^{-1} @ 25°C,
K_{ow} = 1.38; very soluble in ether, soluble in alcohols and hydrocarbons.
K_{oc} = 57 ml g^{-1}.
Specific gravity: 0.9106 (20/4°C).
Vapor density: 2.15 (air = 1).
Vapor pressure: 2530 mmHg @ 20°C.
Viscosity: 0.01702 cP @ 20°C (gas) and 0.280 cP @ −20°C (liquid).
(References: 1–3.)

Composition

Vinyl chloride is a volatile chlorinated hydrocarbon, which, at room temperature, is a colorless gas having a mild, sweet odor. It is typically supplied as a pressurized liquid of technical-grade 99% purity.[4] Phenol is used as a stabilizer to prevent polymerization of vinyl chloride and is generally added at levels of 25–50 mg kg^{-1} vinyl chloride.[5]

Uses/sources

Vinyl chloride can be produced by thermally cracking ethylene dichloride or by chlorinating ethylene.[6] The vinyl chloride monomer is polymerized into polyvinyl chloride and used in the plastics industry to make a variety of products, including food covering, pipe, wire coating, furniture, housewares, wallcovering, packaging materials, car upholstery and car parts. A vinyl chloride–vinyl acetate copolymer is used in floor coverings, phonograph records and flexible film. A vinyl chloride–vinylidine chloride copolymer is used in food-packaging film. Vinyl chloride is also used as a refrigerant gas and as a chemical intermediate. it was formerly used in small amounts as an aerosol propellant and an ingredient in drugs and cosmetics.[2]

Vinyl chloride has the potential to migrate from PVC packaging into food (e.g. bottled drinking water). The migration rate appeared to be linear, and estimated exposure may exceed 100 ng per person per day.[7]

Acute toxicity

Ingestion: Ingestion is an unlikely route of exposure, despite the water solubility of this compound, due to the extremely high vapor pressure. However, the LD_{50} by gavage in rats is reported to be 500 mg kg^{-1}.[6]

Inhalation: Vinyl chloride may be tolerated at levels over 8000 ppm for up to 5 min without developing signs or symptoms of toxicity.[8] Inhalation of vinyl chloride may cause respiratory irritation, bronchitis, dizziness, incoordination, headache, irritability, afferent sensory polyneuritis, cardiac arrythmia, unconsciousness and death. Pulmonary effects include dyspnea, asthma, interstitial pneumonitis and pneumonoconiosis.[8] Vinyl chloride impairs the central nervous system (CNS) at high concentrations. Deaths in humans due to narcosis have been reported. However, the concentrations were unquantified.[2] Slight anesthesia, drowsiness, slight visual disturbances, faltering gait, numbness and tingling in the extremeties have been observed at 1000 ppm.[9] Inhalation of vinyl chloride at concentrations ranging from 8000 to 20000 ppm may produce dizziness, giddiness, euphoria, ataxia, headache and narcosis.[10]

The acute LC_{50} levels for rodents are presented in Table 1. Anesthesia was observed in dogs at 70 000 ppm.[11] Pulmonary edema, hemorrhaging, impaired blood clotting and liver and kidney congestion were also observed in laboratory animals following acute exposure to vinyl chloride.[12]

Eye contact: Vinyl chloride is a severe irritant to the eyes and mucous membranes.[6] Data regarding acute toxicity from eye contact was not located but effects associated with its ability to freeze tissue might be expected.

Skin contact: Liquid vinyl chloride placed on the skin may freeze tissue and produce a chemical burn as it evaporates, causing damage to the underlying tissue (frostbite). This chemical is considered to be a severe skin irritant.[6] Data regarding acute toxicity from skin contact was not located. Significant absorption from incidental skin contact is unlikely to result in systemic toxicity owing to the volatility of the compound.

Chronic toxicity

Ingestion: The liver is the primary target organ for vinyl chloride in humans and animals. Toxic effects in animals subject to chronic ingestion of vinyl chloride include enlargement of the liver and spleen, reduced blood-clotting time and development of angiosarcomas, adenosarcomas and heptacellular carcinomas.[14] Feron *et al.*[15] determined 30 mg kg^{-1} to be the no adverse effect level (NOAEL) and 100 mg kg^{-1} to be the lowest adverse effect level (LOAEL) in rats based upon liver effects during a 15-week gavage study. Slight histological changes in the hepatocytes were observed in the 100 mg kg^{-1} and 300 mg kg^{-1} dose levels. Hypertrophy of the rough endoplasmic reticulum was observed in both sexes in the 300 mg kg^{-1} dose group. A dose related increase in liver weights was also observed, with differences being significant in the 300 mg kg^{-1} dose level.

A lifetime feeding study by Til *et al.*[16] found the NOAEL and LOAEL to be 0.13 and 1.3 mg kg^{-1} day^{-1}, respectively, based upon hepatotoxicity. Heptacellular changes and hepatic cysts were observed in both sexes receiving 1.3 mg kg^{-1} day^{-1}. An increase in basophilic foci was observed in both sexes receiving 1.3 mg kg^{-1} day^{-1} but only in females receiving 0.13 and 0.014 mg kg^{-1} day^{-1}. The authors concluded that histopathological effects observed at 0.13 and 0.014 mg kg^{-1} day^{-1} were

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compound-related but not considered to be adverse effects. Feron *et al.*[17] also observed hepatic effects including preneoplastic changes in all treatment groups receiving dietary polyvinyl chloride at levels between 1.7 and 14.1 mg kg^{-1} day^{-1}. Thus the minimal risk level or Hazard Index, based upon hepatotoxicity, is estimated to be approximately 0.0013 mg kg^{-1} day^{-1},[18] after applying a standard default safety factor of 100 to the NOAEL determined by Til *et al.*[16]

Inhalation: Occupational exposure to vinyl chloride vapor has resulted in deaths due to narcosis and development of conditions known as 'vinyl chloride disease' and 'meat wrapper's asthma'.[8,18,19] Vinyl chloride disease has been reported in workers exposed to several hundred ppm for periods ranging 1 month to 3 years before the onset of symptoms.[8]

Symptoms of vinyl chloride disease include scleroderma of the connective tissue in the fingers with dermal thickening, a Raynaud-like condition with reversible arteriole constriction causing numbness, pallor and cyanosis of the fingers. This condition may be followed by acro-osteolysis (dissolution of the ends of the distal phalanges of the hand). Hematological, respiratory and hepatic effects, and increases in circulating IgG and immune complexes have also been observed.[20,21] However, no new cases of vinyl chloride disease have been reported in the US since 1974, when the occupational exposure levels were reduced to 1 ppm.[8]

Hepatotoxicity was observed in mice found dead after subchronic exposure (5–9 days) to 1000 ppm vinyl chloride.[22] Suzuki[23,24] observed proliferative hypertrophy of terminal bronchiolar cells, hyperplasia of alveolar epithelium, degeneration of alveolar septal cells and peribronchiolar or bronchiolar inflammation in mice exposed to 2500 or 6000 ppm during a 6-month period. Rats exposed to 5000 ppm for 1 year were observed to have hematological effects, reduced growth, increased splenic hematopoiesis, degeneration of the myocardium, thickening of the arterial walls, hyperplasia of the olfactory epithelium, nephrotoxicity and mild alteration of the lungs and zymbal glands.[25]

The LOAEL in a 6-month rat subchronic study was found to be 10 ppm based on hepatic effects.[26] The LOAEL based upon hepatic effects in a 1-year rat study was at 50 ppm, the lowest dose level. Consequently, the NOAEL could not be determined.[22] A subsequent 1-year rat study by Bi *et al.*[26] found the LOAEL and NOAEL to be 100 and 10 ppm respectively, based upon terminal body weights. The minimal risk level for 'intermediate exposures' to vinyl chloride is estimated to be 50 ppm based on these data.[18]

Eye contact: See Acute toxicity, Eye contact.

Skin contact: See Acute toxicity, Skin contact.

Sensitization

No information regarding the skin sensitization potential of vinyl chloride was located. However, Meat wrapper's asthma is thought to be attributed to the thermal degradation products emitted from heat-sealed vinyl chloride wrapping materials.[19]

Target organ toxicity

Cardiovascular effects: Impaired circulation in the extremities and Raynaud's syndrome have been observed in humans suffering from 'vinyl chloride' disease. Mild focal degeneration of the myocardium, portal hypertension, thickening of the arterial walls and tumors of the hepatic blood vessels have been observed in rats subject to chronic vinyl chloride exposure.[27]

Vinyl chloride has sensitized the animal heart to epinephrine-induced arrythmias and may increase ventricular fibrillation.[9]

Increased spleen weight, thrombocytopenia and changes in hematology parameters were observed in rats exposed to high levels of vinyl chloride for 1 year.[25]

Hepatic effects: Vinyl chloride has been associated with hepatotoxic effects in humans and animals. Signs of vinyl chloride liver injury include abdominal pain, weight loss, fatigue and weakness. Fibrosis and cirrhosis may occur and cause hepatomegaly, splenomegaly, portal hypertension, thrombocytopenia and esophageal varices.[22,28] Hepatic fibrosis is often observed in the tumor-free portions of liver in workers dying from angiosarcoma. However, the transition from hepatic fibrosis to angiosarcoma has not been proven.[8]

Increased levels of urinary porphyrin and coproporphyrin correlate with liver damage induced by vinyl chloride and other hepatotoxins.[29] Hepatotoxic effects may be reversible in occupationally exposed individuals. Between 1971 and 1982, 12 of 13 workers in a vinyl chloride polymerization plant were observed to have persistent abnormalities in one or more liver function tests. The vinyl chloride levels during this period ranged from 1 ppm to 21 ppm. In 1983 the vinyl chloride level was maintained below 1 ppm and no further cases of vinyl chloride-induced liver dysfunction were observed.[30]

Scleroderma on the back of the hand at the metacarpal and phalangeal joints and inside of the forearms has been observed in some chronically exposed individuals having angioneurotic disorders.[9]

In experimental animals, Torkelson *et al.*[31] observed hepatic lesions in rabbits and rats exposed for 6 months to 500 and 200 ppm, respectively. Lee *et al.*[22] reported increased hepatic cell turnover rates in rats exposed to 50 ppm for 8–9 months. No inhalation NOAEL for hepatotoxic effects has been reported. The ATSDR holds 50 ppm to be a Frank Effect Level (FEL).[18] Development of hepatotoxicity via ingestion of vinyl chloride appears to be significantly related to the duration of exposure given that the subchronic oral NOAEL is 30 mg kg^{-1},[15] over 200 times greater than the chronic oral NOAEL of 0.13 mg kg^{-1} day^{-1}.[16]

The liver is the primary target of vinyl chloride effects because it is believed that vinyl chloride is biologically activated in the liver by cytochrome P-450 IIE1 in the mixed-function oxidase system.[32] The mixed-function oxidase system oxidizes vinyl chloride into the reactive oxirane, 2-chloroethylene oxide. The oxirane then reacts and covalently bonds to nearby macromolecules, such as proteins or nucleic acids, impairing cell function or causing mutations. This hypothesis is supported by studies showing vinyl chloride metabolites covalently bound to macromolecules and vinyl chloride hepatotoxicity potentiation by P-450 inducers and suppression by P-450 inhibitors.[32,33]

Vinyl chloride has recently been implicated as a 'suicide inhibitor' of cytochrome P-450. Following activation by P-450 the reactive oxirane covalently binds to pyrrole nitrogens in the heme moiety, destroying the heme and reducing P-450 activity.[34]

Absorption–metabolism–excretion

Absorption in rats from the gastrointestinal tract is nearly complete, ranging between 80 and 90%.[35] Withey[36] found that blood levels peaked 10–20 min following gavage of a single 10-ml aliquot of 44–92 mg kg^{-1} vinyl chloride in an aqueous solution. Seventy-two hours after ingestion, the greatest concentration of vinyl chloride was found in the liver at concentrations two- to fivefold greater than in the lung, fat, muscle, skin or plasma.[35]

On average, humans retain 42% of inhaled vinyl chloride.[37] Animal data indicate that vinyl chloride is rapidly absorbed, but is insufficient to quantitate the proportion of dose absorbed.[18]

Butcher *et al.*[38] found that when metabolism was inhibited with 6-nitro-1,2,3-benzothiadiazole, the greatest levels of labeled vinyl chloride occurred in the fat. However, when metabolism was not inhibited the greatest levels of vinyl chloride were found in the liver and kidneys. Duprat *et al.*[39] conducted a study measuring tissue concentrations in rats of radiolabeled vinyl chloride following exposure to 20 000 ppm. Ten minutes after exposure, radioactivity was detected in the liver, bile duct, digestive tract and kidney. In rats, radio-labeled vinyl chloride was detected in order of decreasing activity in the liver, kidney, skin, lung, carcass, plasma and fat.[35]

Dermal absorption of vinyl chloride in animals is minimal at best. The bodies of rhesus monkeys were placed in a chamber in which they were exposed to 800 and 7000 ppm vinyl chloride while their heads were outside the chamber, permitting only dermal absorption. At 800 ppm, dermal absorption was 0.031% and 0.023% at 7000 ppm.[40]

Metabolism of vinyl chloride is believed to proceed by three alternative pathways; the extent of each is dependent upon vinyl chloride concentration. At low concentrations, the primary pathway appears to be sequential oxidation of vinyl chloride to 2-chloroethanol, 2-chloroacetalaldehyde and finally to 2-chloroacetic acid by alcohol dehydrogenase. Pretreatment with ethanol at exposure levels of <100 ppm resulted in an inhibition of >83%. However, pretreatment with ethanol prior to exposures of 1000 ppm resulted in inhibition by <47%, suggesting an alternative metabolic route to the alcohol dehydrogenase pathway.[18]

At greater concentrations, vinyl chloride may be oxidized by mixed-function oxidases to 2-chloroethylene oxide, with spontaneous rearrangement to 2-chloroacetalaldehyde, or oxidized by catalase to 2-chloroethylhydro-peroxide, followed by dehydration to form 2-chloroacetalaldehyde. Pretreatment with SKF-525A had no effect on vinyl chloride metabolism at concentrations of < 100 ppm. Metabolism of vinyl chloride was inhibited by 19% with SKF-525A pretreatment at exposure levels of 1000 ppm, indicating that the mixed-function oxidase pathway is significant only at higher concentrations.[41]

However, pretreatment with 6-nitro-1,2,3-bezothiadiazole completely inhibited vinyl chloride metabolism in rats exposed to 0.45 ppm, vinyl chloride for 5 h, suggesting that metabolism via the alcohol dehydrogenase pathway may require epoxidation by mixed-function oxidases.[42] Buchter *et al.*[41] found saturation of the metabolic pathways in rhesus monkeys to occur at 200 ppm and a V_{max} of 50 μmol h^{-1} kg^{-1}, which is less than that observed in rats exposed to 250 ppm with a V_{max} of 110 μmol h^{-1} kg^{-1}.[42]

Vinyl chloride is believed to be metabolized into a reactive epoxide intermediate (see Chronic Toxicity, Hepatic effects). A subsequent metabolite, 2-chlororacetalaldehyde, readily conjugates with sulfhydryl groups in proteins. If the glutathione-*S* transferase (GSH) detoxification pathway is saturated, then the aldehyde is free to bind to other proteins, impairing cell function.

The route of excretion is determined by the extent of exposure regardless of whether exposure is via ingestion, inhalation or interperitoneal or intravenous injection. Generally, as the dose increases, the proportion of unchanged vinyl chloride exhaled also increases.[43] At low exposure levels, the majority (ca.70% of the vinyl chloride is excreted in the urine.[39]

Exhalation of unchanged vinyl chloride is not a significant route of elimination by humans from exposure to low concentrations. Individuals were exposed to 2.9–23.1 ppm vinyl chloride for 6 h and breath samples were taken 30 min following exposure. Mean concentrations of exhaled vinyl chloride ranged from undetectable to 1.1 ppm.[37]

Genotoxicity

Vinyl chloride has tested positive in *in vitro* and *in vivo* mutagenicity assays. The mutagenicity data implicate 2-chloroethylene oxide and 2-chloroacetalaldehyde as being the mutagenic agents associated with the positive results. Both metabolites have been shown to be more effective than vinyl chloride in inducing mutations in *Salmonella typhimurium*.[44] Metabolic activation of vinyl chloride has been required to obtain maximal or any response.[45] Vinyl chloride has been shown to be mutagenic in *S. typhimurium* strains, which revert by base-pair substitutions from alkylating agents rather than frame-shift reversion strains.[46] Singer *et al.*[47] found that *N*-2,3-ethenoguanine is a product of vinyl chloride reaction with DNA *in vivo* and of chloroacetalaldehyde *in vivo*. This reaction led to increased G–A transition with *Escherichia coli* and *Drosophila melanogaster* polymerases, and human immunodeficiency virus (HIV) reverse transcriptase.

Vinyl chloride was positive in the recessive lethal assay but negative in the dominant lethal assay in *D. melanogaster*.[48] Vinyl chloride was negative in the mouse dominant lethal assay.[49] Vinyl chloride has been found to alkylate DNA in rats and mice.[50,51]

Chromosomal aberrations in human peripheral lymphocytes of exposed workers have been detected in many studies and breaks have been reported to be localized in specific chromosomes.[52,53] Andersen *et al.*[54] observed a decrease in chromosomal aberrations in exposed workers following a reduction in exposure from 50 ppm to <5 ppm.

Neurotoxicity

Inhalation of vinyl chloride produces anesthesia and narcosis. Chronic occupational exposure to levels of < 50 ppm has been associated with distal axonal neuropathy in the legs[55] and changes in electroencephalograms of workers exposed to vinyl chloride and other solvents.[56]

Diffuse degenerative lesions in the gray and white matter of the brain and atrophy of the cerebellar granular layer in rats exposed to 30 000 ppm for 4 h per day, 5 days per week have been reported.[57] Other neurotoxic effects include functional disturbances of the CNS with afferent sensory polyneuritis.[58] Brain neuroblastomas have been ovserved in chronic rat inhalation studies.[15]

Reproductive toxicity

Epidemiological data show a possible correlation between paternal occupational exposure to vinyl chloride and fetal loss.[59,60] An increase in CNS defects, deformaties of the upper alimentary and genital tracts and clubfoot were observed in stillborn and live children in three cities having vinyl chloride plants.[5]

Animal teratogenicity data are equivocal. No adverse maternal or teratogenic effects were observed in rats exposed to >500 ppm for 7–12-day intervals during organogenesis.[61,62] However, Mirkova *et al.*[63] observed evidence of fetotoxicity and teratogenicity in rats subject to a continuous exposure of 2.4 ppm. Evidence of fetotoxicity included early post implantation loss, reduced fetal weight, retarded ossification and fetal hematomas. Evidence of teratogenicity included anomalies of the brain and impaired liver functions.

Occupational exposure has been found to be associated with impaired sexual function in both sexes and impaired gynecological health in women in a Russian study. Ovarian dysfunction, benign uterine growths and prolapsed organs were reported in 77% of exposed women.[64] Bi *et al.*[26] observed a significant reduction in testicular weight in rats exposed to 100 and 300 ppm for 6 months.

Carcinogenicity

Vinyl chloride has been associated with cancer in humans and has been classified

Table 1. Acute lethal concentration values in several mammalian species[13]

Species	Concentration (ppm)
Guinea pig	LC_{100} = 100 000
Rabbit	LC_{50} = 230–800
Mouse	LC_{50} = 117–500
Rat	LC_{50} = 6000

Table 2. Occupational exposure: permissible and recommended worker exposure limits[2,85]

ACGIH:TLV	5 ppm
STEL	10 ppm
OSHA:TWA	1 ppm
PEL	5 ppm
NIOSH	0 ppm

as a Group 1 carcinogen by IARC and a Group A carcinogen by the EPA.[65]

Vinyl chloride was first associated with liver cancer in exposed workers in 1974 when rare liver angiosarcomas were detected in three workers in a single vinyl chloride polymerization plant.[66] Based upon a worldwide register of cases of angiosarcoma, the average length of exposure resulting in angiosarcoma is 18.3 years.[8] Fibrosis is typically found in tumor-free portions of the liver in workers dying of angiosarcoma, but transition from fibrosis to angiosarcoma has not been proven.[8] After 1974 occupational exposure levels were reduced to 1 ppm. No cases of hepatic angiosarcoma in workers exposed solely after this date have been recorded. However, a longer latency period may be required. Data on cases of angiosarcoma allegedly due to environmental contamination are equivocal owing to background incidence and insufficient sample size.[8]

Vinyl chloride has been implicated in other forms of cancer. Increased incidence of cancer of the brain, respiratory tract and digestive tract, hematopoietic/lymphopoietic cancers and malignant skin melanomas have been associated with occupational vinyl chloride exposure.[67,68]

Inhalation and ingestion of vinyl chlorde has produced cancer in laboratory animals. Maltoni *et al.*[69] observed an increased incidence in tumors in mice, rats and hamsters exposed to > 50 ppm. Liver angiosarcoma increased in all species; hepatomas, zymbal gland carcinomas, neuroblastomas, extrahepatic angiosarcomas and skin acanthomas were also noted. Drew *et al.*[70] detected an age effect upon carcinogenic response to vinyl chloride exposure in mice, rats and hamsters. Maximal carcinogenic response occurred if inhalation exposure was during the first year of life. Rats were also observed to have a biologically significant increased incidence of brain cancer.[25,69] Lung tumors were frequently observed in mice as the primary carcinogenic response following acute and chronic exposure.[71,72]

Epidemiology

Occupational exposure to vinyl chloride has been associated with an increased incidence of cancer, particularly liver angiosarcoma.[66] A recent Scandinavian study detected a nearly three-fold increase in liver cancer among vinyl chloride workers, which clearly correlated to time of initial exposure, duration of employment and estimated quantitative exposure.[73] Other cancers associated with occupational vinyl chloride exposure include cancer of the brain/CNS, lung/respiratory tract digestive tract, pancreas and hematopoietic/lymphocytic system, and malignant skin melanoma.[67,68] Cutaneous and epithelioid hemangioendotheliomas have also been reported in occupationally exposed individuals.[74,75]

A syndrome know as 'vinyl chloride disease' has been associated with occupational exposure (see Chronic toxicity, Inhalation).

Environmental fate

Air: Nearly all the vinyl chloride released into the environment reaches the atmosphere owing to its volatility. The primary sources of release are vinyl chloride and polyvinyl chloride manufacturers. Prior to 1975, an estimated 100 million kg $year^{-1}$ were released by PVC plants.[5] Hartmans *et al.*[76] estimated that 400 million lb of vinyl chloride were released worldwide during 1982. Other sources include release from landfills, combustion of PVC and tobacco smoke.[5,77]

Photodegradation of vinyl chloride in a reaction with hydroxide radicals is the primary means of removing vinyl chloride from the air. The EPA[2] estimates the half-life to be ca. 1.5–1.8 days. Breakdown products include hydrochloride, formaldehyde, formyl chloride, carbon monoxide, carbon dioxide, chloroacetalaldehyde, acetylene, chloroethylene, chloroacetylchloranil and water. In areas of photochemical smog, the half-life is substantially shorter (ca. 3–7 h).[9] Reaction with ozone, oxygen atoms and direct photolysis are insignificant degradation mechanisms.[2]

The high partition coefficient between air and water probably makes removal of vinyl chloride from air by wet deposition insignificant. Dry deposition is equally insignificant owing to the high vapor pressure of vinyl chloride.[2]

Water: Vinyl chloride volatizes from water relatively rapidly. The estimated half-life of vinyl chloride in a pond, lake and river is 43.3, 34.7 and 8.7h, respectively.[2] However, Lyman *et al.*[78] estimated the half-life to be 0.805 h. This estimate was based upon a Henry's Law constant of 0.0560 atm m^{-3}, a 1-m deep stream, a 3 m s^{-1} current and a wind velocity of 3 m s^{-1}. Factors affecting the half-life include reaeration rates and salt concentration. Photodegradation may be a significant removal mechanism in waters containing photosensitizers, such as humic acids.[9]

Chemical hydrolysis or oxidation is not expected to be a significant degradation mechanism. Microbial degradation under aerobic conditions has been shown to be insignificant.[2] However, biodegradation may be an important mechanism in groundwater where volatization cannot occur. Under aerobic conditions, 99% of radiolabeled vinyl chloride was degraded after 108 days in an aquifer; 65% was converted to carbon dioxide.[79] Degradation products in groundwater also include trichloroethylene, tetrachloroethylene and 1,1,1-trichloroethane.[80]

Soil: Vinyl chloride is expected to volatize readily from dry or wet soil owing to its high vapor pressure. The half-life of vinyl chloride placed in 1 cm and 10 cm of dry soil is estimated to be 2½ and 12 h, respectively.[2]

The soil adsorption coefficient (K_{oc}) for vinyl chloride is estimated to be in the range 17–131, indicating high soil mobility and potential to reach groundwater.[78,81]

Environmental toxicity

Based solely on water solubility, vinyl chloride is estimated to have a bioconcentration factor (BCF) of 7. Thus, vinyl chloride is not expected to be significantly bioconcentrated in aquatic organisms. Other studies sugggest that the high vapor pressure and rapid volatization from water precludes bioconcentration, except in large releases,[82] and estimation of the median threshold limit (TLm) for aquatic species.

Regulatory status

Vinyl choride is an IARC and EPA carcinogen. However, OSHA does list permissible exposure levels (PELs), which are presented in Table 2.

Risk assessment: The EPA[65] estimated that the unit risk factor is 6.6 ×

10^{-5} ppb. The EPA[82] estimates risk based on vinyl chloride ($\mu g\ l^{-1}$) ingestion as follows:

Risk	2 l of water	6.5 g of fish
10^{-5}	20	5246
10^{-6}	2.0	525
10^{-7}	0.2	52.5

Air: Vinyl chloride has been classified as a hazardous air pollutant by the EPA pursuant to section 112 of the Clean Air Act (Title 40, Code of Federal Regulations (CFR) section 401.15; 40 CFR 61.01; 7/1/88). Vinyl cloride concentration in exhaust from vinyl chloride manufacturing or purification plants is not to exceed 10 ppm (40 CFR 61.65 (a); 7/1/89).

Water: Vinyl chloride has been designated a toxic water pollutant by the EPA under section 307(a)(1) of the Clean Water Act (40 CFR 401.15; 7/1/88). Under the Safe Drinking Water Act the EPA has established a maximum concentration limit (MCL) of 2 ppb (0.002 mg l^{-1}) (40 CFR 141.61; 7/1/88), the acute 1-day health advisory for vinyl chloride ingestion for a 10-kg child ingesting 1 l of water per day is 3 mg day^{-1}.[83] A concentration of 0.15 ppb in drinking water will result in a 10^{-6} risk for a 70kg adult ingesting 2 l per day for a lifetime.[83] The California Department of Health Services (DOHS) action MCL is 0.5 ppb.[83]

The use of vinyl chloride in all pesticide products, as an inert or active ingredient, has been cancelled or suspende.[9] The FDA has also banned the use of vinyl chloride as an aerosol propellant, and eliminated its use in drug products.[84] The FDA has also alerted food manufacturers to the need for monitoring packaging material containing vinyl chloride and has proposed limiting the vinyl chloride monomer in packaging to 5–50 ppm.[8]

The reportable quantity for vinyl chloride release is 1 lb/0.454 kg or more (54 FR 33419; 8/14/89). Vinyl chloride is subject to management as a hazardous waste under RCRA regulations and corresponding state regulations once it becomes a waste (40 CFR 261.33). Any residue, or contaminated soil, water or debris containing the released vinyl chloride is also considered hazardous waste (40 CFR 261.3(b)).

The US EPA, Region IX has promulgated a tiered action-level scheme to protect human health surrounding a California landfill. This scheme considered very young children, especially neonates, to be unusually sensitive to the carcinogenic action of vinyl chlorde.
0.2 ppbv: Long-term in-home exposures should be below this level in order to provide sufficient protection from unreasonable excess cancer risks. If indoor vinyl chloride levels exceed 0.2 ppbv, permanent action should be taken to reduce them to below this level.
0.2–10 ppbv: In the event that indoor vinyl chloride levels are found within this range permanent remedial action should be taken to bring levels below 0.2 ppbv. This action should be instituted and its effectiveness confirmed within 60 days.
10–100 ppbv: In the event that indoor vinyl chloride levels are found within this range, temporary corrective action should be taken to bring levels below 10 ppbv. This temporary action should then be followed by permanent remedial action as specified for the 0.2–10 ppbv range. Temporary action should be instituted and its effectiveness confirmed within 12 days.[86]

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