

Vinyl Chloride (VC)

Chemical Summary Form



U.S. EPA, Toxicity and Exposure Assessment for Children's Health

SPECIAL CONCERNS FOR CHILDREN FROM VINYL CHLORIDE

HUMAN EXPOSURE/EFFECTS

- ▶ A case control study on the possible association between the occurrence of central nervous system defects and parental exposure to vinyl chloride revealed no relationship (1).
- ▶ The incidence of miscarriage has been studied in wives of male workers with occupational exposure to vinyl chloride. Increased incidence of miscarriage was observed in one study (2), but not in another (3).
- ▶ The incidence of developmental defects in children living close to vinyl chloride contaminated industrial sites has been studied. A trend of increased incidence of nervous system defects (4) and birth defects (5) was noted.

EXPERIMENTAL ANIMAL EXPOSURE/EFFECTS

- ▶ Rats exposed prenatally to vinyl chloride showed greater DNA adduct formation (chemically-altered DNA molecules in which a chemical has physically bound to the DNA) during early life than during adulthood (6, 7). Greater DNA adduct formation was seen in liver and brain tissue in prenatally exposed rats during early life than during adulthood (6, 7). Several studies suggested that increased DNA adducts may contribute to greater susceptibility to the carcinogenic effects of vinyl chloride in early life (6, 7, 8).
- ▶ Potential reproductive toxicity of vinyl chloride has been investigated. One study showed that vinyl chloride exposure to male rats did not produce dominant lethal mutation in the sperm cells as measured by either preimplantation or postimplantation losses in pregnant rats (9). In another study, vinyl chloride exposure was found to have no effects on any maturation stage of spermatogenesis in the male mouse (10).
- ▶ Studies in animal models have investigated carcinogenic effects of prenatal and early life vinyl chloride exposure. A review article identified vinyl chloride as a transplacental carcinogen in the rat (11). A study in rats on effects of prenatal and early life inhalation exposure to vinyl chloride resulted in brain neuroblastoma, liver angiosarcoma, and hepatocarcinoma (12).

- ▶ One study in rats found no effects of prenatal vinyl chloride exposure on birth weight or incidence of external malformations (13). This result was also seen in another study using mice, rabbits, and rats, which showed no effects as a result of inhalation exposure to pregnant animals (14). Another study in mice, rabbits, and rats, which evaluated the effects of inhaled vinyl chloride monomer on embryonic and fetal development, found significant developmental anomalies and some adverse reproductive outcomes (15). Embryotoxic effects, such as increased fetal mortality, were seen in one study due to vinyl chloride exposure during the first third of pregnancy (16).
- ▶ No increased incidence of miscarriage was seen in a study of inhalation exposure in pregnant rats (13).
- ▶ One study demonstrated a higher incidence of tumors in adult hamsters, rats, and mice following brief inhalation exposure in early life than in later life (17). In the same three species, a second study found increased neoplasms of the liver, mammary gland, gastrointestinal tract, and skin following inhalation exposure for a duration of six months (8). A higher incidence of tumorigenesis was observed when treatment was initiated at birth rather than later in life (8).

CONSIDERATIONS FOR DECISION-MAKING

- ▶ The National-Scale Air Toxics Assessment, which is based on 1996 emissions data, estimated the median annual average VC concentration (18).
- ▶ The oral slope factor explicitly considers the increased risk of cancer from early lifestage exposure to VC (19).
- ▶ Exposure of children to VC frequently occurs from direct industrial releases. Vinyl chloride has also been found as a degradation product of chloroethylene solvents in landfill gas and groundwater.
- ▶ Vinyl chloride in contaminated groundwater can volatilize and contaminate indoor air. (20, 21)
- ▶ An alternate water supply, e.g. bottled water, should be considered where VC-contaminated ground water may be impacting drinking water.
- ▶ Consult “Child-Specific Exposure Factors Handbook,” EPA-600-00-002B, for factors to assess children’s drinking water consumption and inhalation rates.
- ▶ Childhood is a sequence of lifestages rather than a subpopulation, the distinction being that a subpopulation refers to a portion of the population, whereas a lifestage is inclusive of the entire population.

EXPOSURE¹

Exposure Media	Level of Concern²	Basis
Ambient Air	Medium	Polyvinyl chloride factories and other plastics manufacturing facilities can be a source of VC releases. VC can be released from poorly controlled incineration of chlorinated plastics. VC can also be released through volatilization from some waste landfills, usually as a degradation product from plastics or other chlorinated chemicals (i.e., chlorinated ethylenes).
Groundwater	Medium	Groundwater contamination can occur at hazardous waste sites and from landfills where VC is usually generated as a degradation product of chlorinated plastics or other chlorinated chemicals (i.e., chlorinated ethylenes).
Indoor Air	Medium	In homes/dwellings located above contaminated groundwater, VC is capable of migrating through soil and foundations to enter basements or living spaces. VC could also volatilize to indoor air from contaminated groundwater due to indoor water uses (e.g., showering, dishwashing, laundry).
Diet	Lower	High volatility usually prevents VC from entering the food chain.
Soil	Lower	VC does not partition to or accumulate in soils because of high volatility.
Sediment	Lower	VC does not partition to or accumulate in sediments because of high volatility.

¹ For more information about child-specific exposure factors, please refer to the Children's Exposure Factors Handbook (<http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=52047>).

² The Level of Concern category is a subjective determination by the TEACH Workgroup, U.S. EPA, that reflects potential exposure pathways, frequency of exposure, level of exposure, and current state of knowledge.

TOXICITY SUMMARY³ AND REFERENCE VALUES

Toxicity Summary: Experimental animal studies of partial lifetime exposure suggest VC lifetime cancer risk depends on age at exposure, with higher lifetime risks attributable to exposures at younger ages (19). Like adults with preexisting health conditions, fetuses, infants and young children may be unusually susceptible to the toxic effects of VC (22). Workers exposed to VC developed genetic damage (23). Developmental studies in animals have shown VC-DNA adduct formation in exposed weanlings (6, 7). Although controversial, some work (2) suggests that paternal exposure to vinyl chloride can result in an increased incidence of spontaneous abortions; other evidence is negative in this regard (24, 25). Evidence of immune effects (26) and lung and brain tumors in occupationally-exposed workers have also been reported (27). VC exposure has been associated with liver angiosarcoma in adult humans (28) and in prenatally exposed rats (12).

Carcinogenicity weight-of-evidence classification: Under 1986 Guidelines USEPA classified VC in category "A" (known human carcinogen) based upon epidemiological studies, supporting experimental animal evidence and genotoxicity evidence. Under proposed (1996) Cancer Guidelines, USEPA concluded that VC is a known human carcinogen by inhalation exposure based on human epidemiological data, and by analogy the oral route because of positive animal bioassay data and pharmacokinetic data allowing dose extrapolation across routes. VC is also considered highly likely to be carcinogenic by the dermal route because it is well absorbed and acts systemically (<http://www.epa.gov/iris/subst/1001.htm>, II.A.1).

U.S. EPA RfD for Chronic Oral Exposure: 3E-3 mg/kg-day, based on liver cell polymorphisms in adult rats, with supporting early life animal studies used in the analysis (<http://www.epa.gov/iris/subst/1001.htm>, I.A.1). Last revised 8/7/00.

U.S. EPA RfC for Chronic Inhalation Exposure: 1E-1 mg/m³, based on liver cell polymorphisms in adult rats, with supporting early life animal studies used in the analysis (<http://www.epa.gov/iris/subst/1001.htm>, I.B.1). Last revised 8/7/00.

U.S. EPA Cancer Drinking Water Unit Risk: Continuous lifetime adult exposure, 2.1E-5 µg/L; continuous lifetime exposure from birth, 4.2E-5 µg/L. Derived using LMS and LED 10/linear extrapolation method (<http://www.epa.gov/iris/subst/1001.htm>, II.B.1.2) Last revised 8/7/00.

U.S. EPA Drinking Water Concentrations at Specified Risk Levels: 1E-4, 4.8 µg/L (adult exposure), 2.4 µg/L (exposure from birth); 1E-5, 4.8E-1 µg/L (adult), 2.4E-1 µg/L (from birth); 1E-6, 4.8E-2 µg/L (adult), 2.4E-2 µg/L (from birth) (<http://www.epa.gov/iris/subst/1001.htm>). Last revised 8/7/00.

U.S. EPA Drinking Water Advisories (10 kg child): One day, 3 mg/L; 10-day, 3 mg/L (<http://www.epa.gov/waterscience/drinking/standards/dwstandards.pdf>). Last revised summer, 2002.

U.S. EPA Inhalation Unit Risk: Continuous lifetime exposure during adulthood = 4.4E-6 per µg/m³ (based on LMS and LED 10/linear methods); continuous lifetime exposure from birth = 8.8E-5

per $\mu\text{g}/\text{m}^3$ (based on LMS and LED 10/linear methods)
(<http://www.epa.gov/iris/subst/1001.htm>). Last revised 8/7/00.

U.S. EPA Cancer Oral Slope Factor: Continuous lifetime adult exposure, $7.2\text{E}-1$ mg/kg-day (LMS method), $7.5\text{E}-1$ mg/kg-day (LED10 method); continuous lifetime exposure from birth, 1.4 mg/kg-day (LMS method), 1.5 mg/kg-day (LED10 method). Based on liver angiosarcoma, hepatocellular carcinoma, and neoplastic nodules in adults, with supporting developmental studies in animals (<http://www.epa.gov/iris/subst/1001.htm>). Last revised 8/7/00.

U.S. EPA MCL (drinking water): 0.002 mg/L, based on increased risk of cancer in adults.
<http://www.epa.gov/safewater/mcl.html#mcls>. Last revised 7/02.

U.S. EPA MCLG: 0 (zero; <http://www.epa.gov/safewater/mcl.html#mcls>). Last revised 7/02.

ATSDR Minimal Risk Level (MRL): 0.5 ppm (acute inhalation; developmental endpoint); 0.03 ppm (intermediate inhalation [15 to 364 days]; hepatic endpoint); 0.00002 mg/kg-day (oral chronic; hepatic endpoint) (<http://www.atsdr.cdc.gov/mrls.html#bookmark02>). Last revised 9/97.

³ The TEACH database focuses on information from studies of immature and/or developing organisms, e.g. mostly *excluding* workplace studies of adults. This toxicity summary is likely to *include* information from workplace or other studies of mature humans or experimental animals if child-specific (i.e. human epidemiology or developmental toxicity) information is lacking for the agent of interest.

REGULATORY INFORMATION

- ▶ Vinyl chloride is regulated in public drinking water supplies; the MCL is 0.002 mg/L (ppm). There are currently no ambient or indoor air regulatory levels for vinyl chloride (other than the OSHA standards that apply to workplace exposures).
- ▶ Vinyl chloride is one of the 188 hazardous air pollutants (HAPs) listed under Section 112(b) of the 1990 Clean Air Act Amendments and its emissions are regulated from more than 170 industrial air pollutant source categories.
- ▶ Under the Emergency Planning and Community Right-to-Know Act, the Reportable Quantity for vinyl chloride is 1 pound.

BACKGROUND ON CHEMICAL

CAS Number: 75-01-4

Physicochemical Properties: Go to www.ChemFinder.com and search for vinyl chloride.

Production: Vinyl chloride gas, formed by reacting ethylene or acetylene with hydrochloric acid, was once used as a propellant in aerosols but was banned from that application in 1974. Vinyl chloride is a breakdown product of trichloroethane, trichloroethylene, and tetrachloroethylene.

Uses: Vinyl chloride is used to make polyvinyl chloride (PVC). PVC is used to make a variety of plastic products, including pipes, wire and cable coatings, and furniture upholstery.

Environmental Fate: Liquid VC evaporates easily into air, and when near the surface of soil and water. Vinyl chloride can break down in the air within a few days, and the breakdown products can negatively impact health. Vinyl chloride is minimally soluble in water. Vinyl chloride is a degradation product of other chlorinated chemicals that may be present in groundwater, particularly TCE. It does not bioaccumulate in plants or animals.

Synonyms: Vinyl chloride monomer (VCM); chloroethene; chloroethylene; vinchloroethene; ethylene monochloride; monochloroethene; monochloroethylene.

Additional information on vinyl chloride is available in the TEACH Scientific Review Table for Vinyl Chloride, and at the following websites:

www.atsdr.cdc.gov/mrls.html

www.cdc.gov/default.htm

www.epa.gov/epahome/index.html

www.epa.gov/iris/

www.epa.gov/tri/

www.epa.gov/ttn/atw/nata/

www.epa.gov/safewater/dwh/c-voc/vinylchl.html

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