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Vinyl Chloride (VC)

TEACH Chemical Summary

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

This TEACH Chemical Summary is a compilation of information derived primarily from U.S. EPA and ATSDR resources, and the TEACH Database. The TEACH Database contains summaries of research studies pertaining to developmental exposure and/or health effects for each chemical or chemical group. TEACH does not perform any evaluation of the validity or quality of these research studies. Research studies that are specific for adults are not included in the TEACH Database, and typically are not described in the TEACH Chemical Summary.

I. INTRODUCTION

Vinyl chloride is a colorless organic gas with a sweet odor, and is used to make polyvinyl chloride (PVC) plastic and vinyl products (1, 2). It is used in the manufacture of numerous products in building and construction, the automotive industry, electrical wire insulation and cables, piping, industrial and household equipment, medical supplies, and is depended upon heavily by the rubber, paper, and glass industries (1, 2). Over 15 billion pounds were produced in the U.S. in 1995 (3).

Vinyl chloride is a known human carcinogen (cancer-causing agent). Vinyl chloride is also a known genotoxicant, causing chemical alterations of DNA in tissues that may lead to cancer following exposure of humans and experimental animals (1, 2).

The primary target organ for vinyl chloride exposure is the liver (1, 2). The association between angiosarcoma of the liver and vinyl chloride exposure is well documented for occupational exposures (1, 2). Noncancer liver pathologies have also been associated with vinyl chloride exposure, including liver necrosis and cysts (1). Several studies in experimental animal models have demonstrated that early life exposure to vinyl chloride can increase susceptibility to cancer later in life (1, 2, 4-8). Based on these data, the U.S. ATSDR has characterized fetuses, infants, and young children as a "highly susceptible population" for vinyl chloride exposure (2).

Children are at risk for exposure to vinyl chloride from ambient air contaminated with vinyl chloride by emissions released from polyvinyl chloride (PVC) plastics production and manufacturing facilities, as well as some incinerators. Contamination of groundwater and drinking water with vinyl chloride-contaminated run-off from such manufacturing facilities is also a concern for exposure of children (1, 2).

II. EXPOSURE MEDIA AND POTENTIAL FOR CHILDREN'S EXPOSURE[1]

Exposure Media	Relative Potential for Children's Exposure[2,3]	Basis[4]
Ambient Air	Medium	Polyvinyl chloride factories and other plastics manufacturing facilities can be a source of vinyl chloride releases. Vinyl chloride can be released from poorly controlled incineration of chlorinated plastics. Vinyl chloride 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 vinyl chloride can be generated as a degradation product of chlorinated plastics or other chlorinated chemicals (i.e., chlorinated ethylenes, such as TCE).
Drinking Water	Medium	Drinking water may be contaminated with vinyl chloride, particularly in areas where groundwater is contaminated. Drinking water may contain vinyl chloride from contact with polyvinyl chloride-containing pipes.
Indoor Air	Medium	In homes/dwellings located above contaminated groundwater, vinyl chloride is capable of migrating through soil and foundations to enter basements or living spaces. Vinyl chloride can also volatilize to indoor air from contaminated groundwater due to indoor water uses (e.g., showering, dishwashing, laundry).
Diet	Lower	High volatility usually prevents vinyl chloride from entering the food chain.
Soil	Lower	Vinyl chloride does not partition to or accumulate in soils because of high volatility.
Sediment	Lower	Vinyl chloride 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 Child-Specific Exposure Factors Handbook (http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=55145).

[2] The Relative Potential for Children's Exposure category reflects a judgment by the TEACH Workgroup, U.S. EPA, that incorporates potential exposure pathways, frequency of exposure, level of exposure, and current state of knowledge. Site-specific conditions may vary and influence the relative potential for exposure. For more information on how these determinations were made, go to http://www.epa.gov/teach/teachprotocols_chemsumm.html.

[3] Childhood represents a lifestage 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.

[4] Information described in this column was derived from several resources (e.g., 1, 2) including studies listed in the TEACH Database (http://www.epa.gov/teach).

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III. TOXICITY SUMMARY[5, 6]

Reports of effects of chronic exposure to vinyl chloride focus primarily on occupational adult exposures and experimental animal studies. Chronic vinyl chloride exposure causes angiosarcoma of the liver in occupationally-exposed adults (1, 2). Other cancers of the liver, as well as lung and brain cancer, have been reported in occupational exposure studies (1, 2) and experimental animal studies (1, 2, 4, 6). Experimental animal studies have shown that early life exposure increases susceptibility to cancer later in life (4-8), and these studies form the basis for the U.S. ATSDR determination that fetuses, infants, and children are a highly susceptible population for vinyl chloride exposure (2). Vinyl chloride is also a genotoxicant in adults (1, 2) and in experimental animals during development (9-11).

Other non-cancer adverse effects on the liver have also been reported, including necrosis and cysts (1, 2). Paternal occupational exposure to vinyl chloride was associated with increased incidence of miscarriage in one study (12) but not others (13, 14). Evidence has supported a possible association of prenatal vinyl chloride exposure with birth defects, but remains equivocal based on reported results in human (15-17) and in experimental animal studies (18-20). Evidence of immune system and skin effects in occupationally-exposed adults has also been reported (1, 2).

Acute exposure to vinyl chloride has resulted in central nervous system effects, including dizziness, drowsiness, headaches, and giddiness. Acute high dose exposure in humans and experimental animals has also caused loss of consciousness, lung and kidney irritation, and inhibition of blood clotting (1, 2).

Carcinogenicity weight-of-evidence classification[7]: U.S. EPA classified vinyl chloride as a known human carcinogen by inhalation exposure, and by the oral route because of positive animal bioassay data and pharmacokinetic data allowing dose extrapolation across routes; vinyl chloride 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) (21). The World Health Organization International Agency for Research on Cancer (IARC) classifies vinyl chloride as a known (Group 1) human carcinogen (http://monographs.iarc.fr/ENG/Monographs/suppl7/suppl7.pdf) (22).

[5] Please refer to research article summaries listed in the TEACH Database for details about study design considerations (e.g., dose, sample size, exposure measurements).

[6] This toxicity summary is likely to include information from workplace or other studies of mature (adult) humans or experimental animals if child-specific information is lacking for the chemical of interest. Summaries of articles focusing solely on adults are not listed in the TEACH Database because the TEACH Database contains summaries of articles pertaining to developing organisms.

[7] For recent information pertaining to carcinogen risk assessment during development, consult Guidelines for Carcinogen Risk Assessment and Supplemental Guidance on Risks from Early Life Exposure at *http://www.epa.gov/cancerguidelines*.

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IV. EXPOSURE AND TOXICITY STUDIES FROM THE TEACH DATABASE

This section provides a brief description of human and animal studies listed in the TEACH Database. For more details about study design parameters, e.g., doses and exposure information, please refer to article summaries in the TEACH Database. Any consideration should include an understanding that exposure levels in animal studies, in many cases, are greater than exposure levels normally encountered by humans.

A. *HUMAN EXPOSURE AND EFFECTS*

- Based on maternal exposure via drinking water (23) and inhalation (24), estimates of vinyl chloride exposure of fetuses (23) and nursing neonates (23, 24) have been performed using physiologically-based pharmacokinetic (PBPK) modeling. The greatest difference between concentrations of vinyl chloride or vinyl chloride metabolites in maternal blood and in offspring blood was estimated to occur in late pregnancy and the neonatal period (23, 24). Higher concentrations of vinyl chloride (23) or its metabolites (24) were estimated in infant blood than in maternal blood.
- There have been few reported studies in humans that evaluated possible associations between prenatal or parental exposure to vinyl chloride and the incidence of birth defects (14-17). One case control study found no association between incidence of central nervous system defects in infants at birth and parental occupational exposure to vinyl chloride (15). Another study of families reported no significant association between incidence of birth defects, and proximity of homes to vinyl chloride-contaminated sites (16), or ambient air vinyl chloride concentrations (17).
- 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 (25), but not in others (13, 14).
- The incidence of nervous system defects in children living close to vinyl chloride-contaminated industrial sites has been studied, and a trend of increased incidence was noted, but was not statistically significant (16).

B. *EXPERIMENTAL ANIMAL EXPOSURE AND EFFECTS*

- Several studies of health effects following inhalation exposure to vinyl chloride have been performed in experimental animal models during development. In studies described in this Chemical Summary, animals were exposed to vinyl chloride via inhalation unless indicated otherwise.
- Studies of possible effects of vinyl chloride exposure on sperm have reported no observable effects (26, 27). One study demonstrated that vinyl chloride exposure of male rats did not produce dominant lethal mutations in the sperm cells as measured by either preimplantation or postimplantation losses in pregnant rats (26). In another study, vinyl chloride exposure was found to have no effects on any maturation stage of spermatogenesis in the male mouse (27).
- Vinyl chloride is a transplacental carcinogen in the rat (reviewed in (5). Studies in rats demonstrated that prenatal exposure to vinyl chloride resulted in an increased incidence of brain neuroblastoma, liver angiosarcoma, and hepatocarcinoma in adulthood (6, 7).

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- Embryotoxic effects, including increased fetal mortality and decreased fetal body weight, were demonstrated following vinyl chloride exposure during the first trimester of pregnancy in rats, mice and rabbits (20, 28). However, no increased incidence of fetal resorption (fetal loss) was seen in another study of prenatal vinyl chloride exposure in rats (18).
- Studies reported varied results on the incidence of developmental anomalies (birth defects) following prenatal exposure to vinyl chloride. In one study, following maternal exposure to vinyl chloride at doses that caused maternal toxicity, significant delays in bone development in offspring at birth were observed in mice, rabbits, and rats (20). Delays included delayed ossification (bone hardening) of the skull and sternebrae, and unfused sternebrae (20). In another study, no adverse effects of prenatal vinyl chloride exposure on birth weight or incidence of external malformations were seen with rats (18).
- Early life exposure to vinyl chloride resulted in increased incidence of tumors later in life in hamsters, rats, and mice. Animals briefly exposed to vinyl chloride, starting at birth for a duration of six months, demonstrated increased incidence of tumors of the liver, mammary gland, gastrointestinal tract, and skin following vinyl chloride exposure (4, 6, 7); one of these studies also found increased incidence of neuroblastoma (6). A higher incidence of cancer was observed when treatment was initiated at birth rather than later in life (4). A quantitative assessment of cancer risk from early life exposure has been performed (8).
- Several studies have suggested that developmental exposure to vinyl chloride may result in more chemically-modified DNA than adulthood exposure. Formation of chemically-modified DNA is thought to be an early step in cancer formation (9-11). Young rats exposed to vinyl chloride for 1 week had greater amounts of DNA adduct formation (one type of chemically-altered DNA molecules) in liver and brain than adult rats exposed for the same length of time (10, 11). Another study compared vinyl chloride alkylation of DNA between rats exposed during early postnatal life and during adulthood, and found greater amounts of alkylated DNA in rats exposed while young (9).

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V. CONSIDERATIONS FOR DECISION-MAKERS

This section contains information that may be useful to risk assessors, parents, caregivers, physicians, and other decision-makers who are interested in reducing the exposure and adverse health effects in children for this particular chemical. Information in this section focuses on ways to reduce exposure, assess possible exposure, and, for some chemicals, administer treatment.

- The U.S. EPA used 1999 emissions data for vinyl chloride for all 50 states to report emissions, modeled ambient air concentration estimates, modeled human exposure, and estimated risk (29). Vinyl chloride was defined to be a "Regional Cancer Risk Contributor" defined as posing an estimated upper-bound lifetime cancer risk exceeding 1 in a million to more than 1 million people (30).
- The U.S. Centers for Disease Control provides a document called "Medical Management Guidelines for Vinyl Chloride" for health care professionals (31).
- Unlike reference values for many chemicals of interest in TEACH, the U.S. EPA Cancer Oral Slope Factor explicitly considers the increased risk of cancer from early lifestage exposure to vinyl chloride (1, 21). For more information, see "Toxicity Reference Values" in this Vinyl Chloride Chemical Summary.
- Exposure of children to vinyl chloride may often occur from direct industrial releases. Vinyl chloride has also been found in landfill gas and groundwater as a degradation product of chloroethylene solvents, particularly trichloroethylene (TCE) (1, 2). Thus, vinyl chloride exposure from drinking water should be considered in areas where groundwater is contaminated with TCE.
- Vinyl chloride in contaminated groundwater can volatilize and contaminate indoor air (32, 33). This volatilization is referred to as vapor intrusion, and should be considered in a vinyl chloride risk assessment.
- In view of the U.S. EPA Maximum Contaminant Level Goal (MCLG) of 0 for vinyl chloride (see Toxicity Summary and Reference Values in this Chemical Summary), caregivers may consider an alternate water supply, e.g. bottled water, where vinyl chloride-contaminated groundwater may impact drinking water.
- Detailed compilations and analyses of information pertaining to exposure and health effects of vinyl chloride are available in the Toxicological Review for Vinyl Chloride (1) and in the Toxicological Profile for Vinyl Chloride (2). A Hazard Summary for Vinyl Chloride is also available from the U.S. EPA, which summarizes information primarily derived from these two sources (4, 34).
- Consult the "Child-Specific Exposure Factors Handbook" (EPA-600-P-00-002B), for factors to assess children's drinking water consumption and inhalation rates (35). An updated External Draft of the 2006 version of this handbook is available (36).

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VI. TOXICITY REFERENCE VALUES

A. Oral/Ingestion

U.S. EPA Reference Dose (RfD) for Chronic Oral Exposure: 3E-3 (or 0.003) mg/kg-day, based on liver cell polymorphisms in adult rats, with one supporting early life animal study (http://www.epa.gov/iris/subst/1001.htm, I.A.1) (21). Last Agency Consensus Date 7/20/00.

U.S. EPA Cancer Oral Slope Factor: Continuous lifetime adult exposure, 7.2E-1 (or 0.72) mg/kg-day (LMS method), 7.5E-1 (or 0.75) 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) (21). Last Agency Consensus Date 7/20/00.

U.S. EPA Cancer Drinking Water Unit Risk: Continuous lifetime adult exposure, 2.1E-5 (or 0.000021) µg/L; continuous lifetime exposure from birth, 4.2E-5 (or 0.000042) µg/L. Derived using LMS and LED 10/linear extrapolation method (http://www.epa.gov/iris/subst/1001.htm, II.B.1.2) (21). Last Agency Consensus Date 7/20/00.

U.S. EPA Drinking Water Concentrations at Specified Risk Levels for Cancer: 1E-4 (or 1 in 10,000), 4.8 µg/L (adult exposure), 2.4 µg/L (exposure from birth); 1E-5 (or 1 in 100,000), 4.8E-1 µg/L (adult), 2.4E-1 µg/L (from birth); 1E-6 (or 1 in 1,000,000), 4.8E-2 µg/L (adult), 2.4E-2 µg/L (from birth) (http://www.epa.gov/iris/subst/1001.htm) (21). Last Agency Consensus Date 7/20/00.

U.S. EPA Drinking Water Advisories (10 kg or 22 lb. child): 1 day = 3 mg/L, 10 day = 3 mg/L (http://www.epa.gov/waterscience/drinking/standards/dwstandards.pdf) (37). Last revised August 2006.

U.S. EPA Maximum Contaminant Level (MCL) for Drinking Water: 0.002 mg/L, with potential health effects of increased risk of cancer in adults http://www.epa.gov/safewater/mcl.html#mcls) (38). Last revised 7/02.

U.S. EPA Maximum Contaminant Level Goal (MCLG): 0 mg/L (http://www.epa.gov/safewater/mcl.html#mcls) (38). Last revised 7/02.

B. Inhalation

U.S. EPA Reference Concentration (RfC) for Chronic Inhalation Exposure: 1E-1 (or 0.1) mg/m^3, based on liver cell polymorphisms in adult rats, with three supporting early life animal studies (http://www.epa.gov/iris/subst/1001.htm, I.B.1) (21). Last Agency Consensus Date 8/7/00.

U.S. EPA Carcinogenic Risk from Inhalation Exposure Air Unit Risk: Continuous lifetime exposure during adulthood = 4.4E-6 (or 0.000006) per µg/m^3 (based on LMS and LED 10/linear methods); continuous lifetime exposure from birth = 8.8E-5 (or 0.00005) per µg/m^3 (based on LMS and LED 10/linear methods) (http://www.epa.gov/iris/subst/1001.htm) (21). Last revised 8/7/00.

US EPA ARCHIVE DOCUMENT

U.S. ATSDR Minimal Risk Level (MRL): 0.5 ppm (acute inhalation), based on developmental effects; 0.03 ppm (intermediate inhalation), based on hepatic effects; 0.00002 mg/kg-day (chronic oral) based on hepatic effects (http://www.atsdr.cdc.gov/mrls/index.html) (39). Last revised 9/97.

VII. U.S. FEDERAL REGULATORY INFORMATION

- 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 (40).
- Vinyl chloride is listed as number 4 on the 2005 Priority List of Hazardous Substances for the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) section 104 (i), as amended by the Superfund Amendments and Reauthorization Act (SARA). This is a prioritized list ranking chemicals in order of concern for those most commonly found at sites listed on the National Priorities List (NPL); there are currently 275 substances on this list (41).
- The U.S. EPA requires reporting of quantities of certain chemicals that exceed a defined reportable quantity, and that quantity varies for different chemicals (42). Under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), reporting releases of vinyl chloride of any quantity exceeding 1 pound is required (42).

VIII. BACKGROUND ON CHEMICAL

A. CAS Number: 75-01-4

B. Physicochemical Properties: Vinyl chloride is a colorless gas with a sweet odor. Go to the National Library of Medicine ChemID Web site (http://chem.sis.nlm.nih.gov/chemidplus) and search for vinyl chloride.

C. Production: Approximately 15 billion pounds of vinyl chloride monomer was produced in the U.S. in 1995 (1-3). 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 (3). Vinyl chloride is a breakdown product of trichloroethane, trichloroethylene (TCE), and tetrachloroethylene (2, 3).

D. Uses: Vinyl chloride is used to make polyvinyl chloride (PVC) (1-3). PVC is used to make a variety of plastic products, including pipes, wire and cable coatings, and furniture upholstery. Total reported releases and disposals in the U.S. was 573,388 pounds in 2005; total releases are likely to be greater than this estimate because not all sources of vinyl chloride releases are required to report (43).

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E. Environmental Fate: Vinyl chloride is a colorless gas with a mild, sweet odor at room temperature (1, 3). Vinyl chloride exists in liquid form under high pressure or cold temperatures (1-3). Liquid vinyl chloride evaporates easily into air, and when near the surface of soil and water (1, 3). Vinyl chloride can break down in the air within a few days (half-life is 1-4 days), and the breakdown products can negatively impact health (1-3). Vinyl chloride is minimally soluble in water and is flammable. Vinyl chloride is a degradation product of other chlorinated chemicals, particularly TCE, that may be present in groundwater (2, 3). It does not bioaccumulate in plants or animals.

F. Synonyms and Trade Names: Vinyl chloride monomer (VCM); chloroethene; chloroethylene; vinchloroethene; ethylene monochloride; monochloroethene; monochloroethylene (for a list of synonyms and trade names, go to *http://www.atsdr.cdc.gov/toxprofiles/tp20-c4.pdf*.

Additional information on vinyl chloride is available in the TEACH Database for Vinyl Chloride, and at the following Web sites:

http://www.atsdr.cdc.gov/mrls/index.html
http://www.atsdr.cdc.gov/tfacts20.pdf
www.epa.gov/safewater/dwh/c-voc/vinylchl.html
http://www.epa.gov/safewater/contaminants/dw_contamfs/vinylchl.html

US EPA ARCHIVE DOCUMENT

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US EPA ARCHIVE DOCUMENT

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US EPA ARCHIVE DOCUMENT

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