820K90100

CIS-1, 2-DICHLOROETHYLENE

Health Advisory
Office of Drinking Water
U.S. Environmental Protection Agency

I. INTRODUCTION

The Health Advisory (HA) Program, sponsored by the Office of Drinking Water (ODW), provides information on the health effects, analytical methodology and treatment technology that would be useful in dealing with the contamination of drinking water. Health Advisories describe nonregulatory concentrations of drinking water contaminants at which adverse health effects would not be anticipated to occur over specific exposure durations. Health Advisories contain a margin of safety to protect sensitive members of the population.

Health Advisories serve as informal technical guidance to assist Federal, State and local officials responsible for protecting public health when emergency spills or contamination situations occur. They are not to be construed as legally enforceable Federal standards. The HAs are subject to change as new information becomes available.

Health Advisories are developed for One-day, Ten-day, Longer-term (approximately 7 years, or 10% of an individual's lifetime) and Lifetime exposures based on data describing noncarcinogenic end points of toxicity. Health Advisories do not quantitatively incorporate any potential carcinogenic risk from such exposure. For those substances that are known or probable human carcinogens, according to the Agency classification scheme (Group A or B), Lifetime HAs are not recommended. The chemical concentration values for Group A or B carcinogens are correlated with carcinogenic risk estimates by employing a cancer potency (unit risk) value together with assumptions for lifetime exposure and the consumption of drinking water. The cancer unit risk is usually derived from the linear multistage model with 95% upper confidence limits. This provides a low-dose estimate of cancer risk to humans that is considered unlikely to pose a carcinogenic risk in excess of the stated values. Excess cancer risk estimates may also be calculated using the One-hit, Weibull, Logit or Probit models. There is no current understanding of the biological mechanisms involved in cancer to suggest that any one of these models is able to predict risk more accurately than another. Because each model is based on differing assumptions, the estimates that are derived can differ by several orders of magnitude.

This Health Advisory is based on information presented in the Office of Drinking Water's Health Effects Criteria Document (CD) for the Dichloro-ethylenes (U.S. EPA, 1984a). The HA and CD formats are similar for easy reference. Individuals desiring further information on the toxicological data base or rationale for risk characterization should consult the CD. The CD is available for review at each EPA Regional Office of Drinking Water counterpart (e.g., Water Supply Branch or Drinking Water Branch), or for a fee from the National Technical Information Service, U.S. Department of Commerce, 5285 Port Royal Rd., Springfield, VA 22161, PB #86-117785/AS. The toll-free number is (800) 336-4700; in the Washington, D.C. area: (703) 487-4650.

II. GENERAL INFORMATION and PROPERTIES

CAS No. 156-59-2

Chemical Structure

C1 C1 | | H-C=C-H

Synonyms

1,2-DCE; cis-1,2-DCE; 1,2-dichloroethene

Uses

In a mixture with the trans-1,2- isomer, as a captive intermediate in the manufacture of other chlorinated solvents

Properties (Irish, 1963; Windholz et al., 1976)

Chemical Formula C₂H₂Cl₂
Molecular Weight 96.95

Physical State clear, colorless liquid

Freezing Point -80.5°C
Boiling Point 60°C
Melting Point -Density --

Vapor Pressure 208 mm Hg (25°C) Specific Gravity 1.27 (25°C) Water Solubility 3500 ug/L (20°C)

Log Octanol/Water Partition --

Coefficient

Taste Threshold Not available Odor Threshold Not available

Conversion Factor --

Occurrence

o The 1,2-dichloroethylenes are synthetic chemicals with no known natural sources (U.S. EPA, 1983).

- There is little information on the current production and use of the 1,2-dichloroethylenes. The production volume for 1,2-dichloroethylene (mixed isomers) was 1,000 lbs or less in 1978 (U.S. EPA, 1978).
- The major releases of the 1,2-dichloroethylenes are from the manufacturing plants in the Gulf Coast region of the U.S., where they are used as a captive intermediate. Releases are expected to be small. The 1,2-dichloroethylenes, particularly the cis-isomer, have been identified as the degradation products of trichloroethylene and tetrachloroethylene in ground water (Parsons et al., 1984; Vogel and McCarty, 1985).
- There is little direct information on the fate of the 1,2-dichloroethylenes in the environment. However, the behavior of the compounds
 has been estimated based upon the information on similar chlorinated
 compounds (U.S. EPA 1979). 1,2-Dichloroethylenes released to the
 atmosphere are expected to chemically degrade in a matter of hours;
 when released to surface waters, they are expected to volatilize
 rapidly to air. 1,2-Dichloroethylenes are chemically stable in water
 and mobile in soils. Once released to land the 1,2-dichloroethylenes
 are expected to migrate with ground water. 1,2-Dichloroethylenes
 have been shown to biologically degrade to vinyl chloride in some
 groundwaters. These compounds are not expected to bioaccumulate in
 plants or animals. Based upon their similar physical properties,
 the two isomers of 1,2-dichloroethylene are not expected to behave
 differently in the environment.
- Monitoring studies have found that the 1,2-dichloroethylenes occur as widespread but relatively rare contaminants of ground water. The cis- isomer has been reported to occur at higher levels than the trans- isomer. The majority of the 1,2-dichloroethylenes has been found to co-occur with trichloroethylene. Levels of the 1,2-dichloroethylenes in approximately 1 % of all ground waters are greater than 0.5 ug/L. Levels as high as 300 ug/L have been reported for the trans- isomer, while levels of 800 ug/L have been reported for the cis- isomer. The 1,2-dichloroethylenes occur in surface water at lower amounts. Levels of 1,2-dichloroethylenes in air are in the ppt range except near production sites where they may reach levels in the low ppb range. Based upon these compounds' volatility and limited use, levels of 1,2-dichloroethylenes in food are expected to be negligible (U.S. EPA, 1983).
- The major source of exposure to the 1,2-dichloroethylenes is from contaminated water except in the areas near production sites where air exposures may dominate.

III. PHARMACOKINETICS

Absorption

cis-1,2-Dichloroethylene is a neutral, low molecular weight, lipid soluble material which would be expected to be readily absorbed following exposure by any route (oral, inhalation, dermal) at the levels expected to be encountered in contamination incidents (U.S. EPA, 1984a).

Distribution

Kinetic data to define the tissue distribution of cis-1,2-dichloroethylene after oral exposure are not available. If this isomer, however, follows the same absorption and distribution pattern as observed for 1,1-dichloroethylene, the highest concentrations would be expected to be found in the liver and kidney (McKenna et al., 1978).

Metabolism

- The metabolic end products of chlorinated ethylenes are predominantly alcohols and carboxylic acids. Perfusion of cis-1,2-dichloroethylene through isolated rat liver yielded dichloroethanol and dichloroacetic acid, possibly indicating the initial formation of dichloroacetaldehyde (Bonse et al., 1975).
- The position of the chlorine moeity on the chlorinated ethylenes appears to play an important role in their metabolism. Cis-1,2-dichloroethylene was metabolized at a faster rate than trans-1,2-dichloroethylene (which possesses a relatively greater degree of asymmetry) in an in vitro hepatic microsomal system (Costa, 1983).
- Osing isolated rat liver microsomes, Freundt and Macholz (1978) reported that cis-1,2-dichloroethylene showed competitive and reversible interaction with the mixed function oxygenase system, resulting in decreased drug metabolism.

Excretion

No data concerning the excretion of cis-1,2-dichloroethylene are available. If it is similar to 1,1-dichloroethylene, then the rate of elimination would be expected to be relatively rapid, with most of a single dose being excreted in the urine within 24 to 72 hours after cessation of exposure (Jaeger et al., 1977).

IV. HEALTH EFFECTS

Humans

At high concentrations, the dichloroethylenes, like other chlorinated ethylenes, possess anesthetic properties. cis-1,2-Dichloroethylene was used as an anesthetic with some success prior to introduction of newer anesthetic gases, and appeared to be safe (Irish, 1963).

Animals

Short-term Exposure

 $^{\circ}$ No cis- isomer-specific LD₅₀s have been reported. An oral LD₅₀ of 770 mg/kg of the isomer mixture was reported for rats (NIOSH, 1978).

- At high exposure levels, general anesthetic and narcotic effects are observed (Irish, 1963).
- Administration of a single dose of cis-1,2-dichloroethylene at 400 mg/kg to rats caused a significant elevation of liver alkaline phosphatase (Jenkins et al., 1972).

Long-term Exposure

No information was found in the available literature on the effects of long-term exposures to cis-1,2-dichloroethylene.

Reproductive Effects

No information was found in the available literature on the potential of cis-1,2-dichloroethylene to produce reproductive effects.

Developmental Effects

 No information was found in the available literature on the potential of cis-1,2-dichloroethylene to produce developmental effects.

Mutagenicity

- cis-1,2-Dichloroethylene was not mutagenic, with or without metabolic activation, when assayed in E. coli K12 at a medium concentration of 2.9 mM (Greim et al., 1975)
- Galli et al. (1982a) reported that cis-1,2-dichloroethylene did not induce point mutation, mitotic gene conversion or mitotic recombination in yeast. In addition, they (1982b) reported that cis-1,2-dichloroethylene was not mutagenic in an in vivo (intravenous host-mediated assay) test. (Both manuscripts are in Italian.)

Carcinogenicity

No information was found in the available literature on the carcinogenic potential of cis-1,2-dichloroethylene.

V. QUANTIFICATION OF TOXICOLOGICAL EFFECTS

Health Advisories (HAs) are generally determined for One-day, Ten-day, Longer-term (approximately 7 years) and Lifetime exposures if adequate data are available that identify a sensitive noncarcinogenic end point of toxicity. The HAs for noncarcinogenic toxicants are derived using the following formula:

$$HA = \frac{\text{(NOAEL or LOAEL)} \times \text{(BW)}}{\text{(UF)} \times \text{(} L/\text{day)}} = \frac{\text{mg/L}}{\text{ug/L}}$$

where:

NOAEL or LOAEL = No- or Lowest-Observed-Adverse-Effect-Level in mg/kg bw/day.

UF = uncertainty factor (10, 100 or 1,000), in accordance with NAS/ODW guidelines.

___ L/day = assumed daily water consumption of a child (1 L/day) or an adult (2 L/day).

One-day Health Advisory

There are few animal studies which provide dose-response data on the effects of cis-1,2-dichloroethylene (Irish, 1963; Jenkins et al., 1972; Freundt and Macholz, 1978). Only the study by Jenkins et al. provides sufficient information from which a One-day Health Advisory can be calculated. These authors monitored levels of liver glucose-6-phosphatase, liver alkaline phosphatase, liver tyrosine transaminase, plasma alkaline phosphatase and plasma alkaline transaminase and observed that a single, oral dose of 400 mg/kg to the rat produced a significant change only in liver alkaline phosphatase. The LOAEL of 400 mg/kg reported by Jenkins et al. (1972) will be used for the one-day calculations.

The One-day Health Advisory for the 10 kg child is calculated as follows:

One-day HA =
$$\frac{(400 \text{ mg/kg/day}) (10 \text{ kg})}{(1,000) (1 \text{ L/day})} = 4 \text{ mg/L} (4,000 \text{ ug/L})$$

where:

400 mg/kg/day = LOAEL based on increase in liver alkaline phosphatase.

10 kg = assumed body weight of a child.

1,000 = uncertainty factor, chosen in accordance with NAS/ODW guidelines for use with a LOAEL from an animal study.

1 L/day = assumed daily water consumption of a child.

Ten-day Health Advisory

Appropriate studies for the calculation of a Ten-day Health Advisory are not available. Evaluation of the available toxicological data on cis-1,2-dichloroethylene and 1,1-dichloroethylene suggests that the Longer-term Health Advisory of 1 mg/L should provide adequate protection over a 10-day exposure period as well.

Longer-term Health Advisory

A Longer-term HA for cis-1,2-dichloroethylene cannot be derived directly from compound-specific data since appropriate data do not exist at this time. The available information from shorter-term exposure to 1,1-dichloroethylene and cis- and trans 1,2-dichloroethylene suggests that the non-carcinogenic effects induced by the 1,2- isomers is likely to be no more, and conceivably less, severe than those induced by 1,1-dichloroethylene. Since the non-carcinogenic end-points of toxicity for all three isomers appear to be essentially identical, adopting the Longer-term HA derived for 1,1-dichloroethylene for use as the Longer-term HA for cis-1,2-dichloroethylene may even result in an added margin of safety.

The Longer-term HA will be derived from a 90-day subchronic study in which rats of both sexes were administered 1,1-dichloroethylene at nominal concentrations of 0, 50, 100 or 200 ppm (0-25.6 mg/kg/day) in their drinking water (Rampy et al., 1977). Except for a decreased kidney:body weight ratio in males at the low dose, there were no statistically significant differences in organ weights or organ:body weight ratios at the end of the study. The only histopathology noted was an increased cytoplasmic vacuolization of hepatocytes in the livers of both sexes exposed to the highest dose. A NOAEL of 100 ppm (10 to 12.6 mg/kg) was identified.

The Longer-term HA for the 10-kg child is calculated as follows:

Longer-term HA =
$$\frac{(10 \text{ mg/kg/day}) (10 \text{ kg})}{(100) (1 \text{ L/day})} = 1 \text{ mg/L} (1000 \text{ ug/L})$$

where:

10 mg/kg/day = NOAEL based on the absence of liver effects.

10 kg = assumed body weight of a child.

1 L/day = assumed daily water consumption of a child.

The Longer-term HA for the 70-kg adult is calculated as follows:

Longer-term HA =
$$\frac{(10 \text{ mg/kg/day}) (70 \text{ kg})}{(100) (2 \text{ L/day})}$$
 = 3.5 mg/L (3500 ug/L)

where:

10 mg/kg/day = NOAEL based on the absence of liver effects.

70 kg = assumed body weight of an adult.

100 = uncertainty factor, chosen in accordance with NAS/ODW guidelines for use with a NOAEL from an animal study.

2 L/day = assumed daily water consumption of an adult.

Lifetime Health Advisory

The Lifetime HA represents that portion of an individual's total exposure .that is attributed to drinking water and is considered protective of noncarcinogenic adverse health effects over a lifetime exposure. The Lifetime HA is derived in a three step process. Step 1 determines the Reference Dose (RfD), formerly called the Acceptable Daily Intake (ADI). The RfD is an estimate of a daily exposure to the human population that is likely to be without appreciable risk of deleterious effects over a lifetime, and is derived from the NOAEL (or LOAEL), identified from a chronic (or subchronic) study, divided by an uncertainty factor(s). From the RfD, a Drinking Water Equivalent Level (DWEL) can be determined (Step 2). A DWEL is a medium-specific (i.e., drinking water) lifetime exposure level, assuming 100% exposure from that medium, at which adverse, noncarcinogenic health effects would not be expected to occur. The DWEL is derived from the multiplication of the RfD by the assumed body weight of an adult and divided by the assumed daily water consumption of an adult. The Lifetime HA is determined in Step 3 by factoring in other sources of exposure, the relative source contribution (RSC). The RSC from drinking water is based on actual exposure data or, if data are not available, a value of 20% is assumed for synthetic organic chemicals and a value of 10% is assumed for inorganic chemicals. If the contaminant is classified as a Group A or B carcinogen, according to the Agency's classification scheme of carcinogenic potential (U.S. EPA, 1986), then caution should be exercised in assessing the risks associated with lifetime exposure to this chemical.

Lifetime toxicity data for cis-1,2-dichloroethylene do not exist. Data from the chronic drinking water study in rats as used for the Lifetime Health Advisory for 1,1-dichloroethylene will be used instead. The same caveats and assumptions as were described above for the Longer-term HA also apply here.

The Lifetime HA can be calculated from the 2-year chronic study in rats (Quast et al., 1983). 1,1-Dichloroethylene, at nominal concentrations of 0, 50, 100 or 200 ppm (0 to 20 mg/kg/day) in drinking water, was administered to animals of both sexes. No consistent treatment-related changes were observed in any parameter measured. The only histopathology observed was in the livers of both sexes receiving the highest dose, changes characterized by a minimal amount of mid-zonal fatty accumulation. No liver degeneration was noted. A LOAEL of 100 ppm (10 mg/kg) was identified, based upon a trend towards increased fatty deposition in the liver.

A Drinking Water Equivalent Level (DWEL) and Lifetime HA for the 70-kg adult are calculated as follows:

Step 1: Determination of the Reference Dose (RfD)

RfD =
$$\frac{(10 \text{ mg/kg/day})}{(1,000)}$$
 = 0.01 mg/kg/day

where:

10 mg/kg/day = LOAEL based on adverse liver effects.

1,000 = uncertainty factor, chosen in accordance with NAS/ODW guidelines for use with a LOAEL from an animal study.

Step 2: Determination of the Drinking Water Equivalent Level (DWEL)

DWEL =
$$\frac{(0.01 \text{ mg/kg/day}) (70 \text{ kg})}{(2 \text{ L/day})} = 0.35 \text{ mg/L} (350 \text{ ug/L})$$

where:

0.01 mg/kg/day = RfD.

70 kg = assumed body weight of an adult.

2 L/day = assumed daily water consumption of an adult.

Step 3: Determination of Lifetime Health Advisory

Lifetime HA =
$$(0.35 \text{ mg/L}) (20\%) = 0.07 \text{ mg/L} (70 \text{ ug/L})$$

where:

0.35 mg/L = DWEL.

20% = assumed relative source contribution from water.

Evaluation of Carcinogenic Potential

- There are no data available on the carcinogenic potential of cis-1,2-dichloroethylene.
- Applying the criteria described in EPA's guidelines for assessment of carcinogenic risk (U.S. EPA, 1986), cis-1,2-dichloroethylene is classified in Group D: Not classified. This category is for agents with inadequate animal evidence of carcinogenicity.

VI. OTHER CRITERIA, GUIDANCE AND STANDARDS

The Threshold Limit Value (TLV) in the occupational setting for the 1,2-dichloroethylene isomer mixture is 200 ppm (790 mg/m 3) (ACGIH, 1982).

VII. ANALYTICAL METHODS

Analysis of cis-1,2-dichloroethylene is by a purge-and-trap gas chromatographic procedure used for the determination of volatile organohalides in drinking water (U.S. EPA, 1984b). This method calls for the bubbling of an inert gas through the sample and trapping 1,2-dichloroethylene on an adsorbant material. The adsorbant material is heated to drive off the 1,2-dichloroethylene onto a gas chromatographic column. This method will differentiate between the two isomers of 1,2-dichloroethylene. This method is applicable to the

measurement of 1,2-dichloroethylene over a concentration range of 0.03 to 1500 ug/L. Confirmatory analysis for 1,2-dichloroethylene is by mass spectrometry (U.S. EPA, 1985a). The detection limit for confirmation by mass spectometry 0.2 ug/L.

VIII. TREATMENT TECHNOLOGIES

- Treatment technologies which will remove cis-1,2-dichloroethylene from water include granular activated carbon (GAC) adsorption, aeration and boiling.
- Obbs and Cohen (1980) developed adsorption isotherms for cis-1,2-dichloroethylene It was reported that Filtrasorb® 300 carbon exhibited adsorptive capacities of 1.3 mg and 0.26 mg cis-1,2-dichloroethylene/gm carbon at equilibrium concentrations of 100 and 10 ug/L, respectively.
- USEPA-DWRD installed pilot-scale adsorption columns at three locations in New England (U.S. EPA, 1985b,c). Cis-1,2-dichloroethylene was present in the contaminated groundwater at concentrations ranging from 2 to 18 ug/L. The raw water was passed through a Filtrasorb 400 GAC column until breakthrough concentration of 0.1 ug/L was achieved which after approximately 10 weeks of continuous operation.
- cis-1,2-Dichloroethylene is amenable to removal by aeration on the basis of its Henry's Law Constant of 225 atm (U.S. EPA, 1985b,c). In a pilot-scale diffused air aeration column, removal efficiency of 85% was achieved from original concentrations of 18 to 118 ug/L at an air-to-water ratio of 30:1. At an air-to-water ratio of 5:1 and the same operating conditions, 58% of cis-1,2-dichloroethylene was removed from the same source water (Love, 1983). In another pilot-scale study, a countercurrent diffused air aeration column removed 80% of cis-1,2-dichloroethylene from well water with 0.5 ug/L, at an air-towater ratio of 4:1 (Love and Eilers, 1982). Numerous packed column air stripping plant studies have been performed by EPA. All of the studies (using identical column size) indicated that packed column aeration is effective in removing cis-1,2-dichloroethylene from drinking water supplies at different concentrations. The best removal, i.e., 99%+, was achieved at an optimum air-to-water ratio of 80-85:1 (U.S. EPA, 1985b,c; ESE, 1985).
- Boiling also is effective in eliminating cis-1,2-dichloroethylene from water on a short-term, emergency basis. Studies have shown that five minutes of vigorous boiling will remove 96% of cis-1,2-dichloroethylene present in the water (Love and Eilers, 1982).
- Air stripping is an effective, simple and relatively inexpensive process for removing cis-1,2-dichloroethylene and other volatile organics from water. However, this process transfers the contaminant directly into the air stream. When considering this method as a treatment process, it is suggested that careful consideration be given to the overall environmental occurrence, fate, route of exposure and various hazards associated with the chemical.

IX. REFERENCES

- ACGIH. 1982. American Council of Governmental Industrial Hygienists. TLVs.

 Threshold limit values for chemical substances and physical agents in the workroom environment. Cincinnati, Ohio. p.
- Bonse, G., T. Urban, R. Montessano and L. Tomatis. 1975. Chemical reactivity, metabolic oxirane formation and biological reactivity of chlorinated ethylenes in the isolated perfused rat liver preparation. Biochem. Pharmacol. 24:1829-1834.
- Costa, A.K. 1983. The chlorinated ethylenes: Their hepatic metabolism and carcinogenicity. Diss. Abst. Int [B]. 44(6):1791-B.
- Dobbs, R.A. and J.M. Cohen. 1980. Carbon adsorption isotherms for toxic chemicals. Cincinnati, Ohio. EPA-600/8-80-023.
- ESE. 1985. Environmental Science and Engineering. Technologies and costs for the removal of volatile organic chemicals from potable water supplies. ESE No. 84-912-300. Prepared for U.S. EPA Science and Technology Branch, CSD, PDW, Washington, DC.
- Freundt, J.J., and J. Macholz. 1978. Inhibition of mixed function oxidases in rat liver by trans-and cis-1,2-dichloroethylene. Toxicology. 10:131-139.
- Galli, A., C. Bauer, G. Brenzetti, C. Corsi, R. Del Carratore, R. Nieri and M. Paolini. 1982a. (a) Studio in vitro. Attivita genetica dell' 1,2-dichloroetilene. Boll. Soc. It. Biol. Sper. 58:860-863.
- Galli, A., C. Bauer, G. Brenzetti, C. Corsi, R. Del Carratore, R. Nieri and M. Paolini. 1982b. (a) Studio in vivo. Attivita genetica dell' 1,2-dichloroetilene. Boll. Soc. It. Biol. Sper. 58:864-869.
- Greim, H., G. Bonse, Z. Radwan, D. Reichert and D. Henschler. 1975.

 Mutagenicity in vitro and potential carcinogenicity of chlorinated ethylenes as a function of metabolic oxirane formation. Biochem. Pharmacol. 24:2013-2017.
- Irish, D.D. 1963. Vinylidene chloride. In: F.A. Patty (ed), Industrial
 Hygiene and Toxicology. 2nd ed. Vol. II. John Wiley and Sons, Inc.,
 New York. pp. 1305-1309.
- Jaeger, R.J., L.G. Shoner and L.J. Coffman. 1977. 1,1-Dichloroethylene hepatotoxicity: Proposed mechanism of action of distribution and binding of 14C radioactivity following inhalation exposure in rats. Environ. Health Perspect. 21:113-119.
- Jenkins, L.J., Jr., M.J. Trabulus and S.D. Murphy. 1972. Biochemical effects of 1,1-dichloroethylene in rats: Comparison with carbon tetrachloride and 1,2-dichloroethylene. Toxicol. Appl. Pharmacol. 23:501-510.

- Love, D.T., Jr. 1983. Treatment of volatile organic compounds in drinking water. U.S. Dept. of Commerce. NTIS.
- Love, D.T., Jr., and R.G. Eilers. 1982. Treatment of drinking water containing trichloroethylene and related industrial solvents. J.A.W.W.A. 74:413-425.
- McKenna, M.J., J.A. Zempel, E.O. Madrid and P.J. Gehring. 1978. The pharmacokinetics of (14C) vinylidene chloride in rats following inhalation exposure. Toxicol. Appl. Pharmacol. 45:599-610.
- NIOSH. 1978. National Institute for Occupational Safety and Health. 1,2-Dichloroethylene. Registry of toxic effects of chemical substances. p. 563.
- Parsons, F., P.R. Wood and J. DeMarco. 1984. Transformation of tetrachloroethene and trichloroethene in microcosms and groundwater. J.A.W.W.A. 76:56.
- Quast, J.F., C.G. Humiston, C.E. Wade, J. Ballard, J.E. Beyer, R.W. Schwetz and J.M. Norris. 1983. A chronic toxicity and oncogenicity study in rats and subchronic toxicity study in dogs on ingested vinylidine chloride. Fund. Appl. Toxicol. 3:55-62.
- Rampy, L.W., J.F. Quast, C.G. Humiston, M.F. Blamer and B.A. Schwetz. 1977. Interim results of two-year toxicological studies in rats of vinylidene chloride incorporated in the drinking water or administered by repeated inhalation. Environ. Health Perspect. 21:33-43.
- U.S. EPA. 1978. U.S. Environmental Protection Agency. TSCA Inventory-Non-confidential portion. Office of Toxic Substances.
- U.S. EPA. 1979. U.S. Environmental Protection Agency. Water related environmental fate of 129 priority pollutants. Office of Water Planning and Standards. EPA-440/4-79-029. December.
- U.S. EPA. 1983. U.S. Environmental Protection Agency. 1,2-Dichloroethylene occurrence in drinking water, food, and air. Office of Drinking Water.
- U.S. EPA. 1984a. U.S. Environmental Protection Agency. Draft health effects criteria document for the dichloroethylenes. Criteria and Standards Division, Office of Drinking Water. Washington, DC. December.
- U.S. EPA. 1984b. U.S. Environmental Protection Agency. Method 502.1. Volatile halogenated organic compounds in water by purge and trap gas chromatography. Environmental Monitoring and Support Laboratory, Cincinnati, Ohio 45268. June.
- U.S. EPA. 1985a. U.S. Environmental Protection Agency. Method 524.1.

 Volatile halogenated organic compounds in water by purge and trap gas chromatography/mass spectrometry. Environmental Monitoring and Support Laboratory, Cincinnati, Ohio 45268. June.

- U.S. EPA. 1985b. U.S. Environmental Protection Agency. Office of Drinking Water Health Advisory Program. Prepared by ICAIR, Life Systems, Inc. for the U.S. EPA Office of Drinking Water, Criteria and Standards Division.
- U.S. EPA. 1985c. U.S Environmental Protection Agency. Draft technologies and costs for the removal of synthetic organic chemicals from potable water supplies. Science and Technology Branch, CSD, ODW, Washington, D.C.
- U.S. EPA. 1986. U.S. Environmental Protection Agency. Guidelines for carcinogen risk assessment. Federal Register. 51(185):33992-34003. September 24.
- U.S. ITC. 1983. United States International Trade Commission. Synthetic organic chemicals. United States production. 1982 U.S. ITC Publication 1422, Washington, D.C. 20436.
- Vogel, T.M., and P.L. McCarty. 1985. Biotransformation of tetrachloroethylene to trichloroethylene, dichloroethylene, vinyl chloride, and carbon dioxide under methanogenic conditions. Appl. Environ. Microbiol. 49:1080-1083.
- Windholz, M., ed. 1976. The Merck Index. 10th edition. Merck & Co., Inc. Rahway, NJ.