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1,2-DICHLOROETHANE

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.

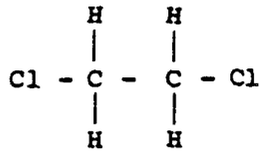
Health Advisories are developed for One-day, Ten-day, Longer-term (approximately 7 years, or 10% of an individual's lifetime) and Lifetime exposures based on data describing noncarcinogenic end points of toxicity. Health Advisories do not quantitatively incorporate any potential carcinogenic risk from 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 Health Assessment Document for 1,2-Dichloroethane (Ethylene Dichloride) (U.S. EPA, 1985a). Individuals desiring further information on the toxicological data should use this document. Information on the Quantification of Toxicological Effects (QTE) section is contained in the QTE Document (PB#86-118080). Both documents are 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 Road, Springfield, VA 22161. The toll-free number is (800) 336-4700; in the Washington, D.C. area: (703) 487-4650.

II. GENERAL INFORMATION AND PROPERTIES

CAS No. 107-06-2

Structural Formula



1,2-Dichloroethane

Synonyms

- Ethylene dichloride, EDC, 1,2-DCE

Uses (U.S. EPA, 1985a)

- The major use for EDC is in the production of vinyl chloride. In addition, it is used as a starting material for the production of other solvents, as an additive (lead scavenger) in gasolines and is widely exported. Some of its minor uses include its use as a solvent in metal degreasing and textile and PVC cleaning, in paints, coatings and adhesives, as a grain fumigant, a varnish and finish remover, in soaps and scouring compound, as a wetting and penetrating agent, in ore flotation and as a chemical intermediate.

Properties (EPA, 1985a; Amoore and Hautala, 1983)

Chemical Formula	$C_2H_2Cl_2$
Molecular Weight	98.96
Physical State	Clear, colorless, volatile, oily liquid
Boiling Point	83.7°C
Melting Point	-35.3°C
Density (20°C)	1.2529 g/mL
Vapor Pressure	64 torr (20°C)
Water Solubility (20°C)	8820 mg/L
Log Octanol/Water Partition Coefficient	1.48
Organoleptic Threshold (water)	29 mg/L
Odor Threshold (air)	3 ppm
Conversion Factor	1 ppm = 4.05 mg/m^3

Occurrence

- Dichloroethane is a synthetic chemical with no natural sources.

- Production of dichloroethane was approximately 12 billion pounds in 1983 (U.S. ITC, 1984). However, the vast bulk of dichloroethane is used as a feed stock for the production of other chlorinated compounds and it is not readily released to the environment. Releases of dichloroethane largely result from the approximately 3 million pounds used as solvents and metal cleaners.

- Releases of dichloroethane are largely to air, with smaller amounts released to surface and ground waters. Because metal working operations are performed nationwide, dichloroethane releases occur in all industrialized areas.

- Dichloroethane released to the air slowly degrades over a few months. Photooxidation is thought to be the predominant environmental process determining the fate of 1,2-dichloroethane (U.S. EPA, 1979). Dichloroethane released to surface waters migrates to the atmosphere in a few days or weeks where it also degrades. Dichloroethane released to the land does not sorb onto soil but migrates readily to ground water where it is expected to remain for months to years.

- Due to dichloroethane's limited releases, it is a relatively rare environmental contaminant. Dichloroethane has been detected in both ground and surface waters but, unlike other volatile organic compounds, higher levels were reported in surface waters than in ground waters. The Agency estimates that 0.3% of all ground water supplies contain concentrations of dichloroethane ranging from 0.5 to 5 ug/L. Surface waters contain higher levels, with 3% of all wells estimated to have from 0.5 to 20 ug/L. Dichloroethane commonly occurs in air in urban and suburban areas at concentrations of less than 0.2 ppb. No information on the levels of dichloroethane in food have been reported.

- For the majority of the U.S. population, the greatest source of dichloroethane exposure is from air. Drinking water is the greatest source only for populations with drinking water levels greater than 6 ug/L.

III. PHARMACOKINETICS

Absorption

- 1,2-Dichloroethane is absorbed by humans and laboratory animals through the lungs (Spencer et al., 1951; Urusova, 1953) gastrointestinal tract (Alumot et al., 1976) and skin (Urusova, 1953).

- The proportions of a dose of 1,2-dichloroethane absorbed through the skin and gastrointestinal tract are unknown. The nature of its other chemical and physical properties would suggest that this substance would be absorbed completely when ingested.

Distribution

- Forty-eight hours after the administration of a single oral dose of 150 mg/kg of 1,2-dichloroethane to rats, the liver and kidneys were reported to have the highest concentration of the chemical. Successively lower concentrations occurred in the forestomach, stomach and spleen (Reitz et al., 1980).

- 1,2-Dichloroethane readily passes the blood/brain barrier. Distribution is also known to occur into milk (Urosova, 1953).

Metabolism

- Following intraperitoneal administration to mice, 1,2-dichloroethane is metabolized to 2-chloroethanol, converted to alcohol and aldehyde dehydrogenases, to monochloroacetic acid, and further dehalogenated by enzyme interaction of monochloroacetate with glutathione or cysteine to yield 5-carboxymethylcysteine and thiodiacetic acid (Yllner, 1971a,b).

- Urinary metabolites of 1,2-dichloroethane intraperitoneally administered to mice include chloroacetic acid, 2-chloroethanol, 5-carboxymethyl cysteine, conjugated 5-carboxymethyl cysteine, thiodiacetic acid and 5,5-ethylene-bis-cysteine (Yllner, 1971a,b).

- Following oral administration of 1,2-dichloroethane (750 mg/kg) or 2-chloroethanol (80 mg/kg) to rats, the blood level of 2-chloroethanol at four hours was 67.8 or 15.8 ug/mL, respectively (Kokarovtseva and Kiseleva, 1978). These levels declined in accordance with first-order kinetics with a half-life of about nine hours. The relatively low blood concentrations found were postulated to be due to initial sequestration of 1,2-dichloroethane in adipose and other tissues with gradual diffusion redistribution as liver metabolism of 1,2-dichloroethane to chloroethanol and chloroethanol to chloroacetic acid proceeded.

Excretion

- Mice intraperitoneally injected with a dose of 0.05 to 0.17 g/kg of 1,2-dichloroethane excreted 11 to 46% of the dose, unchanged, via the lungs; 5 to 13% of the dose was metabolized to carbon dioxide and water; 50 to 73% of the dose was excreted as urinary metabolites (Yllner, 1971a).

- Within 48 hours after dosing, 96% of the radioactivity of a single oral dose of 150 mg/kg was eliminated from the body by rats (Reitz et al., 1980).

IV. HEALTH EFFECTS

Humans

- Clinical symptoms of acute 1,2-dichloroethane poisoning by ingestion usually appear within two hours after exposure and typically include headache, dizziness, general weakness, nausea, vomiting of blood and bile, dilated pupils, heart pains and constriction, pain in the epigastric region, diarrhea and unconsciousness. Pulmonary edema and increasing cyanosis also may occur. These symptoms may disappear if exposure is sufficiently brief (Wirtschafter and Schwartz, 1939; McNally and Fostvedt, 1941).

- A 14-year-old male who drank 15 ml (340 mg/kg) of 1,2-dichloroethane died six days later despite supportive treatment (Yodaiken and Babcock, 1973). During treatment, serum enzyme and calcium levels increased, blood glucose decreased and blood clotting time increased. Autopsy findings revealed extensive liver necrosis and epithelial cell damage in the entire cortico-tubular structure of the kidneys accompanied by degeneration in the proximal tubules.

- While not all instances of 1,2-dichloroethane ingestion are fatal, death has resulted in the majority of reported cases. Death is most often attributed to circulatory and respiratory failure (Budanova, 1965; Yodaiken and Babcock, 1973; Luzhnikov et al., 1974, 1976; and Zhizhonkov, 1976).

- A number of neurological effects following ingestion of 20 to 200 ml of 1,2-dichloroethane have been reported (Akimov et al., 1976, 1978). The most common of these involved disturbances in consciousness, mental disorders and cerebellar and extrapyramidal disorders.

Animals

Short-term Exposure

- Information on the acute oral toxicity of 1,2-dichloroethane indicates the following: rat LD_{50} - 680 mg/kg; rabbit LD_{50} - 860 mg/kg; (NIOSH, 1977).

- The principal acute effect of 1,2-dichloroethane in mammals is central nervous system depression with unconsciousness and coma resulting from exposure to high concentrations (Spencer et al., 1951; Irish, 1963). Visible signs of 1,2-dichloroethane poisoning include restlessness, intolerance to handling, extreme weakness, intoxication, dizziness, muscle incoordination, irregular respiration and loss of consciousness. Deaths occurring within a few hours after recovery from narcosis are usually the result of shock or cardiovascular collapse; deaths delayed by several days most often result from renal damage.

Reproductive Effects

- No reproductive effects, as measured by fertility, gestation, viability or lactation indices, pup survival and weight gain, were indicated in a multigeneration reproduction study using male and female ICR Swiss mice receiving 0, 5, 15 or 50 mg/kg/day in drinking water. No effect on the adult generations was evident after 25 weeks of dosing as measured by body weight, fluid intake or gross pathology (Lane et al., 1982).

Developmental Effects

- In a study in which male and female mice were exposed to 1,2-dichloroethane in drinking water at doses of 0, 5, 15 or 50 mg/kg/day, no statistically significant dose-related developmental effects were observed, as indicated by incidence of fetal visceral or skeletal anomalies (Lane et al., 1982).

Mutagenicity

- 1,2-Dichloroethane has been shown to be weakly mutagenic in Salmonella typhimurium strains TA 1530, 1535 and 1538 and in DNA polymerase-deficient Escherichia coli (Brem et al., 1974).

- 1,2-Dichloroethane has been found to be highly mutagenic in Salmonella typhimurium strains TA 1530 and 1535 with S-9 activation (Rannug and Beije, 1979).

- 1,2-Dichloroethane has been shown to induce sex-linked recessive lethals in Drosophila melanogaster (Rapport, 1960; Shakarnis, 1969).

- 1,2-Dichloroethane was not mutagenic in Salmonella microsome assay system (McCann et al., 1975).

Carcinogenicity

- In an NCI (1978) bioassay, 1,2-dichloroethane was administered by gavage at levels of 47 or 95 mg/kg body weight to Osborne-Mendel rats five times per week for 78 weeks. Statistically significant increases in the incidence of squamous cell carcinomas of the forestomach and hemangiosarcomas of the circulatory system were observed in male rats($p < 0.04$). Female rats had a statistically significant increased incidence of adenocarcinoma of the mammary glands ($p < 0.002$).

- In the same NCI (1978) bioassay, $B6C3F_1$ mice received 1,2-dichloroethane by gavage five times per week for 78 weeks; males were dosed at levels of 97 or 195 mg/kg body weight and females at 149 or 299 mg/kg body weight. Statistically significant increases in the incidence of mammary adenocarcinoma ($p < 0.04$) and endometrial stromal polyps or sarcomas ($p < 0.016$) were seen in female mice. The incidence of alveolar/bronchiolar adenomas was increased in both sexes ($p < 0.028$).

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:

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

where:

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

BW = assumed body weight of a child (10 kg) or an adult (70 kg).

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 and Ten-day Health Advisories

Appropriate data for the derivation of One-day and Ten-day HAs were not located. It is recommended that the Longer-term HA of 0.74 mg/L for the 10 kg child be used as a conservative estimate for One-day and Ten-day exposures.

Longer-term Health Advisory

A combination of three inhalational studies in which various animal species were exposed to 1,2-dichloroethane for up to eight months are considered appropriate to use in calculating a Longer-term HA. In these studies, exposures of rats and guinea pigs to air containing 100 ppm 1,2-dichloroethane for 6 to 7 hours/day, 5 days/week resulted in no mortality and no adverse effects as determined by general appearance, behavior, growth, organ function or blood chemistry. However, similar exposures of rats, guinea pigs, rabbits, and monkeys to air containing 400 or 500 ppm 1,2-dichloroethane resulted in high mortality and varying pathological findings including pulmonary congestion, diffused myocarditis, slight to moderate fatty degeneration of the liver, kidney, adrenal, and heart, and increased plasma prothrombin time (Heppel et al., 1946; Spencer et al., 1951; Hofmann et al., 1971).

The Longer-term HA is calculated as follows:

Step 1: Determination of Total Absorbed Dose (TAD)

$$TAD = \frac{(405 \text{ mg/m}^3) \ (1 \text{ m}^3/\text{hr}) \ (6 \text{ hr}) \ (0.3) \ (5/7)}{70 \text{ kg}} = \frac{521 \text{ mg/day}}{70 \text{ kg}} = 7.4 \text{ mg/kg/day}$$

where:

405 mg/m^3 = NOAEL of 100 ppm (1 ppm = 4.05 mg/m^3) for adverse effects in rats and guinea pigs.

1 m^3/hr = respiratory rate of adult human (pulmonary rate/body weight ratio assumed to be the same for humans and test animals).

6 hr = exposure duration per day.

0.3 = fraction of test substance assumed to be absorbed.

5/7 = conversion of 5-day dosing regimen to full 7-day week.

70 kg = assumed body weight of an adult.

Step 2: Determination of the Longer-term HA

For a 10-kg child:

$$\text{Longer-term HA} = \frac{(7.4 \text{ mg/kg/day})\ (10 \text{ kg})}{(100)\ (1 \text{ L/day})} = 0.74 \text{ mg/L } (740 \text{ ug/L})$$

For a 70-kg adult:

$$\text{Longer-term HA} = \frac{(7.4 \text{ mg/kg/day})\ (70 \text{ kg})}{(100)\ (2 \text{ L/day})} = 2.6 \text{ mg/L } (2600 \text{ ug/L})$$

where:

7.4 mg/kg/day = total absorbed dose (TAD).

10 kg = assumed body weight of a child.

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

70 kg = assumed body weight of an adult.

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

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

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.

No appropriate data are available for determining a reference dose and drinking water equivalent level (DWEL) for 1,2-dichloroethane. A Lifetime Health Advisory is not estimated for this chemical.

Evaluation of Carcinogenic Potential

- 1,2-Dichloroethane was shown to be carcinogenic in rats and mice following gavage exposure in the NCI bioassay (NCI, 1978).

- IARC has not classified 1,2-dichloroethane (IARC, 1982).

- Applying the criteria described in EPA's guidelines for assessment of carcinogenic risk (U.S. EPA, 1986), 1,2-dichloroethane may be classified in Group B2: Probable Human Carcinogen. This category is for agents for which there is inadequate evidence from human studies and sufficient evidence from animal studies.

- The most recent calculations by EPA's Carcinogen Assessment Group (CAG) indicates the cancer risk estimate for 1,2-dichloroethane corresponding to a 10^{-5} risk level is 3.8 ug/L, using the multistage model (95% confidence limit) (U.S. EPA, 1985d).

- The linear multistage model is only one method of estimating carcinogenic risk. Using the 95% upper-bound estimate of risk at 1 mg/kg/day for hemangiosarcomas in male rats, the following comparisons can be made: Multistage, 6.0×10^{-2}; Probit, 2.81×10^{-1}; Weibull, 2.7×10^{-1} (U.S. EPA, 1985a). Each model is based on differing assumptions. No current understanding of the biological mechanisms of carcinogenesis is able to predict which of these models is more accurate than another.

- While recognized as statistically alternative approaches, the range of risks described by using any of these modelling approaches has little biological significance unless data can be used to support the selection of one model over another. In the interest of consistency of approach and in providing an upper bound on the potential cancer risk, the Agency has recommended use of the linearized multistage approach.

VI. OTHER CRITERIA, GUIDANCE AND STANDARDS

- U.S. EPA (1985d) has promulgated a final Recommended Maximum Contaminant Level (RMCL) of zero for 1,2-dichloroethane in drinking water based upon its carcinogenic potential and has proposed a Maximum Contaminant Level (MCL) of 0.005 mg/L.

- Due to the lack of appropriate data, the National Academy of Sciences did not calculate a chronic Suggested-No-Adverse-Response-Level (SNARL) for 1,2-dichloroethane (NAS, 1980).

- ACGIH (1984) has recommended a threshold limit value (TLV) of 10 ppm ($\sim$ 40 mg/m^3) and a short-term exposure level (STEL) of 15 ppm ($\sim$ 60 mg/m^3) due to its hepatotoxic effects.

VII. ANALYTICAL METHODS

- Analysis of 1,2-dichloroethane is by a purge-and-trap gas chromatographic procedure used for the determination of volatile organohalides in drinking water (U.S. EPA, 1985b). This method calls for the bubbling of an inert gas through the sample and trapping 1,2-dichloroethane on the adsorbant material. The adsorbant material is heated to drive off the 1,2-dichloroethane onto a gas chromatographic column. The gas chromatograph is temperature programmed to separate the method analytes which are then detected by a halogen specific detector. This method is applicable to the measurement of 1,2-dichloroethane over a concentration range of 0.2 to 1,500 ug/L. Confirmatory analysis for 1,2-dichloroethane is by mass spectrometry (U.S. EPA, 1985c). The detection limit for confirmation by mass spectrometry is 0.3 ug/L.

VIII. TREATMENT TECHNOLOGIES

- Treatment technologies which will remove 1,2-dichloroethane from water include granular activated carbon (GAC) adsorption, aeration and boiling.

- Dobbs and Cohen (1980) developed adsorption isotherms for several organic chemicals including 1,2-dichloroethane. It was reported that Fibrasorb® 300 carbon exhibited adsorptive capacities of 3.5 mg and 0.5 mg 1,2-dichloroethane/gm carbon at equilibrium concentrations of 1,000 and 100 mg/L, respectively. Also, Love (1983) reported that Witcarb® 950 carbon exhibited adsorptive capacities of 1.9 mg and 0.6 mg 1,2-dichloroethane/gm carbon at equilibrium concentrations of 100 and 10 mg/L, respectively. USEPA-DWRD installed pilot-scale adsorption columns in New Jersey to treat contaminated groundwater (Love and Eilers, 1982). A Witcarb® 950 carbon column removed 1,2-dichloroethane from a concentration as high as 8 mg/L to 0.1 mg/L. Breakthrough occurred at 1,700 bed volumes (BV) with an empty bed contact time (EBCT) of 18 minutes. Similar studies in Louisiana showed removal of 1,2-dichloroethane from a concentration of 8 mg/L

to less than 0.1 mg/L after 39 days of continuous operation by a full-scale GAC column containing Nuchar® WF-G activated carbon (Love, 1983).

- 1,2-Dichloroethane is amenable to aeration on the basis of its Henry's Law Constant of 61 atm (Kavanaugh and Trussell, 1980). In a pilot-scale diffused air aeration column, removal efficiency of 42% of 1,2-dichloroethane was achieved at an air-to-water ratio of 4:1 (Love and Eilers, 1982). In a pilot-scale packed tower aeration study removal efficiencies of 85 to 98.5% for 1,2-dichloroethane were achieved on air-to-water ratios of 5-45, respectively (ESE, 1985).

- Boiling also is effective in eliminating 1,2-dichloroethane from water on a short-term, emergency basis. Studies have shown that 5 to 10 minutes of vigorous boiling will remove 88 to 98% of 1,2-dichloroethane originally present (Love, 1983).

- Air stripping is an effective, simple and relatively inexpensive process for removing 1,2-dichloroethane and other volatile organics from water. However, use of this process then transfers the contaminant directly to the air stream. When considering use of air stripping as a treatment process, it is suggested that careful consideration be given to the overall environmental occurrence, fate, route of exposure and various other hazards associated with the chemical.

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