

March 31, 1987

TRICHLOROETHYLENE

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.

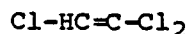
Health Advisories are developed for One-day, Ten-day, Longer-term (approximately 7 years, or 10% of an individual's lifetime) and Lifetime exposures based on data describing noncarcinogenic end points of toxicity. Health Advisories do not quantitatively incorporate any potential carcinogenic risk from 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 upon information presented in the Office of Drinking Water's Health Effects Criteria Document (CD) for Trichloroethylene (U.S. EPA, 1985a). 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 # 86-118106/AS. The toll free number is (800) 336-4700; in Washington, D.C. area: (703) 487-4650.

II. GENERAL INFORMATION AND PROPERTIES

CAS No. 79-01-6

Structural Formula



Trichloroethylene

Synonyms

TCE, trichloroethene, acetylene trichloride, Tri, Trilene

Uses

Industrial solvent and degreaser for metal components

Properties (Torkelson and Rowe, 1981; Windholtz, 1983)

Chemical Formula	C_2HCl_3
Molecular Weight	131.40
Physical State	Colorless liquid
Boiling Point	86.7°C
Vapor Pressure	77 mm (25°C)
Density at 25°C	1.4 g/mL
Water Solubility	0.1 g/100 mL (20°C)
Odor Threshold (water)	0.5 mg/L
Odor Threshold (air)	2.5-900 mg/m ³
Organoleptic Threshold (water)	0.31 mg/L (Amoore and Hautala, 1983)
Conversion Factor	1 ppm = 5.46 mg/m ³

Occurrence

- Trichloroethylene (TCE) is a synthetic chemical with no natural sources.
- Production of TCE was 200 million lbs in 1982 (U.S. ITC, 1983).
- The major source of TCE released to the environment is from its use as a metal degreaser. Since TCE is not consumed during this use, the majority of all TCE production is released to the environment. Most

of the releases occur to the atmosphere by evaporation. However, TCE which is not lost to evaporation becomes heavily contaminated with grease and oil and has been disposed of by burial in landfills, dumping on the ground or into sewers. Because metal working operations are performed nationwide, TCE releases occur in all industrialized areas. Releases of TCE during production and other uses are relatively minor.

- Trichloroethylene released to the air is degraded in a matter of a few days. Trichloroethylene released to surface waters migrates to the atmosphere in a few days or weeks where it also degrades. Photo-oxidation appears to be the predominant fate of this compound (U.S. EPA, 1979). Trichloroethylene which is released to the land does not degrade rapidly, migrates readily to ground water and remains in ground water for months to years. Under certain conditions, TCE in groundwater appears to degrade to dichloroethylene and vinyl chloride. Trichloroethylene also may be formed in ground water by the degradation of tetrachloroethylene (Parsons et al., 1984; Vogel and McCarty, 1985). Trichloroethylene, unlike other chlorinated compounds, does not bioaccumulate in individual animals or food chains.
- Because of the large and dispersed releases, TCE occurs widely in the environment. Trichloroethylene is ubiquitous in the air with levels in the ppt to ppb range. Trichloroethylene is a common contaminant in ground and surface waters with higher levels found in ground water. Surveys of drinking water supplies have found that 3% of all public systems derived from well water contain TCE at levels of 0.5 ug/L or higher. A small number of systems (0.04%) have levels higher than 100 ug/L. Public systems derived from surface water also have been found to contain TCE but at lower levels. Trichloroethylene has been reported to occur in some foods in the ppm range.
- The major sources of exposure to TCE are from contaminated water and to a lesser extent air; food is only a minor source of TCE exposure (U.S. EPA, 1983).

III. PHARMACOKINETICS

Absorption

- Data on absorption of ingested TCE are limited. When a dose of 200 mg/kg of ¹⁴C-TCE in corn oil was administered to rats, 97% of the dose was recovered during 72 hours after dosing (DeKant et al., 1984).

Distribution

- Doses of 0, 10, 100 or 1,000 mg TCE/kg/day were administered by gavage to rats five days/week for six weeks (Zenick et al., 1984). Marginal increases in TCE tissue levels were detected in the 10 mg/kg/day and 100 mg/kg/day dose groups. Compared to controls, a marked increase in TCE levels in most tissues was observed in the highest dose group. Trichloroethylene was distributed in all tissues examined with the highest concentrations in the fat, kidney, lung, adrenals, vas deferens, epididymis, brain and liver.

Metabolism

- Studies indicate that TCE is metabolized to trichloroethylene oxide, trichloroacetaldehyde, trichloroacetic acid, monochloroacetic acid, trichloroethanol and trichloroethanol glucuronide (U.S. EPA, 1985a).

Excretion

- Trichloroethylene and its metabolites are excreted in urine, by exhalation and, to a lesser degree, in sweat, feces and saliva (Soucek and Vlachova, 1959).

IV. HEALTH EFFECTSHumansShort-term Exposure

- Oral exposure of humans to 15 to 25 ml (21 to 35 g) quantities of TCE resulted in vomiting and abdominal pain, followed by transient unconsciousness (Stephans, 1945).

Long-term Exposure

- Studies of humans exposed occupationally have shown an increase in serum transaminases, which indicates damage to the liver parenchyma (Lachnit, 1971). Quantitative exposure levels were not available.

AnimalsShort-term Exposure

- The acute oral LD₅₀ of TCE in rats is 4.92 g/kg (NIOSH, 1980).

Long-term Exposure

- Rats exposed to 300 mg/m³ (55 ppm) TCE five days/week for 14 weeks had elevated liver weights (Kimmerle and Eben, 1973).

Reproductive Effects

- No data were available on the reproductive effects of TCE.

Developmental Effects

- No data were available on the developmental effects of TCE.

Mutagenicity

- Trichloroethylene was mutagenic in Salmonella typhimurium and in the B. coli K-12 strain, utilizing liver microsomes for activation (Greim et al., 1975, 1977).

Carcinogenicity

- Technical TCE (containing epichlorohydrin and other compounds) was found to induce a hepatocellular carcinogenic response in B6C3F₁ mice (NCI, 1976). Under the conditions of this experiment, a carcinogenic response was not observed in Osborne-Mendel rats. The "time-weighted" average doses were 549 and 1,097 mg/kg for both male and female rats. The time-weighted average daily doses were 1,169 and 2,339 mg/kg for male mice and 869 and 1,739 mg/kg for female mice.
- Epichlorohydrin-free TCE was reported to be carcinogenic in B6C3F₁ mice when administered in corn oil at 1,000 mg/kg/day, 5 days/wk, for 103 weeks (NTP, 1982). It was not found to be carcinogenic in female Fischer 344 rats when administered in corn oil at 500 or 1,000 mg/kg/day, 5 days/wk, for 103 weeks. The experiment with male rats was considered to be inadequate since these rats received doses of TCE that exceeded the maximum tolerated dose.
- TCE has been shown to be carcinogenic in mice utilizing the inhalation as well as the oral route of exposure. The National Cancer Institute (1976) and the National Toxicology Program (1982) each conducted an oral gavage study with TCE, one contaminated with epichlorohydrin and the other free of epichlorohydrin, respectively. In these studies, as described above, B6C3F₁ mice were used, and the results were unequivocally positive, showing liver neoplasms.
- In an inhalation study, Henschler et al. (1980) reported dose-related malignant lymphomas in female mice exposed to 100 or 500 ppm TCE vapor 6 hrs/day, 5 days/wk, for 18 months (HAN:NMRI strain). However, the authors downplayed the significance of this observation, indicating that this strain of mice has a high incidence of spontaneous lymphomas.
- Fukuda et al. (1983) found pulmonary adenocarcinomas in female ICR mice on exposure to TCE vapor.
- Henschler et al. (1984) tested Swiss (ICR/HA) mice and reported that when the animals were treated by gavage with TCE in corn oil, no statistical differences were observed in the incidence of cancers. The results of this study can be questioned because the dose schedule was often interrupted even with half of the original dose. Therefore, it is very difficult to assess the exposure. A slight increase in tumors was found in all groups treated with TCE but did not approach statistical significance.
- The Van Duuren study (1979) with skin applications of TCE in ICR/HA mice does not negate the positive findings with other strains of mice and other routes of exposure.

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:

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

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 Advisory

Suitable data were not available to estimate One-day and Ten-day Health Advisories.

Longer-term Health Advisory

No suitable data are available from which to calculate a Longer-term Health Advisory.

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.

Trichloroethylene may be classified in Group B: Probable Human Carcinogen, according to EPA's weight-of-evidence scheme for the classification of carcinogenic potential (U.S. EPA, 1986). Because of this, caution must be exercised in making a decision on how to deal with possible lifetime exposure to this substance. The risk manager must balance this assessment of carcinogenic potential against the likelihood of occurrence of health effects related to non-carcinogenic end-points of toxicity. In order to assist the risk manager in this process, drinking water concentrations associated with estimated excess lifetime cancer risks over the range of one in ten thousand to one in a million for the 70 kg adult, drinking 2 liters of water per day, are provided in the following section. In addition, in this section, a Drinking Water Equivalent Level (DWEL) is derived. A DWEL is defined as the medium-specific (in this case, drinking water) exposure which is interpreted to be protective for non-carcinogenic end-points of toxicity over a lifetime of exposure. The DWEL is determined for the 70 kg adult, ingesting 2 liters of water per day. Also provided is an estimate of the excess cancer risk that would result if exposure were to occur at the DWEL over a lifetime.

Neither the risk estimates nor the DWEL take relative source contribution into account. The risk manager should do this on a case-by-case basis, considering the circumstances of the specific contamination incident that has occurred.

The study by Kimmerle and Eben (1973) is the most appropriate from which to derive the DWEL. This study evaluated the subacute exposure to trichloroethylene via inhalation by adult rats for some 14 weeks following exposure to 55 ppm (300 mg/m³), five days a week. Indices of toxicity include hematological investigation, liver and renal function tests, blood glucose and organ/body weight ratios. Liver weights were shown to be elevated while other test values were not different from controls. The elevated liver weights could be interpreted to be the result of hydropic changes or fatty accumulation. The no-observed-effect level was not identified since only a single concentration was administered. From these results, a LOAEL of 55 ppm (300 mg/m³) was identified. Using the LOAEL, the DWEL is derived as follows:

Step 1: Determination of the Total Absorbed Dose (TAD)

$$\text{TAD} = \frac{(300 \text{ mg/m}^3) (8 \text{ m}^3/\text{day}) (5/7) (0.3)}{(70 \text{ kg})} = 7.35 \text{ mg/kg/day}$$

where:

300 mg/m³ = LOAEL for liver effects in rats

8 m³/day = Volume of air inhaled during the exposure period

5/7 = Conversion factor for adjusting from 5 days/week exposure to a daily dose

0.3 = Ratio of the dose absorbed.

70 kg = Assumed weight of adult.

Step 2: Determination of the Reference Dose (RfD)

$$\text{RfD} = \frac{7.35 \text{ mg/kg/day}}{(100) (10)} = 0.00735 \text{ mg/kg/day}$$

where:

$$7.35 \text{ mg/kg/day} = \text{TAD.}$$

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

Step 3: Determination of the Drinking Water Equivalent Level (DWEL)

$$\text{DWEL} = \frac{(0.00735 \text{ mg/kg/day})(70 \text{ kg})}{2 \text{ L/day}} = 0.26 \text{ mg/L (260 ug/L)}$$

where:

$$0.00735 \text{ mg/kg/day} = \text{RfD.}$$

70 kg = assumed body weight of an adult.

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

The estimated excess cancer risk associated with lifetime exposure to drinking water containing TCE at 260 ug/L is approximately 1×10^{-4} . This estimate represents the upper 95% confidence limit from extrapolations prepared by EPA's Carcinogen Assessment Group using the linearized, multistage model. The actual risk is unlikely to exceed this value, but there is considerable uncertainty as to the accuracy of risks calculated by this methodology.

Evaluation of Carcinogenic Potential

- IARC (1982) has classified TCE in Group 3.
- Trichloroethylene has been classified in Group B2: Probable Human Carcinogen. This classification for carcinogenicity was determined by a technical panel of EPA's Risk Assessment Forum using the EPA risk assessment guidelines for carcinogens (U.S. EPA, 1986). This category is used for agents for which there is "sufficient evidence" for human carcinogenicity from animal studies and for which there is "inadequate evidence" or "no data" from human studies.
- Using the improved multistage linearized model, it can be estimated that water with TCE concentrations of 280 ug/L, 28 ug/L or 2.8 ug/L may increase the risk of one excess cancer per 10⁴, 10⁵ or 10⁶ people exposed, respectively. These estimates were calculated from the 1976 NCI bioassay data, which utilized TCE contaminated with epichlorohydrin. Since then, an NTP (1982) bioassay utilizing epichlorohydrin-free TCE has become available; the data from this bioassay have been reviewed and evaluated for carcinogenicity, and epichlorohydrin-free TCE has been reported to be carcinogenic in mice.

VI. OTHER CRITERIA, GUIDANCE AND STANDARDS

- ACGIH (1984) has recommended a threshold limit value (TLV) of 50 ppm (~ 270 mg/m³) and a short-term exposure limit (STEL) of 150 ppm (~ 805 mg/m³).
- The NAS (1980) recommended One- and Seven-day SNARLS of 105 and 15 mg/L, respectively.
- The WHO (1981) recommended a drinking water guidance level of 30 ug/L based on a carcinogenic end point.
- The EPA (U.S. EPA, 1980) recommended a water quality criterion of 6.77 mg/L for effects other than cancer.
- The EPA (U.S. EPA, 1985d) has promulgated a Recommended Maximum Contaminant Level (RMCL) of zero based upon its classification as a known or probable human carcinogen and has proposed a Maximum Contaminant Level (MCL) of 0.005 mg/L based on its RMCL and appropriate feasibility studies.

VII. ANALYTICAL METHODS

- Analysis of TCE 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 TCE on an adsorbant material. The adsorbant material is heated to drive off the TCE onto a gas chromatographic column. This method is applicable to the measurement of TCE over a concentration range of 0.01 to 1500 ug/L. Confirmatory analysis for TCE is by mass spectrometry (U.S. EPA, 1985c). The detection limit for confirmation by mass spectrometry is 0.2 ug/L.

VIII. TREATMENT TECHNOLOGIES

- Treatment technologies which will remove TCE from water include granular activated carbon (GAC) adsorption, aeration and boiling.
- Dobbs and Cohen (1980) developed adsorption isotherms for several organic chemicals including TCE. It was reported that Fibrasorb® 300 carbon exhibited adsorptive capacities of 7 mg, 1.6 mg and 0.4 mg TCE/gm carbon at equilibrium concentrations of 100, 10 and 1 mg/L, respectively. USEPA-DWRD installed pilot-scale adsorption columns at different sites in New England and Pennsylvania. In New England, contaminated well water with TCE concentrations ranging from 0.4 to 177 mg/L was passed through GAC columns until a breakthrough concentration of 0.1 mg/L was achieved with empty bed contact time (EBCT) of 18 and 9 minutes, respectively (Love and Eilers, 1982). In Pennsylvania, TCE concentrations ranging from 20 to 130 mg/L were reduced to 4.5 mg/L by GAC after 2 months of continuous operation (ESE, 1985).

- TCE is amenable to aeration on the basis of its Henry's Law Constant of 550 atm (Kavanaugh and Trussell, 1980). In a full plant-scale (3.78 MGD) redwood slat tray aeration column, a removal efficiency of 50-60% was achieved from TCE initial concentrations of 8.3-39.5 mg/L at an air-to-water ratio of 30:1 (Hess et al., 1981). In another full plant-scale (6.0 MGD) multiple tray aeration column study, TCE removal of 52% was achieved from 150 mg/L (Hess et al., 1981). A full plant-scale packed tower aeration column removed 97-99% of TCE from 1,500-2,000 mg/L contaminated groundwater at air-to-water ratio of 25:1 (ESE, 1985).
- Boiling also is effective in eliminating TCE from water on a short-term, emergency basis. Studies have shown 5 minutes of vigorous boiling will remove 95% of TCE originally present (Love and Eilers, 1982).
- Air stripping is an effective, simple and relatively inexpensive process for removing TCE 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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