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1,2,3-TRICHLOROPROPANE

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

I. INTRODUCTION

The Health Advisory 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.

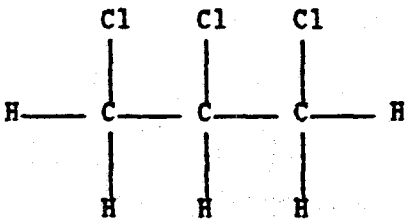
Health Advisories serve as informal technical guidance to assist Federal, State and local officials responsible for protecting public health when emergency spills or contamination situations occur. They are not to be construed as legally enforceable Federal standards. The HAs are subject to change as 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. For those substances that are known or probable human carcinogens, according to the Agency classification scheme (Group A or B), Lifetime Health Advisories 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 lifelong exposure and the ingestion of water. The cancer 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.

II. GENERAL INFORMATION AND PROPERTIES

CAS No. 96-18-4

Structural Formula



1,2,3-Trichloropropane

Synonyms

- Allyl trichloride, glycerol trichlorohydrin, glyceryl trichlorohydrin, trichlorohydrin (NIOSH, 1986).

Uses

- 1,2,3-TCP is used as a paint and varnish remover, solvent, degreasing agent, and crosslinking agent in the clastomer Thiokiol ST (U.S. EPA, 1983).

Properties (Weast, 1972; Verschueren, 1977; Koneman, 1981; U.S. EPA, 1983)

Chemical Formula	$C_3H_5Cl_3$
Molecular Weight	147.43
Physical State (at 25°C)	Colorless clear liquid
Boiling Point (25 mm Hg)	156.85°C
Melting Point	-14.7°C
Density (20°C)	1.387
Vapor Pressure (20°C)	2 mm Hg
Specific Gravity (20°C)	1.3889
Water Solubility (20°C)	1,900 mg/L
Log Octanol Water Partition Coefficient	2.63
Odor Threshold (water)	--
Odor Threshold (air)	--
Taste Threshold	--
Conversion Factor	1 ppm = 6 mg/m^3

Occurrence

- Drinking water from Carrollton Water Plant in New Orleans, LA, contained <0.2 µg/L of 1,2,3-TCP (Keith et al., 1976). It was also detected in Ames, IA, drinking water; however, levels were not given, (U.S. EPA, 1976). Kool et al. (1982) reported that 1,2,3-TCP had been detected in drinking water in unspecified locations.

- Surface water from the Delaware River basin contained trichloropropane (unspecified isomer) at concentrations >1 µg/L in 3% of samples (Dewalle and Chian, 1978). Wakeham et al. (1983) found trichloropropane in seawater of Narragansett Bay, RI, but concentrations were not reported.

Environmental Fate

- Dilling (1977) reported that the half-life for evaporation of 1,2,3-TCP from water was about 1 hour under the following conditions: 0.92 ppm aqueous solution; 6.5 cm deep; 200 rpm stirring; 25°C; <0.2 mph air current.

- Matsui et al. (1975) determined that trichloropropane (unspecified isomer) was relatively easy to decompose by microbes in activated sludge.

III. PHARMACOKINETICS

Absorption

- Data regarding the absorption of 1,2,3-TCP could not be located in the available literature.

Distribution

- Volp et al. (1984) administered 3.6 mg/kg bw C^{14} 1,2,3-TCP (label at 1,3 carbon) intravenously to male Fischer 344 rats. Tissues and excreta were analyzed for total radioactivity and unchanged 1,2,3-TCP at various time periods following the administration of 1,2,3-TCP. The distribution and excretion of 1,2,3-TCP were rapid. 37% of the dose was accounted for in adipose tissue within 15 min. This consisted of primarily unchanged 1,2,3-TCP. The largest fraction of the dose was detected in the liver in the form of metabolites after a 4 hour exposure of 1,2,3-TCP. The kidneys also accumulated radiolabeled 1,2,3-TCP with a peak of 2.8% of the total dose at 2 hours, decreasing thereafter to <1% at the end of the 24 hour period. The small intestine had a concentration of 9.3% of the dose at 1 hour. Brain, lungs, spleen, testes and epidymides contained <0.5% of the total dose at all times. The primary sites of distribution associated with radiolabeled 1,2,3-TCP were initially the adipose tissue, skin and muscle, then subsequently the liver.

Metabolism

- In the Volp et al. (1984) study described above, 1,2,3-TCP was rapidly distributed to all tissues, specifically adipose tissue, skin and muscle. After 4 hours, the concentration of unchanged 1,2,3-TCP was 90% of the total radiolabeled compound in adipose tissue (3.8% of the dose). This concentration was observed to decrease to 37% at the 24 hour period following the administration of compound. The investigators reported that (1) the major metabolite of 1,2,3-TCP was carbon dioxide (25% of dose), and (2) other minor metabolites were also present but not identified.

Excretion

- In the Volp et al. (1984) study described above 99% of the dose was excreted within 6 days. Most of the excretion (90%) occurred in the first 24 hours, with urine being the principal route. Of the total dose of radioactivity, 40% was excreted in urine, 30% in expired air, and 18% in the feces in the first 24 hours. The urine contained no detectable 1,2,3-TCP, indicating that all of the radiolabel was 1,2,3-TCP metabolites. Of the 30% of the initial dose of radioactivity eliminated in expired air, 5% was unchanged TCP and 25% was CO_2. Almost all of the unchanged TCP (85%) was expired within 30 minutes. In the bile, 30% of the total dose appeared within 6 hours, 5% of which was unchanged 1,2,3-TCP. The elimination half-time for unchanged 1,2,3-TCP was 30 to 45 hours for all major tissues. The half-time for elimination of radiolabel by all routes was 44 hours.

IV. HEALTH EFFECTS

Humans

Short-term Exposure

- Silverman et al. (1946) exposed an average of 12 volunteers (males and females) to 1,2,3-TCP and other industrial solvent vapors for 15 minutes, and found that 100 ppm 1,2,3-TCP (600 mg/m^3) caused eye and throat irritation and had an unpleasant odor. A "borderline majority" of the subjects said that 50 ppm (300 mg/m^3) would be acceptable for an 8-hour workday. However, this level is based on organoleptic quality and not toxicity.

Long-term Exposure

- Pertinent data regarding long-term exposure of humans to 1,2,3-TCP could not be located in the available literature.

Animals

Short-term Exposure

- Saito-Suzuki et al. (1982) reported that 500 mg/kg bw 1,2,3-TCP by gastric intubation to male Sprague-Dawley rats was lethal. Smythe

et al. (1962) reported an oral LD_{50} of 0.32 mL/kg bw (444 mg/kg) 1,2,3-TCP for Carworth-Wistar male rats. An oral LD_{50} of 320 mg/kg for 1,2,3-TCP also has been reported in the literature (RTECS, 1978).

- Wright and Schaffer (1932) administered one dose (route not specified) of 0.2 to 0.5 cc/kg (278-694 mg/kg) 1,2,3-TCP to three dogs. All dogs died within 1-2 days after dosing. The major signs of toxicity were narcosis and liver and kidney tissue necrosis.

- Shcherban and Piten'ko (1976) reported the following LD_{50} values for 1,2,3-TCP (route not specified): rats, 505 mg/kg; mice, 369 mg/kg; rabbits, 380 mg/kg; and guinea pigs, 340 mg/kg. They also reported that 0.0035 mg/kg was a completely nontoxic dose. No other details were given.

- Several short-term inhalation studies using 1,2,3-TCP were available. Smythe et al. (1962) reported that 5/6 rats died when exposed to 6,000 mg/m^3 (1,000 ppm) for 4 hours. McOmie and Barnes (1949) reported that exposure to a vapor concentration of 30,000 mg/m^3 (5,000 ppm) for 20 minutes killed several mice (8/15 within 2 days). Four of the remaining seven mice died from liver damage 7 to 10 days later. When exposed to 15,000 mg/m^3 (2,500 ppm), 10 minutes/day for 10 days, 7/10 mice died. Lewis (1979) exposed rats and guinea pigs (five/sex) to 4,800 mg/m^3 (799 ppm), 12,480 mg/m^3 (2,080 ppm) or 30,060 mg/m^3 (5,010 ppm) for 30 minutes, resulting in dose-related central nervous system (CNS) depression. Six guinea pigs and two rats at the high dose 30,060 mg/m^3 (5,010 ppm) died.

- Sidorenko et al. (1979) exposed white male rats (strain not specified) to 1,2,3-TCP 2 to 800 mg/m^3 (0.33 to 133 ppm) for periods ranging from 2 hours to 86 days. An increase in the activity of blood catalase, acetylcholinesterase, and the excitability of nerve centers was reported. These changes were observed in rats after 4 hours of exposure to 800 mg/m^3 (133 ppm) and after 40 days of exposure to 2 mg/m^3 (0.33 ppm).

Dermal/Ocular Effects

- Smythe et al. (1962) reported a single skin penetration LD_{50} of 1.77 mL/kg (2,458 mg/kg) 1,2,3-TCP for rabbits. On a scale of 1 to 10 (1 = least severe, 10 = most severe), 1,2,3-TCP rated 1 for skin irritation and 4 for corneal injury.

- McOmie and Barnes (1949) determined 1,2,3-TCP to be an "intense skin irritant" for rabbits, partially due to its lipid solvent properties. Repeated applications led to sloughing and cracking preceded by irritation and erythrema. In a 15-day period, 10 applications of 2 mL/100 cm^2 led to pain, subdermal hemorrhage and death in 1/7 treated rabbits. The other six rabbits survived and healed within 5 weeks.

Long-term Exposure

- 1,2,3-TCP was administered by gavage in corn oil, 5 days/week for 120 days, to Fischer 344 rats (20/sex/group) at dose levels of 8, 16, 32, 63, 125 and 250 mg/kg bw/day (NTP, 1983a). One control group of 30 rats/sex received corn oil. All animals in the 250 mg/kg dose group died as a result of treatment, with the main findings being renal and hepatic toxicity and necrosis and inflammation of the nasal mucosa. Mortality was also observed in the 125 mg/kg dose group. Dose-related clinical effects (e.g., thin and hunched appearance, depression, abnormal eyes and urine stains) were observed in female rats at doses of ≥125 mg/kg. Hematological effects (decreased hematocrit, hemoglobin and erthrocyte counts) were seen in both sexes at doses of ≥16 mg/kg. There was a dose-related increase in liver and kidney weights. At 125 mg/kg there was also an increase in the weight of testes and a decrease in epididymis weight, but no histomorphologic change was observed. Principal target organs were the liver and kidney, with histomorphological and clinical chemistry changes observed in dose groups ≥63 mg/kg. The nasal turbinates were also a target, but it was suggested that this may have been due to a local effect as opposed to a systemic effect. The NOAEL for this study is 8 mg/kg and the LOAEL is 16 mg/kg based on hematologic effects.

- 1,2,3-TCP was administered by gavage in corn oil, 5 days/week for 120 days, to B6C3F$_1$ mice (20/sex/group) at dose levels of 8, 16, 32, 63, 125 and 250 mg/kg bw/day (NTP, 1983b). One control group of 30 mice/sex received corn oil. Treatment-related deaths due primarily to hepatic toxicity occurred particularly in males at the 250 mg/kg level. The principal target organs were the liver, lung, kidney and stomach, with effects also seen in the spleen and nasal passages. Body weight gain was not affected except for a decrease in two male survivors in the 250 mg/kg group. Evaluation of the hematological and clinical chemistry data revealed no changes of biological importance since findings were sporadic in distribution and noted by the authors as "incidental to compound administration." Increased weights/or ratios were noted in the liver and thymus at doses ≥125 mg/kg. The lowest dose with a statistically significant effect was 16 mg/kg, which resulted in a lower brain weight ratio in female mice. This is the basis for defining 16 mg/kg as the LOAEL for this study. The NOAEL is 8 mg/kg.

Reproductive Effects

- Johannsen et al. (1988) reported the results of reproduction studies in the rat following repeated inhalation exposure. Groups of 10 male and 20 female rats were exposed 6 h/d, 5 d/wk to 5 ppm (30 mg/m^3) or 15 ppm (90 mg/m^3) 1,2,3-trichloropropane vapor during premating and mating. Female rats were also exposed during gestation. Investigators stated that (1) the body weights of both sexes of the 5 ppm (30 mg/m^3) level were comparable to control values, (2) at the 15 ppm (90 mg/m^3) level, both sexes exhibited

lower mean body weights and significantly ($p \leq 0.01$) lower mean weight gains during the premating period, (3) mating performance was low in all groups of female rats including the controls, and (4) all measured progeny indices appeared unaffected by inhalation exposure of 1,2,3-trichloropropane.

Developmental Effects

- No treatment-related effects on incidence of grossly visible internal or external malformations occurred in the offspring of female Sprague-Dawley rats injected intraperitoneally with 37 mg/kg bw 1,2,3-TCP in corn oil on days 1 through 15 of gestation (Hardin et al., 1981).

- Hardin et al. (1981) administered by intraperitoneal (i.p.) injection 37 mg/kg bw 1,2,3-TCP in corn oil to groups of 10 to 15 pregnant Sprague-Dawley rats on days 1 through 15 of gestation. Exposure caused maternal toxicity as indicated by reduced body weight gain or altered organ weights in two or more organs, but did not cause fetotoxicity (reduced fetal size or reduced survival rate).

Mutagenicity

- Stolzenberg and Hime (1980) reported that 1,2,3-TCP was mutagenic to Salmonella typhimurium only with a microsomal activating system (S-9). At the two concentrations evaluated on the tester strain TA100, a dose-dependent increase in revertant colony numbers was observed in the presence of S-9 mix but not in its absence.

- Results were negative in a dominant lethal assay in which 80 mg/kg bw/day 1,2,3-TCP dissolved in olive oil was administered by gastric intubation to Sprague-Dawley rats for 5 consecutive days (Saito-Suzuki et al., 1982).

- In a dominant lethal assay in which 15 male Sprague-Dawley rats received gavage doses of 80 mg/kg bw/day for 5 consecutive days prior to mating, no effects were seen on reproductive performance (frequency of fertile matings) (Saito-Suzuki et al., 1982). No testicular lesions were observed.

Carcinogenicity

- Pertinent data regarding the carcinogenicity of 1,2,3-TCP could not be located at the time of this publication. The National Toxicology Program is currently conducting a 2-year gavage study in rats and mice (NTP, 1988). A judgment of carcinogenicity will be deferred until this study is completed.

V. QUANTIFICATION OF TOXICOLOGICAL EFFECTS

Health Advisories (HAs) are generally determined for one-day, ten-day, longer-term (up to 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:

$$\frac{\text{(NOAEL or LOAEL) x (BW)}}{\text{(UF) (\_\_\_\_ L/day)}} = \text{\_\_\_\_ mg/L (\_\_\_\_ µg/L)}$$

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, 1,000 or 10,000), in accordance with EPA or 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

Sufficient data are not available for the derivation of a One-day HA for 1,2,3-TCP. Available oral data in rats (Saito-Suzuki et al., 1982; Smythe et al., 1962) and dogs (Wright and Schaffer, 1932) define lethal dosages, but sublethal effects were not investigated. In absence of toxicity data, the Longer-term HA value for a child (600 µg/L) is recommended at this time.

Ten-day Health Advisory

Sufficient data are not available for the derivation of a Ten-day HA for 1,2,3-TCP. Several Russian inhalation studies (Sidorenko et al., 1979; Belyaeva et al., 1977; Tarasova, 1975) reported that adverse effects occurred in rats exposed to concentrations as low as 2 mg/m^3, but exposure schedules were not provided and these studies were not available for review. In absence of toxicity data, the Longer-term HA value for a child (600 µg/L) is recommended at this time.

Longer-term Health Advisory

The NTP studies (1983a,b) have been chosen to serve as the basis for the longer-term HA. Fischer 344 rats and B6C3F$_1$ mice were administered 1,2,3-TCP by gavage 5 days/week for 120 days. For both rats and mice, the lowest dose level of 8 mg/kg was a NOAEL, while 16 mg/kg was a LOAEL based on hematological effects in the rats and brain weight changes in the mice.

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

$$\text{Longer-term HA} = \frac{(8\ \text{mg/kg/day})\ (5/7)\ (10\ \text{kg})}{(100)\ (1\ \text{L/day})} = 0.57\ \text{mg/L}\ (600\ \mu\text{g/L})$$

where:

8 mg/kg/day = NOAEL, based on absence of significant effects on body and organ weight, hematology, clinical chemistry and histopathology (NTP, 1983a,b).

5/7 = factor to account for exposure of 5 out of 7 days.

10 kg = assumed body weight of a child.

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

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

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

$$\text{Longer-term HA} = \frac{(8\ \text{mg/kg/day})\ (5/7)\ (70\ \text{kg})}{(100)\ (2\ \text{L/day})} = 2\ \text{mg/L}\ (2{,}000\ \mu\text{g/L})$$

where:

8 mg/kg/day = NOAEL, based on absence of significant effects on body and organ weight, hematology, clinical chemistry and histopathology (NTP, 1983a,b).

5/7 = factor to account for exposure of 5 out of 7 days.

70 kg = assumed body weight of an adult.

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

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 (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious health effects during 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 in drinking water alone 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.

If the contaminant is classified as a known, probable or possible human carcinogen, according to the Agency's classification scheme of carcinogenic potential (U.S. EPA,1986b), then caution must be exercised in making a decision on how to deal with possible lifetime exposure to this substance. For human (A) or probable human (B) carcinogens, a Lifetime HA is not recommended. For possible human carcinogens (C), an additional 10-fold safety factor is used in the calculation of the Lifetime HA. The risk manager must balance this assessment of carcinogenic potential and the quality of the data against the likelihood of occurrence and significance of health effects related to noncarcinogenic end points of toxicity. To assist the risk manager in this process, drinking water concentrations associated with estimated excess lifetime cancer risks over the range of 1 in 10,000 to 1 in 1,000,000 for the 70-kg adult drinking 2 L of water/day are provided in the Evaluation of Carcinogenic Potential section.

There are no studies of suitable duration for the derivation of a DWEL. Therefore, the subchronic studies by NTP (1983a,b) will be used.

Step 1: Determination of the Reference Dose (RfD)

$$RfD = \frac{(8\ mg/kg/day)\ (5/7)}{(1{,}000)} = 0.006\ mg/kg/day$$

where:

8 mg/kg/day = NOAEL, based on absence of significant effects on body and organ weight, hematology, clinical chemistry and histopathology (NTP, 1983a,b).

5/7 = factor to account for exposure of 5 out of 7 days.

1,000 = uncertainty factor, chosen in accordance with EPA or NAS/ODW guidelines for use with a NOAEL from a study in animals of less than lifetime duration.

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

$$DWEL = \frac{(0.006\ mg/kg/day)\ (70\ kg)}{(2\ L/day)} = 0.2\ mg/L\ (200\ \mu g/L)$$

where:

0.006 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 the Lifetime Health Advisory

Lifetime HA = (0.2 mg/L) (20%) = 0.04 mg/L (40 µg/L)

where:

0.2 mg/L = DWEL.

(20%) = assumed relative source contribution from water.

Note: The NTP (1988) is conducting carcinogenicity studies for 1,2,3-TCP in animals. The Lifetime HA for 1,2,3-TCP will be reevaluated following a review of the results on the NTP bioassay in animals when made available by the NTP.

Evaluation of Carcinogenic Potential

- The carcinogenic potential of 1,2,3-TCP has not been reported, but this chemical is being tested for carcinogenicity by the NTP (1988). IARC has not evaluated the carcinogenic potential of 1,2,3-TCP. The evaluation for carcinogenic potential is being deferred until the completion of the NTP studies.

VI. OTHER CRITERIA, GUIDANCE AND STANDARDS

- ACGIH (1980, 1985) recommended a Threshold Limit Value (TLV) of 50 ppm (300 mg/m^3) to prevent hepatotoxicity caused by 1,2,3-TCP, which is typical of many chlorinated hydrocarbons. ACGIH (1980, 1985) recommended a Short-term Exposure Level (STEL) of 75 ppm (450 mg/m^3) to prevent eye and mucosal irritation. ACGIH (1985) proposed changing the TLV to 10 ppm (60 mg/m^3), but no reason was given.

- The OSHA Permissible Exposure Limit (PEL) for 1,2,3-TCP is 50 ppm (300 mg/m^3) (CFR, 1985).

VII. ANALYTICAL METHODS

- Analysis of 1,2,3-TCP is by a purge-and-trap gas chromatographic procedure used for the determination of volatile organohalides in drinking water (U.S. EPA, 1985a). This method calls for the bubbling of an inert gas through the sample and trapping volatile compounds on an adsorbent material. The adsorbent material is

heated to drive off the compounds 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. Confirmatory analysis is by mass spectrometry (U.S. EPA, 1985b). The detection limit has not been determined for either method.

VIII. TREATMENT TECHNOLOGIES

- Leighton and Calo (1981) reported experimental measurements of the distribution coefficients for 21 chlorinated hydrocarbons, including 1,2,3-trichloropropane, in a dilute air-water system. (The distribution coefficient is the ratio of the volume of the compound in air to the volume of the compound in water after purging). They determined the distribution coefficients for 1,2,3-trichloropropane to be approximately 20 at 25°C.

- U.S. EPA (1986a) estimated the feasibility of removing 1,2,3-trichloropropane from water by packed column aeration, employing the engineering design procedure and cost model presented at the 1983 National ASCE Conference on Environmental Engineering. Based on chemical and physical properties and assumed operating conditions, a 90 percent removal effficiency of 1,2,3-trichloropropane was reported for a column with a diameter of 6.7 feet and packed with 16 feet of 1-inch plastic saddles. The air-to-water ratio required to achieve this degree of removal effectiveness is 40.

- No data were presented for the removal of 1,2,3-trichloropropane from drinking water by activated carbon adsorption. However, evaluation of physical/chemical properties indicates that it may be amenable to removal by activated carbon adsorption due to its low solubility.

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