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1,1,2,2--TETRACHLOROETHANE

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

1. 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.

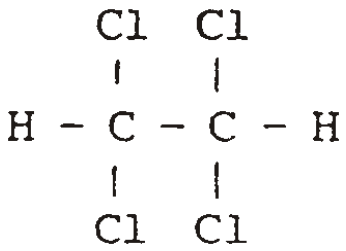
Health Advisories serve as informal technical guidance to assist Federal, State and local officials responsible for protecting public health when emergency spills or contamination situations occur. They are not to be construed as legally enforceable Federal standards. The HAS are subject to change as 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 unit risk is usually derived from a linearized multistage model with 95% upper confidence limits. This provides a low--dose estlimate of cancer risk to humas 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. 79-34-5

Structural Formula



1,1,2,2--Tetrachloroethane

Synonyms

- Acetylene tetrachloride; sym-tetrachloroethane; bonoform; cellon; 1, 1-dichloro--2, 2--dichloroethane tetrachloroethane (Chemline, 1988).

Uses

- 1,1,2,2--Tetrachloroethane is used as a solvent for chlorinated rubber and other organic materials; as a constituent in paint, varnish, lacquer and rust removers; as a metal cleanser and degreaser; as an oil and fat extractant; as an alcohol denaturant; and as a herbicide and soil fumigant. It is also used in photographic films, resins and waxes, in bleach and insecticide manufacture, in organic synthesis and as an intermediate in the manufacture of 1,1-dichloroethylene and other chlorinated hydrocarbons (Hawley, 1981; Verschueren, 1983).

Properties (Banerjee et al., 1980; Amoore and Hautala, 1983; Verschueren, 1983; ACGIH, 1986; Ruth, 1986; Riddick et al., 1986)

Chemical Formula	CHCl2CHCl2
Molecular Weight	167.86
Physical State	Liquid
Boiling Point	146.4ºC
Melting Point	-42.5/-43.8ºC
Density (20ºC)	1.59449 g/mL
Vapor Pressure (20ºC)	5 mm Hg
Specific Gravity (20/4ºC)	1.60
Water Solubility (20ºC)	2,900 mg/L
Log Octanol Water Partition Coefficient	6.64
Odor Threshold (water)	0.50 mg/L
Odor Threshold (air)	1.5 ppm 21 mg/$_{m3}$
Taste Threshold	--
Conversion Factor	1 ppm = 7 mg/m3 1 mg/m3 = 0.143 ppm

Occurrence

- Page (1981) found that 64/1, 072 ground--water and 67/608 surface water samples collected from New Jersey during 1977-1979 showed detectable levels of 1,1,2,2--tetrachloroethane (values ranged from trace to 2.7 ppb and trace to 3.0 ppb, respectively).

- 1,1,2,2--Tetrachloroethane was not detected in samples from 945 nationwide finished water supplies that used ground--water sources (Westrick et al., 1984). The detection limit was 0.5 :g/L.

- Mean atmospheric concentrations of 1,1,2,2--tetracloroethane in Los Angeles and Oakland, CA, and Phoenix, AZ, were determined to be 16.6, 7.1 and 17.0 ppt, respectively (Singh et al., 1981).

Environmental Fate

- Evaporation is likely to be the predominant fate determining process for 1,1,2,2--tetrachloroethane. Dilling (1977) determined a half--life of 55.2 minutes for evaporation from a 0.9 ppm aqueous solution (6.5 cm depth, 200 rpm stirring, 25ºC, still air).

- Significant biodegradation of 1,1,2,2--tetrachloroethane was not demonstrated in a static culture flask test with domestic wastewater inoculum (Tabak et al., 1981); degradation was 0% at 7 days and 23 to 29% at 28 days with 5 to 10 mg/L concentrations.

- Significant chemical degradation (hydrolysis, photolysis, oxidation) of 1,1,2,2--tetrachloroethane in aqueous media is not expected (U.S. EPA, 1979).

- Data regarding the extent of removal of 1,1,2,2--tetrachloroethane by sorption on particulate matter in water are inconclusive (U.S. EPA, 1979).

III. PHARMACOKINETICS

Absorption

- 1,1,2,2--Tetrachloroethane has been reported to be absorbed through intact human skin; dermal exposure has been implicated in one human fatality (ACGIH, 1986).

- Torkelson and Rowe (1981) reported that 1,1,2,2--tetrachloroethane is readily absorbed from the gastrointestinal tract and the lungs, but documentation was not provided.

- Two humans retained approximately 50% of inspired 1,1,2,2--tetrachloroethane. Details on exposure duration and dose were not specified (Lehman and Schmidt--Kehl, 1936).

- Over a 3--hour exposure period, a rabbit absorbed approximately 30% of inhaled 1,1,2,2--tetrachloroethane (Lehmann and Hasegawa, 1910).

Distribution

- Specific information regarding tissue distribution of 1,1,2,2--tetrachloroethane could not be located in the available literature. Widespread distribution is expected due to high solubility in lipids (Morgan et al., 1970).

- At 3 days after an intraperitoneal (i.p.) dose of 0.21 to 0.32 g/kg 14C--1,1,2,2--tetrachloroethane into female albino mice, a mean of 15.5% (11.2 to 19.1%) of the administered dose of radioactivity was retained in the carcass (Yllner, 1971).

Metabolism

- Yllner (1971) administered single intraperitoneal (i.p.) doses of 0.21 to 0.32 g/kg 14C--1,1,2,2--tetrachloroethane in olive oil to female albino mice and collected expired air, urine and feces for 3 days. Expired air contained about 54% of the administered dose of radioactivity; 50% as carbon dioxide, about 4% as 1,1,2,2--tetrachloroethane, and about 0.2 to 0.4% as trichloroethylene and tetrachloroethylene. Radioactivity recovered in the urine within the first 24 hours represented

about 26% of the administered dose. Expressed as a percentage of urinary radioactivity, the following metabolites were quantified: dichloroacetic acid, 27%; trichloroacetic acid, 4%; and urea, 2%. Conversions of 1,1,2,2--tetrachloroethane to glycine in female mice was demonstrated by the recovery of 14C--hippuric acid from the urine of mice treated simultaneously with 14C--1,1,2,2--tetrachloroethane and sodium benzoate. Fecal metabolites (not identified) accounted for about 0.7% of the administered dose of radioactivity. The author proposed, as the principal metabolic scheme for the degradation of 1,1,2,2--tetrachloroethane, a stepvise hydrolytic dechlorination and oxidation of dichloroacetaldehyde to dichloroacetic acid and further to glyoxylic acid and finally to carbon dioxide. Nonenzymatic dehydrochlorination to trichloroethylene followed by conwersion to trichloroacetic acid and trichloroethanol probably represents a minor pathway of degradation.

- Halpert and Neal (1981) incubated 1,1,2,2--tetrachloroethane with hepatic cytochrome P--450 preparations from phenobarbital--treated rats and recovered dichloroacetic acid as a major metabolite. They proposed that cytochrome P--450 dependent hydrolysis of 1,1,2,2--tetrachloroethane occurs followed by spontaneous dechlorination to form dichloroacetyl chloride. The authors postulated that dichloroacetyl chloride is as likely to be an intermediate in the degradation of 1,1,2,2--tetrachloroethane to dichloroacetic acid as is dichloroacetaldehyde, as suggested by Yllner (1971).

- Ikeda and Ohtsuji (1972) determined that trichloroacetic acid and trichloroethanol were important urinary metabolites in male and female Wistar rats exposed to 1,1,2,2--tetrachloroethane through inhalation or i.p. injection. The urine was tested only for trichloro metabolites and expired air was not collected.

Excretion

- After a single i.p. dose of 14C--labeled 1,1,2,2--tetrachloroethane (0.21 to 0.32 g/kg), about 50% of the radioactivity was eliminated as CO2 in the expired air and 15.5% was retained in the carcass by female albino mice in 72 hours (Yllner, 1971). Within 24 hours, about 28% of the administered radioactivity was excreted in the urine and <1% in the feces.

- Mitoma et al. (1985) administered a single oral dose of 14C--labeled 1,1,2,2--tetrachloroethane in corn oil to male Osborn--Mendal rats (100 mg/kg) and B6C3F1 mice (200 mg/kg) following 4 weeks of oral administration of unlabeled compound. Expired air and excreta collected for 48 hours after administration of the 14C--labeled compound, were analyzed. As a percentage of the radioactive dose, 7.03% (rat) and 9.6% (mouse) was expired unchanged, 1.98% (rat) and 10.14% (Irouse) was expired as CO2' 46.01% (rat) and 30.29% (mouse) was recovered from excreta, and 30.75% (rat) and 27.44% (mouse) was recovered from carcasses.

IV. HEALTH EFFECTS

Humans

Short--term Exposure

- Two male volunteers were exposed to 1,1,2,2--tetrachloroethane in a chamber at concentrations of 20, 30 or 90 mg/m3 for 10 minutes; 800 mg/m3 for 20 minutes; 900 mg/m3 for 10 minutes; 1,000 mg/m3 for 30 minutes; 1,800 mg/m3 for 10 minutes; and 2,300 mg/m3 for 10 minutes (Lehmann and Schmidt--Kehl, 1936). After 10 minutes 1,1,2,2--tetrachloroethane odor was detectable at 20 mg/m3, but there were no complaints. After 20 minutes, dizziness, mild vomiting and mucosal irritation were associated with >800 mg/m3. No details were given on whether doses were continuous or if there were recovery periods.

- Prolonged exposure to tetrachloroethane fumes (isomer not specified) produced weakness and nausea associated with acute hepatic damage (Norman et al., 1981). Exposure levels and duration were not reported.

Long--term Exposure

- Norman et al. (1981) retrospectively compared mortality records of men exposed to tetrachloroethane (isomer not specified), in chemical processing plants in which clothing was impregnated for protection against mustard gas exposure during World War II, to those not exposed to tetrachloroethane (i.e., working in chemical processing plants using a water--based solvent instead of tetrachloroethane). Exposure duration ranged from 5 weeks to 1 year. Exposure levels were not quantitated. Although not statistically significant, a slight increase in relative risk (RR) of death due to genital (RR = 4.56) and lymphatic (RR = 5.19) cancers and leukemias (RR = 1.77) was noted in 1,099 tetrachloroethane--exposed workers, compared with 1,319 unexposed workers in the same companies. Overall cancer mortality for tetrachloroethane--exposed workers was 1.26 times that of unexposed workers. Interpretation of these results is difficult, however, since workers were also exposed to the impregnite, N--dichloro--hexachloro--diphenyl--urea and dry--cleaning solvents.

- Jeney et al. (1957) studied a group of about 50 penicillin plant workers who used 1,1,2,2--tetrachloroethane as an extractant for 3 years. Approximately half of the workers developed hepatitis (diagnosed by palpation and liver function tests) during the first year when air concentrations of 1,1,2,2--tetrachloroethane ranged from 2.3 to 247 ppm (16 to 1,700 mg/m3) for most of the workshift, with peaks to 36.4 ppm (250 mg/m3) during cleaning operations; but liver dysfunction still occurred, although at lower frequency and severity (liver enlargement in 5%, urobilinogenuria in 12%, increased serum bilirubin in 7.6%). Exposure--related neurological or hematological alterations were not reported, a control group was not used and the occurrence of dermal contact with liquid solvent is not known (NIOSH, 1976; ACGIH, 1986).

- Lobo--Mendonca (1963) studied 380 workers who were exposed to 1,1,2,2--tetrachloroethane during the manufacture of bracelets from waste cellulose acetate film at 23 factories in Bombay, India. Eighty--five of the workers had dermal contact with a 1:1 1,1,2,2--tetrachloroethane: acetone liquid, 107 had dermal contact with undiluted 1,1,2,2--tetrachloroethane and 188 were exposed only to general atnospheric vapor. Average breathing zone concentrations of 1,1,2,2--tetrachloroethane ranged from 9 to 98 ppm (60 to

670 mg/m3), with most samples between 20 and 65 ppm (140 to 450 mg/m3) (NIOSH, 1976; ACGIH, 1986). There was a high incidence of nervous complaints, with fine tremors of the fingers the predominant finding (35% of the exposed workers). The incidence of these tremors appeared to be dose-related, but controls were not evaluated and the air concentration may not have been representative of actual exposures.

Animals

Short-term Exposure

- A group of 10 male albino rats was given single gavage doses of 100 mg/kg 1,1,2,2-tetrachloroethane in peanut oil (Schmidt et al. 1980). Increased levels of certain liver enzymes, with no changes in liver or body weights, were observed. No other parameters were mentioned.

- Fatty livers occurred in 18 female Cb mice that were exposed to 600 ppm (4,100 mg/m3) 1,1,2,2-tetrachloroethane vapor for 3 hours (Tomokuni, 1969); 8 hours after termination of exposure, liver lipid and triglyceride contents were 216 and 518% of control values, respectively.

- Mult male Wistar rats (6/group) exposed to 10 or 100 ppm (70 or 700 mg/m3) 1,1,2,2-tetrachloroethane for 6 hours showed dose-related increased serum glutamic oxalacetic transaminase (SGOT) levels after 24 to 72 hours when compared with controls (Deguchi, 1972). SGOT levels in rats that were exposed to 10 ppm 1,1,2,2-tetrachloroethane were measured 24, 48, 72, 96 and 120 hours following exposure. Compared with an average value for controls of 110 units, SGOT levels were 144, 214, 245, 160 and 140 units, respectively, following exposure. There were no trends found in serum glutamic pyruvic transaminase (SGPT) levels. Necropsies conducted 24 and 120 hours after exposure showed no gross or histological alterations in the liver, kidney, brain, heart, spleen or bone marrow. Four of 6 rats exposed to 1,000 ppm (7,000 mg/m3) died within 18 hours following exposure.

- Groups of 21 male rats were exposed to 0 or 13.3 mg/m3 1,1,2,2-tetrachloroethane vapor 4 hours/day for 8/10 days (Schmidt et al., 1972). Measurements performed after the second, fourth and eighth exposure indicated no significant differences between the groups in body weight, white blood cell counts, SGOT and SGPT activities, and total fat content of liver. Significant fluctuations in levels of serum proteins and pituitary adrenocorticotrophic hormone (ACTH) content were noted, but these results were inconclusive because no consistent pattern or relationship could be established.

- Groups of 10 male albino rats were given 1,1,2,2-tetrachloroethane by gavage in peanut oil at doses of 0, 8, 20 or 50-mg/kg/day for up to 45 days (Gohlke et al. 1977). Statistically significant ($p \leq 0.05$) enzyme level changes and persistent degeneration of the kidneys, testicles, liver and thyroid gland were observed in some animals at all doses. Specific dose-related details were not provided. A LOAEL of 8 mg/kg/day has been identified from this study.

- Groups of 5 male and female Osborne-Mendel rats were administered 1,1,2,2-tetrachloroethane in corn oil by gavage at dosages of 0, 56, 100, 178, 316 or 562 mg/kg/day, 5 days/week for 6 weeks followed by 2 weeks of observation (NCI, 1978). Dose-related retardation in body weight gain occurred in rats of both sexes at 56, 100 and 178 mg/kg/day (effect at higher dosages not indicated); one male rat at 100 mg/kg/day and all five females at 316 mg/kg/day died. Groups of five male and five famale B6C3F1 mice were similarly exposed at dosages of 0, 32, 56, 100, 178 or 316 mg/kg/day with no effect on body weight gain or survival. No other results or indices of toxicity were mentioned.

Dermal/Ocular Effects

- Smyth (1956) reported an acute dermal LD50 of 6.4 g/kg in rabbits.

- No data were found in the available literature on the ocular effects of 1,1,2,2-tetrachloroethane exposure.

Long-term Exposure

- Truffert et al. (1977) reported that rats exposed to 560 mL/m3 (3,840 mg/m3) for “several” hours/day, 5 days/week for 15 weeks had increased incorporation of 3H-thymidine into liver DNA by the fourth exposure followed by a return to control levels. Unspecified histological alterations in the liver occurred after nine exposures. Lesions were not observed in other organs.

- Brown Norway and Wistar rats exposed to 516 ppm (3,540 mg/m3) 1,1,2,2-tetrachloroethane for 5 hours/day, 5 days/week for 13 weeks had small glomerular lesions (detectable by electron microscopy), lower proteinuria and depressed body weights compared with controls (Danan et al., 1983).

- Over 120 days, groups of 10 male albino rats were given 82 gavage doses of 0, 3.2 or 8 mg/kg of 1,1,2,2-tetrachloroethane in peanut oil (Gohlke et al., 1977). Observations made at 45, 120 and 150 days showed changes in enzyme levels and persistent degeneration of kidneys, testicles, liver and thyroid gland. Although no specific dose-related details were provided, the authors stated that these effects occurred "down to the lowest dosage of 3.2 mg/kg." Therefore, a LOAEL of 3.2 mg/kg/day has been identified from this study.

- Shmuter (1977) observed decreased antibody formation in rabbits exposed to 2 mg/m3 1,1,2,2-tetrachloroethane for 3 hours/day for 8 to 10 months.

Schmidt et al. (1972) exposed 105 male rats (per dose group) daily to 0 or 13.3 mg/m3 1,1,2,2-tetrachloroethane for 4 hours/day for up to 265 days. Seven rats/group were killed and examined after 110 and 265 days of exposure, and an unspecified number were examined 60 days after the end of exposure. Statistically significant treatment-related effects included decreased body weight (only at 110 days), increased white blood cell counts (110 days, no data given for 265 days), increased hepatic fat content (265 days) and increased pituitary ACTH content (110 days, 265 days and 60 days postexposure).

- Rabbits (number not specified) were exposed to 2, 10 or 100 mg/m3 (0.3, 1.46 or 14.6 ppm) of 1,1,2,2-tetrachloroethane for 3 to 4 hours "daily" for 7 to 11 months (Navrotskii et al., 1971). Controls were not specifically mentioned. Effects were not observed at 0.3 ppm but suppression of hemaglutin production and fluctuations in blood acetylcholine levels and cholinesterase activity occurred at 1.46 and 14.6 ppn. Moderate urobilinogenuria, decreased blood hemoglobin and erythrocyte counts and unspecified signs of incipient liver and kidney degeneration were also associated with the 14.6-ppm exposure.

- Groups of 50 male Osborne-Mendel rats were administered time-weighted average (TWA) doses of 1,1,2,2-tetrachloroethane of 62 or 108 mg/kg/day and groups of 50 females were administered TWA doses of 43 or 76 mg/kg/day by gavage in corn oil for 78 weeks (low-dose groups) or 77 weeks (high-dose groups) followed by 32 weeks of observation (NCI, 1978). Vehicle controls consisted of 200 rats/sex and an additional 20 rats/sex were included in the study 5 months after the start as untreated controls. Treatment-related effects included dose-related decreased body weight in both sexes; dose-related increased mortality in the females; increased mortality from approximately weeks 16 to 90 in the high-dose males (survival was similar among treated and control male groups at the end of the study); and overt signs of toxicity such as hunched appearance in females, squinted/reddened eyes and abdominal urine stains in both sexes, duration-related respiratory difficulties in both sexes and dose-related chronic murine pneumonia in females. Treatment-related non-neoplastic histopathological lesions were not observed. A LOAEL of 43 mg/kg/day has been identified from this study.

- Groups of 50 B6C3F1 mice/sex were administered TWA doses of 1,1,2,2-tetrachloroethane of 142 or 284 mg/kg/day by gavage in corn oil 5 days/week for 78 weeks followed by 12 weeks of observation (NCI, 1978). Vehicle and untreated control groups consisted of 20 mice/sex. Body weight gains were not significantly affected by treatment. Dose-related increased mortality occurred in both sexes. Acute toxic tubular necrosis was the apparent cause of death in the high-dose males but most also had hepatocellular carcinomas. Other non-neoplastic histopathological effects were not associated with treatment. A LOAEL of 142 mg/kg/day has been identified from this study.

<u>Reproductive Effects</u>

- Groups of seven male rats were exposed to 0 or 13.3 mg/m3 of 1,1,2,2-tetrachloroethane "daily" for 4 hours/day for 258 days, and were mated with unexposed females (Schmidt et al., 1972);

exposure was continued for 1 week through the mating period. There were no treatment-related effects on percentage of females bearing litters, litter size, sex ratio or offspring weight. Observation of the offspring for 12 weeks showed no effect on growth rate or survival.

Developmental Effects

- Single or several daily i.p. injections of 1,1,2,2-tetrachloroethane into groups of about 25 AB-Jena or DBA mice at doses of 300, 400 or 700 mg/kg/injection during organogenesis were embryolethal and produced a low incidence of developmental anomalies (exencephaly, cleft palate, anopthalmia and fused ribs and vertebrae) (Schmidt, 1976).

- Grossly observable teratogenic or fetotoxic effects were not reported in an inhalation reproductive study where male rats were exposed for about 9 months and then mated to unexposed female rats (Schmidt et al., 1972).

Mutagenicity

- 1,1,2,2-Tetrachloroethane was multagenic to Salmonella typhimurium TA1530 and TA1535, but not TA1538, when assayed in a spot test (chemical applied to filter discs) with unspecified metabolic activation (Brem et al., 1974; Rosenkranz, 1977). 1,1,2,2-Tetrachloroethane preferentially inhibited the growth of INA polymerase-deficient (pol A-) Escherichia coli (Bran et al., 1974; Rosenkranz, 1977).

- 1,1,2,2-Tetrachloroethane induced mitotic gene conversion, recombination and mutations in Saccharomyces cerevisiae D7 when incubated in suspension without an exogenous metabolic activation system (Callen et al., 1980).

- 1,1,2,2-Tetrachloroethane induced sister chromatid exchanges, but no chromoscmal aberrations in Chinese hamster ovary cells with and without metabolic activation (Galloway et al., 1987).

- 1,1,2,2-Tetrachloroethane was nonmutagenic to Salmonella typhurmurium TA1535, TA100, TA1537, TA1538 and TA98 with and without metabolic activation (Nestmann et al., 1980) and to Drosophila without metabolic activation (Woodruff et al., 1985).

- Without metabolic activation, 1,1,2,2-tetrachloroethane did not induce transformation of BALB/c-3T3 mouse cells (Tu et. al., 1985) or cause injury to ELD ascite tumor cells (Holmberg-and Malfors, 1974).

Carcinogenicity

- Groups of 50 male and 50 female B6C3F1 mice were administered TWA doses of 142 or 284 mg/kg/day 1,1,2,2-tetrachloroethane by gavage in corn oil for 78 weeks followed by 12 weeks of observation (NCI, 1978). Vehicle and untreated control groups consisted of 20 mice/sex. Statistically significant increased incidences of hepatocellular carcinoma were observed in both sexes at both dose levels as compared with the vehicle-treated, pooled untreated or historical control group.

- Statistically significant evidence of carcinogenicity was not observed in groups of Osborne-Mendel rats that received TWA doses of 62 or 108 mg/kg/day (50 males/dose) and 43 or 76 mg/kg/day (50 females/dose) in corn oil by gavage for 77 to 78 weeks followed by 32 weeks of observation (NCI, 1978). Vehicle controls consisted of 20 rats/sex and an additional 20 rats/sex were included in the study 5 months after the start as untreated controls.

- 1,1,2,2-Tetrachloroethane was positive in the rat liver foci promotion assay (Story et al., 1986).

- Pulmonary tumors were not found in Strain A mice following i.p. administration of doses of 80, 200 and 400 mg/kg over a 2-week period (total doses of 400, 3,600 and 6,400 mg/kg, respectively) (Theiss et al., 1977).

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:

(NOAEL or LOAEL) × (BW)/ (UF) (___ L/day) = ___ mg/L (___ :g/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

The toxicological data are insufficient to derive a One-day HA. The Ten-day HA of 60 :g/L, derived below, is recommended to be used as a conservative estimate of one-day exposure.

Ten-day Health Advisory

In the Gohlke et al. (1977) study, rats given gavage doses of 8 mg/kg for 32 out of 45 days had enzyme level changes and persistent degeneration of the kidneys, testicles, liver and thyroid gland. Schmidt et al. (1972) exposed male rats (210 to 270 g) to 0 or 13.3 mg/m3 by inhalation for 4 hours/day for 8/10 days and found no treatment-related effects on body weight, white blood cell count, SGOT and SGPT activities, and total fat content in the liver, and inconclusive fluctuations in serum protein and pituitary ACIH; therefore, 13.3 mg/m3 represents a NOAEL. Assuming a rat ventilation rate of 0.18 m3/day, a body weight of 0.24 kg, and an inhalation absorption factor of 0.3, the total exposed dose (TED) can be calculated as follows:

TED = (13.3 mg/m3) (0.18 m3/day) (0.3) (4 hours/24 hours) (8 days/10 days)/(0.24 kg)

= 0.40 mg/kg/day

The Ten-day HA for a 10-kg child is calculated as follows:

Ten-day HA = (0.40 mg/kg) (10 kg)/(100) (1 L/day) = 0.04 mg/L (40 :g/L)

where:

0.40 mg/kg/day = NOAEL expressed as TED, based on the absence of adverse effects in rats exposed to 1,1,2,2-tetrachloroethane (SChmidt et al., 1972).

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 an animal study.

1 L/day = assumed daily water consumption of a 10-kg child.

Longer-term Health Advisory

The Schmuter (1977) and Navrotskii et al. (1971) studies which identified LOAELSs of 2 mg/m3 and 10 mg/m3, respectively, were not used to derive a Longer-term HA value, since details on the experimental protocol are lacking and since the significance of the reported effects is questionable. In the Gohlke et al. (1977) study, rats given gavage doses

of 3.2 mg/kg for 82 out of 120 days had enzyme level changes and persistent degeneration of the kidneys, testicles, liver and thyroid gland. In the Schmidt et al. (1972) study, rats exposed to 13.3 mg/m3 for 4 hours/day for up to 265 days had decreased body weights and increased white blood cell counts, hepatic fat content and pituitary ACTH content; therefore, 13.3 mg/m3 represents a LOAEL. Assuming a rat ventilation rate of 0.24 m3/day, a body weight of 0.35 kg, and an inhalation absorption factor of 0.3 (Lehmann and Hasegawa, 1910), the TED can be calculated as follows:

TED = (13.3 mg/m3) (0.24 m3/day) (4 hours/24 hours) (0.3)/(0.35 kg) = 0.456 mg/kg/day

Therefore, using the 0.456 TED LOAEL (Schmidt et al., 1972), the Longer-term HA for a 10-kg child is calculated as follows:

Longer-term HA = (0.456 mg/kg/day) (10 kg)/(1,000) (1 L/day) = 0.00456 mg/L [rounded to 0.005 mg/L (5 :g/L)]

Where:

0.456 mg/kg/day = LOAEL expressed as TED, based on adverse effects in rats exposed to 1,1,2,2-tetrachloroethane (Schmidt et al., 1972).

10 kg = assumed body weight of a child.

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

1 L/day = assumed daily water consumption of a 10-kg child.

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

Longer-term HA = (0.456 mg/kg/day) (70 kg)/(1,000) (2 L/day) = 0.016 mg/L [rounded to 0.02 mg/L (20 :g/L)]

where:

0.456 mg/kg/day = LOAEL expressed as TED, based on adverse effects in rats exposed to 1,1,2,2-tetrachloroethane (Schmidt et al., 1972).

70 kg = assumed body weight of an adult.

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

2 L/day = assumed daily water consumption of a 70-kg 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 for synthetic organic chemicals and a value of 10% is assumed for inorganic chemicals.

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, 1986), 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 (C) carcinogens, 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.

The lowest dosage tested (43 mg/kg/day, 5 days/week) in the only chronic oral study (NCI, 1978) is a Frank Effect Level (overt signs of morbidity and excess mortality in female rats); therefore, this study was not used for calculating HAs. In the Gohlke et al. (1977) study, rats given gavage doses of 3.2 mg/kg for 82 out of 120 days had enzyme level changes and persistent degeneration of the kidneys, testicles, liver and thyroid gland. In the Schmidt et al. (1972) study, rats exposed to 13.3 mg/m3 for 4 hours/day for up to 265 days had decreased body weights and increased white blood cell counts, hepatic fat content and pituitary ACTH content; therefore, 13.3 mg/m3 (TED = 0.456 mg/kg/day) represents a LOAEL. Use of inhalation data to calculate an oral Lifetime HA for 1,1,2,2-tetrachloroethane is justifiable because the available information indicates that similar effects (primarily on the liver) are produced by both routes of exposure. A provisional DWEL for noncarcinogenic effects could be obtained from the 265-day rat inhalation study (Schmidt et al., 1972).

Step 1: Determination of the Reference Dose (RfD)

RfD = (0.456 mg/kg/day)/(10,000) = 0.0000456 mg/kg/day [rounded to 0.00005 mg/kg/day (0.05 :g/kg/day)]

where:

0.456 mg/kg/day = LOAEL expressed as TED, based on adverse effects in rats exposed to 1,1,2,2-tetrachloroethane (Schmidt et al., 1972).

10,000 = uncertainty factor, chosen in accordance with EPA guidelines for use with a LOAEL from an animal study of less-than-lifetime duration.

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

DWEL = (0.0000456 mg/kg/day) (70 kg)/(2 L/day) = 0.001596 mg/L [rounded to 0.002 mg/L (2 :g/L)]

where:

0.0000456 mg/kg/day = RfD.

70 kg = assumed body weight of an adult.

2 L/day = assumed daily water consumption of a 70-kg adult.

Step 3: Determination of the Lifetime Health Advisory

Lifetime HA = (0.001596 mg/L) (20%)/(1) = 0.0003192 mg/L [rounded to 0.0003 mg/L (0.3 :g/L)]

where:

0.001596 mg/L = DWEL.

20% = assumed relative source contribution from water.

1 = additional uncertainty factor per ODW policy to account for possible carcinogenicity. For this chemical, the quantitative cancer risk assessment indicates that no additional uncertainty factor is necessary to account for possible cancer risk.

Evaluation of Carcinogenic Potential

- The U.S. EPA (1980) derived an upper limit for carcinogenic potency factor (q1*) of 0.2 (mg/kg/day)-1 using the incidences of hepatocellular carcinoma in female mice from the NCI (1978) bioassay.

- IARC (1979) has not evaluated the carcinogenic potential of 1,1,2,2-tetrachloroethane, but regards that limited evidence exists for its carcinogenicity.

- Applying the criteria described in the U.S. EPA's Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1986), 1,1,2,2-tetrachloro-ethane has been classified in Group C: possible human carcinogen. This category is for agents with limited evidence of carcinogenicity in animals in the absence of human data.

VI. OTHER CRITERIA, GUIDANCE AND STANDARDS

- The ACGIH (1986) TLV and NIOSH (1976) criterion for occupational exposure to 1,1,2,2-tetrachloroethane is 1 ppm (7 mg/m3) (TWA). OSHA has promulgated a workplace standard of 5 ppm (35 mg/m3) for 1,1,2,2-tetrachloroethane (Code of Federal Regulation, 1985). Both OSHA and the ACGIH have designated that dermal exposure may significantly contribute to total exposure.

- The U.S. EPA (1980) recommended a criterion of 1.7 :g/L for ambient water. This value is based on the NCI (1978) cancer data as indicated in Section V.

VII. ANALYTICAL METHODS

- Analysis of 1,1,2,2-tetrachloroethane 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-**XXXX**programmmed to separate the method analytes, which are then detected by a halogen-specific detector. This method is applicable to the measurement of 1,1,2,2-tetrachloroethane over a concentration range of 0.1 to 1,500 :g/L. Confirmatory analysis is by mass spectrometry (U.S. EPA, 1985b). The detection limit for confirmation by mass spectrometry has been estimated at 0.28 and 0.41 :g/L by two different analysts.

VIII. TREATMENT TECHNOLOGIES

- Available data indicate that air stripping and granular activated carbon (GAC) adsorption will remove 1,1,2,2-tetrachloroethane from contaminated water.

- One Study investigated the efficiency of GAC for the removal of priority pollutants in a combined municipal/industrial wastewater (McManus et al., 1985). Specifically, a pilot plant program was designed to determine the rate of carbon bed exhaustion for certain pollutants over one regeneration cycle (i.e., up to 6 months). Two columns were used, each 5 inches in diameter and packed with 9.25 feet of Calgon “standard service” carbon (a mixture of Filtrasorb 300 and 400). Each column operated continuously for 6 months at a design flow rate of 0.3 gpm, which corresponds to a surface hydraulic loading of 2.2 gpm/ft2. 1,1,2,2-Tetrachloroethane was present in the plant influent at an average concentration of 22.6 mg/L. The results of this study show that 1,1,2,2-tetrachloroethane breakthrough occurred after 3,000 bed volumes (BV) or 60 days cumulative run time (breakthrough assumed at the limit of detection of 1 mg/L). Also, 50% breakthrough was achieved at the end of the 6-month test run. An observed carbon capacity of 0.35 mg 1,1,2,2-tetrachloroethane/g carbon is reported.

- One pilot plant study evaluated the performance of packed columns in removing certain VOCs discovered in ground water in Tacoma, Washington. 1,1,2,2-Tetrachloroethane concentrations ranged from 17 to 300 mg/L (Byers and Morton, 1985; U.S. EPA, 1986). A packed tower aeration system was selected as the best method for treating the water. Full-scale packed towers were designed from the data produced in pilot-plant testing. The final design consisted of 5 columns, each 12 feet in diameter and packed with 20 feet of 1-inch polypropylene saddles and operating in parallel at an air-to-water ratio of 300:1. During operation, the tower consistently removed an average 95.5% of 1,1,2,2-tetrachloroethane.

- Another study determined removal mechanisms for various priority pollutants from wastewater (Kincannon et al., 1983). Air stripping appeared to be the most effective removal method for 1,1,2,2-tetrachloroethane. A Henry’s Law constant of 23.4 atm is reported. A 94% removal efficiency by air stripping is reported from an initial concentration of 201 mg/L. No other operating parameters are reported.

- In summary, a number of techniques for the removal of 1,1,2,2-tetrachloroethane from water have been examined. While the data are not unequivocal, it appears that adsorption onto GAC is likely to be a successful treatment technique. The amenability of 1,1,2,2-tetrachloroethane to aeration has

been clearly established. Selection of individual or combinations of technologies to attempt 1,1,2,2-tetrachloroethane removal from contaminated drinking water must be based on a case-by-case technical evaluation, and an assessment of the economics involved.

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