Appendix B
Section O

TETRACHLOROETHYLENE
MAXIMUM CONTAMINANL LEVEL
SUPPORT DOCUMENT

Office of Science and Research
New Jersey Department of Environmental Protection

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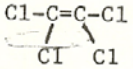
EXECUTIVE SUMMARY

Tetrachloroethylene is a moderately volatile chlorinated hydrocarbon. It i used as a solvent in the dry cleaning of fabrics and in the vapor degreasing o metals. The odor threshold for tetrachloroethylene in water is 300 ug/L Tetrachloroethylene is classified as a probable human carcinogen (EPA Group B2) Based on a mouse bioassay, a quantitative estimate of human cancer risk wa calculated for drinking water. A drinking water level of 0.4 u tetrachloroethylene per liter of water is associated with a lifetime exces cancer risk of one in a million.

TABLE OF CONTENTS

BACKGROUND INFORMATION AND PROPERTIES

Chemical Properties

Chemical Name	Tetrachloroethylene
Synonyms	1,1,2,2-Tetrachloroethylene, Perchloroethylene, PCE, PERC
CAS Number	127-18-4
Chemical formula	C_2Cl_4
Chemical structure	
Molecular weight	165.85
Physical state (room temperature)	colorless liquid
Melting point	-23.4 °C
Boiling point	121.2 °C
Vapor pressure, volatility	19 mm Hg
Specific gravity, density	1.623
Water solubility	150 mg/L at 25 °C
Log octanol/water partition coefficient	2.86
Taste threshold (water)	not available
Odor threshold (water)	300 ug/L
Odor threshold (air)	50 ppm
Conversion factors	1 ppm = 6.8 mg/m^3

Production and Use

Tetrachloroethylene is a colorless liquid used primarily as a solvent in the dry cleaning of fabrics. To a lesser extent it is used as a degreasing solvent in metal industries and as a chemical intermediate in the synthesis of other compounds. Tetrachloroethylene is produced by the oxyhydrochlorination, chlorination, or dehydrochlorination of hydrocarbons or chlorinated hydrocarbons. Production of tetrachloroethylene in 1983, was estimated at 263,000 metric tons.

Guidelines, Regulations, and Standards

An Ambient Water Quality Criteria Document for tetrachloroethylene was published by the U.S. Environmental Protection Agency (U.S.EPA, 1980). Assuming consumption of 2 L of water and 6.5 g of fish per day by a 70 kg adult, a tetrachloroethylene level of 3.5 ug/L was estimated to limit excess lifetime cancer risk to one in a million.

Health Advisory drinking water guidance for tetrachloroethylene describing non-carcinogenic toxicology has been developed by the Office of Drinking Water (U.S.EPA, 1985a). Assuming that a 10 kilogram child consumes one liter of water per day, the Ten-day and Longer-term Health Advisories are 34000 and 1940 ug/L, respectively.

The World Health Organization recommended a tentative guideline value of 10 ug tetrachloroethylene per liter of drinking water (WHO, 1984). This value was based on a 70 kg adult consuming 2 L of water per day and on an acceptable risk level of less than one additional case of cancer per 100,000 population for a lifetime of exposure.

ENVIRONMENTAL EXPOSURE

Fate and Transport

Although tetrachloroethylene is released into water via aqueous effluents from production plants, consumer industries, and household sewage, it is not frequently detected in surface water because of its high volatility. However, it is frequently found as a contaminant in groundwater. Tetrachloroethylene in water slowly decomposes to form trichloroacetic acid, hydrochloric acid, trichloroethylene, dichloroethylene and vinyl chloride.

Ambient Levels

The U.S.EPA's STORET system compiled data on tetrachloroethylene concentrations in water supplies from 66 measurement stations in 10 states for the period from August 1977 to September 1984. The mean and maximum concentrations were 2 and 20 ug/L, respectively.

Information from the Federal Reporting Data System on the United States population served by primary water supply systems was used by the U.S.EPA to estimate exposure to tetrachloroethylene through drinking water (U.S.EPA, 1984). An estimated 874,000 individuals are exposed to tetrachloroethylene in drinking water at or above 5 ug/L (Table I).

The Office of Science and Research, New Jersey Department of Environmenta Protection, surveyed public water supplies statewide for over 100 substance during the period 1978 through 1981. Tetrachloroethylene was detected in 13% o the samples at a mean concentration of 11 ug/L. Recently, A-280 listed chemical were monitored by New Jersey potable water purveyors. Tetrachloroethylene wa found in 5% of the samples. The concentration levels ranged from 0.5 to 14 ug/L.

Table I

Estimated Drinking Water Intake of Tetrachloroethylene

Exposure Level (ug/l)	Population	Intake* (ug/kg/day)
≥0.5	11,430,000	≥ 0.014
>5.0	874,000	> 0.14
>10.0	440,000	>0.29
>50.0	105,000	>1.4

*Intake estimates assume 70 kg adult consumes 2 L water per day.

Source: U.S.EPA, 1984.

METABOLISM AND PHARMACOKINETICS

Absorption

Tetrachloroethylene is completely absorbed from the gastrointestinal tract into the body. Pegg et al. (1979) and Schumann et al. (1980) administered 1 and 500 mg/kg (^{14}C)tetrachloroethylene in corn oil to both rats and mice. More than 90% of the radioactivity was recovered in urine, feces, and exhaled air. Frantz and Watanabe (1983) noted virtually complete absorption of (^{14}C)tetrachloroethylene provided ad libitum in drinking water to rats over a 12-hour period.

Distribution

Tetrachloroethylene is expected to distribute into all body tissues crossing both the blood-brain barrier and placental barrier. Once in the blood stream, tetrachloroethylene is retained in body fat. In humans, the ratio of fat to liver tetrachloroethylene concentrations is greater than 6:1 (McConnell et al., 1975).

Metabolism

The carcinogenic potential of tetrachloroethylene and other halogenated ethylenes depends on the metabolic conversion in the liver of these compounds to reactive epoxides and other biologically reactive intermediates (Bolt et al., 1982; U.S. EPA, 1985b). Metabolism of tetrachloroethylene after oral (mice, rats) or inhalation (mice, rats, humans) exposure is rate-limited and proceeds according to Michaelis-Menten kinetics (Pegg et al., 1979; Schumann et al., 1980; Buben and O'Flaherty, 1985). In humans, the tetrachloroethylene metabolites which has been identified include trichloroethanol and trichloroacetic acid (Ikeda, 1977; Ogata et al., 1971).

Excretion

Unchanged tetrachloroethylene is primarily eliminated from the body via the lungs (Stewart et al., 1970). The respiratory elimination half-life of tetrachloroethylene has been estimated at 65 to 70 hours (Stewart et al., 1970; Ikeda and Imamura, 1973). The mean urinary half-life of trichloroacetic acid, a metabolite of tetrachloroethylene, is 144 hours.

Human Exposure and Body Burden

Three approaches have been developed to estimate tetrachloroethylene body burden: 1) concentration of tetrachloroethylene in exhaled air, 2) blood levels of tetrachloroethylene, and 3) concentrations of trichloroacetic acid in blood and urine (U.S.EPA, 1985b).

HEALTH EFFECTS

Overview

The acute effects of tetrachloroethylene are dominated by central nervous system depression (weakness, ataxia, restlessness), cardiac and respiratory depression, and irritation of eyes and skin. Fatty infiltration of the liver and kidney have been associated with chronic tetrachloroethylene exposure. Tetrachloroethylene induced liver cancer in mice and leukemia in rats.

Human

Chronic exposure to tetrachloroethylene vapors caused irritation of the respiratory tract, nausea, headache, sleeplessness, and abdominal pains (Coler and Rossmiller, 1953; Stewart et al., 1970).

Animal

The oral LD_{50} values of tetrachloroethylene for male and female rats were 3.8 and 3.0 g/kg, respectively (Hayes et al., 1986).

Behavioral and Central Nervous System

The temporary central nervous system effects associated with tetrachloroethylene include dizziness, confusion, headache, and nausea (Coler and Rossmiller, 1953; Stewart, 1969).

Reproductive, Embryotoxic and Teratogenic

No experimental studies on endocrine, gonadal or fertility effects were found. Schwetz et al. (1975) exposed rats and mice to 300 ppm tetrachloroethylene for 7 hours per day on days 6-15 of pregnancy. The dams were killed just before term and the fetuses examined by acceptable teratological methods. Results are provided on a per litter basis only. The number of treated animals in each case was 17, and the number of controls (air-exposed) for both rat and mouse studies was 30. In the mice, there was no effect on implantation sites, live fetuses, or resorption rates, but mean fetal weight was significantly reduced. In the rats, the resorption rate was significantly increased, but fetal body weight was unaffected.

Genetic

In studies conducted by the National Toxicology Program (NTP) (Table II), tetrachloroethylene did not cause mutations in S. typhimurium strains TA98, TA100, TA1535, or TA1537 with or without metabolic activation. Tetrachloroethylene was nonmutagenic to mouse lymphoma L5178Y/TK$^{+/-}$ cells with or without activation. It also did not induce sister-chromatid exchange or chromosomal aberrations in Chinese hamster ovary cells with or without metabolic activation.

Carcinogenicity

The carcinogenic potential of tetrachloroethylene has been adequately reviewed (IARC, 1979; U.S. EPA, 1986; WHO, 1984). Tetrachloroethylene administered by the oral or inhalation route induced hepatocellular carcinoma in both sexes of B6C3F1 mice (NCI, 1977; NTP, 1985). It also induced an increase in mononuclear cell leukemia in male and female F344/N rats (NTP, 1985). Tetrachloroethylene is classified as a probable human carcinogen (EPA Group B2).

The experimental induction of cancer by tetrachloroethylene has been demonstrated in both mice and rats (NCI, 1977; NTP, 1985). In a 2-year study conducted by the NTP (1985), 8-week-old F344/N rats and 9-week-old B6C3F1 mice of both sexes were administered tetrachloroethylene by inhalation. Animals were exposed to the compound 6 hours per day, 5 days per week, for 103 weeks. Dosages (in ppm) were: 0, 100, and 200 for mice, and 0, 200, and 400 ppm for rats. Both rats and mice were randomly distributed so that there were 50 animals per sex per treatment group. Food and water were freely available.

Animals were killed when moribund or at the conclusion of the study. A complete necropsy was performed on each animal unless precluded by cannibalism or autolysis. Selected results are presented in Table III. The National Toxicology Program concluded the following:

> Under the conditions of these inhalation studies, there was some evidence of carcinogenicity of tetrachloroethylene in F344/N rats as shown by increased incidence of mononulcear cell leukemia in males and females and rare tubular cell neoplasms in males. There was clear evidence of carcinogenicity in B6C3F1 mice as shown by increased incidences of both hepatocellular adenomas and carcinomas in males and of hepatocellular carcinomas in females.

Tetrachloroethylene was administered by gavage 5 days per week to Osborne-Mendel rats and B6C3F1 mice for 78 weeks, followed by a 32 and 12 weeks observation period, respectively (NCI, 1977). There were 50 animals per sex per species in each of the treatment groups with 20 controls for each sex and species. Food and water were freely available. Rats were started on the test at the age of 7 weeks and mice at 5 weeks of age. Original doses were the maximum tolerated (MTD) and one-half the MTD (as determined in previous subchronic studies).

Time-weighted-average (TWA) doses for male rats were 471 mg/kg (500 mg/kg for 19 weeks, 700 mg/kg for 6 weeks, 500 mg/kg for 20 weeks, and 500 mg/kg on a 4-weeks-on, 1-week-off cycle for 33 weeks) and 941 mg/kg (1000 mg/kg for 19 weeks, 1400 mg/kg for 6 weeks, 1000 mg/kg for 20 weeks, and 1000 mg/kg on a

Table II

Genetic Toxicology of Tetrachloroethylene

Process	End Point	Test System	Conclusions	References
Gene mutation	Base-pair substitution, Frameshift	Ames *Salmonella* battery with and without metabolic activation.	Negative	NTP unpublished results
		Mouse lymphoma L5178Y/TK$^{+/-}$ cells with and without metabolic activation.	Negative	NTP unpublished results
Chromosomal rearrangement homologous recombination	Sister chromatid exchange	Chinese hamster ovary cells *in vitro* with and without metabolic activation.	Negative	NTP unpublished results
	Gene conversion, Mitotic recombination	*Saccharomyces cerevisiae* (yeast) with and without metabolic activation	Negative	Bronzetti et al., 1983
Chromosomal Rearrangement Non-homologous recombination	Chromosomal aberrations	Chinese hamster ovary cells *in vitro* with and without metabolic activation.	Negative	NTP unpublished results
		Human occupational study	Negative	Ikeda et al.,1980.

Table III

Incidence of Tumors in B6C3F1 Mice
and F344/N Rats Induced by Tetrachloroethylene

Dose Levels		Responses	
Experimental	Adjusted	(# animals with tumor/# animals at risk)	
ppm	mg/kg/day	Male	Female
	NTP-Mouse Hepatocellular Carcinoma		
0	0	7/49	1/45
100	282	25/49	13/43
200	565	26/50	36/49
	NTP-Rat Mononuclear Cell Leukemia		
0	0	28/50	18/50
200	103	37/50	30/50
400	206	37/50	29/50
mg/kg	mg/kg/day	Male	Female
	NCI-Mouse Hepatocellular Carcinoma		
0	0	2/20	
536	215	32/49	
1072	429	27/48	
0	0		0/20
386	155		19/48
772	309		19/48

4-weeks-on, 1-week-off cycle for 33 weeks). Female rats received TWA doses o 474 mg/kg (500 mg/kg for 19 weeks, 600 mg/kg for 3 weeks, 700 mg/kg for 6 weeks 500 mg/kg for 20 weeks, and 500 mg/kg on a 4-weeks-on, 1-week-off cycle for 3 weeks) and 949 mg/kg (1000 mg/kg for 19 weeks, 1200 mg/kg for 3 weeks, 1400 mg/k for 6 weeks, 1000 mg/kg for 20 weeks, and 1000 mg/kg on a 4-weeks-on, 1-week-of cycle for 33 weeks).

Male mice received TWA doses of 536 mg/kg (450 mg/kg for 11 weeks, and 55 mg/kg for 67 weeks) and 1072 mg/kg (900 mg/kg for 11 weeks, and 1100 mg/kg for 6 weeks). Time-weighted-average doses for female mice were 386 mg/kg (300 mg/k for 11 weeks, and 400 mg/kg for 67 weeks) and 772 mg/kg (600 mg/kg for 11 weeks and 800 mg/kg for 67 weeks).

Following dosing, rats were observed for an additional 32 weeks and mice fo 12 weeks, at which time the surviving animals were sacrificed. All animals whether found dead, killed when moribund, or sacrificed at the end of the stud were subject to a complete necropsy unless precluded by autolysis or cannibalism

Tumor incidence in treated rats showed no significant increase ($p<0.05$) ove the incidence in control rats. Compared to controls, treated mice, however, di show a significantly increased ($p<0.05$) incidence of hepatocellular carcinoma The incidence of hepatocellular carcinoma in male control mice was 2/20 with th first tumor appearing after 90 weeks. Low-dose male mice had a hepatocellula carcinoma incidence of 32/49 (27 weeks), while the incidence in high-dose male was 27/48 (40 weeks). The incidence of hepatocellular carcinoma in female mic was 10/48 (50 weeks) in the high-dose group, 19/48 (41 weeks) in the low-dos group, and 0/20 in the controls.

QUANTITATIVE RISK ASSESSMENT

Studies Useful for Risk Assessment

Animal bioassay data (NCI, 1977; NTP, 1985) was considered for th tetrachloroethylene risk assessment. The rationale for the selection of animal laboratory studies for quantitative risk assessment is based on the guideline set forth in Crump and Howe (1980). The experimental study design judged to b most suitable involved tetrachloroethylene treatment of animals for at least 85% of their expected lifespan and observation of animals for at least 90% of thei lifespan. The highest dose level should maximize malignant response without causing overt chronic toxicology. The oral exposure route was considered most relevant for setting drinking water criteria.

In the National Cancer Institute (NCI) study, rats were treated for 60% and mice for 75% of their expected lifespan, while both rats and mice were observed for 85% of their expected lifespan. In the NTP study, rats were observed and treated for only 80% of their expected lifespan, while mice were observed and treated for 100% of their expected lifespan. In the NTP study, animals were exposed to tetrachloroethylene by inhalation, while in the NCI study doses were administered by gavage.

In both the NCI and NTP studies, mice showed a greater increase in tumor incidence than did rats. The male mice in the NTP and NCI studies were more sensitive to tetrachloroethylene than the females.

For these reasons, male B6C3F1 mice with hepatocellular carcinoma from the NCI (1977) study were used for the tetrachloroethylene risk assessment.

Calculation of the Health-Based Maximum Contaminant Level

The dose-response relationship obtained from the NCI (1977) study was modeled by using regression techniques. The multistage model was used in this analysis for low dose extrapolation with quantal data. The multistage model is given by:

$$P(d) = 1 - \exp(-q_o - q_1 d - \ldots - q_k d^k), \quad q_i \geq 0, \; i = 0, 1, \ldots k,$$

where P (d) is the lifetime probability of cancer at dose d and k is set to the number of dose groups less one.

The multistage model was implemented using an updated version of the computer program GLOBAL82. All calculations were provided by K.S. Crump and Company (Crump, 1986).

Risk has been defined as "extra risk", i.e.,

$$[P(d) - P(0)]/[1 - P(0)],$$

where P(d) is the lifetime probability of dying of liver cancer when exposed to tetrachloroethylene dose d and P(0) is the lifetime probability of dying of liver cancer when not exposed to tetrachloroethylene.

A risk level of one in a million was selected for a lifetime exposure scenario. The 95% upper confidence limit on risk is linear at low doses and will be considered a plausible upper bound on risk.

Animal-to-human extrapolation was based on the assumption that both animals and humans are equally susceptible (in terms of extra risk) to the carcinogen when dose is measured in the same units for both species (Crump and Howe, 1980). The unit of mg/m^2 body surface area per day will be used for animal-to-human extrapolation. If D_A represents animal dose in mg/kg per day, then the human dose (D_H) is given by

$$D_H = D_A (W_A/W_H)^{1/3},$$

where W_A and W_H are the weights of animals and humans, respectively. The standard exposure assumption is that a 70 kg adult consumes 2 liters of water per day.

Experimental dose levels are adjusted from mg/kg to mg/kg per day by a factor of 5/7 to account for tetrachloroethylene administration 5 days per week. A factor of $(90/104)^4 = 0.5608$ is further applied for the NCI study because the experiment was terminated before the animals lived out their full lifespan.

The multistage model fitted to the NCI (1977) male mice hepatocellular carcinoma data set with the high dose group deleted provided an animal dose of 1.62×10^{-4} mg/kg per day. Therefore, the human dose (in micrograms per liter) is calculated as follows:

$$\begin{aligned} &= \text{human dose (mg/kg/day)} \times (1000 \text{ ug/mg}) \times (70 \text{ kg})/(2 \text{ L/day}) \\ &= \text{animal dose (mg/kg/day)} \times (W_A/W_H)^{1/3} \times 35000 \\ &= \text{animal dose (mg/kg/day)} \times (0.03 \text{ kg}/70 \text{ kg})^{1/3} \times 35000 \end{aligned}$$

= animal dose (mg/kg/day) x 35000/13
= (1.62×10^{-4}) x 35000/13
= 0.44 ug/L.

A drinking water level of 0.4 ug tetrachloroethylene per liter is associated wit a lifetime excess cancer risk of one in a million.

Assumptions and Uncertainty

The extrapolation of liver cancer risk from bioassay data to human live cancer risk was carried out by assuming that animals and humans were equall sensitive to a particular measure of dose. The interspecies conversion facto applied was mg/m^2 surface area per day. This is equivalent to (mous weight/human weight)$^{1/3}$. Mouse and human weights used are 0.03 and 70 kg respectively.

A 70 kg adult was assumed to consume 2 L of drinking water per day for life The 95% upper confidence level on risk was considered a plausible upper bound o risk. The risk level applied was one in a million excess cancer risk.

Conclusions

Tetrachloroethylene was classified as a probable human carcinogen (EPA Grou B2). It induced liver neoplasms in mice and leukemia in rats. The quantitativ estimation of liver cancer hazard was based on the NCI (1977) mouse bioassay. drinking water level of 0.4 ug tetrachloroethylene per liter of water was associ- ated with a lifetime excess cancer risk of one in a million.

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