1,1,2-TRICHLOROETHANE

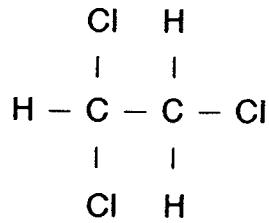
This substance was considered by a previous Working Group, in 1978 (IARC, 1979). Since that time, new data have become available, and these have been incorporated into the monograph and taken into consideration in the evaluation.

1. Chemical and Physical Data

1.1 Synonyms

Chem. Abstr. Services Reg. No.: 79-00-5
Chem. Abstr. Name: 1,1,2-Trichloroethane
IUPAC Systematic Name: 1,1,2-Trichloroethane
Synonyms: Ethane trichloride; trichloroethane; *beta*-trichloroethane; 1,2,2-trichloroethane; vinyl trichloride

1.2 Structural and molecular formulae and molecular weight



$C_2H_3Cl_3$ Mol. wt: 133.41

1.3 Chemical and physical properties of the pure substance

(*a*) *Description*: Clear, colourless liquid with a sweet odour (Verschueren, 1983; Sax & Lewis, 1987; Budavari, 1989)
(*b*) *Boiling-point*: 113.8°C (Weast, 1989)
(*c*) *Melting-point*: -36.5°C (Weast, 1989)
(*d*) *Density*: 1.4397 at 20/4°C (Weast, 1989)

(*e*) *Spectroscopy data*[1]: Infrared (Sadtler Research Laboratories, 1980, prism [9721], grating [29465]; Pouchert, 1981, 1985a,b), nuclear magnetic resonance (Sadtler Research Laboratories, 1980, proton [16882, V2], C-13 [344]; Pouchert, 1974, 1983) and mass spectral data [617] have been reported.

(*f*) *Solubility*: Slightly soluble in water (4.50 g/l at 20°C); soluble in ethanol, chloroform and diethyl ether (Verschueren, 1983; Weast, 1989)

(*g*) *Volatility*: Vapour pressure, 19 mm Hg at 20°C, 40 mm Hg at 35°C; relative vapour density (air = 1), 4.63 (Verschueren, 1983)

(*h*) *Reactivity*: Reacts with strong oxidizers, strong caustics and metals such as sodium, potassium, powdered aluminium and magnesium (Sittig, 1985)

(*i*) *Octanol/water partition coefficient (P)*: log P, 2.07 (Chemical Information Systems, Inc., 1990)

(*j*) *Conversion factor*[2]: $mg/m^3 = 5.46 \times ppm$

1.4 Technical products and impurities

1,1,2-Trichloroethane is available in the USA as a commercial-grade product, either stabilized or unstabilized, at a purity of > 99%. The stabilized product contains *sec*-butanol (0.5%) and 1,2-butylene oxide (0.25%) (Dow Chemical Co., 1990). 1,1,2-Trichloroethane is also available in research quantities at 95-99.9% purity for calibration of liquid density meters and as a gas chromatography standard (Riedel-de Haën, 1986; Eastman Kodak Co., 1987; American Tokyo Kasei, 1988; Pfaltz & Bauer, 1988; Aldrich Chemical Co., 1990).

2. Production, Use, Occurrence and Analysis

2.1 Production and use

(*a*) *Production*

1,1,2-Trichloroethane is produced in the USA by the chlorination of ethylene (see IARC, 1987a). In a two-stage manufacturing process, this initially yields 1,2-dichloroethane (see IARC, 1987b); subsequent chlorination yields

---

[1]In square brackets, spectrum number in compilation

[2]Calculated from: mg/m^3 = (molecular weight/24.45) × ppm, assuming standard temperature (25°C) and pressure (760 mm Hg)

1,1,2-trichloroethane and hydrochloric acid. In an alternative production method, ethylene is combined with hydrochloric acid and oxygen at 280-370 °C on a catalyst of cupric chloride and potassium chloride to yield 1,1,2-trichloroethane and other chlorinated ethanes (Reed *et al.*, 1988). 1,1,2-Trichloroethane has also been made by the chlorination of vinyl chloride (see IARC, 1987c) in a liquid phase at 300-320 °C (Thomas *et al.*, 1982).

There is only one producer in the USA, with an annual production estimated to be 186 000 tonnes (Reed *et al.*, 1988). 1,1,2-Trichloroethane is also produced by one company each in France and the UK (Chemical Information Services Ltd, 1988).

(*b*) *Use*

Over 95% of the 1,1,2-trichloroethane produced in the USA is used as an intermediate in the production of vinylidene chloride (see IARC, 1987d). Vinylidene chloride is made by dehydrochlorination of 1,1,2-trichloroethane with an aqueous suspension of calcium hydroxide at about 320 °C or with lime or sodium hydroxide (Wiseman, 1972; Reed *et al.*, 1988). 1,1,2-Trichloroethane has two minor uses: as a solvent for the coating laid down on films and as a chemical intermediate or process solvent in pharmaceutical manufacture. It has been used as a solvent for fats, oils, waxes and resins and, in small amounts, as a solvent for chlorinated rubber and adhesives (US Environmental Protection Agency, 1980; Sax & Lewis, 1987; Strobel & Grummt, 1987).

(*c*) *Regulatory status and guidelines*

Occupational exposure limits and guidelines for 1,1,2-trichloroethane are presented in Table 1.

Table 1. Occupational exposure limits and guidelines for 1,1,2-trichloroethane[a]

Country	Year	Concentration (mg/m^3)	Interpretation[b]
Australia	1985	45 (skin)[c]	TWA
		90	STEL
Austria	1987	55 (skin)[d]	TWA
Belgium	1989	45 (skin)	TWA
Brazil	1982	35	TWA
Canada	1986	45 (skin)	TWA
Chile	1987	36 (skin)	TWA
Denmark	1987	45 (skin)	TWA
Finland	1987	45	TWA
		90 (skin)	STEL

Table 1 (contd)

Country	Year	Concentration (mg/m^3)	Interpretation[b]
Germany	1988	55[d]	TWA
		110[d]	STEL (30 min)
Hungary	1985	10	TWA
		50	STEL
Indonesia	1987	45 (skin)	TWA
Japan	1988	45 (skin)	TWA
Mexico	1987	45 (skin)	TWA
Netherlands	1986	45 (skin)	TWA
Poland	1985	100	TWA
Switzerland	1987	55 (skin)	TWA
UK	1987	45 (skin)	TWA
		90 (skin)	STEL (10 min)
USA			
ACGIH	1989	55 (skin)	TWA
OSHA	1989	45 (skin)	TWA
Venezuela	1987	35 (skin)	TWA
		90	STEL

[a]From Cook (1987); Health and Safety Executive (1987); American Conference of Governmental Industrial Hygienists (ACGIH) (1989); US Occupational Safety and Health Administration (OSHA) (1989); United Nations Environment Programme (1990)

[b]TWA, time-weighted average; STEL, short-term exposure limit

[c]With a notation of skin absorption

[d]Suspected of carcinogenic potential

2.2 Occurrence

(*a*) *Natural occurrence*

1,1,2-Trichloroethane is not known to occur as a natural product.

(*b*) *Occupational exposure*

The US National Occupational Hazard Survey estimated that in 1972-74 112 000 workers were potentially exposed to 1,1,2-trichloroethane (National Institute for Occupational Safety and Health, 1978)

(*c*) *Multimedia exposure assessment*

Nine volunteers in New Jersey (four chemical and oil company workers) and three in North Carolina (none considered to be occupationally exposed) were

monitored for exposure to 1,1,2-trichloroethane between July and December 1980. Breathing-zone air, drinking-water, food and breath were monitored. 1,1,2-Trichloroethane was detected in 10 (seven at trace) of the 161 breathing-zone air samples in New Jersey at 0.14-34.70 μg/m^3 and in one of the 60 samples in North Carolina at about 0.54 μg/m^3. It was detected in five (four at trace) exhaled breath samples in New Jersey at 0.07-5.13 μg/m^3, with a median concentration of 0.2 μg/m^3, and in none of the 17 exhaled breath samples in North Carolina (limit of detection, 0.20 μg/m^3). It was not detected in any of the 13 home drinking-water samples for the volunteers in New Jersey (limit of detection, 0.01 μg/l) [no information was provided on home samples for the volunteers in North Carolina] (Wallace *et al.*, 1984).

(*d*) *Air*

According to the Toxic Chemical Release Inventory, total emissions of 1,1,2-trichloroethane into the air in the USA in 1987 were approximately 935 tonnes from 49 locations. Industrial releases to other media were 5.5 tonnes to ambient water from 12 locations and 4 kg to the land from two locations (National Library of Medicine, 1989).

Estimated levels of 1,1,2-trichloroethane in ambient air collected in 1977 from four sites in Iberville Parish, LA, USA, ranged from trace to 1.8 μg/m^3 (Pellizzari, 1982).

1,1,2-Trichloroethane was detected in one of ten air samples collected in 1978 at Bochum-University, Germany, at a concentration of 0.6 μg/m^3, in three of 12 air samples from Bochum-Kemnade at 0.3-0.8 μg/m^3, and in three of 12 air samples from Bochum City at 0.8-17 μg/m^3; the mean concentration of 1,1,2-trichloroethane in air samples from Bochum and vicinity was 0.4 μg/m^3 (Bauer, 1981).

In a review of data on the presence of volatile organic chemicals in the US atmosphere in 1970-80, a median concentration of 0.082 μg/m^3 1,1,2-trichloroethane was reported for the 760 data points examined, 0.076 μg/m^3 (667 data points) for urban/suburban areas, and 0.5 μg/m^3 (91 data points) close to industrial sources (Brodzinsky & Singh, 1983).

Air samples collected in 10 US cities in 1979-81 contained mean concentrations of 0.033-0.022 μg/m^3 1,1,2-trichloroethane (Singh *et al.*, 1983).

The mean concentration of 1,1,2-trichloroethane in indoor winter air samples collected in 1982 from 11 homes in Knoxville, TN, USA, was 14.1 μg/m^3; the compound was not detected (< 1 μg/m^3) in any outdoor air sample (Gupta *et al.*, 1984).

(e) Water and sediments

Of 10 water supplies surveyed by the US Environmental Protection Agency (1980), only one contained 1,1,2-trichloroethane; a study of finished water from a metropolitan area reported concentrations of 0.1-8.5 µg/l. The highest concentrations of 1,1,2-trichloroethane in samples collected in 1977-79 in ground- and surface water in New Jersey, USA, were 31.1 µg/l and 18.7 µg/l, respectively; the compound was detected in 72 of 1069 groundwater samples and in 53 of 603 surface water samples (Page, 1981).

Samples of Rhine River water collected near Lobith, Germany, between April and December 1976 contained levels of 1,1,2-trichloroethane ranging from not detected to 1.0 µg/l, with a mean of 0.2 µg/l. Levels in drinking-water samples collected from 100 cities in western Germany in 1977 ranged from not detected to 5.8 µg/l (Bauer, 1981).

1,1,2-Trichloroethane was not detected in raw water samples collected in 1979 from 30 Canadian water treatment facilities but was found in two samples of treated water at a mean concentration of < 1 µg/l (max, 7 µg/l) (Otson *et al.*, 1982).

Tap-water samples (30 each) collected in 1981 in Tübingen, Germany, from the local water supply contained an average concentration of 1,1,2-trichloroethane of 0.05 µg/l (range, 0.03-0.09 µg/l); and samples from the Lake Constance water supply (sampled in Tübingen) contained an average of 0.01 µg/l (range, 0.00-0.02 µg/l) (Hagenmaier *et al.*, 1982).

1,1,2-Trichloroethane was detected at concentrations greater than 10 µg/l in 58 of 1982 US industrial wastewater samples. The minimum and maximum concentrations found were 12 µg/l and 3400 µg/l, respectively. The industrial wastewaters in which 1,1,2-trichloroethane was identified include those from factories producing adhesives/sealants, iron/steel foundries, laundries, factories producing, mechanical products, organics/plastics and paint/ink, petroleum refineries, plants for pharmaceuticals and phosphates, printing/publishing establishments and timber mills (Thomas *et al.*, 1982).

According to the US Environmental Protection Agency, the entrance of 1,1,2-trichloroethane into groundwater and surface water most probably occurs through the direct discharge of chemical wastes into water and the disposal of waste into landfills, with subsequent leaching of the chemical to groundwater. 1,1,2-Trichloroethane was found in 66% of 399 drinking-water samples from New Jersey, USA, with groundwater sources. Concentrations were distributed as follows: 203 samples, < 1 µg/l; 141 samples, 1-10 µg/l; 55 samples, 10-100 µg/l; one sample, > 100 µg/l. 1,1,2-Trichloroethane was also detected in 50 of 372 well-water samples (maximum, 300 µg/l) from Long Island, NY, in 1978 (Thomas *et al.*, 1982).

Tap-water samples collected in 1981 from Port Robinson, Niagara Falls and Chippawa, Ontario, Canada, contained concentrations of 0.48, 0.045-0.12 and 0.15 µg/l 1,1,2-trichloroethane, respectively. Surface water samples collected from 17 sites in 1980 and 19 sites in 1981 in the same vicinity (Welland River watershed) contained concentrations ranging from not detected to trace (< 0.04 µg/l) (Kaiser & Comba, 1983).

In the 1982 Nationwide Urban Runoff Program, 1,1,2-trichloroethane was detected in samples from one of the 15 reporting cities at a range of 2-3 µg/l (Cole *et al.*, 1984).

Of approximately 20 000 groundwater samples collected in Suffolk County, NY, USA, 5.9% from community water systems and 4.5% from other water systems (non-community and private wells) contained 1,1,2-trichloroethane. The maximal concentration was 13 µg/l in community water systems and 1 µg/l in non-community water systems; none was detected in private wells (Zaki, 1986).

In 1985, the California Department of Health Services carried out a survey in which the water samples from 2949 wells throughout California were analysed for organic contaminants; 1,1,2-trichloroethane was found at concentrations ranging from 0.7 to 1.1 µg/l in four wells (Reed *et al.*, 1988).

(f) Human tissues

The mean concentrations (micrograms per kilogram fresh weight) of 1,1,2-trichloroethane in human tissues samples collected in 1978 from the general population in the Ruhr District of Germany were: kidney capsule fat, 6.1 (max., 35.7); hypodermal fat, 13.9 (max., 158.6); lung, 2.3 (max., 34.0); liver, 2.6 (max., 25.3); and muscle, 17.2 (Bauer, 1981).

(g) Other

1,1,2-Trichloroethane has been identified as a by-product in the production of polyvinyl chloride plastics; it constituted 40% of the volatile components of a by-product known as EDC-tar collected from a Swedish factory (Rosenberg *et al.*, 1975).

1,1,2-Trichloroethane was identified as an impurity in nine of 22 samples of commercial 1,1,1-trichloroethane, at concentrations ranging from 300 to 3015 µg/ml (Henschler *et al.*, 1980).

2.3 Analysis

Selected methods for the analysis of 1,1,2-trichloroethane in air and water are identified in Table 2. The US Environmental Protection Agency methods for

analysing water (Methods 8010 and 8240) have also been applied to liquid and solid wastes. Volatile components of solid-waste samples are first extracted with methanol prior to purge-and-trap concentration and analysis by gas chromatography-electrolytic conductivity detection (Method 8010) or gas chromatography-

Table 2. Methods for the analysis of 1,1,2-trichloroethane

Sample matrix	Sample preparation[a]	Assay procedure[b]	Limit of detection[c]	Reference
Air	Adsorb on activated charcoal; desorb (carbon disulfide); inject aliquot	GC/FID	0.01 mg per sample	Eller (1987); Lodge (1989a)
	Collect whole air sample in passivated stainless steel canister	GC/FID	10 ppm (54.6 mg/m^3)	US Environmental Protection Agency (1988a) [Method TO-14]; Lodge (1989b)
	Draw air through tube; compare reaction with standard chart	Colorimetric	NR	SKC Inc. (1990)
Breath	Collect in plastic bag; evacuate cell; draw sample in and scan	FT-IR	10 ppm (54.6 mg/m^3)	Lodge (1989c)
Water	Purge (inert gas); trap (OV-1 on Chromosorb-W/Tenax/silica gel); desorb as vapour (heat to 180°C, backflush with inert gas) onto packed GC column	GC/ECD	0.02 µg/l	US Environmental Protection Agency (1988b) [Method 601]
		GC/MS	5.0 µg/l	US Environmental Protection Agency (1988c) [Method 624]
Water	Purge (inert gas); trap (OV-1 on Chromosorb-W/Tenax/silica gel); desorb as vapour (heat to 180°C, backflush with inert gas) onto capillary GC column	GC/ECD	0.04 µg/l	US Environmental Protection Agency (1988d) [Method 502.2]
		GC/MS	0.10 µg/l	US Environmental Protection Agency (1988e) [Method 524.2]
	Add internal standard (isotope-labelled 1,1,2-trichloroethane); purge; trap and desorb as above	GC/MS	10 µg/l	US Environmental Protection Agency (1988f) [Method 1624]

[a]GC, gas chromatograph

[b]GC/FID, gas chromatography/flame ionization detection; FT-IR, Fourier transform/infrared spectroscopy; GC/ECD, gas chromatography/electrolytic conductivity detection; GC/MS, gas chromatography/mass spectrometry

[c]NR, not reported

mass spectrometry (Method 8240). The detection limit using Method 8010 is 0.02 μg/l, and the practical quantification limit using Method 8240 is 5 μg/l for groundwater and 5 μg/kg for soil/sediment samples (US Environmental Protection Agency, 1986a,b). Method 624 has also been adapted to the analysis of 1,1,2-trichloroethane in fish, with an estimated detection limit of 10 μg/kg (Easley *et al.*, 1981).

3. Biological Data Relevant to the Evaluation of Carcinogenic Risk to Humans

3.1 Carcinogenicity studies in animals (Table 3)

(*a*) *Oral administration*

Mouse: Groups of 50 male and 50 female B6C3F$_1$ mice, five weeks of age, received technical-grade 1,1,2-trichloroethane (one batch; purity, 99, 91 and 95% in three analyses over a one-year period [impurities unspecified]) in corn oil by gavage on five consecutive days per week for 78 weeks. Low-dose and high-dose animals received, respectively, 150 and 300 mg/kg bw per day for eight weeks and then 200 and 400 mg/kg bw per day for 70 weeks, followed by 12-13 weeks without treatment, after which the experiment was terminated. The time-weighted average doses were 195 and 390 mg/kg bw per day, respectively [calculated over seven days per week]. Groups of 20 male and 20 female mice either received corn oil alone and served as matched vehicle controls or remained untreated and served as matched untreated controls. At least 50% of the male mice in each group were alive at week 86; 50% of the female mice were still alive after 90, 89, 58 and 81 weeks in the untreated control, vehicle control, low-dose and high-dose groups, respectively. The incidence of hepatocellular neoplasms [reported as carcinomas] was increased significantly ($p < 0.01$) in all treated groups: males—2/17 (untreated controls, 2/20 vehicle controls, 18/49 low-dose animals and 37/49 high-dose animals; in females—2/20 untreated controls, 0/20 vehicle controls, 16/48 low-dose animals and 40/45 high-dose animals. Adrenal phaeochromocytomas were present in 8/48 high-dose males and in 12/43 high-dose females, but in no other group (National Cancer Institute, 1978).

Rat: Groups of 50 male and 50 female Osborne-Mendel rats, six weeks of age, received technical-grade 1,1,2-trichloroethane (see above) in corn oil by gavage on five consecutive days a week for 78 weeks. Low-dose and high-dose groups received, respectively, 35 and 70 mg/kg bw per day for 20 weeks, then 50 and 100 mg/kg bw per day for 58 weeks and were left untreated for the subsequent 34-35 weeks. The

time-weighted average doses were 46 and 92 mg/kg bw per day [calculated over seven days per week]. Groups of 20 male and 20 female rats either received corn oil alone and served as matched vehicle controls or remained untreated and served as matched untreated controls. At least 50% of the male rats in untreated, low-dose and high-dose control groups survived more than 96 weeks; 50% of the females in the untreated control, low-dose and high-dose groups survived more than 105 weeks. Vehicle control groups had unexpectedly poor survival, with only 5% (1/20) of males and 20% (4/20) of females still alive at the end of the study; the authors did not, therefore, include them in statistical comparisons. No statistically significant increase in tumour incidence was found, either in males or in females (National Cancer Institute, 1978).

In a screening assay based on the production of γ-glutamyltranspeptidase-positive foci in rat liver, 10 male Osborne-Mendel rats (weighing 180-230 g when received and observed for a further five days before the start of the experiment) were given a single dose of 0.52 mmol/kg bw ([69.4 mg] maximum tolerated dose) 1,1,2-trichloroethane (98% pure) in corn oil by gavage 24 h following a two-thirds partial hepatectomy. Ten male rats given 2 ml/kg bw corn oil following partial hepatectomy served as vehicle controls. Six days after partial hepatectomy, the rats were given 0.05% (w/w) phenobarbital in the diet for seven weeks; they were then transferred to their regular diet for seven more days, at which time they were sacrificed. The number of foci/cm^2 liver in rats given 1,1,2-trichloroethane was not greater than that in the vehicle controls [numbers of foci not reported]. In further studies, groups of 10 male rats were given a single initiating dose of 30 mg/kg bw *N*-nitrosodiethylamine in 5 ml water or water alone by intraperitoneal injection 24 h after a two-thirds partial hepatectomy. Six days later, the rats were given 0.52 mmol/kg bw 1,1,2-trichloroethane in corn oil or corn oil alone by gavage on five days per week for seven weeks. In rats initiated with *N*-nitrosodiethylamine, 1,1,2-trichloroethane significantly increased the incidence of γ-glutamyltranspeptidase-positive foci/cm2 liver: control (*N*-nitrosodiethylamine plus corn oil), 1.6 ± 0.3 (SD); treated, 6.3 ± 2.2 ($p < 0.05$, Student's *t* test). In rats not initiated with *N*-nitrosodiethylamine, 1,1,2-trichloroethane also produced a significant increase in the number of foci/cm2 liver: control (water plus corn oil), 0.4 ± 0.2; treated, 4.4 ± 1.3 ($p < 0.05$) (Story *et al.*, 1986).

(*b*) *Subcutaneous injection*

Rat: Groups of 50 male and 50 female Sprague-Dawley rats [200-250 g] were given 15.37 or 46.77 μmol [2.05 or 6.24 mg] 1,1,2-trichloroethane (> 99% pure) in 0.25 ml dimethylsulfoxide by subcutaneous injection once a week for two years. Groups of 35 male and 50 female rats given 0.25 ml dimethylsulfoxide on the same dosing schedule served as vehicle controls; groups of the same size without

treatment served as untreated controls. The median survival time was: males—untreated control, 100 weeks; vehicle control, 87 weeks; low-dose, 90 weeks; high-dose, 85 weeks; females—untreated control, 91 weeks; vehicle control, 95 weeks; low-dose, 86 weeks; high-dose, 83 weeks. Sarcomas occurred at various sites in none of the untreated controls, in 2/35 and 3/50 vehicle control, in 4/50 and 3/50 low-dose and in 8/50 and 5/50 high-dose rats. The proportion of low- or high-dose rats with sarcomas was not significantly larger than that of vehicle controls (Norpoth *et al.*, 1988).

3.2 Other relevant data

(*a*) *Experimental systems*

(i) *Absorption, distribution, excretion and metabolism*

The blood/gas partition coefficient (at 37°C) of 1,1,2-trichloroethane in rats was 58 (Gargas *et al.*, 1989), and the blood serum/gas partition coefficient (at 25°C) in humans was 56 (Morgan *et al.*, 1972); these findings predict that this compound is readily absorbed by inhalation.

1,1,2-Trichloroethane is absorbed through mouse skin (Tsuruta, 1975): when 5.4 mmol (0.5 ml) of undiluted 1,1,2-trichloroethane were applied to a 2.92-cm^2 area of mouse skin, 5.7 μmol (763 μg) were absorbed after 15 min, 0.015 μmol (2 μg) was eliminated in expired air, and the balance was retained in the body. The percutaneous absorption rate was 130 nmol/min per cm^2 of skin. Skin absorption was confirmed in guinea-pigs (Jakobson *et al.*, 1977).

In mice exposed to 1000 ppm (5445 mg/m^3) 1,1,2-trichloroethane by whole-body inhalation for 1 h, the highest concentrations of the halocarbon immediately after exposure were found in fat, followed by liver and kidney (Takahara, 1986).

In dogs given undiluted 1,1,2-trichloroethane (0.375 or 0.75 mmol/kg, 50 or 100 mg/kg) intravenously, about 20 or 30%, respectively, was eliminated in the expired air within 60 min (Hobara *et al.*, 1981).

The metabolic kinetic constants for 1,1,2-trichloroethane in rats exposed *in vivo* by inhalation to 501 ppm [2.7 g/m^3] for 6 h were K_m = 0.75 mg/l (5.63 μM) and V_{max} = 7.70 mg/kg per h (57.7 μmol/kg per h) (Gargas & Andersen, 1989).

^{14}C-1,1,2-Trichloroethane (0.75-1.5 mmol/kg, 100-200 mg/kg) was given in olive oil to mice by intraperitoneal injection, and the elimination of radiolabel was followed for three days (Yllner, 1971); 73-87% of the dose was eliminated in the urine, 0.1-2% in the feces and 16-22% in expired air, largely (60%) as ^{14}C-carbon dioxide. Several urinary metabolites were identified: chloroacetic acid (6-31% of urinary radioactivity), free *S*-carboxymethylcysteine (29-46%), conjugated *S*-carboxymethylcysteine (3-10%), thiodiacetic acid (38-42%), 2,2-dichloroethanol

Table 3. Summary of carcinogenicity studies of 1,1,2–trichloroethane in experimental animals

Reference	Species/ strain	Sex	Dose schedule	Experimental parameter/ observation[a]	Group				Significance
					0	1	2	3	
National Cancer Institute (1978)	Mouse $B6C3F_1$	M	5 d/week, gavage, corn oil, 78 weeks	Dose (mg/kg TWA)	0*	0*	195	390	
				Survival (91 weeks)	11/20	10/20	25/50	18/50	
				Hepatocellular carcinoma	2/17	2/20	18/49	37/49	$p < 0.001$
				Adrenal phaeochromocytoma	0/18	0/20	0/49	8/48	$p = 0.003$
		F		Dose (mg/kg TWA)	0*	0*	195	390	
				Survival (91 weeks)	11/20	18/20	21/50	15/50	
				Hepatocellular carcinoma	2/20	0/20	16/48	40/45	$p < 0.001$
				Adrenal phaeochromocytoma	0/20	0/20	0/48	12/43	$p = 0.006$
National Cancer Institute (1978)	Rat Osborne-Mendel	M	5 d/week, gavage, corn oil, 78 weeks	Dose (mg/kg TWA)	0*	0*	46	92	
				Survival (111 weeks)	6/20	1/20	11/50	15/50	
		F		Dose (mg/kg TWA)	0*	0*	46	92	
				Survival (111 weeks)	8/20	4/20	32/50	21/50	
Norpoth *et al.* (1988)	Rat Sprague-Dawley	M	1/week, s.c. inj., dimethyl sulfoxide, 2 years	Dose (μmol/rat)	0**	0**	15.37	46.77	
				Survivl (median days)	696	605	633	594	
				Sarcoma (not necessarily at injection site)	0/35	2/35	4/50	8/50	
		F		Dose (μmol/rat)	0**	0**	15.37	46.77	
				Survival (median days)	639	668	602	584	
				Sarcoma (not necessarily at injection site)	0/50	3/50	3/50	5/50	

[a]TWA, time-weighted average

*Group 0, untreated; group 1, corn oil

**Group 0, untreated; group 1, 0.25 ml dimethylsulfoxide

(1-2%), oxalic acid (about 0.5%) and trichloroacetic acid (1.4-2.3%). The latter compound may be formed from an impurity or may be a metabolite of 1,1,2-trichloroethane.

Thiodiglycolic acid (about 20% of the dose) was identified as a urinary metabolite of 1,1,2-trichloroethane given intraperitoneally (40 or 160 μmol [5.3 or 21.3 mg]/rat) in corn oil to rats (Norpoth *et al.*, 1988).

Daily oral doses of 1,1,2-trichloroethane (0.52 mmol/kg [70 mg/kg] to rats and 2.24 μmol/kg [300 mg/kg] to mice) on five days per week for four weeks followed by a single dose of ^{14}C-1,1,2-trichloroethane in corn oil resulted in elimination of about 7-9 or 3-5% of the dose in the expired air as 1,1,2-trichloroethane and volatile metabolites or as ^{14}C-carbon dioxide, respectively, by 48 h (Mitoma *et al.*, 1985). The excreta contained about 75% of the administered radiolabel, and 2-4% was retained in the carcass. *S*-Carboxymethylcysteine, thiodiacetic acid and chloroacetic acid were identified as urinary metabolites in both rats and mice.

1,1,2-Trichloroethane is metabolized by rat hepatic cytochromes P450 to chloroacetic acid (Ivanetich & Van den Honert, 1981) and to inorganic chloride (Van Dyke & Wineman, 1971; Van Dyke & Gandolfi, 1975).

When ^{14}C-1,1,2-trichloroethane was incubated in air with primary cultures of rat hepatocytes isolated from phenobarbital-treated rats, covalent binding of 1,1,2-trichloroethane metabolites to protein (about 1.6 nmol/mg protein per 30 min) and to lipid (about 5 nmol/mg lipid phosphorus) was detected (DiRenzo *et al.*, 1984).

(ii) *Toxic effects*

The single-dose oral LD_{50}s (in 10% Emulphor in water) of 1,1,2-trichloroethane in male and female CD-1 mice were estimated to be 378 and 491 mg/kg bw, respectively (White *et al.*, 1985). Six-hour LC_{50} values of 1654 (9 g/m^3) and 416 ppm (2.3 g/m^3) 1,1,2-trichloroethane by whole-body inhalation have been reported for male Sprague-Dawley rats and for mice [sex and strain not specified], respectively (Bonnet *et al.*, 1980). Percutaneous applications of undiluted 1,1,2-trichloroethane at doses of 0.5 or 2.0 ml/3.1 cm^2 skin to male and female guinea-pigs resulted in 100% mortality; 0.25 ml was lethal for 25% of the animals (Wahlberg, 1976). Intraperitoneal administration of 0.25-2.0 ml/animal 1,1,2-trichloroethane to male and female guinea-pigs was also lethal to all animals (Wahlberg & Boman, 1979). Signs of toxicity included sedation, gastric irritation, lung haemorrhage and liver and kidney damage (White *et al.*, 1985).

Daily administration of 3.8 or 38 mg/kg bw 1,1,2-trichloroethane by gavage (in 10% Emulphor in water) to male and female CD-1 mice for 14 days had no significant toxic effect (Sanders *et al.*, 1985; White *et al.*, 1985).

Administration of 0.02, 0.2 or 2 g/l 1,1,2-trichloroethane in the drinking-water of male and female CD-1 mice for 90 days [calculated daily doses: males—4.4, 46 or 305 mg/kg bw; females—3.9, 44 or 384 mg/kg bw] resulted in body weight reduction, depressed peritoneal macrophage function and decreased liver glutathione levels in males; decreased haematocrit and haemoglobin levels in females; and increased serum alkaline phosphatase activities and decreased haemagglutination titres in males and females at some doses. No other significant toxic effect was observed (Sanders *et al.*, 1985; White *et al.*, 1985).

1,1,2-Trichloroethane significantly increased the total number of enzyme-altered foci in livers of Osborne-Mendel rats when administered after treatment with *N*-nitrosodiethylamine (Milman *et al.*, 1988; see also section 3.1).

(iii) *Effects on reproduction and prenatal toxicity*

In a developmental toxicity screening study, 1,1,2-trichloroethane given orally in corn oil at a dose that killed 3/30 pregnant mice caused no developmental toxicity in offspring of survivors (Seidenberg *et al.*, 1986).

(iv) *Genetic and related effects* (Table 4)

The genetic and related effects of 1,1,2-trichloroethane have been reviewed (Infante & Tsongas, 1982).

1,1,2-Trichloroethane did not induce mutation in *Salmonella typhimurium*. It caused chromosome malsegregation in *Aspergillus nidulans* and morphological transformation of BALB/c 3T3 cells. 1,1,2-Trichloroethane bound to DNA *in vitro* (DiRenzo *et al.*, 1982) and to DNA, RNA and protein of various organs following treatment of rodents *in vivo* (Mazzullo *et al.*, 1986). Strong S-phase induction but no unscheduled DNA synthesis was observed in livers of treated mice.

A study in which a negative response was reported in *S. typhimurium* and positive responses in tests for unscheduled DNA synthesis in rat hepatocytes and for transformation in BALB/c 3T3 cells could not be evaluated [details not given] (Milman *et al.*, 1988).

(*b*) *Humans*

(i) *Absorption, distribution, excretion and metabolism*

When ^{38}Cl-1,1,2-trichloroethane was administered by inhalation at a dose of about 5 mg/subject in a single breath to human volunteers [subject weight and gender not stated], about 3% of the administered radiolabel was eliminated in the breath within 1 h. Urinary excretion of ^{38}Cl amounted to < 0.01% of the dose/min (Morgan *et al.*, 1970).

(ii) *Toxic effects*

No data were available to the Working Group.

Table 4. Genetic and related effects of 1,1,2–trichloroethane

Test system	Result		Dose LED/HID	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
SA0, *Salmonella typhimurium* TA100, reverse mutation	–	0	0.0000	Simmon *et al.* (1977)[a]
SA0, *Salmonella typhimurium* TA100, reverse mutation	–	–	5300.0000	Barber *et al.* (1981)[a]
SA0, *Salmonella typhimurium* TA100, reverse mutation	–	–	0.0000	Mersch–Sunderman (1989)[b]
SA2, *Salmonella typhimurium* TA102, reverse mutation	–	–	0.0000	Mersch–Sunderman (1989)[c]
SA5, *Salmonella typhimurium* TA1535, reverse mutation	–	–	3900.0000	Rannug *et al.* (1978)
SA5, *Salmonella typhimurium* TA1535, reverse mutation	–	–	5300.0000	Barber *et al.* (1981)[a]
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	5300.0000	Barber *et al.* (1981)[a]
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	0.0000	Mersch–Sunderman (1989)[b]
SAS, *Salmonella typhimurium* TA97, reverse mutation	–	–	0.0000	Mersch–Sunderman (1989)[b]
ANN, *Aspergillus nidulans*, chromosome malsegregation	+	0	750.0000	Crebelli *et al.* (1988)
ANG, *Aspergillus nidulans*, genetic crossing over	–	0	750.0000	Crebelli *et al.* (1988)
TBM, Cell transformation, BALB/c3T3 mouse cells *in vitro*	(+)	0	25.0000	Tu *et al.* (1985)[a]
???, S–phase synthesis induction, mouse hepatocytes *in vivo*	+	0	0.0000	Mirsalis *et al.* (1989)
UVM, Unscheduled DNA synthesis, mouse hepatocytes *in vivo*	–	0	1000.0000	Mirsalis *et al.* (1989)
BVD, Binding to DNA, mouse cells *in vivo*	+	0	0.8000	Mazzullo *et al.* (1986)
BVD, Binding to DNA, rat cells *in vivo*	+	0	0.8000	Mazzullo *et al.* (1986)
BVP, Binding to RNA and protein, rat cells *in vivo*	+	0	0.8000	Mazzullo *et al.* (1986)
BVP, Binding to RNA and protein, mouse cells *in vivo*	+	0	0.8000	Mazzullo *et al.* (1986)

[a]Closed container
[b]Negative in closed container, standard test, or spot test
[c]Negative in standard test, spot test

(iii) *Effects on reproduction and prenatal toxicity*

No data were available to the Working Group.

(iv) *Genetic and related effects*

No data were available to the Working Group.

3.3 Case reports and epidemiological studies of carcinogenicity to humans

No data were available to the Working Group.

4. Summary of Data Reported and Evaluation

4.1 Exposure data

1,1,2-Trichloroethane is used as an intermediate in the production of vinylidene chloride and, to a lesser extent, as a special-purpose industrial solvent and as a chemical intermediate in other processes. It has been detected in drinking-water as well as in untreated groundwater and surface water in some locations; it may occur mainly as a result of industrial emissions.

4.2 Experimental carcinogenicity data

1,1,2-Trichloroethane was tested for carcinogenicity in a two-year study in male and female B6C3F$_1$ mice and Osborne-Mendel rats by oral administration and in Sprague-Dawley rats by subcutaneous injection. In the studies by oral administration, 1,1,2-trichloroethane produced hepatocellular neoplasms and adrenal phaeochromocytomas in mice of each sex but did not significantly increase the proportion of rats with neoplasms at any site relative to untreated controls. In the study in rats by subcutaneous injection, 1,1,2-trichloroethane did not increase the incidence of neoplasms.

In a screening assay for γ-glutamyltranspeptidase-positive foci in the liver of male Osborne-Mendel rats, 1,1,2-trichloroethane did not increase the number of foci in the liver in the initiation protocol (single injection), but the number was increased in the promotion protocol (repeated injections), with or without initiation by *N*-nitrosodiethylamine.

4.3 Human carcinogenicity data

No data were available to the Working Group.

4.4 Other relevant data

1,1,2-Trichloroethane was not mutagenic to bacteria. In single studies, it induced chromosomal malsegregation in a fungus and transformation in cultured mammalian cells. S-Phase induction, but not unscheduled DNA synthesis, was observed in mice after treatment *in vivo*.

4.5 Evaluation[1]

There is *limited evidence* for the carcinogenicity of 1,1,2-trichloroethane in experimental animals.

No data were available from studies in humans on the carcinogenicity of 1,1,2-trichloroethane.

Overall evaluation

1,1,2-Trichloroethane *is not classifiable as to its carcinogenicity to humans (Group 3).*

5. References

Aldrich Chemical Co. (1990) *1990-1991 Aldrich Handbook of Fine Chemicals*, Milwaukee, WI, p. 1269

American Conference of Governmental Industrial Hygienists (1989) *TLVs Threshold Limit Values and Biological Exposure Indices for 1989-1990*, Cincinnati, OH, p. 40

American Tokyo Kasei (1988) *TCI American Organic Chemicals 88/89 Catalog*, Portland, OR, p. 1218

Barber, E.D., Donish, W.H. & Mueller, K.R. (1981) A procedure for the quantitative measurement of the mutagenicity of volatile liquids in the Ames *Salmonella*/microsome assay. *Mutat. Res.*, *90*, 31-48

Bauer, U. (1981) Human exposure to environmental chemicals—Investigations on volatile organic halogenated compounds in water, air, food, and human tissues. III. Communication: results of investigations. *Z. Bakteriol. Mikrobiol. Hyg. Abt. 1 Orig. B*, *174*, 200-237

Bonnet, P., Francin, J.-M., Gradiski, D., Raoult, G. & Zissu, D. (1980) Determination of the median lethal concentration of the main chlorinated aliphatic hydrocarbons in the rat (Fr.). *Arch. Mal. prof.*, *41*, 317-321

[1]For definition of the italicized terms, see Preamble, pp. 30-33.

Brodzinsky, R. & Singh, H.B. (1983) *Volatile Organic Chemicals in the Atmosphere: An Assessment of Available Data* (EPA-600/3-83-027a; NTIS PB83-195503), Research Triangle Park, NC, US Environmental Protection Agency

Budavari, S., ed. (1989) *The Merck Index*, 11th ed., Rahway, NJ, Merck & Co., p. 1516

Chemical Information Services Ltd (1988) *Directory of World Chemical Producers—1989/90 Edition*, Oceanside, NY, p. 567

Chemical Information Systems, Inc. (1990) *ISHOW Database*, Baltimore, MD

Cole, R.H., Frederick, R.E., Healy, R.P. & Rolan, R.G. (1984) Preliminary findings of the Priority Pollutant Monitoring Project of the Nationwide Urban Runoff Program. *J. Water Pollut. Control Fed.*, *56*, 898-908

Cook, W.A. (1987) *Occupational Exposure Limits—Worldwide*, Cincinnati, OH, American Industrial Hygiene Association, pp. 126, 155, 220

Crebelli, R., Benigni, R., Franekic, J., Conti, G., Conti, L. & Carere, A. (1988) Induction of chromosome malsegregation by halogenated organic solvents in *Aspergillus nidulans*: unspecific or specific mechanism? *Mutat. Res.*, *201*, 401-411

DiRenzo, A.B., Gandolfi, A.J. & Sipes, I.G. (1982) Microsomal bioactivation and covalent binding of aliphatic halides to DNA. *Toxicol. Lett.*, *11*, 243-252

DiRenzo, A.B., Gandolfi, A.J., Sipes, I.G., Brendel, K. & Byard, J.L. (1984) Effect of O_2 tension on the bioactivation and metabolism of aliphatic halides by primary rat-hepatocyte cultures. *Xenobiotica*, *14*, 521-525

Dow Chemical Co. (1990) *Material Safety Data Sheet: 1,1,2-Trichloroethane*, Midland, MI

Easley, D.M., Kleopfer, R.D. & Carasea, A.M. (1981) Gas chromatographic-mass spectrometric determination of volatile organic compounds in fish. *J. Assoc. off. anal. Chem.*, *64*, 653-656

Eastman Kodak Co. (1987) *Kodak Laboratory and Research Products* (Catalog No. 53), Rochester, NY, pp. 257, 420

Eller, P.M. (1987) *NIOSH Manual of Analytical Methods*, 3rd ed., Vol. 2, rev. 1 (DHHS (NIOSH) Publ. No. 84-100), Washington DC, US Government Printing Office, pp. 1003-1-1003-6

Gargas, M.L. & Andersen, M.E. (1989) Determining kinetic constants of chlorinated ethane metabolism in the rat from rates of exhalation. *Toxicol. appl. Pharmacol.*, *99*, 344-353

Gargas, M.L., Burgess, R.J., Voisard, D.E., Cason, G.H. & Andersen, M.E. (1989) Partition coefficients of low-molecular-weight volatile chemicals in various liquids and tissues. *Toxicol. appl. Pharmacol.*, *98*, 87-99

Gupta, K.C., Ulsamer, A.G. & Gammage, R. (1984) Volatile organic compounds in residential air: levels, sources and toxicity. In: *Proceedings of the 77th Annual Meeting of the Air Pollution Control Association, San Francisco, CA*, Pittsburgh, PA, Air Pollution Control Association, Section 84-1.3, pp. 1-9

Hagenmaier, H., Werner, G. & Jäger, W. (1982) Quantitative determination of volatile halogenated hydrocarbons in water samples by capillary gas chromatography and electron capture detection. *Z. Wasser Abwasser Forsch.*, *15*, 195-198

Health and Safety Executive (1987) *Occupational Exposure Limits (1985)* (Guidance Note EH 40/87), London, Her Majesty's Stationary Office, p. 21

Henschler, D., Reichert, D. & Metzler, M. (1980) Identification of potential carcinogens in technical grade 1,1,1-trichloroethane. *Int. Arch. occup. environ. Health*, *47*, 263-268

Hobara, T., Kobayashi, H., Iwamoto, S. & Sakai, T. (1981) Diminution of 1,1,1- and 1,1,2-trichloroethane in the blood and their excretion by the lungs (Jpn.). *Jpn. J. ind. Health*, *23*, 377-382

IARC (1979) *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans*, Vol. 20, *Some Halogenated Hydrocarbons*, Lyon, pp. 533-543

IARC (1987a) *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*, Suppl. 7, *Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 43*, Lyon, p. 63

IARC (1987b) *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*, Suppl. 7, *Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 43*, Lyon, p. 62

IARC (1987c) *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*, Suppl. 7, *Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 43*, Lyon, pp. 373-376

IARC (1987d) *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*, Suppl. 7, *Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 43*, Lyon, pp. 376-377

Infante, P.F. & Tsongas, T.A. (1982) Mutagenic and oncogenic effects of chloromethanes, chloroethanes, and halogenated analogues of vinyl chloride. *Environ. Sci. Res.*, *25*, 301-327

Ivanetich, K.M. & Van den Honert, L.H. (1981) Chloroethanes: their metabolism by hepatic cytochrome P-450 *in vitro*. *Carcinogenesis*, *2*, 697-702

Jakobson, I., Holmberg, B. & Wahlberg, J.E. (1977) Variations in the blood concentration of 1,1,2-trichloroethane by percutaneous absorption and other routes of administration in the guinea pig. *Acta pharmacol. toxicol.*, *41*, 497-506

Kaiser, K.L.E. & Comba, M.E. (1983) Volatile contaminants in the Welland River watershed. *J. Great Lakes Res.*, *9*, 274-280

Lodge, J.P., Jr, ed. (1989a) *Methods of Air Sampling and Analysis*, 3rd ed., Chelsea, MI, Lewis Publishers, pp. 678-685

Lodge, J.P., Jr, ed. (1989b) *Methods of Air Sampling and Analysis*, 3rd ed., Chelsea, MI, Lewis Publishers, pp. 78-83

Lodge, J.P., Jr, ed. (1989c) *Methods of Air Sampling and Analysis*, 3rd ed., Chelsea, MI, Lewis Publishers, pp. 171-187

Mazzullo, M., Colacci, A., Grilli, S., Prodi, G. & Arfellini, G. (1986) 1,1,2-Trichloroethane: evidence of genotoxicity from short-term tests. *Jpn. J. Cancer Res. (Gann)*, *77*, 532-539

Mersch-Sunderman, V. (1989) Examination of the mutagenicity of organic micro-contaminations in the environment. II. The mutagenicity of halogenated aliphatic hydrocarbons with the *Salmonella*-microsome test (Ames test) in relation to contamination of ground- and drinking-water (Ger.). *Zbl. Bakt. Hyg. B.*, *187*, 230-243

Milman, H.A., Story, D.L., Riccio, E.S., Sivak, A., Tu, A.S., Williams, G.M., Tong, C. & Tyson, C.A. (1988) Rat liver foci and *in vitro* assays to detect initiating and promoting effects of chlorinated ethanes and ethylenes. *Ann. N.Y. Acad. Sci., 534*, 521-530

Mirsalis, J.C., Tyson, C.K., Steinmetz, K.L., Loh, E.K., Hamilton, C.M., Bakke, J.P. & Spalding, J.W. (1989) Measurement of unscheduled DNA synthesis and S-phase synthesis in rodent hepatocytes following in vivo treatment: testing of 24 compounds. *Environ. mol. Mutagenesis, 14*, 155-164

Mitoma, C., Steeger, T., Jackson, S.E., Wheeler, K.P., Rogers, J.H. & Milman, H.A. (1985) Metabolic disposition study of chlorinated hydrocarbons in rats and mice. *Drug chem. Toxicol., 8*, 183-194

Morgan, A., Black, A. & Belcher, D.R. (1970) The excretion in breath of some aliphatic halogenated hydrocarbons following administration by inhalation. *Ann. occup. Hyg., 13*, 219-233

Morgan, A., Black, A. & Belcher, D.R. (1972) Studies on the absorption of halogenated hydrocarbons and their excretion in breath using ^{38}Cl tracer techniques. *Ann. occup. Hyg., 15*, 273-282

National Cancer Institute (1978) *Bioassay of 1,1,2-Trichloroethane for Possible Carcinogenicity. CAS No. 79-00-5* (Technical Report Series No. 74; DHEW (NIH) Publ. No. 78-1324), Washington DC, US Department of Health, Education, and Welfare

National Institute for Occupational Safety and Health (1978) *Chloroethanes: Review of Toxicity. Current Intelligence Bulletin 27* (DHEW (NIOSH) Publ. No. 78-181), Cincinnati, OH, US Department of Health, Education, and Welfare

National Library of Medicine (1989) *Toxic Chemical Release Inventory (TRI) Data Bank: 1,1,2-Trichloroethane*, Bethesda, MD

Norpoth, K., Heger, M., Müller, G., Mohtashamipur, E., Kemena, A. & Witting, C. (1988) Investigations on metabolism and carcinogenicity of 1,1,2-trichloroethane. *J. Cancer Res. clin. Oncol., 114*, 158-162

Otson, R., Williams, D.T. & Bothwell, P.D. (1982) Volatile organic chemicals in water at thirty Canadian potable water treatment facilities. *J. Assoc. off. anal. Chem., 65*, 1370-1374

Page, G.W. (1981) Comparison of groundwater and surface water for patterns and levels of contamination by toxic substances. *Environ. Sci. Technol., 15*, 1475-1481

Pellizzari, E.D. (1982) Analysis for organic vapor emissions near industrial and chemical waste disposal sites. *Environ. Sci. Technol., 16*, 781-785

Pfaltz & Bauer (1988) *Organic and Inorganic Chemicals for Research*, Waterbury, CT, p. 378

Pouchert, C.J., ed. (1974) *The Aldrich Library of NMR Spectra*, Vol. 1, Milwaukee, WI, Aldrich Chemical Co., p. 65B

Pouchert, C.J., ed. (1981) *The Aldrich Library of Infrared Spectra*, 3rd ed., Milwaukee, WI, Aldrich Chemical Co., p. 52F

Pouchert, C.J., ed. (1983) *The Aldrich Library of NMR Spectra*, 2nd ed., Vol. 1, Milwaukee, WI, Aldrich Chemical Co., p. 83A

Pouchert, C.J., ed. (1985a) *The Aldrich Library of FT-IR Spectra*, Vol. 1, Milwaukee, WI, Aldrich Chemical Co., p. 85A

Pouchert, C.J., ed. (1985b) *The Aldrich Library of FT-IR Spectra*, Vol. 3, Milwaukee, WI, Aldrich Chemical Co., p. 118B

Rannug, U., Sundvall, A. & Ramel, C. (1978) The mutagenic effect of 1,2-dichloroethane on *Salmonella typhimurium*. I. Activation through conjugation with glutathione *in vitro*. *Chem.-biol. Interact.*, *20*, 1-16

Reed, N.R., Reed, W., Beltran, L., Babapour, R. & Hsieh, D.P.H. (1988) *Health Risk Assessment of 1,1,2-Trichloroethane (1,1,2-TCA) in California Drinking Water* (UCD/ET-88/2; PB89-131999), Davis, CA, Department of Environmental Toxicology, University of California-Davis

Riedel-de Haën (1986) *Laboratory Chemicals 1986*, Seelze 1/Hannover, p. 1072

Rosenberg, R., Grahn, O. & Johansson, L. (1975) Toxic effects of aliphatic chlorinated by-products from vinyl chloride production on marine animals. *Water Res.*, *9*, 607-612

Sadtler Research Laboratories (1980) *The Sadtler Standard Spectra, 1980 Cumulative Index*, Philadelphia, PA

Sanders, V.M., White, K.L., Jr, Shopp, G.M., Jr & Munson, A.E. (1985) Humoral and cell-mediated immune status of mice exposed to 1,1,2-trichloroethane. *Drug Chem. Toxicol.*, *8*, 357-372

Sax, N.I. & Lewis, R.J., Sr (1987) *Hawley's Condensed Chemical Dictionary*, 11th ed., New York, Van Nostrand Reinhold, p. 1176

Seidenberg, J.M., Anderson, D.G. & Becker, R.A. (1986) Validation of an in vivo developmental toxicity screen in the mouse. *Teratog. Carcinog. Mutagenesis*, *6*, 361-374

Simmon V.F., Kauhanen, K. & Tardiff, R.G. (1977) Mutagenic activity of chemicals identified in drinking water. In: Scott, D., Bridges, B.A. & Sobels, F.H., eds, *Progress in Genetic Toxicology*, Amsterdam, Elsevier/North-Holland Biomedical Press, pp. 249-258

Singh, H.B., Salas, L.J., Stiles, R. & Shigeishi, H. (1983) *Measurements of Hazardous Organic Chemicals in the Ambient Atmosphere* (EPA-600/3-83-002; NTIS PB83-156935), Research Triangle Park, NC, US Environmental Protection Agency

Sittig, M. (1985) *Handbook of Toxic and Hazardous Chemicals and Carcinogens*, 2nd ed., Park Ridge, NJ, Noyes Publications, pp. 881-883

SKC Inc. (1990) *Comprehensive Catalog and Guide. Air Sampling Products for Worker Monitoring, Chemical Hazard Detection and Industrial Hygiene*, Eighty Four, PA

Story, D.L., Meierhenry, E.F., Tyson, C.A. & Milman, H.A. (1986) Differences in rat liver enzyme-altered foci produced by chlorinated aliphatics and phenobarbital. *Toxicol. ind. Health*, *2*, 351-362

Strobel, K. & Grummt, T. (1987) Aliphatic and aromatic halocarbons as potential mutagens in drinking water. III. Halogenated ethanes and ethenes. *Toxicol. environ. Chem.*, *15*, 101-128

Takahara, K. (1986) Experimental study on toxicity of trichloroethane. Part 1. Organ distribution of 1,1,1- and 1,1,2-trichloroethanes in exposed mice (Jpn.). *Okayama Igakkai Zasshi*, *98*, 1079-1089

Thomas, R., Byrne, M., Gilbert, D., Goyer, M. & Wood, M. (1982) *An Exposure and Risk Assessment for Trichloroethanes* (EPA-440/4-85-018; PB85-220598), Washington DC, Office of Water Regulations and Standards, US Environmental Protection Agency

Tsuruta, H. (1975) Percutaneous absorption of organic solvents. 1. Comparative study of the in vivo percutaneous absorption of chlorinated solvents in mice. *Ind. Health, 13*, 227-236

Tu, A.S., Murray, T.A., Hatch, K.M., Sivak, A. & Milman, H.A. (1985) In vitro transformation of BALB/c-3T3 cells by chlorinated ethanes and ethylenes. *Cancer Lett., 28*, 85-92

United Nations Environment Programme (1990) *International Register of Potentially Toxic Chemicals, Recommendations—Legal Mechanisms*, Geneva

US Environmental Protection Agency (1980) *Ambient Water Quality Criteria for Chlorinated Ethanes* (EPA-440/5-80-029; PB81-117400), Washington DC, Office of Water Regulations and Standards

US Environmental Protection Agency (1986a) Method 8010. Halogenated volatile organics. In: *Test Methods for Evaluating Solid Waste—Physical/Chemical Methods*, 3rd ed. (*EPA No. SW-846*), Washington DC, Office of Solid Waste and Emergency Response

US Environmental Protection Agency (1986b) Method 8240. Gas chromatography/mass spectrometry for volatile organics. In: *Test Methods for Evaluating Solid Waste—Physical/Chemical Methods*, 3rd ed. (EPA No. SW-846), Washington DC, Office of Solid Waste and Emergency Response

US Environmental Protection Agency (1988a) Method TO-14. The determination of volatile organic compounds (VOCs) in ambient air using SUMMA(R) passivated canister sampling and gas chromatographic analysis. In: *Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air* (EPA-600/4-89-017; NTIS PB90-116989), Research Triangle Park, NC, Atmospheric Research and Exposure Laboratory, Office of Research and Development

US Environmental Protection Agency (1988b) Methods for organic chemical analysis of municipal and industrial wastewater. Method 601. Purgeable halocarbons. *US Code fed. Regul., Title 40*, Part 136, Appendix A, pp. 267-281

US Environmental Protection Agency (1988c) Methods for organic chemical analysis of municipal and industrial wastewater. Method 624. Purgeables. *US Code fed. Regul., Title 40*, Part 136, Appendix A, pp. 432-446

US Environmental Protection Agency (1988d) Method 502.2. Volatile organic compounds in water by purge and trap capillary column gas chromatography with photoionization and electrolytic conductivity detectors in series. In: *Methods for the Determination of Organic Compounds in Drinking Water* (EPA-600/4-88/039; PB89-220461), Cincinnati, OH, Office of Research and Development, pp. 31-62

US Environmental Protection Agency (1988e) Method 524.2. Measurement of purgeable organic compounds in water by capillary column gas chromatography/mass spectrometry. In: *Methods for the Determination of Organic Compounds in Drinking Water* (EPA-600/4-88/039; PB89-220461), Cincinnati, OH, Office of Research and Development, pp. 285-323

US Environmental Protection Agency (1988f) Methods for organic chemical analysis of municipal and industrial wastewater. Method 1624 Revision B. Volatile organic compounds by isotope dilution GC/MS. *US Code fed. Regul., Title 40*, Part 136, Appendix A, pp. 475-488

US Occupational Safety and Health Administration (1989) Air contaminants—permissible exposure limits (Report No. OSHA 3112). *US Code fed. Regul., Title 29*, Part 1910.1000, pp. 10-73

Van Dyke, R.A. & Gandolfi, A.J. (1975) Characteristics of a microsomal dechlorination system. *Mol. Pharmacol., 11*, 809-817

Van Dyke, R.A. & Wineman, C.G. (1971) Enzymatic dechlorination: dechlorination of chloroethanes and propanes *in vitro*. *Biochem. Pharmacol., 20*, 463-470

Verschueren, K. (1983) *Handbook of Environmental Data on Organic Chemicals*, 2nd ed., New York, Van Nostrand Reinhold, pp. 1128-1129

Wahlberg, J.E. (1976) Percutaneous toxicity of solvents. A comparative investigation in the guinea pig with benzene, toluene and 1,1,2-trichloroethane. *Ann. occup. Hyg., 19*, 115-119

Wahlberg, J.E. & Boman, A. (1979) Comparative percutaneous toxicity of ten industrial solvents in the guinea pig. *Scand. J. Work Environ. Health, 5*, 345-351

Wallace, L.A., Pellizzari, E., Hartwell, T., Rosenzweig, M., Erickson, M., Sparacino, C. & Zelon, H. (1984) Personal exposure to volatile organic compounds. I. Direct measurements in breathing-zone air, drinking water, food, and exhaled breath. *Environ. Res., 35*, 293-319

Weast, R.C., ed. (1989) *CRC Handbook of Chemistry and Physics*, 70th ed., Boca Raton, FL, CRC Press, pp. C-266, D-199

White, K.L., Jr, Sanders, V.M., Barnes, D.W., Shopp, G.M., Jr & Munson, A.E. (1985) Toxicology of 1,1,2-trichloroethane in the mouse. *Drug chem. Toxicol., 8*, 333-355

Wiseman, P. (1972) *An Introduction to Industrial Organic Chemistry*, New York, Wiley Interscience, p. 125

Yllner, S. (1971) Metabolism of 1,1,2-trichloroethane-1,2-^{14}C in the mouse. *Acta pharmacol. toxicol., 30*, 248-256

Zaki, M.H. (1986) Groundwater contamination with synthetic organic compounds and pesticides in Suffolk County. *Northeast. environ. Sci., 5*, 15-22