United States Environmental Protection Agency | Pollution Prevention and Toxics (7407) | November 1994 EPA 749-F-95-020a

OPPT Chemical Fact Sheets

1,2,4-Trichlorobenzene (TCB) Fact Sheet: Support Document (CAS No. 120-82-1)

This summary is based on information retrieved from a systematic search limited to secondary sources (see Appendix A). These sources include online databases, unpublished EPA information, government publications, review documents, and standard reference materials. The literature search done in January of 1995. No attempt has been made to verify information in these databases and secondary sources.

I. CHEMICAL IDENTITY AND PHYSICAL/CHEMICAL PROPERTIES

The chemical identity and physical/chemical properties of 1,2,4-trichlorobenzene (TCB) are summarized in Table 1.

TABLE 1. CHEMICAL IDENTITY AND CHEMICAL/PHYSICAL PROPERTIES OF 1, 2, 4-TRICHLOROBENZENE

Characteristic/Property	Data	Reference
CAS No.	120-82-1	
Common Synonyms	TCB; Unsym-trichlorobenzene	U.S. Air Force 1989
Molecular Formula	$C_6H_3Cl_3$	Budavari et al. 1989
Chemical Structure		Budavari et al. 1989
Physical State	colorless liquid	Keith and Walters 1985
Molecular Weight	181.46	Budavari et al. 1989
Melting Point	17°C	Budavari et al. 1989
Boiling Point	213°C	Budavari et al. 1989
Water Solubility	31 mg/L at 25°C	Darling 1995
Density	1.46 at 25°	Budavari et al. 1989
Vapor Density (air = 1)	6.26	Keith and Walters 1985
K_{OC}	6350	U.S. Air Force 1989
Log K_{OW}	4.12	U.S. Air Force 1989
Vapor Pressure	0.27 mm Hg at 20°C	U.S. Air Force 1989
Reactivity		
Flash Point	110°C	Budavari et al. 1989
Henry's Law Constant	4.33×10^{-3} atm·m³/mol	U.S. Air Force 1989
Fish Bioconcentration Factor	182-815 (bluegill) 1200-3200 (rainbow trout)	U.S. EPA 1987
Odor Threshold	3 ppm	U.S. Air Force 1989
Conversion Factors (in air)	1 ppm = 7.41 mg/m³ 1 mg/m³ = 0.135 ppm	U.S. Air Force 1989

II. PRODUCTION, USE, AND TRENDS

A. Production

In 1990, the total United States production and import volume of 1,2,4-trichlorobenzene was between 22 million and 32 million pounds. More exact figures on production and import volume are not available. Table 2 lists the two producers of 1,2,4-trichlorobenzene in 1992 (PPG Industries, Inc. and Standard Chlorine Chemical Company Inc. of Delaware) along with their plant locations. In 1990, the importers of 1,2,4-trichlorobenzene included Mobay, Inc. and Sandoz Crop Protection of Beaumont, Texas. The TCB production capacity of these two companies are not available.

TABLE 2. UNITED STATES PRODUCERS OF 1,2,4-TRICHLOROBENZENE IN 1992

Company Name	Plant Site
PPG Industries, Inc.	New Martinsville, WV
Standard Chlorine Chemical Company	Delaware City, DE

Source: USITC 1994

B. Uses

Major applications of 1,2,4-trichlorobenzene include its use as a solvent in chemical manufacturing, dyes and intermediates, as a dielectric fluid, as a component of synthetic transformer oils, as a component of lubricants, as a heat-transfer medium, as an ingredient in insecticides and herbicides, as degreasing agents, as septic tank and drain cleaners, as an ingredient in wood preservatives, and as an ingredient in abrasive formulations (HSDB 1994; Sax and Lewis 1987). It is also used as a comonomer with p-dichlorobenzene in the production of arylene sulfide polymers (HSDB 1994).

C. Trends

The use of chlorinated hydrocarbon solvents, of which 1,2,4-trichlorobenzene is one, is being phased out due to more stringent environmental standards. U.S. demand is expected to decrease in the near future (The Freedonia Group, Inc. 1990).

III. ENVIRONMENTAL FATE

A. Environmental Release

Of the total 420 thousand pounds of 1,2,4-trichlorobenzene released to the environment in 1992, as reported to the Toxics Release Inventory by certain types of U.S. industries, 415 thousand pounds were released into the atmosphere, 1 thousand pounds each into surface waters and underground injection sites, and 2.7 thousand pounds onto land (TRI92 1994). As much as 0.25 micrograms/m^3 of TCB has been measured in the air of Los Angeles (IPCS 1991). Concentrations ranging from 0.007 to 275 micrograms/L (ppb) have been measured in the drinking water supplies of U.S. cities (IPCS 1991).

B. Transport

TCB is classified as a bioaccumulator and is persistent in the environment (TRI92 1994). The chemical strongly adsorbs to soils with 1-2% organic content, as predicted by its K_{OC} value, but leaching into ground waters can occur from deep soils (U.S. Air Force 1989). Slow evaporation into the atmosphere occurs from surface water and, to a lesser extent, from soils (IPCS 1991).

C. Transformation/Persistence

1. Air — In the atmosphere, TCB reacts with photochemically produced hydroxyl radicals with an estimated half-life of approximately 18.8 days (U.S. EPA 1987).

2. Soil — One study suggests that TCB may be biodegraded slowly in aerobic soils (U.S. EPA 1987). In another report, a degradation rate of only 1.0 nmole/day per 20 g of soil was measured (IPCS 1991). Generally, the chemical is expected to persist adsorbed to soils (U.S. EPA 1987; U.S. Air Force 1989).

3. Water — Half-lives of TCB in water range from 1 day in rivers to 10 days in lakes and 100 days in ground waters (IPCS 1991). A volatilization half-life of 11 to 22 days has been measured for aerated seawater (U.S. EPA 1987). Biodegradation of TCB in water is not an important removal route (U.S. EPA 1987; IPCS 1991).

4. Biota — Based on the bioconcentration factor of TCB in fish (ranging from 182 to 3200), the chemical is expected to bioaccumulate in aquatic organisms. Biomagnification of trichlorobenzene in the food chain has not been investigated (IPCS 1991).

IV. HUMAN HEALTH EFFECTS

A. Pharmacokinetics

1. Absorption — TCB is readily absorbed following oral, inhalation, and dermal exposure (IPCS 1991). Based on recovery in the feces and urine of radioactivity associated with a single oral dose of TCB to rats or monkeys, these species absorbed 89 and 99%, respectively, of the administered dose (U.S. EPA 1987).

2. Distribution — TCB is preferentially distributed to the fat and liver following oral administration to rats. Starvation of rats for 4 days had no effect on TCB concentrations in fat (IPCS 1991).

3. Metabolism — Metabolism of TCB is mediated by microsomal oxidation to form chlorophenols which are conjugated to glutathione, glucuronic acid or sulfate (IPCS 1991). Following oral administration of 10 mg/kg to rats, 60% of the urinary metabolites consisted of isomers of N-acetyl-S-(trichlorophenyl)-L-cysteine and 33% consisted of isomers of trichlorothiophenol (IPCS 1991).

4. Excretion — Rats and monkeys were given an oral dose of 10 mg/kg radiolabled TCB. In 24 hours, rats had excreted 84 and 11% of the dose in urine and feces, respectively, while monkeys had excreted 40 and 1% of the dose in urine and feces, respectively (U.S. EPA 1987). The half-life of TCB has been reported as 93 hours in the rat (IPCS 1991).

B. Acute Toxicity

TCB is irritating to the eye and respiratory tract. Altered liver enzymes and hepatic porphyria occurs in experimental animals after oral exposure to relatively high doses. At high doses, death can occur following either oral or dermal exposure.

1. Humans — An individual developed eye and respiratory tract irritation from exposure to 3-5 ppm TCB for an unspecified duration (U.S. Air force 1989). The concentration of 4 ppm is roughly equivalent to 4.24 mg/kg over an eight-hour period[1].

2. Animals — Acute oral LD_{50} values of 756 and 766 mg/kg have been reported for rats and mice, respectively. Death occurred within 3 days for mice and 5 days for rats (U.S. Air Force 1989). Reversible hepatic porphyria was induced in rats fed 730 mg/kg/day for 15 days and in rats exposed to 30-100 ppm 7 hours/ day, 5 days/week, for 30 days (U.S. Air Force 1989).

   The acute dermal LD_{50} for TCB in rats is >5 mg/kg. Focal necrosis of the liver of guinea pigs resulted from dermal application of 0.5 mL, 5 days/week, and death of all animals occurred within 3 weeks (IPCS 1991).

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[1]For dose comparison purposes this has been calculated using the factor 7.41 (U.S. Air Force 1989) to convert 4 ppm to 29.64 mg/m^3. This value is multiplied by 0.143 (the occupational standard 8-hour breathing rate, 10 m^3, divided by the assumed adult body weight, 70 kg and assuming 100% absorption) to obtain the dose in mg/kg (U.S. EPA 1988).

C. Subchronic/Chronic Toxicity

Chronic dermal contact with TCB can cause dermatitis in humans. Increased liver weights and altered hepatic enzymes occurred in animals after chronic oral or inhalation exposure to moderate to high levels of TCB.

1. Humans — Prolonged dermal contact with TCB can cause dermatitis (IPCS 1991). Anemia has been reported in two individuals chronically exposed to TCB, although the possibility exists that the workers were exposed to multiple chemicals (U.S. Air Force 1989).

2. Animals — Male and female rats were given 53 mg/kg/day for 1 month by gavage. Increased adrenal gland weight was associated with vacuolization of the zona fasciculata (U.S. EPA 1994a). Rats exposed orally to 40 mg/kg for 90 days had altered hepatic enzyme activities and increased liver weights (U.S. EPA 1987). Female rats were given TCB by gavage for up to 120 days. After 30 days, increased liver porphyrins were observed at $\geq$100 mg/kg and increased liver weights were observed at 200 mg/kg. After 120 days, increased liver porphyrins were observed at $\geq$50 mg/kg (U.S. EPA 1987).

   Rats, rabbits, and monkeys were exposed to 0, 25, 50, or 100 ppm, 7 hours/day, 5 days/week, for up to 26 weeks. No effects were observed in rabbits or monkeys. Rats had a dose-related hepatocytomegaly and vacuolization of hepatocytes, granuloma formation, biliary hyperplasia, and hyaline degeneration of the kidney after 4 and 13 weeks but not after 26 weeks of exposure (U.S. EPA 1994a). Rats, rabbits, and dogs were exposed to 0, 30, or 100 ppm, 7 hours/day, 5 days/week, for 30 exposures in 44 days. At 100 ppm, increased liver weights were observed for rats and dogs and increased kidney weights were observed in rats (U.S. EPA 1987). Urinary porphyrin excretion was elevated in rats exposed to 10 ppm, 6 hours/day, 5 days/week, for 3 months (IPCS 1991).

D. Carcinogenicity

The chlorinated benzene industry has completed cancer studies on TCB in animals in response to an EPA request for testing. 1,2,4-Trichlorobenzene in the diet of animals causes cancer in mice but not in rats. The significance of these results in assessing the potential for TCB to cause cancer in humans is not known.

1. Humans — No information was found in the secondary sources searched concerning the carcinogenicity of TCB in humans.

2. Animals — In 1986, EPA issued a final rule under section 4 of the Toxic Substances Control Act that manufacturers and processors of TCB conduct oncogenicity tests (U.S. EPA 1986). The tests were conducted in $B6C3F_1$ mice (groups of 50/sex) and in F344 rats (groups of 50/sex). 1,2,4-trichlorobenzene was administered for 104 weeks in the diet at 150 ppm, 700 ppm, and 3200 ppm in mice; for 104 weeks in the diet at 100 ppm, 350 ppm, and 1200 ppm in rats (Lai 1994a and 1994b). Results have been submitted to EPA and reviewed. The design and conduct of the studies appear adequate. Under conditions of the test TCB was carcinogenic to mice, inducing liver tumors (hepatocellular adenomas and carcinomas) in both males and females. The incidences of hepatocellular carcinomas (malignant liver tumors) was 8/49, 50/50, 27/50, and 50/50 in the control, low dose, mid dose and high dose male mouse. They were 1/50, 1/50, 28/50, and 46/50, respectively, in the female mouse. For hepatocellular adenomas (benign liver tumors) the incidences were 4/49, 7/50, 16/50, 2/50 for male mice and 3/50, 4/50, 16/50, and 8/50 for female mice. The increased incidences of these liver tumors combined appeared to be statistically significant for the mid-and high-dose male and female mice. TCB was not carcinogenic in either sex of rats. The validity of these findings for assessing human cancer risk is questionable. The mouse liver tumors could have been caused by a secondary mechanism due to liver toxicity (Lai 1994b).

   In another study 0.03 mL of a TCB solution was applied to the skin of male and female mice at concentrations of 30 or 60% twice a week for 2 years. No increase in a specific tumor type was observed in either sex (U.S. EPA 1987, 1994a). The chemical is not listed among the chemicals studied or to be studied by the National Toxicology Program (NTP 1994).

E. Genotoxicity

Results of short-term mutation tests are mixed. The chlorinated benzene industry has completed an unscheduled DNA synthesis study on 1,2,4 trichlorobenzene in response to an EPA request for testing. Results show that TCB does not induce *in vitro* DNA repair at concentrations up to 1% (v/v). Other studies with up to seven strains of *Salmonella typhimurium* were negative for mutation with or without metabolic activation (U.S. EPA 1987; U.S. Air Force 1989). Eight-week-old mice injected i.p. with up to 420 mg/kg of TCB had a dose-related increase in the number of micronucleated cells in the bone marrow (U.S. Air Force 1989).

F. Developmental/Reproductive Toxicity

EPA has derived an oral reference dose (RfD[2]) for TCB of 0.01 mg/kg/day, based on increased adrenal gland weights observed in a multigeneration reproductive study in rats exposed to 400 ppm in the drinking water. No information was found on the developmental or reproductive toxicity of TCB to humans. Developmental toxicity, such as growth retardation, occurred in experimental animals only at maternally toxic doses.

1. Humans — No information was found in the secondary sources searched concerning the developmental or reproductive toxicity of TCB to humans.

2. Animals — The derivation of the oral RfD is based on a multigeneration reproductive study. Male and female rats were exposed to TCB in the drinking water at concentrations of 0, 25, 100, or 400 ppm. At the highest concentration, a significant increase in adrenal gland weight occurred in males and females of the F0 and F1 generations; a no-observed-adverse-effect level (NOAEL) for the study was 100 ppm. No effect was seen in gestation index, fertility,

[2]The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during the time period of concern.

neonate weight, maternal weight, litter size, viability, or growth in either the F0 or F1 generations (U.S. EPA 1994a). Based on these data for the effects on the adrenal gland, the U.S. EPA (1994a) calculated a chronic RfD for TCB of 0.01 mg/kg/day.

Retarded embryonic development associated with maternal toxicity was observed in rat pups following maternal oral doses of 360 mg/kg on gestation days 9-13 (U.S. EPA 1987; U.S. Air Force 1989). No effects were observed on offspring from mice given 130 mg/kg by gavage on gestation days 8-12 (U.S. Air Force 1989).

G. Neurotoxicity

At lethal doses to experimental animals, TCB causes tremors and convulsions.

1. Humans — No information was found in the secondary sources searched concerning the neurotoxicity of TCB to humans.
2. Animals — During LD_{50} studies, rats and mice had symptoms of depressed activity at lower doses and extensor convulsions at lethal doses (IPCS 1991). Tremors followed by death within 20-30 days occurred in monkeys exposed orally to 174 mg/kg/day (U.S. EPA 1994a). No mortality was observed in rats exposed by inhalation to 418 ppm TCB for 4 hours, however, clinical signs included lacrimation, salivation, pink ears, labored breathing, and discoordination (U.S. EPA 1994b).

V. ENVIRONMENTAL EFFECTS

The chlorinated benzene industry has submitted to EPA results of aquatic toxicity testing on 1,2,4-trichlorobenzene. Results show that TCB is highly toxic to aquatic organisms. Based on studies in laboratory animals, it is unlikely that TCB would be toxic to terrestrial animals at environmental levels.

A. Toxicity to Aquatic Organisms

TCB is moderately to highly toxicity to aquatic organisms. The high toxicity is based on a measured algal 96-h EC50 value of 0.37 mg/L (Newsome 1995). The chlorinated benzene industry has completed aquatic toxicity studies in response to an EPA request for testing. Results show that the 96-hr LC_{50} for TCB in mysid shrimp is 0.49 mg/L. TCB adversely affects survival, growth, and reproduction in mysid shrimp. The 28-day chronic toxicity value for mysid shrimp is 0.06 mg/L (Newsome 1995).

Ninety-six-hour LC_{50} values for *Cyprinodon variegatus* (sheepshead minnow) and *Lepomis macrochirus* (bluegill) are 10 mg/L and 3.4 mg/L, respectively. The 48-hour LC_{50} for *Salmo gairdneri* (rainbow trout) is 1.95 mg/L (IPCS 1991). Based on the bioconcentration factor in fish (182-3200), TCB also has the potential to bioconcentrate in the tissues of aquatic organisms (U.S. Air Force 1989).

B. Toxicity to Terrestrial Organisms

No information was found in the secondary sources searched concerning the toxicity of TCB to terrestrial organisms. However based on the LD_{50} values of 756 mg/kg and 766 mg/kg for rats and mice, respectively (U.S. Air Force 1989) it is unlikely that TCB would be toxic to terrestrial animals at environmental levels. Developmental toxicity, such as growth retardation, occurred in experimental animals at maternally toxic doses (U.S. Air Force 1989; U.S. EPA 1987) which are unlikely to occur in the environment.

C. Abiotic Effects

Most TCB in the atmosphere is removed by reaction with photochemically produced hydroxyl radicals (U.S. EPA 1987). TCB is not expected to react to an appreciable amount with ozone (IPCS 1991).

VI. EPA/OTHER FEDERAL/OTHER GROUP ACTIVITY

The Clean Air Act Amendments of 1990 list TCB as a hazardous air pollutant. Federal agencies and groups may develop recommendations to assist in controlling workplace exposure. These agencies and groups (listed in Tables 3 and 4) should be contacted regarding workplace exposures and for additional information on TCB.

TABLE 3. EPA OFFICES AND CONTACT NUMBERS FOR INFORMATION ON TCB

EPA Office	Statute	Contact Number
Pollution Prevention & Toxics	PPA[a]	(202) 260-1023
	EPCRA (§313/TRI)[b]	(800) 535-0202
	TSCA (§4, 8A, 8D)[c]	(800) 554-1404
Air	Clean Air Act (§111.112B)[d]	(919) 541-0888
Solid Waste & Emergency Response	RCRA[e] (Action levels: air, 1E+01 µg/m^3 water, 7E-01 mg/L soil, 2E+03 mg/kg)	(800) 535-0202
	CERCLA[f] (RQ: 100 pounds)	(800) 535-0202
Water	Safe Drinking Water Act[g] (MCL: 70 µg/L; MCLG: 0.07 µg/L; Health Advisories: 0.01 mg/L [ch/1d]; 0.01 mg/L [ch/10d]; 0.1 mg/L [ch/lt]; 0.5 mg/L [a/lt]); 0.07 mg/L [lifetime])	(800) 426-4791
	Clean Water Act(§304B, §307A)[h]	(202) 260-7588
Pesticides	FIFRA[i]	(800) 858-7378

[a]**PPA**: Pollution Prevention Act
[b]**EPCRA**: Emergency Planning and Community Right to Know Act of 1986
[c]**TSCA:** Toxic Substances Control Act
[d]Listed as hazardous air pollutant under §112 of Clean Air Act [42 U.S.C. 7401 et seq. (1990)]
[e]**RCRA**: Resource Conservation and Recovery Act [40 CFR §264.94 (1990)]. Action Levels = Health and environmental-based levels used by the EPA as indicators for the protection of human health and the environment and as triggers for a Corrective Measure Study.
[f]**CERCLA**: Comprehensive Environmental Response, Compensation, and Liability Act of 1980, as amended; **RQ**: level of hazardous substance, which, if equaled or exceeded in a spill or release, necessitates the immediate reporting of that release to the National Response Center [40 CFR Part 302 (1991)].
[g]**MCL**: Maximum contaminant level [40 CFR Part 141 (1994)]; **MCLG**: Maximum contaminant level goal [40 CFR Part 141 (1994)]; Drinking Water Health Advisories. Estimated for a 10-kg child or a 70-kg adult consuming 2 L of water per day. **(ch/1d)**: Child, one-day health advisory = the concentration of a chemical in drinking water that is not expected to cause any adverse noncarcinogenic effects for up to 5 consecutive days of exposure, with a margin of safety. **(ch/10d)**: Child, ten-day health advisory = the concentration of a chemical in drinking water that is not expected to cause any adverse noncarcinogenic effects up to 14 consecutive days of exposure, with a margin of safety. **(ch/lt)**: Child, longer-term health advisory = the concentration of a chemical in drinking water that is not expected to cause any adverse noncarcinogenic effects up to approximately 7 yr (10% of an individual's lifetime) of exposure, with a margin of safety. **(a/lt)**: Adult, longer-term health advisory. **Lifetime**: lifetime health advisory, the concentration of a chemical in drinking water that is not expected to cause any adverse noncarcinogenic effects over a lifetime of exposure, with a margin of safety.
[h]Clean Water Act; regulates waters of the United States, including surface waters, ground waters, and wetlands [40 CFR Part 131 (1994)].
[i]**FIFRA**: Federal Insecticide, Fungicide, and Rodenticide Act

TABLE 4. OTHER FEDERAL OFFICES/CONTACT NUMBERS FOR INFORMATION ON 1,2,4-TRICHLOROBENZENE

Other Agency/Department/Group	**Contact Number**
Agency of Toxic Substances & Disease Registry	(404) 639-6000
American Conference of Governmental Industrial Hygienists (Ceiling limit: 5 ppm [37 mg/m^3])[a]	(513) 742-2020
Consumer Product Safety Commission	(301) 504-0994
Food & Drug Administration	(301) 443-3170
National Institute for Occupational Safety & Health (Ceiling limit: 5 ppm [40 mg/m^3])[a]	(800) 356-4674
Occupational Safety & Health Administration [TWA[b]: 1ppm (7 mg/m^3)] (Check local phone book for phone number under Department of Labor)	

[a] **Ceiling limit**: concentration that should not be exceeded during any part of the working exposure.

[b] **TWA**: Time-weighted-average that must not be exceeded during any 8-hour work shift of a 40-hour workweek. Standard promulgated pursuant to the Occupational Safety and Health Act, 29 CFR 1910 (OSHA 1993).

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APPENDIX A. SOURCES SEARCHED FOR FACT SHEET PREPARATION

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