United States Environmental Protection Agency | Pollution Prevention and Toxics (7407) | February 1995 EPA 749-F-95-010a

OPPT Chemical Fact Sheets

1, 4-Dioxane Fact Sheet: Support Document (CAS No. 123-9-1)

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 These sources include online databases, unpublished EPA information, government publications, review documents, and standard reference materials.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 was done in February 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 1,4-dioxane are summarized in Table 1.

Characteristic/Property	Data	Reference
CAS No.	123-91-1	
Common Synonyms	1,4-diethylene oxide	Budavari et al. 1989
Molecular Formula	$C_4H_8O_2$	Budavari et al. 1989
Chemical Structure		
Physical State	flammable liquid, faint pleasant odor	Budavari et al. 1989
Molecular Weight	88.10	Budavari et al. 1989
Melting Point	11.80ºC	Budavari et al. 1989
Boiling Point	101.1ºC at 760 mm Hg	Budavari et al. 1989
Water Solubility	soluble in water	Budavari et al. 1989
Density	1.0329 g/mL at 20ºC	Keith and Walters 1985
Vapor Density (air = 1)	3.03	Verschueren 1983
K_{OC}	1.23	Howard 1990
Log K_{OW}	-0.27	Howard 1990
Vapor Pressure	30 mm Hg at 20ºC	Verschueren 1983
Reactivity	Tends to form explosive peroxides, especially when anhydrous	Budavari et al. 1989
Flash Point	5-18ºC	Budavari et al. 1989
Henry's Law Constant	4.88×10^{-6} atm•m^3/mol	Howard 1990
Log Bioconcentration Factor	-0.44, calculated	CHEMFATE 1994
Odor Threshold	9.8 mg/m^3 (2.7 ppm)	Verschueren 1983
Conversion Factors	1 mg/ m^3 = 0.278 ppm; 1 ppm = 3.6 mg/m^3	Verschueren 1983

II. PRODUCTION, USE, AND TRENDS

A. Production

In 1992, there were three producers of 1,4-dioxane in the United States: Ferro Corporation, Dow Chemical, and Stepan Co. Dow Chemical was also an importer of 1,4-dioxane in 1992 (U.S. EPA 1987-92). In 1990, the total U.S. production volume of 1,4-dioxane was between 10,500,000 and 18,300,000 pounds (U.S. EPA 1995a). No information on the current capacities of individual producers is available.

Table 2 Producers of 1,4-Dioxane and Plant Sites 1992

Company Name	Plant Site	Plant Capacity
Ferro Corp.	Zachary, LA	NA
Dow Chemical Corp.	Plaquemine, LA	NA
Stepan Chemical Co.	Elwood, IL	NA

Source: U.S. EPA 1987-1992
NA: Not available

B. Uses

In 1985, 90 percent of 1,4-dioxane produced in the U.S. was used as a stabilizer for chlorinated solvents such as 1,1,1-trichloroethane (HSDB 1995). Although this application continues to be the primary one, it is quickly being phased out (Ferro Corp. 1995). In the future, the primary use of 1,4-dioxane will be as a solvent for various applications, primarily in the manufacturing sector. Other solvent applications include those for cellulose acetate, dyes, fats, greases, lacquers, mineral oil, paints, resins, varnishes, and waxes. 1,4-Dioxane is also used in paint and varnish strippers, as a wetting agent and dispersing agent in textile processing, dye baths, stain and printing compositions, and in the preparation of histological slides. Additionally, 1,4-dioxane is used in cosmetics, deodorants, fumigants, automotive coolant liquid, and scintillation counters (Chemical Marketing Reporter 1988; Sax and Lewis 1993; Sittig 1991; USITC 1994).

C. Trends

The total U.S. production volume of 1,4-dioxane for 1982 was estimated at 15 million pounds, up from 12 million pounds reported in 1977. No recent information on production and demand is available.

III. ENVIRONMENTAL FATE

A. Environmental Release

Of the total 1.13 million pounds of 1,4-dioxane released into the U.S. environment in 1992, as reported to the Toxics Release Inventory by certain types of U.S. industries, 680 thousand pounds were released into the atmosphere, 450 thousand pounds were released into surface waters, and 33 hundred pounds were released onto the land (TRI92 1994). 1,4-Dioxane at a concentration of 1 microgram/L has been detected in drinking water in the U.S., (no specific locations given); the chemical was detected in 37% of well water samples collected near a solid waste landfill located 60 miles southwest of Wilmington, DE. 1,4-Dioxane at 1 microgram/L was detected in the Chicago Sanitary and Ship Channel. In 1981, 51% of air samples from three urban New Jersey sites were positive for 1,4-dioxane with mean concentrations ranging from 0.01-0.02 ppb. Only 20% of samples from the same sites were positive in 1982 with a geometric mean concentration of 0-0.01 ppb (Howard 1990).

B. Transport

The low estimated soil-sorption coefficient (K_{oc}), indicates that 1,4-dioxane should readily leach to ground water. The estimated Henry's Law constant suggests that volatilization from moist soils will be slow. Based on its vapor pressure, volatilization from dry soils should be fast. 1,4-Dioxane is not expected to adsorb significantly to suspended sediments (Howard 1990).

C. Transformation/Persistence

1. Air — 1,4-Dioxane in the atmosphere is expected to degrade fairly quickly. The half-life of the reaction of 1,4-dioxane with photochemically produced hydroxyl radicals in the atmosphere was estimated to be 6.7-9.6 hours. Experimental results of sunlight-irradiated mixtures of 1,4-dioxane/NO suggest similar half-lives (Howard 1990).

2. Soil — No adsorption data are available, but the low estimated log soil-sorption coefficient (K_{oc}) suggests that 1,4-dioxane should readily leach to ground water. No data concerning the volatilization of 1,4-dioxane are available, but the estimated Henry's Law constant suggests that volatilization from moist soils will be slow; however, based on its vapor pressure, volatilization from dry soils should be fast. 1,4-dioxane is not expected to biodegrade in soil (Howard 1990).

3. Water — No hydrolysis data on 1,4-dioxane are available. Because ethers in general have been classified as generally resistant to hydrolysis, 1,4-dioxane is not expected to hydrolyze significantly. The estimated Henry's Law constant for 1,4-dioxane and its miscibility in water suggest that volatilization will be slow. From its estimated Koc, 1,4-dioxane is not expected to significantly adsorb to suspended sediments. 1,4-Dioxane is not expected to biodegrade in water (Howard 1990).

4. Biota — Based on its log P, 1,4-Dioxane is not expected to bioconcentrate in fish (Howard 1990).

IV. HEALTH EFFECTS

A. Pharmacokinetics

1. Absorption — No specific information on the absorption of 1,4-dioxane was found in the secondary sources searched. However, systemic toxicity has been observed following oral, inhalation and dermal exposure, indicating that absorption occurs by these routes.

2. Distribution — No specific information on the distribution of 1,4-dioxane was found in the secondary sources searched.

3. Metabolism — The major urinary metabolite of 1,4-dioxane is β-hydroxyethoxyacetic acid, as determined in studies with rats and with human volunteers and dioxane plant personnel (Rowe and Wolf 1982). A second minor urinary metabolite may be diethylene glycol. There is evidence that high doses of 1,4-dioxane can improve the body's ability to metabolize it. One study showed that 1000 mg/kg/day, but not 10 mg/kg/day, caused more rapid excretion of metabolites, and that the 1000 mg/kg/day doses increased the capacity of the rat to metabolize 1,4-dioxane by 62-fold (Rowe and Wolf 1982).

   During an epidemiologic study, urine samples of 5 workers exposed to ≤32 ppm 1,4-dioxane for up to 5 years, were analyzed for 1,4-dioxane and hydroxyethoxyacetic acid (Rowe and Wolf 1982). Essentially all of the dioxane was metabolized to hydroxyethoxyacetic acid, suggesting that the metabolic pathway was not saturated at that dose (Rowe and Wolf 1982).

4. Excretion — Rowe and Wolf (1981) report that 1,4-dioxane and its metabolites are excreted in urine, and that unchanged 1,4-dioxane is found in expired air (IARC 1976).

B. Acute Effects

Dioxane has low acute toxicity. The liquid is painful and irritating to the eyes, irritating to the skin upon prolonged or repeated contact, and can be absorbed through the skin in toxic amounts. Dioxane vapor has poor warning properties and can be inhaled in amounts that may cause serious systemic injury, particularly to the liver and kidneys.

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1. Humans — Exposure of 12 volunteers to a concentration of 1080 mg/m^3 (300 ppm) 1,4-dioxane in air for 15 minutes produced irritation of the eyes, nose and throat (IARC 1976).

   Five deaths due to acute inhalation exposure to 1,4-dioxane have been reported; hemorrhagic nephritis and liver necrosis were recorded at autopsy (IARC 1976). Other workers in the same plant suffered from nausea, vomiting, and irritation of the eyes and respiratory passages (ACGIH 1991).

   Death of a worker, probably attributable to one week's inhalation exposure to about 1800 mg/m^3 (500 ppm in air, which is roughly equivalent to 257 mg/kg over an 8-hour work day)[1] has been reported. In that case, there was also the possibility of skin absorption since the dioxane was also used as a solvent to remove glue from hands (IARC 1976).

   Dioxane vapor has poor warning properties and can be inhaled in amounts that may cause serious systemic injury, particularly to the liver and kidneys (Rowe and Wolf 1982). Serious and fatal exposures can be experienced without forewarning; illness sometimes becomes apparent hours after exposure (Rowe and Wolf 1982).

2. Animals — The oral LD_{50} in mice, rats, and guinea pigs is 5.7, 5.2, and 3.9 g/kg body weight, respectively (IARC 1976).

   Three-hour inhalation exposures at concentrations of 1000-30,000 ppm to guinea pigs, and 8-hour exposures at concentrations of 4000-11,000 ppm to rats, mice, guinea pigs, and rabbits, resulted in irritation of the mucous membranes at the higher doses (Rowe and Wolf 1982). Deaths during or shortly after exposure were from respiratory failure due to lung edema, with animals also exhibiting brain edema. Liver and kidney injuries were almost always apparent upon microscopic examination of animals dying days after exposure, or sacrificed after apparent recovery. The 4-hour inhalation LC_{50} for rats is 14,260 ppm (Rowe and Wolf 1982).

   The dermal LD_{50} for rabbits is 7.6 g/kg (Rowe and Wolf 1982).

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[1] For dose comparison purposes, this has been calculated by multiplying 1800 mg/m^3 by 0.143 (the occupational standard 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/day (U.S. EPA 1988).

C. Subchronic/Chronic Effects

EPA has not published an RfD or RfC for 1,4-dioxane. Epidemiologic studies of workers exposed to low levels of 1,4-dioxane have not shown adverse effects. Dose-related liver and kidney damage have been observed in several species of animals exposed by oral, inhalation, and dermal routes.

1. Humans — No evidence of adverse effects attributable to 1,4-dioxane exposure was found in three epidemiologic studies of dioxane workers. The studies included: (1) 74 workers exposed 3-41 years to levels up to 14.24 ppm dioxane; (2) 165 employees exposed between 1954-1959 to levels up to 32 ppm dioxane; and (3) 80 workers with exposure levels ranging from 0.05 - 51 ppm dioxane (Rowe and Wolf 1982).

   Dioxane is not considered to be a skin irritant in the workplace, but prolonged and repeated contact can cause eczema, as can any effective fat solvent (Rowe and Wolf 1982).

2. Animals — Dropsical changes (excessive fluid retention) in the liver were observed in rabbits and guinea pigs after 10 gavage feedings of 0.1 mL/kg (0.1 g/kg) of 1,4-dioxane. Some animals died following repeated (5, 16, or 20) feedings of 0.5 mL/kg (0.52 g/kg) (Rowe and Wolf 1982).

   1,4-Dioxane at levels of 0.01-1.0% (9.6-1000 mg/kg/day, males; 19.0-1600 mg/kg/day, females) was administered in drinking water to rats for 2 years (Rowe and Wolf 1982). Effects at the high dose included decreased weight gains, high mortality, (only 1 of 60 male rats survived), and decreased water consumption. Kidney and liver damage were observed. At the 0.1% level, the rats developed variable degrees of kidney and liver changes. No adverse effects were observed at the 0.01% level. The authors concluded that toxicity of dioxane is dose-related (Rowe and Wolf 1982).

Rats, mice, guinea pigs, and rabbits were exposed by inhalation to 1,4-dioxane in concentrations ranging from 1000-10,000 ppm for 1.5 hours/day [140-1,400 mg/kg/day for rats][2] (reported as chronic exposure, but no duration specified, Rowe and Wolf 1982). Mortality, usually due to lung injury, was high at the higher levels. Marked liver and kidney injury was seen in all animals surviving repeated exposures. In another experiment, cats, rabbits and guinea pigs (two each), were exposed to 1350 ppm 1,4-dioxane for 8 hours/day for 45 days. Typical liver and kidney damage was seen in the one cat sacrificed after becoming ill, but either slight or no injury was evident in other animals sacrificed after exposure. A similar group plus 2 mice, was exposed to 2700 ppm, 8 hours/day. Seven of the 10 animals died after 4-26 exposures, the rest survived 34 exposures. Observed effects included irritation of the mucous membranes, emaciation, sporadic cramps, narcosis, and albuminuria. In some cases, blood urea nitrogen doubled in concentration (Rowe and Wolf 1982).

Kidney and liver injury has been observed in rabbits and guinea pigs as a result of repeated dermal application of dioxane (Rowe and Wolf 1982).

D. Carcinogenicity

EPA classifies 1,4-dioxane as B2, a probable human carcinogen, based on the induction of nasal cavity and liver carcinomas in multiple strains of rats, liver carcinomas in mice, and gall bladder carcinomas in guinea pigs. IARC classifies 1,4-dioxane as Group 2B, possible human carcinogen.

1. Humans — Human carcinogenicity data for 1,4-dioxane are inadequate. In three epidemiologic studies on workers exposed to 1,4-dioxane, no increase was observed in the number of cancer deaths over that expected (U.S. EPA 1995b).

2. Animals — EPA classifies 1,4-dioxane as B2, a probable human carcinogen (U.S. EPA 1995b). IARC classifies 1,4-dioxane as Group 2B, possible human carcinogen (IARC 1987). 1,4-Dioxane was administered in drinking water to rats (240 to 640 mg/kg/day for 110 weeks) or mice (380 to 830 mg/kg/day for 90 weeks) (U.S. EPA 1995b). Male and female rats had a statistically elevated incidence of nasal cavity squamous cell carcinomas, and female rats had a statistically elevated incidence of liver adenomas, both dose-related. Male and female mice developed a dose-related, statistically significant elevated incidence of liver carcinomas and liver carcinomas or adenomas (U.S. EPA 1995b).

   In other experiments, 1,4-dioxane was administered in drinking water to Sherman rats (0.01-1% for up to 716 days), and guinea pigs (0.5-2.0% for 23 months) (U.S. EPA 1995b). There was a significant increase in the incidence of hepatocellular carcinomas, liver cholangiomas, and nasal cavity carcinomas in high-dose Sherman rats. In guinea pigs, treatment-related gall bladder carcinomas (2/22) and liver hepatomas (3/22) were seen. Male Sprague-Dawley rats were treated with 0.75-1.8% dioxane for 13 months. Nasal cavity carcinomas were observed at all dose levels, and hepatocellular carcinomas were observed at the higher levels. Liver tumors (7/26) were induced in male Wistar rats administered 1% dioxane in drinking water for 63 weeks (U.S. EPA 1995b).

   1,4-Dioxane at levels of 0.01-1.0% (9.6-1000 mg/kg/day, males; 19.0-1600 mg/kg/day, females) was administered in drinking water to rats for 2 years (Rowe and Wolf 1982). At the high dose nasal carcinomas (3/66) and hepatic tumors (10/66) were observed. The authors concluded that toxicity of dioxane is dose-related and that liver damage precedes liver tumor development (Rowe and Wolf 1982).

   No treatment-related lesions were observed in gross and microscopic examination of major

[2] For dose comparison purposes, this has been calculated using the factor 3.6 to convert 1000 ppm to 3600 mg/m^3 which is multiplied by 0.040 (the 1.5 hour breathing rate, 0.014 m^3 [standard 24 hour breathing rate, 0.223] divided by the assumed adult rat body weight 0.350 kg, and assuming 100% absorption) to obtain the dose in mg/kg/day (U.S. EPA 1985).

tissues and organs of Wistar rats exposed to 111 ppm 1,4-dioxane vapor in a two-year inhalation study (U.S. EPA 1995b).

E. Genotoxicity

No evidence of adverse effects was found in chromosomal analyses performed on 6 members of a cohort in an epidemiologic study of dioxane workers (Rowe and Wolf 1982).

1,4-Dioxane induced DNA strand breaks in rat hepatocytes *in vitro*. It did not induce sex-linked recessive lethal mutations in *Drosophila*, or aneuploidy in yeast. It induced chromosomal abnormalities in plants. It was not mutagenic to bacteria (IARC 1976). The chemical was negative in the mouse lymphoma assay, negative for the induction of chromosomal aberrations in cultured Chinese hamster ovary (CHO) cells and weakly positive in the CHO sister-chromatid exchange assay (ACGIH 1991).

F. Developmental/Reproductive Toxicity

There is very little information on the developmental/reproductive toxicity of 1,4-dioxane. There is evidence of fetal toxicity in one rat study at high doses.

1. Humans — No information was found in the secondary sources searched regarding the developmental/reproductive toxicity of 1,4-dioxane in humans.

2. Animals — Rats were administered 0.25-1.0 mL/kg/day (0.25-1.0 g/kg/day) dioxane by gavage on days 6-15 of gestation. At 1.0 mL/kg (1.0 g/kg) reduced fetal weight and reduced maternal body weight were observed (U.S. EPA 1987).

   Rats and mice given 1,1,1-trichloroethane containing 3.5% (32 ppm) dioxane by inhalation showed no evidence of maternal, embryonic, or fetal toxicity, nor any signs of a teratogenic response. These studies are inconclusive with respect to 1,4-dioxane because of the nature of the product tested (Rowe and Wolf 1982).

G. Neurotoxicity

Impaired neurological function has been observed in humans exposed to high levels of 1,4-dioxane by inhalation, and in animals exposed by the inhalation, oral, or dermal routes. These effects include incoordination, narcosis, vertigo, behavioral effects, and coma.

1. Humans — Among symptoms observed in cases of fatal industrial 1,4-dioxane inhalation poisoning (possibly combined with dermal absorption) were drowsiness, vertigo, headache, and coma (Rowe and Wolf 1982).

2. Animals — Cats exposed by inhalation to 1,4-dioxane in concentrations ranging from 12,000 ppm for 7 hours to 31,000 ppm for 3 hours exhibited loss of equilibrium, increased salivation, lacrimation, and narcotic effects. Rapidity of symptom development was concentration-dependent. Activity of all cats decreased gradually after exposure, followed by their death. Similar narcotic effects have been reported in the rabbit (Rowe and Wolf 1982).

   Trained rats were exposed by inhalation to 1500, 3000, or 6000 ppm 1,4-dioxane for 4 hrs/day, 5 days/week for 2 weeks. Apparently dose-related, temporary, and reversible inhibition effects on behavior (avoidance and escape response) were observed (Rowe and Wolf 1982).

   Symptoms observed in single-dose, oral toxicity studies in several species progress from weakness, depression, incoordination, and coma to death (Rowe and Wolf 1982).

   Unsteadiness and incoordination have been observed in rabbits and guinea pigs administered a “sufficient amount” of 1,4-dioxane by repeated topical application (Rowe and Wolf 1982),

V. ENVIRONMENTAL EFFECTS

1,4-Dioxane has low toxicity to aquatic organisms; toxicity values are greater than 100 mg/L. 1,4-Dioxane is not likely to be acutely toxic to aquatic or terrestrial animals at levels found in the environment. Long-term exposure to terrestrial animals may increase tumor incidence.

A. Toxicity to Aquatic Organisms

A report describing acute and chronic toxicity of 1,4-dioxane to *Pimephales promelas* (fathead minnows) reported acute effects at concentrations of 10,000 mg/L. The highest NOAEL was 6000 mg/L. A 32-day embryo-larval test identified a Maximum Allowable Toxicant Concentration (MATC) >145 mg/L (U.S. EPA 1989). The 96-hour LC_{50} value for *Lepomis macrochirus* (bluegill) is 10,000 ppm (10,000 mg/L) in fresh water. The 96-hour LC_{50} value for the fish *Mendia beryllina* in synthetic seawater is reported to be 6,777 ppm (6777 mg/L) (Verschueren 1983).

B. Toxicity to Terrestrial Organisms

No information was found in the secondary sources searched regarding the toxicity of 1,4-dioxane to terrestrial organisms. Based on the oral LD_{50} of 1,4-dioxane for rats of 5.2 g/kg, the chemical is not expected to be acutely toxic to terrestrial animals at levels normally found in the environment. However, long-term exposure from residues in water may increase tumor incidence based on chronic drinking water studies in rats.

C. Abiotic Effects

No information was found in the secondary sources searched regarding the abiotic effects of 1,4-dioxane.

VI. EPA/OTHER FEDERAL/OTHER GROUP ACTIVITY

The Clean Air Act Amendments of 1990 list 1,4-dioxane as a hazardous air pollutant. Occupational exposure to 1,4-dioxane is regulated by the Occupational Safety and Health Administration (OSHA). The OSHA permissible exposure limit (PEL) is 100 parts per million of air (ppm) as an 8-hour time-weighted average (TWA) (29 CFR 1910.000). In addition to OSHA, other 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 1,4-dioxane.

TABLE 3. EPA OFFICES AND CONTACT NUMBERS INFORMATION ON 1.4-DIOXANE

EPA Office	Statute	Contact Number
Pollution Prevention & Toxics	PPA[a] EPCRA (§313/TRI)[b] TSCA (§8A)	(800) 535-0202
Air	Clean Air Act (111, 112B)[c]	(919) 541-0808
Solid Waste & Emergency Response	RCRA (action level: water: 3E-3 mg/L soil: 6E+1 mg/kg) RCRA U Waste[d] CERCLA (RQ: 100 pounds)[e] EPCRA (§304/311/312)[f]	(415) 744-2074 (800) 535-0202
Water	Safe Drinking Water Act (Health Advisories: 4 mg/L [ch/1d] 0.4 mg/L [ch/10d]; 0.7 mg/L [ca risk])[g] PARA-4C[h]	(800) 426-4791

[a] PPA: Pollution Prevention Act

[b] EPCRA: Emergency Planning and Community Right to Know Act of 1986. §313: Toxic Chemicals (TRI Chemicals).

[C] CAA: Clean Air Act. §111: Standards of performance for new stationary sources of air pollutants - Equipment leaks chemical list. §112B: National emissions standards

[d] RCRA: The Resource Conservation and Recovery Act of 1976, (codified as amended at 42 U.S.C. §6901 et seq. 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 Measures Study (U.S. EPA 1990). RCRA U Waste: Other discarded commercial chemical products.

[e] 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)].

[f] EPCRA: Emergency Planning and Community Right to Know Act of 1986.

[g] Drinking Water Health Advisories: estimated for a 10-kg child or a 70-kg adult consuming 2 L of water per day. (ch/1d) (one-day health advisory for a child): 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) (for a child): 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. ca. risk: mg/L at 10^{-4} cancer risk.

[h] PARA-4C: Pretreatment pollutants.

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

Other Agency/Department/Group	Contact Number
Agency of Toxic Substances & Disease Registry	(404) 639-6000
American Conference of Governmental Industrial Hygienists (TLV-TWA:25 ppm [90 mg/m^3] *)[a]	(513) 742-2020
National Institute for Occupational Safety & Health (1 ppm [3.6 mg/m^3] 30 minutes; ca , LF; IDLH: 500 ppm)[b]	(800) 356-4674
Occupational Safety & Health Administration (TWA: 100 ppm [360 mg/m^3] *)[c] (Check local phone book for phone number under Department of Labor)	

[a] TLV-TWA : Time-Weighted-Average concentration for a normal 8-hr workday and a 40-hr workweek to which nearly all workers may be repeatedly exposed without adverse effects (ACGIH 1994-1995). *: air sampling alone is insufficient to accurately quantitate exposure. Measures to prevent significant cutaneous absorption may be required.

[b] C: This concentration that should not be exceeded during any part of the working exposure. Ca: potential human carcinogen. LF: reduce exposure to lowest feasible concentration; when Ca designation accompanies lowest feasible designation, use of only the most reliable and protective respirators is recommended. After a limited review, NIOSH concluded that adverse health effects could occur at the proposed OSHA PELS and NIOSH did not establish a TWA (NIOSH 1994). IDLH: level immediately dangerous to life and health.

[c] TWA: Time-Weighted-Average concentrations that must not be exceeded during any 8-hour work shift of a 40-hour workweek. OSHA standards 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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