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CHEMICAL SUMMARY FOR METHANOL

prepared by
OFFICE OF POLLUTION PREVENTION AND TOXICS
U.S. ENVIRONMENTAL PROTECTION AGENCY
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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 These sources include online databases, unpublished EPA information, government publications, review documents, and standard reference materials. No attempt has been made to verify information contained in these databases and secondary sources.

I. CHEMICAL IDENTITY AND PHYSICAL/CHEMICAL PROPERTIES

The chemical identity and physical/chemical properties of methanol are summarized in Table 1.

TABLE 1. CHEMICAL IDENTITY AND CHEMICAL/PHYSICAL PROPERTIES OF METHANOL

Characteristic/Property	Data	Reference
CAS No.	67-56-1	
Common Synonyms	methyl alcohol, wood alcohol, wood spirit	Budavari et al. 1989
Molecular Formula	CH4O	
Chemical Structure	H | H - C - OH | H	
Physical State	colorless liquid	Verschueren 1983
Molecular Weight	32.04	Budavari et al. 1989
Melting Point	-97.8øC	Budavari et al. 1989
Boiling Point	64.7øC at 760 mm Hg	Budavari et al. 1989
Water Solubility	miscible	Budavari et al. 1989
Density	d20/4, 0.7915 g/mL	Budavari et al. 1989
Vapor Density (air = 1)	1.11	Budavari et al. 1989
KOC	9	CHEMFATE 1994
Log KOW	-0.77	HSDB 1994
Vapor Pressure	126 mm Hg at 25øC	CHEMFATE 1994
Reactivity	Flammable; may explode when exposed to flame	HSDB 1994
Flash Point	12øC	Budavari et al. 1989
Henry's Law Constant	4.55 x 10-6 atm m3/mol	CHEMFATE 1994
Fish Bioconcentration Factor	<1 (estimated)	HSDB 1994
Odor Threshold	highly variable, ranges over several orders of magnitude (10 ppm to 20,000 ppm in air)	HSDB 1994
Conversion Factors	1 ppm = 1.33 mg/m3 1 mg/m3 = 0.76 ppm	Verschueren 1983

II. PRODUCTION, USE, AND TRENDS

A. Production

Methanol, also called methyl alcohol, is manufactured by 13 producers in the United States. Table 2 lists producers, plant locations, and plant capacities. Annual production capacity is approximately 1,626 million gallons. In 1992, 1,345 million gallons of methanol were produced in the US, 495 million gallons were imported into the US, and 50 million gallons were exported (Mannsville 1993).

B. Use

Methanol is used in a variety of industrial applications. Its largest use is as a raw material for the production of methyl t-butyl ether (MTBE), a gasoline additive. It is also used in the production of formaldehyde, acetic acid, chloromethanes, methyl methacrylate, methylamines, dimethyl terephthalate, and as a solvent or antifreeze in paint strippers, aerosol spray paints, wall paints, carburetor cleaners, and car windshield washer compounds. Table 3 shows the estimated 1993 US end-use pattern for methanol.

C. Trends

US consumption of methanol reached an all-time high in 1992, with estimated 1993 consumption surpassing this level. Demand is expected to continue to rise, due primarily to predicted increases in demand for MTBE as a gasoline additive. Demand for MTBE is expected to at least double by 1995 (Mannsville 1993).

TABLE 2. United States Producers of Methanol

Company	Plant Location	Plant Capacity (in millions of gallons)
Air Products	Pensacola, FL	60
Ashland Chemical Co.	Plaquemine, LA	130
Beaumont Methanol	Beaumont, TX	250
Borden	Geismar, LA	210
Coastal Chemical	Cheyenne, WY	26
Enron (Tenneco)	Pasadena, TX	140
Georgia Gulf	Plaquemine, LA	140
Hoechst Celanese	Bishop, TX	160
Lyondell Petrochemicals	Channelview, TX	220
Quantum Chemical	Deer Park, TX	100
Sand Creek Chemical	Commerce City, CO	25
Tennessee Eastman	Kingsport, TN	65
Texaco Chemical	Delaware City, DE	100

Source: Mannsville 1993.

TABLE 3. Estimated 1993 Unites States End-Use Pattern of Methanol

Use of Methanol (typical Standard Industrial Classification (SIC) Code) (see end note 1)	Percentage of US Methanol Use
Methyl t-butyl ether (production, SIC 2911)	37%
Formaldehyde (production, SIC 2869)	24%
Acetic acid (production, SIC 2869)	10%
Chloromethanes (SIC Codes unknown)	6%
Methyl methacrylate (production, SIC 2821)	3%
Methylamines (SIC Codes unknown)	3%
Dimethyl terephthalate (SIC Codes unknown)	2%
Solvents and automotive chemicals (production, SIC 2842)	8%
Miscellaneous (no applicable SIC Code(s))	7%

Source: Mannsville 1993.

III. ENVIRONMENTAL FATE

A. Environmental Release

Methanol ranked third in the U.S. among all chemicals for total releases into the environment in 1992. Of the total released, 195 million pounds were into the atmosphere, 16.4 million pounds were into surface water, 27 million pounds into underground injection sites, and 3.3 million pounds were onto land (TRI92 1994). Methanol detected in the air from Point Barrow, Alaska averaged 0.77 ppb (CHEMFATE 1994). Ambient concentrations from Stockholm, Sweden ranged from 3.83 to 26.7 ppb while concentrations from two remote locations in Arizona were 7.9 and 2.6 ppb (HSDB 1994). In one survey, methanol was detected in drinking waters from 6 of 10 U.S. cities (HSDB 1994) but levels were not included. The chemical has also been detected in rainwater collected from Santa Rita, Arizona (HSDB 1994).

B. Transport

The miscibility of methanol in water and a low KOC (9) indicate that the chemical will be highly mobile in soil (HSDB 1994). Volatilization half-lives from a model river and an environmental pond were estimated at 4.8 days and 51.7 days, respectively (HSDB 1994). Methanol can be removed from the atmosphere in rain water (HSDB 1994).

C. Transformation/Persistence

1. Air - Once in the atmosphere, methanol exists in the vapor phase with a half life of 17.8 days (HSDB 1994). The chemical reacts with photochemically produced hydroxyl radicals to produce formaldehyde (HSDB 1994). Methanol can also react with nitrogen dioxide in polluted air to form methyl nitrite

(HSDB 1994).

2. Soil - Biodegradation is the major route of removal of methanol from soils. Several species of Methylobacterium and Methylomonas isolated from soils are capable of utilizing methanol as a sole carbon source (CHEMFATE 1994).

3. Water - Most methanol is removed from water by biodegradation. The degradation products of methane and carbon dioxide were detected from aqueous cultures of mixed bacteria isolated from sewage sludge (CHEMFATE 1994). Aerobic, Gram-negative bacteria (65 strains) isolated from seawater, sand, mud, and weeds of marine origin utilized methanol as a sole carbon source (CHEMFATE 1994). Aquatic hydrolysis, oxidation, and photolysis are not significant fate processes for methanol (HSDB 1994).

4. Biota - Bioaccumulation of methanol in aquatic organisms is not expected to be significant based on an estimated bioconcentration factor of 0.2 (HSDB 1994).

V. HUMAN HEALTH EFFECTS

A. Pharmacokinetics

1. Absorption - Methanol is readily absorbed after oral, inhalation, or dermal exposure. Oral doses in humans of 71 to 84 mg/kg resulted in blood levels of 4.7 to 7.6 mg/100 mL of blood within 3 hours (Rowe and McCollister 1981). Inhalation of 500 to 1000 ppm methanol for 3 to 4 hours gave urine concentrations of 1 to 3 mg methanol/100 mL of urine at the end of exposure (Rowe and McCollister 1981). Based on urinary methanol levels, the rate of absorption of the chemical appears to be proportional to the concentration of vapor inhaled (HSDB 1994). The rate of dermal absorption increased for 35 minutes then decreased over the next 25 minutes (no other details given) (HSDB 1994).

2. Distribution - Methanol distributes rapidly in dogs exposed to 4000 to 15,000 ppm for 12 hours to 5 days; the highest concentrations of the chemical were found in blood, eye fluid, bile, and urine (HSDB 1994).

3. Metabolism - Methanol is oxidized in the human liver by the enzyme alcohol dehydrogenase (Rowe and McCollister 1981). Metabolic products include formaldehyde and formic acid (HSDB 1994). The rate of metabolism for methanol (25 mg/kg/hr) is much slower than for ethanol (175 mg/kg/hr) and is independent of concentrations in the blood (HSDB 1994). Formic acid is responsible for the toxic effects of methanol (ACGIH 1991).

4. Excretion - Methanol is excreted either as parent compound in the urine or expired air, or as the formic acid metabolite in urine (Rowe and McCollister 1981; HSDB 1994). The amount of formic acid excreted varies greatly with species from 1% in rabbits to 20% in dogs; humans are intermediate (HSDB 1994). In humans, the half-life of methanol elimination in expired air after oral or dermal exposure is 1.5 hours (HSDB 1994).

B. Acute Toxicity

Acute methanol intoxication is manifested initially by signs of narcosis. This is followed by a latent period in which formic acid accumulates in the body causing metabolic acidosis. Severe abdominal, leg, and back pain occur and visual degeneration can lead to blindness.

1. Humans - Ingestion of 80 to 150 mL of methanol is usually fatal to humans (HSDB 1994). One worker died from exposure to vapor ranging from 4000 to 13,000 ppm over 12 hours (ACGIH 1991). The concentration of 4000 ppm is roughly equivalent to a total of 1140 mg/kg over the 12 hour period (see end note 2). Poisoning by nonlethal doses can be described in three stages: (1) narcotic stage similar to ethanol; (2) latent period of 10-15 hours; (3) visual disturbances and central nervous system lesions (Rowe and McCollister 1981). Visual disturbances can lead to blindness due to edema of the retina and atrophy of the optic nerve head (HSDB 1994). Third-stage CNS lesions include headache, dizziness, abdominal, back, and leg pain, delirium that can lead to coma, and nausea (HSDB 1994). Formic acid production causes severe metabolic acidosis (Rowe and McCollister 1981).

2. Animals - Oral LD50 values for methanol in animals are 0.4 g/kg in the mouse, 6.2 to 13 g/kg in the rat, 14.4 g/kg in the rabbit, and 2 to 7 g/kg in the monkey (Rowe and McCollister 1981). The LD50 for dermal application to rabbits is 20 mL/kg (approximately 16 g/kg) (Rowe and McCollister 1981). Dose-response data for inhalation vary with species, dose, and duration (8800 ppm for 8 hours to 152,800 ppm for 94 minutes). Symptoms of intoxication include incoordination, salivation, lethargy, narcosis, and death (Rowe and McCollister 1981).

C. Subchronic/Chronic Toxicity

Chronic exposure to methanol, either orally or by inhalation, causes headache, insomnia, gastrointestinal problems, and blindness in humans and hepatic and brain alterations in animals. EPA has derived an oral RfD (reference dose) (see end note 3) for methanol of 0.5 mg/kg/day, based on the absence of liver and brain effects in animals exposed by mouth to 500 mg/kg/day.

1. Humans - "Chronic" exposure to methanol vapors (no time or dose given) caused conjunctivitis, headache, giddiness, insomnia, gastric disturbances, and bilateral blindness (ACGIH 1991). Marked vision loss occurred in one worker exposed to 1200 to 8000 ppm vapor for 4 years (ACGIH 1991).

2. Animals - No effects were seen in rats given 1% (approximately 140 mg/kg/day) methanol in drinking water for 6 months (Rowe and McCollister 1981). Hepatic abnormalities (proteinic degeneration, altered RNA metabolism) occurred in rhesus monkeys given 3 to 6 g/kg for 3 to 20 weeks and in rats given 10, 100, or 500 mg/kg/day for one month (Rowe and McCollister 1981). Rabbits chronically fed methanol (no dose or time given) had increasing blood levels, brain and eye edema, and myelin

thinning (HSDB 1994). Male and female rats were gavaged with 100, 500, or 2500 mg/kg/day for 90 days (U.S. EPA 1994). Increased levels of SGPT and SAP as well as decreased brain weights were seen in both sexes at the highest dose; a no-observed-adverse effect level (NOAEL) for the study was 500 mg/kg/day. Based on these data, the U.S. EPA (1994) calculated a chronic RfD (see end note 4) for methanol of 0.5 mg/kg/day. No toxic effects were seen in dogs exposed by inhalation to either 10,000 ppm for 3 minutes, 3x/day, for 100 days or to 450 or 500 ppm, 8 hours/day for 379 days (Rowe and McCollister 1981). Ultrastructural changes were observed in the photoreceptor cells of rabbits exposed to 46.6 ppm for 6 months (Rowe and McCollister 1981). Rowe and McCollister (1981) concluded that the effects of combined oral and inhalation exposure appear to be additive. Rats exposed by inhalation to 16.8 ppm, 4 hours/day, for 6 months and administered 0.7 mg/kg/day orally had changes in blood morphology, oxidation-reduction processes, and liver function (Rowe and McCollister 1981).

D. Carcinogenicity

No information was found on the carcinogenicity of methanol in the secondary sources searched.

1. Humans - No information was found in the secondary sources searched concerning the carcinogenicity of methanol to humans.

2. Animals - No information was found in the secondary sources searched concerning the carcinogenicity of methanol to animals. The NTP has assigned a project leader for methanol and the design of the study is in progress (NTP 1994).

E. Genotoxicity

Methanol was negative for cell transformation in Syrian hamster embryo cells (clonal assay and viral enhanced), sister chromatid exchange in vitro, and for aneuploidy and chromosome aberrations in Neurospora crassa (GENETOX 1992). The micronucleus test and the assay for chromosome aberrations in mammalian polychromatic erythrocytes were inconclusive (GENETOX 1992).

F. Developmental/Reproductive Toxicity

No information was found on the developmental toxicity of methanol in humans. Methanol can cause adverse effects in the developing offspring in rats at doses that cause overt maternal intoxication.

1. Humans - No information was found in the secondary sources searched regarding the developmental or reproductive toxicity of methanol to humans. However, one of the breakdown products of the artificial sweetener asparatame is methanol. Increased blood methanol levels did not lead to increased formic acid levels in women receiving up to 200 mg/kg asparatame (no other details reported) and no evidence of fetal risk was detected (HSDB 1994).

2. Animals - Rats were exposed by inhalation, 7 hours/day, to 5000

or 10,000 ppm methanol on gestation days 1-19 or to 20,000 ppm on days 7-15. Maternal intoxication (unsteadiness) occurred at the highest dose and coincided with extra or rudimentary ribs and urinary or cardiovascular defects in the fetuses (ACGIH 1991). Male rats had significantly lowered testosterone levels after inhalation exposure to 200 ppm methanol for 6 weeks; at 10,000 ppm a change in luteinizing hormone was also observed (HSDB 1994).

G. Neurotoxicity

Methanol causes central nervous system depression in humans and animals as well as degenerative changes in the brain and visual system.

1. Humans - Methanol causes narcosis similar to ethanol intoxication and nonlethal doses can lead to blindness. Autopsy of individuals after lethal doses revealed edema and hyperemia of the brain and degeneration of the ganglion cells of the retina (Rowe and McCollister 1981).

2. Animals - Acute methanol intoxication in animals causes CNS depression as observed by narcosis, incoordination, lethargy, drowsiness, and prostration (Rowe and McCollister 1981).

V. ENVIRONMENTAL EFFECTS

A. Toxicity to Aquatic Organisms

Methanol has low acute toxicity to aquatic organisms; lethal concentrations are much greater than 100 mg/L. Ninety-six hour LC50 values for fish are 28,100 mg/L for Pimephales promelas (fathead minnow), 20,100 mg/L for Oncorhynchus mykiss (rainbow trout), and >28,000 mg/L for Alburnus alburnus (bleak) (AQUIRE 1994). Forty-eight hour LC50 values for Cyprinus carpio (common carp) and Carassius auratus (goldfish) are 28,000 mg/L and 1,700 mg/L, respectively (AQUIRE 1994). Growth inhibition occurred for 4 strains of Anabaena (blue-green algae) over a range of EC50's of 2.57-3.13% for 10-14 days (AQUIRE 1994). The LC50 for Artemia salina (brine shrimp) is >10,000 mg/L in 24 hours and that for Culex restuans (mosquito) is 20,000 mg/L in 18 hours (AQUIRE 1994).

B. Toxicity to Terrestrial Organisms

No information was found in the secondary sources searched regarding the toxicity of methanol to terrestrial organisms. However, based on the range of oral LD50's, 0.4 to 14.2 g/kg, for monkeys, rats, mice, and rabbits (Rowe and McCollister 1981), it is unlikely that methanol would be toxic to terrestrial animals at environmental levels.

C. Abiotic Effects

Methanol reacts with nitrogen dioxide in polluted atmospheres to produce methyl nitrite (HSDB 1994). According to the definition provided in the Federal Register (1992), methanol is a volatile organic compound (VOC) substance. As a VOC, methanol can contribute to the formation of photochemical smog in the presence of other VOCs.

VI. EPA/OTHER FEDERAL/OTHER GROUP ACTIVITY

The Clean Air Act Amendments of 1990 list methanol as a hazardous air pollutant. Occupational exposure to methanol is regulated by the Occupational Safety and Health Administration. The permissible exposure limit (PEL) is 200 parts per million parts of air (ppm) as an 8-hour time weighted average (29 CFR 1910.1000).

Federal agency and other group activities for methanol are summarized in Tables 3 and 4.

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

EPA OFFICE	LAW	PHONE NUMBER
Pollution Prevention & Toxics	Toxic Substances Control Act (Sec. 8E)	(202) 554-1404
	Emergency Planning and Community Right-to-Know Act (EPCRA) Regulations (Sec. 313)	(800) 535-0202
	Toxics Release Inventory data	(202) 260-1531
Air	Clean Air Act	(919) 541-0888
Solid Waste & Emergency Response	Comprehensive Environmental Response, Compensation, and Liability Act (Superfund)/ Resource Conservation and Recovery Act / EPCRA (Sec. 304/311/312)	(800) 535-0202

TABLE 4. OTHER FEDERAL OFFICE/OTHER GROUP CONTACT NUMBERS FOR INFORMATION ON METHANOL

Other Agency/Department/Group	Contact Number
Agency for Toxic Substances & Disease Registry	(404) 639-6000
American Conference of Governmental Industrial Hygienists (Recommended Exposure Limit (see end note 5): 200 ppm) (Recommended Short Term Exposure Limit (see end note 6): 250 ppm)	(513) 742-2020
Consumer Product Safety Commission	(301) 817-0994
Food & Drug Administration	(301) 443-3170
National Institute for Occupational Safety & Health (Recommended Exposure Limit (see end note 5): 200 ppm)	(800) 356-4674
Occupational Safety & Health Administration (Permissible Exposure Limit (see end note 7): 200 ppm)	Check local phone book for phone number under Department of Labor

VII. END NOTES

1. Standard Industrial Classification code is the statistical classification standard for all Federal economic statistics. The code provides a convenient way to reference economic data on industries of interest to the researcher. SIC codes presented here are not intended to be an exhaustive listing; rather, the codes listed should provide an indication of where a chemical may be most likely to be found in commerce.

2. Calculated using the factor 1.33 (Verschueren 1983) to convert 4000 ppm to 5320 mg/m3 which is multiplied by 0.214 (the 12-hour breathing rate, 15 m3 [from the occupational standard 8-hour breathing rate, 10 m3] divided by the assumed adult body weight, 70 kg) to obtain the dose in mg/kg (U.S. EPA 1988).

3. 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.

4. 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.

5. The ACGIH/NIOSH exposure limits are time-weighted average (TWA) concentrations for an 8-hour workday (ACGIH) and up to a 10-hour workday (NIOSH) for a 40-hour workweek.

6. This is a recommended 15-minute exposure limit value that should not be exceeded at any time.

7. The OSHA exposure limit is a time-weighted-average (TWA) concentration that must not be exceeded during any 8-hour workshift during a 40-hour workweek.

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APPENDIX A. SOURCES SEARCHED FOR FACT SHEET PREPARATION

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