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CHEMICAL SUMMARY FOR METHYL-TERT-BUTYL ETHER
 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 These sources include online databases, unpublished EPA information, government publications, review documents, and standard reference materials.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 in these databases and secondary sources.

I. CHEMICAL IDENTITY AND PHYSICAL/CHEMICAL PROPERTIES

The chemical identity and physical/chemical properties of methyl tertiary-butyl ether are summarized in Table 1.

TABLE 1. CHEMICAL IDENTITY AND CHEMICAL/PHYSICAL
 PROPERTIES OF METHYL-tert-BUTYL ETHER

| Characteristic/Property | Data | Reference |
|---------------------------------|--|----------------------|
| CAS No. | 1634-04-4 | |
| Common Synonyms | MTBE; 2-Methoxy- 2-methyl-propane | U.S. EPA 1993a |
| Molecular Formula | C ₅ H ₁₂ O | |
| Chemical Structure | $\begin{array}{c} \text{CH}_3 \\ \\ \text{CH}_3\text{-O-C-CH}_3 \\ \\ \text{CH}_3 \end{array}$ | |
| Physical State | Colorless liquid | U.S. EPA 1993a |
| Molecular Weight | 88.15 | Budavari et al. 1989 |
| Melting Point | -109°C | Budavari et al. 1989 |
| Boiling Point | 55.2°C | Budavari et al. 1989 |
| Water Solubility | 51.26 g/L at 25°C | U.S. EPA 1993a |
| Density | d _{20/4} , 0.7404 g/mL | Budavari et al. 1989 |
| Vapor Density (air = 1) | 3.1 | U.S. EPA 1993a |
| KOC | 12.3; 11.0 (estimated) | U.S. EPA 1993a |
| Log KOW | 1.24 | CHEMFATE 1994 |
| Vapor Pressure | 245 mm Hg at 25°C | Budavari et al. 1989 |
| Reactivity | | |
| Flash Point | Flammable | HSDB 1994 |
| Henry's Law Constant | 5.5 x 10 ⁻⁴ atm m ³ /mol at 25°C | U.S. EPA 1993a |
| Fish Bioconcentration Factor | <2 (measured); <4 (estimated) | U.S. EPA 1993a |
| Odor Threshold | 0.32-0.47 mg/m ³ | U.S. EPA 1993c |
| Conversion Factors | 1 ppm = 3.605 mg/m ³ ; 1 mg/m ³ = 0.277 ppm | U.S. EPA, 1993 |

II. PRODUCTION, USE, AND TRENDS

A. Production

There are 27 companies producing methyl-tert-butyl ether (MTBE) at

32 facilities in the United States. Table 2 lists U.S. producers, plant locations, and plant capacities. In 1992, 9.1 billion pounds of MTBE were produced in the United States. During that same year, capacity was estimated at 11.6 billion pounds.

B. Use

The largest use for MTBE is as a gasoline additive, accounting for almost all U.S. consumption. Small amounts of MTBE are used as a chemical intermediate to produce high purity isobutylene. The estimated 1993 end-use pattern for MTBE reports that 100 percent of MTBE use was in gasoline additives (produced in SIC Code 2911, used in many industries).

C. Trends

The U.S. market for MTBE is expected to grow rapidly well into the 1990s. This growth is due, in part, to Clean Air Act provisions regarding gasoline reformulation.

TABLE 2. U.S. Producers of MTBE

| Company | Plant Location(s) | Plant Capacity (in millions of pounds) |
|-------------------------------------|---------------------|---|
| Amoco | Whiting, IN | 285 |
| | Yorktown, VA | 50 |
| ARCO Chemical | Channelview, TX | 3,610 |
| | Corpus Christi, TX | 1,140 |
| ARCO Petroleum | Carson, CA | 240 |
| Ashland Oil | Catlettsburg, KY | 305 |
| Champlin Refining Co. | Corpus Christi, TX | 165 |
| Chevron | El Segundo, CA | 190 |
| | Pascagoula, MS | 200 |
| Citgo | Lake Charles, LA | 255 |
| Conoco, Inc. | Ponca City, OK | 133a |
| | Westlake, LA | 133a |
| Crown Central Petroleum | Pasadena, TX | 285 |
| Diamond Shamrock | Sunray, TX | 200 |
| Enron | LaPorte, TX | 1,425 |
| Exxon Chemical | Baytown, TX | 285 |
| Fina Oil & Chemical | Big Spring, TX | 48 |
| Global Octanes (Mitsui) | Deer Park, TX | 1,188 |
| Hill Petroleum (Phibro) | Houston, TX | 130 |
| Kerr McGee | Corpus Christi, TX | 171 |
| Lyondell Petrochemical | Channelview, TX | 285 |
| Marathon Oil (USX) | Detroit, MI | 133a |
| | Robinson, IL | 133a |
| Mark West | South Shore, KY | 162 |
| Mobil | Beaumont, TX | 240 |
| Oxychem | Chocolate Bayou, TX | 190 |
| Phillips | Sweeny, TX | 285 |
| Star Enterprises (Texaco/Aramco) | Convent, LA | 190 |
| Sun Refining & Marketing Co. | Marcus Hook, PA | 240 |
| Texaco | Port Neches, TX | 950 |
| Texas Petrochemicals Corp. | Houston, TX | 2,090 |
| Valero Refining Co. | Corpus Christi, TX | 160 |

a Estimated. Combined capacity was reported for both locations listed.

Source: Mannsville 1993.

III. ENVIRONMENTAL FATE

A. Environmental Release

Environmental releases of MTBE may occur at industrial sites involved in the manufacture of MTBE or in the blending of MTBE with gasoline; during the storage, distribution and transfer of MTBE-blended gasoline; and from spills or leaks or fugitive emissions at automotive service stations. Total annual industrial emissions of MTBE in the U.S. in 1992, as reported on the Toxics Release Inventory, were 3 million pounds: 2.8 million pounds to air, 100 thousand pounds to surface water, 68 thousand pounds to underground injection sites, and 288 lb to land (TRI92 1994). Vapor emissions of MTBE from gasoline blended with MTBE may also contribute substantially to atmospheric levels (U.S. EPA 1988a). The annual mean concentration of MTBE in the atmosphere in the United States during 1987-1988 was estimated to be <0.2 ppb (U.S. EPA 1993b). MTBE has been detected in potable well water samples in several locations; maximum reported concentrations ranged from 20 micrograms/L to more than 200 mg/L (U.S. EPA 1993b).

B. Transport

MTBE is highly volatile (vapor pressure 245 mm Hg, Henry's Law Constant 5.5×10^{-4} atm-m³/mole), and would be expected to volatilize rapidly from soil surfaces or water (U.S. EPA, 1993). Calculations based on the environmental partitioning model (ENPART) in the Graphical Exposure Modeling System (GEMS) indicate that 99.99% of MTBE will partition to air (U.S. EPA 1993a). However, MTBE leaking from underground gasoline storage tanks may not readily reach the atmosphere. With estimated organic carbon partitioning coefficients (K_{oc}) of 12.3 (derived from water solubility data) and 10.96 (derived from K_{ow} data) (U.S. EPA 1993a), MTBE is expected to be highly mobile in soils, and leaching of the chemical into groundwater is likely (U.S. EPA 1993a).

C. Transformation/Persistence

1. Air - MTBE is not expected to persist in the atmosphere because of its rapid reaction with hydroxyl radicals. The rate constant for this reaction is 2.84×10^{-12} cm³/molecule-sec at 25°C (HSDB 1994). MTBE does not absorb light of greater than 210 nm; therefore, direct photolysis by UV absorption is not expected to be environmentally significant. Atmospheric half-lives of 3.0 days in polluted air and 6.1 days in non-polluted air have been calculated (U.S. EPA 1993a).
2. Soil - MTBE is expected to volatilize rapidly from soil surfaces (U.S. EPA 1993a). MTBE released in subsoils as a result of leaks from underground storage tanks may be persistent. There is little evidence that MTBE is susceptible to either aerobic or anaerobic biodegradation (U.S. EPA 1993a).
3. Water - MTBE is expected to volatilize from surface waters (U.S. EPA 1993a). Volatilization half-lives of MTBE from streams, rivers and lakes were estimated to be 2.5 h, 9.5 h and 3,296 h (137 days), respectively (U.S. EPA 1993a). MTBE is not expected to hydrolyze, photolyze, or be adsorbed to sediments or suspended particulate matter (HSDB 1994).

4. Biota - Bioconcentration factors (BCF) of 1.5 and 1.4 were reported for Japanese carp exposed to 10 and 80 mg/L MTBE (U.S. EPA 1993a). Bioconcentration factors of 3.70 and 1.56 were estimated from regression equations based on Kow values (U.S. EPA 1993a). These BCFs indicate a low potential for bioconcentration.

IV. HEALTH EFFECTS

A. Pharmacokinetics

1. Absorption - Animal studies have shown that MTBE is rapidly absorbed following oral or inhalation exposures. Bioavailability following dermal exposure is reported to be 39% or less than that for oral exposures (U.S. EPA 1993a)
2. Distribution - Animal studies indicate that MTBE is rapidly distributed in the blood to all parts of the body including the brain. Peak blood levels occur within 15 min of i.p. injection, and highest tissue levels were reported to occur in the liver and kidneys (U.S. EPA 1993a).
3. Metabolism - The major metabolites of MTBE are tertiary butyl alcohol (TBA) and formaldehyde (U.S. EPA 1993a). The formaldehyde is likely further metabolized to formic acid and carbon dioxide, with the possible formation of methanol as well. 2-Methyl-1,2-propanediol and α -hydroxyisobutyric acid have also been identified in the urine of MTBE-exposed animals.
4. Excretion - Animal studies have shown that MTBE is rapidly excreted following oral or inhalation exposures (U.S. EPA 1993a).

Following oral exposures, MTBE is eliminated mainly in expired air (46-69%) with smaller amounts excreted in the urine (11-36%).

Following inhalation exposures, most MTBE and metabolites are excreted in the urine (53-72%) with smaller amounts in expired air (17-22%) (U.S. EPA 1993a).

B. Acute Effects

Limited information indicate that ambient levels of MTBE do not pose a health risk to healthy individuals; however, susceptible subpopulations living under unique climatic condition (i.e., subarctic) may be adversely affected by volatile emissions from MTBE-blended gasoline. Animal lethality data indicate that MTBE is low in acute toxicity. The main target organ of acutely toxic doses of MTBE is the nervous system.

1. Humans - In controlled clinical tests in which healthy individuals were exposed to 5 mg MTBE/m³ for 1 hour, no symptoms of adverse effects were observed (U.S. EPA 1993c). The concentration of 5 mg/m³ for 1 hour is roughly equivalent to a dose of 0.09 mg/kg (see end note 1). The individuals were evaluated for symptomatic responses (headaches, throat and nasal irritation, cough, and dizziness), neurobehavioral changes, upper airway inflammation, and eye inflammation. Complaints of headaches, eye irritation, nose and throat irritation, cough, nausea, dizziness, and spaciness were recorded in two cities in Alaska following the introduction of MTBE-blended gasoline during the fall of 1992 (U.S. EPA 1993c). Similar effects could not be identified in populations in New Jersey or Connecticut where MTBE-blended gasoline was also used. U.S. EPA (1993c) notes that "There is unlikely to be a substantial risk of acute health symptoms among members of the public receiving 'typical' environmental exposures under

temperate conditions (i.e., not subarctic temperature). This leaves open the question about more subtle health risks, especially among susceptible subpopulations. If acute symptoms are being caused by MTBE, they appear to be mild and transient".

Humans are acutely exposed to MTBE as part of a medical treatment to dissolve cholesterol gallstones (U.S. EPA 1994). Injection of the gall bladder with MTBE can result in nausea, vomiting, sleepiness, and minor transient mucosal damage in the gallbladder.

Intravascular hemolysis and renal failure have occurred following inadvertent extravasation of a large bolus of MTBE (U.S. EPA 1994).

2. Animals - Oral LD50 values of 1.6-3.9 g/kg have been reported for rodents (U.S. EPA 1993a). Acutely toxic oral doses can result in nervous system effects (see section G) as well as muscular weakness and inflammation of the stomach and small intestines. Inhalation LC50 values of 85-142 g/m³ have been reported in rodents (U.S. EPA 1993a). Symptoms of inhalation exposure include nervous system effects (see section G), as well as inflammation of the nasal mucosa and trachea. MTBE also causes mild skin irritation (slight erythema and edema) and moderate eye irritation (corneal opacities, chemosis and conjunctival redness) (U.S. EPA 1993a). A skin penetration LD50 of >10 mL/kg has been reported for rabbits (Clayton and Clayton 1981-82). MTBE is not expected to be a primary skin irritant (HSDB 1994).

C. Subchronic/Chronic Effects

Information on the subchronic and chronic toxicity of MTBE to humans was not found in the secondary sources searched. Laboratory rodents exposed to high doses or concentrations of MTBE exhibit blood chemistry changes and kidney abnormalities.

1. Humans - No information specific to MTBE was found in the available literature.
2. Animals - The subchronic oral toxicity of MTBE was evaluated in a 90-day bioassay in which Sprague-Dawley rats were dosed by gavage with 0, 100, 300, 900 or 1200 mg MTBE/kg/day. Animals in all treatment groups exhibited diarrhea. A dose level of 1200 mg/kg/day resulted in an increase in hyaline droplets and blockage of renal tubules with granular cysts; increased kidney and liver weights; increases in hemoglobin, hematocrit, and erythrocytes; decreases in blood glucose, blood urea nitrogen (BUN), and calcium; and elevations in cholesterol and aspartate aminotransferase (AST) (U.S. EPA 1993a). Pathological changes in the kidney were not seen at the lower dose levels; however, males and females treated with 900 mg/kg MTBE and females treated with 300 mg/kg/day exhibited increases in relative kidney weight as well as several changes in blood chemistry such as a decrease in BUN. The oral reference dose (RfD) for MTBE is currently under review by EPA (U.S. EPA 1994).

In tests in which rats were exposed to MTBE concentrations of 797, 3920, or 8043 ppm (6 h/day, 5 days/wk for 13 wk), the two highest concentrations resulted in increased liver, kidney and adrenal weights; changes in hematologic parameters (increased hemoglobin, decreased erythrocyte counts and increased reticulocyte counts); altered serum chemistry [increases in calcium and protein, and decreases in blood glucose, and AST and alanine aminotransferase (ALT) activities]; lymphoidal hyperplasia; splenic hemosiderosis;

and an increase in the size of hyaline droplets in the renal proximal tubular epithelial cells (U.S. EPA 1993a). No adverse effects were seen at 797 ppm. The concentration of 8043 ppm is roughly equivalent to 4,639 mg/kg/day (see end note 2).

Exposure of mice and rats to concentrations of 0, 403, 3023, or 7977 ppm MTBE for 6 h/day, 5 days/week, for up to 18 months (mice) or 24 months (rats) resulted in clinical signs of toxicity at the two highest exposure levels (U.S. EPA 1993a). Effects seen in rats included: increased incidences of swollen periocular tissue; decreases in absolute body weight and body weight gain; increases in absolute and relative liver weight in females; an increase in kidney weight; and an increase in the incidence of chronic nephropathy (glomerulosclerosis, tubular proteinosis, interstitial nephritis, and interstitial fibrosis). Effects seen in mice included increases in absolute and relative liver and kidney weights; increases in absolute and relative adrenal weights in males; increases in hepatocellular hypertrophy and liver masses; and a slightly increased frequency of urinary bladder dilation/distension in males. The 403 ppm exposure level was considered a NOAEL. Based on the results of the rat study, the U.S. EPA (1994) calculated a chronic RfC (reference concentration) of 3 mg/m³ for MTBE.

D. Carcinogenicity

Information on the potential carcinogenicity of MTBE in humans is lacking and the results of animal studies are still under review within EPA; however, the current unfinished assessment supports a hazard classification of "possible" human carcinogen based upon "limited" animal evidence (U.S. EPA 1993c; Anderson 1994).

1. Humans - No studies were found in the available secondary sources evaluating the potential carcinogenicity of MTBE to humans.
2. Animals - The carcinogenicity of MTBE has been evaluated in inhalation exposure studies using CD-1 mice and F344 rats (U.S. EPA 1993a). In female mice, the incidence of liver adenomas was significantly increased over control values (10/50 vs. 2/50) in animals exposed to 7973 ppm MTBE (6 h/day, 5 days/week, for 18 months). Male mice exposed to the same MTBE concentration exhibited a slight increase in the combined incidence of liver adenomas and carcinomas (16/49 vs. 12/49 in controls), but this increase was not statistically significant and was within the range of historical control values (316/891). In male rats, the incidence of renal tubular cell tumors (adenomas and carcinomas) was 8/50 for animals exposed to 3023 ppm (6 h/day, 5 days/week, for up to 97 weeks), 3/50 for animals exposed to 7977 ppm (for up to 82 weeks), but only 1/50 in the control group (statistical significance not reported). In female rats, the only reported kidney tumor was a renal cell adenoma in an animal exposed to 3023 ppm MTBE. The incidence of testicular tumors (interstitial cell adenomas) was increased above control values (41/50 in the 3023 ppm group and 47/50 in the 7977 ppm group vs. 32/50 for the controls). It was noted by the investigators that the frequency of testicular tumors in the MTBE-exposed animals was within the range (64-98%) reported for aged Fischer 344 rats, and that the frequency of these tumors in the control group was lower than historical controls from the same laboratory.

E. Genotoxicity

The genotoxicity of MTBE has been evaluated in microbial mutation

assays, a sister chromatid exchange (SCE) assay, a mouse lymphoma assay, and in a Drosophila sex-linked recessive lethal test (U.S. EPA 1993a). The only study in which MTBE gave a positive response was the mouse lymphoma forward mutation assay, and this response occurred only in the presence of a metabolic activation system.

F. Developmental/Reproductive Toxicity

Information on the developmental or reproductive toxicity of MTBE in humans was not found in the available secondary sources. In animal studies, high concentrations of MTBE produced developmental and reproductive toxicity in mice and rats (U.S. EPA 1993a).

1. Humans - No data were found in the secondary sources searched to indicate that MTBE is a developmental/reproductive toxicant in humans.
2. Animals - In tests on CD-1 mice, exposure to 14,400 mg/m³ and 28,800 mg/m³ on gestation days 6-15 resulted in reduced pup viability. An increase in the incidence of cleft palate was seen at the higher concentration. Exposure to 3,600 mg/m³ resulted in no adverse effects (EPA, 1993c). In a one-generation study on Sprague-Dawley rats, no adverse developmental effects occurred at 1,082 mg/m³, but reduced pup viability was seen at 4,470 mg/m³. In a two-generation study conducted on the same strain of rats, an exposure level of 1,453 mg/m³ caused no adverse effects; however, 10,899 mg/m³ (3019 ppm) resulted in maternal toxicity, a significant increase in dead pups on day 4, a significant reduction in litter size on days 21 and 28, and a significant reduction in mean pup weight throughout lactation (Anderson, 1994; EPA, 1993c). EPA (1993c) applied an uncertainty factors of 3 (to extrapolate from rats to humans) and 10 (for sensitive subpopulations) to the rat NOAEL of 1,453 mg/m³, to derive a human NOAEL for developmental effects of 48 mg/m³ (with uncertainty spanning at least an order of magnitude).

G. Neurotoxicity

Information on the potential neurotoxicity of MTBE to humans was not found in the secondary sources searched. Laboratory rodents exposed to high concentrations of MTBE exhibit neurotoxic effects.

1. Humans - No information available.
2. Animals - Acutely toxic oral doses can result in hypoactivity; muscular weakness; hyperpnea; prostration; lacrimation; and a general anesthetic effect at doses of \geq 1200 mg/kg (U.S. EPA 1993a). Symptoms of inhalation exposures include: hyperactivity; ataxia; loss of righting; unconsciousness; clonia; sporadic convulsive seizures. The median effective concentration (EC₅₀) for anesthesia is 200 g/m³ (55,400 ppm) in mice. Rats exposed to \geq 4000 ppm (6 h/day, 5 days/wk for 13 wk) exhibited mild neurotoxic effects (transient episodes of ataxia, reduced grip strength, and altered motor activity) (U.S. EPA 1993a).

Exposure of mice and rats to target concentrations of 0, 400, 3000, or 8000 ppm MTBE for 6 h/day, 5 days/week, for up to 18 months (mice) or 24 months (rats) resulted in several clinical signs of neurotoxicity at the two highest exposure levels. Effects seen in rats included: blepharospasm; hypoactivity; ataxia, lack of a startle reflex; salivation; and tremors. Effects seen in mice included blepharospasm, hypoactivity, ataxia, lack of a startle reflex, stereotypy, and

prostration (U.S. EPA 1993a).

V. ENVIRONMENTAL EFFECTS

A. Toxicity to Aquatic Organisms

Limited information indicate that MTBE has low acute toxicity to aquatic organisms; lethal concentrations are generally greater than 100 mg/L. Ninety-six-hour LC50 values of 706 mg/L for the fathead minnow (*Pimephales promelas*), and >1000 mg/L for the bleak *Alburnus alburnus* and for the harpacticoid copepod, *Nitocra spinipes*, have been reported (U.S. EPA 1993a). Quantitative Structure Activity Relationship (QSAR) calculations predict a fish acute LC50 of 510 mg/L; a fish chronic value (ChV) of 57 mg/L; a daphnid acute LC50 of 510 mg/L; a daphnid ChV of 17 mg/L; a green algal acute LC50 of 300 mg/L; and a green algal ChV of 18 mg/L (U.S. EPA 1993a). An LC50 of 2,500 mg/L have been reported for tadpoles of the frog *Rana temporaria*; a concentration of 200 mg/L was not lethal, and a concentration of 100 mg/L resulted in accelerated tadpole development and marked increases in body weight of tadpoles and frogs that had undergone metamorphosis (U.S. EPA 1993a).

B. Toxicity to Terrestrial Organisms

No information was found in the available secondary sources on the toxicity of MTBE to terrestrial organisms. The reported rat oral LD50 values of 1.6-3.9 g/kg and LC50 values of 85-142 g/m³ suggest that the chemical would not be acutely toxic to terrestrial animals unless present in very high concentrations.

C. Abiotic Effects

Although MTBE is added to gasoline to improve air quality by enhancing combustion and reducing emissions of carbon monoxide and benzene, emissions of other pollutants, such as formaldehyde may increase (U.S. EPA 1993c). The ozone forming potential of MTBE is estimated to be lower than that for most other non-methane components of urban air including alkenes, aldehydes, nontoluene aromatics, and ethene (U.S. EPA 1993a). According to the definition provided in the Federal Register (1992), MTBE is a volatile organic compound (VOC) substance. As a VOC, MTBE 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 MTBE as a hazardous air pollutant.

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

TABLE 3. EPA OFFICES AND CONTACT NUMBERS
FOR INFORMATION ON METHYL TERTIARY-BUTYL ETHER

| EPA OFFICE | LAW | PHONE NUMBER |
|----------------------------------|---|----------------|
| Pollution Prevention & Toxics | Toxic Substances Control Act (Sec. 4/8A/8D/8E) | (202) 554-1404 |
| | Emergency Planning and Community Right-to-Know Act (EPCRA) | |
| | Regulations (Sec. 313) | (800) 424-9346 |
| | 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) 424-9346 |
| Water | Safe Drinking Water Act | (800) 426-4791 |

TABLE 4. OTHER FEDERAL OFFICES/OTHER GROUPS
CONTACT NUMBERS FOR INFORMATION ON MTBE

| Other Agency/Department/Group | Contact Number |
|---|----------------|
| Agency of Toxic Substances & Disease Registry | (404) 639-6000 |
| American Industrial Hygiene Association Workplace Environmental Exposure Level (AIHAWHEEL) Guide (Recommended Exposure Limit (see end note 3): 100 ppm) | (703) 849-8888 |
| Consumer Product Safety Commission | (301) 504-0994 |

VII. END NOTES

1. Calculated using a factor of 0.0179 (the standard 8-hour occupational breathing rate of 10 m³ divided by 8, divided by the assumed adult body weight of 70 kg) to obtain the dose in mg/kg (U.S. EPA 1988b).
2. Calculated using the factor 3,605 (U.S. EPA 1993) to convert 8043 ppm to 28,995 mg/m³ which is multiplied by 0.16 (the 6-hour breathing rate, 0.056 m³ [standard 24-hour breathing rate, 0.223 m³] 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).
3. The AIHAWHEEL exposure limit is a time-weighted average (TWA) concentration that should not be exceeded during an 8-hour workday during a 40-hour workweek.

VIII. CITED REFERENCES

- Anderson L. 1994. Methyl Tertiary-Butyl Ether Risk Management 1 Screening Document, April 1994 Draft. Chemical Screening and Risk Assessment Division, U.S. EPA, Washington, DC.
- Budavari S, O'Neil MJ, Smith A, Heckelman PE (Eds.). 1989. The Merck Index, 11th ed. Merck & Co., Inc., Rahway, NJ, p. 951.
- CHEMFATE. 1994. Syracuse Research Corporation's Environmental Fate Data base. Syracuse Research Corporation, Syracuse, NY.
- Clayton GD, Clayton FE. 1981-1982. Patty's Industrial Hygiene and Toxicology, 3rd ed., Vol. 2C. New York: John Wiley & Sons. p. 2503.
- Federal Register. 1992. Part 51 - Requirements for Preparation, Adoption, and Submittal of Implementation Plans. Fed. Reg. 57:3945.
- HSDB. 1994. Hazardous Substances Data Bank. MEDLARS Online Information Retrieval System, National Library of Medicine. Retrieved August, 1994.
- Mannsville. 1993. Chemical Products Synopsis, MTBE. Mannsville Chemical Products Corporation. January, 1993.
- TRI92. 1994. 1992 Toxics Release Inventory. Office of Pollution

Prevention and Toxics, U.S. EPA, Washington, DC. EPA 745-R-94-001.

U.S. EPA. 1985. U.S. Environmental Protection Agency. Reference Values for Risk Assessment. Environmental Criteria and Assessment Office, U.S. EPA, Cincinnati, OH, Table 1-2.

U.S. EPA. 1988a. U.S. Environmental Protection Agency. Fuel and Fuel Additives: waiver decision. Notice. Federal Register 44:33846-33847. (cited in U.S. EPA 1993a)

U.S. EPA. 1988b. U.S. Environmental Protection Agency. Methodology for Evaluating Potential Carcinogenicity in Support of Reportable Quantity Adjustments Pursuant to CERCLA Section 102. Carcinogen Assessment Group, Office of Health and Environmental Assessment, U.S. EPA, Washington, D.C., pp. 21, 22. OHEA-C-073.

U.S. EPA. 1993a. U.S. Environmental Protection Agency. Technical Information Review. Methyl tertiary Butyl Ether (CAS No. 1634-04-4). Office of Pollution Prevention and Toxics, U.S. EPA, Washington, D.C.

U.S. EPA. 1993b. U.S. Environmental Protection Agency. Revised and Updated Drinking Water Quantification of Toxicological Effects for Methyl tert-Butyl Ether (MTBE). Final Draft. Environmental Criteria and Assessment Office, U.S. EPA, Cincinnati, OH. ECAO-CIN-DO23.

U.S. EPA. 1993c. U.S. Environmental Protection Agency. Assessment of Potential Health Risks of Gasoline Oxygenated with Methyl Tertiary Butyl Ether (MTBE). Office of Research and Development, U.S. EPA, Washington, DC. EPA/600/R-93/206.

U.S. EPA. 1994. Integrated Risk Information System (IRIS) Online. Coversheet for Methyl tertiary Butyl Ether. Office of Health and Environmental Assessment, U.S. EPA, Cincinnati, OH, Retrieved 8/2/94.

APPENDIX A. SOURCES SEARCHED FOR FACT SHEET PREPARATION

AQUIRE. 1994. Aquatic Information Retrieval online data base. Chemical Information Systems, Inc., a subsidiary of Fein-Marquart Assoc.

ATSDR. 1989-1994. Agency for Toxic Substances and Disease Registry. Toxicological Profiles. Chamblee, GA: ATSDR.

Budavari S, O'Neil MJ, Smith A, Heckelman PE (Eds.). 1989. The Merck Index, 11th ed. Rahway, N.J.: Merck & Co., Inc.

Clayton GD, Clayton FE. 1981-1982. Patty's Industrial Hygiene and Toxicology, 3rd ed., Vol. 2C. New York: John Wiley & Sons.

GENETOX. 1994. U.S. EPA GENETOX Program, computerized database.

HSDB. 1994. Hazardous Substances Data Bank. MEDLARS Online Information Retrieval System, National Library of Medicine.

IARC. 1979-1994. International Agency for Research on Cancer. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Lyon: IARC.

NIOSH (National Institute for Occupational Safety and Health). 1992. NIOSH Recommendations for Occupational Safety and Health. Compendium of Policy Documents and Statements. Cincinnati, OH: NIOSH.

NTP. 1994. National Toxicology Program. Toxicology and Carcinogenesis Studies. Tech Rep Ser.

NTP. 1994. National Toxicology Program. Management Status Report. Produced from NTP Chemtrack system. April 8, 1994. National Toxicology Program, Research Triangle Park, NC.

OSHA. 1994. Occupational Safety and Health Administration. Table Z-2. Limits for Air Contaminants.

RTECS. 1994. Registry of Toxic Effects of Chemical Substances. MEDLARS Online Information Retrieval System, National Library of Medicine.

U.S. Air Force. 1989. The Installation Restoration Toxicology Guide, Vols. 1-5. Wright-Patterson Air Force Base, OH.

U.S. EPA (U.S. Environmental Protection Agency). 1991. Table 302.4 List of Hazardous Substances and Reportable Quantities 40 CFR, part 302.4:3-271.

U.S. EPA. Most current. Drinking Water Regulations and Health Advisories. Office of Drinking Water, U.S. Environmental Protection Agency, Washington, D.C.

U.S. EPA. Most Current. Health Effects Assessment Summary Tables. Cincinnati, OH: Environmental Criteria and Assessment Office, U.S.EPA. U.S. EPA reviews such as Health and Environmental Effects Documents, Health and Environmental Effect Profiles, and Health and Environmental Assessments.

U.S. EPA. 1994. Integrated Risk Information System (IRIS) Online. Cincinnati, OH: Office of Health and Environmental Assessment.