March 31, 1987

TOLUENE

Health Advisory
Office of Drinking Water
U.S. Environmental Protection Agency

I. INTRODUCTION

The Health Advisory (HA) Program, sponsored by the Office of Drinking Water (ODW), provides information on the health effects, analytical methodology and treatment technology that would be useful in dealing with the contamination of drinking water. Health Advisories describe nonregulatory concentrations of drinking water contaminants at which adverse health effects would not be anticipated to occur over specific exposure durations. Health Advisories contain a margin of safety to protect sensitive members of the population.

Health Advisories serve as informal technical guidance to assist Federal, State and local officials responsible for protecting public health when emergency spills or contamination situations occur. They are not to be construed as legally enforceable Federal standards. The HAs are subject to change as new information becomes available.

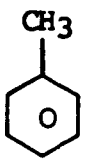
Health Advisories are developed for One-day, Ten-day, Longer-term (approximately 7 years, or 10% of an individual's lifetime) and Lifetime exposures based on data describing noncarcinogenic end points of toxicity. Health Advisories do not quantitatively incorporate any potential carcinogenic risk from such exposure. For those substances that are known or probable human carcinogens, according to the Agency classification scheme (Group A or B), Lifetime HAs are not recommended. The chemical concentration values for Group A or B carcinogens are correlated with carcinogenic risk estimates by employing a cancer potency (unit risk) value together with assumptions for lifetime exposure and the consumption of drinking water. The cancer unit risk is usually derived from the linear multistage model with 95% upper confidence limits. This provides a low-dose estimate of cancer risk to humans that is considered unlikely to pose a carcinogenic risk in excess of the stated values. Excess cancer risk estimates may also be calculated using the One-hit, Weibull, Logit or Probit models. There is no current understanding of the biological mechanisms involved in cancer to suggest that any one of these models is able to predict risk more accurately than another. Because each model is based on differing assumptions, the estimates that are derived can differ by several orders of magnitude.

This Health Advisory is based on information presented in the Office of Drinking Water's Health Effects Criteria Document (CD) for Toluene (U.S. EPA, 1985a). The HA and CD formats are similar for easy reference. Individuals desiring further information on the toxicological data base or rationale for risk characterization should consult the CD. The CD is available for review at each EPA Regional Office of Drinking Water counterpart (e.g., Water Supply Branch or Drinking Water Branch), or for a fee from the National Technical Information Service, U.S. Department of Commerce, 5285 Port Royal Rd., Springfield, VA 22161, PB # 86-117975/REB. The toll-free number is (800) 336-4700; in the Washington, D.C. area: (703) 487-4650.

II. GENERAL INFORMATION AND PROPERTIES

CAS No. 108-88-3

Structural Formula



Synonyms

- Methylbenzene, phenylmethane, toluol, methylbenzol, methacide

Uses

- Raw material in the production of benzene and other organic solvents
  Solvent (especially for paints, coatings, gums, oils and resins)
  Gasoline additive to elevate octane ratings

Properties (Amoore and Hautala, 1983; Cier, 1969; Sutton and Calder, 1975; Tute, 1971; Weast, 1977; Zoeteman et al., 1971)

Chemical Formula	C_7H_8
Molecular Weight	92.15
Physical State (room temp.)	Clear, colorless liquid
Melting Point	-94.9°C
Boiling Point	110.6°C
Vapor Pressure	28.7 mm Hg at 25°C
Specific Gravity	0.8623 at 15.6°C
Water Solubility	
Fresh Water	535 mg/L
Sea Water	379 mg/L
Log Octanol/Water Partition Coefficient	2.69
Taste Threshold (water)	0.04 mg/L; 1 mg/L
Odor Threshold (water)	0.04 mg/L; 1 mg/L
Odor Threshold (air)	0.6-140 mg/m^3
Conversion Factor	1 ppm = 3.77 mg/m^3

Occurrence

- Toluene occurs naturally as a component of petroleum oil.

- Toluene is produced in large amounts (5.1 billion lbs in 1981). Toluene also is produced indirectly in large volumes during gasoline refining and other operations. Toluene content of gasoline can be as high as several percent.

- Releases of toluene to the environment are mainly to air due to toluene's volatile nature, with smaller amounts to water and soil. Releases of toluene to water are due to spills and leaks of gasoline and other petroleum products and from the disposal of waste from paints, inks and other products containing toluene. Because of the widespread use of petroleum products, releases of toluene occur nationwide.

- Toluene degrades rapidly in air with a half life of a few days (Mabey et al., 1981). Toluene released to surface water rapidly volatilizes to air. Toluene released to the ground binds somewhat to soil and slowly migrates with ground water. Toluene is biodegraded readily in soils and surface waters. In the absence of biodegradation, toluene is expected to be stable in ground water (Marion and Malaney, 1963; Lutin et al., 1965; Price et al., 1974; Bridie et al., 1979; Patterson and Kodukala, 1981; Tabak et al., 1981).

- Toluene occurs at low levels in drinking water, food and air. Toluene occurs in both ground and surface public water supplies, with higher levels occurring in surface water supplies. Based upon EPA's Ground Water Supply Survey (U.S. EPA, 1983), approximately 1% of all ground water-derived public drinking water systems have levels greater than 0.5 ug/L. The highest level reported in ground water was 1.4 ug/L. Based upon EPA's National Screening Program Survey, approximately 3% of all surface water-derived drinking water systems are contaminated at levels higher than 0.1 ug/L. None of the systems were reported to contain levels higher than 1.4 ug/L. Toluene is found in foods as a naturally occurring compound at ppb levels and in the air of urban and suburban areas at levels of approximately 10 ppb. Toluene has been reported to occur in indoor air at levels higher than outside. Based upon the available data, the major source of toluene exposure is from air.

III. PHARMACOKINETICS

Absorption

- Studies in humans showed that toluene is absorbed quickly through the respiratory tract (Astrand et al., 1972; Astrand, 1975). Toluene was detected in arterial blood within the first 10 seconds after exposure to 100 or 200 ppm toluene (Astrand et al., 1972).

- In humans, inhalation exposure at 115 ppm (430 mg/m^3) resulted in a pulmonary absorption of 57% after 1 hour which decreased to a stable 37% of inspired dose after 2-4 hours of exposure (Nomiyama and Nomiyama, 1974).

- Absorption from the GI tract in male rats was relatively rapid, with maximal blood-toluene levels being reached within 2 hours after gastric intubation with 100 uL toluene in 400 uL peanut oil. The oil may have slowed absorption (Pyykko et al., 1977).

- Dermal absorption of aqueous toluene (180 to 600 mg/L) across human hand skin was 160 to 600 ug/cm^2/hour. Absorption was related directly to concentration (Dutkiewicz and Tyras, 1968a,b).

Distribution

- Little is known about the tissue distribution of toluene in humans. Due to its lipophilic nature and low water solubility, toluene would be expected to distribute to and accumulate in lipid tissue (U.S. EPA, 1985a).

- In male rats, tissue distribution of toluene and its metabolites is similar following inhalation of high concentrations of toluene (17,340 mg/m^3) or oral administration of a single dose of labelled toluene (100 uL in 400 uL peanut oil) (Pyykko et al., 1977; Bergman, 1979). Toluene is distributed throughout the body with greatest accumulation in lipid tissues (adipose, bone marrow). Toluene and its metabolites also were found in relatively high concentration in tissues active in its metabolism and excretion (i.e., liver and kidney).

Metabolism

- Toluene is metabolized in humans, rats and rabbits by side-chain hydroxylation to benzyl alcohol, which is conjugated with glycine to form hippuric acid (70% of the dose) and then excreted in the urine (Daley et al., 1968; Ogata et al., 1970).

- In rats dosed orally with toluene, minor amounts of toluene undergo ring hydroxylation, probably via arene oxide intermediates, to form o-cresol and p-cresol (0.04-1.0% of the dose) which are excreted in the urine as sulphate or glucuronide conjugates (Bakke and Scheline, 1970; Angerer, 1979).

Excretion

- Following oral or inhalational exposure in both humans and animals, toluene is excreted rapidly as the unchanged compound in expired air and mainly as the metabolite, hippuric acid, in the urine (Smith et al., 1954; El Masri et al., 1956; Ogata et al., 1970).

- Most of the urinary excretion of toluene occurs within 12 hours of the termination of exposure. The concentration of toluene in exhaled air of human subjects declined rapidly as soon as inhalation exposure was terminated (Astrand et al., 1972).

- The supply of glycine needed to conjugate with toluene in hippuric acid formation may be a limiting factor in the rate of toluene excretion. Riihimaki (1979) suggested that toluene at 780 ppm (2,940

mg/m^3) during light work or 270 ppm (1,010 mg/m^3) during heavy work would saturate the capacity for glycine conjugation in humans.

IV. HEALTH EFFECTS

Humans

- Exposures of humans to toluene are usually the result of inhalation of toluene vapors in experimental or occupational settings or during episodes of intentional abuse.

- Acute exposure to toluene at approximately 200 ppm (754 mg/m^3) for 8 hours caused symptoms indicating CNS toxicity (fatigue, headache, nausea, muscular weakness, confusion and incoordination (von Oettingen et al., 1942a,b; Carpenter et al., 1944). These effects generally increased in severity with increases in toluene concentration (von Oettingen et al., 1942a,b). Toluene vapor at 100 ppm for 8-hour exposures appeared to be the NOAEL for these effects (von Oettingen et al., 1942a,b).

- Subacute occupational exposure to toluene (for 1 to 3 weeks) at levels of 50 to 1500 ppm (189 to 5660 mg/m^3) resulted in symptoms similar to those seen in acute exposure studies and which were related to level of exposure (Wilson, 1943).

- Chronic exposure to toluene vapors at levels of approximately 200 to 800 ppm have been associated primarily with CNS (von Oettingen et al., 1942a,b) and, possibly, peripheral nervous system effects (Matsushita et al., 1975; Seppalainen et al., 1978). Disturbances in memory, thinking, psychomotor skills, visual accuracy and sensorimotor speed were reported in a significant number of workers exposed to 200 to 800 ppm for "many years" (Munchinger, 1964). Hanninen et al. (1976) reported many differences in performance test results between non-exposed workers and painters exposed to approximately 30.6 ppm toluene for an average of 14.8 years. Effects indicative of cerebral and cerebellar dysfunction, such as ataxia, tremors, equilibrium disorders, impaired speech, vision and hearing, and impaired memory and coordination have been reported in chronic abusers of toluene (Knox and Nelson, 1966; Boor and Hurtig, 1977; Sasa et al., 1978).

- Chronic abuse of and occupational exposures to toluene (approximately 200 to 800 ppm) for periods ranging from 2 weeks to 6 years have been associated with hepatomegaly and hepatic function changes (Greenburg et al., 1942; Grabski, 1961). Renal function also appears to be affected in chronic abusers of toluene (Kroeger et al., 1980; Moss et al., 1980).

Animals

Short-term Exposure

- The oral toxicity of toluene is relatively low, with an LD_{50} between 6.4 and 7.53 g/kg in adult rats (Wolf et al., 1956; Smyth et al.,

1969; Kimura et al., 1971). The earliest observable sign of acute oral toluene toxicity in adult rats is inhibition of the functions of the CNS, which become evident at approximately 2.0 g/kg (Kimura et al., 1971).

- The LC_{50} for inhaled toluene is 4,618 ppm (17,400 mg/m^3) after a 6-hour exposure in rats (Bonnet et al., 1982). No effects were reported after acute exposures to 620 or 1,100 ppm (2,340 or 4,150 mg/m^3) toluene, but 1250 ppm (4710 mg/m^3) affected coordination and irritated the mucous membranes in rats.

- The dermal LD_{50} in rabbits is 12.2 g/kg (Smyth et al., 1969).

Long-term Exposure

- Subchronic oral administration of toluene to female rats at 118, 354 or 590 mg/kg/day for 193 days (5 days/week for 138 total doses) resulted in no effects at any level (hematological, clinical, gross or histopathological) and a NOAEL $\geq$590 mg/kg/day (Wolf et al., 1956).

- Subchronic inhalation of toluene for 6 weeks resulted in slight pulmonary irritation in rats exposed at 200 ppm (754 mg/m^3) for 7 hours/day, 5 days/week (von Oettingen et al., 1942a). Renal effects were evident in rats treated at 600 ppm (2260 mg/m^3) for 7 hours/day, 5 days/week for 6 weeks.

- Chronic inhalation of toluene was studied in F344 rats exposed to 30, 100 or 300 ppm (113, 377 or 1,130 mg/m^3) toluene 6 hr/day, 5 days/week for 24 months (CIIT, 1980). Reduced hematocrit values were reported in females exposed to 100 and 300 ppm. Increased corpuscular hemoglobin concentration was reported in females exposed to 300 ppm.

Reproductive Effects

- Data regarding the reproductive effects of toluene have not been located.

Developmental Effects

- Based on data reported in an abstract, oral administration of 1.0 mL/kg toluene in cottonseed oil to pregnant CD-1 mice, 3 times daily on days 6 through 15 of gestation, resulted in a statistically significant increase in the incidence of cleft palate (Nawrot and Staples, 1979). Maternal toxicity was not seen after exposure to toluene but a significant increase in embryonic lethality occurred at doses of 0.3 ml/kg and up.

- Inhalation exposures to 1,000 mg/m^3 by pregnant rats for 8 hours per day on gestational days 1 through 21 resulted in a significant increase in signs of skeletal retardation but did not cause internal or external malformations (Hudak and Ungvary, 1978).

Mutagenicity

- Toluene has been tested for mutagenicity by many investigators using various assay methods (reverse mutation, mitotic gene conversion and mitotic crossing-over) and has not been demonstrated to be genotoxic or mutagenic.

Carcinogenicity

- CIIT (1980) concluded that exposures to 0, 30, 100 or 300 ppm toluene for 24 months did not produce an increased incidence of neoplastic, proliferative, inflammatory or degenerative lesions in F344 rats. However, the highest dose used did not approach the Maximum Tolerated Dose (MTD) and, therefore, it has been suggested that toluene may not have been adequately tested for carcinogenicity (Powers, 1979).

- Other studies suggest that toluene is not carcinogenic when applied topically (twice weekly applications of 0.1 ml toluene for 20 weeks) to the shaved skin of mice (Frei and Stephens, 1968).

- No evidence of a promotion effect was noted when toluene (0.1 ml) was painted on the skin of mice twice weekly for 20 weeks following initiation with 7,12-dimethyl-benz(a)anthracene (Frei and Kingsley, 1968; Frei and Stephens, 1968).

- Toluene is used extensively as a solvent for lipophilic chemicals being tested for carcinogenic potential. Negative control studies employing 100% toluene were negative.

V. QUANTIFICATION OF TOXICOLOGICAL EFFECTS

Health Advisories (HAs) are generally determined for One-day, Ten-day, Longer-term (approximately 7 years) and Lifetime exposures if adequate data are available that identify a sensitive noncarcinogenic end point of toxicity. The HAs for noncarcinogenic toxicants are derived using the following formula:

$$HA = \frac{(NOAEL \text{ or } LOAEL) \times (BW)}{(UF) \times (\_\_\_ \text{ L/day})} = \_\_\_ \text{ mg/L } (\_\_\_ \text{ ug/L})$$

where:

NOAEL or LOAEL = No- or Lowest-Observed-Adverse-Effect-Level in mg/kg bw/day.

BW = assumed body weight of a child (10 kg) or an adult (70 kg).

UF = uncertainty factor (10, 100 or 1,000), in accordance with NAS/ODW guidelines.

___ L/day = assumed daily water consumption of a child (1 L/day) or an adult (2 L/day).

One-day Health Advisory

The effects of single inhalation exposures of humans to toluene for periods up to 8 hours have been reported by several investigators (von Oettingen et al., 1942a,b; Carpenter et al., 1944; Ogata et al., 1970; Gamberale and Hultengren, 1972). Based on the consistent dose-response data from a combination of these studies, it is evident that toluene at approximately 100 ppm for up to 8 hours/day causes no apparent adverse effects in humans. Complaints of headache and drowsiness were reported by one volunteer exposed to 50 and 100 ppm, while consistent toluene-induced effects (fatigue, muscular weakness, incoordination) were evident in persons exposed to 200 ppm for 8 hours. Gamberale and Hultengren (1972) reported that a 20-minute exposure to 100 ppm toluene was a no-effect level when determined by perceptual speed and reaction time tests. At 200 ppm, toluene was noted as clearly causing toxic effects such as incoordination, exhilaration and prolonged reaction time (von Oettingen et al., 1942a,b; Carpenter et al., 1944; Ogata et al., 1970). These data substantiate the selection of 100 ppm (377 mg/m^3) toluene as the NOAEL in humans exposed for up to 8 hours.

Using a NOAEL of 100 ppm (377 mg/m^3), a One-day HA is calculated as follows:

Step 1: Determination of the Total Absorbed Dose (TAD)

$$TAD = \frac{(377\ mg/m^3)(20\ m^3/day)(0.6)(8\ hr/24\ hr)}{70\ kg} = 21.5\ mg/kg/day$$

where:

377 mg/m^3 = NOAEL (converted from 100 ppm) for absence of toxic effects in humans (von Oettingen et al., 1942a,b).

8 hours/24 hours = duration of exposure in one day.

20 m^3/day = assumed daily ventilation volume for 70 kg adult

0.6 = estimated ratio of dose absorbed (Nomiyama and Nomiyama, 1974).

70 kg = assumed body weight of an adult.

Step 2:

The One-day HA for a 10-kg child is derived from the TAD as follows:

$$\text{One-day HA} = \frac{(21.5\ mg/kg/day)(10\ kg)}{(10)\ (1\ L/day)} = 21.5\ mg/L\ (21{,}500\ ug/L)$$

where:

21.5 mg/kg/day = TAD

10 kg = assumed body weight of a child.

10 = uncertainty factor, chosen in accordance with NAS/ODW guidelines for use with a NOAEL from a human study.

1 L/day = assumed daily water consumption of a child.

Ten-day Health Advisory

No information was found in the available literature that was suitable for determination of a Ten-day HA value. It is therefore recommended that the DWEL, adjusted for a 10 kg child (3.46 mg/L), be used at this time as a conservative estimate of the Ten-day HA value.

Longer-term Health Advisory

No information was found in the available literature that was suitable for determination of the Longer-term HA values. It is therefore recommended that the DWEL, adjusted for a 10 kg child (3.46 mg/L), be used at this time as a conservative estimate of the Longer-term HA values.

Lifetime Health Advisory

The Lifetime HA represents that portion of an individual's total exposure that is attributed to drinking water and is considered protective of noncarcinogenic adverse health effects over a lifetime exposure. The Lifetime HA is derived in a three step process. Step 1 determines the Reference Dose (RfD), formerly called the Acceptable Daily Intake (ADI). The RfD is an estimate of a daily exposure to the human population that is likely to be without appreciable risk of deleterious effects over a lifetime, and is derived from the NOAEL (or LOAEL), identified from a chronic (or subchronic) study, divided by an uncertainty factor(s). From the RfD, a Drinking Water Equivalent Level (DWEL) can be determined (Step 2). A DWEL is a medium-specific (i.e., drinking water) lifetime exposure level, assuming 100% exposure from that medium, at which adverse, noncarcinogenic health effects would not be expected to occur. The DWEL is derived from the multiplication of the RfD by the assumed body weight of an adult and divided by the assumed daily water consumption of an adult. The Lifetime HA is determined in Step 3 by factoring in other sources of exposure, the relative source contribution (RSC). The RSC from drinking water is based on actual exposure data or, if data are not available, a value of 20% is assumed for synthetic organic chemicals and a value of 10% is assumed for inorganic chemicals. If the contaminant is classified as a Group A or B carcinogen, according to the Agency's classification scheme of carcinogenic potential (U.S. EPA, 1986), then caution should be exercised in assessing the risks associated with lifetime exposure to this chemical.

The study by CIIT (1980) is the most appropriate from which to derive the Lifetime Health Advisory. Rats were exposed to toluene via inhalation at 0, 113, 337 or 1130 mg/m^3 for 6 hrs/day 5 days/wk for two years. All parameters measured at the end of the study, to include clinical chemistry, hematology and urinalysis, were normal with the exception of a decreased hematocrit in females exposed at 100 and 300 ppm (377 and 1130 mg/m^3, respectively) and an increased corpuscular hemoglobin concentration in the high-dosed females.

Similar changes did not occur in the males nor were they related to any pathological findings. From these results, a NOAEL of 300 ppm (1130 mg/kg) was identified.

Using this NOAEL, the Lifetime Health Advisory is derived as follows:

Step 1: Determination of the Total Absorbed Dose (TAD)

$$TAD = \frac{(1130\ mg/m^3)\ (6\ hours/24\ hours)\ (20\ m^3/day)\ (5/7)\ (0.6)}{70\ kg} = 34.6\ mg/kg/day$$

where:

1130 mg/m^3 = NOAEL from animal data.

6 hours/24 hours = exposure duration in one day.

20 m^3/day = assumed daily respiratory volume of an adult.

5/7 = conversion of 5 day/week dosing regimen to 7 day/week continuous exposure.

0.6 = estimated ratio of dose absorbed (Nomiyama and Nomiyama, 1974).

70 kg = assumed body weight of an adult.

Step 2: Determination of the Reference Dose (RfD)

$$RfD = \frac{(34.6\ mg/kg/day)}{100} = 0.346\ mg/kg/day$$

Where:

28.8 mg/kg/day = TAD.

100 = uncertainty factor, chosen in accordance with NAS/ODW guidelines for use with a NOAEL from an animal study.

Step 3: Determination of the Drinking Water Equivalent Level (DWEL)

$$DWEL = \frac{(0.346\ mg/kg/day)\ (70\ kg)}{(2\ L/day)} = 12.1\ mg/L\ (12{,}100\ ug/L)$$

where:

0.346 mg/kg/day = RfD.

70 kg = assumed body weight of an adult.

2 L/day = assumed daily water consumption of an adult.

Step 4: Determination of the Lifetime Health Advisory

Lifetime HA = (12.1 mg/L) (20%) = 2.42 mg/L (2,420 ug/L)

where:

12.1 mg/L = DWEL.

20% = assumed relative source contribution from water.

Evaluation of Carcinogenic Potential

- IARC (1982) has not classified toluene into various categories of carcinogenic risk to humans.
- Applying the criteria described in EPA's guidelines for assessment of carcinogenic risk (U.S. EPA, 1986), toluene may be classified in Group D: Not classified. This category is for agents with inadequate animal evidence of carcinogenicity.
- The chronic (106-week) bioassay of toluene in F-344 rats of both sexes resulted in no carcinogenic effects (CIIT, 1980). Gross and microscopic examination of tissues and organs revealed no increase in neoplastic tissue or tumor masses among rats treated at 30, 100 or 300 ppm when compared with controls. This bioassay, however, could have been performed at higher exposure levels, since the highest dose administered (300 ppm) was not a Maximum Tolerated Dose (MTD).
- Prechronic carcinogenicity testing of commercial toluene administered by gavage to F344 rats and $B6C3F_1$ mice has been conducted, but a technical report on the data has not been issued (NCI, 1983). The NTP (NCI, 1983) also has started a chronic bioassay of commercial toluene in rats and mice exposed by inhalation. Testing is in progress, but neither preliminary nor final data are available. The assessment of the carcinogenic potential of toluene must await the completion of these tests.

VI. OTHER CRITERIA, GUIDANCE AND STANDARDS

- TLV = 100 ppm (375 mg/m^3); STEL = 150 ppm (560 mg/m^3) for skin (ACGIH, 1981).
- EPA's ambient water quality criterion for toluene is 14.3 mg/L (U.S. EPA, 1980).
- The EPA has proposed a Recommended Maximum Contaminant Level (RMCL) of 2.0 mg/L based upon the Adjusted Acceptable Daily Intake (AADI) of 10.1 mg/L for noncarcinogenic effects assuming 20% contribution from drinking water (U.S. EPA, 1985d).

VII. ANALYTICAL METHODS

- Analysis of toluene is by a purge-and-trap gas chromatographic procedure used for the determination of volatile aromatic and unsaturated organic compounds in water (U.S. EPA, 1985b). This method calls for the bubbling of an inert gas through the sample and trapping toluene on an adsorbant material. The adsorbant material is heated to drive off toluene onto a gas chromatographic column. The gas chromatograph is temperature programmed to separate the method analytes which are then detected by the photoionization detector. This method is applicable to the measurement of toluene over a concentration range of 0.02 to 1500 ug/L. Confirmatory analysis for toluene is by mass spectrometry (U.S. EPA, 1985c). The detection limit for confirmation by mass spectrometry is 0.2 ug/L.

VIII. TREATMENT TECHNOLOGIES

- Treatment options for removing toluene from drinking water sources include aeration and adsorption onto granular activated carbon (GAC). Conventional treatment methods have been found to be ineffective for the removal of toluene from drinking water (ESE, 1982).

- The Henry's Law Constant for toluene (288 atm at 20°C) indicates it is amenable to removal by aeration. In a pilot-scale study, a packed column aerator, operated at 50 to 90% of its flooded condition, removed toluene from contaminated water (ESE, 1982). A field study by Cummins (1985) also demonstrated the efficacy of aeration treatment.

  Water containing 62 ug/L toluene from a gasoline spill was decontaminated successfully by air stripping (air to water ration was 30:1 or greater). The process was less effective at lower air to water ratios (i.e., 8:1) but even at this ratio about 70% of the toluene was removed.

- Air stripping is an effective, simple and relatively inexpensive process for removing toluene and other volatile organics from water. However, use of this process then transfers the contaminant directly to the air stream. When considering use of air stripping as a treatment process, it is suggested that careful consideration be given to the overall environmental occurrence, fate, route of exposure and various hazards associated with the chemical.

- Carbon adsorption isotherms developed by Dobbs and Cohen (1980) showed that GAC can remove toluene from water effectively. However, with Freundlich constants of 26 for K and 0.44 for 1/n, carbon usage rates would be relatively high (U.S. EPA, 1985b). Toluene was also successfully removed from a light hydrocarbon cracking quench using GAC. The solution treated contained 8.3 mg/L toluene. Breakthrough on a 6 ft x 4 inch GAC column (Filtrasorb® 300) occurred after the processing of about 1,200 gallons. Suffet et al., as cited by ESE (1982) found that GAC (Filtrasorb® 400) adsorbed toluene from water containing a mixture of contaminants. However, in this pilot study, breakthrough occurred after 10 weeks, whereas levels of the other contaminants remained below detection for 18 weeks.

IX. REFERENCES

ACGIH. 1984. American Conference of Governmental Industrial Hygienists. Toluene. Documentation of threshold limit values for substances in workroom air. 3rd ed. Cincinnati, OH. p. 400.

Amoore, J.E., and E. Hautala. 1983. Odor as an aid to chemical safety: Odor threshold compared with threshold limit values and volatilities for 214 industrial chemicals in air and water dilution. J. Appl. Tox. 3:272-290.

Angerer, J. 1979. Occupational chronic exposure to organic solvents. VII. Metabolism of toluene in man. Int. Arch. Occup. Environ. Health. 43(1): 63-67.

Astrand, I. 1975. Uptake of solvents in the blood and tissues of man. A review. Scand. J. Work Environ. Health. 1(4):199-218.

Astrand, I., H. Ehrner-Samuel, A. Kilbom and P. Ovrum. 1972. Toluene exposure. I. Concentration in alveolar air and blood at rest and during exercise. Work Environ. Health. 72(3):119-130.

Bakke, O.H., and R.R. Scheline. 1970. Hydroxylation of aromatic hydrocarbons in the rat. Toxicol. Appl. Pharmacol. 16:691-700.

Bergman, K. 1979. Whole-body autoradiography and applied tracer techniques in distribution and elimination studies of some organic solvents. Benzene, toluene, xylene, styrene, methylene chloride, chloroform, carbon tetrachloride and trichloroethylene. Scand. J. Work Environ. Health. 5: Suppl. 1. (263 pp.).

Bonnet, P., Y. Morele, G. Raoult, D. Zissu and D. Gradiski. 1982. Determination of the median lethal concentration of the main aromatic hydrocarbons in the rats. Arch. Mal. Prof. Med. Trav. Secur. Soc. 43(4):261-265.

Boor, J.W., and H.I. Hurtig. 1977. Persistent cerebellar ataxia after exposure to toluene. Ann. Neurol. 2(5):440-442.

Bridie, A.L., et al. 1979. BOD and COD of some photochemicals. Water Research. 13:627-630.

Carpenter, C.P., C.B. Shaffer, C.S. Weil and H.F. Smyth, Jr. 1944. Studies on the inhalation of 2,3-butadiene; with a comparison of its narcotic effect with benzol, toluol and styrene, and a note on the elimination of styrene by the human. J. Ind. Hyg. Toxicol. 26:69-78.

CIIT. 1980. Chemical Industry Institute of Toxicology. A twenty-four month inhalation toxicology study in Fischer-344 rats exposed to atmospheric toluene. Executive Summary and Data Tables. October 15, 1980.

Cier, H.E. 1969. Toluene. In: Kirk-Othmer Encyclopedia of Chemical Technology, Vol. 20, 2nd ed., A. Standen, ed. John Wiley and Sons, Inc., N.Y., p. 528.

Cummins, M.D. 1985. Field evaluation of packed column air stripping. U.S. Environmental Protection Agency, Office of Drinking Water, Technical Support Division, Cincinnati, Ohio 45268.

Daley, J., D. Jerina and B. Witkop. 1968. Migration of deuterium drinking hydroxylation of aromatic substrates by liver microsomes. I. Influence of ring substituents. Arch. Biochem. Biophys. 128(2):517-527.

Dobbs, R.A., and J.M. Cohen. 1980. Carbon adsorption isotherms for toxic organics. EPA 600/8-80-023. MERL, U.S. EPA, Cincinnati, Ohio.

Dutkiewicz, T., and H. Tyras. 1968a. The quantitative estimation of toluene skin absorption in man. Arch. Gewerbepath Gewerbehyg. 24:253-257.

Dutkiewicz, T., and H. Tyras. 1968b. Skin adsorption of toluene, styrene and xylene by man. Br. J. Med. 25(3):243.

El Masri, A.M., J.N. Smith and R.T. Williams. 1956. Studies in detoxication. 69. The metabolism of alkylbenzenes: n-propylbenzene and n-butylbenzene with further observations on ethylbenzene. Biochem. J. 64:50-56.

ESE. 1982. Environmental Science and Engineering, Inc. ESE review of organic contaminants in ODW data base for summary of all available treatment techniques: Toluene. Office of Drinking Water, U.S. Environmental Protection Agency. EPA No. 68-01-6494.

Frei, J.V., and W.F. Kingsley. 1968. Observations on chemically induced regressing tumors of mouse epidermis. J. Natl. Cancer Inst. 41:1307-1313.

Frei, J.V., and P. Stephens. 1968. The correlation of promotion of tumor growth and of induction of hyperplasia in epidermal two-stage carcinogenesis. Br. J. Cancer. 22:83-92.

Gamberale, F., and M. Hultengren. 1972. Toluene exposure. II. Psychophysiological functions. Work Environ. Health. 9(3):131-139. (CA 79:950-1973).

Grabski, D.A. 1961. Toluene sniffing producing cerebellar degeneration. Am. J. Psychiatry. 118:461-462.

Greenburg, L., M.R. Mayers, H. Heimann and S. Moskowitz. 1942. The effects of exposure to toluene in industry. J. Am. Med. Assoc. 118:573-578.

Hanninen, H., L. Eskelinen, K. Husman and M. Nurmineen. 1976. Behavioral effects of long-term exposure to a mixture of organic solvents. Scand. J. Work Environ. Health. 2(4):240-255.

Hudak, A., and G. Ungvary. 1978. Embryotoxic effects of benzene and its methyl derivatives: toluene, xylene. Toxicology. 11:55-63.

IARC. 1982. International Agency for Research on Cancer. IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Supplement 4. Lyon, France.

Kimura, E.T., D.M. Ebert and P.W. Dodge. 1971. Acute toxicity and limits of solvent residue for sixteen organic solvents. Toxicol. Appl. Pharmacol. 19(4):699-704.

Knox, J.W., and J.R. Nelson. 1966. Permanent encephalopathy from toluene inhalation. N. Engl. J. Med. 275:1494-1496.

Kroeger, R.M., R.J. Moore, T.H. Lehman, J.D. Giesy and E.D. Skeeters. 1980. Recurrent urinary calculi associated with toluene sniffing. J. Urol. 123(1):89-91.

Lutin, P.A., J.J. Cibulka and G.W. Malaney. 1965. Oxidation of selected carcinogenic compounds by activated sludge. Purdue Univ., Eng. Bull. Ext. Ser. 118:131-145.

Mabey, W.R., J.H. Smith, R.T. Podoll et al. 1981. Aquatic fate process data for organic priority pollutants: Final draft report. U.S. EPA, Washington, D.C. EPA 440/4-81-014.

Marion, C.V., and G.W. Malaney. 1963. Ability of activated sludge microorganisms to oxidize aromatic organic compounds. Proc. Indus. Waste Conf. 18:297-308. (CA 62:1437a, 1965)

Matsushita, T., Y. Arimatsu, A. Ueda, K. Satoh and S. Nomura. 1975. Hematological and neuro-muscular response of workers exposed to low concentration of toluene vapor. Ind. Health. 13:115-121.

Moss, A.H., P.A. Gabow, W.D. Kaehny, S.I. Goodman and L.L. Haut. 1980. Fanconi's syndrome and distal renal tubular acidosis after glue sniffing. Ann. Intern. Med. 92:69-70.

Munchinger, R. 1964. Der nachweis central nervoser storungen bei losungsmitt el-exponierten arbeitern. Excerpta Medica Series, Madrid; 16-21. 2(62):687-689. (Ger.)

Nawrot, P.S., and R.E. Staples. 1979. Embryo-fetal toxicity and teratogenicity of benzene and toluene in the mouse. Teratology. 19:41A. (Abst.)

NCI. 1983. National Cancer Institute. National Toxicology Program/Carcinogenesis Testing Program. Chemicals on Standard Protocol: Management Status, June 15. Tech. Info. Sec. CTP/NTP. Bethesda, Md.

Nomiyama, K., and H. Nomiyama. 1974. Respiratory retention, uptake and excretion of organic solvents in man. Benzene, toluene, n-hexane, trichloroethylene, acetone, ethyl acetate and ethyl alcohol. Int. Arch. Arbeitsmed. 32(1-2):75-83.

Ogata, M., K. Tomokuni and Y. Takatsuka. 1970. Urinary excretion of hippuric acid and m- or p-methylhippuric acid in the urine of persons exposed to vapours of toluene and m- or p-xylene as a test of exposure. Br. J. Ind. Med. 27(1):43-50.

Patterson, J.W., and P.S. Kodukala. 1981. Biodegradation of hazardous organic pollutants. Chem. Eng. Prog. 77(4):48-55.

Powers, M.B. 1979. Chemical selection meetings on toluene. Memorandum for the record from the NTP Chemical Selection Group, Toxicology Branch, CGT, DCCP, National Institute, Washington, D.C., May 25.

Price, K.S., G.T. Waggy and R.A. Conway. 1974. Brine shrimp bioassay and seawater BOD of petrochemicals. J. Water Pollut. Control Fed. 46(1):63-77.

Pyykko, K., H. Tahti and H. Vapaatalo. 1977. Toluene concentrations in various tissues of rats after inhalation and oral administration. Arch. Toxicol. 38:169-176.

Riihimaki, V. 1979. Conjugation and urinary excretion of toluene and m-xylene matabolites in a man. Scand. J. Work Environ. Health. 4(1):135-142.

Sasa, M., S. Igarashi, T. Miyazaki, K. Miyazaki, S. Nakano and I. Matsuoka. 1978. Equilibrium disorders with diffuse brain atrophy in long-term toluene sniffing. Arch. Oto-Rhino-Laryngol. 221(3):163-169.

Seppalainen, A.M., K. Husman and C. Martenson. 1978. Neurophysiological effects of long-term exposure to a mixture of organic solvents. Scand. J. Work Environ. Health. 4(4):304-314.

Smith, J.N., R.H. Smithies and R.T. Williams. 1954. Studies in detoxication. 55. The metabolism of alkylbenzenes: (a) Glucuronic acid excretion following the administration of alkylbenzenes: (b) Elimination of toluene in the expired air of rabbits. Biochem. J. 56:317-320.

Smyth, H.F., Jr., C.P. Carpenter, C.S. Weil, U.C. Pozzani, J.A. Striegel, and J.S. Nycum. 1969. Range-finding toxicity data. List. VII. Am. Ind. Hyg. Assoc. J. 30(5):470-476.

Sutton, C., and J.A. Calder. 1975. Solubility of alkylbenzenes in distilled water and seawater at 25°C. J. Chem. Eng. Data. 2(3):320-322. (CA 83:104181q, 1975)

Tabak, H.H., S.A. Quave, C.I. Mashni and E.F. Barth. 1981. Biodegradability studies with organic priority pollutants compounds. J. Water Pollut. Control Fed. 53:1503-1518.

Tute, M.S. 1971. Principles and practice of Hansch analysis: A guide to structure-activity correlation for the medicinal chemist. Adv. Drug. Res. 5:1-77.

U.S. EPA. 1980. United States Environmental Protection Agency. Water quality criteria documents; availability. Federal Register 45(231): 79318-79379.

U.S. EPA. 1983. U.S. Environmental Protection Agency. Ground Water Supply Survey. Computer data file provided by Office of Drinking Water, Technical Support Division, U.S. EPA, Cincinnati, OH.

U.S. EPA. 1985a. U.S. Environmental Protection Agency. Drinking water criteria document for toluene (Final Draft). March, 1985.

U.S. EPA. 1985b. United States Environmental Protection Agency. Method 503.1. Volatile aromatic and unsaturated organic compounds in water by purge and trap gas chromatography. Environmental Monitoring and Support Laboratory, Cincinnati, Ohio 45268.

U.S. EPA. 1985c. U.S. Environmental Protection Agency. Method 524.1. Volatile organic compounds in water by purge and trap gas chromatography/mass spectrometry. Environmental Monitoring and Support Laboratory, Cincinnati, Ohio 45268.

U.S. EPA. 1985d. U.S. Environmental Protection Agency. National primary drinking water regulations; Synthetic organic chemicals, inorganic chemicals and microorganisms; Proposed Rule. Federal Register. 50(219):46936-47022. November 13.

U.S. EPA. 1986. U.S. Environmental Protection Agency. Guidelines for carcinogen risk assessment. Federal Register 51(185):33992-34003. September 24.

von Oettingen, W.F., P.A. Neal, D.D. Donahue, et al. 1942a. The toxicity and potential dangers of toluene, with special reference to its maximal permissible concentration. U.S. Public Health Service Pub., Bull. No. 279. p. 50.

von Oettingen, W.F., P.A. Neal, and D.D. Donahue. 1942b. The toxicity and potential dangers of toluene -- Preliminary report. J. Am. Med. Assoc. 118:579-584.

Weast, R.C., ed. 1977. CRC handbook of chemistry and physics, 58th ed. Chemical Rubber Co., Cleveland, OH.

Wilson, R.H. 1943. Toluene poisoning. JAMA. 123:1106-1108.

Wolf, M.A., V.K. Rowe, D.D. McCollister, R.C. Hollingsworth and F. Oyen. 1956. Toxicological studies of certain alkylated benzenes and benzene. Arch. Ind. Health. 14:387-398.

Zoeteman, B.C.J., A.J.A. Kraayeveld and C.J. Piet. 1971. Oil pollution and drinking water odors. Water 4(16):367-371.