

Appendix B
Section K

KEROSENE
HEALTH-BASED MAXIMUM CONTAMINANT LEVEL
SUPPORT DOCUMENT

Office of Science and Research
New Jersey Department of Environmental Protection

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EXECUTIVE SUMMARY

Kerosene, a product of crude oil refining, is used as a jet fuel and for home heating. Because of its multichemical composition, kerosene's environmental and metabolic fates vary with its individual components. Its taste threshold is 100 ug/L. Short term toxicity is characterized by chemical pneumonitis followed by bacterial infection. Chronic toxicity of kerosene is not well defined. Therefore, a risk assessment based on toxicity of kerosene was not done, instead it is recommended that kerosene be regulated on the basis of its most toxic component, benzene and its most prevalent component, naphthalene.

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BACKGROUND INFORMATION AND PROPERTIES

Chemical Properties

Chemical Name	Kerosene
Synonyms	Fuel oil No. 1, Range oil
CAS Number	8008-20-6
Boiling point	200-260 °C
Physical state	liquid at room temperature, slightly brown
Specific gravity, density	less than 0.80 at 15 °C
Water solubility	varies with the individual components, not significantly soluble
Odor threshold, air	1 ppm
Taste threshold	0.1 mg/L (Stofen, 1973)
Conversion	1 ppm = 7.14 mg/m ³

Kerosene is a mixture of aliphatic and aromatic hydrocarbons and may contain sulfur and mercaptans. Composition varies by source of origin but generally the percentage of all components is as follows: 25% normal paraffins, 12% branched paraffins, 30% monocycloparaffins, 12% dicycloparaffins, 1% tricycloparaffins, 16% mononuclear aromatics, and 5% dinuclear aromatics. Paraffins consist of C10-C16 carbon lengths. The total aromatics may vary from 5-20% (NIOSH, 1977).

Kerosene can also be treated by fuming sulfuric acid to produce deodorized kerosene, Diobase (Merck, 1976).

Production and Use

Kerosene is prepared by the fractional distillation of crude petroleum. The primary use is for jet fuel (83%) and range oil (13%) for space heating. Production and use of kerosene has been declining in recent years and only 55,673,000 barrels of kerosene were produced in 1975 (NIOSH, 1977).

Deodorized kerosene is used primarily as a solvent for insect sprays.

Guidelines, Regulations, and Standards

The Office of Drinking Water, U.S.EPA has recommended that the taste threshold level for kerosene, 0.1 mg/L should be used as the action level for transient exposures (U.S.EPA, 1980). Levels of kerosene below 0.1 mg/L would then be acceptable in the event of emergency. In addition, this office recommended that 0.35 mg/L of benzene and 25 ug/L for total polynuclear aromatic hydrocarbons be used as the controlling factors for the amount of kerosene in drinking water.

ENVIRONMENTAL EXPOSURE LEVELS

Fate and Transport

The environmental fate and distribution of kerosene depends on the mobility of its components. The U.S. EPA (Coleman, 1984) has provided the following analysis of the component distribution of kerosene before and after mixing in tap water and 17 hrs later (approximately 0.7% of kerosene is soluble in water, (w/w basis)):

Table I

Kerosene and its water soluble fraction^a

Chemical class	original product	water soluble	fraction
		0.5 hr	17 hrs
Alkanes & Cycloalkanes	68.6	4.5	0.5
Benzene & Substituted Benzenes	13.7	63.5	53.2
Naphthalene & Substituted Naphthalenes	5.7	29.5	44.8

^a estimated weight percent

(adapted from Coleman, 1984)

The aromatic fraction is more water soluble than the aliphatics and over 17 hrs, this differential increases, especially with the naphthalene fraction.

Ambient Levels

No surveys of ground water identify a kerosene fraction. Approximately 0.7% of kerosene partitions into the aqueous phase (Coleman et al, 1984). The soluble components are aromatics which belong to the following groups: substituted benzenes, naphthalene, and substituted naphthalenes. The single most predominant component is naphthalene (Coleman, 1985).

The concentration of naphthalene in the drinking water supplies of several major U.S. cities ranges from nondetectable to 5 ppb (Coleman, 1984). Ground water from Hoe Creek, N.Y. contained 380-1800 ppb, probably emanating from an underground coal gasification site. Naphthalene (Stuermer, 1982) was found in the aquifer of an English town in amounts of 150-1000 ppb. The concentration of naphthalene in the aquifer was inversely proportional to the distance from petrol storage tanks.

METABOLISM AND PHARMACOKINETICS

The absorption, metabolism, and excretion properties of kerosene are dependent upon its components. Generally, the aromatic fractions are more absorbable and metabolized better than the aliphatic fractions.

Human Exposure and Body Burden

Because of the continued heavy use of petroleum, kerosene as well as other fuels may pose a human or environmental hazard by leakage or accidental spills. Petroleum fractions can seep into the aquifer and be found in ground water.

Persons working with kerosene, such as refinery workers, distributors, and those using kerosene as a fuel, will have a higher degree of exposure than the general population.

HEALTH EFFECTS

Overview

The toxicity of kerosene to humans has been characterized by central nervous system depression, dermatitis, bone marrow depression, and pneumonia. The pneumonia is the result of aspiration of kerosene into the lungs from ingestion followed by vomiting. The effects of kerosene exposure on animals are rapid respiration, convulsions, pneumonia and death. No chronic kerosene ingestion studies have been found. A two-year dermal study of JP-5 Jet fuel (similar composition to kerosene) was done by the National Toxicology Program (1986). No evidence of carcinogenicity was reported.

Human

In several cases of acute and chronic exposure to kerosene, both in percutaneous and ingestion modes, aplastic anemia was reported. Children exposed to kerosene while playing with kerosene pumps and cans contracted severe dermatitis. The prolonged skin contact with kerosene led to blistering and pus formation (NIOSH, 1977).

When children accidentally ingested kerosene, they exhibited nausea, vomiting, drowsiness, stupor and occasionally, convulsions. These patients remained feverish for several days. Diarrhea and pneumonitis were often present. Severe or untreated cases developed confluent pneumonia, cardiac dilation, hepatosplenomegaly, and cardiac failure (NIOSH, 1977).

It is estimated that ingestion of 2.5 mL could cause fatal pneumonia (Arena, 1979).

Hickel et al. (1963) (as cited by NIOSH, 1977) reported three occurrences of bone marrow depression from dermal application or ingestion of kerosene.

Epidemiologic studies of refined petroleum solvents (including kerosene) indicate dermal, nose and throat irritation. Michael et al. (1975) (as cited by NIOSH, 1977) studied the health of rubber workers exposed to solvents (not specified). A positive association was shown between solvent exposure and lymphatic leukemia. The authors did not attribute the incidence of this leukemia to benzene exposure because leukemias associated with benzene toxicity are usually of the hemocytoblastic or myeloblastic type.

Animal

Acute. Kerosene was given to Swiss Albino mice in a single, oral 1 mL dose (Narasimhan and Ganla, 1967). The mice became drowsy 12-15 minutes later and had difficulty breathing with rapid respiration. If a second dose was given, all died in 8-10 hrs, otherwise they recovered in 4 hrs. On gross examination, kidneys and livers had cloudy degeneration and vacuolation but, histologically lungs appeared normal.

In the same study (Narasimham and Ganla, 1967) 16 dogs were given 50 mL/kg of kerosene by intraperitoneal injection. The dogs became drowsy, had labored respiration, and difficulty breathing; eventually all of the dogs died. Rabbits orally administered 70 mL/kg of body weight also had difficulty breathing, then went into convulsions and died.

Wolfsdorf and Kundig (1972) examined the effects of kerosene on vervet monkeys to determine whether lung effects were the result of absorption and excretion of the solvent or as the result of aspiration. The monkeys were divided into three groups, one of which had a tracheostomy. One group received 45 mL/kg body weight via nasogastric tube. The group with the tracheostomy also received 45 mL/kg via nasogastric tube. Another group received kerosene at a dose of 1.0 mL i.v. or 0.2 mL in 5 mL of normal saline, endotracheally. Only the group with the tracheostomy had no lung lesions. The authors therefore concluded that the effect was due to aspiration.

Volkova et al. (1969) described the effects of various types of kerosene (lamp fuel) on rats, mice, rabbits, and cats. The animals were exposed to aerosol concentrations of 500, 1,000, 2,500, or 12,000 mg/m³ for 2 hrs per day for either 1 day or 2-4 weeks. Lamp fuel kerosene, which had the highest aromatic content, caused tracheitis, bronchitis, leukocytosis and an increase in the erythrocyte sedimentation rate after exposure to 500 mg/m³. An aerosol of the more purified lamp fuel at 500 mg/m³ failed to induce a detectable toxic effect. These results suggest that purified (less aromatic in content) kerosene was less toxic than the unpurified. It was impossible, however, to correlate aromatic content with toxicity because no compositional information was given.

Chronic. Separate groups containing 25 male rats and 4 male beagles were exposed to deodorized kerosene at mean airborne concentrations of 20, 48, or 100 mg/m³ or solvent-free air for 6 hours per day 5 days per week for 67 days (Carpenter et al., 1978). The animals were examined for body weight change, and blood and urine analyses were performed. An elevated serum alkaline phosphatase at 100 mg/m³ at 8 weeks was the only abnormal blood chemistry change in rats. Two rats died from bronchopneumonia. Dogs exposed to 20 mg/m³ displayed a slight increase in body weight after 13 weeks. Occasional lesions were seen in the organs of both control and exposed dogs, but the authors did not attribute them to deodorized kerosene exposure.

Starek and Kaminsky (1981) exposed 42 Wistar rats to 75 and 300 mg/m³ of deodorized kerosene 6 days per week for 14 weeks. They found morphologic and cytoplasmic changes in the lungs. In addition, variations in enzyme activity (decrease in succinic dehydrogenase) were found, along with metabolic acidosis at 75 mg/m³ and 300 mg/m³.

Behavioral and Central Nervous System

Pilots and aircraft factory workers exposed to jet fuels reported that they experienced dizziness, headache and mild neurological symptoms (polyneuropathy). In addition, surveys of workers exposed chronically to kerosene showed that they had signs of neurasthenic neurosis, functional neurosis and mild neuropathy (NIOSH, 1977).

Carcinogenicity

No reports that have been found that investigate the carcinogenic potential of heating-fuel grade kerosene. A 2 year carcinogenicity/toxicology study on marine diesel fuel and JP-5 navy fuel (similar to kerosene) was conducted by the National Toxicology Program (NTP) on the skin of 49 or 50 female B6C3F3 mice. Doses selected for the 2-year studies did not cause deaths, decrease body weight gain, or produce excessive dermatitis in 14-day or 13-week studies. The doses of 0, 250, or 500 mg/kg were administered in an acetone vehicle in a dose volume of 0.1 mL. There was a marked increase in chronic dermatitis in the group that received the JP-5 navy fuel or marine diesel fuel. There was equivocal evidence of carcinogenicity reported for the diesel fuel, but no evidence of carcinogenicity for the JP-5 navy fuel.

Certain components of kerosene are known to be carcinogenic, i.e., benzene.

QUANTITATIVE RISK ASSESSMENT

Studies Useful for Risk Assessment

There are no adequate toxicity studies available that could be used to develop a risk assessment for kerosene in drinking water. The most

appropriate studies for risk assessment would be those that evaluate the toxicity of chronic ingestion of kerosene, but these are not available. Studies using deodorized kerosene have been judged unsuitable for risk assessment because the deodorized form has a negligible portion of aromatic hydrocarbons. The aliphatic and naphthenic portions of kerosene are virtually insoluble in water and are known to be less toxic than the aromatics.

Of the 0.7% of kerosene that can be dissolved in tap water, this fraction consists almost entirely of substituted benzenes, naphthalene and substituted naphthalenes (Coleman et al, 1984). Therefore, any risk assessment for kerosene in drinking water must address the risk of exposure to the aromatic fraction.

Assumptions and Uncertainty

Benzene and benzo(a)pyrene are the components of kerosene which were proposed by the Office of Drinking Water, U.S. EPA (1980) as the controlling factors for the amount of kerosene in drinking water. Benzene has been used to regulate petroleum fuel contamination for two reasons: it is the most well-known toxic entity found in petroleum and it is probably the most water soluble of the petroleum hydrocarbons. The amount of benzene in kerosene would be expected to be minimal since most of the benzene is extracted into a higher boiling fraction in crude petroleum refining. No information was obtained on the benzo(a)pyrene content of kerosene.

Nevertheless, naphthalene is the most predominant single aromatic species in kerosene and its water soluble fraction (Coleman, 1985). The acute toxicity of naphthalene is well known but very little is known about its chronic toxicity or carcinogenicity. There are limited studies available at present that could be used for a risk assessment of naphthalene in drinking water. A chronic naphthalene exposure study is currently being evaluated by the National Toxicology Program.

Conclusion, and Recommendations

To regulate a safe concentration of kerosene in drinking water, a recommended health based maximum contaminant level (MCL) should be determined on the basis of the most toxic and abundant components of kerosene. An MCL has been recommended for benzene, however a level for naphthalene still needs to be developed. Therefore it is recommended that naphthalene be evaluated under the 2B program.

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