TOXICOLOGICAL PROFILE FOR
FUEL OILS

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

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DISCLAIMER

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UPDATE STATEMENT

A Toxicological Profile for Fuel Oils was released on November 1993. This edition supersedes any previously released draft or final profile.

Toxicological profiles are revised and republished as necessary, but no less than once every three years. For information regarding the update status of previously released profiles, contact ATSDR at:

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FOREWORD

This toxicological profile is prepared in accordance with guidelines developed by ATSDR and the Environmental Protection Agency (EPA) and in support of Department of Defense information needs. The original guidelines were published in the Federal Register on April 17, 1987. Each profile will be revised and republished as necessary.

The ATSDR toxicological profile succinctly characterizes the toxicologic and adverse health effects information for the hazardous substance being described. Each profile identifies and reviews the key literature (that has been peer-reviewed) that describes a hazardous substance's toxicologic properties. Other pertinent literature is also presented, but described in less detail than the key studies. The profile is not intended to be an exhaustive document; however, more comprehensive sources of specialty information are referenced.

Each toxicological profile begins with a public health statement, which describes in nontechnical language a substance's relevant toxicological properties. Following the public health statement is information concerning levels of significant human exposure and, when known, significant health effects. The adequacy of information to determine a substance's health effects is described in a health effects summary. Data needs that are significant to protect public health will be identified by ATSDR and the EPA. The focus of the profiles is on health and toxicologic information; therefore, we have included this information in the beginning of the document.

Each profile must include the following:

(A) The examination, summary, and interpretation of available toxicologic information and epidemiologic evaluations on a hazardous substance in order to ascertain the levels of significant human exposure for the substance and the associated acute, subacute, and chronic health effects.

(B) A determination of whether adequate information on the health effects of each substance is available or in the process of development to determine levels of exposure that present a significant risk to human health of acute, subacute, and chronic health effects.

(C) When appropriate, identification of toxicologic testing needed to identify the types or levels of exposure that might present significant risk of adverse health effects in humans.

The principal audiences for the toxicological profiles are health professionals at the federal, state, and local levels, interested private sector organizations and groups, and members of the public.

The toxicological profiles are developed in response to the Superfund Amendments and Reauthorization Act (SARA) of 1986 (Public Law 99-499) which amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or Superfund). Section 211 of SARA also amended Title 10 of the U. S. Code, creating the Defense Environmental Restoration Program. Section 2704(a) of Title 10 of the U. S. Code directs the Secretary of Defense to notify the Secretary of Health and Human Services of not less than 25 of the most commonly found unregulated hazardous substances at defense facilities.

Section 2704(b) of Title 10 of the U. S. Code directs the Administrator of the Agency for Toxic Substances and Disease Registry (ATSDR) to prepare a toxicological profile for each substance on the list provided by the Secretary of Defense under subsection (b).

Foreword

This profile reflects our assessment of all relevant toxicologic testing and information that has been peer reviewed. It has been reviewed by scientists from ATSDR, the Centers for Disease Control and Prevention (CDC), and other federal agencies. It has also been reviewed by a panel of nongovernment peer reviewers and was made available for public review. Final responsibility for the contents and views expressed in this toxicological profile resides with ATSDR.

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THE PROFILE HAS UNDERGONE THE FOLLOWING ATSDR INTERNAL REVIEWS:

1.   Green Border Review. Green Border review assures the consistency with ATSDR policy.

2.   Health Effects Review. The Health Effects Review Committee examines the health effects chapter of each profile for consistency and accuracy in interpreting health effects and classifying endpoints.

3.   Minimal Risk Level Review. The Minimal Risk Level Workgroup considers issues relevant to substance-specific minimal risk levels (MRLs), reviews the health effects database of each profile, and makes recommendations for derivation of MRLs.

4.   Quality Assurance Review. The Quality Assurance Branch of the Division of Toxicology assures that consistency across profiles is maintained, identifies any significant problems in format or content, and establishes that Guidance has been followed.

PEER REVIEW

A peer review panel was assembled for fuel oils. The panel consisted of the following members:

1.   Dr. Rick Cardwell, Head of Toxicology, Parametrix, Inc., Kirkland, Washington

2.   Dr. Charles Ward, Private Consultant, Pittsburgh, Pennsylvania

3.   Dr. Laurence M. Holland, Laboratory Associate, Los Alamos National Laboratory, Los Alamos, New Mexico

These experts collectively have knowledge of fuel oils' physical and chemical properties, toxico-kinetics, key health end points, mechanisms of action, human and animal exposure, and quantification of risk to humans. All reviewers were selected in conformity with the conditions for peer review specified in Section 104(i)(13) of the Comprehensive Environmental Response, Compensation, and Liability Act, as amended.

Scientists from the Agency for Toxic Substances and Disease Registry (ATSDR) have reviewed the peer reviewers' comments and determined which comments will be included in the profile. A listing of the peer reviewers' comments not incorporated in the profile, with a brief explanation of the rationale for their exclusion, exists as part of the administrative record for this compound. A list of databases reviewed and a list of unpublished documents cited are also included in the administrative record.

The citation of the peer review panel should not be understood to imply its approval of the profile's final content. The responsibility for the content of this profile lies with the ATSDR.

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1. PUBLIC HEALTH STATEMENT

This Statement was prepared to give you information about fuel oils and to emphasize the human health effects that may result from exposure to them. The Environmental Protection Agency (EPA) has identified 1,397 sites on its National Priorities List (NPL). Fuel oils have been found in 2% (26 out of the 1,397) of the NPL sites. However, we do not know how many of the 1,397 NPL sites have been evaluated for fuel oils. As EPA evaluates more sites, the number of sites at which fuel oils are found may change. This information is important for you to know because fuel oils may cause harmful health effects and because these sites are potential or actual sources of human exposure to fuel oils.

When a chemical is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to hazardous chemicals such as fuel oils, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, lifestyle, and state of health.

1.1 WHAT ARE FUEL OILS?

Fuel oils are petroleum products that are used in many types of engines, lamps, heaters, furnaces, stoves, and as solvents. Fuel oils come from crude petroleum and are refined to

meet specifications for each use. Fuel oils are mixtures of aliphatic (open chain and cyclic compounds that are similar to open chain compounds) and aromatic (benzene and compounds similar to benzene) petroleum hydrocarbons. In addition, they may contain small amounts of nitrogen, sulfur, and other elements as additives. The exact chemical composition (i.e., precise percentage of each constituent) of each of the fuel oils discussed in this profile may vary somewhat, depending on the source and other factors. Fuel oils are distinguished from each other primarily by their boiling point ranges, chemical additives, and uses. In this profile, six fuel oils are discussed. The fuel oils of interest and common synonyms follow:

- fuel oil no. 1 (the most widely used fuel oil)
    - kerosene
    - straight-run kerosene
    - kerosene
    - range oil
    - Deobase (the trade name of a clear, white, deodorized kerosene)
    - coal oil
    - JP-5 (jet fuel)
- fuel oil no. 1-D
    - diesel fuel
    - diesel fuel oil no. 1
- fuel oil no. 2
    - home heating oil
    - gas oil
    - no. 2 burner oil
- fuel oil no. 2-D
    - diesel fuel oil no. 2
    - diesel fuel no. 2
    - diesel oil no. 2
    - no. 2 diesel

    - fuel oil no. 4

        - diesel fuel oil no. 4

        - heavy residual fuel oil

        - marine diesel fuel

        - residual fuel oil no. 4

    - fuel oil UNSP (which is not referred to by any synonyms)

In this profile, a fuel oil is referred to by the name used in the cited study. That is, if one study identifies a fuel oil as fuel oil no. 1, and another study identifies the same fuel oil as kerosene, the names "fuel oil no. 1" and "kerosene" will be used, respectively. All fuel oils are liquids at room temperature, although they can evaporate. The rates at which the various fuel oils will evaporate is dependent on the temperature and the composition of the individual fuel oil. Most fuel oils are yellowish to light brown in color. They generally have a kerosene-like odor, are flammable, and burn at temperatures between 177°C and 329°C.

In this profile, fuel oils are discussed together because of the similarities in their chemical and physical properties. More information on the chemical and physical properties of fuel oils is provided in Chapter 3. More information on the production and use of fuel oils is found in Chapter 4.

1.2 WHAT HAPPENS TO FUEL OILS WHEN THEY ENTER THE ENVIRONMENT?

Fuel oils are composed of a large number of different chemicals, and each fuel oil is a slightly different mixture of these chemicals. Some of these chemicals evaporate into the air when fuel oils are spilled onto soils or surface waters (e.g., streams, rivers, lakes, or oceans) or are stored in open containers. Other chemicals in the fuel oils dissolve in water following spills to surface waters or leaks from underground storage tanks. Some of the chemical constituents of fuel oils may slowly move down through the soil to the groundwater. Another group of chemicals in fuel oils can attach to particles in the soil or water and, in water, may sink down into the sediment. The chemicals that evaporate may break down in air by

reacting with sunlight, *e.g.*, photooxidation, or other chemicals in the air. The chemicals that dissolve in water may also be broken down by organisms (primarily bacteria and fungi) in the soil or water. However, this may take up to a year to occur, if ever, depending on the environmental conditions. Chemicals that attach to soil or other matter (e.g., marsh sediment) may remain in the environment for more than a decade. Benzene, toluene, and xylenes (single-ring aromatic compounds), as well as polycyclic aromatic compounds, are the fuel oil components about which we have the greatest amount of information. You can find this information in the ATSDR toxicological profiles for these specific chemicals. See Chapter 5 for more information on what happens to fuel oils when they enter the environment.

1.3 HOW MIGHT I BE EXPOSED TO FUEL OILS?

The most likely way for you to be exposed to fuel oils in the home is if you use a kerosene heater. If you handle fuel oils or use a fuel oil to clean equipment at your job, or if fuel oils are stored at your workplace, you may also be exposed to them through contact with the skin or in the air. Some workers may be exposed to fuel oils through their skin if they come into contact with them without adequate protection, such as gloves, boots, coveralls, or other protective clothing. There are no data on background levels of fuel oils that may be found in the environment or workplace.

You may also be exposed to fuel oils if you swim in waters where fuel oils have been spilled. If fuel oils have leaked from underground storage tanks and entered underground water, you may drink contaminated water from a well containing fuel oils. The vapor (the gas phase) of fuel oils can also move through the soil and enter basements of homes or buildings near areas where leaks have occurred. Children may also be exposed by playing in soil contaminated with fuel oils. A major pathway of exposure is washing one's hands with fuel oils to remove paint, grease, etc. For more information on how you might be exposed to fuel oils, see Chapter 5.

1.4 HOW CAN FUEL OILS ENTER AND LEAVE MY BODY?

Fuel oils can enter and leave your body when you breathe them in the air, when you drink water or eat food containing them, and when your skin comes into contact with them. This can occur in the workplace or if you live near an area where fuel oils have been dumped or spilled. We do not know how much of a fuel oil might be taken up by your body if you inhale fuel oil vapor, drink contaminated water, or come in contact with fuel oils. We have no information on what happens to fuel oils once they enter your body. Kerosene has been found in small amounts in the brain, lung, liver, spleen, and kidney of exposed animals. We do not know if fuel oils are broken down and leave the body in the urine or the feces. For more information on how fuel oils can enter and leave your body, see Chapter 2.

1.5 HOW CAN FUEL OILS AFFECT MY HEALTH?

We know very little of the human health effects caused by fuel oils. Daily use of a kerosene stove for cooking should not cause any breathing problems for most people. People who use kerosene stoves to cook do not have more colds than people who have other types of stoves. Breathing moderate amounts of deodorized kerosene (fuel oil no. 1) has been shown to slightly affect the ability to smell and to cause a taste sensation. Numerous case-studies have reported accidental poisoning in children as the result of drinking kerosene. These accidents are probably much more frequent in areas where kerosene is commonly used for cooking and heating. Drinking kerosene may cause vomiting, diarrhea, swelling of the stomach, stomach cramps, coughing, drowsiness, restlessness, irritability, and unconsciousness; also, it may be difficult to breathe, and breathing may be painful. Coughing, pneumonia, and difficult or painful breathing after drinking kerosene suggest that kerosene has entered the lungs. In addition, drinking large amounts of kerosene can put you into a coma, cause convulsions, and may even cause death. When kerosene gets on your skin for short periods, it can make your skin itchy, red, and sore; sometimes blisters may occur and your skin may peel.

Breathing fuel oil no. 1 vapor for periods as short as 1 hour may make you feel nauseous, increase your blood pressure, be irritating to your eyes, or make your eyes bloodshot. Breathing kerosene or JP-5 vapors can also affect your nervous system. Some of the effects that have been noted in case studies include headache, light-headedness, anorexia (loss of appetite), poor coordination, and difficulty concentrating. Breathing diesel fuel vapors for a long time may damage your kidneys, increase your blood pressure, or lower your blood's ability to clot. Constant skin contact (for example, washing) with diesel fuel may also damage your kidneys.

It appears that repeated contact with fuel oils can cause skin cancer in mice and may cause liver cancer in mice. However, there is some conflicting information. Further, the fuel oils were tested only on mice. We do not know if fuel oils can cause cancer in humans. The International Agency for Research on Cancer (IARC) has determined that residual (heavy) fuel oils and marine diesel fuel are possibly carcinogenic to humans (Group 2B classification). In addition, IARC considers that there is not enough information (Group 3 classification) available to determine if distillate (light) fuel oils or distillate (light) diesel fuels cause cancer. They have also determined that occupational exposures to fuel oils during petroleum refining are probably carcinogenic to humans (Group 2A classification). We do not know if fuel oils can cause birth defects or if they affect reproduction. See Chapter 2 for more information on the health effects of fuel oils.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO FUEL OILS?

There is no medical test that shows if you have been exposed to fuel oils. There are methods to determine if your blood contains some fuel oil components such as benzene, toluene, and xylenes; however, the concentrations of these compounds in distilled fuels are so low that if they were detected in your blood, it might not indicate specific or exclusive exposure to fuel oils. For information on tests for measuring exposure to some individual components of fuel oils, see the ATSDR toxicological profiles on benzene, toluene, total xylenes, and polycyclic

aromatic hydrocarbons. See Chapters 2 and 6 for information on symptoms that suggest exposure to fuel oils.

1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The government has developed regulations and guidelines for fuel oils and some of the chemicals in them. These are designed to protect the public from the possible harmful health effects of these chemicals. The Department of Transportation also regulates the transportation of fuel oils, because they are classified as hazardous materials that are considered to pose a risk to health, safety, or property when transported.

The Occupational Safety and Health Administration (OSHA) and the Air Force Office of Safety and Health (AFOSH) regulate levels of petroleum products in the private sector and Air Force workplaces, respectively. The maximum allowable amount of petroleum products in the workroom air during an &hour workday, 40-hour workweek, is 400 parts of petroleum distillates (naphtha) per million parts of air, or more simply stated, 400 ppm.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

> Agency for Toxic Substances and Disease Registry
> Division of Toxicology
> 1600 Clifton Road NE, E-29
> Atlanta, Georgia 30333

This agency can also provide you with information on the location of the nearest occupational and environmental health clinic. These clinics specialize in the recognition, evaluation, and treatment of illnesses resulting from exposure to hazardous substances.

2. HEALTH EFFECTS

2.1 INTRODUCTION

The primary purpose of this chapter is to provide public health officials, physicians, toxicologists, and other interested individuals and groups with an overall perspective of the toxicology of fuel oils and a depiction of significant exposure levels associated with various adverse health effects. It contains descriptions and evaluations of studies and presents levels of significant exposure for fuel oils based on toxicological studies and epidemiological investigations.

2.2 DISCUSSION OF HEALTH EFFECTS BY ROUTE OF EXPOSURE

To help public health professionals address the needs of persons living or working near hazardous waste sites, the information in this section is organized first by route of exposure--inhalation, oral, and dermal--and then by health effect--death, systemic, immunological, neurological, developmental, reproductive, genotoxic, and carcinogenic effects. These data are discussed in terms of three exposure periods--acute (14 days or less), intermediate (15-364 days), and chronic (365 days or more).

Levels of significant exposure for each route and duration are presented in tables and illustrated in figures. The points in the figures showing no-observed-adverse-effect levels (NOAELs) or lowest observed-adverse-effect levels (LOAELs) reflect the actual doses (levels of exposure) used in the studies, LOAELs have been classified into "less serious" or "serious" effects. These distinctions are intended to help the users of the document identify the levels of exposure at which adverse health effects start to appear. They should also help to determine whether or not the effects vary with dose and/or duration, and place into perspective the possible significance of these effects to human health.

The significance of the exposure levels shown in the tables and figures may differ depending on the user's perspective. For example, physicians concerned with the interpretation of clinical findings in exposed persons may be interested in levels of exposure associated with "serious" effects. Public health officials and project managers concerned with appropriate actions to take at hazardous waste sites may want information on the lowest levels of exposure associated with more subtle effects in humans or animals (LOAEL) or exposure levels below which no adverse effects (NOAEL) have been

observed. Estimates of levels posing minimal risk to humans (Minimal Risk Levels, MRLs) may be of interest to health professionals and citizens alike.

Levels of exposure associated with the carcinogenic effects of fuel oils are indicated in Table 2-3. Estimates of exposure levels posing minimal risk to humans (MRLs) have been made, where data were believed reliable, for the most sensitive non-cancer effect for each exposure duration. MRLs include adjustments to reflect human variability and extrapolation of data from laboratory animals to humans.

Although methods have been established to derive these levels (Barnes and Dourson 1988; EPA 1990e), uncertainties are associated with these techniques. Furthermore, ATSDR acknowledges additional uncertainties inherent in the application of the procedures to derive less than lifetime MRLs. As an example, acute inhalation MRLs may not be protective for health effects that are delayed in development or are acquired following repeated acute insults, such as hypersensitivity reactions, asthma, or chronic bronchitis. As these kinds of health effects data become available and methods to assess levels of significant human exposure improve, these MRLs may be revised.

Fuel oils are petroleum products whose composition varies with the refinery streams from which they are blended (Air Force 1989). Fuel oils, which have auto-ignition temperatures between 177°C and 329°C (Coast Guard 1985), are composed primarily of aliphatic hydrocarbons (64%), aromatic hydrocarbons (35%), and olefinic hydrocarbons (l-2%) (Air Force 1989). The aliphatic constituents consist of *n*-alkanes (*n*-paraffins), branched alkanes (isoparaffins), and cyclic alkanes (cycloparaffins or naphthenes). Aromatic hydrocarbons include benzene and polycyclic hydrocarbons. These petroleum products are used both for residential heating oil and for diesel fuel. Diesel fuels are graded according to the type of engine in which they are used and range from no. 1-D for higher speed and frequent load changes to no. 4-D for low speed engines.

The purpose of this chapter is to discuss the toxicological effects of fuel oils. There are six types of fuel oils discussed in this profile: (1) fuel oil no. 1, (2) fuel oil no. I-D, (3) fuel oil no. 2, (4) fuel oil no. 2-D, (5) fuel oil no. 4, and (6) fuel oil UNSP. However, there are no toxicity data for fuel oils in general; the available toxicity data are specific for particular fuel oils. Therefore, the toxicity of fuel oils will be discussed in this chapter by referring to the following fuel oils for which there are data: (1) fuel oil no. 1, which is also called kerosene, straight-run kerosene, kerosene, range oil, Deobase®, deodorized kerosene, coal oil, and JP-5 (jet fuel); (2) fuel oil no. l-D, which is also known as diesel

fuel and diesel fuel oil no. 1; (3) fuel oil no. 2, which is also called home heating oil, gas oil, and no. 2 burner oil; (4) fuel oil no. 2-D, which is also known as diesel fuel oil no. 2, diesel fuel no. 2, diesel oil no. 2, and no. 2 diesel; (5) fuel oil no. 4, which is also known as diesel fuel oil no. 4, heavy residual fuel oil, marine diesel fuel, and residual fuel oil no. 4; and (6) fuel oil UNSP. Exposure to individual fuel oil components or combustion products will not be discussed as it is not known whether the components or combustion products behave in a toxicologically similar manner to the fuel oils from which they are derived. For more information regarding the potential toxicity of some fuel oil components, the Air Force document (1989) and previous ATSDR profiles on benzene (ATSDR 1989), toluene (ATSDR 1990a), xylenes (ATSDR 1991a), and polycyclic aromatic hydrocarbons (ATSDR 1991b) can be consulted. Since fuel oils are complex and somewhat variable mixtures, it is not possible to identify the molecular weight for these oils. As such, airborne concentrations of fuel oils are reported as milligrams per cubic meter (mg/m^3).

2.2.1 Inhalation Exposure

Fuel oils can enter the respiratory system as a vapor or an aerosol; in addition, products formed during the combustion of fuel oils can be inhaled in smoke. A vapor is the gaseous phase of a substance that is a liquid at standard temperature. Fuel oils have a low vapor pressure (e.g., the saturation concentration of kerosene in air is approximately 100 mg/m^3). An aerosol is the suspension of solid or liquid particles in a gas (usually air), with particles ranging in size from 0.0001 to over 100 μm. Smoke is an aerosol formed during incomplete combustion. Due to the low volatility of fuel oils, human exposure to vapor concentrations above 100 mg/m^3 is unlikely. Higher concentrations can be achieved, however, by increasing the ambient temperature or modifying other physical parameters. Thus, exposure of the general population to high concentrations of airborne fuel oils would probably occur only in unusual situations.

2.2.1.1 Death

No studies were located regarding death in humans after inhalation exposure to fuel oils.

Mortality occurred in 3 of 10 mice exposed by inhalation 8 hours/day for 5 days to diesel fuel no. 2 vapors at a concentration of 204 mg/m^3; inhalation of 65 or 135 mg/m^3 was not lethal to mice (Kainz and White 1984). Acute inhalation of either 86.9 or 408.8 ppm fuel oil UNSP vapor or 101.8 or

401.5 ppm diesel fuel vapor did not induce death in rats (API 1979c, 19798). Inhalation of 8,000-16,000 mg/m^3 diesel fuel aerosol for 2 or 4 hours was lethal to rats (Dalbey and Lock 1983). No lethality occurred following single-exposure inhalation of 6,000 mg/m^3 diesel fuel aerosol for 4 hours or 2,700 mg/m^3 for 6 hours. However, inhalation of 4,000 mg/m^3 for 6 hours was lethal (Dalbey and Lock 1983).

No deaths occurred in rats exposed to 5,000 mg/m3 kerosene (physical form not specified) for 4 hours (Vernot et al. 1990d). This study is limited because only one concentration level was tested. It is useful, however, because the concentration used is very high. Inhalation of diesel fuel aerosol 4 hours/day, 2 days/week, for 13 weeks at concentrations up to 1,500 mg/m^3 did not induce mortality in rats (Lock et al. 1984). Intermediate exposures (once per week for 9 weeks or 3 times per week for 3 weeks) to diesel fuel aerosol induced mortality in 6.25% of the rats exposed to a concentration time product (Ct) of 12,000 mg hour/m^3 (Ct = [airborne concentration of aerosolized diesel fuel in mg/m^3] x [duration of exposure in hours]). However, no rats died at a Ct of 8,000 mg hour/m^3 (Dalbey et al. 1987). The Ct of 8,000 mg hour/m^3, under these conditions, was based on repeated 2- or 6-hour exposures to 4,000 or 1,330 mg/m^3, respectively. The Ct of 12,000 mg hour/m^3 was based on repeated 2- or 6-hour exposures to 6,000 or 2,000 mg/m^3, respectively. However, dose-response data were not reported for the individual exposure concentrations used to produce each Ct.

No rats died during 90-day inhalation exposures to 50 or 300 mg/m^3 marine diesel fuel vapor (Cowan and Jenkins 1981) or to 150 or 750 mg/m^3 JP-5 vapor (Cowan and Jenkins 1981; Gaworski et al. 1984). No mice died during a 90-day inhalation exposure of 150 or 750 mg/m^3 JP-5 vapor (Cowan and Jenkins 1981; Gaworski et al. 1984). One rat died of pneumonia out of 25 male rats exposed to 100 mg/m3 deodorized kerosene vapor (the maximally achievable vapor concentration at standard temperature and pressure) for 6 hours per day, 5 days per week for 13 weeks (Carpenter et al. 1976).

The NOAEL and LOAEL values for death after inhalation exposure to fuel oils are recorded in Table 2-l and plotted in Figures 2-1 and 2-2.

TABLE 2-1. Levels of Significant Exposure to Fuel Oils - Inhalation

Key[a] to figure	Species/ (strain)	Exposure/ duration/ frequency	System	NOAEL[b] (mg/m3)	LOAEL[b] Less serious (mg/m3)	LOAEL[b] Serious (mg/m3)	Reference/ Chemical Form
ACUTE EXPOSURE							
Death							
1	Rat Sprague-Dawley	1 d 6 hr/d				4000 (sex not specified, 30% mortality)	Dalbey and Lock 1983 FO5DFU
2	Mouse CD-1	5 d 8 hr/d				204 M (30% mortality)	Kainz and White 1984 FO4DF2
Systemic							
3	Rat	10 d 6 hr/d	Other	408.8 F (ppm)			API 1979c FO5
4	Rat [CRL: COBS CD(SD) BR]	10 d 6 hr/d	Other		401.5 F (decreased food intake) (ppm)		API 1979g FO5DFU
5	Rat NS	10 d 6 hr/d	Other	400 F (ppm)			Beliles and Mecler 1983 FO2HHO
6	Mouse CD-1	5 d 8 hr/d	Cardio	65 M	135 M (vasodilation)		Kainz and White 1984 FO4DF2
			Bd Wt	135 M	204 M (decreased water and food consumption with subsequent weight loss of 30%)		
Neurological							
7	Mouse CD-1	5 d 8 hr/d			65[c] M (ataxia; disturbed gait)		Kainz and White 1984 FO4DF2

TABLE 2-1. Levels of Significant Exposure to Fuel Oils - Inhalation (continued)

Key[a] to figure	Species/ (strain)	Exposure/ duration/ frequency	System	NOAEL[b] (mg/m3)	LOAEL[b]		Reference/ Chemical Form
					Less serious (mg/m3)	Serious (mg/m3)	
Developmental							
8	Rat	10 d 6 hr/d		408.8 (ppm) F			API 1979c FO5
9	Rat [CRL: COBS CD(SD) BR]	10 d 6 hr/d		401.5 (ppm) F			API 1979g FO1DOK
10	Rat NS	10 d 6 hr/d		400 (ppm) F			Beliles and Mecler 1983 FO2HHO
INTERMEDIATE EXPOSURE							
Systemic							
11	Rat Albino Harlan- Wistar	13 wk 5 d/wk 6 hr/d	Resp	100 M			Carpenter et al. 1976 FO1DOK
			Cardio	100 M			
			Gastro	100 M			
			Hemato	100 M			
			Musc/skel	100 M			
			Hepatic	100 M			
			Renal	100 M			
			Other	100 M			

TABLE 2-1. Levels of Significant Exposure to Fuel Oils - Inhalation (continued)

Key [a] to figure	Species/ (strain)	Exposure/ duration/ frequency	System	NOAEL[b] (mg/m3)	LOAEL[b]		Reference/ Chemical Form
					Less serious (mg/m3)	Serious (mg/m3)	
12	Rat Sprague-Dawley	13 wk 2d/wk 4hr/d	Resp	750	1500 (increase in relative weight of right lobe of lung)		Lock et al. 1984 FO5DFU
			Cardio	1500			
			Gastro	1500			
			Hemato	1500			
			Hepatic	1500			
			Renal	1500			
			Dermal	1500			
			Bd Wt		250 (decreased body weight)		
13	Rat Wistar	14 wk 6 d/wk 6 hr/d	Hepatic		58[d] M (decreased blood glucose levels)		Starek and Vojtisek 1986 FO-1
			Other		231 M (decreased metabolism of phenacetin; decrease in lactate and pyruvate)		
14	Mouse C57BL/6	90 d 24 hr/d	Hepatic		150 F (hepatocellular fatty changes and vacuolization)		Gaworski et al. 1984 FD1JP5
			Other	750 F			
15	Dog Beagle	13 wk 5 d/wk 6 hr/d	Resp	100 M			Carpenter et al. 1976 FO1DOK
			Cardio	100 M			
			Gastro	100 M			
			Hemato	100 M			
			Musc/skel	100 M			
			Hepatic	100 M			
			Renal	100 M			
			Bd Wt		20 M (increased body weight)		

TABLE 2-1. Levels of Significant Exposure to Fuel Oils - Inhalation (continued)

Key[a] to figure	Species/ (strain)	Exposure/ duration/ frequency	System	NOAEL[b] (mg/m3)	LOAEL[b]		Reference/ Chemical Form
					Less serious (mg/m3)	Serious (mg/m3)	
Neurological							
16	Rat Harlan- Wistar	13 wk 5 d/wk 6 hr/d		100			Carpenter et al. 1976 FO1DOK
17	Rat Sprague- Dawley	13 wk 2d/wk 4hr/d			250 (increased response time/startle reflex assay)		Lock et al. 1984 FO5DFU
18	Dog Beagle	13 wk 5 d/wk 6 hr/d		100 M			Carpenter et al. 1976 FO1DOK
Reproductive							
19	Rat Sprague- Dawley	13 wk 2d/wk 4hr/d		1500			Lock et al. 1984 FO5DFU

[a]The number corresponds to entries in Figures 2-1 or 2-2.

[b]Units are in mg/m^3, unless specified otherwise. In some studies, the units were given in ppm. Since the molecular weight of fuel oils is not known, the ppm units could not be converted to mg/m^3. Data from the studies with exposures in ppm are shown in Figure 2-1; data from studies with exposures in mg/m^3 are shown in Figure 2-2.

[c]Used to derive an acute inhalation Minimal Risk level (MRL) of 0.02 mg/m^3; exposure concentration adjusted for 24 hours per day exposure divided by an uncertainty factor of 1000 (10 for interspecies variability, 10 for intraspecies variability, and 10 for use of a LOAEL (less serious effect) for MRL derivation).

[d]Used to determine an intermediate Minimal Risk Level (MRL) of 0.01 mg/m^3; exposure concentration adjusted for 24 hours per day, 7 days per week exposure divided by an uncertainty factor of 1000 (10 for interspecies variability, 10 for intraspecies variability, and 10 for use of a LOAEL (less serious effect) for MRL derivation).

Cardio = cardiovascular; d = day(s); F = female; FO-1 = fuel oil no. 1; FO1DOK = deodorized kersoene; FO5DFU = unspecified diesel fuel; FO2HHO = home heating oil no. 2; FO1JP5 = JP-5 (jet fuel); FO4DF2 = fuel oil no. 2-D; FO5 = unspecified fuel oil; Gastro = gastrointestinal; Hemato = hematological; hr = hour(s); LOAEL = lowest-observed-adverse-effect level; M = male; Musc/skel = musculoskeletal; NOAEL = no-observed-adverse-effect level; Resp = respiratory; wk = week(s)

Figure 2-1. Levels of Significant Exposure to Fuel Oils – Inhalation

Acute
(≤14 days)

Systemic

(ppm)

Other

Developmental

10,000					
1,000					
	○ 3r	◖ 4r	○ 5r	○ 8r	○ 9r	○ 10r
100					
10					
1					
0.1					
0.01					

Key

r   Rat

◖   LOAEL for less serious effects (animals)

○   NOAEL (animals)

The number next to each point
corresponds to entries in the
accompanying table.

0.001
0.0001
0.00001
0.000001
0.0000001

Figure 2-2. Levels of Significant Exposure to Fuel Oils – Inhalation

Acute
(≤14 days)

Systemic

(mg/m³)

Death

Cardiovascular

Body Weight

Neurological

| 10,000 |
| 1,000 |
| 100 |
| 10 |
| 1 |
| 0.1 |
| 0.01 |
| 0.001 |
| 0.0001 |
| 0.00001 |
| 0.000001 |
| 0.0000001 |

1r

2m

6m

6m

6m

6m

7m

Key
r Rat   ● LOAEL for serious effects (animals)
m Mouse ◐ LOAEL for less serious effects (animals)
d Dog   ○ NOAEL (animals)

The number next to each point corresponds to entries in the accompanying table.

Figure 2-2. Levels of Significant Exposure to Fuel Oils – Inhalation (continued)

Intermediate
(15-364 days)

2.2.1.2 Systemic Effects

The highest NOAEL values and all reliable LOAEL values for systemic effects in each species and
duration category for inhalation exposure to fuel oils are recorded in Table 2-l and plotted in
Figures 2-l and 2-2.

Respiratory Effects. Pleural effusions and alveolar infiltrations were noted in a man who had
washed his hair with an unknown amount of diesel fuel (Barrientos et al. 1977). The relative
contributions from inhalation and dermal exposure could not be distinguished in this case. There was
no throat irritation in six volunteers following a 15-minute exposure to a concentration reported to be
140 mg/m^3 of deodorized kerosene vapor (Carpenter et al. 1976). The authors used a hot nichrome
wire for the volatilization of their test material and reported that the concentration was probably the
"highest attainable concentration at which vapor analysis is representative of liquid analysis." The air
saturating concentration of kerosene is considered to approximate 100 mg/m^3 (room temperature and
760 mmHg) and is dependent on the constituents of the mixture.

An epidemiological study examined the effects of chronic exposure to jet fuels in factory workers
(Knave et al. 1978). This study found a significant increase in a feeling of heaviness in the chests of
exposed subjects when compared to unexposed controls from the same factory. The data are limited
because the jet fuels were not specified and may not include JP-5, which is the jet fuel of concern in
this profile, and the study did not adjust for the presence of other chemicals. Inhalation exposure is
likely, since jet fuel vapor was detected by the authors; however, dermal and oral (i.e., from eating
contaminated food) exposures cannot be excluded. An estimated time-weighted average of
128-423 mg/m^3 was detected in the breathing zones of the workers. However, it is not possible to
associate the specific concentrations with specific effects.

Limited epidemiological data suggest that chronic human inhalation exposure to kerosene vapor and/or
kerosene combustion products from cooking with kerosene stoves does not induce asthmatic
respiratory effects. The presence of kerosene stoves in the homes of Malaysian children was not
associated with chronic cough, persistent wheeze, asthma, or chest illness (Azizi and Henry 1991).
Asthmatic bronchitis and frequent common colds in 3-year-old Japanese children were not associated
with the presence of kerosene stoves in their homes (Tominaga and Itoh 1985). The latter study

corrected for exposure to passive smoke. These data are of limited usefulness because the duration of exposure was not reported and the levels of kerosene exposure could not be quantified. Finally, it cannot be determined whether actual exposure to kerosene occurred in these individuals because kerosene exposure was assumed to occur if kerosene was used during cooking or if a kerosene stove was present in the home.

Animal data that pertain to respiratory effects following acute exposure to kerosene by inhalation are limited, because only one concentration level was tested in each study. Reductions in tidal volume and dynamic lung compliance, bronchoconstriction, and an increase in pulmonary resistance occurred in rabbits following inhalation of 32,500 mg/m^3 kerosene aerosol (Casaco et al. 1982). Bronchoconstriction was also induced in guinea pigs that were exposed to 20,400 mg/m^3 kerosene aerosol (Garcia et al. 1988b).

No histopathological changes were noted in the respiratory system of rats or dogs exposed to up to 100 mg/m^3 deodorized kerosene vapor for 13 weeks (Carpenter et al. 1976). Inhalation of diesel fuel aerosol 4 hours/day, 2 days/week, for 13 weeks at concentrations up to 1,500 mg/m^3 did not impair pulmonary function or induce histopathological changes in rats (Lock et al. 1984). However, there was a dose-related increase in the relative weight of the right lobe of the lung, which was only significant at the highest exposure level. Elevated numbers of alveolar macrophages in the low- and high-dose groups were not dose related. Intermediate exposures to diesel fuel aerosol induced damage to the lung parenchyma of rats exposed to a Ct of 8,000 or 12,000 mg hour/m^3 (Dalbey et al. 1987). In general, respiratory effects were more severe after three exposures/week for 3 weeks than one exposure/week for 9 weeks at various exposure levels and durations. Thus, the study found that the respiratory effects were generally more dependent upon the frequency of exposure than the exposure dose or duration. Dose-response data were not reported for the individual exposure concentrations used to produce each concentration time product (see discussion in Section 2.2.1.1).

Cardiovascular Effects. Two case studies were found that reported mild hypertension in humans from acute inhalation exposures to fuel oils. Mild hypertension was noted for 4 days in one of two individuals following a l-hour exposure to JP-5 vapor while flying a small airplane (Porter 1990). Delayed mild hypertension was also noted in a man who was exposed to diesel fuel vapor for 10 days while driving a truck with a fuel injector leak (Reidenberg et al. 1964). The concentration of vapor was not reported in either study. Palpitations were noted in workers chronically exposed to jet fuel

according to one epidemiological study (Knave et al. 1978). The limitations of this study are discussed in detail in Section 2.2.1.2 under Respiratory Effects.

Vasodilation was seen in the ear and tail veins of mice exposed via acute inhalation to 204 or 135 mg/m^3 diesel fuel no. 2 vapors. The effect was not seen at 65 mg/m^3 (Kainz and White 1984). Inhalation of kerosene aerosol for an intermediate duration induced aortic plaques in guinea pigs that resemble those seen in atherosclerosis (Noa and Illnait 1987a). Significant increases in total serum cholesterol and decreases in high-density lipoprotein (HDL) were also noted. Similar effects were induced following exposure to kerosene aerosol or kerosene smoke. This study was limited because only one concentration of kerosene aerosol, within a range of 20,400-34,000 mg/m^3, was tested and the actual exposure level of kerosene smoke was not reported. Inhalation of diesel fuel aerosol 4 hours/day, 2 days/week, for 13 weeks at concentrations up to 1,500 mg/m^3 did not induce histopathological changes in the cardiovascular system of rats (Lock et al. 1984). However, a statistically significant decrease in blood cholesterol levels was reported in females of the high-dose group immediately after exposure compared to controls. The authors did not consider this effect to be treatment related; no data were presented. No microscopic or histopathological changes were noted in the cardiovascular system of rats or dogs exposed to up to 100 mg/m^3 deodorized kerosene vapor for 13 weeks (Carpenter et al. 1976).

Gastrointestinal Effects. Several studies were identified that described gastrointestinal effects in humans after inhalation exposure to unknown quantities of fuel oils. In one case study, one of two individuals that were exposed to JP-5 vapor for approximately 1 hour while flying a small airplane experienced nausea after landing (Porter 1990). This effect subsided within 24 hours. Abdominal cramps, vomiting, and diarrhea occurred in a man who was exposed to diesel fuel vapor for 10 days while driving a truck with a fuel injector leak (Reidenberg et al. 1964). Also, nausea, abdominal cramps, and diarrhea were reported for a man who had washed his hair with a diesel fuel. "On examination" (no further description of the examination was provided), the abdomen was normal (Barrientos et al. 1977). A history of epigastric (upper abdominal) pain was noted in a male subsequent to washing his hands with diesel fuel over several weeks (Crisp et al. 1979). Effects resulting from inhalation versus dermal exposure could not be distinguished in the two latter cases.

No histopathological changes were noted in the gastrointestinal system of rats or dogs exposed to up to 100 mg/m^3 deodorized kerosene vapor for 13 weeks (Carpenter et al. 1976). Inhalation of diesel

fuel aerosol 4 hours/day, 2 days/week, for 13 weeks at concentrations up to 1,500 mg/m^3 did not induce histopathological changes in the gastrointestinal system of rats (Lock et al. 1984).

Hematological Effects. Three case studies were found that addressed possible hematological effects due to acute inhalation exposure to unknown quantities of fuel oils by humans. There were no blood chemistry changes in either of two individuals following a l-hour exposure to JP-5 vapor while flying a small airplane (Porter 1990). Subcutaneous hemorrhage, mild nose bleeds, low platelet counts, and retinal arteriole constriction were reported for a man who was exposed to diesel fuel vapor for 10 days while driving a truck with a fuel injector leak (Reidenberg et al. 1964). The latter effect was delayed, occurring 4 weeks after initial exposure. These effects may be indicative of blood clotting problems. Decreased hemoglobin concentration and an increase in erythrocyte sedimentation rate were noted in one man after washing his hands with diesel fuel over several weeks (Crisp et al. 1979). Effects resulting from inhalation versus dermal exposure cannot be distinguished in this case.

No exposure-related hematological effects were noted in rats or dogs exposed to up to 100 mg/m^3 deodorized kerosene vapor for 13 weeks (Carpenter et al. 1976). Inhalation of diesel fuel aerosol (4 hours/day, 2 days/week, for 13 weeks) at concentrations ranging up to 1,500 mg/m^3 failed to produce histopathological changes, splenic weight changes, or other hematological effects in rats (Lock et al. 1984). Intermediate exposures to diesel fuel aerosol induced decreases in the mean red blood cell count in rats exposed to a Ct of 8,000 or 12,000 mg hour/m^3 3 times/week for 3 weeks (Dalbey et al. 1987). However, the statistical significance of this effect was not clearly reported in the study. There was no significant effect on white blood cell count in these rats. Dose-response data were not reported for the individual exposure concentrations used to produce each Ct (see discussion in Section 2.2.1.1).

Musculoskeletal Effects. No studies were located regarding musculoskeletal effects in humans after inhalation exposure to fuel oils.

No histopathological changes were noted in the musculoskeletal system of rats or dogs exposed to up to 100 mg/m^3 deodorized kerosene vapor for 13 weeks (Carpenter et al. 1976). Only one study of this effect was located.

Hepatic Effects. No studies were located regarding hepatic effects in humans after inhalation exposure to fuel oils.

In a study designed to evaluate the effects of kerosene hydrocarbons on the tissue metabolism of rats after acute and subchronic exposure, Starek and Vojtisek (1986) exposed groups of male Wistar rats (131 total animals for both acute and subchronic studies) to kerosene vapors in average concentrations of 58 (range 33.3-75.0) or 231 (range 181.3-250.2) mg/m^3 for 6 hours/day, 6 days/week for 14 consecutive weeks. Food and water were available *ad lib* throughout exposure, but animals were fasted for 18 hours prior to post-dosing tests. Twenty hours after termination of exposure, two types of tests were conducted: (1) the effects of kerosene on the rate of biotransformation of hexobarbital and phenacetin were examined by looking at hexobarbital sleeping time and measuring the antipyritic activity of phenacetin in intact animals; and (2) blood, liver, and muscle tissue were obtained for *in vitro* tests to examine pyruvate, lactate, and glucose activity/levels. In the first experiments, hexabarbital sleeping time remained unchanged in both treatment groups, and the antipyretic activity of phenacetin was significantly prolonged only in the high-dose (concentration) group. In the second group of tests, the blood glucose concentration was found to be decreased in both the 58 mg/m^3 and 231 mg/m^3 exposure groups, while lactate and pyruvate were found to be increased only in the high concentration group. (This study was used as the basis for derivation of an intermediate MRL for fuel oil no. 1 for the inhalation route of exposure.) The authors suggest that the decreased circulating glucose levels may be associated with both increased glycolysis and the inhibition of gluconeogenesis (kerosene exposure effecting increased glycolysis is supported by the findings of increased concentrations of lactate and pyruvate in the blood and liver, as well as increased lactate dehydrogenase activity in the liver). Further, the authors suggest that the increased glycolysis may be the result of the inhibition of cellular respiration by kerosene, and it was noted that cellular respiration was inhibited in liver and kidney slices subsequent to the addition of kerosene to the incubation solution.

When exposed to up to 100 mg/m^3 deodorized kerosene vapor for 13 weeks, no histopathological changes in the liver were noted in rats or dogs, and no liver weight changes were noted in dogs (Carpenter et al. 1976). Inhalation of diesel fuel aerosol 4 hours/day, 2 days/week for 13 weeks at concentrations up to 1,500 mg/m^3 did not induce histopathological or organ weight changes in the livers of rats (Lock et al. 1984).

Renal Effects. Renal effects were tested by urinalysis in two individuals who were exposed to JP-5 vapor for approximately 1 hour while flying a small airplane (Porter 1990). No abnormalities in the urine were reported in this case study for either of these individuals. Acute renal failure was reported in a man who was exposed to diesel fuel vapor for 10 days while driving a truck with a fuel injector leak (Reidenberg et al. 1964). Acute renal failure also occurred in a man after washing his hair with an unknown amount of diesel fuel (Barrientos et al. 1977). In addition, he had oliguria; biopsy revealed mitosis and vacuolization in renal cells, tubular dilation, and some cellular proliferation in the glomerulus. Another man developed acute tubular renal necrosis after washing his hands with an unspecified diesel fuel over several weeks (Crisp et al. 1979). Specifically, patchy degeneration and necrosis of the proximal and distal tubular epithelium with preservation of the basement membranes were noted. Also, increased blood urea and serum creatinine levels were noted in this individual. Effects resulting from inhalation versus dermal exposure could not be distinguished in the two latter cases. Based upon these case studies and the finding of kidney damage in mice dermally exposed to fuel oils (see Section 2.2.3.2 under Renal Effects), it may be possible to absorb fuel oil vapor through the skin. However, no studies were located that tested dermal absorption of fuel oil vapor in humans (see Section 2.3.1.3).

Several studies have identified a hydrocarbon-induced nephropathy in male rats that is associated with exposure to hydrocarbon vapors, including some fuel oils (Bruner 1984; Cowan and Jenkins 1981; Gaworski et al. 1984). This hydrocarbon-induced nephropathy has only been demonstrated in adult male rats and has been linked to a specific protein, $\alpha_{2\mu}$-globulin, which is produced under hormonal control by the liver (Alden 1986). When male rats are exposed to certain hydrocarbons, including JP-5 and marine diesel fuel, $\alpha_{2\mu}$-globulin accumulates in hyaline droplets, which can be visualized in proximal tubule cells. This buildup of $\alpha_{2\mu}$-globulin-containing hyaline droplets is thought to lead to cell necrosis; the cellular debris accumulates at the corticomedullary junction, causing tubule dilation and mineralization of the tubules. Studies of 90-day continuous inhalation of 150 or 750 mg/m^3 JP-5 vapor (Bruner 1984; Cowan and Jenkins 1981; Gaworski et al. 1984) and 50 or 300 mg/m^3 marine diesel fuel vapor (Bruner 1984; Cowan and Jenkins 1981) have shown that a dose-response relationship exists for multifocal tubular atrophy and focal tubular necrosis at the corticomedullary junction in male rats. Granular cysts form from the necrotic debris, which then plug and dilate the proximal tubules, resulting in chronic necrosis. In all cases of JP-5-induced and marine diesel fuel-induced nephropathy, dose-dependent formation of cytoplasmic hyaline droplets of the proximal tubules in the renal cortex is prominent. Increased blood urea nitrogen and creatinine levels were

found to be associated with this nephropathy in male rats following inhalation of 150 or 750 mg/m^3 JP-5 (Cowan and Jenkins 1981). Since humans do not possess $\alpha_{2\mu}$ -globulin, this effect in male rats is not considered germane to human health risk assessment.

This nephropathy has also been identified in male rats exposed to JP-5 by the oral route (see the discussion of Renal Effects in Section 2.2.2.2). This nephropathy does not appear to be induced by deodorized kerosene or diesel fuel, at least not under subchronic exposure conditions. No histopathological changes were noted in the renal system of rats or dogs exposed to up to 100 mg/m^3 deodorized kerosene vapor for 13 weeks (Carpenter et al. 1976). No organ weight or histopathological changes of the renal system were noted in rats following inhalation of diesel fuel aerosol 4 hours/day, 2 days/week, for 13 weeks at concentrations up to 1,500 mg/m^3 (Lock et al. 1984). This lesion has not been noted in female rats, female mice (studies conducted on male mice were not located), or dogs of either sex when exposed under similar conditions to either JP-5 or marine diesel fuel vapors (Bruner 1984; Cowan and Jenkins 1981; Gaworski et al. 1984).

Dermal Effects. No studies were located regarding dermal effects in humans after inhalation exposure to fuel oils.

Whole-body inhalation exposure to diesel fuel aerosol 4 hours/day, 2 days/week, for 13 weeks at concentrations up to 1,500 mg/m^3 did not induce skin lesions in rats (Lock et al. 1984).

Ocular Effects. One case study describes eye irritation in two individuals exposed to JP-5 vapor for approximately 1 hour while flying a small airplane (Porter 1990). Both individuals experienced a burning sensation in their eyes, and one had itchy, watery eyes 1 day after the exposure. These effects subsided within 24 hours. Hyperemic conjunctiva were also reported for one of the individuals; this effect subsided after 4 days. Another case study describes subconjunctival hemorrhages in a man whom had washed his hair with an unknown amount of diesel fuel (Barrientos et al. 1977). Effects resulting from inhalation versus dermal exposure could not be distinguished in this case. Eye irritation was not induced in six volunteers by a 15-minute exposure to 140 mg/m^3 deodorized kerosene vapor (Carpenter et al. 1976). This study is limited since only one concentration was tested. Eye irritation was also noted in factory workers who were chronically exposed to jet fuel (Knave et al. 1978). The limitations of the study are discussed in detail in Section 2.2.1.2 under Respiratory Effects.

No studies were located regarding ocular effects in animals after inhalation exposure to fuel oils.

Body Weight Effects. No studies were located regarding body weight effects for humans after inhalation exposure to fuel oils.

There was no change in body weight gain in rats exposed to up to 100 mg/m^3 deodorized kerosene vapor for 13 weeks (Carpenter et al. 1976). Inhalation of diesel fuel aerosol 4 hours/day, 2 days/week for 13 weeks at concentrations of 250-1,500 mg/m^3 induced reversible body weight loss at all exposure levels and decreased food consumption in rats exposed to the mid- and high-exposure levels (Lock et al. 1984). There were no histopathological or relative adrenal gland weight changes in these animals. Intermediate exposures to diesel fuel aerosol induced decreases in the mean body weights of rats exposed to a Ct of 8,000 or 12,000 mg hour/m^3 (Dalbey et al. 1987). Weight loss was found to be dependent upon exposure concentration, frequency, and duration in this study. Dose-response data were not reported for the individual exposure concentrations used to produce each Ct (see discussion in Section 2.2.1.1). There was no change in body weight gain in mice or female rats following 90-day inhalation exposure to 750 mg/m^3 JP-5 vapor (Gaworski et al. 1984).

Other Systemic Effects. A man exposed to diesel fuel vapor for 10 days while driving a truck with a fuel injector leak exhibited systemic edema (Reidenberg et al. 1964). Edema of the scrotum and ankle were reported in a man who washed his hands with diesel fuel over several weeks (Crisp et al. 1979). Other effects noted in this man were loin pains, thirst, and severe exhaustion. Effects resulting from inhalation versus dermal exposure could not be distinguished in this case. Inhalation of 400 ppm home heating oil no. 2 vapor, 408.8 ppm fuel oil UNSP vapor, or 401.5 ppm diesel fuel vapor by pregnant rats for 10 days did not induce maternal body weight changes (API 1979c, 1979g; Beliles and Mecler 1983). A dose-response relationship was noted for decreased food and water consumption with subsequent weight loss and dehydration in mice following 5-day, 8-hour/day acute inhalation of 204 mg/m^3 diesel fuel no. 2 vapors (Kainz and White 1984). These effects were not induced by 135 mg/m^3 of the diesel fuel. Decreased food intake was also noted in pregnant rats following inhalation of 401.5 ppm diesel fuel vapor; no effects on maternal food intake were noted following inhalation of 101.8 ppm diesel fuel vapor, 100 or 400 ppm home heating oil vapor, or 86.9 or 408.8 ppm fuel oil UNSP vapor (API 1979c, 1979g; Beliles and Mecler 1983).

2.2.1.3 Immunological and Lymphoreticular Effects

No studies were located regarding immunological or Lymphoreticular effects in humans or animals after inhalation exposure to fuel oils.

2.2.1.4 Neurological Effects

In one case study, neurological effects in humans resulting from acute exposure to JP-5 vapor were reported (Porter 1990). Coordination and concentration difficulties and fatigue were noted in each of two individuals following a l-hour exposure to JP-5. Other effects included headache, apparent intoxication, and anorexia. Neither experienced any sensory impairment. The effects subsided within 24 hours for one individual, and within 4 days for the other. Anorexia was also reported in a man who washed his hands with diesel fuel over several weeks (Crisp et al. 1979). Effects resulting from inhalation versus dermal exposure could not be distinguished in this case. A man exposed to diesel fuel vapor for 10 days while driving a truck with a fuel injector leak exhibited severe headaches approximately 4 weeks after exposure (Reidenberg et al. 1964). In a study of six volunteers, slight olfactory fatigue was induced in three, while one reported "tasting something," following a 15-minute exposure to 140 mg/m3 deodorized kerosene vapor (Carpenter et al. 1976). This study is limited since only one concentration was tested.

An epidemiological study tested the effects of chronic exposure to jet fuel in factory workers (Knave et al. 1978). This study found significant increases in neurasthenia (i.e., fatigue, depressed mood, lack of initiative, dizziness, and sleep disturbances) in the exposed subjects when compared to unexposed controls from the same factory. Also, attention and sensorimotor speed were impaired in the exposed workers, but no effects were found on memory function or manual dexterity. Results of an electroencephalograph (EEG) suggested that the exposed workers may have had instability in the thalamocortical system. The limitations of the study, which include lack of specification of type of jet fuel and no adjustment for other chemicals, were discussed in greater detail in Section 2.2.1.2 under Respiratory Effects.

Kainz and White (1984) exposed (nose only) groups of CD-l mice to concentrations of 65, 135, or 204 mg diesel vapor/m3 of air for 8 hours/day for 5 consecutive days. General appearance and behavior were observed, as was performance on a series of neurobehavioral tests (square box activity

test, rotating rod test, inclined plane test, cornea1 reflex test, hot plate test) administered 24 hours before exposure, on each day of exposure, and 24 hours after the last exposure. Results of the square box test revealed increased activity at the lowest concentration, little change at the mid-concentration, and decreased activity (up to 50%) in the high-concentration exposed animals. The rotating rod test showed decreased performance at the mid- and high-exposures. Results of the hot plate test indicated an increased sensitivity to heat of the mid- and high-concentration groups on day 1 of exposure, but tolerance was reported on day 6. The corneal reflex and inclined plane tests showed no differences from controls at any air concentration. Some degree of ataxia/disturbed gait was observed in all exposure groups immediately after removal from the exposure chamber, with the severity and duration of the symptom being dose dependent (all mice returned to normal before the next day's exposure). Grooming habits were reported to be poor in the mid- and high-exposure groups. Water consumption was decreased in the high-dose group, which was reported to appear dehydrated on day 3 of exposure, and a 30% loss in body weight was also observed in this group. Vasodilation of the ear and tail veins was seen in the mid- and high-concentration groups on the third day of the experiment. Tremors were reported in 3 of 10 mice in the mid-exposure group and in 5 of 10 mice in the high-concentration group while in motion, and 3 of the high-exposure animals died before the end of the experiment. The results of the Kainz and White (1984) study indicated a time- and dose-dependent response to diesel vapor, with nonlethal effects all being completely reversible within 24 hours of cessation of exposure. (This study was used to derive an acute inhalation MRL for fuel oil no. 2-D.)

No histopathological changes were noted in the nervous system of rats or dogs exposed to up to 100 mg/m^3 deodorized kerosene vapor for 13 weeks (Carpenter et al. 1975, 1976). Inhalation of diesel fuel aerosol 4 hours/day, 2 days/week, for 13 weeks at concentrations up to 1,500 mg/m^3 did not induce histopathological changes in the nervous system of rats (Lock et al. 1984). However, peak response time using the startle reflex assay was increased in rats at all exposure levels, but the greatest increase occurred in the high-dose group. Neurotoxicity, as measured using the landing footspread, tail flick, forelimb grip strength, and startle reflex assays, did not occur in rats exposed to a Ct of 8,000 or 12,000 mg hour/m^3 diesel fuel aerosol for 3 or 9 weeks (Dalbey et al. 1987).

The highest NOAEL and all reliable LOAEL values for neurological effects after inhalation exposure to fuel oils are recorded in Table 2-1 and plotted in Figure 2-2.

2.2.1.5 Reproductive Effects

No studies were located regarding reproductive effects in humans after inhalation exposure to fuel oils.

Inhalation of diesel fuel aerosol 4 hours/day, 2 days/week, for 13 weeks at concentrations up to 1,500 mg/m^3 did not induce relative testes weight changes nor histopathological changes in the reproductive organs of rats (Lock et al. 1984).

The highest NOAEL value for reproductive effects after inhalation exposure to fuel oils is recorded in Table 2-l and plotted in Figure 2-2.

2.2.1.6 Developmental Effects

No studies were located regarding developmental effects in humans after inhalation exposure to fuel oils.

No developmental effects (soft tissue changes, skeletal abnormalities, inhibition of fetal growth) were noted in the fetuses of female rats exposed to 400 ppm home heating oil no. 2, 408.8 ppm fuel oil UNSP, or 401.5 ppm diesel fuel vapor by inhalation during gestation days 6-15 (API 1979c 1979g; Beliles and Mecler 1983). Only one study was located for each fuel oil for these effects.

The highest NOAEL values for developmental effects after inhalation exposure to fuel oils are recorded in Table 2-1 and plotted in Figure 2-l.

2.2.1.7 Genotoxic Effects

No studies were located regarding genotoxic effects in humans after inhalation exposure to fuel oils.

In the only *in vivo* animal study located regarding genotoxic effects using inhalation as the route of exposure, male CD-l mice (12/group) exposed to 100 or 400 ppm diesel fuel vapor (6 hours/day, 5 days/week, for 8 weeks) showed no adverse effects with respect to the frequency of dominant lethal mutations (API 1981).

Other genotoxicity studies are discussed in Section 2.4.

2.2.1.8 Cancer

There are limited epidemiological data regarding carcinogenicity in humans following chronic inhalation exposure to kerosene. In one case-control study, there was no association between the use of kerosene stoves for cooking and bronchial cancer in nonsmoking women (Chan et al. 1979). In another case-control study, there was no association between renal cell cancer and occupational exposure to fuel oils, including diesel fuel (Partanen et al. 1991). In the first study, it cannot be determined whether the individuals were actually exposed to kerosene vapor. Also, there may be additional or alternative effects resulting from exposure to the combustion products of kerosene, which may be toxicologically different from the kerosene itself. In the latter study, exposure to other chemicals may have affected the results, since fuel oil exposure was based upon occupation. Both studies are limited because they do not quantify the levels of exposure and cannot accurately determine the duration of exposure to fuel oils. In another study, increased risk of laryngeal cancer was associated with self-reported exposure to diesel oil (Ahrens et al. 1991). However, the data were equivocal. Similarly, exposure to "petroleum products," including diesel fuel, has been associated with acute leukemia (Lindquist et al. 1991), but the study was very limited in that type of product exposed to was not described, and included a wide range of product types. A third study associated the use of kerosene stoves and exposure to "petroleum products" with oral and pharyngeal cancer (Zheng et al. 1992), but suffers the same limitations as Lindquist et al., with the addition that use of kerosene stoves involves exposure to both kerosene vapor and combustion products.

In a study conducted on rats, no renal tumors were observed during life-time observation following a 90-day continuous exposure to 750 mg/m^3 JP-5 vapor or to 300 mg/m^3 marine diesel fuel vapor (Bruner 1984). Since this study was not designed to test carcinogenicity, these data have limited usefulness.

2.2.2 Oral Exposure

2.2.2.1 Death

Numerous case studies have described death following the accidental ingestion of kerosene by children (usually under the age of 5 but as old 15 years). The deaths are usually attributed to lipoidal pneumonia (Morrison and Sprague 1976; Santhanakrishnan and Chithra 1978; Zucker et al. 1986) that was probably induced by the aspiration of the kerosene. Specific respiratory effects associated with death from kerosene ingestion include pneumothorax (Mahdi 1988; Zucker et al. 1986), emphysema (Mahdi 1988), and pneumonitis (Singh et al. 1981). Cardiac arrhythmia was reported as the cause of death in one child; however, it was suspected that myocarditis and pulmonary edema may have been the cause of the rapid deterioration and death of the child (Dudin et al. 1991).

Estimated ingested doses of kerosene associated with death are as low as 1,890 mg/kg, based on ingestion of 30 mL of kerosene by children 11 months to 2 years of age (Dudin et al. 1991; Santhanakrishnan and Chithra 1978), and as high as 16,789 mg/kg, based on ingestion of 200 mL of kerosene by a l-year-old child (Santhanakrishnan and Chithra 1978). No lethality was reported for children from 10 months to 5 years old following ingestion of estimated doses ranging from 120 to 870 mg/kg and in one instance a dose as high as 1,700 mg/kg of kerosene (Dudin et al. 1991).

Death in rats occurred after acute oral exposure to 12,000 mg/kg kerosene but not after exposures to 8,000-11,200 mg/kg kerosene or 12,150 mg/kg Deobase® (Muralidhara et al. 1982). Oral exposure to 4,000 mg/kg kerosene was lethal to 10-day-old rats; this dose level was not tested in adult rats (Deichmann et al. 1944). Death occurred in two out of six rats subsequent to a single gavage dose of 47,280 mg/kg JP-5, but none died from single doses of 18,912-29,944 mg/kg JP-5 (Parker et al. 1981). One rat exposed to 37,824 mg/kg JP-5 died from a gavage accident. There were no other deaths in that treatment group.

The acute oral LD_{50} values for kerosene in guinea pigs and rabbits have been reported to be 16,320 and 22,720 mg/kg, respectively (Deichmann et al. 1944). In guinea pigs, death occurred following acute oral exposure to 3,760-19,200 mg/kg kerosene. Death in rabbits occurred after acute oral exposure to 12,800-28,800 mg/kg kerosene but not after exposure to 8,000 mg/kg. These data for guinea pigs and rabbits are limited, because the methodologies and experimental conditions of this

study were poorly described. Oral gavage of 6,400 mg/kg/day kerosene, administered for 7-10 days, was lethal to four out of five male calves; only one dose was tested in this study (Rowe et al. 1973). Mortality in rats was induced by acute aspiration of 0.05-0.25 mL of kerosene; there was a dose response relationship for death in this study (Gerarde 1963). Aspiration is induced by placing the test material into the back of the throat which causes the animal to choke, forcing the test compound into the respiratory tract. The purpose of using aspiration as a route of exposure in animals is to mimic human respiratory exposure which occurs during vomiting after ingestion of kerosene. Mortality in mice was noted following a single exposure to 20 µL kerosene by aspiration (Nouri et al. 1983). This latter study is limited because only one dose was tested.

All reliable LOAEL values for death in each species and duration category after oral exposure to fuel oils are recorded in Table 2-2 and plotted in Figure 2-3.

2.2.2.2 Systemic Effects

No studies were located regarding musculoskeletal effects in humans or animals after oral exposure to fuel oils.

The highest NOAEL and all reliable LOAEL values for systemic effects in each species and duration category for oral exposure to fuel oils are recorded in Table 2-2 and plotted in Figure 2-3.

Respiratory Effects. Even if the kerosene is initially ingested (accidental ingestion of fuel oils is most often noted in children under 5 years of age), the respiratory toxicity is usually attributable to the aspiration of kerosene into the lungs during vomiting (Coruh and Inal 1966; Majeed et al. 1981; Nouri and Al-Rahim 1970). Based on those case studies that examined at least 50 cases of kerosene ingestion by children, the respiratory effects that primarily occur from kerosene ingestion are bronchopneumonia, bronchitis, pneumonitis, lung infiltrates and effusions, cough, dyspnea, and tachypnea (Akamaguna and Odita 1983; Aldy et al. 1978; Annobil 1983; Annobil and Ogunbiyi 1991; Mahdi 1988; Santhanakrishnan and Chithra 1978; St. John 1982). Pneumonitis, pulmonary edema, and/or pneumonia were reported for children and adults who had ingested kerosene (Subcommittee on Accidental Poisoning 1962). Hypoxia has also been noted in some cases (Dudin et al. 1991). An epidemiological study found a significant increase in a feeling of heaviness in the chests of workers

TABLE 2-2. Levels of Significant Exposure to Fuel Oils ‐ Oral

Key[a] to figure	Species/ (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL		Reference/ Chemical Form
					Less Serious (mg/kg/day)	Serious (mg/kg/day)	
ACUTE EXPOSURE							
Death							
1	Rat Wistar-CFTRI	1 d (G)				12000 F (minimum lethal dose; 33% mortality)	Muralidhara et al. 1982 FO-1
2	Rat Sprague-Dawley	1 d (G)				47280 M (33% mortality)	Parker et al. 1981 FO1JP5
Systemic							
3	Rat Wistar-CFTRI	1 d (G)	Cardio	12000 F			Muralidhara et al. 1982 FO-1
			Gastro	12000 F			
			Hemato	12000 F			
			Renal	12000 F	NS F (slightly dilated kidney tubules)		
			Hepatic	12000 F	NS F (cellular vacuolization; infiltration)		
			Bd Wt	12000 F	NS F (decreased body weight and food intake)		

TABLE 2-2. Levels of Significant Exposure to Fuel Oils - Oral (continued)

Key [a] to figure	Species/ (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference/ Chemical Form
4	Rat Sprague-Dawley	1 d (G)	Resp		NS M (congestion of the lung)		Parker et al. 1981 FO1JP5
			Cardio	NS M	NS M (epicardium congestion)		
			Gastro	18912 M	NS M (mottled liver; swollen liver; hepatocyte changes)		
			Hepatic		18912 M (hepatocyte necrosis)		
			Renal	37824 M	47280 M (hyaline droplets)		
			Derm		NS M (subcutis congestion; alopecia)		
Neurological							
5	Rat Wistar-CFTRI	1 d (G)		8000 F	NS F (anorexia)	9600 F (unsteady gait; drowsiness)	Muralidhara et al. 1982 FO-1

[a]The number corresponds to entries in Figure 2-3.

Cardio = cardiovascular; Derm - dermal; d = day(s); F = female; FO-1 = fuel oil no. 1; FO1JP5 = JP-5 (jet fuel); (G) = gavage; Gastro = gastrointestinal; Hemato = hematological; LOAEL = lowest-observed-adverse-effect level; M = male; NOAEL = no-observed-adverse-effect level; Resp = respiratory

Figure 2-3. Levels of Significant Exposure to Fuel Oils – Oral
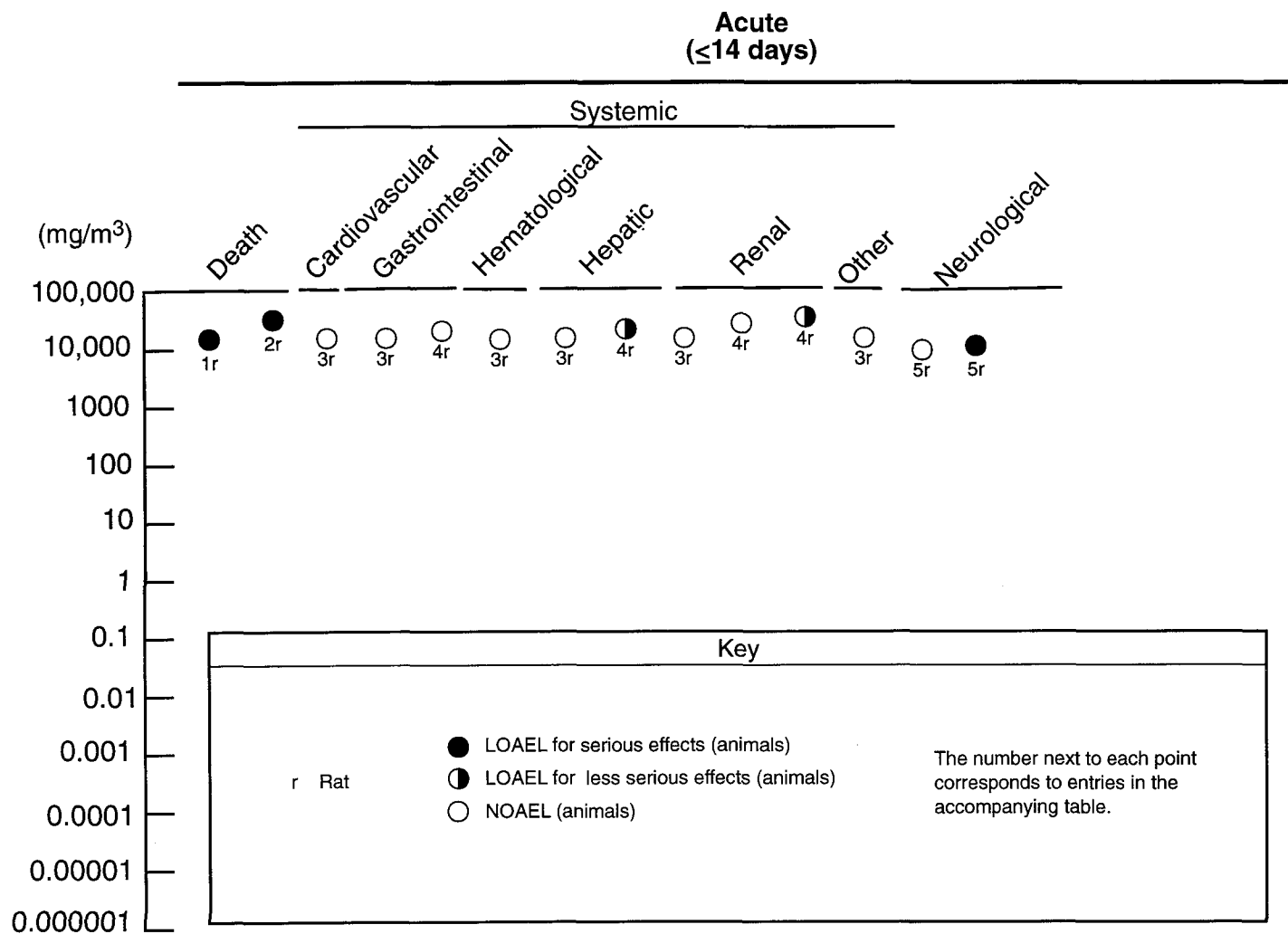
Acute
(≤14 days)

who were chronically exposed to jet fuels by the inhalation, oral, and/or dermal exposure routes (limitations of the study are discussed in detail in Section 2.2.1.2 in Respiratory Effects) (Knave et al. 1978). A follow-up study was conducted on children who 10 years earlier had been diagnosed with pneumonitis due to kerosene ingestion and who had abnormal chest radiographs at the time (Tal et al. 1984).

Researchers found an increase in volume of isoflow, a decrease in change in flow while breathing helium compared to air at 50% vital capacity, and the continued presence of abnormal chest radiographs. The study suggests that there may be long-term respiratory effects following aspiration of ingested kerosene.

Several studies have reported estimated levels of exposure which are usually based on the finding of an empty container near the poisoned child (Agarwal and Gupta 1974; Akamaguna and Odita 1983; Aldy et al. 1978; Coruh and Inal 1966; Dudin et al. 1991; Nouri and Al-Rahim 1970; Saksena 1969; Santhanakrishnan and Chithra 1978), although the effects associated with specific doses were not reported. The Subcommittee on Accidental Poisoning (1962) estimated that ingestion of 10-30 mL kerosene was associated with pulmonary complications in 11 of the 422 cases studied (the incidence of the effects, ages associated with the effects, and doses were not reported). These effects also occurred at doses beyond this range. An estimated oral dose of less than 5,300 mg/kg kerosene resulted in the death of a 10-month-old girl. Pneumothorax, pneumomediastinum, and death were believed to be the results of respiratory distress from aspiration of kerosene (Zucker et al. 1986). Respiratory distress was reported to have resulted in the deaths of a 2-year-old child and a l-year-old child after ingestion of 30 mL (1,890-1,959 mg/kg) and 200 mL (15,340-16,789 mg/kg) of kerosene, respectively (Santhanakrishnan and Chithra 1978).

Not all cases of kerosene ingestion result in toxicity. For instance, as many as 56% of the cases studied were asymptomatic in two of the study populations (Mahdi 1988; Santhanakrishnan and Chithra 1978). Also, 39% of one population of children had normal lung x-rays following kerosene ingestion (Annobil and Ogunbiyi 1991). No doses were reported in these cases, although the authors estimated them as small.

Mononuclear and polymorphonuclear cell infiltration and unspecified pathological lesions were noted in the lungs of guinea pigs after gavage administration of 3,200-8,000 mg/kg kerosene (Brown et al.

1974). In mice, aspiration of 20 µL of Kerosene induced pulmonary consolidation and hemorrhage, pneumonitis, a decrease in pulmonary clearance of *Staphylococcus aureus*, and an increase in relative lung weight (Nouri et al. 1983). Dogs exposed to 0.5 mL/kg kerosene by aspiration exhibited increases in oxygen utilization, intrapulmonary physiologic shunt fraction, and respiratory rate and decreases in arterial oxygen tension (Goodwin et al 1988). In the aspiration studies, the actual dose entering the lungs cannot be determined.

Cardiovascular Effects. Tachycardia was noted in children following acute ingestion of kerosene (Akamaguna and Odita 1983; Coruh and Inal, 1966). In one case study, cardiomegaly, but not heart failure, occurred in 20% of the cases of kerosene poisoning (Akamaguna and Odita 1983). An epidemiological study found a significant increase in cardiac palpitations in workers who were chronically exposed to jet fuels by the inhalation, oral, and/or dermal exposure routes (Knave et al. 1978). The limitations of the study are discussed in detail in Section 2.2.1.2 under Respiratory Effects.

There were no histopathological changes and no change in the relative heart weight in rats following exposure by gavage to single doses of up to 12,000 mg/kg kerosene or 12,150 mg/kg Deobase[®] (Muralidhara et al. 1982). Data for Deobase[®] are limited because effects were reported for only one dose.

In another study, decreases in heart rate and mean arterial blood pressure occurred in dogs following a single exposure to 0.5 mL/kg kerosene by aspiration, although these values returned to the control values within 60 minutes (Goodwin et al. 1988). The actual dose entering the lungs by aspiration cannot be determined. The study is limited, however, because only one dose was tested.

Gatrotestinal Effects. The most commonly reported gastrointerinal effect in children following acute ingestion of kerosene in vomiting (Akamaguna and Odita 1983; Aldy et al. 1978; Mahdi 1988; Majeed et al. 1981; Nouri and Al-Rahim 1970; Saksena 1969; St. Johns 1982), including bloody vomit (Nouri and Al-Rahmin 1970). Other effects noted are abdominal pain and/or distension (Akamaguna and Odita 1983; Mahdi 1988; Majeed et al. 1981; Nouri and Al-Rahim 1970; Sakena 1969), gastroenteritis (Sakena 1969), and diarrhea (Majeed et al. 1981).

No diarrhea was noted in rats following exposure by gavage to single doses of up to 12,000 mg/kg kerosene or 12,150 mg/kg Deobase® (Muralidhara et al. 1982).

Hematological Effects. Several case studies reported hematological effects in children following acute ingestion of kerosene. Increases in leukocyte counts were reported for 37-80% of the respective study populations (Dudin et al. 1991; Majeed et al. 1981; Nouri and Al-Rahim 1970).

In rats exposed by gavage to single doses of up to 12,000 mg/kg/ kerosene or 12,150 mg/kg Deobase®, there was no change in relative spleen weight and no histopathological changes of the spleen occurred (Muralidhara et al. 1982). Rats had increased hematocrit, decreased white blood cell counts, and increased erythrocyte counts following exposure by gavage to a single dose of 18,912 mg JP-5/kg (Parker et al. 1981).

Hepatic Effects. No studies were located regarding hepatic effects in humans after oral exposure to fuel oils.

There was no change in the relative organ weight of the liver in rats following exposure by gavage to single doses of up to 12,000 mg/kg kerosene or 12,150 mg/kg Deobase® (Muralidhara et al. 1982). Histopathological examination revealed slight cellular infiltration and mild vacuolization of the liver, but the kerosene and Deobase exposure levels that induced these effects were not specified. A single exposure to JP-5 induced necrosis in the hepatocytes of rats exposed to 18,912-47,280 mg/kg by gavage (Parker et al. 1981). In another experiment, a single exposure to 18,912 mg JP-5/kg induced vacuolization of the periportal hepatocytes within 2 days of gavage, as well as statistically significant increases in serum glutamic pyruvic transaminase (SGPT), glutamic oxaloacetic transaminase (SGOT), and lactate dehydrogenase levels, suggesting hepatic damage (Parker et al. 1981).

Renal Effects. Several case studies reported normal urinalysis tests in children following acute ingestion of kerosene (Dudin et al. 1991; Mahdi 1988; Nouri and Al-Rahim 1970), although albuminuria was occasionally noted (Dudin 1991; Nouri and Al-Rahim 1970).

There were no changes in relative kidney weights in rats following exposure by gavage to single doses of up to 12,000 mg/kg kerosene or 12,150 mg/kg Deobase® (Muralidhara et al. 1982). Histopathological examination revealed slight cellular infiltration and mild vacuolization in the kidney

tissues and slight dilation of the kidney tubules, but the kerosene and Deobase® exposure levels that induced these effects were not specified. Only one study was located that tested for kidney weight changes.

In another study, hyaline droplets were detected in the kidneys of two male rats that died 48 hours after a single exposure to 47,280 mg/kg JP-5 by gavage (Parker et al. 1981). This effect was not apparent in male rats that died within 48 hours of exposure to 47,280 mg/kg or in rats that survived for 14 days following exposures to 18,912-37,824 mg/kg JP-5. However, hyaline droplets were apparent in rats that were killed within 2-3 days of exposure to 18,912 mg/kg JP-5. Thus, the effect appears to be induced within a specific period, between 2 and 14 days, following exposure. A single exposure to 18,912 mg/kg JP-5 also induced a statistically significant increase in creatinine levels (Parker et al. 1981). These effects are apparently unique to male rats and are not expected to occur in humans (see discussion in Section 2.2.1.2 under Renal Effects).

Dermal Effects. Large blisters, erythema, and peeling skin were reported in two cases of apparent oral exposure to kerosene (Annobil 1988). However, the strong odor of kerosene on one of the individuals and the kerosene-stained clothing of the other indicate that dermal exposure may have also occurred in these cases. Exposure levels were not reported.

Alopecia and congestion of the subcutis were noted in rats following exposure by gavage to single doses of 24 mL JP-5/kg (Parker et al. 1981).

Ocular Effects. No studies were located regarding ocular effects in humans or animals after oral exposure to fuel oils.

Other Systemic Effects. Fever has been reported in children following ingestion of kerosene (Akamaguna and Odita 1983; Aldy et al. 1978; Dudin et al. 1991; Mahdi 1988; Majeed et al. 1981; Nouri and Al-Rahim 1970; Saksena 1969; St. John 1982). In one study, fever was reported with pulmonary complications for children and adults who had ingested kerosene (Subcommittee on Accidental Poisoning 1962). It is not known whether the fever was secondary to the pulmonary effects.

There were no histopathological changes and no changes in the relative adrenal gland weights in rats
following exposure by gavage to single doses of up to 12,000 mg/kg kerosene or 12,150 mg/kg
Deobase® (Muralidhara et al. 1982). Only one study was located that tested for these effects.

2.2.2.3 Immunological and Lymphoreticular Effects

No studies were located regarding immunological effects in humans or animals after oral exposure to
fuel oils.

2.2.2.4 Neurological Effects

In one study, lethargy, semicoma, and/or coma were reported for children and adults who had ingested
kerosene (Subcommittee on Accidental Poisoning 1962). Estimated exposure levels of 10-30 mL
kerosene were associated with complications of the central nervous system in 18 of the 422 study
participants. However, these effects also occurred at doses beyond this range, but the exact exposure
levels are not known. Incidences of the effects and the ages associated with the effects or ingested
doses were not reported.

Several case studies reported neurological effects in children following acute ingestion of kerosene. In
studies that examined 50-205 kerosene poisoning cases, the neurological effects noted most frequently
were unconsciousness or semiconsciousness, drowsiness, restlessness, and irritability (Akamaguna and
Odita 1983; Aldy et al. 1978; Coruh and Inal 1966; Dudin et al. 1991; Mahdi 1988; Majeed et al.
1981; Nouri and Al-Rahim 1970; Saksena 1969; Santhanakrishnan and Chithra 1978; St. John 1982).
Coma and convulsions were also noted in numerous studies, but were usually evident in only one or
two individuals per study population (Coruh and Inal 1966; Dudin et al. 1991; Majeed et al. 1981;
Nouri and Al-Rahim 1970; Saksena 1969; Santhanakrishnan and Chithra 1978).

In one study of 78 children known to have ingested kerosene, coma, convulsions, and death were
noted in two children (aged 11-48 months) after each ingested a quantity of kerosene estimated to be
between 30 mL (1,890 mg/kg) and 50 mL (4,255 mg/kg) (Dudin et al. 1991). The cause of death was
not neurological for these children. Neither coma nor convulsions occurred in children that ingested
3-20 mL of kerosene (equivalent to 126-1,754 mg/kg in children aged 10 months to 5 years).
However, in the majority of the cases of kerosene ingestion, neurological effects were not associated

with specific reported quantities. There are limited data that suggest that the central nervous system effects following ingestion of kerosene are due to hypoxia from kerosene-induced respiratory impairment (Majeed et al. 1981).

An epidemiological study found significant increases in neurasthenia (i.e., fatigue, depressed mood, lack of initiative, dizziness, and sleep disturbances) in workers who were chronically exposed to jet fuels by inhalation, oral, and/or dermal exposure (Knave et al. 1978). Also, attention and sensorimotor speed were impaired, but no effects were found on memory function or manual dexterity. EEG results suggest that the exposed workers may have had instability in the thalamocortical system. The limitations of the study are discussed in detail in Section 2.2.1.2 under Respiratory Effects.

Single exposures to 12,000 mg/kg kerosene and 12,150 mg/kg Deobase by oral gavage induced unsteady gait and drowsiness in rats; however, no neurological effects occurred from exposure to 8,000 mg/kg kerosene (Muralidhara et al. 1982). These data are limited since statistical analysis was not conducted and effects in the controls were not described. Also, a dose-response relationship cannot be identified from the Deobase$^®$ data, since only one dose was tested.

In another study in which mice were exposed to a single dose of 20 μL of kerosene followed by aspiration, drowsiness, lack of coordination, and behavioral changes occurred (Nouri et al. 1983). The study is limited because only one dose was tested. The actual dose entering the lungs by aspiration cannot be determined.

The highest NOAEL and all reliable LOAEL values for neurological effects after oral exposure to fuel oils are recorded in Table 2-2 and plotted in Figure 2-3.

2.2.2.5 Reproductive Effects

No studies were located regarding reproductive effects in humans or animals after oral exposure to fuel oils.

2.2.2.6 Developmental Effects

No studies were located regarding developmental effects in humans or animals after oral exposure to fuel oils.

2.2.2.7 Genotoxic Effects

No studies were located regarding genotoxic effects in humans after oral exposure to fuel oils.

Inconclusive evidence that fuel oil no. 2 is clastogenic in rats has been reported (Conaway et al. 1984). Briefly, a bone marrow assay was used in which groups of four Sprague-Dawley rats (sex not specified) received oral doses of 125, 417, or 1,250 mg/kg/day fuel oil no. 2 for 5 consecutive days. Animals were then sacrificed, and bone marrow cells were examined for abnormal chromosome morphology. Marked, but not dose-related, increases in the percentage of aberrant cells and the percentage of cells with chromatid breaks were seen in all treatment groups. The effect was reported to be significant at the low and high dose, and the greatest yield of aberrant chromosome figures occurred in the animals treated with 125 mg/kg/day. Alternatively, cyclohexane/DMSO extract and DMSO extract of diesel 1 (CAS no. 8008-20-6) and home heating oil (CAS no. 68476-30-2), administered orally at doses of 1.0, 2.0, and 5.0 g/kg, did not induce increased frequency of micronuclei in a mouse bone marrow micronucleus assay (McKee et al 1994). It should be noted that the extraction procedure was used to concentrate the aromatic fraction (with particular interest in the polynuclear aromatics) of the fuel oils tested.

Other genotoxicity studies are discussed in Section 2.4.

2.2.2.8 Cancer

No studies were located regarding cancer in humans or animals after oral exposure to fuel oils.

2.2.3 Dermal Exposure

2.2.3.1 Death

No studies were located regarding death in humans after dermal exposure to fuel oils.

Single dermal exposures to 0.5 mL/kg home heating oil no. 2 or diesel fuel were not lethal to rabbits (API 1979a, 1980a). Daily dermal exposures for 1 week to 0.1 mL kerosene were not lethal to male mice (Upreti et al. 1989). Death in mice occurred after acute dermal exposures to 20,000-40,000 mg/kg/day marine diesel fuel and 30,000-40,000 mg/kg/day JP-5, but not after exposures to 2,000-8,000 mg/kg/day marine diesel fuel or 5,000-20,000 mg/kg/day JP-5 (NTP/NIH 1986). Intermediate exposures to 2,000-8,000 mg/kg/day JP-5 for 13 weeks (NTP/NIH 1986) and to 42.2 mg per application JP-5 or 45.5 mg per application marine diesel fuel 3 times per week for 40 weeks (Schultz et al. 1981), were also lethal to mice. Intermediate exposures (13 weeks) to 500 or 1,000 mg/kg/day JP-5, or 2504,000 mg/kg/day marine diesel fuel (NTP/NIH 1986), or 21.1 mg per application JP-5 3 times per week for 40 weeks, or 22.9 mg per application marine diesel fuel 3 times per week for 40 weeks (Schultz et al. 1981) were not lethal in mice.

A statistically significant increase in the number of deaths was noted only in female mice following chronic exposure to marine diesel fuel and JP-5 at doses of 250 and 500 mg/kg/day for both fuel oils (NTP/NIH 1986). Although the number of deaths in males under these conditions was increased over that of the controls, the effect was not significant. Deaths were observed as early as week 1 of exposure to marine diesel fuel and week 2 of exposure to JP-5. The data are limited for each of these experiments because it was not specified whether the animals were protected against oral exposure and/or removal of the test material.

The highest NOAEL and all reliable LOAEL values for death in each species and duration category after dermal exposure to fuel oils are recorded in Table 2-3.

2.2.3.2 Systemic ,Effects

The highest NOAEL and all reliable LOAEL values for systemic effects in each species and duration category for dermal exposure to fuel oils are recorded in Table 2-3.

TABLE 2-3. Levels of Significant Exposure to Fuel Oils - Dermal

Species/ (Strain)	Exposure/ Duration/ Frequency/ (Specific Route)	System	NOAEL[a] (mg/kg/day)	LOAEL[a]		Reference/ Chemical Form
				Less Serious (mg/kg/day)	Serious (mg/kg/day)	
ACUTE EXPOSURE						
Death						
Mouse B6C3F1	2 wk 7 d/wk				20000 (100% mortality)	NTP/NIH 1986 FO1DFM
Mouse B6C3F1	2 wk 7 d/wk				30000 F (100% mortality) M (0% mortality)	NTP/NIH 1986 FO1JP5
Systemic						
Mouse B6C3F1	2 wk 7 d/wk	Derm		2000 (acanthosis; inflammation; parakerotosis; hyperteratosis)		NTP/NIH 1986 FO1DFM
		Other	8000			
Mouse B6C3F1	2 wk 7 d/wk	Derm		NS (scaly skin; hair loss; inflammation; acanthosis; hyperkeratosis)		NTP/NIH 1986 FO1JP5
		Bd Wt	5000	10000 (17% decrease in body weight gain)		
INTERMEDIATE EXPOSURE						
Death						
Mouse B6C3F1	13 wk 7 d/wk				2000 F (60% mortality) M (0% mortality)	NTP/NIH 1986 FO1JP5
Mouse C3HF/Bd	40 wk 3x/wk				45.5 F (40% mortality) (mg/app) M (20% mortality)	Schultz et al. 1981 FO1DFM

TABLE 2-3. Levels of Significant Exposure to Fuel Oils - Dermal (continued)

Species/ (Strain)	Exposure/ Duration/ Frequency/ (Specific Route)	System	NOAEL[a] (mg/kg/day)	LOAEL[a]		Reference/ Chemical Form
				Less Serious (mg/kg/day)	Serious (mg/kg/day)	
Mouse C3HF/Bd	40 wk 3x/wk				42.2 F (40% mortality) (mg/app) M (13% mortality)	Schultz et al. 1981 FO1JP5
Systemic						
Mouse B6C3F1	13 wk 7 d/wk	Resp	8000			NTP/NIH 1986 FO1JP5
		Cardio	8000			
		Gastro	8000			
		Hemato		500 (splenic hematopoiesis)		
		Hepatic		500 (karyomegaly)		
		Renal	8000			
		Derm		500 (slight to moderate dermatosis)		
		Bd Wt	2000	4000 (decrease in body weight gain)		
Mouse B6C3F1	13 wk 7 d/wk	Resp	4000			NTP/NIH 1986 FO1DFM
		Cardio	4000			
		Gastro	4000			
		Hemato	4000			
		Hepatic	4000			
		Renal	4000			
		Dermal	2000	4000 (mild chronic active dermatitis)		
		Bd Wt	250M	500 M (9% decrease in body weight gain)		

TABLE 2-3. Levels of Significant Exposure to Fuel Oils - Dermal (continued)

Species/ (Strain)	Exposure/ Duration/ Frequency/ (Specific Route)	System	NOAEL[a] (mg/kg/day)	LOAEL[a]		Reference/ Chemical Form
				Less Serious (mg/kg/day)	Serious (mg/kg/day)	
Mouse BALC/C	40 wk 3x/wk	Hemato		21.1 (mg/app) (increased spleen weight)		Schultz et al. 1981 FO1JP5
		Hepatic	42.2 (mg/app)			
		Renal		21.1 F (increased kidney weight) (mg/app) 21.1 M (decreased kidney weight) (mg/app)		
		Bd Wt		21.1 (7-11% decrease in body (mg/app) weight)		
Mouse BALB/C	40 wk 3x/wk	Hemato		22.9 (increased spleen weight) (mg/app)		Schultz et al. 1981 FO1DFM
		Hepatic	45.5 (mg/app)			
		Renal		22.9 F (increased kidney weight) (mg/app) 22.9 M (decreased kidney weight) (mg/app)		
		Bd Wt		22.9 (4-21% decrease in body (mg/app) weight)		

Neurological

| Mouse B6C3F1 | 13 wk 7 d/wk | | 8000M | | | NTP/NIH 1986 FO1JP5 |

Reproductive

| Mouse B6C3F1 | 13 wk 7 d/wk | | 8000 | | | NTP/NIH 1986 FO1JP5 |

TABLE 2-3. Levels of Significant Exposure to Fuel Oils - Dermal (continued)

Species/ (Strain)	Exposure/ Duration/ Frequency/ (Specific Route)	System	NOAEL[a] (mg/kg/day)	LOAEL[a]		Reference/ Chemical Form
				Less Serious (mg/kg/day)	Serious (mg/kg/day)	
CHRONIC EXPOSURE						
Death						
Mouse B6C3F1	84 -103 wk 5 d/wk				250 F (74% mortality) 250 M (54% mortality)	NTP/NIH 1986 FO1DFM
Mouse B6C3F1	90 - 103 wk 5 d/wk				250 F (30% mortality) 250 M (34% mortality)	NTP/NIH 1986 FO1JP5
Systemic						
Mouse B6C3F1	90 - 103 wk 5 d/wk	Resp	500			NTP/NIH 1986 FO1JP5
		Cardio	500			
		Gastro	500			
		Hemato	250	500 (amyloid spleen)		
		Musc/skel	500			
		Hepatic	250	500 (amyloid liver)		
		Renal	250	500 (amyloid kidney)	250	
		Derm			(ulcers; dermatitis)	
		Bd Wt	250	500 (12-25% decrease in body weight gain)		

TABLE 2-3. Levels of Significant Exposure to Fuel Oils ⁻ Dermal (continued)

Species/ (Strain)	Exposure/ Duration/ Frequency/ (Specific Route)	System	NOAEL[a] (mg/kg/day)	LOAEL[a]				Reference/ Chemical Form
				Less Serious (mg/kg/day)		Serious (mg/kg/day)		
Mouse B6C3F1	84 - 103 wk 5 d/wk	Resp	500					NTP/NIH 1986 FO1DFM
		Cardio	500					
		Gastro	500					
		Hemato		250	(hematopoiesis of spleen and liver)			
		Musc/skel	500					
		Renal		250	(inflammation of urinary bladder)	250		
		Dermal					(ulcers; dermatitis)	
		Bd Wt		250	(14-23% decrease in body weight gain)			

Immuno/Lymphoret

| Mouse B6C3F1 | 90 - 103 wk 5 d/wk | | 250 | 500 | (granulocyte hyperplasia in the bone marrow; hyperplasia in the lymph nodes) | | | NTP/NIH 1986 FO1JP5 |
| Mouse B6C3F1 | 84 - 103 wk 5 d/wk | | | 250 | (lymph node plasmocyosis) | | | NTP/NIH 1986 FO1DFM |

Neurological

| Mouse B6C3F1 | 90 - 103 wk 5 d/wk | | 500 | | | | | NTP/NIH 1986 FO1JP5 |
| Mouse B6C3F1 | 84 - 103 wk 5 d/wk | | 500 | | | | | NTP/NIH 1986 FO1DFM |

TABLE 2-3. Levels of Significant Exposure to Fuel Oils ⁻ Dermal (continued)

Species/ (Strain)	Exposure/ Duration/ Frequency/ (Specific Route)	System	NOAEL[a] (mg/kg/day)	LOAEL[a]		Reference/ Chemical Form
				Less Serious (mg/kg/day)	Serious (mg/kg/day)	
Reproductive						
Mouse B6C3F1	84 - 103 wk 5 d/wk		500			NTP/NIH 1986 FO1DFM
Mouse B6C3F1	90 - 103 wk 5 d/wk		500			NTP/NIH 1986 FO1JP5
Cancer						
Mouse B6C3F1	84 - 103 wk 5 d/wk				250 M (hepatocellular adenoma or carcinoma)	NTP/NIH 1986 FO1DFM
Mouse B6C3F1	90 - 103 wk 5 d/wk				250 (malignant lymphomas)	NTP/NIH 1986 FO1JP5

[a]Units are in mg/kg/day, unless specified otherwise.

Bd Wt = body weight: Cardio = cardiovascular; d = day(s); Derm = dermal; F = females; FO1DFM = diesel marine fuel; FO1JP5 = JP-5 (jet fuel); Gastro = gastrointestinal; Hemato = hemotological; Immuno/Lymphoret = immunological/lymphoreticular; LOAEL = lowest-observed-adverse-effect level; M = males; mg/app = mg per application; Musc/skel = musculoskeletal; NOAEL = no-observed-adverse-effect level; Resp = respiratory; wk = week(s); x = time(s)

Respiratory Effects. Effusions and alveolar infiltrations of the lung occurred in a man who had washed his hair with an unknown amount of diesel fuel (Barrientos et al. 1977). Effects resulting from inhalation versus dermal exposure could not be distinguished in this case. An epidemiological study found a significant increase in feelings of "thoracic oppression" (no description provided) in workers who were chronically exposed to jet fuels by the inhalation, oral, and/or dermal exposure routes (Knave et al. 1978). The limitations of the study are discussed in detail in Section 2.2.1.2 under Respiratory Effects.

No histopathological or organ weight changes were noted in the respiratory system of male mice following daily dermal exposures for 1 week to 0.1 mL kerosene (Upreti et al. 1989); 13-week exposures to 2,000-8,000 mg/kg/day JP-5; or chronic exposures to 250 or 500 mg/kg/day of either marine diesel fuel or JP-5 (NTP/NIH 1986).

Cardiovascular Effects. An epidemiological study found a significant increase in heart palpitations in workers who were chronically exposed to jet fuels by inhalation, oral, and/or dermal exposure routes (Knave et al. 1978). The limitations of the study are discussed in detail in Section 2.2.1.2 under Respiratory Effects.

No histopathological changes were noted in the cardiovascular system of mice exposed to 2,000-8,000 mg/kg/day JP-5 for 13 weeks or mice chronically exposed to 250 or 500 mg/kg/day of either marine diesel fuel or JP-5 (NTP/NIH 1986).

Gastrointestinal Effects. Nausea, abdominal cramps, and diarrhea occurred in a man who had washed his hair with an unknown amount of diesel fuel (Barrientos et al. 1977). Clinical examination revealed a normal abdomen. Another man had epigastric pain after washing his hands with diesel fuel over several weeks (Crisp et al. 1979). Effects resulting from inhalation versus dermal exposure could not be distinguished in these cases.

No histopathological changes were noted in the gastrointestinal system of mice exposed to 2,000-8,000 mg/kg/day JP-5 for 13 weeks or mice chronically exposed to 250 or 500 mg/kg/day of either marine diesel fuel or JP-5 (NTP/NIH 1986).

Hematological Effects. One case study reported that decreased hemoglobin concentration and an increase in erythrocyte sedimentation rate were noted in one man after washing his hands with diesel fuel over several weeks (Crisp et al. 1979). Effects resulting from inhalation versus dermal exposure could not be distinguished in this case.

A decrease in the splenic relative weight, which was not accompanied by histopathological changes, was noted in male mice following daily dermal exposures for 1 week to 0.1 mL kerosene (Upreti et al. 1989). In addition, decreases in hemoglobin concentration and increases in erythrocyte, white blood cell, and polymorphonuclear leukocyte concentrations were noted. Females were not tested in this study. Hematopoiesis of the spleen (extramedullary hematopoiesis) was noted in mice exposed to 500-8,000 mg/kg/day JP-5 for 13 weeks (NTP/NIH 1986). This effect was dose related and was found to be secondary to dermatitis in mice chronically exposed to 250 or 500 mg/kg/day marine diesel fuel (NTP/NIH 1986). The appearance of extramedullary hematopoiesis indicates a response to a hematological change or effect. Hematopoiesis in the liver in female mice was dose dependent and was directly related to chronic exposures to 250 or 500 mg/kg/day marine diesel fuel (NTP/NIH 1986).

Musculoskeletal Effects. No studies were located regarding musculoskeletal effects in humans after dermal exposure to fuel oils.

No histopathological changes were noted in the musculoskeletal system of mice chronically exposed to 250 or 500 mg/kg/day of either marine diesel fuel or JP-5 (NTP/NIH 1986).

Hepatic Effects. No studies were located regarding hepatic effects in humans after dermal exposure to fuel oils.

No histopathological or organ weight changes were noted in the livers of male mice following daily dermal exposures for 1 week to 0.1 mL kerosene (Upreti et al. 1989). Slight hepatic karyomegaly was noted in mice exposed to 500-8,000 mg/kg/day JP-5 for 13 weeks (NTP/NIH 1986). Amyloidosis of the liver occurred in mice chronically exposed to 500 mg/kg/day JP-5 but not in those exposed to 250 mg/kg/day.

Renal Effects. Acute renal failure occurred in a man who washed his hair with an unknown amount of diesel fuel (Barrientos et al. 1977). In addition, he had oliguria; biopsy revealed mitosis and vacuolization in renal cells, tubular dilation, and some cellular proliferation in the glomerulus. Another man developed acute tubular renal necrosis after washing his hands with an unspecified diesel fuel over several weeks (Crisp et al. 1979). Specifically, patchy degeneration and necrosis of the proximal and distal tubular epithelium with preservation of the basement membranes were noted. Also, increased blood urea nitrogen and serum creatinine levels were noted in this individual. Effects resulting from inhalation versus dermal exposure could not be distinguished in these cases.

No histopathological or organ weight changes were noted in the kidneys of male mice following daily dermal exposures for 1 week to 0.1 mL kerosene (Upreti et al. 1989) nor following exposure to 2,000-8,000 mg/kg/day JP-5 for 13 weeks (NTP/NIH 1986). Renal lesions were produced in at least one sex and at one or both doses levels (100% or 50%) in mice dermally treated three times per week for 60 weeks with JP-5 or marine diesel fuel (Easley et al. 1982). However, the lesions could not be duplicated in mice injected intraperitoneally with 100 mg/kg (using a corn oil vehicle) for 3 times per week for days or in mice injected intraperitoneally with 25 microliters of JP-5 for 2 to 8 weeks. In contrast to the case study (Barrientos et al. 1977) in which oliguria was manifested as a symptom of acute diesel fuel toxicity, the dermally treated test animals demonstrated increased urine output, increased insensitive water loss, and increased water consumption. The inability to reproduce the lesions and the increased water consumption and loss by intraperitoneal injection led the authors to speculate that dermal application may be the necessary route of exposure to cause the renal toxicity (Easley et al. 1982). It should be noted that only abbreviated results were reported. Intermediate and chronic exposure to petroleum oils was reported to induce a nodular appearance of the kidney as well as induce tubular atrophy of the renal cortex in mice (Schultz et al. 1981). However, it was not reported what petroleum fuels induced the kidney injury, although marine diesel fuel and JP-5 were among those studied. From calculations of the kidney to body weight ratios in mice exposed to 21.l and 42.2 mg JP-5 or 22.9 and 45.5 mg marine diesel fuel for 40 weeks, dose-related trends were noted in female mice for increased relative kidney weights following JP-5 exposure (right kidney only) and marine diesel fuel (both kidneys) (Schultz et al. 1981). There were no dose-response trends for the decreased relative kidney weights in males exposed to either fuel oil. Statistical analysis was not conducted on the changes in kidney to body weight ratios. Therefore, the significance of the dose-response trends cannot be confirmed.

Lymphocytic inflammation of the urinary bladder was noted in mice chronically exposed to 250 or 500 mg/kg/day marine diesel fuel. Amyloidosis of the kidney was found to be secondary to dermatitis in mice chronically exposed to 500 mg/kg/day JP-5 (NTP/NIH 1986).

Dermal Effects. Experimental data regarding dermal exposure of humans to fuel oils are limited. In one study, there was a dose-dependent increase in dermatitis from acute exposures to 55-85% solutions of kerosene (Tagami and Ogino 1973). No effects were noted in these subjects from exposure to the 40% solution of kerosene. This study is limited because no vehicle controls were used. Also, each subject was exposed to all test solutions (i.e., four different concentrations of kerosene), but the chronological spacing of the four treatments is not known. In another study, acute dermal exposure to 1 mL of kerosene impaired protein synthesis, but not deoxyribonucleic acid (DNA) or collagen synthesis, in the epidermis (Lupulescu and Birmingham 1975). Hyperemia, cellular damage of the epidermis, and mild edema also occurred from acute exposure to 1 mL kerosene (Lupulescu and Birmingham 1976; Lupulescu et al. 1973). Histological changes included disorganization of the cells, cytolysis, and enlarged intercellular spaces in the stratum corneum and spinous cells of the epidermis (Lupulescu and Birmingham 1976). Effects had subsided within 72 hours in some individuals (Lupulescu et al. 1973). These studies are limited because each tested only one dose.

Dermal effects of fuel oils from known or suspected short-term dermal exposures are described in several case studies. In one study, erythema, bullae, burning, and itching were reported in a 45-year-old man following a 20-minute dermal exposure to kerosene (Mosconi et al. 1988). In another case study, three males (2-15 years old) and 1 female (2 years old) exhibited blisters, reddening, flaccid bullae, pustules, soreness, burning, swelling, and denudation of the skin following dermal exposures to unknown volumes of kerosene (Tagami and Ogino 1973). A third study reported large blisters, erythema, and peeling skin in two cases of apparent oral exposure to kerosene (Annobil 1988). However, the strong odor of kerosene on one of the individuals and the kerosene-stained clothing of the other strongly indicate that dermal exposure may have also occurred in these cases. Exposure levels were not specified. Dermatosis and erythema were evident in factory workers who were exposed to kerosene for up to 5 hours daily by handling kerosene-soaked steel parts; exposure levels were not reported (Jee et al. 1985).

Male mice treated daily for 1 week with 0.1 mL kerosene had rough skin, edema, and inflammation at the exposure sites (Upreti et al. 1989). Females were not tested in this study. Female mice treated with middle distillates including straight-run kerosene for six weeks developed hyperplasia and necrosis in the epidermis (Ingram et al. 1993) and increased sebocyte counts (Lesnik et al. 1992). Acute dermal exposures to 1% JP-5 or marine diesel fuel induced mild dermal sensitization in guinea pigs (Cowan and Jenkins 1981). Skin irritation was not induced in male rabbits by acute exposure to 0.5 mL JP-5 or marine diesel fuel (Schultz et al. 1981). These studies are limited because only one exposure dose was used in each. Acute dermal exposures to 2,000-40,000 mg/kg marine diesel fuel and unspecified concentrations of JP-5-induced dermatitis (acanthosis, scaly skin, hair loss, inflammation, parakeratosis, and/or hyperkeratosis of the skin) in mice (NTP/NIH 1986). This effect also occurred following 13-week exposures to 4,000 mg/kg/day marine diesel fuel. Intermediate exposure to 500-8,000 mg/kg/day JP-5 induced slight-to-moderate dermatosis in mice which increased with dose. Dermal sensitization did not occur in guinea pigs that were dermally exposed to 9 or 10 doses of diesel fuel (API 1979f; Schultz et al. 1981) or 9 doses of JP-5 (Schultz et al. 1981) over a 3-week period. Dermal application of three types of no. 2 fuel oils (low-catalytic cracked [10%], medium-catalytic cracked [30%], and high-catalytic cracked [50%]) and diesel fuel did not produce skin sensitization in the guinea pig (Beck et al. 1984), although doses were not reported. Erythema and edema occurred during the induction phase in the animals exposed to diesel fuel (API 1979f).

Chronic exposures to 250 and 500 mg/kg/day of both marine diesel fuel and JP-5 induced dermatitis and ulcerations of the skin in mice (NTP/NIH 1986). The incidence and severity of dermatitis and the incidence of ulcers induced by marine diesel fuel were dose dependent for the chronic exposures. The severity, but not the incidence, of dermatitis induced by JP-5 was dose dependent for the chronic exposures. Also, the incidence of ulcers was dose dependent in chronic studies with JP-5. Dermatitis was also noted in another study in mice that were chronically exposed to either JP-5 or marine diesel fuel; effective doses were not reported (Easley et al. 1982).

Ocular Effects. Acute exposure to diesel fuel induced ocular effects in one case (Barrientos et al. 1977). Subconjunctival hemorrhages occurred in a man who had washed his hair in an unknown amount of diesel fuel. Effects resulting from inhalation versus dermal exposure could not be distinguished in this case. Eye irritation was also noted in factory workers who were chronically exposed to jet fuel (Knave et al. 1978). The limitations of this study are discussed in detail in Section 2.2.1.2 under Respiratory Effects.

Ocular irritation was not induced in rabbits by diesel fuel (API 1979b; Beck et al. 1984), marine diesel fuel, JP-5 (Cowan and Jenkins 1981; Schultz et al. 1981), or three types of no. 2 fuel oils (low-catalytic cracked [10%], medium-catalytic cracked [30%], an high-catalytic cracked [50%]) (Beck et al. 1984). Draize scores were not reported in the Cowan and Jenkins (1981) study.

Body Weight Effects. No studies were located regarding body weight effects in humans after dermal exposure to fuel oils.

There was no change in body weight in male mice following daily dermal exposures for 1 week to 0.1 mL kerosene (Upreti et al. 1989). There was no effect on body weight in mice following acute exposures to 2,000-8,000 mg/kg/day marine diesel fuel (NTP/NIH 1986). Acute exposure to at least 10,000 mg/kg/day JP-5, but not 5,000 mg/kg/day, induced decreases in body weight in mice. A dose-related trend in decreased body weight gain was noted in male, but not female, mice exposed to 4,000 and 8,000 mg/kg/day JP-5 and 500-4,000 mg/kg/day marine diesel fuel for 13 weeks. For JP-5 exposure, the changes in weight gain compared to the controls were not large, i.e., 5-7%, and therefore, the significance of the effect could not be determined (NTP/NIH 1986). Dermal application of total weekly doses of 126.6 and 63.3 mg of JP-5 or 136.5 and 68.2 mg of diesel fuel marine three times per week for 40 weeks produced significant weight reduction in mice (Schultz et al. 1981); however, the authors failed to accurately describe the methods and doses. Chronic exposures to 500 (but not 250) mg/kg/day JP-5 and 250 and 500 mg/kg/day marine diesel fuel induced decreases in body weight relative to controls (NTP/NIH 1986). An increased incidence of amyloid in the adrenal cortex was found to be secondary to dermatitis in mice chronically exposed to 500 mg/kg/day JP-5 (NTP/NIH 1986).

Other Systemic Effects. Edema of the scrotum and ankle, loin pains, thirst, and severe exhaustion were reported in a man who washed his hands with diesel fuel over several weeks (Crisp et al. 1979). Effects resulting from inhalation versus dermal exposure could not be distinguished in this case.

There were no abnormal clinical signs, no histopathological or organ weight changes in the adrenal glands, and no effects on food or water intake in male mice following daily dermal exposures for 1 week to 0.1 mL kerosene (Upreti et al. 1989). Amyloidosis of the spleen was found secondary to dermatitis in mice chronically exposed to 500 mg/kg/day JP-5; this effect was not noted following exposure to 250 mg/kg/day JP-5 (NTP/NIH 1986). Mice exposed to a 50% solution of marine diesel

fuel for 60 weeks had increases in daily water consumption (Easley et al. 1982). This effect also occurred in mice exposed to JP-5, but dose levels were not reported. Similarly, dermal application of JP-5 and diesel marine fuel increased water consumption and urine output (accompanied by a loss in osmolarity) in mice. Easley and coworkers (1982) speculated that the increased water consumption may have been the result of impaired renal function (see Section 2.2.3.2 under Renal Effects) and/or the dehydration of these animals.

2.2.3.3 Immunological and Lymphoreticular Effects

No studies were located regarding immunological effects in humans after dermal exposure to fuel oils.

Decreases in the relative weights of the lymph nodes and thymus were noted in male mice following daily dermal exposures for 1 week to 0.1 mL kerosene (Upreti et al. 1989). In addition, thymocyte counts, bone marrow nucleated cell counts, thymic cortical lymphocytes, and the cellularity of the thymic lobules were decreased. Increases in the cellular populations of the popliteal lymph nodes and the axial lymph nodes were also present. This study is limited because females were not tested. Chronic exposure to 500 mg/kg/day JP-5 induced granulocytic hyperplasia in the bone marrow in male and female mice and hyperplasia in the lymph nodes of female mice (NTP/NIH 1986). Plasmacytosis of the lymph nodes was found to be secondary to dermatitis in mice chronically exposed to 250 and 500 mg/kg/day of marine diesel fuel.

The highest NOAEL and all reliable LOAEL values for immunological effects after dermal exposure to fuel oils are recorded in Table 2-3.

2.2.3.4 Neurological Effects

In one case study, anorexia was reported in a man who washed his hands with diesel fuel over several weeks (Crisp et al. 1979). Effects resulting from inhalation versus dermal exposure could not be distinguished in this case. An epidemiological study found a significant increase in neurasthenia (i.e., fatigue, depressed mood, lack of initiative, dizziness, and sleep disturbances) in workers who were chronically exposed to jet fuels by either inhalation, oral, and/or dermal exposure (Knave et al. 1978). Also, attention and sensorimotor speed were impaired in the exposed workers, but no effects were found on memory function or manual dexterity. Results of EEG tests suggest that the exposed

workers may have instability in the thalamocortical system. The limitations of the study were discussed in detail in Section 2.2.1.2 under Respiratory Effects.

Increased response to tactile stimuli and hyperactivity occurred in male mice at initiation of daily dermal exposures for 1 week to 0.1 mL kerosene (Upreti et al. 1989). Females were not tested in this study. No histopathological changes were noted in the nervous system of mice exposed to 2,000-8,000 mg/kg/day JP-5 for 13 weeks or mice chronically exposed to 250 or 500 mg/kg/day of either marine diesel fuel or JP-5 (NTP/NIH 1986).

The highest NOAEL values for neurological effects after dermal exposure to fuel oils are recorded in Table 2-3.

2.2.3.5 Reproductive Effects

No studies were located regarding reproductive effects in humans after dermal exposure to fuel oils.

No histological changes were noted in the reproductive system of mice dermally exposed to 2,000-8,000 mg/kg/day JP-5 for 13 weeks or in mice chronically exposed to 250 or 500 mg/kg/day of either marine diesel fuel or JP-5 (NTP/NIH 1986).

The highest NOAEL values for reproductive effects after dermal exposure to fuel oils are recorded in Table 2-3.

2.2.3.6 Developmental Effects

No studies were located regarding developmental effects in humans or animals after dermal exposure to fuel oils.

2.2.3.7 Genotoxic Effects

No studies were located regarding genotoxic effects in humans or animals after dermal exposure to fuel oils.

Genotoxicity studies are discussed in Section 2.4.

2.2.3.8 Cancer

No studies were located regarding cancer in humans after dermal exposure to fuel oils. Unspecified skin tumors were identified in C3HF/Bd mice under the following exposure conditions: a 40-week exposure to 22.9 mg (but not 42.2 mg) JP-5 or 23.8 and 45.5 mg marine diesel fuel; a 60-week exposure to 5.7-42.2 mg (the highest incidence was at 11.4 mg) JP-5 or 11.9 and 23.8 mg (but not 45.5 mg) marine diesel fuel (Schultz et al. 1981). Tumors were more prevalent in females than males for JP-5 exposure. None of the control animals developed skin tumors and statistical analysis was not conducted. The tumor incidence was not dose related, and historical control data for this strain of mouse were not provided.

No skin cancer was reported in $B6C3F_1$ mice chronically exposed to 250 and 500 mg/kg/day of JP-5 (NTP/NIH 1986). There was a 2-6% incidence of squamous cell papilloma and/or carcinoma of the skin in $B6C3F_1$ mice chronically exposed to 250 (females only) or 500 (both sexes) mg/kg/day marine diesel fuel. The effect did not occur in the control groups; the statistical significance of these effects was not reported. Hepatocellular adenoma or carcinoma were noted in males exposed to 250 and 500 mg/kg/day marine diesel fuel. These effects did not occur in female mice at these doses. Malignant lymphomas were noted in females exposed to 250 mg/kg/day, but not 500 mg/kg/day, JP-5; therefore, no dose-response relationship was apparent for this effect. A significant negative trend in the incidence of malignant lymphomas was noted in males of the high-dose group.

The tumorigenic potential of API no. 2 fuel oil was evaluated by dermal application in male and female C3H/Bd$_f$ mice (Witschi et al. 1987). Fifty microliters of the fuel oil was applied neat, or as a 50% or 25% dilution (w/v, in acetone) three times per week. Negative controls consisted of mice treated with acetone or animals that received no treatment. Positive controls received 50, 25, or 12.5 mg of benzo[a]pyrene dissolved in 50 microliters of acetone. Over all the doses, 15 of the 150 mice developed skin tumors (the first tumor appeared at 75 to 80 weeks), while 299 of the 300 mice treated with benzo[a]pyrene developed skin tumors (first tumor appeared at 19 weeks). Neither of the negative control groups developed neoplastic lesions).

Similarly, the dermal carcinogenic activity of 6 Commercial no. 2 heating oils with varied boiling ranges, points of origin, and composition have been evaluated (Biles et al 1988). The heating oils were applied neat to male C3H/HeJ mice in 25 microliter aliquots, 3 times per week for lifetime or until all mice in the test group developed frank carcinomas. The negative controls received a "highly refined white mineral oil," and the positive controls received four concentrations of catalytically cracked clarified oil (known to produce a positive dermal tumorigenic response) diluted in white mineral oil. None of the negative control mice (140 animals) developed tumors, while all of the positive control groups displayed significantly increased incidence of tumor production. All of the Commercial no. 2 heating oils induced a significantly increased incidence of tumors (an incidence of 5 animals with tumor per 50 animals was statistically significant, and the heating oil incidence ranged from 5 to 11 per 50).

No increase in the incidence of tumor-bearing mice was noted in animals treated with 25 mg of undiluted petroleum diesel three times per week for up to 105 weeks in male and female C3H/HeN (diesel-treated had an incidence of 2/27 and mineral oil-treated negative controls did not develop any tumors). Jet A did, however, produce an increased incidence (26%) of tumors (primarily squamous cell carcinoma and fibrosarcoma) (Clark et al. 1988). It was noted that both types of fuels produced inflammatory and degenerative changes at the application that led to "early mortality" and that the nonneoplastic lesions and their attendant effects were so severe that the application of Jet A was discontinued at week 62.

Furnace oil (CAS no. 68474-30-2) was evaluated for skin carcinogenicity with both lifetime skinpainting and an initiation/promotion bioassay (Gerhart et al. 1988). Briefly, in the lifetime skinpainting study, 50 microliters of undiluted furnace oil was applied to 50 male C3H/HeN mice twice weekly for the lifetime of the animals. A sham negative control group of equal size was run concomitantly. In the initiation portion of the initiation/promotion bioassay, 30 CD-l mice received five treatments of 25 microliters or five treatments of 50 microliters of furnace oil, and both groups were subsequently treated with 50 microliters of 0.1 mg/mL phorbol-12-myristate-13-acetate (PMA) twice weekly for 25 weeks. Thirty mice received one treatment of 50 microliters of 9,10-dimethyl-1,2-benzanthracene (DMBA) or 50 microliters of acetone and subsequently were treated with 50 microliters of furnace oil twice weekly for 25 weeks in the promotion component of the investigation. (Note that Gerhart and coworkers [1988] indicated that the two different strains of mice were used: C3H mice, which are not commonly used in initiation/promotion studies, and CD-l mice,

which are considered to be a sensitive model and used extensively in initiation/promotion studies of carcinogenesis.) In the skin-painting studies, furnace oil produced a significant increase in the incidence (9/43 in furnace oil group and 0/49 in negative controls) of confirmed skin tumors (squamous cell carcinoma and fibrosarcoma). Two of the nine tumor-bearing animals developed squamous cell carcinoma in untreated areas of skin. In contrast, when furnace oil as an initiating agent, no increase in tumor incidence was observed in animals treated with either 25 or 50 microliters. However, a significant increase (12/30 vs l/30 in acetone initiated) in histologically confirmed tumor incidence (squamous cell carcinoma) was observed in mice that were treated with DMBA as an initiating agent and received furnace oil as the promoting agent.

The tumorigenic activity of no. 2 type fuel oil and hydrodesulfurized kerosene was examined utilizing an initiation/promotion assay with CD-l mice (API 1989). In the initiation phase of the study, mice received 5-50 microliter consecutive daily applications of the fuel oils and subsequently were treated with PMA (0.1 mg/mL) twice weekly for 25 weeks. In the promotion phase of the study, mice received a single application of DMBA (1.0 mg/mL) and were subsequently treated with one of the fuel oils twice weekly for 25 weeks. No significant difference in tumor incidence was noted in those animals treated with either of the fuel oils, when compared to the negative control (initiation with 50 microliters of acetone). However, both the incidence of confirmed tumor (18/30 in animals treated with no. 2 fuel oil as a promoting agent, 22/30 in animals receiving hydrodesulfurized kerosine as a promoting agent, and 0/30 in acetone-promoted negative controls) and the latency period were significantly different than those of the positive controls. The most prevalent tumors were squamous cell papillomas and keratoacanthomas.

The dermal carcinogenicity of mixtures of petroleum products that have a boiling range approximately equal to or greater than 370°C has been reported to be primarily related to the polycyclic aromatic hydrocarbon (PAH) content of the material (Biles 1988). The boiling ranges of the various fuel oils are as follows: fuel oil no. 1, 175-300°C; fuel oil no. l-D, 193-293°C; fuel oil no. 2, 160-360°C; fuel oil no. 2-D 282-338°C; fuel oil no. 4, l0l-588°C; fuel oil UNSP, 151-588°C (see Table 3-3). Whereas some fuel oils contain cracked stocks that are known to contain biologically active PAH, virgin distillate petroleum products (boiling range approximately 177°C to 370°C), which include various middle distillate fuel oils, primarily contain saturated species (Biles 1988). Although these virgin petroleum materials contain low concentrations of PAH, repeated application can induce dermal tumors. It has been reported that the tumorigenicity of three petroleum- and four coal-derived liquids

were not consistent with the PAH content of the test materials (Witschi et al. 1987). Similarly, the dermal carcinogenicity of 10 petroleum-derived fuels (including 6 formulations of no. 2 heating oil) did not appear to be "directly related" to the aromatic content or the presence of PAH in the materials (Biles et al. 1988). Gerhart and colleagues (1988) reported that furnace oil induced tumors through epigenetic or promotional means, even though it contained no detectable concentrations of four- and five-ring aromatics. In a 2-year skin-painting study of four petroleum middle distillates (including jet fuel), the authors suggested aromatic and sulfur heterocycles tested were not the source of tumorigenicity in middle distillates (Freeman et al. 1993). These results suggest that the tumorigenic potential of the middle distillates is not related to their PAH content.

It has been alternatively hypothesized that the carcinogenic activity of fuel oils is a secondary effect associated with dermal irritation (Biles et al. 1988; Clark et al. 1988; McKee et al. 1989). Biles and coworkers (1988) speculated that the irritating properties of middle distillate petroleum fuels played a role in the mechanism of dermal carcinogenesis in a lifetime skin-painting assay, although the data did not demonstrate a relationship. In fact, they noted that the test groups with the most severe "degree of epidermal degeneration and necrosis" demonstrated the lowest tumor yields. Repeated application of four petroleum-derived distillates (including Jet A and diesel) to mouse skin induced severe inflammation and degenerative changes; however, the severity and early onset of inflammation was not always predictive of tumorigenicity (Clark et al. 1988). Similarly, even though dermal application of dewaxed heavy paraffinic distillate led to a greater incidence of confirmed tumors than did furnace oil (26/48 and 9/43, respectively), furnace oil induced a greater incidence of nonneoplastic lesions (37/43 vs. 14/48) (Gerhart et al. 1988). McKee and coworkers (1989) ascribed the weak promotional activity of a lightly refined paraffinic oil to the irritation caused by its repeated application to mouse skin, partially based on findings that the whole oil and various fractions of the oil were negative for both mutagenic activity in bacteria and carcinogenic initiating activity.

The role of chronic acanthosis and inflammation in tumor promotion by a middle distillate has been investigated (Skisak 1991). Briefly, male CD-l mice received a single dermal treatment of 50 microliters of DMBA (initiation) and subsequently were treated with 25, 50, or 100 microliters (twice weekly for 25 weeks) of hydrodesulfurized kerosine (HK). Dose, washing after treatment, and topical application of dexamethasone were used to control inflammation. The mice treated with 100 microliters of HK had the greatest tumor incidence (35/53) and the highest degree of acanthosis throughout the study. While the tumor responses of the 25 and 50 microliter treated groups were

similar (14/54 and 13/54, respectively), the degree of acanthosis was much more pronounced in the mice treated with 50 microliters HK. Application of dexamethasone to animals treated with 50 microliters reduced the tumor incidence to 0, although acanthosis was still observed. It is interesting to note that washing the mice (1 to 2 hours after treatment) with an Ivory soap solution with 50 microliters of HK increased tumor incidence (22/53) compared to the group treated with 50 microliters HK but not washed. The authors concluded that although hyperplasia may play a role in the promoting activity, there are other factors involved.

In a 2-year skin-painting study designed to evaluate the role of skin irritation in the tumorigenicity of middle distillates, 37.5 microliters of jet fuel and steam cracked gas oil were applied two times per week, and jet fuel was also applied in an intermittent fashion (dosing was suspended when irritation was noted in 20% of the group and resumed when it was resolved in all but 20%) (Freeman et al. 1993). Intermittent dosing produced irritation that was less severe than dosing two times per week, and only l/50 intermittently dosed animals developed tumors, compared with 22/50 in the twice-weekly dosed group. Freeman and coworkers (1993) stated that chronic skin irritation was necessary, but insufficient for tumor induction. These data indicate that the irritant properties of fuel oils are not the ultimate cause of dermal carcinogenicity, although the irritation induced by the repeated application may play an important role in the mechanism of tumor formation.

Reliable LOAEL values for cancer effects after dermal exposure to fuel oils are recorded in Table 2-3.

2 . 3 TOXICOKINETICS

Few data were available concerning the absorption, distribution, metabolism, and excretion of fuel oils. Indirect evidence suggests that some fuel oils may be absorbed through the respiratory tract, the gastrointestinal tract, and percutaneously. Although data concerning the metabolism of fuel oils in humans could not be located, a single animal study suggested that cytochrome P-448 may be involved in the metabolism of fuel oil no. 2 (Rahimtula et al. 1982). No quantitative data were found regarding the excretion of fuel oils.

2.3.1 Absorption

2.3.1.1 Inhalation Exposure

No studies were located regarding the absorption of fuel oils in humans or animals after inhalation exposure. However, indirect evidence of gastrointestinal, cardiovascular, hematological, renal, and/or dermal/ocular effects from case reports in which two pilots were exposed to JP-5 vapor while flying a small airplane and another man was exposed to diesel fuel vapor while driving a truck indicate that these fuel oils can be absorbed following inhalation exposure in humans (Porter 1990; Reidenberg et al. 1964). Effects on animals acutely exposed to fuel oils by inhalation provide indirect evidence for inhalation absorption in animals (Casaco et al. 1985c; Garcia et al. 1988b; Kainz and White 1984).

2.3.1.2 Oral Exposure

No quantitative data were located regarding the absorption of fuel oils in humans after oral exposure. However, there is evidence that absorption from the gastrointestinal tract occurs following ingestion of kerosene by humans (Subcommittee on Accidental Poisoning 1962). In a study of 422 cases, pulmonary complications were noted in 11 individuals, even though gastric lavage was not administered nor was vomiting reported (vomiting and gastric lavage could cause aspiration of the kerosene, thus contributing to the respiratory effects). In this same report, pulmonary complications occurred in a higher percentage of the individuals that did not receive lavage than in those that were treated by gastric lavage. This suggests that pulmonary effects of fuel oils may also be the result of systemic toxicity. Further, administration of gastric lavage within 30 minutes of ingestion further decreased the number of affected individuals, suggesting that removal of kerosene from the stomach may have prevented its absorption and subsequent toxicity in these cases. In this same study, there is also indirect evidence that aspiration from vomiting may induce pulmonary effects since there were more individuals with respiratory complications when vomiting occurred than when it did not, regardless of the administration of gastric lavage. It is possible that both absorption and aspiration contribute to the respiratory effects in these individuals.

Limited animal data and indirect evidence indicate kerosene is poorly absorbed from the gastrointestinal tract. In one study, kerosene labeled with ^{3}H-toluene or ^{14}C-hexadecane was given to tracheotomized baboons (15 mL/kg) by nasogastric tube (Mann et al. 1977). The isotopes were

recovered after 6 hours from the brain, lung, liver, spleen, heart, and kidney; however, the amounts distributed to the tissue were very small, suggesting that gastrointestinal tract absorption was slight. The potential absorption of ingested kerosene into the lungs was tested by comparing respiratory effects from oral exposures in nontracheotomized and tracheotomized monkeys (Wolfsdorf and Kundig 1972). The tracheotomized monkeys who received the kerosene via nasogastric tube could not aspirate the kerosene; thus, the potential for respiratory exposure by aspiration was prevented. Lung lesions were seen in the nontracheotomized monkeys, but no lesions were seen in the tracheotomized monkeys. These data suggest that aspiration of fuel oils, not absorption, is the underlying cause of the respiratory effects. A lack of pulmonary toxicity was reported in dogs in which aspiration was prevented. This study supports the theory that pulmonary toxicity following kerosene ingestion is the result of aspiration of kerosene into the lungs rather than absorption from the gastrointestinal tract (Dice et al. 1982).

2.3.1.3 Dermal Exposure

Studies of effects on animals following acute, intermediate, and chronic dermal exposure to marine diesel fuel and JP-5 fuel provide evidence for dermal absorption (NTP/NIH 1986). Case reports concerning a man who washed his hair with an unknown amount of diesel fuel (Barrientos et al. 1977) and a man who washed his hands with an unspecified diesel fuel over several weeks (Crisp et al. 1979) provide possible evidence for dermal absorption, but effects resulting from inhalation versus dermal exposure could not be distinguished in these cases. No other data on the absorption of fuel oils following dermal exposure in humans or animals were located.

Some case studies suggest that dermal exposure to the vapor of diesel fuel may also result in absorption via the skin. The studies identify one individual with only vapor exposure and two others with vapor and/or direct dermal contact with diesel fuel; individuals developed acute renal failure or renal necrosis (Barrientos et al. 1977; Crisp et al. 1979; Reidenberg et al. 1964). Also, dermal exposures to marine diesel fuel and JP-5 in mice induced renal damage (Easely et al. 1982). No studies were located that directly tested dermal absorption of fuel oil vapor.

2.3.1.4 Other Routes of Exposure

Dogs that had undergone transection of the esophagus exhibited no respiratory or gastrointestinal effects after injection by syringe of 5, 10, 20, or 30 mL of kerosene (4-17.9 rnL/kg) directly into the stomach. This study provides indirect evidence that kerosene is poorly absorbed from the gastrointestinal tract (Wolfe et al. 1970), or alternatively, kerosene is adsorbed but not grossly toxic unless it is aspirated.

2.3.2 Distribution

2.3.2.1 Inhalation Exposure

No studies were located regarding the distribution of fuel oils in humans or animals after inhalation exposure.

2.3.2.2 Oral Exposure

No studies were located regarding the distribution of fuel oils in humans after oral exposure.

Very limited animal data indicate that kerosene is absorbed and distributed to various tissues (Mann et al. 1977). Kerosene, labelled with ^{3}H-toluene or ^{14}C-hexadecane, was given to tracheotomized baboons (15 mL/kg) by nasogastric tube (Mann et al. 1977). Radioactivity was recovered from the brain, lung, liver, spleen, heart, and kidney after 6 hours. ^{3}H-Toluene was absorbed and taken up by most tissues to a greater extent than was ^{14}C-hexadecane; however, the amounts absorbed and distributed were very small (Mann et al. 1977).

2.3.2.3 Dermal Exposure

No studies were located regarding the distribution of fuel oils in humans or animals after dermal exposure.

2.3.3 Metabolism

No studies were located regarding the metabolic pathway of fuel oils in humans. In one animal study, fuel oil no. 2 applied to the skin of rats induced cutaneous aryl hydrocarbon hydroxylase activity in rat skin microsomal preparations by causing a three-fold induction of benzo(a)pyrene (BaP) 3-hydroxylase activity (Rahimtula et al. 1982). In addition, BaP 3-hydroxylase activity was selectively inhibited by α-naphthoflavone, but not by metyrapone, suggesting that cytochrome P-448 enzymes are induced and may participate in the metabolism of this fuel oil (Rahimtula et al. 1982).

2.3.4 Excretion

There is no quantitative information on the excretion of fuel oils following inhalation, oral, or dermal exposure in humans or animals.

2.3.5 Mechanisms of Action

The primary risk from ingestion of kerosene is aspiration during emesis, which may cause pneumonitis. A number of studies have investigated the biochemical mechanism of the lung response to the exposure of large concentrations of aerosolized kerosene (Casaco et al. 1982, 1985b, 1985c). It was suggested that kerosene may induce asthma-like symptoms by acting on the parasympathetic nervous system either through a direct effect on the vagus nerve or by inhibiting acetylcholinesterase, resulting in bronchoconstriction. Garcia and Gonzalez (1985), based on their observations that kerosene caused an ''increase in Ca^{2+}-dependent ATP hydrolysis without increase in the rate of net calcium accumulation,'' concluded that kerosene induced an effect on the membrane of the sarcoplasmic reticulum vesicles and suggested that the mechanism of kerosene-induced bronchoconstriction may involve changes in the ionic flow across the cellular membranes to prolong muscle contraction.

Although generalizations regarding the hematological effects of fuel oils on humans cannot be made, the effect of kerosene on the first two steps of the heme synthetic pathway has been studied in an animal model. Both hepatic δ-aminolevulinic acid (δ-ALA) dehydratase and δ-ALA synthetase activities were decreased in female rats after intraperitoneal injection of kerosene, while heme oxygenase was unaffected (Rao and Pandya 1980). Since δ-ALA synthetase is the rate-limiting enzyme of the heme biosynthesis pathway, hepatic heme biosynthesis may be inhibited by kerosene.

It is conceivable that this may be related to the extramedullary hematopoiesis reported in other studies (NTWNIH 1986); however, there are no direct data to support this.

The biochemical mechanism of CNS depression seen with fuel oils and common to many organic solvents has not been elucidated. The mechanism of carcinogenesis associated with various formulations of fuel oils is unknown.

2.4 RELEVANCE TO PUBLIC HEALTH

Information regarding health effects of fuel oils in humans and animals is available for the inhalation, oral, and dermal routes of exposure. Most of the information in humans is from cases of accidental ingestion of kerosene that resulted in respiratory, neurotoxic, and to a lesser extent, gastrointestinal effects. In addition, a few case studies have identified these effects as well as cardiovascular, hematological, and renal effects in humans after inhalation and/or dermal exposures to fuel oils. Fuel oils appear to be eye and skin irritants in both animals and humans following direct contact. Animal data exist for most systemic effects; however, the data are inconclusive for many of the endpoints. Further, a number of the animal studies utilized an aerosol for exposure, and it should be noted that the toxicity from an aerosol will typically vary from that of a vapor (the probable form of exposure). The available epidemiological studies are generally inconclusive, since they cannot exclusively associate exposures to fuel oils with the adverse effects reported.

Minimal Risk Levels for Fuel Oils

Inhalation

- An MRL of 0.02 mg/m^3 has been derived for acute inhalation exposure to diesel fuel (fuel oil no. 2). The MRL is based on dose-related neurobehavioral effects (mild transient ataxia and CNS depression), beginning at 65 mg/m^3, in mice exposed to airborne concentrations of fuel oil of 65, 135, and 204 mg/m^3.

- An MRL of 0.01 mg/m^3 has been derived for the intermediate inhalation exposure to kerosene (also termed fuel oil no. 1). The MRL is based on decreased blood glucose levels noted in male rats subsequent to exposure to airborne concentrations of kerosene averaging

58 mg/m^3 for 6 hours/day, 6 days/week for 14 weeks. Rats exposed to an airborne concentration of kerosene averaging 231 mg/m^3 showed a decrease in blood glucose titers, as well as increased circulating levels of lactate and pyruvate. In addition, mice exposed to 150 or 750 mg/m^3 or airborne JP-5 showed hepatocellular fatty changes and vacuolization at the LOAEL of 150 mg/m^3 (Gawororski et al. 1984). Finally, no systemic or neurological effects were observed in rats or dogs exposed to a deodorized kerosene concentration of 100 mg/m^3 (Carpenter et al. 1976).

No chronic inhalation MRLs were derived for fuel oils because available data were not suitable for MRL derivation. Studies that report lethality or biochemical alterations without attendant pathology cannot be used for MRL determination

Oral

No acute, intermediate, or chronic oral MRLs were derived for fuel oils because available data were not suitable for MRL derivation. Studies that report lethality at the lowest dose tested cannot be used for MRL determinations. Hepatocyte necrosis reported by Parker et al. (1981) occurred at a dose greater than dose levels at which serious effects occurred in other studies, making these data unsuitable for derivation of an MRL.

Death. No quantitative lethality data for humans were located from inhalation or dermal exposure to fuel oils. Based on case studies reporting deaths in humans following ingestion of kerosene, estimated lethal doses of kerosene range from 1,890 to 16,789 mg/kg (Dudin et al. 1991; Santhanakrishnan and Chithra 1978). These lethal doses are based upon specific cases in which 30 or 200 mL of kerosene were ingested by l- and 2-year-old children. No lethality was reported for children from 10 months to 5 years old following ingestion of 126-877.2 mg/kg or 1,754 mg/kg of kerosene, respectively (Dudin et al. 1991). There are no human data that identify lethal oral doses in adults, and no dose-response data are available for humans; therefore, it is not possible to determine a specific oral dose of kerosene at which lethality in humans would not be expected to occur.

A single 6-hour exposure to 4,000 mg/m^3 diesel fuel aerosol (Dalbey and Lock 1983) and repeated exposures (once or three times per week for a total of nine exposures) for 2 hours to 6,000 mg/m^3 or 6 hours to 2,000 mg/m^3 diesel fuel aerosol (Dalbey et al. 1987) were lethal to rats. However, a single

4-hour exposure to 6,000 mg/m^3 diesel fuel aerosol was not lethal to rats (Dalbey and Lock 1983). The variability of these diesel fuel data appear to indicate that the cumulative dose may be more important than the airborne concentration of fuel oil aerosol, although human exposure to such high concentration of a fuel oil aerosol seems unlikely. Acute and intermediate exposures to moderate through high concentrations of marine diesel fuel, diesel fuel vapor or aerosol, fuel oil UNSP, JP-5, and kerosene (API 1979c, 19798; Cowan and Jenkins 1981; Gaworski et al. 1984; Lock et al. 1984; Vernot et al. 1990d) ranging from 50 mg/m^3 marine diesel vapor to 5,000 mg/m^3 kerosene were not lethal to rats. Although it appears that fuel oils may be lethal to humans only at vapor concentrations that occur at elevated temperatures or as the result of exposure to an aerosol, these data are not sufficient for such generalizations to be drawn concerning the lethal concentration or cumulative dose of fuel oils.

The acute oral LD$_{50}$ in guinea pigs and rabbits for kerosene has been reported to be 16,320 mg/kg and 22,720 mg/kg, respectively (Deichmann et al. 1944). These data suggest that guinea pigs may be more sensitive to kerosene than rabbits. Similarly, a lethal dose of kerosene of 6,400 mg/kg has been reported in calves (Rowe et al. 1973), but the lethal dose for rats is 12,000 mg/kg (Muralidhara et al. 1982). Comparison of these data is problematic; however, they do suggest that species differences and age sensitivity may exist for oral kerosene toxicity, although such differences have not been established.

For oral exposures, different fuel oils have differing lethality profiles in rats. Acute lethal doses in rats were reported to be 12,000 mg/kg for kerosene (Muralidhara et al. 1982) and 47,300 mg/kg for JP-5 (Parker et al. 1981). However, an oral dose of 12,200 mg/kg of Deobase$^®$ was not lethal in rats (Muralidhara et al. 1982). Although differences in the oral toxicity of fuel oils and differences in species thresholds of toxicity may exist, the oral toxicity of fuel oils is relatively low. The intestinal absorption of fuel oils is also relatively low, and aspiration, with its resultant pulmonary effects, is the primary risk from the ingestion of fuel oils.

Daily dermal exposures for 1 week to 0.1 mL kerosene were not lethal to male mice (Upreti et al. 1989). A minimum lethal dose of 30,000 mg/kg/day was reported for JP-5 from acute dermal exposure in mice, but this dose was decreased to 2,000 and 250 mg/kg/day following intermediate and chronic exposures, respectively (NTP/NIH 1986). A similar trend was also reported for dermal

toxicity in mice exposed to marine diesel fuels (NTP/NIH 1986). Conclusions cannot be drawn from the available data regarding dermal exposure by humans to fuel oils near hazardous waste sites.

Systemic Effects

Respiratory Effects. There are epidemiological data that found no evidence of respiratory toxicity in children from exposure to kerosene vapor and combustion products from kerosene stoves used for cooking (Azizi and Henry 1991; Tominaga and Itoh 1985); however, the importance of such exposures to individuals living near hazardous waste sites or in the workplace is uncertain. Another epidemiological study noted thoracic oppression in workers that were chronically exposed to jet fuel by the inhalation, oral, and/or dermal routes (Knave et al. 1978). However, the jet fuels were not specified in this study, and therefore, these exposures may not necessarily include fuel oils such as JP-5. Only one case study was found that reported effusions and alveolar infiltrations from dermal and/or inhalation exposure to diesel fuel when used as a shampoo (Barrientos et al. 1977). A low concentration of deodorized kerosene vapor was not irritating to the throat in humans (Carpenter et al. 1976). Animal data indicate that functional parameters of the lung may be affected (Casaco et al. 1982) and bronchoconstriction may occur (Casaco et al. 1982; Garcia et al. 1988b) from acute inhalation of kerosene aerosol. In one study, intermediate exposure to diesel fuel aerosol induced damage to the lung parenchyma of rats (Dalbey et al. 1987). This study found that an increase in the frequency of exposure was more likely to induce respiratory effects than the exposure dose or duration. However, in each of these cases of respiratory toxicity, relatively high exposure levels were used. Other animal studies have found no histopathological evidence of respiratory toxicity following relatively low to moderate intermediate inhalation or acute, intermediate, and chronic dermal exposures to various fuel oils (Carpenter et al. 1976; Lock et al. 1984; NTP/NIH 1986; Upreti et al. 1989). These data suggest that bronchoconstriction or respiratory impairment may occur in humans at high inhalation or dermal exposure levels of kerosene or diesel fuel. Relatively low or moderate exposure levels may also affect sensitive members of the population, but this cannot be determined from the data. The data also indicate that humans who are frequently exposed to fuel oils, such as those exposed occupationally, may be at greater risk of developing respiratory lesions than those with single or less frequent exposures.

Ingestion of fuel oils, kerosene in particular, has been shown to induce respiratory effects in humans, although it appears that aspiration is the primary cause of the pulmonary toxicity and the most serious

feature of ingestion. Numerous studies in animals and humans have evidenced the introduction of kerosene into the lungs during vomitus and subsequent manifestation of deleterious effects in the respiratory tract (Coruh and Inal 1966; Dice et al. 1982; Majeed et al. 1981; Nouri and Al-Rahim 1970; Wolfe et al. 1970; Wolfsdorf and Kundig 1972). However, limited absorption from the gastrointestinal tract into the lungs may also occur (Mann et al. 1977).

Specific effects that have occurred in humans following ingestion of kerosene include bronchopneumonia, bronchitis, pneumonitis, lung infiltrates and effusions, cough, dyspnea, hypoxia, and tachypnea (Akamaguna and Odita 1983; Aldy et al. 1978; Annobil 1983; Annobil and Ogunbiyi 1991; Dudin et al. 1991; Mahdi 1988; Santhanakrishnan and Chithra 1978; St. John 1982). The animal data describing respiratory toxicity are limited but are consistent with the findings in humans. Oral exposure data for humans are only available for kerosene; therefore, no conclusions can be made regarding the respiratory toxicity of other fuel oils. However, the similar composition of the various fuel oils suggests that the effects may also be similar.

A number of studies have investigated the biochemical mechanism of lung response to concentrations of aerosolized kerosene (ranging up to a mean of 32.5 mg/L). It was indicated that kerosene may induce asthma-like symptoms by acting on the parasympathetic pathway involving a direct effect on the vagus nerve or by inhibiting acetylcholinesterase, thus increasing the acetylcholine level in the trachea, resulting in bronchoconstriction (Casaco et al. 1982, 1985b, 1985c). It has also been reported that kerosene can affect the calcium pump of the rabbit sarcoplasmic reticulum (Garcia and Gonzalez 1985), suggesting that the mechanism of kerosene-induced bronchoconstriction may involve changes in the ionic flow across the cellular membranes to prolong muscle contraction.

Curdiovasculur Effects. Mild hypertension from acute inhalation of JP-5 vapor (Porter 1990) or diesel fuel vapor (Reidenberg et al. 1964) and palpitations from chronic inhalation, dermal, and/or oral exposures to unspecified jet fuels have been reported in humans (Knave et al. 1978). Tachycardia and cardiomegaly were reported in children following acute ingestion of kerosene (Akamaguna and Odita 1983; Coruh and Inal 1966). Most of the available animal studies found no histopathological changes or organ weight changes in the cardiovascular system of rats and mice following inhalation, oral, or dermal exposures to various fuel oils, including kerosene (Carpenter et al. 1976; Lock et al. 1984; Muralidhara et al. 1982; NTP/NIH 1986). However, there are some limited data regarding cardiac effects. Inhalation of 20,400-34,000 mg/m^3 of kerosene vapor induced aortic plaques in guinea pigs.

Further, significant increases in total serum cholesterol and decreases in HDL were noted (Noa and Illnait 1987a). Vasodilation was reported in mice exposed by inhalation to diesel fuel no. 2 aerosol (Kainz and White 1984). Aspiration of kerosene decreased heart rate and mean arterial blood pressure in dogs (Goodwin et al. 1988). Because most of the toxicity findings are specific for kerosene, it is difficult to draw conclusions regarding the cardiac effects of other fuel oils. However, the similar composition of the various fuel oils suggests that the effects may also be similar. The potential for cardiac effects in humans at hazardous waste sites exists; the risk is increased if the individuals are exposed in confined spaces.

Gastrointestinal Effects. Inhalation of JP-5 vapor induced nausea in one individual. Abdominal cramps, vomiting, and diarrhea occurred in another man who was exposed to diesel fuel vapor for 10 days (Reidenberg et al. 1964). Inhalation and/or dermal exposure to diesel fuel induced epigastric pains in a man who washed his hands with diesel fuel (Crisp et al. 1979) and nausea, abdominal cramps, and diarrhea in another individual who used it as a shampoo (Barrientos et al. 1977). Ingestion of kerosene induces more severe effects: vomiting, abdominal pain and/or distension, gastroenteritis, bleeding, and diarrhea (Akamaguna and Odita 1983; Aldy et al. 1978; Mahdi 1988; Majeed et al. 1981; Saksena 1969; St. John 1982; Nouri and Al-Rahim 1970). No histopathological changes in the gastrointestinal system were reported in animals exposed to various fuel oils by the inhalation and dermal routes of exposure (Carpenter et al. 1976; Lock et al. 1984; NTP/NIH 1986). Also, acute oral exposure to kerosene or Deobase did not induce diarrhea in rats (Muralidhara et al. 1982). These data, though limited, indicate that species variations may exist between humans and rats following oral exposure to kerosene. Although the data are largely anecdotal, they strongly suggest that gastrointestinal effects are induced in humans by ingestion of kerosene, inhalation of JP-5 or diesel fuel vapor, and dermal contact with or inhalation of diesel fuel. It has not been determined whether these effects would occur as the result of exposure to other fuel oils and if they did at what exposure levels such effects would occur.

Hematological Effects. Subcutaneous hemorrhage, mild nose bleeds, low platelet counts, and retinal arteriole constriction were reported for a man who was exposed to diesel fuel vapor for 10 days (Reidenberg et al. 1964). These effects may be indicative of blood clotting problems. Decreased hemoglobin concentration and an increase in erythrocyte sedimentation rate were noted in one man after washing his hands with diesel fuel over several weeks (Crisp et al. 1979). Of 12 patients admitted to the pediatric intensive care unit of a children's hospital during a 5-year period due to

respiratory distress associated with hydrocarbon aspiration, 3 showed signs of intravascular hemolysis. A fourth patient, who had ingested kerosene, had clinically insignificant hemolysis (Algren and Rodgers 1992). Increases in leukocyte counts from acute ingestion of kerosene (Dudin et al. 1991; Majeed et al. 1981; Nouri and Al-Rahim 1970) have; also been reported in humans. No hematological effects were noted two individuals exposed to JP-5 for a few hours (Porter 1990).

No hematological or splenic effects were reported in rats following oral exposure to kerosene (Muralidhara et al. 1982), nor were hematological and splenic effects noted in rats and dogs following inhalation of deodorized kerosene (Carpenter et al. 1976), in rats that inhaled diesel fuel (Lock et al. 1984), or in rats following oral administration of Deobase (Muralidhara et al. 1982).

Decreases in hemoglobin concentration and increases in erythrocyte, white blood cell, and polymorphonuclear leukocyte concentrations were noted in mice after acute dermal exposure to kerosene. A decrease in the splenic relative weight, which was not accompanied by histopathological changes, was also noted (Upreti et al. 1989). Oral exposure to JP-5 increased hematocrit levels, decreased white blood cell counts, and increased erythrocyte counts in rats (Parker et al. 1981). However, inhalation of diesel fuel aerosol induced decreases in the mean red blood cell count in rats and had no effect on white blood cells (Dalbey et al. 1987).

The effect of kerosene on the first two steps of the heme synthetic pathway was studied in an animal model and demonstrated that hepatic δ-aminolevulinic acid (δ-ALA) dehydratase and δ-ALA synthetase activities were decreased in female rats after intraperitoneal injection of kerosene. Further, heme oxygenase was not affected by kerosene under these conditions (Rao and Pandya 1980). Since δ-ALA synthetase is the rate-limiting enzyme of the heme biosynthesis pathway, hepatic heme biosynthesis may be inhibited by kerosene.

Generalizations regarding the hematological effects of fuel oils on humans cannot be made because the available data suggest that each fuel oil may behave differently, that species variation may exist, and that exposure route may play a role. The similar effects in humans and mice exposed to kerosene suggest that kerosene may not be species specific and may act similarly on the hematopoietic system regardless of the exposure route. However, there are not enough data to determine whether these generalizations can be made. The limited data suggest that inhalation or dermal exposure to diesel fuel and ingestion of kerosene can induce hematological effects in some individuals; however, it is not

known if these effects would occur in most individuals, although Algren and Rodgers suggest that hydrocarbon ingestion may frequently effect intravascular hemolysis.

Musculoskeletal Effects. No studies were located regarding musculoskeletal effects in humans after inhalation, oral, or dermal exposure to fuel oils. No histopathological changes were noted in the musculoskeletal systems of rats and dogs exposed by inhalation to up to 100 mg/m^3 deodorized kerosene vapor for 13 weeks (Carpenter et al. 1976). Mice treated dermally with marine diesel fuel and JP-5 (up to 500 mg/kg/day) also displayed no detectable adverse effects to the musculoskeletal system (NTP/NIH 1986). The limited information available on animals is not sufficient to assess its relevance to human health.

Hepatic Effects. Histopathological examination revealed slight cellular infiltration and mild vacuolization of the livers of rats following gavage with kerosene or Deobase$^®$ organ weights were not affected (Muralidhara et al. 1982). Gavage with JP-5 induced increases in serum levels of hepatic enzymes, hepatocyte necrosis, and vacuolization of the periportal hepatocytes in rats (Parker et al. 1981). Inhalation of 231 mg/m^3 kerosene vapor induced increases in blood lactate and pyruvate levels; exposure to 58 mg/m^3 kerosene vapor induced decreases in blood glucose levels in rats (Starek and Vojtisek 1986). Neither rats nor dogs exhibited histopathological changes in the liver following inhalation exposure to 20, 48, or 100 mg/m^3 deodorized kerosene vapor (Carpenter et al. 1976) or following inhalation of up to 1,500 mg/m^3 diesel fuel aerosol by rats (Lock et al. 1984). No histopathological changes were noted in the livers of mice following acute dermal exposures to 0.1 mL kerosene (Upreti et al. 1989). Slight hepatic karyomegaly was noted in mice dermally exposed to 500-8,000 mg/kg/day JP-5 for 13 weeks (NTP/NIH 1986). These data suggest that fuel oils may be of concern to humans because they affected rats by inhalation and oral exposures and mice by dermal exposures. However, no human data are available for inhalation, oral, or dermal exposures to fuel oils with regard to hepatic toxicity. Therefore, the available information is not sufficient to assess the relevance to human health.

Renal Effects. In one individual, acute renal failure was noted from inhalation and/or dermal exposure to diesel fuel which was used as a shampoo. Biopsy detected tubular dilation, mitosis, and vacuolization in renal cells, and some cellular proliferation in the glomerulus in this individual (Barrientos et al. 1977). Another individual experienced renal failure following inhalation of diesel fuel vapor for 10 days (Reidenberg et al. 1964). Renal necrosis developed in one man after washing

his hands with diesel fuel over several weeks (Crisp et al. 1979). Urinalysis was normal following inhalation of JP-5 by two individuals or ingestion of kerosene by numerous individuals (Dudin et al. 1991; Mahdi 1988; Nouri and Al-Rahim 1970; Porter 1990).

Renal lesions have been produced in mice by dermal application of JP-5 or marine diesel fuel. The inability to duplicate these lesions with intraperitoneal administration suggested that skin application, in particular the alteration of skin following repeated dermal application, was necessary to produce the renal toxicity, and that the renal effects appeared to be secondary to skin injury (Easley et al. 1982). Lymphocytic inflammation has been induced in the urinary bladder of mice with chronic dermal application of JP-5 or marine diesel fuel (NTP/NIH 1986). However, acute and intermediate dermal exposures to kerosene and JP-5, respectively, were not toxic to the renal system of mice (Upreti et al. 1989; NTP/NIH 1986). Although renal damage has occurred in a few individuals exposed to diesel fuel, it is not known if the general population would exhibit these effects following exposure to these fuel oils. The effects of JP-5 and kerosene on humans is not known.

Inhalation of JP-5 or marine diesel fuel vapors and oral exposure to JP-5 induce a nephropathy that is unique to male rats (Bruner 1984; Cowan and Jenkins 1981; Gaworski et al. 1984; Parker et al. 1981). The progression of this lesion has been noted in several studies, including studies conducted on the hydrocarbon decalin (decahydronaphthalene) (Alden 1986; Bruner 1984; Cowan and Jenkins 1981; Gaworski et al. 1984; Parker et al. 1981). Specifically, hyaline droplets are formed in the cytoplasm of the proximal tubule cells of the cortex. The hyaline droplets contain high concentrations of the protein $\alpha_{2\mu}$-globulin. It is believed that this protein accumulates in the cytoplasm of the renal tubule cells because the degradation of $\alpha_{2\mu}$-globulin is slowed as a result of to binding with specific substances, such as fuel oils, or their metabolites. The cells die and are sloughed off. The tubules near the corticomedullary junction become dilated and are eventually filled with coarsely granular casts and necrotic debris. This results in nephron obstruction and chronic necrosis.

The nephropathy induced by accumulation of this protein has not been noted in female rats, female mice (studies conducted on male mice were not located), or dogs of either sex when exposed under similar conditions to either JP-5 or marine diesel fuel vapors (Bruner 1984; Cowan and Jenkins 1981; Gaworski et al. 1984). There is no evidence of renal necrosis in humans acutely exposed to JP-5 vapor (Porter 1990). In a case report, one individual exposed to an unspecified diesel fuel for several weeks exhibited acute tubular necrosis (Crisp et al. 1979). However, renal necrosis did not occur in

two other individuals acutely exposed to diesel fuel vapor (Barrientos et al. 1977; Reidenberg et al. 1964), although they did exhibit acute renal failure. Based on available data, it does not appear that the nephrotoxicity attributable to the $\alpha_{2\mu}$-globulin syndrome observed in male rats is relevant to humans (Olson et al. 1990).

Dermal Effects. Oral and/or dermal exposure to kerosene induced blisters, erythema, and peeling skin in two cases (Annobil 1988). Dose-related effects in humans from dermal exposures to fuel oils are based upon limited information. Case studies describe numerous effects in or on the skin following dermal exposure to kerosene. These effects are itching, blisters, reddening, flaccid bullae, pustules, soreness, burning, swelling, and denudation (Annobil 1988; Jee et al. 1985; Mosconi et al. 1988; Tagami and Ogino 1973). There are limited data suggesting that epidermal damage may be induced by kerosene by impairing protein synthesis, but not DNA or collagen synthesis, in the epidermis (Lupulescu and Birmingham 1975). However, these data are insufficient to identify the toxic effects that may occur in humans following dermal exposure to kerosene. Also, cellular destruction was noted in humans from dermal exposure to kerosene (Lupulescu and Birmingham 1976; Lupulescu et al. 1973), but the implications of these effects have yet to be determined.

Acute, intermediate, and chronic dermal exposures to marine diesel fuel and JP-5 have induced various degrees of dose-dependent dermatitis in mice (Easley et al. 1982; NTP/NIH 1986). Manifestations of the dermatitis include: acanthosis, inflammation, parakeratosis, and hyperkeratosis (NTP/NIH 1986). Dermal irritation was induced in mice by acute dermal exposure to kerosene (Upreti et al. 1989). It is possible that dermal sensitization by fuel oils occurs only following acute exposures since sensitization was noted in guinea pigs acutely exposed to JP-5 or marine diesel fuel vapors but not following intermediate exposures to JP-5 or diesel fuel vapors (API 1979f; Cowan and Jenkins 1981). However, delayed sensitization was not induced in guinea pigs treated with diesel fuel (Beck et al. 1984), three formulations of No. 2 fuel oil (Beck et al. 1984), JP-5 (Schultz et al. 1981), or diesel fuel marine (Schultz et al. 1981). Diesel fuel marine and JP-5 did induce skin irritation in guinea pigs (API 1979f). No signs of dermal lesions were noted in rats following repeated whole-body inhalation exposure to diesel fuel aerosol (Lock et al. 1984). Whereas dermal exposure to fuel oils (liquid or vapor) would be expected to induce irritation or possibly dermatitis, the data are not adequate to evaluate delayed skin sensitization. Furthermore, the data are insufficient to assess the dermal effects of fuel oils when exposure occurs via the oral or respiratory routes.

Ocular Effects. JP-5 vapor were irritating to the eyes of two individuals and were associated with hyperemic conjunctiva in one case (Porter 1990). Eye irritation was also reported in workers who were chronically exposed to unspecified jet fuels, which may or may not include JP-5 (Knave et al. 1978). Deodorized kerosene vapor were shown to induce eye irritation in some individuals (Carpenter et al. 1976). Dermal exposure to diesel fuel and/or inhalation of its vapor was associated with subconjunctival hemorrhages in an individual who used it as a shampoo (Barrientos et al. 1977). In the only available animal studies, rabbits exposed dermally to diesel fuel (API 1979b), marine diesel fuel, or JP-5 (Cowan and Jenkins 1981; Schultz et al. 1981) showed no signs of ocular irritation. These data suggest that fuel oils, in general, may induce eye irritation in some individuals, although only one or two individuals exhibited ocular or dermal effects from airborne exposures to fuel oils. Irritation is likely to be more severe if exposure occurs in confined spaces.

Body Weight Effects. In mice, dose-dependent decreases in body weight were induced by intermediate and chronic dermal exposures to marine diesel fuel. In addition, dose-dependent decreases in body weight were induced in mice by acute and intermediate dermal exposures to JP-5 (NTP/NIH 1986; Schultz et al. 1981). Both food and water consumption were decreased in mice exposed to diesel fuel no. 2 aerosol for 8 hours/day, 5 consecutive days (Kainz and White 1984); in rats exposed to diesel fuel for 2 or 6 hours, once or 3 times per week (for a total of 9 exposures) (Dalbey et al. 1987); and in rats exposed for 4 hours per day, twice per week for 13 weeks (Lock et al. 1984). None of these effects were noted in mice following acute dermal exposures to kerosene (Upreti et al. 1989). No conclusions can be made regarding human health from the animal data since the significance of decreased body weight and food and water consumption with regard to humans cannot be determined.

Other Systemic Effects. Inhalation and/or dermal exposure to diesel fuel has been associated with edema in two individuals (Crisp et al. 1979; Reidenberg et al. 1964). In one of these cases, loin pains, thirst, and severe exhaustion were also reported (Crisp et al. 1979). Several case studies reported fever in children following acute ingestion of kerosene (Akamaguna and Odita 1983; Aldy et al. 1978; Dudin et al. 1991; Mahdi 1988; Majeed et al. 1981; Nouri and Al-Rahim 1970; Saksena 1969; St. John 1982; Subcommittee on Accidental Poisoning 1962). The effects of oral exposure to kerosene in children cannot be used to predict possible effects in adults or the effects of other fuel oils by this route without additional information. Similarly, it cannot be determined whether the effects of diesel

fuel noted in humans would occur from exposure to other fuel oils or in the general population since limited information is available.

Immunological and Lymphoreticular Effects. No studies were located regarding immunotoxicity in humans after inhalation, oral, or dermal exposure or in animals following inhalation or oral exposure to fuel oils. Dermal application of JP-5 induced granulocytic hyperplasia in the bone marrow and hyperplasia in the lymph nodes of mice. Dermal treatment of mice with marine diesel fuel induced plasmacytosis in the lymph nodes; this effect was secondary to dermatitis (NTP/NIH 1986). Decreases in the relative weights of the lymph nodes and thymus were noted in mice following dermal exposure to kerosene (Upreti et al. 1989). In addition, thymocyte counts, bone marrow nucleated cell counts, thymic cortical lymphocytes, and the cellularity of the thymic lobules were decreased. Increases in the cellular populations of the popliteal lymph nodes and the cell population of the axial lymph nodes were also present. These studies indicate that fuel oils may have an effect on the immune system of mice, although the toxicological significance of these effects cannot be determined from the data. There are not enough data to determine whether fuel oils would induce immunological effects in humans.

Data regarding changes in white blood cell counts were found; however, it cannot be determined whether these changes indicate hematological or immunological toxicity. Increases in leukocyte counts from acute ingestion of kerosene (Dudin et al. 1991; Majeed et al. 1981; Nouri and Al-Rahim 1970) have been reported in humans. Increases white blood cell and polymorphonuclear leukocyte concentrations were noted in mice after acute dermal exposure to kerosene (Upreti et al. 1989). Oral exposure to JP-5 decreased white blood cell counts in rats (Parker et al. 1981). However, inhalation of diesel fuel aerosol had no effect on white blood cells in rats (Dalbey et al. 1987). The conflicting changes in white blood cell levels may be due to differences in the toxicity of these fuel oils or to differences in exposure route or both. The similar effects in humans and mice exposed to kerosene suggests that kerosene may not be species specific and that this fuel oil affects white blood cells in a similar manner regardless of the exposure route. However, there are not enough data to determine whether these generalizations can be made.

Neurological Effects. Numerous neurological effects were reported from kerosene ingestion by children: unconsciousness or semiconsciousness, drowsiness, restlessness, irritability, and in fewer cases, coma and convulsions (Akamaguna and Odita 1983; Aldy et al. 1978; Coruh and Inal 1966; Dudin et al. 1991; Mahdi 1988; Majeed et al. 1981; Nouri and Al-Rahim 1970; Saksena 1969; Santhanakrishnan and Chithra 1978; St. John 1982; Subcommittee on Accidental Poisoning 1962). Neither coma nor convulsions occurred in children that ingested 3-20 mL of kerosene. This dose is equivalent to 126-1,754 mg/kg in children aged 10 months to 5 years (Dudin et al. 1991). There are limited data that suggest that the central nervous system effects noted from ingestion of kerosene are due to hypoxia which results from kerosene-induced respiratory impairment (Majeed et al. 1981).

Severe headaches occurred in an individual exposed to diesel fuel vapor for 10 days (Reidenberg et al. 1964). Anorexia occurred in a man following dermal and/or inhalation exposure to diesel fuel over several weeks (Crisp et al. 1979). Other neurological effects were reported following inhalation of JP-5 vapor in two individuals who had fatigue and coordination and concentration difficulties; other effects included headache, apparent intoxication, and anorexia. Effects subsided within 24 hours for one individual and within 4 days for the other (Porter 1990). Sensory impairment did not occur in these individuals. However, experimental data indicate that olfactory fatigue and taste sensation may occur in some individuals after a 15-minute inhalation exposure to 140 mg/m^3 deodorized kerosene vapor (Carpenter et al. 1976). These data suggest that the different types of fuel oils may behave differently under inhalation exposure conditions. The effect of deodorized kerosene may also occur at lower doses, but this cannot be determined from these data.

Neurasthenia (i.e., fatigue, depressed mood, lack of initiative, dizziness, sleep disturbances) and impairment of attention and sensorimotor speed were associated with chronic inhalation, oral, and/or dermal exposures to jet fuel by factory workers (Knave et al. 1978). Nevertheless, it is not known to which jet fuels the workers were exposed, and confounding by exposure to other chemicals may have occurred.

Acute inhalation of diesel fuel no. 2 vapor induced dose-dependent ataxia, increased sensitivity to heat, changes in behavior, and tremors in mice. Also, while ataxia occurred, there was no affect on the spinal cord reflex for blink response nor on the integrity of the neuromuscular junction based on responses to the rota rod and inclined plane tests (Kainz and White 1984). In rats, intermediate inhalation of diesel fuel aerosol induced increased peak response time using the startle reflex assay

(Lock et al. 1984); however, histopathological changes of the nervous system were not noted in these rats. Neurotoxicity was not induced using the landing footspread, tail flick, forelimb grip strength, and startle reflex assays under similar exposure conditions (Dalbey et al. 1987). Oral exposure to kerosene and Deobase® induced ataxia and drowsiness in rats (Muralidhara et al. 1982). Aspiration of kerosene induced drowsiness, lack of muscular coordination, and behavioral changes (Nouri et al. 1983) and dermal exposure induced an increased response to tactile stimuli and hyperactivity (Upreti et al. 1989) in mice. No histopathological changes were noted in the nervous system of mice following dermal exposures to JP-5 or marine diesel fuel (NTP/NIH 1986). The information from human and animal studies indicate that neurotoxicity may occur by all routes of exposure and that all fuel oils may be neurotoxic. As is common with hydrocarbons, the primary acute neurotoxic effect is central nervous depression that may be manifest in a number of symptoms.

Developmental Effects. No studies were located regarding developmental effects in humans after inhalation, oral, or dermal exposure to fuel oils. No developmental effects were noted in rat fetuses after inhalation exposure of the gestating female to home heating oil no. 2, fuel oil UNSP, or diesel fuel vapor (API 1979c, 19798; Beliles and Mecler 1983). Since negative effects were noted for several fuel oils in one species, it is possible that none of the fuel oils induce developmental effects by inhalation. However, additional data are needed to assess whether developmental effects would occur in other species, including humans, and/or by oral and dermal exposures.

Reproductive Effects. No studies were located regarding reproductive effects in humans after inhalation, oral, or dermal exposure to fuel oils. No histological changes were noted in the reproductive system of mice dermally exposed to JP-5 for 13 weeks or chronically exposed to marine diesel fuel or JP-5 (NTP/NIH 1986) or in rats following intermediate-duration inhalation of diesel fuel aerosol (Lock et al. 1984). There is not enough information to assess the human reproductive toxicity to fuel oils by oral, inhalation, or dermal exposures.

Genotoxic Effects. No genotoxicity studies involving human exposure to fuel oils were identified. The results from a study employing a human cell line showed that neither 5 nor 50 ppm petroleum-derived JP-5 (PD-JPS) interfered with Snyder-Theilen feline sarcoma virus (ST-FeSV)-directed transformation of human foreskin fibroblastic cells (Blakeslee et al. 1983). Higher concentrations (2100 ppm) were cytotoxic. It was reported that marine diesel fuel failed to inhibit transformation in this assay, but data were not shown. The study authors consider this *in vitro* assay to be a useful

predictor of carcinogenesis since several known carcinogens have been shown to suppress transformation in cells infected with the ST-FeSV virus by blocking a specific virus gene function (i.e., transformation); noncarcinogens do not inhibit virus-induced cell transformation in this test system.

Animal models have primarily yielded negative genotoxicity data. Inhalation of 100 to 400 ppm diesel fuel 6 hours/day, 5 days/week for 8 weeks did not increase the frequency of dominant lethal mutations. Cyclohexane/DMSO extract and DMSO extract of diesel 1 (CAS no. 8008-20-6), diesel 2 (CAS no. 64742-47-7), and home heating oil (CAS no. 68476-30-2), administered orally at doses of 1.0, 2.0, and 5.0 g/kg, did not induce increased frequency of micronuclei in a mouse bone marrow micronucleus assay (McKee et al. 1994). It should be noted that the extraction procedure was used to concentrate the aromatic fraction (with particular interest in the polynuclear aromatics) of the fuel oils tested. Kerosene, administered intraperitoneally , did not increase the frequency of chromosomal aberrations in bone marrow cells harvested from rats following a one-time exposure to 0.04, 0.13, or 0.4 mL or a 5-day exposure to 0.02, 0.06, or 0.18 ml/day (Conaway et al. 1984). Since the rationale for selection of 0.4 mL (LD,) as the high dose was not provided and there was no information regarding toxic effects in the treated animals or cytotoxic effects on the target organ (i.e., bone marrow cells), the findings do not fully support a negative conclusion for kerosene.

Some data has, however, suggested that fuel oils may have genotoxic activity. Evidence that fuel oil no. 2 is clastogenic in rat bone marrow has been reported (Conaway et al. 1984). Significant increases (Wilcoxon rank test) in the percentage of aberrant cells were observed in a rat bone marrow cytogenetic assay in rats receiving single intraperitoneal (i.p.) injections (2.0 or 6.0 mL/kg diesel fuel) and in rats receiving daily i.p. injections of 6.0 ml/kg/day for 5 days, although the response was not dose-related. Similarly, rats that received doses of fuel oil no. 2 (oral gavage; 125, 417, or 1,250 g/kg/day) for 5 consecutive days demonstrated nondose-related increases in the percentage of aberrant cells and the percentage of cells with chromatid breaks (Conaway et al. 1984). The effect was significant at the low and high dose, and the greatest yield of aberrant chromosome figures occurred in the rats treated with 125 g/kg/day.

The genotoxicity of fuel oil no. 2, kerosene, and diesel fuel was also evaluated with the mouse lymphoma $TK^{+/-}$ forward mutation assay (Conaway et al. 1984). The data reported was insufficient to

permit a full evaluation of the results; however, the authors considered diesel fuel and kerosene to be negative and fuel oil no. 2 to be positive.

The cyclohexane/DMSO extract and DMSO extract of diesel 1 (CAS no. 8008-20-6), diesel 2 (CAS no. 64742-47-7), and home heating oil (CAS no. 68476-30-2) were evaluated for genotoxicity with the Ames assay (McKee et al. 1994). Diesel 1 extracts did not produce significant increases in revertants. The DMSO extract of diesel 2 produced a significant increase in the number of revertants, although the increase was not dose-related. The cyclohexane/DMSO extract of diesel 2 failed to produce a significant increase in the number of revertants. The DMSO-extract of home heating oil produce a significant increase in revertants, that was dose-dependent, while the cyclohexane/DMSO extract induced a dose-related increase in revertants that was less than two times greater than the control. It should be noted that the modifications to the standard Ames mutagenesis included not only the extraction step but also the use of S9 at eight times the recommended concentration, and the use of the TA98 strain exclusively. Kerosene was mutagenic in S. *typhimurium* TA98 in the presence of increased concentrations of hamster S9 and nicotinamide adenine dinucleotide phosphate (NADP) in the S9-cofactor mix (Blackburn et al. 1986).

In contrast to the positive results with kerosene, neither JP-5 nor a marine diesel fuel product were mutagenic in the Ames assay when activated with S9 (Arochlor-induced rat liver enzymes) (Schultz et al. 1981). Similarly, neither marine diesel fuel nor JP-5 were mutagenic in well-conducted *S. typhimurium* preincubation assays (NTP/NIH 1986). Doses of each agent evaluated without S9 activation and with rat or hamster liver fractions ranged from 3 to 333 μg/plate -S9, from 33 to 3,333 μg/plate +S9 (marine diesel fuel), and from 100 to 10,000 μg/plate +/-S9 (JP-5). Further, fuel oil no. 2 was not mutagenic up to the limit of solubility (42 mg/plate) in the *Salmonella/mammalian* microsome mutagenicity assay (Conaway et al. 1984). It was also reported that kerosene (0.001-5 μL/plate +/-S9 [plate test] and 6.25-50 μL/mL +/-S9 [preincubation assay]) and diesel fuel (0.001-5 μL/plate +/-S9 [plate test] and 3.38-25 μL/mL [preincubation assay]) were negative in this microbial test system.

The inconsistent data reported for the animal models, the human cell assays, and the Ames tests with the various fuel oils preclude the use of the data for the prediction of genotoxic hazards to humans (refer to Tables 2-4 and 2-5 for a further summary of these studies).

TABLE 2-4. Genotoxicity of Fuel Oils *In Vivo*

Species (test system)	End point	Results	Reference
Fuel Oil No. 2			
Mammalian cells:			
Rat (bone marrow)	Chromosome aberrations	+/–[a]	Conaway et al. 1984
Diesel Fuel			
Mammalian cells:			
Rat (bone marrow)	Chromosome aberrations	+	Conaway et al. 1984
Mouse (all stages of spermato-	Dominant lethal		
and spermiogenesis)	mutations	–[b]	API 1981
Kerosene			
Mammalian cells:			
Rat (bone marrow)	Chromosome aberrations	–[c]	Conaway et al. 1984
Diesel Fuel No. 1			
Mammalian cells:			
Rat (bone marrow)	Micronucleus increase	–	McKee et al. 1994
Diesel Fuel No. 2			
Mammalian cells:			
Rat (bone marrow)	Micronucleus increase	–	McKee et al. 1994
Home Heating Oil			
Mammalian cells:			
Rat (bone marrow)	Micronucleus increase	–	McKee et al. 1994

[a]Positive after oral exposure but no dose response
[b]Negative after inhalation exposure
[c]Negative after intraperitoneal exposure but study was compromised

+/– = inconclusive result, + = positive result; – = negative result

TABLE 2-5. Genotoxicity of Fuel Oils *In Vitro*

Species (test system)	End point	Results		Reference
		With activation	Without activation	

Kerosene

Prokaryotic organisms:

Salmonella typhimurium (TA98)	Gene mutation	+	No data	Blackburn et al. 1986

Marine Diesel Fuel

Prokaryotic organisms:

S. typhimurium (TA1535, TA1537, TA98, TA100)	Gene mutation	–	–	NTP/NIH 1986
S. typhimurium (TA98)	Gene mutation	–	–	Schultz et al. 1981

Mammalian cells:

ST-FeSV-infected human foreskin fibroblasts	Inhibition of morphological transformation	No data	–	Blakeslee et al. 1983

JP-5 Fuel

Prokaryotic organisms:

S. typhimurium (TA1535, TA97, TA98, TA100)	Gene mutation	–	–	NTP/NIH 1986
S. typhimurium (TA98)	Gene mutation	–	–	Schultz et al. 1981

Mammalian cells:

ST-FeSV-infected human foreskin fibroblasts	Inhibition of morphological transformation	No data	–	Blakeslee et al. 1983

Fuel Oil No. 2

Prokaryotic organisms:

S. typhimurium (TA1535, TA1537, TA1538, TA98, TA100)	Gene mutations	–	–	Conaway et al. 1984

TABLE 2-5. Genotoxicity of Fuel Oils *In Vitro* (*continued*)

Species (test system)	End point	Results		Reference
		With activation	Without activation	
Mammalian cells:				
Mouse lymphoma (L5178Y)	Gene mutations	–	+/–	Conaway et al. 1984
Kerosene				
Prokaryotic organisms:				
S. typhimurium (TA1535, TA1537, TA1538, TA98, TA100)	Gene mutations	–	–	Conaway et al. 1984
Mammalian cells:				
Mouse lymphoma (L5178Y)	Gene mutations	–	–	Conaway et al. 1984
Diesel Fuel				
Prokaryotic organisms:				
S. typhimurium (TA1535, TA1537, TA1538, TA98, TA100)	Gene mutations	–	–	Conaway et al. 1984
Mammalian cells:				
Mouse lymphoma (L5178Y)	Gene mutations	–	–	Conaway et al. 1984
Diesel Fuel No. 1				
Prokaryotic organisms:				
S. typhimurium (TA98)	Gene mutation	–	No data	McKee et al. 1994
Diesel Fuel No. 2				
Prokaryotic organisms:				
S. typhimurium (TA98)	Gene mutation	+/-	No data	McKee et al. 1994

TABLE 2-5. Genotoxicity of Fuel Oils *In Vitro* (*continued*)

Species (test system)	End point	Results		Reference
		With activation	Without activation	
Home Heating Oil				
Prokaryotic organisms:				
S. typhimurium (TA98)	Gene mutation	+	No data	McKee et al. 1994

+ = positive result; – = negative result; +/– = inconclusive result; ST-FeSV = Snyder-Theilen feline sarcoma virus

Cancer. Human cancer data from epidemiological studies found only equivocal evidence of an association between cancer and exposures to fuel oils. Several studies examined the association between "fuel and oil expenditures for farm purposes" and various forms of cancer in central Canadian farmers (Morrison et al. 1992, 1994; Semenciw et al. 1993). They reported an association between such expenditures and non-Hodgkin's lymphoma and multiple myeloma incidence, but the association was equivocal and not statistically significant. Furthermore, the type of fuels and oils was not specified, nor was the exposure route described. Scherr and colleagues (1992) reported no additional relative risk for non-Hodgkin's lymphoma for subjects occupationally exposed to "gasoline or kerosene." No significant increased relative risk for any type of cancer was noted in Swedish Air Force personnel exposed to military aircraft fuels (including an "unleaded kerosene type jet fuel"). One study (Partanen et al. 1991) suggests that other chemicals could be present in the occupational setting, which could alter fuel oil toxicity, though this same study found no significant association between fuel oil exposure and cancer. Chan and coworkers (1979) examined exposure to kerosene from kerosene cooking stoves, but exposure to kerosene combustion products may have occurred instead of, or in addition to, inhalation of kerosene vapor. Therefore, no firm conclusions may be drawn from this data for human health.

No dermal cancer was noted in $B6C3F_1$ mice following chronic dermal exposure to 250 or 500 mg/kg/day JP-5 (NTP/NIH 1986). However, unspecified skin tumors were noted in C3HF/Bd mice, but the tumors were not dose related in most exposure conditions (Schultz et al. 1981). There was an increased incidence of squamous cell papilloma and/or carcinoma in mice chronically exposed to 250 or 500 mg/kg/day marine diesel fuel (NTP/NIH 1986). Hepatocellular adenoma and carcinoma were noted in male, but not female, mice exposed to 250 or 500 mg/kg/day marine diesel fuel (NTP/NIH 1986). Although a significant increase in hepatocellular carcinomas were observed in mice dermally treated with middle distillates, the increase was not substantially greater than the incidence noted in "historical" data from negative control groups (Biles et al. 1988). API no. 2 fuel oil demonstrated low tumorigenic activity (15/150) in male and female mice dermally treated with the undiluted material or as a 50% or 25% solution in acetone (Witschi et al. 1987). A low, but significant increase in the incidence of dermal tumor was noted in male mice treated with six no. 2 fuel oils that varied in composition (Biles et al. 1988). No increase in tumor incidence occurred in mice receiving dermal applications of diesel fuel; however, dermal application of Jet A induced an increased incidence (26%) of neoplastic lesions (Clark et al. 1988). An increase in tumor incidence was noted in mice receiving

DMBA as an initiating agent and furnace oil as a promoting agent; further, furnace oil produced a significant increase in the incidence of confirmed skin tumors in a skin-painting assay (Gerhart et al. 1988). An increase in the incidence of confirmed tumor was also noted in animals receiving DMBA as an initiator and either hydrodesulfurized kerosine or no. 2 fuel oil as a promoting agent (API 1989). These data suggest that fuel oils can act as a skin or liver carcinogen. However, only one species has been investigated, limiting the data. Further investigation utilizing other species is required to more fully elucidate the mechanism of dermal carcinogenesis and the impact of dermal exposure of fuel oils on humans. (See Section 2.2.3.8 for a more complete review of carcinogenesis data.)

2.5 BIOMARKERS OF EXPOSURE AND EFFECT

Biomarkers are broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility (NAS/NRC 1989).

A biomarker of exposure is a xenobiotic substance, its metabolite(s), or the product of an interaction between a xenobiotic agent and some target molecule(s) or cell(s) that is measured within a compartment of an organism (NAS/NRC 1989). The preferred biomarkers of exposure are generally the substance itself or substance-specific metabolites in readily obtainable body fluid(s) or excreta. Examples of the types of biomarkers indicated above include blood lead (the xenobiotic), urinary excretion of 2-thiothizolidine-4-carboxylic acid (a metabolite of carbon disulfide), or a DNA adduct (the product of an interaction between an exogenous material and a macromolecule). Several factors can confound the use and interpretation of biomarkers of exposure. The body burden of a substance may be the result of exposures from more than one source. The substance being measured may be a metabolite of another xenobiotic substance (e.g., high urinary levels of phenol can result from exposure to several different aromatic compounds). Depending on the properties of the substance (e.g., biologic half-life) and environmental conditions (e.g., duration and route of exposure), the substance and all of its metabolites may have left the body by the time biologic samples can be taken. It may be difficult to identify individuals exposed to hazardous substances that are commonly found in body tissues and fluids (e.g., essential mineral nutrients such as copper, zinc, and selenium). Biomarkers of exposure to fuel oils are discussed in Section 2.5.1.

Biomarkers of effect are defined as any measurable biochemical, physiologic, or other alteration within an organism that, depending on magnitude, can be recognized as an established or potential health impairment or disease (NAS/NRC 1989). This definition encompasses biochemical or cellular signals of tissue dysfunction (e.g., increased liver enzyme activity or pathologic changes in female genital epithelial cells), as well as physiologic signs of dysfunction such as increased blood pressure or decreased lung capacity. Note that these markers are often not substance specific. They also may not be directly adverse, but can indicate potential health impairment (e.g., DNA adducts). Biomarkers of effects caused by fuel oils are discussed in Section 2.5.2.

A biomarker of susceptibility is an indicator of an inherent or acquired limitation of an organism's ability to respond to the challenge of exposure to a specific xenobiotic substance. It can be an intrinsic genetic or other characteristic or a preexisting disease that results in an increase in absorbed dose, a decrease in the biologically effective dose, or a change in target tissue response. If biomarkers of susceptibility exist, they are discussed in Section 2.7, "Populations That Are Unusually Susceptible."

2.5.1 Biomarkers Used to Identify or Quantify Exposure to Fuel Oils

No biomarkers of exposure were identified for fuel oils in general. However, there have been suggestions for potential markers for kerosene exposure. These include the odor of kerosene on the breath suggesting ingestion (Annobil 1988; Zucker et al. 1986) and the odor of kerosene on clothing suggesting dermal exposure (Annobil 1988; Tagami and Ogino 1973). The odor of distillate fuels are so similar, however, that the sensitivity and specificity of these markers would be extraordinarily low. Some components of kerosene, other fuel oils, and their metabolites may be detected in the blood and urine, although neither the route of exposure nor the origin can be determined. For information on biomarkers of exposure for some of the constituents of fuel oils, the ATSDR toxicological profiles on benzene, toluene, total xylenes, and polycyclic aromatic hydrocarbons (ATSDR 1989, 1990a, 1991a, 1991b) can be consulted.

2.5.2 Biomarkers Used to Characterize Effects Caused by Fuel Oils

No specific, quantitative biomarkers of effect for fuel oils were identified.

2.6 INTERACTIONS WITH OTHER CHEMICALS

Exposures to two or more substances may cause effects that are additive (the combined effect of the mixture is equal to the sum of the effects of the agents), synergistic (causing an effect that is greater than the sum of the effects of the agents), or antagonistic (one substance interferes with the action of another). No information was located regarding the influence of other chemicals on the toxicity of fuel oils. However, kerosene vapor has been shown to increase the effects of hexobarbital (a sleeping agent), following acute exposure, and phenacetin (an antipyretic), following subchronic exposure, in rats (Starek and Vojtisek 1986).

2.7 POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE

A susceptible population will exhibit a different or enhanced response to fuel oils than will most persons exposed to the same level of fuel oils in the environment. Reasons include genetic make-up, developmental stage, health and nutritional status, and chemical exposure history. These parameters result in decreased function of the detoxification and excretory processes (mainly hepatic and renal) or the pre-existing compromised function of target organs. For these reasons we expect the elderly with declining organ function and the youngest of the population with immature and developing organs will generally be more vulnerable to toxic substances than healthy adults. Populations who are at greater risk due to their unusually high exposure are discussed in Section 5.6, "Populations With Potentially High Exposure."

No information was located regarding the toxicity of fuel oils in susceptible populations. The human data, in general, were based upon case studies that reported ingestion of kerosene by children. Although children were not shown to be particularly susceptible to kerosene in these studies, it was obvious that children are more likely to be exposed to kerosene accidentally than adults. In particular, children that are 5 years old or younger often mistakenly drank kerosene because it was accessible to them.

In one animal study, it was found that younger rats are more susceptible to kerosene toxicity than are older rats. A single oral dose of 22,400 mg/kg kerosene killed 27% of the adult rats, 66% of the 5-week-old rats, and 100% of the 10-day-old rats (Deichmann et al. 1944). It is not known whether kerosene would also be more toxic in younger humans as compared to older humans.

2.8 METHODS OF REDUCING TOXIC EFFECTS

This section will describe clinical practice and research concerning methods for reducing toxic effects of exposure to fuel oils. However, because some of the treatments discussed may be experimental and unproven, this section should not be used as a guide for treatment of exposures to fuel oils. When specific exposures have occurred, poison control centers and medical toxicologists should be consulted for medical advice.

2.8.1 Reducing Peak Absorption Following Exposure

The mitigation procedures for fuel oils parallel those for hydrocarbon poisoning in general. Inhalation and ingestion appear to be the most serious routes of exposure. In the case of overexposure by inhalation, it is suggested that the patient be moved to an area of fresh air and given basic supportive treatment (CONCAWE 1985; HSDB 1991) including 100% humidified supplemental oxygen as required (HSDB 1991).

For poisoning by ingestion, the treatment protocol is more complex. As with inhalation, it is recommended that the patient receive prompt supportive medical care (Bronstein and Currance 1988; CONCAWE 1985; Goldfrank et al. 1990; Haddad and Winchester 1990; Stutz and Janusz 1988; Zieserl 1979). The primary concern for the person who has ingested hydrocarbons such as fuel oils or kerosene is hydrocarbon aspiration either during ingestion or during gastric decontamination. Aspiration of the hydrocarbon into the lungs can cause hydrocarbon pneumonitis and secondary infections including pneumonia.

Because of the aspiration risk, a controversy has developed over which (if either) of two gastric decontamination treatments is better: induced vomiting or gastric lavage. In general, the recommendation is that no form of gastric emptying be used if the amount of hydrocarbon ingestion is small (Bronstein and Currance 1988; Ellenhorn and Barceloux 1988; Goldfrank et al. 1990; HSDB 1991; Litovitz and Greene 1988; Shirkey 1971; Zieserl 1979). This is usually the case with accidental poisonings. If unknown or large amounts (volumes greater than 100 mL) have been ingested, then the decision of how and/or whether to decontaminate the stomach should be based on the state of the patient, the hydrocarbon's viscosity, and the involvement of other more dangerous chemicals. For conscious patients with operational gag reflexes and without spontaneous emesis, induced vomiting

seems to be the preferred method of gastric emptying (Ellenhorn and Barceloux 1988; Goldfrank et al. 1990; Ng et al. 1974; Shirkey 1971; Zieserl 1979); otherwise, endotracheal intubation followed by gastric lavage has been suggested (Ellenhorn and Barceloux 1988; Haddad and Winchester 1990).

For ingestion of large amounts (greater than 30 mL in children) of home or diesel fuel oils, gastric decontamination has been contraindicated since these hydrocarbons have a high viscosity and are poorly absorbed (Ellenhorn and Barceloux 1988). The low viscosity of kerosene, however, has produced conflicting opinions. Some recommend induced emesis to prevent gastrointestinal absorption (Ellenhorn and Barceloux 1988). On the other hand, others suggest that the low viscosity of kerosene increases the risk of aspiration (Gerarde 1959; Litovitz and Greene 1988) and therefore do not recommend gastric decontamination regardless of volume (Bronstein and Currance 1988; CONCAWE 1985; Haddad and Winchester 1990; Litovitz and Greene 1988; Macnamara 1968).

Controversy also exists over whether or not to administer activated charcoal (to bind the hydrocarbon) or cathartics (Ellenhorn and Barceloux 1988; Goldfrank et al. 1990; Haddad and Winchester 1990; HSDB 1991; Litovitz and Greene 1988; Shirkey 1971; Stutz and Janusz 1988; Zieserl 1979). Some question the overall effectiveness of activated charcoal and cathartics (Goldfrank et al. 1990; Litovitz and Greene 1988; Zieserl 1979). In addition, activated charcoal may cause vomiting (HSDB 1991) which may or may not be desired. Most agree, however, that if cathartics are administered, they should be saline cathartics such as magnesium or sodium sulfate or citrate and not oil-based cathartics such as mineral oil (Ellenhorn and Barceloux 1988; Goldfrank et al. 1990; Haddad and Winchester 1990; Stutz and Janusz 1988).

In general, administration of antibiotics and/or corticosteroids does not appear useful in treating hydrocarbon pneumonitis (Brown et al. 1974; Goldfrank et al. 1990; Haddad and Winchester 1990; HSDB 1991; Steele et al. 1972; Wolfsdorf and Kundig 1974; Zieserl 1979). In fact, one study has suggested that steroid administration may increase bacterial colonization in the lungs (Brown et al. 1974). The use of antibiotics is recommended only to treat secondary lung infections (Haddad and Winchester 1990; HSDB 1991; Zieserl 1979).

If the skin is exposed to fuel oils, washing the area of contact with large amounts of soapy water is recommended (CONCAWE 1985; Ellenhorn and Barceloux 1988; Goldfrank et al. 1990; HSDB 1991; Stutz and Janusz 1988). If blistering or skin loss occurs, then the use of sterile water alone is

suggested (CONCAWE 1985). For ocular exposure, flushing the eyes liberally with water (CONCAWE 1985; HSDB 1991; Stutz and Janusz 1988) and, if necessary, using proparacaine hydrochloride to assist the irrigation (Bronstein and Currance 1988) are the recommended treatment protocols.

2.8.2 Reducing Body Burden

Little is known about the toxicokinetics of fuel oils, and there are no known methods for the reduction of body burden.

2.8.3 Interfering with the Mechanism of Action for Toxic Effects

Although lung response to aerosolized kerosene and the effect of kerosene on heme biosynthesis have been partially investigated, the toxicities of fuel oils as well as their mechanisms are not well defined. As such, no known therapies are available that disrupt the mechanisms of action.

2.9 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of fuel oils is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of fuel oils.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that, if met, would reduce or eliminate the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda may be proposed.

2.9.1 Existing Information on Health Effects of Fuel Oils

The existing data on health effects of inhalation, oral, and dermal exposure of humans and animals to fuel oils are summarized in Figure 2-4. The purpose of this figure is to illustrate the existing information concerning the health effects of fuel oils. Each dot in the figure indicates that one or more studies provide information associated with that particular effect. The dot does not imply anything about the quality of the study or studies. Gaps in this figure should not be interpreted as "data needs" information (i.e., data gaps that must necessarily be filled).

Information is available on acute, intermediate, and chronic systemic effects, as well as neurological and cancer effects, following inhalation exposure to fuel oils; death, acute systemic, and neurological effects following oral exposure to fuel oils; and acute, intermediate, and chronic systemic and neurological effects following dermal exposure to fuel oils in humans. Information is available on death, and acute and intermediate systemic effects, as well as neurological, developmental, reproductive, genotoxic, and cancer effects following inhalation exposure to fuel oils; death, acute systemic effects, as well as neurological and genotoxic effects following oral exposure to fuel oils; and death, acute, intermediate, and chronic systemic effects, as well as immunological, neurological, reproductive, and cancer effects following dermal exposure to fuel oils in animals. Therefore, as Figure 2-4 shows, the majority of the data on health effects of fuel oils concern inhalation or dermal exposure of animals; however, there are some data for all routes of exposure in both animals and humans.

2.9.2 Identification of Data Needs

The following are topical sections that identify gaps in the present state of knowledge concerning the toxicology of fuel oils. Each of the sections identifies specific areas in which additional data are needed to gain a greater understanding of the toxicity of fuel oils and its constituents as well as of the biochemical mechanisms of their toxicity. It must be noted, however, that there are finite monies available for all toxicological research. Hard decisions must be made to determine how (e.g., the material to be studied, the effect to be investigated, whether human study or animal model) these funds would best be invested.

FIGURE 2-4. Existing Information on Health Effects of Fuel Oils

	Death	Acute	SYSTEMIC Intermediate	SYSTEMIC Chronic	Immunological/Lymphoreticular	Neurological	Reproductive	Developmental	Genotoxic	Cancer
Inhalation	●	●	●	●		●				●
Oral	●	●		●		●				
Dermal		●	●	●		●				●

Human

	Death	Acute	SYSTEMIC Intermediate	SYSTEMIC Chronic	Immunological/Lymphoreticular	Neurological	Reproductive	Developmental	Genotoxic	Cancer
Inhalation	●	●	●		●	●	●	●	●	●
Oral	●	●	●		●	●	●		●	
Dermal	●	●	●	●	●	●	●			●

Animal

● Existing Studies

Acute-Duration Exposure. There are many case studies that identify respiratory, neurological, and gastrointestinal effects as the primary effects in humans induced by acute exposures to fuel oils, particularly by the oral route (Akamaguna and Odita 1983; Aldy et al. 1978; Annobil 1983; Annobil and Ogunbiyi 1991; Mahdi 1988; Santhanakrishnan and Chithra 1978; St. John 1982; Subcommittee on Accidental Poisoning 1962) and, to a lesser extent, by inhalation exposure (Barrientos et al. 1977; Porter 1990). Dermal irritation is also well documented for both humans (Annobil 1988; Barrientos et al. 1977; Mosconi et al. 1988; Tagami and Ogino 1973) and animals (NTP/NIH 1986; Upreti et al. 1989) by the dermal route of exposure. A few case studies indicate that cardiovascular, hematological, and renal effects may occur in humans exposed to the vapors of JP-5 or diesel fuel (Barrientos et al. 1977; Porter 1990; Reidenberg et al. 1964). Renal toxicity may also occur following dermal contact with diesel fuel (Barrientos et al. 1977).

Dose-response data are largely lacking for the effects noted in both humans and animals. A few animal studies do contain dose-response data. Decreased food and water consumption, vasodilation, and neurological effects (reduced coordination, increased sensitivity to heat, changes in behavior, tremors) were found to be dose-dependent in mice exposed to diesel fuel no. 2 aerosol (Kainz and White 1984). Dose-response lethality data were found for inhalation exposures to diesel fuel aerosols (Dalbey and Lock 1983). In addition, there was a dose-response relationship following a single exposure to kerosene by oral gavage for death, unsteady gait, and drowsiness in rats (Muralidhara et al. 1982). However, the majority of the animal studies contain negative data (Beliles and Mecler 1983) that have not been verified by more than one study using the same fuel oil, species, and/or route of exposure, or the studies only tested one dose (Brown et al. 1974; Casaco et al. 1982; Garcia et al. 1988b; Goodwin et al. 1988; Nouri et al. 1983; Upreti et al. 1989). Acute oral LD_{50} data are available for kerosene in guinea pigs and rabbits (Deichmann et al. 1944). Additional data are needed regarding inhalation and dermal exposures in various species to verify the renal toxicity of fuel oils noted in a few individuals.

Intermediate-Duration Exposure. Only one case study was identified that described intermediate exposure in one individual who washed his hands with diesel fuel over several weeks (Crisp et al. 1979). The man exhibited epigastral pain, hematological effects, renal necrosis, edema of the scrotum and ankle, loin pains, thirst, and severe exhaustion. Effects resulting from inhalation versus dermal exposure could not be distinguished in this case. This is the only study found that identifies renal

necrosis in humans. The renal necrosis described in this individual resembles a renal nephropathy that was previously found only in male rats from vapor inhalation and oral exposure to JP-5 and marine diesel fuel (Bruner 1984; Cowan and Jenkins 1981; Gaworski et al. 1984; Parker et al. 1981). There are data that show the $\alpha_{2\mu}$-globulin protein, which is responsible for the necrosis in rats, may not exist in humans (Alden 1986). Also, data are needed to determine whether mechanisms of toxicity, other than those involving this protein, may exist for the induction of this lesion in both humans and rats. Also, data from well conducted studies are needed to determine which fuel oils induce this lesion in various species. Finally, in future cases of human exposure to fuel oils, signs of renal toxicity should be carefully monitored and results from histological examinations of renal tissue should be reported, if available.

Animal data are available for intermediate exposures by the inhalation and dermal routes of exposure. No animal data were located by the oral route. Most of these studies found no evidence of toxicity in any of the exposure conditions used in each (Carpenter et al. 1976; Bruner 1984; Lock et al. 1984; NTP/NIH 1986). However, the lack of toxicity in these studies has not been verified by more than one study using the same fuel oil, species, and/or route of exposure. In one aerosol inhalation study (Dalbey et al. 1987) there were positive findings for respiratory, hematological, and body weight effects at higher doses than those used in the studies by Carpenter et al. (vapor) (1979) and Lock et al. (aerosol) (1984). However, MRLs cannot be derived from these data because the Dalbey et al. study was not designed to test for a dose-response relationship, and therefore, the exact LOAEL(s) could not be determined for these effects. In another aerosol study with positive findings, only one concentration level was tested (Noa and Illnait 1987a).

One well-conducted study in mice describes effects (death, hepatic karyomegaly, and dermatitis) from dermal exposures to either JP-5 or marine diesel fuel (NTP/NIH 1986). Another study found dose-dependent increases in blood lactate and pyruvate levels and decreases in blood glucose levels in rats after inhalation of kerosene vapor (Starek and Vojtisek 1986). In a third study, dose-related increases in the relative weight of the right lobe of the lung were noted from inhalation of diesel fuel aerosol (Lock et al. 1984). None of these studies can be used for MRL derivation since the data were obtained by dermal exposures in one study and the biochemical and organ weight effects induced by inhalation of the fuel oils were not supported by pathological changes. More data are needed in animals, and especially in humans, for all routes of exposure to identify the primary toxic effects of fuel oils from intermediate exposures.

Chronic-Duration Exposure and Cancer. Epidemiological data regarding respiratory and dermal effects from chronic exposures to fuel oils in humans are described elsewhere (see Epidemiological and Human Dosimetry Studies in this section). No other information is available for humans regarding chronic inhalation or oral exposures to fuel oils. A single animal study addressed carcinogenicity in animals via inhalation (Bruner 1984); however, the study did not adequately investigate the subject. Animal model data were available for the carcinogenic effects of chronic dermal exposure. It is apparent that chronic dermal application of fuel oils can induce tumorigenesis; however, both the mechanism of induction and the relevance of fuel oil tumor induction to humans are poorly defined. Equivocal data were available for the induction of hepatic tumors following dermal exposure. The data were so limited that the effect could not be evaluated. As such, further elucidation of the biochemical pathway, the relevance of dermal exposure to humans, and the incidence of induction of systemic tumorigenesis subsequent to dermal exposure would be of value.

The demonstration of renal toxicity in animal models has been considered significant due, at least in part, to case studies reporting such toxicity. However, data exist that appear to associate the renal toxicity with water loss due to skin lesions induced by chronic dermal application of fuel oils rather than systemic toxicity. Data that clarifies this effect would be of interest.

Genotoxicity. No definite conclusions can be reached from the *in vitro* human cell and whole animal genetic toxicology studies that have been performed with fuel oils. Data from bacterial *in vitro* assays are inconsistent (see Section 2.4, Genotoxic Effects). A study of the genotoxicity/mutagenicity of commercially available fuel oils and the various component petroleum streams used in their formulation would be of value.

Reproductive Toxicity. No information was found regarding reproductive toxicity in humans from inhalation, oral, or dermal exposures to fuel oils. There were no pathological changes on the reproductive organs of mice following chronic and/or intermediate dermal exposures to marine diesel fuel and JP-5 (NTP/NIH 1986) or in rats following intermediate inhalation of diesel fuel aerosol (Lock et al. 1984). Additional data are needed to identify the toxic potential of fuel oils on the reproductive system by all routes of exposure.

Developmental Toxicity. No information was found regarding developmental toxicity in humans from inhalation, oral, or dermal exposures to fuel oils. Several studies were identified that tested developmental effects in animals, but only using the inhalation route of exposure. These studies found no developmental effects in the fetuses of female rats that had been exposed to heating oil, fuel oil UNSP, or diesel fuel vapors by inhalation during gestation days 6-15 (API 1979c, 19798; Beliles and Mecler 1983). Additional data are needed to identify the toxic potential of fuel oils regarding developmental effects by all routes of exposure.

Immunotoxicity. No information was found regarding immunotoxicity in humans from inhalation, oral, or dermal exposures to fuel oils. Only two animal studies were identified that tested immunological effects, both using mice. These studies identified cellular effects in the bone marrow, lymph nodes, and/or thymus and decreases in the relative weights of the lymph nodes and thymus from acute dermal exposures to kerosene (Upreti et al. 1989) and chronic dermal exposures to JP-5 and marine diesel fuel (NTP/NIH 1986). However, the toxicological significance of these effects on the immune system cannot be determined from these data. Additional data are needed to identify the toxic potential of fuel oils on the immune system by all routes of exposure and in different animal systems.

Neurotoxicity. Epidemiological data regarding neurological effects from chronic exposures to fuel oils in humans are described elsewhere (see Epidemiological and Human Dosimetry Studies in this section). Neurological effects from oral exposures are well documented in humans by case studies (Akamaguna and Odita 1983; Aldy et al. 1978; Coruh and Inal 1966; Dudin et al. 1991; Mahdi 1988; Majeed et al. 1981; Nouri and Al-Rahim 1970; Saksena 1969; Santhanakrishnan and Chithra 1978; St. John 1982; Subcommittee on Accidental Poisoning 1962). There is limited information in animals regarding neurotoxic effects following oral exposure (Muralidhara et al. 1982) or aspiration (Nouri et al. 1983).

Some information is available to identify neurological effects in humans from inhalation exposures. The available data indicate that coordination and concentration difficulties, headache, intoxication, and/or anorexia may be induced by inhalation of JP-5 vapor (Porter 1990), headaches may be induced by diesel fuel vapor (Reidenberg et al. 1964), and sensory impairment may be induced by deodorized kerosene vapor (Carpenter et al. 1976). In animals, a few studies were found that document neurological effects from inhalation of fuel oils. Acute inhalation of diesel fuel no. 2 vapor produced

behavioral changes, tremors, ataxia, reduced coordination, and increased sensitivity to heat in mice (Kainz and White 1984). In another study (Lock et al. 1984), peak response time, based on the startle reflex assay, was increased in rats after intermediate inhalation of diesel fuel aerosol, but at higher exposure levels than those used in the Kainz study. These studies conflict with the negative neurotoxicity findings of a second intermediate-duration study in which diesel fuel aerosol was tested in rats at even higher concentrations (Dalbey et al. 1987). Thus, MRLs cannot be derived from these data.

Neurotoxicity in humans from dermal exposures has been reported in 1 case study in which anorexia was noted (Crisp et al. 1979); inhalation exposure may have also occurred. One animal study found no histopathological changes in the organs of the nervous system in mice following chronic and/or intermediate dermal exposures to marine diesel fuel and JP-5 (NTP/NIH 1986). However, increased response to tactile stimuli and hyperactivity occurred in mice from acute dermal exposures to kerosene (Upreti et al. 1989).

In summary, there is much information regarding the specific neurological effects that may be induced by oral exposures to kerosene in humans, but dose-response data are lacking for both animals and humans. More information is needed to identify the inhalation and dermal effects of fuel oils on the nervous system in both animals and humans.

Epidemiological and Human Dosimetry Studies. There were limited data that indicated that the use kerosene stoves in the home is not associated with increased respiratory illness (Azizi and Henry 1991; Tominaga and Itoh 1985), although chronic dermal exposure to kerosene has been related to Dermatosis (Jee et al. 1985). These studies are of limited use, however, since neither exposure nor duration of exposure were reported.

A number of effects have been associated with chronic exposure to jet fuel in factory workers (Knave et al. 1978). These effects included increases in the occurrence of neurasthenia (anxiety and/or mental depression, fatigue, depressed mood, lack of initiative, dizziness, palpitations, thoracic oppression, sleep disturbances) and eye irritation. Psychological tests found that attention and sensorimotor speed were impaired in exposed workers, but there were no effects on memory functions or manual dexterity. EEG tests suggested that there may have been instability in the thalamocortical system in the exposed group. However, the type of jet fuels were not noted nor was there a control for exposure to other

compounds. Inhalation exposure is likely since jet fuel vapor was detected by the study authors; however, dermal and oral (i.e., eating with contaminated hands) exposures may also be possible.

Limited epidemiological information exists for carcinogenicity in humans following inhalation exposure to kerosene (vapor) (Chan et al. 1979) and other fuel oils such as diesel fuel (vapor) (Partanen et al. 1991). These studies either test kerosene exposure by use of kerosene stoves, and so are limited for the same reasons as the respiratory studies described above, or measure fuel oil exposures according to occupation. In the latter case, confounding from exposure to other chemicals, such as gasoline, exists. Both studies are limited since the duration and level of fuel oil exposure were not identified. Other available data are also reported to be inadequate to assess the carcinogenic potential of fuel oils (IARC 1989; Lam and Du 1988).

Exposures to fuel oils generally occur in the occupational setting. For this reason, it is difficult to control for confounding by other chemicals and to identify levels and durations of exposure to specific fuel oils. Exposure to kerosene may occur in the general population through the use of kerosene stoves and kerosene heaters. Aside from accidental poisonings in children, however, quantitative exposures to kerosene are difficult to determine because exposures are likely to be by inhalation or dermal routes. Also, there is much variability in the ventilation systems, cooking patterns, and smoking habits in individual homes of the general population, which makes determination of the level of exposure difficult. Finally, it is not possible to control for confounding by combustion products of kerosene when testing the effects of kerosene by the inhalation route. Therefore, if future studies are going to yield useful data concerning the toxicity of fuel oils in humans, rigorous controls must be planned for any confounding factors.

Biomarkers of Exposure and Effect. No biomarkers of exposure or effect were identified for fuel oils. Although no standard procedures exist for identifying and quantifying exposure to fuel oils in general, procedures do exist for identifying and quantifying the hydrocarbon components of fuel oils, specifically kerosene, in blood, urine, and stomach contents (Hara et al. 1988; Kimura et al. 1988, 1991; Yamaguchi et al. 1992). Another potential biomarker of exposure to kerosene is the distinct odor of kerosene on the breath or clothing (Annobil 1988; Tagami and Ogino 1973; Zucker et al. 1986). Studies delineating the metabolism and excretion of fuel oils are needed to identify potential biomarkers of exposure.

Although not specific for kerosene, aminolevulinic acid (ALA) could potentially be used as an adjunct or supplemental biomarker for kerosene exposure. Kerosene may affect heme metabolism by decreasing the activities of enzymes in the heme biosynthetic pathway (hepatic δ-ALA dehydratase and δ-ALA synthetase) (Rao and Pandya 1980). Therefore, it may be possible that this effect would generate increased ALA in the urine of exposed individuals. Additional studies of acute, intermediate, and chronic exposure are needed to identify biomarkers of effects for specific target organs following exposure to fuel oils.

Absorption, Distribution, Metabolism, and Excretion. No quantitative data were located regarding the absorption, distribution, metabolism, or excretion of fuel oils following inhalation, oral, or dermal exposure in humans. No quantitative data were located regarding absorption and distribution of fuel oils following inhalation or dermal exposure in animals. Very limited data indicate that kerosene is poorly absorbed from the gastrointestinal tract and is distributed to various tissues, although accumulation is low (Mann et al. 1977). Another study in humans suggests that respiratory toxicity may result from both aspiration from vomiting and gastrointestinal absorption (Subcommittee on Accidental Poisoning 1962). However, aspiration is the primary concern following ingestion. There is also some suggestion from case studies that renal toxicity may occur in humans following exposure to diesel fuel vapor (Barrientos et al. 1977; Reidenberg et al 1964), although this possibility appears remote. Renal toxicity may occur following dermal contact with diesel fuel (Barrientos et al. 1977; Easely et al. 1982). No data were located regarding the metabolism or excretion of fuel oils following any of the three routes of exposure. Acute, intermediate, and chronic data are needed to assess the relative rates and extent of absorption, distribution, and excretion of fuel oils with respect to all three routes of exposure, as well as with respect to time or dose. Also, data are needed to determine whether dermal absorption of diesel fuel vapor can occur to induce renal toxicity.

Comparative Toxicokinetics. Limited data are available regarding comparative toxicokinetics. The acute oral LD_{50} values in guinea pigs and rabbits for kerosene has been reported to be 16,320 mg/kg and 22,720 mg/kg, respectively (Deichmann et al. 1944). These data suggest that there may be species differences in the oral toxicity of kerosene; however, more data would be needed to thoroughly examine species variation in toxicokinetics. This information would be useful to identify similar target organs and to adequately assess which animals can serve as the best models for humans, as well as to define mechanisms of action.

Methods for Reducing Toxic Effects. The mitigation procedures for fuel oils parallel those for hydrocarbon poisoning. Several treatments for hydrocarbon poisoning have been considered controversial: gastric decontamination, induced emesis versus gastric lavage, and administration of activated charcoal, cathartics, antibiotics, and corticosteroids. Most studies indicate that antibiotics and corticosteroids are not effective treatments for hydrocarbon-induced, and specifically kerosene-induced, pneumonitis (Brown et al. 1974; Goldfrank et al. 1990; Haddad and Winchester 1990; HSDB 1991; Steele et al. 1972; Wolfsdorf and Kundig 1974; Zieserl 1979). However, more research regarding the usefulness of cathartics and activated charcoal is needed. In addition, elucidating kerosene's toxicokinetic properties of absorption in the gastrointestinal tract would help determine whether gastric decontamination is worth the risk of pulmonary aspiration. Related to gastric decontamination is the question of whether induced emesis is safer than gastric lavage. Since there are presently no known antidotes for hydrocarbon poisoning, research in this area would be beneficial as well.

2.9.3 On-going Studies

No on-going studies evaluating the health effects or toxicokinetics of fuel oils were located.

3. CHEMICAL AND PHYSICAL INFORMATION

3.1 CHEMICAL IDENTITY

Information regarding the chemical identity of fuel oils is located in Table 3-l. Information on the composition of selected fuel oils, specifically fuel oil no. 2 and kerosene, is presented in Table 3-2.

All of the fuel oil classes discussed in this Profile are refined from crude petroleum and may be categorized as either a distillate fuel or a residual fuel depending on the method of production. Fuel oils no. 1 and no. 2 are distillate fuels which consist of distilled process streams. Residual fuel oils such as fuel oil no. 4 are residues remaining after distillation or cracking, or blends of such residues with distillates (IARC 1989). Diesel fuels are approximately similar to fuel oils used for heating (fuel oils no. 1, no. 2, and no. 4). All fuel oils consist of complex mixtures of aliphatic and aromatic hydrocarbons. The aliphatic alkanes (paraffins) and cycloalkanes (naphthenes) are hydrogen saturated and compose approximately 80-90% of the fuel oils. Aromatics (e.g., benzene) and olefins (e.g., styrene and indene) compose 10-20% and l%, respectively, of the fuel oils. Fuel oil no. 1 (straightrun kerosene) is a light distillate which consists primarily of hydrocarbons in the C_9—C_{16} range; fuel oil no. 2 is a heavier, usually blended, distillate with hydrocarbons in the C_{11}-C_{20} range. Straight-run distillates may also be used to produce fuel oil no. 1 and diesel fuel oil no. 1. Diesel fuel no. 1 and no. 2 are similar in chemical composition to fuel oil no. 1 and fuel oil no. 2, respectively, with the exception of the additives. Diesel fuels predominantly contain a mixture of C_{10} through C_{19} hydrocarbons, which include approximately 64% aliphatic hydrocarbons, l-2% olefinic hydrocarbons, and 35% aromatic hydrocarbons (Air Force 1989). Jet fuels are based primarily on straight-run kerosene, as well as additives. All of the above fuel oils contain less than 5% polycyclic aromatic hydrocarbons. Fuel no. 4 (marine diesel fuel) is less volatile than diesel fuel no. 2 and may contain up to 15% residual process streams, in addition to more than 5% polycyclic aromatic hydrocarbons (IARC 1989). Residual fuel oils are generally more complex in composition and impurities than distillate fuel oils; therefore, a specific composition cannot be determined (Air Force 1989). Sulfur content in residual fuel oils has been reported to be from 0.18% to 4.36% by weight.

TABLE 3-1. Chemical Identity of Fuel Oils

Characteristic	Fuel oil no. 1	Fuel oil no. 1-D	Fuel oil no. 2	Fuel oil no. 2-D	Fuel oil no. 4	Fuel oil UNSP
Synonym(s)	Kerosene; coal oil; kerosine; range oil; straight run kerosene; distil-late fuel oils, light; furnace oil no. 1; Deobase®; JP-5; JP-1; range oil[a,b,c]	Diesel fuel; diesel fuel oil no. 1; diesel oil no. 1; no. 1 diesel; diesel oil (light); Arctic diesel[a,d,e]	API no. 2 fuel oil; gas oil; home heating oil no. 2; number 2 burner oil; diesel fuel; furnace oil no. 2[b]	Diesel fuel; diesel fuel oil no. 2; diesel oil no. 2; no. 2 diesel; diesel oil (medium)[a,d,e]	Oil, fuel, no. 4; residual fuel oil no. 4; no. 4 fuel oil; residual fuel oil; marine boiler fuel; marine diesel fuel; diesel fuel no. 4; grade 4[a,d,e]	No data
Registered trade name(s)	Deobase®[c]	No data	No data	No data	No data	No data
Chemical formula[f]	No data	No data	No data	No data	No data	No data
Chemical structure[f]	No data	No data	No data	No data	No data	No data
Identification numbers:						
CAS registry	8008-20-6[c]/70892-10-3[e]	No data	68476-30-2[d]	68476-34-6[a]	68476-31-3[b]	No data
NIOSH RTECS	OA5500000[c]	No data	HZ1800000[d]	No data	No data	LS8950000[d]
EPA hazardous waste	No data	No data	No data	No data	No data	No data
OHM/TADS	7217063[e]	No data	No data	No data	7217065[e]	No data
DOT/UN/NA/IMCO shipping	UN 1223; IMO 3.3[c]	No data	No data	No data	No data	No data
HSDB	632[c]	No data	No data	No data	No data	No data
NCI	No data	No data	C54795[g]	No data	No data	No data

[a]IARC 1989
[b]RTECS 1991
[c]HSDB 1991
[d]Air Force 1989
[e]OHM/TADS 1985
[f]Fuel oils are mixtures of various hydrocarbons designed to meet specifications set forth by the American Society for Testing and Materials (IARC 1989); therefore, chemical structure, chemical formula, and molecular weight cannot be determined.
[g]Sax and Lewis 1989

CAS = Chemical Abstracts Services; DOT/UN/NA/IMCO = Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code; EPA = Environmental Protection Agency; HSDB = Hazardous Substances Data Bank; NCI = National Cancer Institute; NIOSH = National Institute for Occupational Safety and Health; OHM/TADS = Oil and Hazardous Materials/Technical Assistance Data System; RTECS = Registry of Toxic Effects of Chemical Substances

CHEMICAL AND PHYSICAL INFORMATION

TABLE 3-2. Analysis of Fuel Oils[a]

Hydrocarbon type	Volume %	
	Fuel oil no. 1	Fuel oil no. 2
Paraffins (n- and iso-)	52.4	41.3
Monocycloparaffins	21.3	22.1
Bicycloparaffins	5.1	9.6
Tricycloparaffins	0.8	2.3
Total saturated hydrocarbons	79.7	75.3
Olefins	No data	No data
Alkylbenzenes	13.5	5.9
Indans/tetralins	3.3	4.1
Dinaphthenobenzenes/indenes	0.9	1.8
Naphthalenes	2.8	8.2
Biphenyls/acenaphthenes	0.4	2.6
Fluorenes/acenaphthylenes	No data	1.4
Phenanthrenes	No data	0.7
Total aromatic hydrocarbons	23.6	24.7

[a]Derived from IARC 1989; provided by the American Petroleum Institute

3.2   PHYSICAL AND CHEMICAL PROPERTIES

Information regarding the physical and chemical properties of fuel oils is located in Table 3-3.

TABLE 3-3. Physical and Chemical Properties of Fuel Oils[a]

Characteristic	Fuel oil no. 1	Fuel oil no. 1-D	Fuel oil no. 2	Fuel oil no. 2-D	Fuel oil no. 4	Fuel oil no. UNSP
Molecular weight	No data	No data	No data	No data	No data	No data
Color	Pale yellow[b]; Colorless to brown[c,d]	Colorless to brown[c]	Colorless to brown[c]	Colorless to brown[c]	Colorless to brown[c]	Colorless to brown[c]
Physical state	Liquid[c]	Liquid[c]	Liquid[c]	Liquid[c]	Liquid[c]	Liquid[c]
Melting point	-45.6°C[d]	-34°C[d]	-29°C[d]	18°C[d]	-29°C to -9°C[d]; -46°C[c]	-48°C–18°C[c]
Boiling point	175–325°C[b]; 200–260°C[d]	193–293°C[d]	160–360°C[f] 282–338°C[d]	282–338°C[d]	101–588°C[d]	151–>588°C[c]
Density:						
At 15°C	0.810–0.9360 g/mL[c]	0.810–0.9360 g/mL[c]	No data	No data	0.810–0.9360 g/mL[c]	No data
At 20°C	0.80 g/mL[e,f]	No data	0.8700–0.9500[c]	0.8700–0.9500 g/mL[c]	1 g/mL[e]	No data
Odor	Kerosene-like[c]	Kerosene-like[c]	Kerosene-like[c]	Kerosene-like[c]	Kerosene-like[c]	Kerosene-like[c]
Odor threshold (ppm)	0.082[f]; 1[d]	0.7[d]	No data	No data	0.5[e]	No data
Solubility						
Water at 20°C	≈5 mg/L[c]	≈5 mg/L[c]	≈5 mg/L[c]	≈5 mg/L[c]	≈5 mg/L[c]	≈5 mg/L[c]
Organic solvent(s)	Miscible with other petroleum solvents[b]	No data	No data	No data	No data	No data
Partition coefficients:						
Log K_{ow}	3.3–7.06[c]	3.3–7.06[c]	3.3–7.06[c]	3.3–7.06[c]	3.3–7.06[c]	3.3–7.06[c]
Log K_{oc}	3.0–6.7[c]	3.0–6.7[c]	3.0–5.7[c]	3.0–6.7[c]	3.0–6.7[c]	3.0–6.7[c]
Vapor pressure at 21°C	2.12–26.4 mmHg[c]	2.12–26.4 mmHg[c]	2.12–26.4 mmHg[c]	2.12–26.4 mmHg[c]	2.12–26.4 mmHg[c]	2.12–26.4 mmHg[c]
Henry's law constant at 20°C - atm-m^3/mol	5.9 x 10^{-5}–7.4[c]	5.9 x 10^{-5}–7.4[c]	5.9 x 10^{-5}–7.4[c]	5.9 x 10^{-5}–7.4[c]	5.9 x 10^{-5}–7.4[c]	5.9 x 10^{-5}–7.4[c]
Autoignition temperature	229°C[d]	177.0–329.0°C[d]	257°C[d]	254–285°C[d]	263°C[d]	No data
Flashpoint (close cup)	38°C[c,d]	38°C[d,f]	58°C[d]	52°C[d,f]	55°C[f];>54°C[d]	No data
Flammability limits (% volume in air)	0.7%–5%[d]	1.3–6.0%[d]	0.6–7.5%[d]	1.3–6.0%[d]	1.0–5.0%[d]	No data
Conversion factors	No data	No data	No data	No data	No data	No data
Explosive limits	0.7%–5%[b]	No data	No data	No data	No data	No data

[a]Values listed are specifications required or general characteristics of each class of fuel oils.
[b]HSDB 1991
[c]Air Force 1989
[e]OHM/TADs 1985
[d]Coast Guard 1985
[f]IARC 1989

4. PRODUCTION, IMPORT, USE, AND DISPOSAL

4.1 PRODUCTION

Fuel oils are produced from refined crude petroleum to meet specifications for a particular use (Air Force 1989; IARC 1989). These specifications are designated by the American Society for Testing and Materials (ASTM) (IARC 1989). Light fuel oils such as fuel oil no. 1 and no. 2 are manufactured from straight distillation of crude oil, or distillation of crude oil in the presence of a catalyst, and are chemically enhanced with antioxidants, dispersants, or corrosion inhibitors to meet the requirements for a specific application. Fuel oil no. 1 is a product of the straight-run distillation of crude petroleum (HSDB 1991). It consists of a mixture of petroleum hydrocarbons, chiefly of the methane series, which typically have from 10 to 16 carbon atoms per molecule (HSDB 1991; IARC 1989). The typical components of the end product of fuel oil no. 1 include paraffins (*n*-, iso-, monocycle-, bicycle- and tricycle-), olefins, aromatics, and nitrogen and sulfur impurities (Air Force 1989; IARC 1989). Fuel oil no. 2 is manufactured from the blending of straight-run and catalytically cracked distillates (IARC 1989). The types of each stream and the proportioned amounts in the resulting fuel oil depend on the quality of the crude petroleum used for the distillations.

The distillate manufacturing processes of diesel fuel oils, such as fuel oil 1-D and fuel oil 2-D, are similar to those of fuel oil no. 1 and fuel oil no. 2, respectively (IARC 1989). Fuel oil no. 1-D is manufactured from a straight-run distillate process. Diesel fuel oil (l-D, 2-D) is defined as the fraction of petroleum that distills after kerosene (Air Force 1989). Fuel oil no. 2-D is also made from mixing of straight-run and catalytically cracked distillates (IARC 1989).

Residual fuel oils, such as fuel oil no. 4, are manufactured by the addition of blending stocks to distillation residues in order to meet viscosity specifications set by ASTM (IARC 1989).   No data were located for the manufacturing process of fuel oil UNSP.

Although most facilities that refine crude petroleum in the United States produce a fuel oil. no. 1 fraction (HSDB 1991), only producers that market fuel oil no. 1 as an end product are listed as commercial manufacturers. These manufacturers include Claiborne Gasoline Company (Claiborne and Union Parish, Louisiana), Continental Oil Company (Acadia Parish, Louisiana), Sun Production

Company (Starr County, Texas), Exxon Corporation (Pledger County, Texas), Atlantic Richfield Company (New York, New York), and Shell Oil Company (Houston, Texas) (HSDB 1991). Since fuel oils nos. 1, 1D, 2, 2-D, and 4, and fuel oil UNSP are not required to be reported under SARA Section 313, there are no data for these fuel oils in the 1990 Toxics Release Inventory (TRI90 1992).

Between 1970 and 1980, distillate fuel production, which includes the production of diesel fuels, increased steadily (IARC 1989). The total production volume in 1970 was 2,460,000 barrels, which increased to 3,167,000 barrels in 1979; however, between 1980 and 1986, distillate fuel production volumes fluctuated. After 1986, production volume of distillate fuels steadily increased to a maximum to 3,167,000 barrels in 1990 (API 1991).

Production of kerosene has steadily decreased since 1970 (API 1991). The supply of kerosene produced in 1970 was 95,600,000 barrels. By 1975, production volume had dropped to 55,500,000 barrels. As of 1990, only 16,400,000 barrels of kerosene were produced. Production volumes of residual fuel oils showed a sharp increase between 1970 and 1980 and a sharp decline between 1980 and 1985 (IARC 1989). The total production volume of residual fuels in 1970 was 262,000,000 barrels, which increased to 456,000,000 barrels in 1975, 605,000,000 barrels in 1980, decreased to 321,000,000 barrels in 1985, and increased slightly to 346,000,000 barrels in 1990 (API 1991). No data were located for production volumes of fuel oil UNSP.

4.2 IMPORT/EXPORT

Imports of distillate fuels have varied from year to year since the 1970s. Since 1975, imports of distillate fuel oils such as fuel oils no. 1 and no. 2 into the United States have been low compared to the amount of distillate fuel oils produced in the United States (API 1991). Annual import volumes fluctuated between 57,000,000 barrels in 1975 and 64,000,000 barrels in 1983 with a maximum of 91,250,000 barrels in 1977. From 1984 to 1989, imports of distillate fuels steadily increased from 99,439,000 barrels in 1984 to 111,000,000 barrels in 1989. Imports of kerosene fluctuated between 1975 and 1984 and then showed a steady increase from 1985 to 1987, attaining an annual maximum of 6,935,000 barrels in 1987. From 1988 to 1990, imports of kerosene deceased to a low of 1,825,000 barrels in 1990 (API 1991).

Imports of residual fuel oils such as fuel oil no. 4 generally decreased in the period between 1975 and 1989 (API 1991). In 1975, total imports of residual fuel oil were 447,000,000 barrels; however, imports gradually decreased over this 15-year period to 230,000,000 barrels in 1985. No information was located regarding diesel fuel imports or unspecified fuel oil imports.

Exports of fuel oil no. 1 between 1972 and 1975 ranged from 14,000 tons in 1972 to 98,000 tons in 1975 (HSDB 1991). Exports of distillate fuel oils (which include fuel oil no. 1, fuel oil no. 2, diesel fuel, and fuel oil no. 4) increased almost 100-fold between 1975 and 1990 (API 1991). In 1975, a yearly average of 365,000 barrels of distillate fuel oils were exported out of the United States. By 1990, exports had increased to an average of 3,900,000 barrels. Little kerosene has been exported from the United States since the 1970s. In 1971, approximately 365,000 barrels were exported from the United States. The next 2 years for which export volumes were reported for kerosene were 1983 and 1984, when 365,000 barrels were exported each year. However, between 1986 and 1990, export volumes doubled from 730,000 barrels in 1986 to 1,820,000 barrels in 1990 (API 1991). Comprehensive export data for kerosene prior to 1986 are not available. Kerosene exportation between 1987 and 1989 remained relatively constant with a yearly export average of approximately 547,500 barrels. However, by 1990, the annual export of kerosene was 2,190,000 barrels (API 1991), an increase of approximately 400%. Residual fuel oil exports also increased, from a daily average of 15,000 barrels in 1975 to 215,000 barrels in 1989 (API 1990).

4.3 USE

Fuel oils have many commercial and military uses. Kerosene, a type of fuel oil no. 1, was chosen as a jet fuel during the development of the first jet engines, largely because gasoline was in short supply during wartime (IARC 1989). Fuels that are used currently in jet engines, such as JP-5, are very similar to kerosene, and are also included in the fuel oil no. 1 designation. At present, fuel oil no. 1 is used almost exclusively for domestic heating (Air Force 1989) with burners of the vaporizing type (IARC 1989); although fuel oil no. 1 was used as a jet fuel in the first jet engines (IARC 1989). In the petroleum industry, fuel oil no. 1 has also been used as an illuminating fuel, a motor fuel, and a heating fuel (HSDB 1991). In the pesticide industry, fuel oil no. 1 has been used as a vehicle for insecticides and fungicides (HSDB 1991). Fuel oil no. 1 is also used in kerosene lamps, flares, and stoves. Fuel oil no. 2 has been primarily used as a home heating oil and as an industrial heating oil

(IARC 1989). Fuel oil no. 2 is also reported to be used in atomizing-type burners (Air Force 1989; IARC 1989).

Diesel fuels are most commonly used as transportation fuels for diesel engines (Air Force 1989; IARC 1989). Fuel oil no. 1-D is used in engines that require frequent load and speed changes (Air Force 1989). Fuel oil no. 2-D has been used for engines that are in industrial or heavy mobile service (Air Force 1989). Diesel fuels have been used in stationary gas turbines to generate electric power (IARC 1989).

In electric utilities, residual fuel oils, such as no. 4, have been used to process steam for electric plants (IARC 1989). Fuel oil no. 4 has been used in commercial and industrial burner installations that are not equipped with preheating facilities (Air Force 1989). In other industries, such as the maritime industry, plants and factories, and the petroleum industry, residual fuel oils have been used for space and water heating, pipeline pumping, and gas compression, as well as in road oils, and in the manufacture.

U.S. consumption of distillate fuels used for heating decreased gradually between 1979 and 1983 (IARC 1989). The annual domestic demand for distillate fuel oils was approximately 1,214,355,000 barrels in 1979, decreasing to 981,850,000 barrels in 1983. The U.S. demand for distillate fuels subsequently rose from 1984 to 1989, reaching an annual maximum of 1,248,700,000 barrels in 1989 (API 1991). The annual domestic demand for residual fuel oils also decreased between 1979 (1,034,800,000 barrels) and 1985 (475,600,000 barrels). After 1985, the demand varied from an annual average of 561,000,000 barrels in 1986 to 486,180,000 barrels in 1990 (API 1991). Finally, the average annual domestic demand for kerosene decreased from 68,990,000 barrels in 1979 to 16,790,000 barrels in 1990 (API 1991).

4.4 DISPOSAL

Incineration is one method of disposal proposed for fuel oils no. 1 and no. 2 (OHM/TADS 1985). Other methods may be used to disposal of oil spills, including absorption (straw, polyurethane foam, activated carbon, and peat have been used as absorbents), gelling agents, combustion promoters, dispersants, and mechanical systems (OHM/TADS 1985). Biodegradation has also been suggested as a means of disposal for spills onto soil (OHM/TADS 1985). Hydrocarbon-degrading bacteria have been

shown to degrade petroleum products into smaller units and eventually into nonseparable particles
(Butt et al. 1988). Soil contaminated with fuel oil no. 1 was found to have a growth response of
10 E+6 colony forming units per mL in 7 out of 21 types of bacteria isolated for sample study (Butt et al.
1988). For more information on biodegradation, refer to Chapter 5.

5. POTENTIAL FOR HUMAN EXPOSURE

5.1 OVERVIEW

Fuel oils are complex mixtures of aliphatic and aromatic hydrocarbons whose exposure potentials are based on the environmental fate of the individual components of the mixtures, particularly n-alkanes, branched alkanes, benzene and alkylbenzenes, naphthalenes, and PAHs.

Fuel oils may enter the water or soil environment as a result of spills during use or transportation or from leaking storage facilities or pipelines. The more volatile components of fuel oils (low molecular weight alkanes) will evaporate from the soil or water and enter the atmosphere where they will be degraded. The higher molecular weight aliphatic components ($>C_{20}$) of fuel oils have very low water solubility and will not volatilize from soils or surface waters. Consequently, these heavier compounds will remain on the soil or in the water column where they may be adsorbed to particulate organic matter in water or soil and, in water, will settle to the sediment. They will eventually be biodegraded by microorganisms in the soils and sediments. The rate and extent of biodegradation are dependent on the ambient temperature, the presence of a sufficient number of microorganisms capable of metabolizing these hydrocarbons, the amount of aromatic species in a given oil, and the concentration of fuel oil. The aromatic components (benzene and alkylbenzenes) of fuel oils tend to partition into the polar phase of the environment and migrate through the soil to the groundwater. However, these components of fuel oil are also the most biodegradable.

The National Occupational Exposure Survey conducted by NIOSH between 1980 and 1983 estimated that 96,345 employees were exposed to fuel oil no. 2, 1,526 workers were exposed to fuel oil no. 4, and 1,076,518 employees (including 96,255 females) were exposed to kerosene in the workplace. Worker exposure was most likely in industries associated with machinery and special trade contractors. General population exposure is potentially the greatest for persons living near an area where fuel oils have been dumped and have migrated into the groundwater or when fuel oil vapor has penetrated the soil and may enter basements of buildings.

Fuel oil no. 1 has been identified in 23 of the 1,397 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL), and fuel oil no. 2 has been identified in at least 4

of the NPL sites (HAZDAT 1991). The frequency of these sites within the United States can be seen in Figures 5-l and 5-2.

5.2 RELEASES TO THE ENVIRONMENT

Releases of fuel oils are not required to be reported under SARA Section 313; consequently, there are no data for these compounds in the 1990 Toxics Release Inventory (TR190 1992).

5.2.1 Air

Fuel oils may be released to air as fugitive emissions from storage facilities or as a result of their use as fuel for combustion engines; in stoves, lamps, and furnaces; and as solvents. Atmospheric emissions of a fuel oil may be determined primarily by detection of its volatile hydrocarbon components. Atmospheric releases of fuel oil no. 1 (kerosene) may be expected as a result of its use as a solvent for insecticides and fungicides and by evaporation from open containers when used as a fuel. Indoors, unvented kerosene heaters are a source of sulfur dioxide, nitrogen dioxide, carbon monoxide, respirable suspended particulate matter, and acids in the vapor and particulate phase (Koutrakis et al. 1992; WHO 1991). In mobile homes in particular, use of kerosene heaters resulted in an increase in CO and airborne unburned kerosene fuel, an increase in mutagenicity of particle-phase organics, and little mutagenicity in the semivolatile organics (Mumford et al. 1991). Emission rates of carbon monoxide from unvented kerosene heaters in mobile homes were measured to be 10 to 272 μg/kJ for convective heaters, and 57 to 264 μg/kJ for radiant heaters; concentrations of CO averaged 7.4 ppm with a peak of 11.5 ppm. Hydrocarbon emissions detected at fixed-roof fuel oil and low vapor pressure distillate storage tanks were only 8% of the emissions estimated for these facilities using the equations developed by the American Petroleum Institute (Wilson et al. 1978). The relationships have been quantified between the components in diesel fuel blends, the variables of the emissions when the fuel is burned, and their biological effects (Westerholm and Egerback 1991). Combustion from kerosene heaters contributes to the radon progeny particle size distribution in particles in the size range of 0.02-0.08 μm (Tu et al. 1991). Fuel oil combustion was determined to be a negligible contributor to atmospheric aerosol particles in the Toyama Prefecture of Japan (Toriyama et al. 1991). For residential oil furnaces, the mutagenicity of emissions in revertants per megajoule increases as particulate emissions increase, and emissions are up to 3 orders of magnitude

FIGURE 5-1. FREQUENCY OF NPL SITES WITH FUEL OIL NO. 1 CONTAMINATION *

FREQUENCY ▦ 1 SITE ▨ 2 SITES
■ 9 SITES
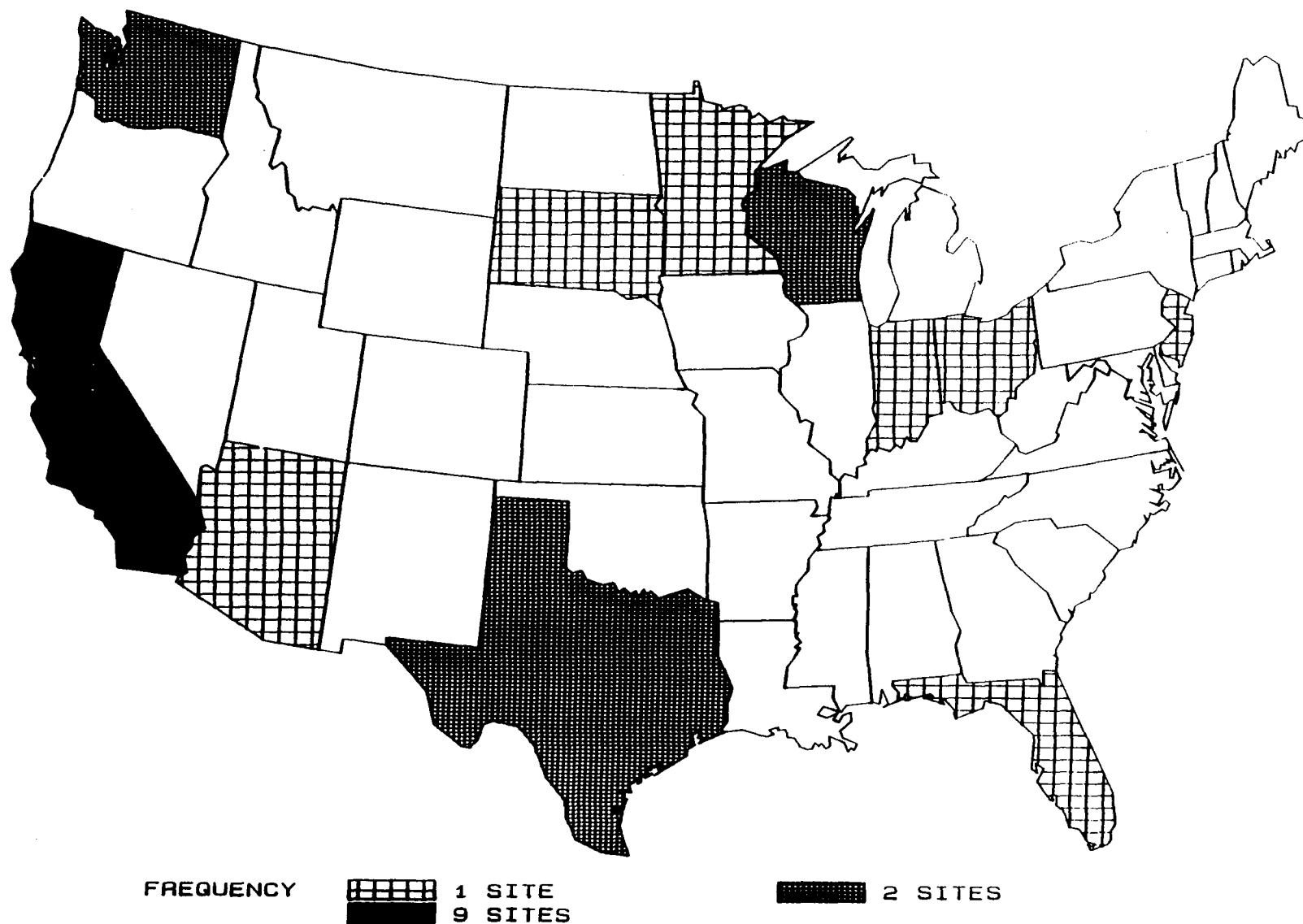
*Derived from HazDat 1994

FIGURE 5-2. FREQUENCY OF NPL SITES WITH FUEL OIL NO. 2 CONTAMINATION *

FREQUENCY ⊞⊞ 1 SITE
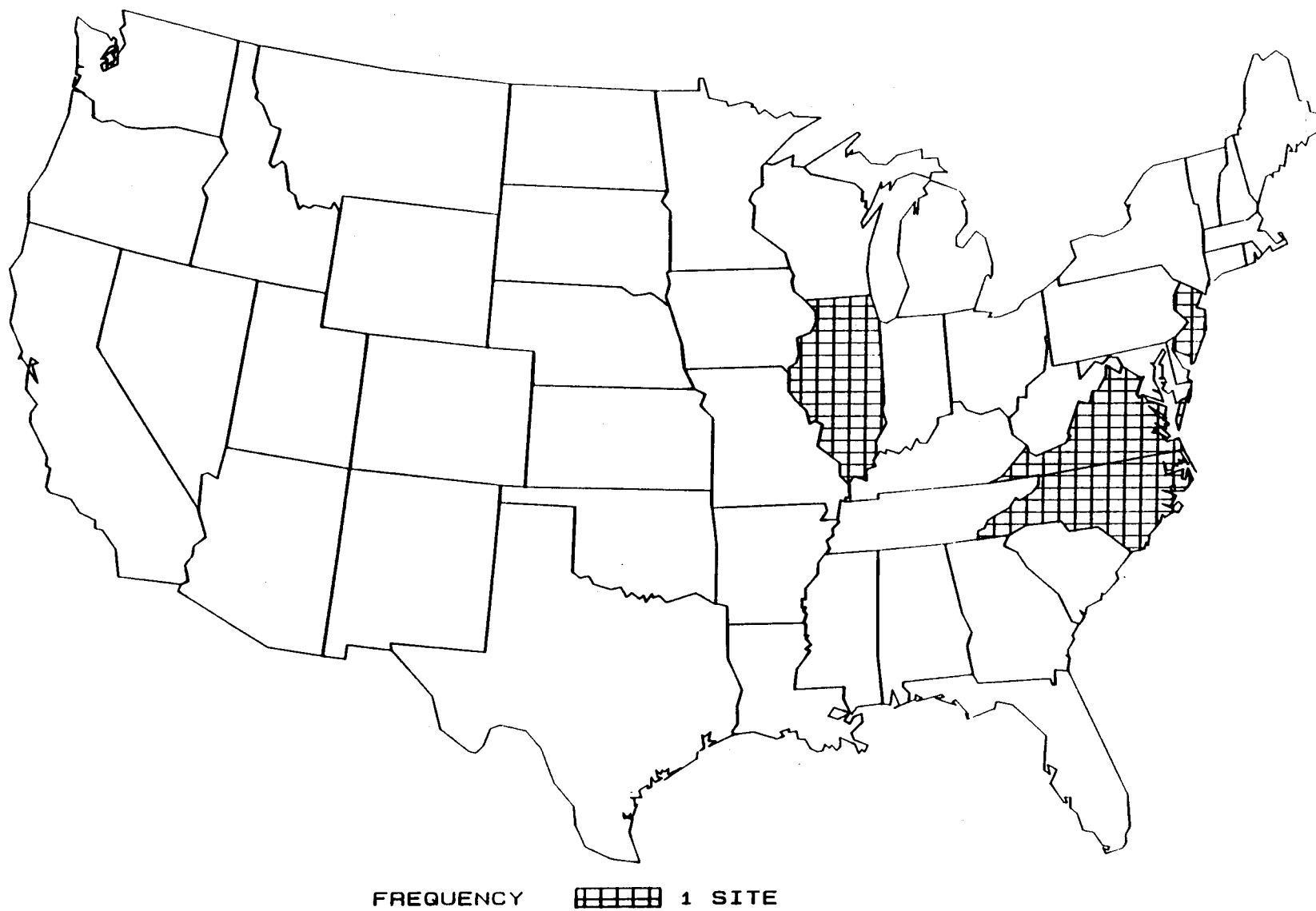
*Derived from HazDat 1994

less mutagenic than wood smoke from conventional, uncontrolled woodstoves on a per unit of fuel energy value (EPA 1992).

Fuel oils are released to the atmosphere as vapor when used as smoke screens or fogs by the U.S. Army; concentrations may range from 13 to 2,000 mg/m^3. Deposition of these particles to soil and water may result in fallout concentrations of 6-60 g/m^2 (Army 1986).

5.2.2 Water

Fuel oils may be released to surface waters as a result of fuel spills from transport vessels or from facilities located adjacent to surface waters, in runoff from industrial facilities where they are used as fuels or solvents, or from the intentional disposal of excess fuel oils down drains. Kerosene may enter surface and groundwater as a result of leaking aboveground and underground storage tanks, including those found at disposal facilities for spent nuclear fuel reprocessing operations (DOE 1989c). In 1988, a rupture of an aboveground storage tank outside of Pittsburgh, Pennsylvania, released more than l,000,000 gallons of fuel oil no. 2 into the Monongahela and Ohio Rivers and disrupted local drinking water supplies (MacKerron and Kiesche 1988). Fuel oils may enter the marine ecosystem as a result of accidental spills from transport vessels, such as occurred when an oil barge released more than 650,000 liters of fuel oil no. 2 into Buzzards Bay, Massachusetts, in 1969 (EPA 1981); a second spill occurred in this area in 1974 (Bums and Teal 1979; Teal et al. 1978). Since 1915, ships and barges containing fuel or oil have sunk in Puget Sound or adjacent waters. Nine of these vessels have been classified as posing a potential hazard to human health or the aquatic environment because they have not been salvaged and contain large known reservoirs of fuel or oil, some which may have been discharged after sinking (EPA 1991d). Between 1986 and 1991, 4.6 million gallons of fuel oil no. 2 and 12.6 million gallons of fuel oil no. 6 were reported released into Newark Bay, New Jersey (Gunster et al 1993a). A significant source of petroleum to the bay appears to be municipal and industrial wastewater treatment facilities; over 600,000 gallons of petroleum products have been discharged since 1982 (Gunster et al 1993b). Approximately 170,000 gallons of diesel fuel arctic entered the marine ecosystem surrounding Arthur Harbor on the Antarctic Peninsula in 1989 when an Argentine resupply/tourist ship sank (Karl 1992), and in 1988, 230,000 gallons of Bunker C fuel oil was released from a barge off the Washington coast (Strand et al 1992).

Fuel oils that are accidentally released to inland waters are reported to the Emergency Response
Notification System (ERNS). Total spill volumes of fuel oils released in 1991 were as follows (ERNS
1992):

Fuel oil	No. of notifications	Gallons released
No. 1	8	43,190
No. 1-D	20	3,292
No. 2	916	14,558,313
No. 2-D	28	2,769
No. 4	35	12,239

Storm runoff from an industrial site in Rhode Island used by oil distributors, scrap metal dealers, and
metal finishers contained a hydrocarbon product resembling fuel oil no. 2. This product comprised 4%
of the total petroleum hydrocarbons detected in the runoff, most of which were associated with
crankcase oil (Latimer et al. 1990). Two freighters collided off the coast of South Africa in 1992; the
freighter transporting 160 tons of marine fuel oil and 53 tons of gas oil sank (Molden 1992).

5.2.3 Soil

Fuel oils may be released to soil as a result of accidental spills (Strayer et al. 1983; Rosenblatt and
Montemagno 1992) and leaking from underground storage tanks or pipelines. Hydrocarbon
contamination (due to diesel fuel spillage and leakage related to ship and boating activities) has been
measured in sediment samples in Arthur Harbor on the Antarctic Peninsula (Kennicutt et al.
Also, approximately 200,000 gallons of liquid hydrocarbons were released into the underlying soil at a
fuel-storage terminal in northern Virginia. The soil vapor above the resultant hydrocarbon plume and
the groundwater below were analyzed for total and individual petroleum hydrocarbons (Mushrush et al.
1994).

5.3 ENVIRONMENTAL FATE

5.3.1 Transport and Partitioning

The transport and dispersion of fuel oils are dependent on the water solubility and volatility of the aliphatic and aromatic hydrocarbon fractions. Lower molecular weight hydrocarbons such as *n*-alkanes may volatilize relatively quickly from both water and soil, while larger aliphatics (greater than C_9 chain length) may be sorbed to organic particles in water or soil. Aromatic hydrocarbons will be dissolved in the aqueous phase in both soil and water and may undergo some volatilization.

Aerosol oil particles of SGF-1, a distillate oil chemically similar to fuel oil no. 2, used by the military to generate oil fogs (particle diameter of 0.5-1.2 μm) may remain aloft for approximately 1 hour, may be transported 1-10 km (average of 5 km) downwind during this time, and, with the exception of evaporative losses, will be deposited to soil or surface waters (Army 1986; Liss-Suter et al. 1978).

The solubility of fuel oil no. 2, particularly the alkane and isoprenoid fractions, in seawater is increased by the presence of fulvic acid, although the solubilities of phenanthrene or anthracene, both polycyclic aromatic hydrocarbons, are unaffected by the presence of humic materials (Boehm and Quinn 1973). Unfiltered Narragansett Bay water was able to dissolve 1,560 μg/L of fuel oil no. 2, although removal of dissolved organic material from the water reduced the solubility by 33-60% (Boehm and Quinn 1974). The water-dissolved fraction of JP-5 fuel was stable for 24 days (Edgerton et al. 1987).

The microlayer at the air-water interface may be composed of different fuel oil hydrocarbons than the subsurface waters. When fuel oil no. 2 was added below the surface of a marine ecosystem, the microlayer was enriched for both saturated and aromatic hydrocarbons compared with the subsurface water, with a significantly greater proportion of total saturates compared with aromatics. The hydrocarbons in the microlayer were predominantly higher molecular weight hydrocarbons, greater than C_{18} for saturates and phenanthrene (F_2) for aromatics. This indicates that there is transport of all the hydrocarbons from the water column to the microlayer and weathering of the hydrocarbons by evaporation once in the microlayer (Gearing and Gearing 1982b). Weathering of fuel oil spills on water surfaces is dependent on the vapor pressures of the hydrocarbon components and can be correlated with the evaporation rates of the alkanes present in the fuel oil mixture. Components with

vapor pressures higher than n-octane rapidly evaporate from the surface of the fuel oil spill while those with vapor pressures lower than n-octadecane persist and may result in a more viscous residue, eventually retarding the evaporation of other components (Regnier and Scott 1975.

Addition of fuel oil no. 2 to a laboratory marine ecosystem showed that the insoluble, saturated hydrocarbons in the oil were slowly transported to the sediment on suspended particulate material. The particulate material contained 40-50% of the total amount of aliphatics added to the system and only 3-21% of the aromatic fraction (Oviatt et al. 1982). This indicates that most aromatic hydrocarbons are dissolved in the water (Coleman et al. 1984), whereas the aliphatic hydrocarbons are not (Gearing et al, 1980; Oviatt et al. 1982). In a similar experiment, when fuel oil no. 2 was added continuously to a marine ecosystem for 24 weeks, oil concentrations in the sediment remained low until 135 days after the additions began, but then increased dramatically to levels that were 9% of the total fuel oil added (108 g/tank) and 12% of the total fuel oil saturated hydrocarbons. The fuel oil concentrations in the sediment began to decrease quite rapidly after the maximum levels were reached. The highest sediment concentrations of saturated hydrocarbons (106-527 μg/g) were found in the surface flocculent layer, with concentrations decreasing with sediment depth from 22 μg/g to not detectable at 2-3 cm below the sediment surface. Fuel oil was being transported to the sediments adsorbed to suspended material in the water column with smaller particles (<45 μm) containing 2.2 times more fuel oil than larger particles (>45 μm) (Wade and Quinn 1980). Although biodegradation may remove many of the soluble, aromatic hydrocarbons, a residue of 10-20% of the total added oil, composed primarily of branched alkanes, cycloalkanes, and aromatics, may remain in the top 2 cm of the sediment for over a year (Gearing et al. 1980; Oviatt et al. 1982). A spill of fuel oil no. 2 to waters off Massachusetts showed that the oil was detected at distant monitoring locations 46 months after the spill, although the concentration in the sediments remained the greatest in the vicinity of the original spill. Oil continued to move in pulses from more polluted areas to less polluted areas for several years after the spill, with much of the oil being exhumed from sediments in the shallower waters as a result of storm action (EPA 1981). Movement of the oil to adjacent salt marshes indicated that the oil was sorbed to the anoxic sediments where it persisted (Bums and Teal 1979).

The partitioning of fuel oil no. 2 and kerosene into drinking water after 17 hours of incubation resulted in only 1.0% of the fuel oil and 0.7% of the kerosene being dissolved in the water. Further analysis of these fuels indicated that although each compound contains approximately 50% aliphatic

hydrocarbons (by weight percent), the water-soluble fractions contained primarily aromatic constituents (>93%) including benzenes and naphthalenes as shown below (Coleman et al. 1984):

	Water-soluble fraction		
	Whole product*	1/2 hour	17 hours
Kerosene			
Alkanes + cycloalkanes	68.6	4.5	0.5
Benzene + substituted benzenes	13.7	63.5	53.2
Naphthalene + substituted naphthalenes	5.7	29.6	44.8
Fuel oil no. 2			
Alkanes + cycloalkanes	48.0	<0.5	<0.5
Benzene + substituted benzenes	4.5	25.8	19.4
Naphthalene + substituted naphthalenes	32.4	73.4	79.7

*Estimated weight percent

Partitioning of eight PAHs between four diesel fuels and water can be described using Raoult's law for activity coefficients (Lee et al. 1992).

A coal-liquid spill resulted in soil penetration to a depth of 8 meters; the soil was described as crushed stone mixed with glacial outwash sand and gravel. Soil columns collected 6 months after the spill were leached with a synthetic rain solution, and the leachates were collected and analyzed for total phenolics. Over the 12-month leaching period, the phenol fraction of the residual coal-derived fuel decreased by 98%, primarily by dissolution. Groundwater monitoring down-gradient from the spill detected phenols even though the surface of the spill had been sealed to prevent leaching of water-soluble components by precipitation (Strayer et al. 1983).

The movement of a synthetic kerosene through soil was found to be dependent on the moisture content of the soil. The greater the moisture content (i.e., 4% compared with 0.8%) of the soil, the less the adsorption of the more volatile components of the kerosene and the greater and the more rapid the penetration of the liquid component through the soil. Conversely, the upward mobility of both the liquid and vapor phases of kerosene through soil decreased with increased moisture content, and at field capacity, the upward capillary movement of the kerosene was completely inhibited (Acher et al.

1989). Desorption of a simulated kerosene applied to three types of soil, each with a moisture content at 70% of field capacity, was found to be complete after 30 days of exposure to the atmosphere with the slowest desorption from the soil having the greatest organic content (Yaron et al. 1989). Kerosene loss from a dune sand, a loamy sand, and a silty loam soil after 50 days showed that volatilization of all kerosene components was greatest from the dune sand and loamy sand soils. The larger pore size of these types of soil compared with the silty loam soil was thought to be the reason for the increased volatilization (Galin et al. 1990a). Movement of kerosene through three grades of sand was affected mainly by volatilization of the C_9—C_{13} components with a subsequent increase in the viscosity of the remaining kerosene residue and a decrease in the infiltration rate through the inert porous media (Galin et al. 1990b).

The movement of kerosene through various types of soil over a 12 hour period was studied. Upward, downward, and lateral movement was greatest in soil of the mica/kaolinite type (11% clay content) 40, 102, and 45 cm, respectively. Movement through soils that were primarily kaolinite (clay content of 26-52%), regardless of the direction, ranged between 20 and 33 cm (EPA 1986b). Application of herbicides such as *S*-ethyl dibutylthiocarbamate to a field using kerosene or fuel oil no. 2 as solvents (up to a volume of 40 gallons per acre) increased the inactivation of the herbicide on soil, whereas acetone, benzene, or xylene did not. The accelerated inactivation possibly resulted from a change in surface tension that facilitated the volatilization of the herbicide from the soil (Danielson and Gentner 1970).

Studies on the permeability of compacted micaceous soil used as a potential liner for landfills found that the permeability of the soil to kerosene and diesel fuel was 3-4 orders of magnitude greater for kerosene and 1-1..5 orders greater for diesel fuel compared with water (EPA 1984).

Aquatic organisms are known to bioconcentrate hydrocarbons. Addition of fuel oil no. 2 (containing 38% aromatics) to a commercial shrimp pond gave an initial concentration of approximately 0.03 ppm for total naphthalenes. The concentration of total naphthalenes (toxic components of fuel oil) peaked at approximately 0.3 ppm in the water after 48 hours and slowly decreased to background levels after 38 days. Sediment concentrations of the total naphthalenes rose from 0 ppm at 6 hours after the application of the fuel oil to 2 ppm at day 1, peaked at 14 days (9 ppm), and slowly declined to 1.5 ppm at 296 days. The concentrations of total naphthalenes in shrimp, clams, and oysters peaked at 24, 48, and 72 hours, respectively, with maximum concentrations in each organism exceeding the

naphthalene concentrations in the ambient water. When the contaminated organisms were placed in clean water after 38 days of exposure in the shrimp pond, depuration (to background levels) was complete after 10 days for shrimp and after 47 days for oysters, indicating that the concentrations in marine organisms are more closely correlated with water concentrations than with sediment concentrations of aromatic hydrocarbons (Cox et al. 1975). Lobsters exposed to a pulse of fuel oil no. 2, which simulated a small spill, were found to bioconcentrate PAHs (components of the fuel oil) in the hepatopancreas and muscle tissue within 34 days after exposure. PAH levels continued to be elevated at days 10 and 11; however, depuration to control levels occurred by day 20-21 (Williams et al. 1989, 1991).

Mussels *(Mytilis edulis)* exposed to a small spill (approximately 6,000 liters) of fuel oil no. 2 were followed for 86 days post-spill to assess the uptake and retention of the fuel oil components. Alkanes, cycloalkanes, and aromatic concentrations increased significantly in the mussel tissue the 1st day; however, by day 5 post-spill, the *n*-alkanes were barely detectable, and by day 21, the concentration of the unresolved complex mixture of alkanes-cycloalkanes was 30% of the day 1 concentrations. Concentrations of lower molecular weight aromatics (e.g., naphthalenes) decreased before the higher molecular weight aromatics (e.g., phenanthrenes). The biological half-lives of some fuel oil no. 2 components in mussels were as follows: n-C_{16}--0.2 days; n-C_{23}--0.8 days; C-2 naphthalene--0.9 days; C-3 naphthalene--1.5 days; phenanthrene--2.1 days; and unresolved complex mixture--2.8-3.9 days (Farrington et al. 1982a). Similar results were found in sea scallops (*Aequipecten irrudians),* which showed a gradual depletion of straight chain and branched chain hydrocarbons after exposure to fuel oil no. 2. However, the more toxic aromatic hydrocarbons were retained in scallop tissue for several months after exposure (Blumer et al. 1970). Sea mullet exposed to 5 ppm kerosene in sea water had a kerosene-like taint after 24 hours of exposure. In another study, oysters exposed to fuel oil no. 2 at 400 ppm for 8 hours accumulated 312 ppm of PAHs and 8.7-21.8 ppm of naphthalenes in their tissues; however, over 90% of the *n*-paraffins were released within 24 hours when the oysters were returned to oil-free water. Depuration of the aromatic hydrocarbons was much slower. When shrimp, clams, and fish were exposed to the water-soluble fraction of fuel oil no. 2, shrimp rapidly accumulated total naphthalenes for an hour then released them, clams accumulated the naphthalenes at a slower but constant rate for 24 hours, and the fish accumulated the naphthalenes very rapidly during 2 hours of exposure; all three species rapidly released the accumulated naphthalenes when placed in oil-free water with low or undetectable levels at 14 days (Anderson and Neff 1977).

5.3.2 Transformation and Degradation

The fate of four petroleum mixtures has been evaluated using three approaches-evaluating the fate of (1) indicator chemicals, (2) the mixture of the whole with a surrogate, and (3) the hydrocarbon mixture as a whole. The four mixtures were crude oil, JP-5, mineral spirits, and diesel. The choice of approach requires the consideration of availability and quality of data on the mixture, the mobility and toxicity of the mixture, and the availability of site data (Custance et al. 1992).

5.3.2.1 Air

No studies on the transformation or degradation of fuel oils in the atmosphere were located. However, volatile components of fuel oils such as benzene, toluene, xylenes, and PAHs may be expected to enter the atmosphere where they are subjected to degradation processes. Further information on the atmospheric degradation of selected volatile hydrocarbons are presented in the ATSDR toxicological profiles for these chemicals (ATSDR 1989, 1990a, 1991a, 1991b).

5.3.2.2 Water

The photooxidation of fuel oil no. 2 in water is quite rapid in sunlight. The rate is four times that of coal liquids, producing two photooxidant pools with reaction rate constants of 0.58/minute and 0.037/minute. The faster reacting photooxidant was tentatively identified as hydrogen peroxide and the slower reacting pool as a mixture of indane and tetralin hydroperoxides (Herbes and Whitley 1983). Two mechanisms appear to result in the formation of the peroxides: (1) reaction of free radicals with triplet oxygen to produce peroxy radicals and hydroperoxides, which in turn continue the chain reaction; and (2) formation of hydroperoxides by direct addition of excited singlet oxygen to reactive acceptors in the oil such as olefins. These reactions reached a maximum after approximately 90 hours of irradiation. Other photodegradation products of fuel oil exposed to ultraviolet radiation include phenols (linear increase in concentration during 165 hours of irradiation), naphthols, and carboxylic acids (Larson et al. 1977, 1979).

Biodegradation of fuel oils is dependent on the degradation of the various hydrocarbon fractions present in the oils. The relative order for biodegradation of the hydrocarbon fractions from the mostreadily degraded to the least is as follows: *n*-alkanes, iso-alkanes, olefins, low molecular weight

aromatics (at low, non-toxic concentrations), PAHs, and cycloalkanes (Bartha and Atlas 1977; Edgerton et al. 1987).

Water and sediment samples taken from Muddy Creek in the Rhode River of Chesapeake Bay were inoculated with 0.1% (volume/volume) of fuel oil no. 2 and incubated for 28 days. Growth of aerobic bacteria and fungi was enhanced by the addition of the fuel oil, although growth of yeast was inhibited (Walker and Colwell 1975). A mixed culture of estuarine bacteria was observed to degrade fuel oil no. 2 by 55% in 28 days with the primary microorganisms being *Bacillus* and *Pseudomonas.* Degradation of aromatic components was significantly greater than the n-alkanes (Walker et al. 1976).

Microorganisms readily able to degrade hydrocarbons were found in the Neuse River estuary in North Carolina. Although the estuary was relatively free of hydrocarbon contamination, 63% of the bacteria and 71% of the fungi isolated from surface water samples were able to utilize kerosene as the sole carbon source (Buckley et al. 1976). Weathered kerosene (volatile components were allowed to escape prior to testing) was spiked with four marker hydrocarbons, and the degradation of the markers was monitored. All four markers were degraded by a water-sediment mixture from an "oiled arm" of an Ohio lake; more rapid degradation was associated with mixtures taken from relatively polluted areas of the lake (Cooney et al. 1985), suggesting that biodegradation is enhanced by the presence of acclimated microorganisms. Marine bacteria capable of using fuel oil nos. 1, 2, and 4 were isolated from Narragansett Bay, Rhode Island. Most of the bacteria were found to utilize the aliphatic components of the fuel oils, primarily hexadecane, while only a few of the bacteria were able to degrade the aromatic components. The bacteria were able to degrade the hexadecane at 0°C but degradation was significantly improved when the incubation temperature was increased to 8°C and 16°C; similar but not such dramatic effects were seen in the degradation of naphthalene with increased temperature (Cundell and Traxler 1976).

The degradation of hydrocarbons fractions of fuel oil no. 2 was studied using a marine microcosm under different temperature, light, and biological activity regimes. Initial fuel oil concentrations ranging from 151 to 189 μg/L were found to decrease exponentially under all regimes, with temperature having the greatest effect on the half-lives of the component hydrocarbons. In cold water (0-2 °C), the half-life for total hydrocarbons was greater than 10 days, while in warmer water (17-21°C) the half-life decreased to approximately 30 hours. In cold water, saturated hydrocarbons were removed more rapidly than aromatic hydrocarbons, but in warmer water the half-lives of the

fractions were similar. For the saturated hydrocarbons, the half-life increased with increasing

molecular weight or with branched or cyclic moieties, i.e., small *n*-alkanes (C-12) had the shortest

half-life in both warm and cold water. Poisoning the microcosm to eliminate biodegradation had no

effect on the degradation of saturated hydrocarbons other than *n*-alkanes, but decreased the rate of

aromatic degradation by half, indicating that biodegradation is a significant removal process for both

n-alkanes and aromatics. Saturated hydrocarbons other than *n*-alkanes are most likely to be removed

by adsorption to particulate material in the water followed by sedimentation or the formation of a

micelle, which may rise to the surface of the water and evaporate. In cold water, aromatics are most

likely to be removed by volatilization, whereas in warmer water, biodegradation is the more important

removal process. Photooxidation did not appear to be a significant removal process for either

hydrocarbon fraction (Gearing and Gearing 1982a).

Fuel oil no. 2 (average water column concentration 182 μg/L) was added to the Marine Ecosystems

Research Laboratory (MERL) system for 5.5 months with a 2-month recovery period. Hydrocarbon

concentrations were then measured in the sediment and benthic organisms (*Glyceru americana,* a

carnivorous polychaete, and *Crepidulu sp.,* the common slipper shell). The alkane-cycloalkane and

aromatic fractions in the benthic organisms were suggestive of partially degraded fuel oil no. 2, and

for the aromatic hydrocarbons, were at least an order of magnitude greater than in the control tank.

Phenanthrenes, although present at lower concentrations in fuel oil no. 2 than naphthalenes, were

present at greater concentrations in the benthic organisms, suggesting that there is a bioaccumulation

potential for these compounds. Organism-specific differences show that organisms in close contact

with the sediment had relatively higher concentrations of lower molecular weight naphthalenes, as

these compounds are more readily retained in the sediment than the water column. Similarly,

organisms that live in the water column contained fewer naphthalenes. These results indicate that fuel

oil no. 2 degradation products are taken up by benthic organisms, that they may be selectively retained

in both sediments and aquatic organisms, and may thus enter the food chain (Farrington et al. 1982b).

After 3 months of exposure in a marine intertidal zone, the concentration of fuel oil no. 2 in the mean

lower low water (MLLW) area and 2 feet above the MLLW area had decreased by 30% and 53%,

respectively (NOAA 1982). Straight-chain alkanes degrade more readily than branched alkanes with a

ratio of 1.67 (*n*-C$_{17}$/pristine), although this ratio will decrease as biodegradation of the n-alkanes

progresses (Blumer et al. 1970; EPA 1981). Following spills of fuel oil no. 2 into Buzzards Bay,

Massachusetts, the concentrations of aromatic hydrocarbons decreased by a factor of 5 within

6 months of the spills with lighter molecular weight compounds decreasing at a faster rate than the

heavier molecular weight compounds. Aromatics such as naphthalenes and phenanthrenes were still
present in the marsh sediments at concentrations above background, 6.5 years after the spills (Teal et
al. 1978). The oil was sorbed to the anoxic marsh sediments, although biodegradation and dissipation
resulted in the decrease of *n*- and branched alkanes from the surface sediment within 4 years. Even
8 years after the spill, some sediments contained over 1,200 ppm petroleum hydrocarbons, with
naphthalene and heavier aromatics expected to persist for many more years (Burns and Teal 1979).
Twenty years after the spill, some sediments contained trace amounts of biodegraded fuel oil (Teal et
al. 1992).

Petroleum residues were measured in the northern Arabian Sea to assess the contamination following
the oil spills resulting from the Gulf War in 1991. Little change in variables related to oil pollution
took place in any compartment of the marine environment-water, plankton, fish, and sediments
(Sengupta et al. 1993).

Groundwater contamination by fuel oil no. 2 and gasoline was detected in wells of a residential area of
Rhode Island. While the total hydrocarbon content of the groundwater samples taken at one particular
well decreased from 2,350 to 1,580 μg/L over a 19-month period and the percentage of hydrocarbons
associated with gasoline also decreased from 58% to 22%, the percentage of hydrocarbons attributable
to fuel oil no. 2 increased from 42% to 78% These data indicate that the gasoline hydrocarbons are
more readily removed compared with the higher molecular weight hydrocarbons from fuel oils (Zheng
and Quinn 1988).

5.3.2.3 Soil

Microbial degradation in soils is greatest for the aromatic fractions of fuel oils, while the
biodegradation of the aliphatic hydrocarbons decreases with increasing carbon chain length.
Evaporation is the primary fate process for these aliphatics (Air Force 1989).

A single application of approximately 21, 14, or 13 g/kg soil of home heating oil no. 2 to outdoor
plots in Pennsylvania (silt loam), Oklahoma (sandy loam), and Texas (clay loam) was degraded by
86%, 90%, and 86%, respectively, after 1 year, with degradation being independent of temperature
differences. Very little oil was present in runoff and leachate water from the sites; however, analysis
of ether-extractable compounds in the leachate at the plots suggested that incomplete degradation of

some individual components in the oils was taking place. Of the six oils tested, no. 2 home heating oil resulted in the largest increase in the number of hydrocarbon-utilizing microorganisms in the plots, and it was the most lethal to soil nematodes (Raymond et al. 1975, 1976). The degradation of kerosene in soil was further studied when a pipeline ruptured and showered a wheat field with kerosene. After 6 months, the kerosene concentration began to decrease in the upper 30 cm of soil (with C13-Cl7 *n*-alkanes disappearing more rapidly compared with C10-C12 *n*-alkanes) and at 21 months was reduced to trace amounts; however, kerosene was still detected at soil depths of 30-45 +cm. The authors interpreted this as indicating reduced aerobic biodegradation at this depth, especially since the compounds disappeared in the order of their preferential microbial utilization. Seed germination studies using the contaminated soil 1 year after the spill (0.34% kerosene concentrations) showed that kerosene delayed seed germination but that the percent germination was unaffected (Dibble and Bartha 1979). Landfarming techniques (tillage of soil using agricultural implements), developed in the Netherlands to enhance biodegradation of contaminants, demonstrated that after one growing season, kerosene (initial concentration of l,000-10,000 mg/kg dry matter) was significantly degraded (final concentration of 500 mg/kg dry matter) in 40 cm of soil (Soczo and Staps 1988).

Application of diesel oil or fuel oil (type unspecified) to soil at 1% or 10% showed that, based on carbon dioxide evolution, degradation did occur. After 12 weeks, the applications of 1% diesel oil and fuel oil were degraded by 45% and 23%, respectively, whereas the 10% applications showed that only 10% of each oil was degraded in this time. Carbon dioxide evolution did not increase with increasing time, indicating that microbial populations were not increasing (Flowers et al. 1984). Addition of nitrogen (as urea) to the soil increases the biodegradation potential of diesel oil and kerosene; however, both oils were found to inhibit the urease activity of soil microbes by up to 47% and 35%, respectively, suggesting that sources of nitrogen other than urea should be used (Frankenberger 1988).

The bacterial species in the genera *Achromobacter, Pseudomonas,* and *Alcaligenes*, isolated from the soil of an active oil field in Louisiana, were able to aerobically degrade kerosene as determined by oxygen uptake (Cooper and Hedrick 1976). Soil *Pseudomonas* were able to degrade kerosene to a greater extent than were *Enterobacter* with stationary phases occurring after 10 days and 8 days, respectively (Butt et al. 1988). Seven years after the dumping of sludge containing kerosene at two sites, vegetation at each site showed little recovery. Although the bacterial biomass had declined at

both sites, microbial activity, as determined by carbon dioxide evolution, was greater at the site that had received more precipitation and had the more aerated soil (Jones 1977).

Oxidation of kerosene (fuel oil no. 1) and diesel fuel (fuel oil no. 2) by soil microbes, as determined by dehydrogenase activity, increased with increasing loading rates for both fuel oils (up to 60% w/w oil/dry soil) for up to 7 days of incubation but decreased thereafter. Dehydrogenase activity in soil treated with diesel fuel was almost twice that of soils treated with kerosene (56 and 32 pg formazan/g soil/24 hours, respectively) (Frankenberger and Johanson 1982).

Biodegradation of fuel oils in sediments is inhibited under anaerobic conditions (Bartha and Atlas 1977). Under anaerobic conditions, some soil microorganisms are capable of nitrate reduction using fuel oils as the carbon source, although nitrite may be an unwanted by-product. However, the addition of a small amount of oxygen (0.2 volume percent oxygen) to the medium can accelerate the degradation of the oil without the concomitant production of nitrite (Riss and Schweisfurth 1987). Thirteen months after the spill in 1988 of 230,000 gallons of Bunker C fuel oil off the Washington coast, only trace levels of oil were found in surface sediments (Strand et al. 1992).

5.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

5.4.1 Air

No data were located on concentrations of fuel oils in workplace or ambient air.

5.4.2 Water

Analysis of drinking water from Cincinnati, Ohio, in February of 1980, showed the presence of numerous hydrocarbons associated with petroleum products at concentrations ranging from 5 ng/L, for naphthalene to 843 ng/L for benzene (Coleman et al. 1984).

Kerosene was detected at monitoring wells (concentrations were not reported) located at the perimeter of a spent nuclear fuel processing plant in western New York State in 1983. The kerosene had been used as an extractant during plant operations from 1966 to 1972 (DOE 1989c).

Groundwater samples taken from monitoring wells at gasoline stations undergoing remediation in Florida contained both kerosene and fuel oil at unspecified concentrations (Thomas and Delfino 1991a).

Fuel oil no. 2 was detected along with gasoline in groundwater wells in Tiverton, Rhode Island. Over a 19-month period, total hydrocarbon concentrations in the water from one well decreased from 2,350 to 1,580 μg/L during which time the proportion of hydrocarbons associated with fuel oil increased from 42% (987 μg/L) to 78% (1,232 μg/L), probably as a result of the more rapid degradation of the gasoline (Zheng and Quinn 1988).

Kerosene was detected in a whole water sample from monitoring wells for municipal intakes in California in the μg/L range (STORET 1992).

Background concentrations of total hydrocarbons in Narragansett Bay, Rhode Island, ranged from 0.7 to 4.0 μg/L (Gearing and Gearing 1982a).

5.4.3 Soil

No data were located on levels of fuel oils detected in soils.

5.4.4 Other Environmental Media

Shellfish taken from unpolluted waters have been found to contain between 1 and 12 μg/g wet weight of total hydrocarbons while fish have been found to contain between 4 and 14 μg/g total hydrocarbons (steam distillables) (Connell and Miller 1980). Following a spill of fuel oil no. 2 in the Cape Cod Canal in Massachusetts, edible mussels (*Mytilus edulis*) contained average concentrations of various hydrocarbons up to 4.69 μg/g dry weight on day 1 of the spill; background hydrocarbon levels in the controls did not exceed 0.29 μg/g (Farrington et al. 1982a). Limpets in close proximity to onshore accumulations of hydrocarbon contaminants caused by diesel fuel spillage and leakage related to ship and boating activities in Arthur Harbor on the Antarctic Peninsula have incorporated PAHs into their tissues (Kennicutt et al. 1992b). However, 2 years after the release of 150,000 gallons of diesel fuel in the harbor, little spill-related contamination could be detected in intertidal limpets (Kennicutt and Sweet 1992).

No data were located that discussed concentrations of fuel oils in other environmental media such as food or terrestrial plants and animals.

5.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

The National Occupational Exposure Survey, conducted by NIOSH between 1980 and 1983, estimated that 96,345 employees (including 590 females) were exposed to fuel oil no. 2, 1,526 employees were exposed to fuel oil no. 4 and 1,076,518 employees were exposed to kerosene in the workplace (NOES 1992). Most exposure to fuel oil no. 2 was in the electric, gas, and sanitary services industries; exposure to fuel oil no. 4 was greatest in the primary metal industries, and exposure to kerosene was greatest for machinists.

Exposure of the general population to fuel oils is most likely to occur as a result of the use of kerosene and other fuel oils in heaters, furnaces, and combustion engines. Spills of number 2 fuel oil in residential basements can pose a significant health risk; a spill of 21 gallons would present a risk for 8 days or longer (Kaplan et al. 1993). Unintentional exposure to fuel oils may occur as a result of groundwater contamination from spilled fuel oils or contact with soils that have been contaminated with fuel oils.

5.6 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

The extent of potential high exposures to various segments of the population appears to be largely unknown. Workers in the petroleum industry may receive intermittent skin exposure to kerosene and other fuel oils during the refining process. Exposure is most likely to occur during the distillation of crude oil, when monitoring and servicing of equipment are carried out, or when sampling must be done (Runion 1988).

The use of kerosene as a solvent in paints and insecticides increases the likelihood of exposure by painters, particularly when spray applicators are used (Fidler et al. 1987), and in exterminators. Use of a respirator, alternate application methods (brush or roller), and increased ventilation can all reduce worker exposure to the solvent vapor.

Workers in a ball-bearing manufacturing plant in Taiwan had severe dermatitis as a result of exposure to kerosene that was used as a degreasing agent. Eczema and erythema were also prevalent among the workers (Jee et al. 1985).

5.7 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of fuel oils is available. Where adequate information is not available, ATSDR, in conjunction with NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of fuel oils.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that, if met, would reduce or eliminate the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda may be proposed.

5.7.1 Identification of Data Needs

Physical and Chemical Properties. The physical and chemical properties of some fuel oils and their primary component chemicals, specifically kerosene and fuel oil no. 2, are well defined and can be used to estimate the fate of these fuel oils following release to the environment (Air Force 1989; IARC 1989). However, the physical and chemical properties of other fuel oils such as no. l-D, no. 2-D, and no. 4, are not well defined, and data should be gathered in order to estimate the fate of these oils in the environment. Data needs associated with specific compounds that are components of fuel oils (e.g., benzene, toluene, xylene, and PAHs) are presented in the ATSDR toxicological profiles for these chemicals (ATSDR 1989, 1990a, 1991a, 1991b).

Production, Import/Export, Use, and Release and Disposal. Fuel oils are used primarily as heating oils and in engines (Air Force 1989). Most releases of fuel oils are the result of spills either on land or water (EPA 1981; Strayer et al. 1983). Few data are available on current production volumes for specific fuel oils such as fuel oil no. 4. Further information on the production volumes for each fuel oil, environmental releases, and disposal of fuel oils would aid in assessing the potential for human exposure as a result of accidental or intentional release.

Environmental Fate. The environmental fate of fuel oils is based on the environmental partitioning of the major hydrocarbon fractions. For aliphatic hydrocarbons, volatilization of lower molecular weight alkanes and sorption to organic matter for larger aliphatics, followed by photooxidation and biodegradation, respectively, are the primary degradation processes (Gearing et al. 1980; Oviatt et al. 1982). Aromatic components are most susceptible to biodegradation in warm water or soil, although some volatilization may occur in colder waters (Walker et al. 1976; Gearing and Gearing 1982a). Aromatics, however, are also water soluble and therefore are the most likely fuel oil components to leach through soil into groundwater (Strayer et al. 1983). Aliphatics that sediment out of the water column may persist for over a year (Oviatt et al. 1982). Photooxidation may also be a significant degradation process for fuel oils in surface waters (Larson et al. 1977, 1979). The movement and persistence of fuel oils in water and soils is well studied. Further data on the atmospheric reactions of fuel oils would be helpful in determining the transport of fuel oils through air.

Bioavailability from Environmental Media. The extent of absorption of fuel oils by inhalation, oral, and/or dermal routes is unknown. However, toxicity data are available for humans exposed to various fuel oils by each of these routes (Porter 1990; Reidenberg et al. 1964; Subcommittee on Accidental Poisoning 1962). These data indicate that absorption does occur. The extent of absorption by these routes depends on the volatility, solubility, lipophilicity, and other properties of the specific fuel oil components. Several of these component compounds have been discussed in their individual ATSDR toxicological profiles (e.g., benzene, toluene, xylene, PAHs, and lead), which should be consulted for further information (ATSDR 1989, 1990a, 1990b, 1991a, 1991b). More data linking exposure levels of fuel oils with biological levels of component chemicals would be useful in determining which chemicals in the mixture are most likely to be absorbed and by which routes. This information would aid in determining daily human exposure levels and more accurately assessing the risk associated with exposure to fuel oils.

Food Chain Bioaccumulation. Studies of the accidental and intentional release of fuel oils to the aquatic environment indicate that aquatic organisms are able to bioaccumulate some hydrocarbon fractions, particularly PAHs (Farrington et al. 1982b); however, depuration does occur if the source of the contamination is removed (Cox et al. 1975; Williams et al. 1989). Further studies are needed to determine the biomagnification potential of the hydrocarbon components of fuel oils, particularly PAHs, up the food chain within aquatic and terrestrial ecosystems. Specific research needs are presented in the individual ATSDR toxicological profiles on specific hydrocarbon components such as benzene, toluene, total xylenes, and polycyclic aromatic hydrocarbons (ATSDR 1989, 1990a, 1991a, 1991b). Research on the biomagnification of fuel oils as actual mixtures would not be useful because they are not available to the food chain as mixtures.

Exposure Levels in Environmental Media. There is limited information available on the levels of fuel oils found in soil or water where fuel oils are used or stored. Most monitoring studies have been conducted in the aquatic environment following an accidental spill (EPA 1981; Teal et al. 1978). More data on levels of fuel oils or their components in the air, water, and soil around facilities where fuel oils are produced, stored, and used would be useful. Data on levels in contaminated surface water, groundwater, and soil are needed to assess the potential risk from these likely sources of exposure.

Exposure Levels in Humans. Workers who use fuel oils in manufacturing and those involved in their transfer may experience increased dermal and inhalation exposures. Workers in the petroleum refining industry, particularly those involved with monitoring and servicing unit equipment, including fuel oil storage tanks, are known to have increased exposure to fuel oils such as kerosene (Runion 1988). Further information is needed to assess the approximate levels of exposure for these populations.

Exposure Registries. No exposure registries for fuel oils were located. These substances are not currently compounds for which a subregistry has been established in the National Exposure Registry. These substances will be considered in the future when chemical selection is made for subregistries to be established. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to the exposure to these substances.

A registry does exist for benzene, a component of fuel oils. More information on the benzene exposure registry can be found in the ATSDR toxicological profile for benzene (ATSDR 1989).

5.7.2 On-going Studies

No on-going studies on the exposure or environmental fate of fuel oils were located.

6. ANALYTICAL METHODS

The purpose of this chapter is to describe the analytical methods that are available for detecting and/or measuring and monitoring fuel oils in environmental media and in biological samples. The intention is not to provide an exhaustive list of analytical methods that could be used to detect and quantify fuel oils. Rather, the intent is to identify well-established methods that are used as the standard methods of analysis. Many of the analytical methods used to detect fuel oils in environmental samples are the methods approved by federal organizations such as EPA and the National Institute for Occupational Safety and Health (NIOSH). Other methods presented in this chapter are those that are approved by groups such as the Association of Official Analytical Chemists (AOAC) and the American Public Health Association (APHA). Additionally, analytical methods are included that refine previously used methods to obtain lower detection limits, and/or to improve accuracy and precision.

6.1 BIOLOGICAL MATERIALS

Fuel oils are mixtures of aliphatic and aromatic hydrocarbons (Air Force 1989). Most analytical methods for detecting fuel oils in biological media focus on the detection of kerosene components, as this is a commonly used fuel for residential heaters. Analytical methods for detecting other fuel oils in biological media were not located. See Table 6-l for a summary of the analytical methods most commonly used to measure kerosene in biological materials. For more analytical methods information, see the previous profiles on some of the individual components of fuel oils (e.g., benzene, toluene, total xylenes, and PAHs) (ATSDR 1989, 1990a, 1991a, 1991b).

The primary method for detecting kerosene in biological materials such as blood is gas chromatography (GC). GC may be combined with mass spectroscopy (MS) for peak identification with the gas chromatograph in the electron impact mode (Kimura et al. 1988, 1991). Quantification methods include the use of mass fragmentography (Kimura et al. 1988). Hydrocarbon components of kerosene are determined based on analysis of headspace gas above the sample (Kimura et al. 1991). This method is useful for distinguishing between kerosene intoxication and gasoline intoxication as kerosene gives a high toluene peak and has a pseudocumene-to-toluene ratio only half that of gasoline. Capillary columns are used, with either Porapak, Chromosorb®, or Chemipak®, giving acceptable results (Kimura et al. 1988). The percent recoveries of these methods were not provided. Wide-bore

TABLE 6-1. Analytical Methods for Determining Fuel Oils in Biological Materials

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Blood	Add internal standard; extract with *n*-pentane; centrifuge; freeze; decant solvent; concentrate; inject to GC	GC/MS	50 pg	NR	Kimura et al. 1988
Blood	Mix sample with internal standard; add salt solution; equilibrate; aspirate headspace vapor and inject to GC	GC/MS	50 pg (toluene)	NR	Kimura et al. 1991
Stomach contents, blood, urine	Extract sample with ethyl acetate; condense; inject to GC	GC/FID/MS	0.2 μg/mL	93–100	Hara et al. 1988

FID = flame ionization detector; GC = gas chromatography; MS = mass spectrometry; NR = not reported

capillary columns have also been used (Hara et al. 1988) for GC/MS analysis combined with flame ionization detectors (FID). This method determined levels of *m*- and *o*-xylene (components of kerosene) in the blood, urine, and stomach contents. The sensitivity and precision of this method was generally good (93-100% recovery).

No analytical methods studies were located for detecting fuel oils in biological samples other than blood, urine, or stomach contents.

6.2 ENVIRONMENTAL SAMPLES

Because fuel oils are composed of a mixture of hydrocarbons, there are few methods for the environmental analysis of fuel oils as a whole, but methods are reported for the analysis of their component hydrocarbons. The methods most commonly used to detect the major hydrocarbon components of fuel oils in environmental samples are GC/FID and GC/MS. See Table 6-2 for a summary of the analytical methods used to determine fuel oils in environmental samples. Several of the components of fuel oils have been discussed in detail in their individual toxicological profiles (e.g., benzene, toluene, total xylenes, and PAHs), which should be consulted for more information on analytical methods (ATSDR 1989, 1990a, 1991a, 1991b).

GC is the most commonly used method to selectively detect, identify, and quantify the volatile hydrocarbon components of fuel oils in air (Andrasko 1983; Baldwin 1977; NIOSH 1994). Air samples may be collected on adsorbent tubes such as charcoal, Florisil[®], Tenax[®], Porapak[®], or Chromosorb[®]. Active carbons wires have also been used (Andrasko 1983). The hydrocarbons are extracted from the tubes by thermal desorption or with a liquid solvent such as carbon disulfide and analyzed on the gas chromatograph. Precision is good (relative standard deviation = 0.052) using the charcoal tubes (NIOSH 1994); recovery data were not reported for the other types of adsorption tubes, although desorption from the active carbon wires ranged between 90% and 99% recovery, with a detection limit in the ppb range. A Tenax-TA[®] sorbent trap has been used with subsequent thermal desorption (Andrasko 1983). Combining sample concentration with the headspace method allows for sampling of smaller air volumes and for other environmental samples, such as kerosene combustion debris, that have undergone significant evaporation; the headspace method requires concentrating the sample prior to analysis (Andrasko 1983; Baldwin 1977). An exploratory study indicates that, with

TABLE 6-2. Analytical Methods for Determining Fuel Oils in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Air	Adsorb to solid sorbent tube (e.g., charcoal); desorb in CS_2; equilibrate; inject aliquot to GC	GC/FID	0.1 mg/ 5–10 mL sample	96–106	NIOSH 1994
Air	Adsorb to quartz filter	Evolved Gas Analysis	10 μg	NR	Daisey and Gundel 1991
Air	Adsorb to Florisil filter; elute with CS_2; evaporate under vacuum	GC	NR	NR	Baldwin 1977
Water	Strip sample in sparger with helium; adsorb effluent gas to adsorption tube; thermally desorb to GC	GC/FID/MS	10 μg/L	89.7–95.7	Bianchi et al. 1991
Water	Acidify sample; extract with hexane; dry solvent phase; inject to GC	GC/FID	0.25μL/L	NR	Dell'Acqua and Bush 1973
Water (purgeable aromatics)	Purge sample with inert gas; adsorb vapor in trap; heat trap; backflush to GC	GC/PID	0.2 μg/L	92–96	EPA 1991c

TABLE 6-2. Analytical Methods for Determining Fuel Oils in Environmental Samples (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Water	Purge sample with helium; collect vapor on adsorption tube; thermally desorb; concentrate; backflush to GC	GC/FID	10 µg/L	99–114 (fuel oil no. 2); 91–112 (kerosene)	Belkin and Esposito 1986
Water	Purge sample with ambient air, adsorb to charcoal filter; extract filter with CS_2; inject to GC	GC/MS	5 ng/L	0.4–89 (75% average)	Coleman et al. 1981
Water	Extract aqueous sample with pentane; equilibrate; inject to GC	GC/MS	NR	NR	Coleman et al. 1984
Water (base/ neutral and acids)	Adjust sample pH to >11; extract sample with CH_2Cl_2 solvent; adjust pH to <2; reextract; dry; concentrate; inject to GC	GC/MS	1.5–7.8 µg/L (varies with actual compound)	NR	EPA 1991c
Seawater	Extract aqueous phase of sample with pentane; evaporate; inject to GC	GC/MS	NR	NR	Boylan and Tripp 1971
Soil (other solid materials)	Extract sample with CCl_4; inject extract	GLC	NR	NR	Midkiff and Washington 1972

TABLE 6-2. Analytical Methods for Determining Fuel Oils in Environmental Samples (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Soil	Extract sample with CCl₄; centrifuge; remove water and humic materials with Na₂SO₄ and Al₂O₃; inject extract	GC/FID	NR	NR	Galin et al. 1990a
Soil	Purge at elevated temperatures; heat trap to desorb material into GC column	GC	NR	NR	Chang et al. 1990
Soil	Sample added to substrate and placed in cup mounted on optical fiber	Fluorescence Spectroscopy	NR	NR	Apitz et al. 1993
Soil	Sample extracted using water and cyclohexane	Synchronous Scanning Fluorescence Spectroscopy	NR	NR	Pharr et al. 1992
Sediment	Add internal sample to sample; extract with KOH in methanol; partition into petroleum ether; concentrate; purify and isolate hydrocarbon fractions using TLC or column chromatography	GLC/FID	NR	NR	Gearing et al. 1980
Shellfish tissue (mussel)	Extract with NaOH; isolate fractions with column chromatography; inject fractions to GC	GC or GC/MS	NR	34–120 (GC); 36–87 (GC/MS)	Farrington et al. 1982a

TABLE 6-2. Analytical Methods for Determining Fuel Oils in Environmental Samples (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Shellfish tissue	Extract with methanol then pentane; dry; concentrate; inject to GC	GC	NR	NR	Blumer et al. 1970

Al_2O_3 = aluminum oxide; CCl_4 = carbon tetrachloride; CH_2Cl_2 = dichloromethane (methylene chloride); CS_2 = carbon disulfide; FID = flame ionization detector; GC = gas chromatography; GLC = gas liquid chromatography; KOH = potassium hydroxide; MS = mass spectrometry; Na_2SO_4 = sodium sulfate; NaOH = sodium hydroxide; NR = not reported; PID = photoionization detector; TLC = thin-layer chromatography

additional research, the microanalytical evolved gas analysis technique (EGA) can be further developed
to measure kerosene soot in indoor aerosols (Daisey and Gundel 1991).

GC/FID and GC/MS have been used to measure the water-soluble components of fuel oils, particularly
kerosene, in industrial effluents and estuarine water (Bianchi et al. 1991), sea water (Boyland and
Tripp 1971), drinking water (Coleman et al. 1984; Dell'Acqua and Bush 1973), and groundwater
(Thomas and Delfino 1991a). Purge-and-trap sample preparation methods have been used to
determine purgeable (volatile) aromatic components of fuel oils. This method requires a trap with a
Tenax®/Chromosorb® absorbent and the use of a gas chromatograph with a photoionization detector
(PID) (EPA 1991c), an ion trap detector (ITD), or FID (Thomas and Delfino 1991a). A modification
of the purge-and-trap method uses ambient temperatures, has the advantage of being applicable to a
variety of waters, requires virtually no sample preparation (no solvents are required), and has an
analysis time of approximately 30 minutes (Bianchi et al. 1991). While this method may be used for
determining the presence of petroleum contaminants in water, it cannot distinguish between various
sources of this contamination, e.g., between gasoline, kerosene, and diesel oil.

Distinctions between water-soluble fractions of mixed hydrocarbons may be made by using solvent
extraction of the water-soluble base/neutral and acid fractions with methylene chloride (EPA 1991c;
Thomas and Delfino 1991a). This separation of base/neutral and acid fractions will permit the GC
resolution of the type of water soluble hydrocarbons present in the aqueous phase. Hexane has also
been used as a solvent (Dell'Acqua and Bush 1973), as has pentane (Coleman et al. 1984).

A dynamic thermal stripper has also been used to detect low levels (ppb range) of fuel oil no. 2 and
kerosene present in water samples (Belkin and Esposito 1986). This method traps the fuels on an
adsorption tube using helium gas for purging. The fuel is then thermally desorbed and backflushed to
a gas chromatograph with FID. This method also does not require any solvent and needs only a
15 mL sample. Recovery for this method is good (91-114%) with precision ranging from 6.4 to
14.3% relative standard deviation. A modified Grob closed-loop-stripping method, which uses a wall-coated
open tubular glass capillary column combined with GC/MS, has been used to extract and
quantify low levels (ppt) of hydrocarbons in water samples. The method continually recirculates an
ambient air stream through the 3.8-liter water sample for approximately 2 hours and collects the vapor
on an activated carbon filter, followed by extraction with carbon disulfide and analysis (Coleman et al.
1981). An optical fiber fluorescence spectroscopy system has been used for real-time *in situ*

measurements of low levels (at ppb of diesel fuel marine equivalent) of petroleum hydrocarbons in seawater, showing temporal and spatial variability (Lieberman et al 1993).

A qualitative method for determining diesel oil in water has been proposed that is based on changes in the internal reflection of an optical fiber coated with an organophilic compound caused by the presence of hydrocarbons. The method does not require any sample preparation but is limited to relatively high concentrations of contaminants, e.g., 17 mg/L for diesel oil (Kawahara et al. 1983). An alternative method uses a Fourier transform infrared spectrometer (FTIR). This method has the advantage of no sample preparation, a short analysis time (20-30 seconds), and good accuracy (±20%). A detection limit of 0.5 ppb has been determined for a l-liter sample of sea water; 10 mL is sufficient if a detection limit of 0.05 ppm is acceptable. The FTIR may be coupled with a GC or liquid chromatography for the analysis of complex mixtures (Mille et al. 1985).

GC/FID (Galin et al. 1990a), gas liquid chromatography (GLC) with FID (Midkiff and Washington 1972), and elevated temperature purge and trap with GC (Chang et al. 1990) have been used to measure fuel oils in soils. An enzyme immunoassay has been developed using a monoclonal antibody reagent that detects gasoline and diesel fuel in soil; commercialization of this assay will offer significant advantages over current testing methods of gasoline and fuel contamination levels in soil (Allen et al. 1992b). GLC has also been used to determine fuel oils in marine sediments (Gearing et al. 1980) and other environmental samples such as paper, cloth, and wood (Midkiff and Washington 1972). Extraction is used to concentrate the sample because fuel oils do not provide sufficient vapors to allow the use of a headspace sampling method. Carbon tetrachloride is the recommended solvent as it causes less interference with the chromatographic peaks of the fuel oils (Galin et al. 1990a; Midkiff and Washington 1972). Quantification of fuel oil hydrocarbons from sediments is a more elaborate process. Following extraction, the saturated and olefinic hydrocarbon fraction is separated from the aromatic hydrocarbon fraction using thin-layer chromatography or column chromatography. Fractions are subsequently analyzed by GLC (Gearing et al. 1980). Recovery, sensitivity, and levels of detection data were not reported. Quantification of oils and grease, by gross weight only, in soils and sludges may be accomplished by extraction with a Soxhlet apparatus using either trichlorotrifluoroethane (APHA 1985) or methylene chloride (Martin et al. 1991) as the solvent, although this method may not be used to identify the specific type of oil or grease present in the soil sample. Synchronous scanning fluorescence spectroscopy can be used to identify kerosene, fuel oil number 2, fuel oil number 5, and other aromatic-containing products in groundwater and soil samples. This analytical method is more

efficient than chromatographic methods, and its spectra are easier to interpret for identification purposes (Pharr et al. 1992). Fluorescence spectroscopy has been used for *in situ* detection of petroleum hydrocarbon plumes in soil; this technique allows for measurements in soils before monitoring wells are drilled and is thus independent of the fractionation and transport problems inherent when sampling well fluids (Apitz et al. 1992).

The age of diesel oil in the subsurface soil environment can be determined by utilizing the fact that the composition of the diesel oil (the ratio between *n*-alkanes and isoprenoids) changes due to biodegradation. In one study, the ratio of C_{17} to pristane was highly correlated with the residence time of diesel fuel at 12 test locations (Christensen et al 1993).

A set of neural networks has been trained to identify seven classes of petroleum hydrocarbon based fuels from their fluorescence emission spectra; this technique correctly identified at least 90% of the test spectra (Andrews and Lieberman 1994).

High-performance liquid chromatography (HPLC), followed by GC/MS, has been used to fractionate and then quantitate the aliphatic and aromatic hydrocarbons present in liquid fuel precursors in order to determine the fuel potential of the compounds. Kerosene had the advantage of not requiring any sample preparation. Other light fuel oils may require the use of methylene chloride as a solvent prior to HPLC analysis (Lamey et al. 1991). The sensitivity, precision, and recovery of this method were not reported. An alternative method for fractionating and purifying petroleum hydrocarbons prior to GC or HPLC separation has been developed (Theobald 1988). The method uses small, prepacked, silica or C_{18} columns that offer the advantage of rapid separation (approximately I5 minutes for a run); good recovery of hydrocarbons (85% for the C_{18} column and 92% for the silica column); reusability of the columns; and for the silica column in particular, good separation of hydrocarbon from nonhydrocarbon matrices as may occur with environmental samples. Infrared analysis and ultraviolet spectroscopy were used to analyze the aromatic content in diesel fuels; these methods are relatively inexpensive and faster than other available methods, such as mass spectrometry, supercritical fluid chromatography, and nuclear magnetic resonance (Bailey and Kohl 1991).

Due to the tendency of hydrocarbons in the soil to undergo subsurface oxidation, measuring CO_2 levels in the soil gas could be used as a cost-effective field screening tool. In one soil-gas survey, CO_2 levels in soil gas correlated well with petroleum hydrocarbons in the soil (Diem et al. 1988).

A two-dimensional supercritical fluid chromatography (SFC) system has been developed for the determination of saturates, alkenes, and mono-, di-, and tri-aromatics in diesel fuel. This technique results in a short analysis time (less than 8 minutes) and good relative standard deviations at low alkene content (Andersson et al. 1992).

The principal method for detecting kerosene or its components in biota is GC (Blamer et al. 1970; Farrington et al. 1982a; Newton et al. 1991). Aliphatic and aromatic hydrocarbon components of fuel oils taken up by shellfish (whole mussels without shells) were isolated by column chromatography following extraction. Both the alka.ne/cycloalkane and alkene/aromatic fractions were analyzed by GC with recoveries in the range of 67-100% for alkanes and 71-78% for some aromatics; these aromatics were also analyzed using GC/MS with recoveries between 49% and 74% (Farrington et al. 1982a). Determination of hydrocarbons may also be accomplished by fractionating the hydrocarbon components. Extraction of hydrocarbons from contaminated shellfish may be accomplished using Soxhlet extraction with methanol followed by reextraction with pentane. The extracts are then dried and concentrated prior to injection into the GC (Blumer et al. 1970). Other data on detection limits and precision were not provided.

6.3 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of fuel oils is available. Where adequate information is not available, ATSDR, in conjunction with NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of fuel oils.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that, if met, would reduce or eliminate the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda may be proposed.

6.3.1 Identification of Data Needs

Methods for Determining Biomarkers of Exposure and Effect. No biomarkers of exposure
were identified for fuel oils because, while standard procedures exist for identifying or quantifying
exposure to fuel oils based on hydrocarbon components in blood, urine, and stomach contents (Hara et
al. 1988; Kimura et al. 1988, 1991), none of these are applicable solely to fuel oils. These methods
are sensitive enough to measure the levels at which health effects occur and may be adequate for
determining background levels in the population; however, they cannot distinguish between exposure
to different fuel oils or other types of hydrocarbon mixtures. Analytical methods are needed for
measuring the hydrocarbon components of fuel oils in lungs. Biomonitoring studies are needed to
adequately assess exposure to fuel oils.

No biomarkers of effects were identified for fuel oils because the effects associated with exposure to
fuel oils are not unique for them, i.e., the effects may be caused by other chemicals or hydrocarbon
mixtures. Analytical methods do exist for determining angiotensin-converting enzyme activity in the
lungs. This enzyme may be used to determine the lung damage caused by a fuel oil. Analytical
methods are needed to determine whether the tissue damage is specific to fuel oils and the target
organs.

**Methods for Determining Parent Compounds and Degradation Products in
Environmental Media.** Methods exist to detect major hydrocarbon components of fuel oils in air
(Andrasko 1983; Baldwin 1977; NIOSH 1994), water (Bianchi et al. 1991; Boyland and Tripp 1971;
Dell'Acqua and Bush 1973; EPA 1991c), sediment (Gearing et al. 1980), and soil (Galin et al. 1990a;
Midkiff and Washington 1972). The most commonly used methods are GC/FID and GC/MS. These
methods are relatively sensitive, selective, and reliable, and can be used to detect the levels of the
various components of fuel oils found in the environment and levels at which health effects occur.

6.3.2 On-going Studies

No on-going analytical methods studies were located.

7. REGULATIONS AND ADVISORIES

The international, national, and state regulations and guidelines regarding fuel oils in air, water, and other media are summarized in Table 7-l. There are only a few regulations that are specific to a particular fuel oil and these are indicated in the table.

An acute inhalation MRL of 0.02 mg/m^3 was derived for fuel oil no. 2. The MRL is based on a LOAEL value of 65 mg/m^3 for neurobehavioral effects (mild transient ataxia and CNS depression) in mice exposed to airborne concentrations of fuel oil no. 2.

An intermediate inhalation MRL of 0.01 mg/m^3 was derived for fuel oil no. 1. The MRL is based on decreased blood glucose levels in male rats exposed to airborne concentrations of kerosene averaging 58 mg/m3 for 6 hours/day, 6 days/week for 14 weeks. Rats exposed to airborne concentrations of kerosene averaging 231 mg/m^3 showed a decrease in blood glucose titers, as well as increased circulating levels of lactate and pyruvate.

The EPA has no oral reference dose (RfD) or inhalation reference concentration (RfC) for any of the fuel oils.

Under the Hazardous Materials Transportation Act, fuel oils are designated as hazardous substances subject to special requirements for packaging, labeling, and transportation (DOT 1989a, 1989b).

7. REGULATIONS AND ADVISORIES

TABLE 7-1. Regulations and Guidelines Applicable to Fuel Oils

Agency	Description	Information	References
INTERNATIONAL			
IARC	Carcinogenic classification for occupational exposures in petroleum refining	Group 2A[a]	IARC 1989
	Carcinogenic classification marine diesel fuel	Group 2B[b]	
	residual (heavy) fuel oils	Group 2B	
	jet fuels	Group 3[c]	
	distillate (light) diesel fuels	Group 3	
NATIONAL			
Regulations:			
a. Air:			
AFOSH	PEL TWA		Air Force 1989
	Petroleum distillates (naphtha)	400 ppm	
	STEL (15 minutes)		
	Petroleum distillates (naphtha)	500 ppm	
NIOSH	TWA		
	Petroleum distillates (naphtha)	85 ppm (350 mg/m^3)	NIOSH 1992
	Ceiling REL (15 minutes)		
	Petroleum distillates (naphtha)	438 ppm (1,800 mg/m^3)	
	IDLH		
	Petroleum distillates (naphtha)	10,000 ppm	NIOSH 1990
OSHA	PEL TWA		OSHA 1989a (29 CFR
	Petroleum distillates (naphtha)	400 ppm	1910.1000); OSHA 1989b
b. Other:			
DOT	Hazardous Material Transportation Act: Fuel oils are designated as hazardous materials which are subject to requirements for packaging, shipping, and transporting	Yes	DOT 1989a (49 CFR 172.101 Appendix A); DOT 1989b
EPA	Toxic Substances Control Act: Manufacturers and processors of the C$_9$ aromatic hydrocarbon fraction must test this fraction for the following: neurotoxicity, mutagenicity, developmental toxicity, reproductive effects, and oncogenicity	Yes	EPA 1991a (40 CFR 799.2175); EPA 1987c
EPA	Marine Protection Research and Sanctuaries Act: Ocean dumping of organohalogen compounds or oils of any kind which are known or suspected carcinogens, mutagens, or teratogens is prohibited except when they are present as trace contaminants	Yes	EPA 1991b (40 CFR 227.6)
Guidelines:			
a. Other:			
EPA	Domestic water supply must be virtually free from oil and grease, particularly from the tastes and odors that emanate from petroleum products	Yes	EPA 1986c
	For aquatic life, levels must be ≤ 0.01 of the lowest continuous flow 96-h LC$_{50}$	Yes	EPA 1986c

7. REGULATIONS AND ADVISORIES

TABLE 7-1. Regulations and Guidelines Applicable to Fuel Oils (*continued*)

Agency	Description	Information	References
NATIONAL (Cont.)			
	Clean Water Act: Oil and grease are designated as conventional pollutants. Effluent limitations for oil and grease (polynuclear aromatic hydrocarbons) exist for almost all point sources under the general pretreatment standards for new and existing sources	Yes	EPA 1988b (40 CFR 403.2); EPA 1988c
	Pesticide subject to registration and/or reregistration (kerosene)	Yes	EPA 1989c (40 CFR 152.146); EPA 1989b
STATE			
Regulations and Guidelines:			
a. Air:			
	Acceptable ambient air concentrations (Navy Fuels JP-5)		NATICH 1991
Connecticut	(8 hour)	2.00×10^3 µg/m^3	
Maryland		0.00	
Oklahoma	(24 hour)	1.00×10^4 µg/m^3	
Texas	(30 minutes)	1.00×10^3 µg/m^3	
Texas	(annual)	1.00×10^2 µg/m^3	
	Regulations on hydrocarbon emissions (kerosene, petroleum distillates, diesel, fuel oil)	Yes	CELDS 1991
Connecticut			
Kansas			
Wisconsin			
	Regulations on volatile organic carbon emissions (VOC)	Yes	CELDS 1991
Alabama			
Arizona			
Florida			
Maine			
Maryland			
Michigan			
New Jersey			
South Carolina			
Virginia			
Texas			
Washington, DC			
Maine	Regulations on the open burning of fuel oils (kerosene)	Yes	CELDS 1991
Texas	Regulations for gas processing plants	Yes	CELDS 1991
b. Water:			
Alaska	Aquatic life criterion for total hydrocarbons in marine and surface waters	15 µg/L	State of Alaska 1989
	Aquatic life criterion for aromatic hydrocarbons in marine and surface waters	10 µg/L	State of Alaska 1989

7. REGULATIONS AND ADVISORIES

TABLE 7-1. Regulations and Guidelines Applicable to Fuel Oils (*continued*)

Agency	Description	Information	References
STATE (Cont.)			
Arkansas	Average or maximum allowable quantity of oil or grease discharged into surface waters	10 mg/L (average) 15 mg/L (maximum)	State of Arkansas 1991
Florida	Average or maximum allowable quantity of oil or grease discharged Class V waters (navigation, industrial use)	10 mg/L	State of Florida 1992
	Average or maximum allowable quantity of oil or grease discharged into all other surface waters	5 mg/L	State of Florida 1992
Maine	Drinking water quality guidelines and standards (fuel oil no. 2)	100 μg/L	FSTRAC 1988
Massachusetts	Maximum discharge concentration of oil or grease of petroleum origin in surface waters	15 mg/L	Commonwealth of Massachusetts 1988
Nebraska	Maximum petroleum oil concentration in surface waters	10 mg/L	State of Nebraska 1991
New York	Maximum contaminant level of kerosone in drinking water	50 μg/mL	State of New York 1989
South Dakota	Water quality standard for all petroleum products in surface waters	10 mg/L	State of South Dakota 1989
Virginia	Water quality standard for petroleum hydrocarbons in ground water	1 mg/L	Commonwealth of Virginia 1988
Wyoming	Water quality standard for all surface waters classes	10 mg/L	State of Wyoming 1990
c. Other:			
	Regulations on the transport of flammable/hazardous liquids (petroleum distillates or VOC)	Yes	CELDS 1991
Colorado Maryland Massachusetts Wisconsin			
Maine	Regulations on the disposal of special wastes including diesel fuels	Yes	CELDS 1991
California	Regulations on leaking underground fuel tanks	Yes	CELDS 1991
Wyoming	Regulations on well drilling for oil	Yes	CELDS 1991
Rhode Island	Regulations on oil pollution	Yes	CELDS 1991

[a]Group 2A = Probably carcinogenic to humans. This classification is not specified for the fuel oils discussed in this profile, nor for exposure to fuel oils by the general population; rather, this classification applies only to occupational exposures in petroleum refining.
[b]Group 2B = Possibly carcinogenic to humans
[c]Group 3 = Not classifiable as to its carcinogenicity to humans

AFOSH = Air Force Office of Health and Safety; CELDS = Computer-Environmental Legislative Data System; DOT = Department of Transportation; EPA = Environmental Protection Agency; IARC = International Agency for Research on Cancer; IDLH = Immediately Dangerous to Life or Health; NATICH = National Air Toxics Information Clearinghouse; NIOSH = National Institute for Occupational Safety and Health; OSHA = Occupational Safety and Health Administration; PEL = Permissible Exposure Limit; REL = Recommended Exposure Limit; STEL = Short Term Exposure Limit; TWA = Time-Weighted Average; VOC = Volatile Organic Compounds

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9. GLOSSARY

Acute Exposure -- Exposure to a chemical for a duration of 14 days or less, as specified in the Toxicological Profiles.

Adsorption Coefficient (K_{oc}) -- The ratio of the amount of a chemical adsorbed per unit weight of organic carbon in the soil or sediment to the concentration of the chemical in solution at equilibrium.

Adsorption Ratio (Kd) -- The amount of a chemical adsorbed by a sediment or soil (i.e., the solid phase) divided by the amount of chemical in the solution phase, which is in equilibrium with the solid phase, at a fixed solid/solution ratio. It is generally expressed in micrograms of chemical sorbed per gram of soil or sediment.

Bioconcentration Factor (BCF) -- The quotient of the concentration of a chemical in aquatic organisms at a specific time or during a discrete time period of exposure divided by the concentration in the surrounding water at the same time or during the same period.

Cancer Effect Level (CEL) -- The lowest dose of chemical in a study, or group of studies, that produces significant increases in the incidence of cancer (or tumors) between the exposed population and its appropriate control.

Carcinogen -- A chemical capable of inducing cancer.

Ceiling Value -- A concentration of a substance that should not be exceeded, even instantaneously.

Chronic Exposure -- Exposure to a chemical for 365 days or more, as specified in the Toxicological Profiles.

Ct -- A means of expressing concentration as a convenient mechanism for establishing an exposure mechanism. Specifically, it is the product of airborne concentration of aerosolized diesel fuel (mg/m3) and duration of exposure (hours).

Developmental Toxicity -- The occurrence of adverse effects on the developing organism that may result from exposure to a chemical prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism.

Embryotoxicity and Fetotoxicity -- Any toxic effect on the conceptus as a result of prenatal exposure to a chemical; the distinguishing feature between the two terms is the stage of development during which the insult occurred. The terms, as used here, include malformations and variations, altered growth, and in utero death.

EPA Health Advisory -- An estimate of acceptable drinking water levels for a chemical substance based on health effects information. A health advisory is not a legally enforceable federal standard, but serves as technical guidance to assist federal, state, and local officials.

Immediately Dangerous to Life or Health (IDLH) -- The maximum environmental concentration of a contaminant from which one could escape within 30 min without any escape-impairing symptoms or irreversible health effects.

Intermediate Exposure -- Exposure to a chemical for a duration of 15-364 days, as specified in the Toxicological Profiles.

Immunologic Toxicity -- The occurrence of adverse effects on the immune system that may result from exposure to environmental agents such as chemicals.

In vitro -- Isolated from the living organism and artificially maintained, as in a test tube.

In vivo -- Occurring within the living organism.

Lethal Concentration$_{(LO)}$ (LC$_{Lo}$) -- The lowest concentration of a chemical in air which has been reported to have caused death in humans or animals.

Lethal Concentration$_{(50)}$ (LC$_{50}$) -- A calculated concentration of a chemical in air to which exposure for a specific length of time is expected to cause death in 50% of a defined experimental animal population.

Lethal Dose$_{(Lo)}$ (LD$_{Lo}$) -- The lowest dose of a chemical introduced by a route other than inhalation that is expected to have caused death in humans or animals.

Lethal Dose$_{(50)}$ (LD$_{50}$) -- The dose of a chemical which has been calculated to cause death in 50% of a defined experimental animal population.

Lethal Time$_{(50)}$, (LT$_{50}$) -- A calculated period of time within which a specific concentration of a chemical is expected to cause death in 50% of a defined experimental animal population.

Lowest-Observed-Adverse-Effect Level (LOAEL) -- The lowest dose of chemical in a study, or group of studies, that produces statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control.

Malformations -- Permanent structural changes that may adversely affect survival, development, or function.

Minimal Risk Level -- An estimate of daily human exposure to a dose of a chemical that is likely to be without an appreciable risk of adverse noncancerous effects over a specified duration of exposure.

Mutagen -- A substance that causes mutations. A mutation is a change in the genetic material in a body cell. Mutations can lead to birth defects, miscarriages, or cancer.

Neurotoxicity -- The occurrence of adverse effects on the nervous system following exposure to chemical.

No-Observed-Adverse-Effect Level (NOAEL) -- The dose of chemical at which there were no statistically or biologically significant increases in frequency or severity of adverse effects seen

between the exposed population and its appropriate control. Effects may be produced at this dose, but they are not considered to be adverse.

Octanol-Water Partition Coefficient (K_{ow}) -- The equilibrium ratio of the concentrations of a chemical in n-octanol and water, in dilute solution.

Permissible Exposure Limit (PEL) -- An allowable exposure level in workplace air averaged over an 8-hour shift.

q_1* -- The upper-bound estimate of the low-dose slope of the dose-response curve as determined by the multistage procedure. The q_1* can be used to calculate an estimate of carcinogenic potency, the incremental excess cancer risk per unit of exposure (usually µg/L for water, mg/kg/day for food, and µg/m^3 for air).

Reference Dose (RfD) -- An estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure of the human population to a potential hazard that is likely to be without risk of deleterious effects during a lifetime. The RfD is operationally derived from the NOAEL (from animal and human studies) by a consistent application of uncertainty factors that reflect various types of data used to estimate RfDs and an additional modifying factor, which is based on a professional judgment of the entire database on the chemical. The RfDs are not applicable to nonthreshold effects such as cancer.

Reportable Quantity (RQ) -- The quantity of a hazardous substance that is considered reportable under CERCLA. Reportable quantities are (1) 1 pound or greater or (2) for selected substances, an amount established by regulation either under CERCLA or under Sect. 311 of the Clean Water Act. Quantities are measured over a 24-hour period.

Reproductive Toxicity -- The occurrence of adverse effects on the reproductive system that may result from exposure to a chemical. The toxicity may be directed to the reproductive organs and/or the related endocrine system. The manifestation of such toxicity may be noted as alterations in sexual behavior, fertility, pregnancy outcomes, or modifications in other functions that are dependent on the integrity of this system.

Short-Term Exposure Limit (STEL) -- The maximum concentration to which workers can be exposed for up to 15 min continually. No more than four excursions are allowed per day, and there must be at least 60 min between exposure periods. The daily TLV-TWA may not be exceeded.

Target Organ Toxicity -- This term covers a broad range of adverse effects on target organs or physiological systems (e.g., renal, cardiovascular) extending from those arising through a single limited exposure to those assumed over a lifetime of exposure to a chemical.

Teratogen -- A chemical that causes structural defects that affect the development of an organism.

Threshold Limit Value (TLV) -- A concentration of a substance to which most workers can be exposed without adverse effect. The TLV may be expressed as a TWA, as a STEL, or as a CL.

Time-Weighted Average (TWA) -- An allowable exposure concentration averaged over a normal 8-hour workday or 40-hour workweek.

Toxic Dose (TD$_{50}$) -- A calculated dose of a chemical, introduced by a route other than inhalation, which is expected to cause a specific toxic effect in 50% of a defined experimental animal population.

Uncertainty Factor (UF) -- A factor used in operationally deriving the RfD from experimental data. UFs are intended to account for (1) the variation in sensitivity among the members of the human population, (2) the uncertainty in extrapolating animal data to the case of human, (3) the uncertainty in extrapolating from data obtained in a study that is of less than lifetime exposure, and (4) the uncertainty in using LOAEL data rather than NOAEL data. Usually each of these factors is set equal to 10.

APPENDIX A

USER'S GUIDE

Chapter 1

Public Health Statement

This chapter of the profile is a health effects summary written in nontechnical language. Its intended audience is the general public especially people living in the vicinity of a hazardous waste site or substance release. If the Public Health Statement were removed from the rest of the document, it would still communicate to the lay public essential information about the substance.

The major headings in the Public Health Statement are useful to find specific topics of concern. The topics are written in a question and answer format. The answer to each question includes a sentence that will direct the reader to chapters in the profile that will provide more information on the given topic.

Chapter 2

Tables and Figures for Levels of Significant Exposure (LSE)

Tables (2-1, 2-2, and 2-3) and figures (2-l and 2-2) are used to summarize health effects by duration of exposure and end point and to illustrate graphically levels of exposure associated with those effects. All entries in these tables and figures represent studies that provide reliable, quantitative estimates of No-Observed-Adverse-Effect Levels (NOAELs), Lowest-Observed-Adverse-Effect Levels (LOAELs) for Less Serious and Serious health effects, or Cancer Effect Levels (CELs). In addition, these tables and figures illustrate differences in response by species, Minimal Risk Levels (MRLs) to humans for noncancer end points, and EPA's estimated range associated with an upper-bound individual lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. The LSE tables and figures can be used for a quick review of the health effects and to locate data for a specific exposure scenario. The LSE tables and figures should always be used in conjunction with the text.

The legends presented below demonstrate the application of these tables and figures. A representative example of LSE Table 2-l and Figure 2-l are shown. The numbers in the left column of the legends correspond to the numbers in the example table and figure.

LEGEND

See LSE Table 2-1

(1).   Route of Exposure One of the first considerations when reviewing the toxicity of a substance using these tables and figures should be the relevant and appropriate route of exposure. When sufficient data exist, three LSE tables and two LSE figures are presented in the document. The three LSE tables present data on the three principal routes of exposure, i.e., inhalation, oral, and dermal (LSE Table 2-1, 2-2, and 2-3, respectively). LSE figures are limited to the inhalation (LSE Figure 2-l) and oral (LSE Figure 2-2) routes.

(2).   Exposure Duration Three exposure periods: acute (14 days or less); intermediate (15 to 364 days); and chronic (365 days or more) are presented within each route of exposure. In this example, an inhalation study of intermediate duration exposure is reported.

(3).   Health Effect The major categories of health effects included in LSE tables and figures are
        death, systemic, immunological, neurological, developmental, reproductive, and cancer.
        NOAELs and LOAELs can be reported in the tables and figures for all effects but cancer.
        Systemic effects are further defined in the "System" column of the LSE table.

(4).   Key to Figure Each key number in the LSE table links study information to one or more data
        points using the same key number in the corresponding LSE figure. In this example, the study
        represented by key number 18 has been used to define a NOAEL and a Less Serious LOAEL
        (also see the two "18r" data points in Figure 2-l).

(5).   Species The test species, whether animal or human, are identified in this column. Species

(6).   Exposure Frequency/Duration The duration of the study and the weekly and daily exposure
        regimen are provided in this column. This permits comparison of NOAELs and LOAELs from
        different studies. In this case (key number 18), rats were exposed to [substance x] via
        inhalation for 13 weeks, 5 days per week, for 6 hours per day.

(7).   System This column further defines the systemic effects. These systems include: respiratory,
        cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, and
        dermal/ocular. "Other" refers to any systemic effect (e.g., a decrease in body weight) not
        covered in these systems. In the example of key number 18, one systemic effect (respiratory)
        was investigated in this study.

(8).   NOAEL A No-Observed-Adverse-Effect Level (NOAEL) is the highest exposure level at which
        no harmful effects were seen in the organ system studied. Key number 18 reports a NOAEL of
        3 ppm for the respiratory system which was used to derive an intermediate exposure, inhalation
        MRL of 0.005 ppm (see footnote "b").

(9).   LOAEL A Lowest-Observed-Adverse-Effect Level (LOAEL) is the lowest exposure level used
        in the study that caused a harmful health effect. LOAELs have been classified into "Less
        Serious" and "Serious" effects. These distinctions help readers identify the levels of exposure at
        which adverse health effects first appear and the gradation of effects with increasing dose. A
        brief description of the specific end point used to quantify the adverse effect accompanies the
        LOAEL. The "Less Serious" respiratory effect reported in key number 18 (hyperplasia)
        occurred at a LOAEL of 10 ppm.

(10).   Reference The complete reference citation is given in Chapter 8 of the profile.

(11).   CEL A Cancer Effect Level (CEL) is the lowest exposure level associated with the onset of
        carcinogenesis in experimental or epidemiological studies. CELs are always considered serious
        effects. The LSE tables and figures do not contain NOAELs for cancer, but the text may report
        doses which did not cause a measurable increase in cancer.

(12).   Footnotes Explanations of abbreviations or reference notes for data in the LSE tables are found
        in the footnotes. Footnote "b" indicates the NOAEL of 3 ppm in key number 18 was used to
        derive an MRL of 0.005 ppm.

LEGEND

See LSE Figure 2-1

LSE figures graphically illustrate the data presented in the corresponding LSE tables. Figures help the
reader quickly compare health effects according to exposure levels for particular exposure duration,

① ────────────────────────────────▶ **TABLE 2-1. Levels of Significant Exposure to [Chemical x] - Inhalation**

Key to figure[a]	Species	Exposure frequency/ duration	System	NOAEL (ppm)	LOAEL (effect)		Reference
					Less serious (ppm)	Serious (ppm)	

② ─▶ INTERMEDIATE EXPOSURE

③ ─▶ Systemic ⑤ ⑥ ⑦ ⑧ ⑨ ⑩

④ ─▶ 18 Rat 13 wk 5d/wk 6hr/d Resp 3[b] 10 (hyperplasia) Nitschke et al. 1981

CHRONIC EXPOSURE ⑪

Cancer

38 Rat 18 mo 5d/wk 7hr/d 20 (CEL, multiple organs) Wong et al. 1982

39 Rat 89-104 wk 5d/wk 6hr/d 10 (CEL, lung tumors, nasal tumors) NTP 1982

40 Mouse 79-103 wk 5d/wk 6hr/d 10 (CEL, lung tumors, hemangiosarcomas) NTP 1982

[a] The number corresponds to entries in Figure 2-1.

⑫ ─▶ [b] Used to derive an intermediate inhalation Minimal Risk Level (MRL) of 5×10^{-3} ppm; dose adjusted for intermittent exposure and divided by an uncertainty factor of 100 (10 for extrapolation from animal to humans, 10 for human variability).

CEL = cancer effect level; d = day(s); hr = hour(s); LOAEL = lowest-observed-adverse-effect level; mo = month(s); NOAEL = no-observed-adverse-effect level; Resp = respiratory; wk = week(s)

SAMPLE

FIGURE 2-1. Levels of Significant Exposure to [Chemical X] - Inhalation

13 ──────────► INTERMEDIATE
(15-364 Days)

CHRONIC
(≥ 365 Days)

Systemic

Systemic

14 ──────► Death Respiratory Hematological Hepatic   Death Respiratory Hematological Hepatic Renal Other Reproduction Cancer*

15 ──► (ppm)

10,000	
1,000	
100	●10r ●17r ◖24g ◖25m ○21r ◖24g ◖21r ○22h ●32r ○36m ◖34r ◖34r ◖34r ◖34r ◆38r
10	○25m ◖18r ◖19r ●33m ○32r ◖36m ◖35r ○34r ○34r ○34r ○34r ◖37r ◆40m ◆38r
16 ──	○18r ○19r
1	
0.1	
0.01	
0.001	
0.0001	
0.00001	

17 ──────

10-4
10-5 ─ Estimated Upper- ◄── 18
Bound Human
Cancer Risk
10-6 ─ Levels
10-7 ─

Key

r	Rat	● LOAEL for serious effects (animals)	Minimal risk level for
m	Mouse	◖ LOAEL for less serious effects (animals)	effects other than cancer
h	Rabbit	○ NOAEL (animals)	
g	Guinea pig	◆ CEL - Cancer Effect Level (animals)	
h	Monkey		

The number next to each point corresponds to entries in Table 2-1.

* Doses represent the lowest dose tested per study that produced a tumorigenic
response and do not imply the existence of a threshold for the cancer end point.

19 ──────

f39017-1

(13).   <u>Exposure Duration</u> The same exposure periods appear as in the LSE table. In this example,
         health effects observed within the intermediate and chronic exposure periods are illustrated.

(14).   <u>Health Effect</u> These are the categories of health effects for which reliable quantitative data
         exist. The same health effects appear in the LSE table.

(15).   <u>Levels of Exposure</u> Exposure levels for each health effect in the LSE tables are graphically
         displayed in the LSE figures. Exposure levels are reported on the log scale "y" axis.
         Inhalation exposure is reported in mg/m^3 or ppm and oral exposure is reported in mg/kg/day.

(16).   <u>NOAEL</u> In this example, 18r NOAEL is the critical end point for which an intermediate
         inhalation exposure MRL is based. As you can see from the LSE figure key, the
         open-circle symbol indicates a NOAEL for the test species (rat). The key number 18
         corresponds to the entry in the LSE table. The dashed descending arrow indicates the
         extrapolation from the exposure level of 3 ppm (see entry 18 in the Table) to the MRL of
         0.005 ppm (see footnote "b" in the LSE table).

(17).   <u>CEL</u> Key number 38r is one of three studies for which Cancer Effect Levels (CELs) were
         derived. The diamond symbol refers to a CEL for the test species (rat). The number 38
         corresponds to the entry in the LSE table.

(18).   <u>Estimated Upper-Bound Human Cancer Risk Levels</u> This is the range associated with the
         upper-bound for lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. These risk levels are
         derived from EPA's Human Health Assessment Group's upper-bound estimates of the slope
         of the cancer dose response curve at low dose levels (q_l*).

(19).   <u>Key to LSE Figure</u> The Key explains the abbreviations and symbols used in the figure.

Chapter 2 (Section 2.4)

Relevance to Public Health

The Relevance to Public Health section provides a health effects summary based on evaluations of
existing toxicological, epidemiological, and toxicokinetic information. This summary is designed to
present interpretive, weight-of-evidence discussions for human health end points by addressing the
following questions.

         1 . What effects are known to occur in humans?

         2 . What effects observed in animals are likely to be of concern to humans?

         3 . What exposure conditions are likely to be of concern to humans, especially around hazardous waste
         sites?

The section discusses health effects by end point. Human data are presented first, then animal data.
Both are organized by route of exposure (inhalation, oral, and dermal) and by duration (acute,
intermediate, and chronic). *In vitro* data and data from parenteral routes (intramuscular, intravenous,
subcutaneous, etc.) are also considered in this section. If data are located in the scientific literature, a
table of genotoxicity information is included.

The carcinogenic potential of the profiled substance is qualitatively evaluated, when appropriate, using existing toxicokinetic, genotoxic, and carcinogenic data. ATSDR does not currently assess cancer potency or perform cancer risk assessments. MRLs for noncancer end points if derived, and the end points from which they were derived are indicated and discussed in the appropriate section(s).

Limitations to existing scientific literature that prevent a satisfactory evaluation of the relevance to public health are identified in the Identification of Data Needs section.

Interpretation of Minimal Risk Levels

Where sufficient toxicologic information was available, MRLs were derived. MRLs are specific for route (inhalation or oral) and duration (acute, intermediate, or chronic) of exposure. Ideally, MRLs can be derived from all six exposure scenarios (e.g., Inhalation - acute, -intermediate, -chronic; Oral - acute, -intermediate, - chronic). These MRLs are not meant to support regulatory action, but to acquaint health professionals with exposure levels at which adverse health effects are not expected to occur in humans. They should help physicians and public health officials determine the safety of a community living near a substance emission, given the concentration of a contaminant in air or the estimated daily dose received via food or water. MRLs are based largely on toxicological studies in animals and on reports of human occupational exposure.

MRL users should be familiar with the toxicological information on which the number is based. Section 2.4, "Relevance to Public Health," contains basic information known about the substance. Other sections such as 2.6, "Interactions with Other Chemicals" and 2.7, "Populations that are Unusually Susceptible" provide important supplemental information.

MRL users should also understand the MRL derivation methodology. MRLs are derived using a modified version of the risk assessment methodology used by the Environmental Protection Agency (EPA) (Barnes and Dourson 1988; EPA 1989a) to derive reference doses (RfDs) for lifetime exposure.

To derive an MRL, ATSDR generally selects the end point which, in its best judgement, represents the most sensitive human health effect for a given exposure route and duration. ATSDR cannot make this judgement or derive an MRL unless information (quantitative or qualitative) is available for all potential effects (e.g., systemic, neurological, and developmental). In order to compare NOAELs and LOAELs for specific end points, all inhalation exposure levels are adjusted for 24 hr exposures and all intermittent exposures for inhalation and oral routes of intermediate and chronic duration are adjusted for continuous exposure (i.e., 7 days/week).   If the information and reliable quantitative data on the chosen end point are available, ATSDR derives an MRL using the most sensitive species (when information from multiple species is available) with the highest NOAEL that does not exceed any adverse effect levels. The NOAEL is the most suitable end point for deriving an MRL. When a NOAEL is not available, a Less Serious LOAEL can be used to derive an MRL, and an uncertainty factor of (1, 3, or 10) is employed. MRLs are not derived from Serious LOAELs. Additional uncertainty factors of (1, 3, or 10) are used for human variability to protect sensitive subpopulations (people who are most susceptible to the health effects caused by the substance) and (1, 3, or 10) are used for interspecies variability (extrapolation from animals to humans). In deriving an MRL, these individual uncertainty factors are multiplied together. Generally an uncertainty factor of 10 is used; however, the MRL Workgroup reserves the right to use uncertainty factors of (1, 3, or 10) based on scientific judgement. The product is then divided into the adjusted inhalation concentration or oral dosage selected from the study. Uncertainty factors used in developing a substance-specific MRL are provided in the footnotes of the LSE Tables.

APPENDIX B

ACRONYMS, ABBREVIATIONS, AND SYMBOLS

ACGIH	American Conference of Governmental Industrial Hygienists
ADME	Absorption, Distribution, Metabolism, and Excretion
atm	atmosphere
ATSDR	Agency for Toxic Substances and Disease Registry
BCF	bioconcentration factor
BSC	Board of Scientific Counselors
C	Centigrade
CDC	Centers for Disease Control
CEL	Cancer Effect Level
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
CLP	Contract Laboratory Program
cm	centimeter
CNS	central nervous system
d	day
DHEW	Department of Health, Education, and Welfare
DHHS	Department of Health and Human Services
DOL	Department of Labor
ECG	electrocardiogram
EEG	electroencephalogram
EPA	Environmental Protection Agency
EKG	see ECG
F	Fahrenheit
F_1	first filial generation
FAO	Food and Agricultural Organization of the United Nations
FEMA	Federal Emergency Management Agency
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
fpm	feet per minute
ft	foot
FR	Federal Register
g	gram
GC	gas chromatography
gen	generation
HPLC	high-performance liquid chromatography
hr	hour
IDLH	Immediately Dangerous to Life and Health
IARC	International Agency for Research on Cancer
ILO	International Labor Organization
in	inch
Kd	adsorption ratio
kg	kilogram
kkg	metric ton
K_{oc}	organic carbon partition coefficient
K_{ow}	octanol-water partition coefficient
L	liter
LC	liquid chromatography
LC_{Lo}	lethal concentration, low
LC_{50}	lethal concentration, 50% kill
LD_{Lo}	lethal dose, low
LD_{50}	lethal dose, 50% kill
LOAEL	lowest-observed-adverse-effect level
LSE	Levels of Significant Exposure

m	meter
mg	milligram
min	minute
mL	milliliter
mm	millimeter
mmHg	millimeters of mercury
mmol	millimole
mo	month
mppcf	millions of particles per cubic foot
MRL	Minimal Risk Level
MS	mass spectrometry
NIEHS	National Institute of Environmental Health Sciences
NIOSH	National Institute for Occupational Safety and Health
NIOSHTIC	NIOSH's Computerized Information Retrieval System
ng	nanogram
nm	nanometer
NHANES	National Health and Nutrition Examination Survey
nmol	nanomole
NOAEL	no-observed-adverse-effect level
NOES	National Occupational Exposure Survey
NOHS	National Occupational Hazard Survey
NPL	National Priorities List
NRC	National Research Council
NTIS	National Technical Information Service
NTP	National Toxicology Program
OSHA	Occupational Safety and Health Administration
PEL	permissible exposure limit
pg	picogram
pmol	picomole
PHS	Public Health Service
PMR	proportionate mortality ratio
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion
REL	recommended exposure limit
RfD	Reference Dose
RTECS	Registry of Toxic Effects of Chemical Substances
sec	second
SCE	sister chromatid exchange
SIC	Standard Industrial Classification
SMR	standard mortality ratio
STEL	short term exposure limit
STORET	STORAGE and RETRIEVAL
TLV	threshold limit value
TSCA	Toxic Substances Control Act
TRI	Toxics Release Inventory
TWA	time-weighted average
U.S.	United States
UF	uncertainty factor
yr	year
WHO	World Health Organization
wk	week
>	greater than
$\geq$	greater than or equal to

APPENDIX B

$=$	equal to
$<$	less than
$\leq$	less than or equal to
$\%$	percent
α	alpha
β	beta
δ	delta
γ	gamma
μm	micron
μg	microgram