TOXICOLOGICAL PROFILE FOR USED MINERAL-BASED CRANKCASE OIL

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

September 1997

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UPDATE STATEMENT

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 the Agency for Toxic Substances and Disease Registry (ATSDR) and the Environmental Protection Agency (EPA). 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 described therein. Each peer-reviewed profile identifies and reviews the key literature that describes a hazardous substance's toxicologic properties. Other pertinent literature is also presented, but is 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.

The focus of the profiles is on health and toxicologic information; therefore, each toxicological profile begins with a public health statement that describes, in nontechnical language, a substance's relevant toxicological properties. Following the public health statement is information concerning levels of significant human exposure and, where 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 of significance to protection of public health are identified by ATSDR and EPA.

Each profile includes the following:

(A) The examination, summary, and interpretation of available toxicologic information and epidemiologic evaluations on a hazardous substance 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; and

(C) Where appropriate, identification of toxicologic testing needed to identify the types or levels of exposure that may 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.

This profile reflects ATSDR's assessment of all relevant toxicologic testing and information that has been peer-reviewed. Staff of the Centers for Disease Control and Prevention and other Federal scientists have also reviewed the profile. In addition, this profile has been peer-reviewed by a nongovernmental panel 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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*Legislative Background

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).

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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 end points.

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.

PEER REVIEW

A peer review panel was assembled for Used Mineral-Based Crankcase Oil. The panel consisted of the following members:

1. Dr. Carson Conaway, Research Scientist, Mahopac, NY
2. Dr. Walter Decker, Private Consultant, El Paso, TX
3. Dr. David Warshawsky, Professor, Department of Environmental Health, University of Cincinnati, Cincinnati, OH

These experts collectively have knowledge of Used Mineral-Based Crankcase Oil's physical and chemical properties, toxicokinetics, 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.

CONTENTS

LIST OF FIGURES

LIST OF TABLES

1. PUBLIC HEALTH STATEMENT

This Statement was prepared to give you information about used mineral-based crankcase oil and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) identifies the most serious waste sites in the nation. sites make up the National Priorities List (NPL) and are the sites targeted for long-term federal cleanup activities. Used mineral-based crankcase oil has been found in at least 85 of 1,430 current or former NPL sites. However, the total number of NPL sites evaluated is not known. As more sites are evaluated, the number of sites at which used mineral-based crankcase oil is found may increase. This information is important because exposure to used mineral-based crankcase oil may cause harmful health effects and because these sites are potential or actual sources of human exposure to used mineral-based crankcase oil.

When a substance 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. This release does not always lead to exposure. You can be exposed to a substance only when you come in contact with it. You may be exposed by breathing, eating, or drinking substances containing the substance or by skin contact with it.

If you are exposed to a substance such as used mineral-based crankcase oil, many 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, gender, nutritional status, family traits, life-style, and state of health.

1.1 WHAT IS USED MINERAL-BASED CRANKCASE OIL?

Used mineral-based crankcase oil is another name for used motor oil or used engine oil. It is the brown-to-black, oily liquid removed from the engine of a motor vehicle when the oil is

changed. Used mineral-based crankcase oil is similar to unused oil except that it contains additional chemicals that are produced or that build up in the oil when it is used as an engine lubricant. Used mineral-based crankcase oil has many of the characteristics of unused oil. It smells like unused oil and contains the chemicals found in unused oil. These include straight chain (aliphatic) hydrocarbons and aromatic or polycyclic aromatic hydrocarbons (PAHs), which are distilled from crude oil, and various additives that improve the performance of the oil in the engine. Aliphatic hydrocarbons are defined as molecules with carbon atoms in simple or branched chains. Aromatic hydrocarbons are compounds with unsaturated carbons in six-membered rings and with properties similar to benzene. PAHs are complex organic compounds containing three or more aromatic rings. In addition to the chemicals found in unused oil, used mineral-based crankcase oil contains chemicals that are formed when the oil is exposed to the high temperatures and pressures inside an engine as it runs. It also contains metals such as aluminum, chromium, copper, iron, lead, manganese, nickel, silicon, and tin, that come from engine parts as they wear down. In addition, used mineral-based crankcase oil contains small amounts of water, gasoline, antifreeze, and chemicals that come from gasoline when it burns inside the engine. The chemicals found in used mineral-based crankcase oil vary depending on the brand(s) and type of engine oil used, whether gasoline or diesel fuel was used, the mechanical condition of the engine that the oil came from, the various sources of used mineral-based crankcase oil (e.g., automobiles, airplanes, trains, ships, tractors, lawn mowers), and the number of miles driven between oil changes. Used mineralbased crankcase oil is not naturally found in the environment, but most of the chemicals found in the oil do occur naturally.

A large amount of used mineral-based crankcase oil is generated each year when motor oil is changed. It is usually discarded into the environment or recycled, but some other uses for it exist. Some industries mix used mineral-based crankcase oil with other oils to produce cutting oils or other lubricating oils. Used mineral-based crankcase oil can also be burned. It burns at about 300-400°C, depending on the mixture of chemicals in the used mineral-based crankcase oil. It has been used as a fuel in oil burners in homes, as well as in industrial steam boilers, municipal incinerators, and rotary cement kilns. It is also used in producing

asphalt. In the past, used mineral-based crankcase oil was also used on dirt roads to control dust. However, most states currently restrict this use.

Please refer to Chapters 3 and 4 for more information on used mineral-based crankcase oil.

1.2 WHAT HAPPENS TO USED MINERAL-BASED CRANKCASE OIL WHEN IT ENTERS THE ENVIRONMENT?

Some used mineral-based crankcase oil enters the air through the exhaust system during engine use. Used mineral-based crankcase oil may also enter the water or soil when it is disposed of improperly, for example, when it is poured into sewers or directly onto the ground. It may also enter the environment as a result of disposal in landfills, engine oil leaks, automobile or truck exhaust, and application onto rural roads for dust control. Some chemicals found in used mineral-based crankcase oil may be released into the air when it is used as a fuel in boilers, incinerators, and cement kilns. The movement in the environment of the chemicals found in used mineral-based crankcase oil depends on their individual properties. The hydrocarbon components of the oil generally stick to the soil surface and do not move through the soil. If oil is spilled, some hydrocarbons evaporate into the air very quickly, and others evaporate more slowly. Hydrocarbons that do not evaporate may stay in the soil for a long time because they do not dissolve in water and do not generally break down. Hydrocarbon components of the oil that enter surface water bind to small particles in the water. They eventually settle to the bottom where they can stay for many years. These hydrocarbons may build up in shellfish or other organisms. Some metals in used mineralbased crankcase oil dissolve in water and move through the soil easily and may be found in surface water and groundwater. Groundwater flows slowly underground and then drains into surface waters such as marshes and lakes. Most metals found in used mineral-based crankcase oil stay in the environment for a long time. Thus, metals from used oils can build up in plants, animals, soil, sediments, and non-flowing surface water.

Please refer to Chapter 5 for more information on what happens to used mineral-based crankcase oil when it enters the environment.

1.3 HOW MIGHT I BE EXPOSED TO USED MINERAL-BASED CRANKCASE OIL?

You are most likely to be exposed to used mineral-based crankcase oil when you change the engine oil in your car. A small amount of the chemicals in exhaust fumes comes from used mineral-based crankcase oil. Therefore, you may be exposed to used mineral-based crankcase oil when you breathe in exhaust fumes. You may also be exposed to used mineral-based crankcase oil when it is used to control dust on rural roads. The waste crankcase oil used for road oiling comes from service stations or fleet shops. It is mixed with industrial oils which contain polychlorinated biphenyls (PCBs), chlorodibenzo dioxins (CDDs), and chlorodibenzo furans (CDFs) which are bad for your health. You may be exposed to these compounds when you breathe in these compounds as wind-blown dust. However, most states currently either prohibit or restrict its use for that purpose. Most people are exposed to very low levels of used mineral-based crankcase oil. People who live or work in the vicinity of an active or inactive recycling facility that recycles used mineral-based crankcase oil may either breathe fumes found near the facility or come in contact with contaminated soil, sludge, or sediment. People who work or live in or near buildings where used mineral-based crankcase oil is burned for heating fuel may breathe high levels of metal particles and PAHs.

People who live near landfills can also be exposed to used mineral-based crankcase oil. A common practice of people who change the oil in their own vehicles (or do-it-yourselfers) is to dump it together with other household wastes, which usually end up in landfills. The waste oil can then enter the environment and may contaminate crops, farm animals, or fish and shellfish. If you eat the contaminated food, you may be exposed to the hydrocarbons and heavy metals present in the waste oil. You may also be exposed to used mineral-based crankcase oil by accidental spills during transport. However, since very small amounts are transported, the environmental impact of spilled used oil is limited.

There is very little information on the levels of chemicals in the environment resulting from releases of used mineral-based crankcase oil. Used mineral-based crankcase oil is a source of hydrocarbons in storm runoff from bridges, rivers, streams, and deep as well as surface

sediments. However, in several studies, used mineral-based crankcase oil may be only one of many sources of the environmental contamination. Such studies have shown increased levels of various components of used mineral-based crankcase oil in dust, surface water, storm runoff, and roadside soil in industrial, commercial, and residential areas and near highways. Heavy metals, possibly from used crankcase oil sources and from motor vehicle exhausts, have been found in the soil near extensively traveled roads and highways. Surface water samples taken from ponds at an NPL site that was a recycling facility for used mineral-based crankcase oil and other used petroleum products were found to contain chromium, lead, and xylene, respectively.

Please refer to Chapter 5 for more information.

1.4 HOW CAN USED MINERAL-BASED CRANKCASE OIL ENTER AND LEAVE MY BODY?

Used mineral-based crankcase oil is a mixture of several different chemicals. Whether the chemicals in used mineral-based crankcase oil are taken up, stored in, or excreted by the body after exposure depends on the individual properties of the chemicals in the oil. Studies of cattle that swallowed used mineral-based crankcase oil showed that lead and other metals in the oil are absorbed and distributed to various tissues, such as the liver and kidneys. Studies in mice showed that the PAHs that build up in used mineral-based crankcase oil are absorbed when used mineral-based crankcase oil is applied to the skin. A study in rats showed that used mineral-based crankcase oil that is swallowed is eliminated in the feces. Medicinalgrade mineral oil is also eliminated rapidly from the body in the feces. Thus, it is likely that the chemicals that are in both medicinal-grade mineral oil and used mineral-based crankcase oil are eliminated rapidly in the feces, but we do not know the timeframe for elimination from the body of other chemicals found in used mineral-based crankcase oil. We also do not know whether other routes of elimination exist. Additional information on the movement of the Individual chemicals into, through, and from the body can be found in the toxicological profiles for those chemicals.

Please refer to Chapter 2 for more information.

1.5 HOW CAN USED MINERAL-BASED CRANKCASE OIL AFFECT MY HEALTH?

The health effects of exposure to used mineral-based crankcase oil vary depending on the properties of the chemicals found in the oil. Each brand of oil contains slightly different mixtures of oils and additives. Also, the characteristics of the engine in which the oil is used affect its final composition. Thus, effects experienced after exposure to one batch of used mineral-based crankcase oil may not be the same after exposure to another batch. Mechanics and other auto workers who are exposed to used mineral-based crankcase oil from a large number of motor vehicles have experienced effects on the skin (rashes), blood (anemia), and nervous system (headaches and tremors). However, these workers are also exposed to a large number of other chemicals in the workplace. Exposures to some of these other substances are known to cause rashes, anemia, headaches, and tremors. Therefore, it is possible that the effects the workers experienced may have been caused by chemicals from sources other than used mineral-based crankcase oil.

Volunteers who breathed mists of used mineral-based crankcase oil for a few minutes had slightly irritated noses and throats, and the mists were irritating to the eyes of some people. Animals that ate large amounts of used mineral-based crankcase oil developed diarrhea. Thus, people who swallow used mineral-based crankcase oil may also have diarrhea. Some cows that ate used oil containing metals such as molybdenum and lead in contaminated pastures experienced harmful effects on the blood, such as anemia, and on the nervous system, such as tremors. Some of the cows died. There is a possibility that anemia and tremors may occur in people exposed to used mineral-based crankcase oil. Used mineral-based crankcase oil was only slightly irritating to the skin of rabbits, guinea pigs, and mice. We do not know if exposure to used mineral-based crankcase oil affects the reproductive ability of men or women or whether it causes birth defects.

Long-term exposure of the skin to used mineral-based crankcase oil causes skin cancer in mice. PAHs in the oil have been identified as the cancer-causing agents because some PAHs are known to cause cancer and because the carcinogenicity of various batches of the used oil increased with increasing amounts of PAHs in the oil. The Department of Health and Human Services, the International Agency for Research on Cancer (IARC), and EPA have not classified used mineral-based crankcase oil with regard to its carcinogenicity in people.

The health effects of many of the individual chemicals found in used mineral-based crankcase oil (PAHs, lead, zinc, cadmium, copper, chromium, nickel, barium, boron, manganese, tin, and aluminum) are discussed in the toxicological profiles for those chemicals. Please refer to Chapter 2 for more information on the health effects associated with exposure to used mineral-based crankcase oil.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO USED MINERAL-BASED CRANKCASE OIL?

Used mineral-based crankcase oil is a mixture of a large number of chemicals. Its composition depends on the brand of oil and the characteristics of the engine in which it was used. Therefore, no one medical test will show if you have been exposed to used mineralbased crankcase oil. However, there are methods for determining if you have been exposed to some of the chemicals in used mineral-based crankcase oil. These include testing for the presence of metals such as lead or molybdenum in your blood, or testing for deoxyribonucleic acid (DNA) adducts (chemical addition products) with PAHs in your skin cells. These tests are not part of routine medical examinations. However, your doctor can collect blood or tissue samples and send them to most university medical centers or medical laboratories for analysis. It should be noted that molybdenum and PAHs are common environmental contaminants and elevated levels of these materials may be the result of exposures to materials not associated with used mineral-based crankcase oil. Also, testing for exposure to specific chemicals in the oil cannot be used to find out exactly how much used mineral-based crankcase oil you have been exposed to. However, the testing is very useful because the amounts of individual chemicals identified can be used to determine the health effects that

you may experience. Health professionals with expertise in occupational medicine or industrial hygiene should be able to identify chemicals that may have been in the oil and recommend tests for those chemicals. Some of the tests for specific chemicals provide information about previous exposure levels and about whether harmful health effects may occur. For more information on the tests available for specific chemical components of used mineral-based crankcase oil, please refer to the toxicological profiles for those chemicals.

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

Regulations for used mineral-based crankcase oil are still being created and revised. Thus, federal guidelines or standards for inhalation, drinking water, food, or dermal exposure to used mineral-based crankcase oil are not yet available. However, the EPA and most states have developed regulations regarding disposal of used oil, its recycling, spraying used oil onto road surfaces for dust control, or burning it as a fuel. See Chapter 7 for further information.

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
(404) 639-6000

This agency can also provide you with inforrnation on the location of occupational and environmental health clinics. These clinics specialize in the recognition, evaluation, and treatment of illness 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 on the toxicology of used mineral-based crankcase oil. It contains descriptions and evaluations of toxicological studies and epidemiological investigations and provides conclusions, where possible, on the relevance of toxicity and toxicokinetic data to public health.

A glossary and list of acronyms, abbreviations, and symbols can be found at the end of this profile.

This profile will focus on the properties of *used* mineral-based crankcase oil because most of the mineral-based crankcase oil at hazardous waste sites is expected to be previously used oil. It is important to make the distinction between new and used mineral-based crankcase oil because the characteristics of mineral-based crankcase oil change with use (Vazquez-Duhalt 1989). An important difference between new and used motor oil is the heavy metal content. This difference is extremely important because many of the metals are harmful to human health and living organisms. These metals originate from the fuel and from motor wear. Used oil contains high concentrations of lead, zinc, calcium, barium, and magnesium along with lower concentrations of iron, sodium, copper, aluminium, chromium, manganese, potassium, nickel, tin, silicon, boron, and molybdenum (Vasquez-Duhalt 1989). Concentrations of lead in used mineral-based crankcase oil were likely higher when leaded gasoline was used.

Mineral-based crankcase oils are manufactured using highly refined base oils and contain up to 20% of a variety of additives such as viscosity index improvers, detergents/dispersants, antiwear additives, pour-point depressants, and antioxidants (IARC 1984; Kirk-Othmer 1981). During use, the high temperatures and friction cause changes such as oxidation, nitration, and cracking of polymers in the component chemicals (Vazquez-Duhalt 1989). In addition, a variety of substances such as fuel, water, antifreeze, dust, and various combustion products such as polycyclic aromatic hydrocarbons (PAHs), metals, and metallic oxides accumulate in the oil. The degree of chemical change and accumulation of

contaminants in the oil increases with use and varies depending on the type of fuel used and the mechanical properties of the engine.

In an engine lubricating system, the required quantity of lubricant is transported where it is needed in the engine. The lubricant protects against wear, reduces friction, cleans the engine of dirt and residue (detergent), protects against corrosion, cools the engine, and seals the pistons (Van Donkelaar 1990). Additives are added to lubricating oils to improve its physical and chemical properties. Consequently, lubricating oils have high additive contents (up to 20%), especially detergents and dispersants which constitute 2-15% of oil weight (Vasquez-Duhalt 1989). However, several of the oil additives are toxic environmental contaminants, e.g., zinc dithiophosphate and zinc diary1 or dialkyl dithiophosphates (ZDTPs); calcium alkyl phenates; magnesium, sodium, and calcium sulphonates; tricresyl phosphates; molybdenum disulfide; heavy metal soaps; and other organometallic compounds that contain heavy metals. Hence, very high levels of zinc and cadmium are found in new motor oil-approximately 1,500 μg/g of zinc and 87 μg/kg of cadmium (Hewstone 1994a; Vasquez-Duhalt 1989). Although ZDTPs have a low acute systemic toxicity, they can cause eye damage and skin irritation (Hewstone 1994a). Prolonged exposure to high concentrations of ZDTPs, calcium alkyl phenates, and magnesium, sodium, and calcium sulphonates had significant effects on the reproductive organs of male rabbits (testicular atrophy and reduction or absence of spermatozoa) which appeared to be species specific. The absorption of tricresyl phosphates caused peripheral nervous system damage, leading to neuromuscular problems (Hewstone 1994a).

In a crankcase-lubricated engine, the oil compartment acts as a sink for heavy molecular incomplete combustion products such as PAHs and their analogs (Scheepers and Bos 1992). Thus, contaminants such as PAHs, which are formed via combustion, can accumulate in the oil by a factor of up to 1,000. PAHs are known to be highly toxic environmental contaminants with carcinogenic and mutagenic properties. They leave the engine in various ways, such as via particulates, oil leaks, and uncontrolled oil changes, which then accumulates in the environment. One hundred and forty different PAHs have been found in the used oil of crankcase-lubricated engines. These PAHs are also preseni'in much lower quantities in new or fresh oil (Van Donkelaar 1990).

Used mineral-based crankcase oil is a complex mixture of metals and PAHs. When motor oils undergo thermal decomposition, gasoline combustion products are formed, significantly increasing the levels of PAHs which contribute to the carcinogenic and mutagenic properties of the oils (Bingham

1988; Ingram et al. 1994). Hence, it is difficult to define the precise composition of used mineralbased crankcase oil because of the variety of chemical additives that may be present and the varying degrees of chemical decomposition and contaminant accumulation. Therefore, rather than describing toxicities associated with individual components, the following discussion focuses on information obtained in studies that have examined the effects of exposure to samples of used mineral-based crankcase oil. In several studies, composite samples of used mineral-based crankcase oil have been employed; these studies may therefore provide a more generalized picture of toxicities associated with exposure. However, it should be noted that the results of any one study may not be representative of effects occurring with similar exposures to other samples of used mineral-based crankcase oil.

2.2 DISCUSSION OF HEALTH EFFECTS BY ROUTE OF EXPOSURE

To help public health professionals and others 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, reproductive, developmental, 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. “Serious” effects are those that evoke failure in a biological system and can lead to morbidity or mortality (e.g., acute respiratory distress or death). “Less serious” effects are those that are not expected to cause significant dysfunction or death, or those whose significance to the organism is not entirely clear. ATSDR acknowledges that a considerable amount of judgment may be required in establishing whether an end point should be classified as a NOAEL, “less serious” LOAEL, or “serious” LOAEL, and’that in some cases, there will be insufficient data to decide whether the effect is indicative of significant dysfunction. However, the Agency has established guidelines and policies that are used to classify these end points. ATSDR believes that there is sufficient merit in this approach to warrant an attempt at distinguishing between “less serious” and “serious” effects. The distinction between “less serious” effects and “serious” effects is considered to be important because it helps the users of the profiles to

identify levels of exposure at which major health effects start to appear. LOAELs or NOAELs should also help in determining 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 Levels of Significant Exposure (LSE) tables and figures may differ depending on the user's perspective. Public health officials and others concerned with appropriate actions to take at hazardous waste sites may want information on levels of exposure associated with more subtle effects in humans or animals or exposure levels below which no adverse effects have been observed. Estimates of levels posing minimal risk to humans (Minimal Risk Levels or MRLs) may be of interest to health professionals and citizens alike.

Levels of exposure associated with carcinogenic effects (Cancer Effect Levels, CELs) of used mineral-based crankcase oil are indicated in Table 2-3.

A User's Guide has been provided at the end of this profile (see Appendix B). This guide should aid in the interpretation of the tables and figures for Levels of Significant Exposure and the MRLs.

2.2.1 Inhalation Exposure

2.2.1.1 Death

No studies were located regarding death in humans or animals after inhalation exposure to used mineral-based crankcase oil.

2.2.1.2 Systemic Effects

No studies were located regarding musculoskeletal or renal effects in humans or animals after inhalation exposure to used mineral-based crankcase oil.

The systemic effects observed after inhalation exposure are discussed below. Although some qualitative information was located for cardiovascular, gastrointestinal, hematological, hepatic, dermal and ocular effects, NOAELs and/or LOAELs for these effects were not identified. The highest

NOAEL value for respiratory effects in guinea pigs after an acute-duration inhalation exposure to used mineral-based crankcase oil is recorded in Table 2-1 and plotted in Figure 2-1.

Respiratory Effects. Ten-minute exposures of volunteers to aerosols of used mineral-based crankcase oils resulted in mild-to-moderate nose and throat irritation (Dautrebande and Capps 1950). If a respiratory filter mask was worn, the degree of irritation was decreased to none-to-mild even at twofold higher concentrations. Insufficient information was provided in the report to convert the exposure levels used in this study, expressed as ppm by weight, to mg/m^3. In another study in which inhalation exposures occurred as the result of leakage of the aerosol from inside goggles designed to assess ocular exposures, chest tightness (but no nose irritation) was reported (Dautrebande et al. 1951). Concentrations of the used mineral-based crankcase oil in ambient air were not measured in this study; however effects were observed in one of three volunteers exposed within the goggles to 42 mg/m^3 and in another to 84 mg/m^3. Both of these studies are limited in that they were conducted over 40 years ago and are of limited value in predicting effects of current formulations that have been used in present-day engines. No studies were located regarding the respiratory effects in humans after longer term exposure to aerosols of used mineral-based crankcase oil.

Guinea pigs exposed for 1 hour to an aerosol of used mineral-based crankcase oil at concentrations as high as 222 mg/m^3 showed no adverse effects on pulmonary function (Costa and Amdur 1979a). Studies in animals on the respiratory effects of longer-term exposures were not found.

Cardiovascular Effects. Blood pressure was elevated in 37% of the mechanics, 7% of the apprentice mechanics, and 18% of the miscellaneous workers from 10 auto shops in Denmark (Clausen and Rastogi 1977). However, interpretation of these results is severely limited because of the absence of comparison with a control population, the likelihood of dermal as well as inhalation exposures, the high probability of exposure to other causative substances, and the failure to consider other confounding factors.

No studies were located regarding cardiovascular effects in animals after inhalation exposure to used mineral-based crankcase oil.

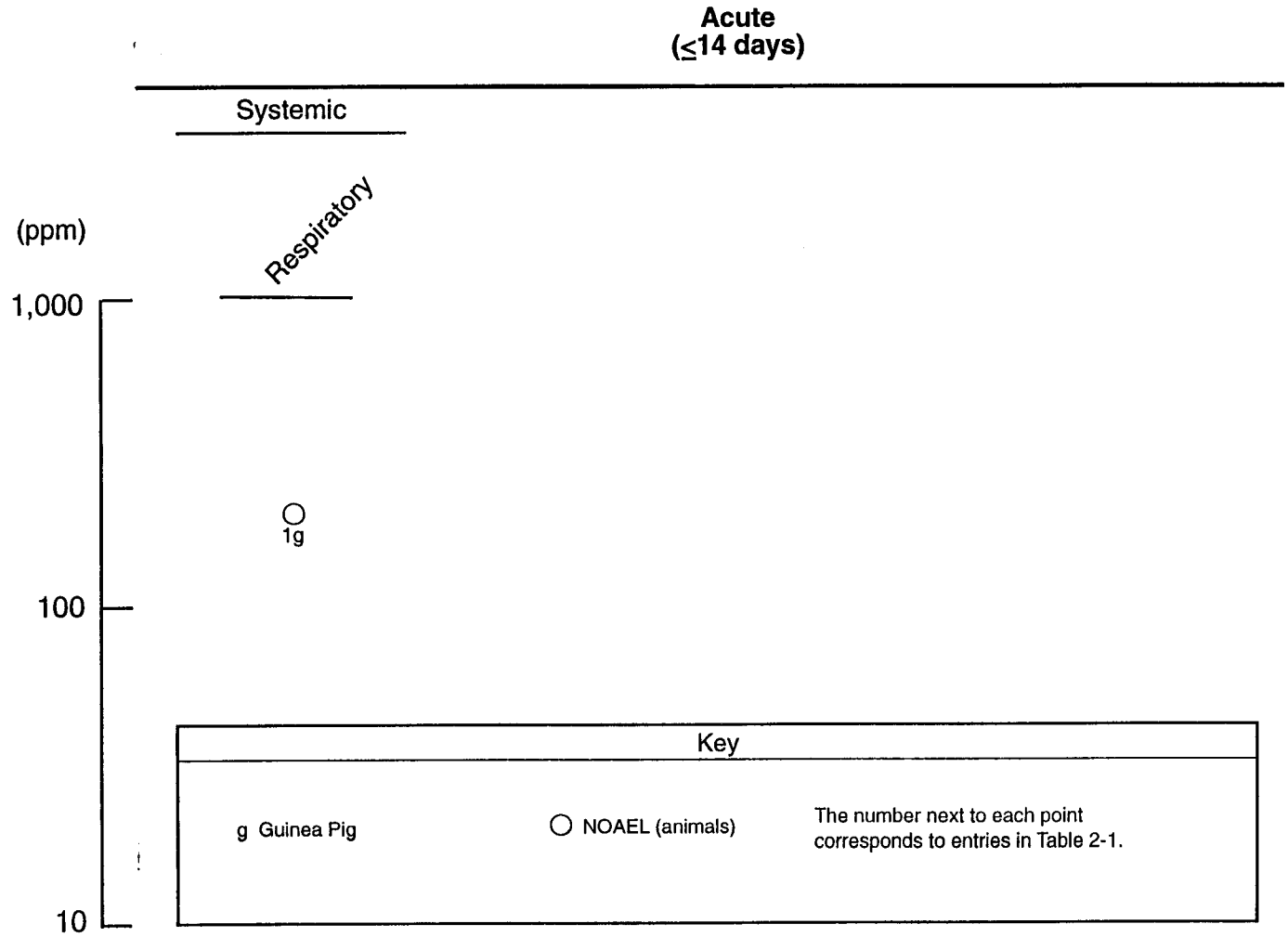
TABLE 2-1. Levels of Significant Exposure to Used Mineral-based Crankcase Oil - Inhalation

Key [a] to figure	Species (strain)	Exposure duration/ frequency	System	NOAEL (ppm)	LOAEL (effect)		Reference
					Less serious (ppm)	Serious (ppm)	
ACUTE EXPOSURE							
Systemic							
1	Gn pig (NS)	1 hr	Resp	222			Costa and Amdur 1979a

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

Gn pig = guinea pig; hr = hour; LOAEL = lowest-observed-adverse-effect level; NOAEL = no-observed-adverse-effect level; NS = not specified; Resp = respiratory

Figure 2-1. Levels of Significant Exposure to Used Mineral-based Crankcase Oil - Inhalation



Gastrointestinal Effects. The incidence of gastrointestinal effects (stomach pain, constipation, anorexia) did not appear to be increased in an epidemiologic study of mechanics and apprentice mechanics from auto shops in Denmark (Clausen and Rastogi 1977). As indicated above under Cardiovascular Effects, interpretation of these results is severely limited because of the absence of a comparison with a control population, the likelihood of dermal as well as inhalation exposures, the high probability of exposure to other causative substances, and the failure to consider other confounding factors.

No studies were located regarding gastrointestinal effects in animals after inhalation exposure to used mineral-based crankcase oil.

Hematological Effects. Lower than normal hematocrit and mean corpuscular hemoglobin were observed in several mechanics and apprentice mechanics in an epidemiologic study examining health effects among auto shop workers (Clausen and Rastogi 1977). A significant correlation was seen between the lead level and delta-aminolevulinic acid dehydratase (δ-ALAD) activity in the blood of both normal subjects and autoworkers (Spear-man's rank analysis, $p < 0.001$). In the blood of autoworkers, the δ-ALAD activity was depressed because of the higher blood lead levels. The lead levels in whole blood of all autoworkers was significantly higher than that of control subjects (Wilcoxon's test, $p < 0.002$) (Clausen and Rastogi 1977). Elevated blood lead levels were observed in 52% of the mechanics when compared to levels in controls not employed in the auto industry (Wilcoxon's test, $p < 0.002$). In several cases, the elevated lead levels correlated with decreases in hematocrit and mean corpuscular hemoglobin, suggesting that the effects may have been related to blood lead levels (Clausen and Rastogi 1977). Eastin et al. (1983) demonstrated that in ducks and pheasants fed diets containing up to 4.5% of used mineral-based crankcase oil, δ-ALAD was significantly decreased (ANOVA and Tukey's HSD test, $p <0.05$). No effects on hematocrit or hemoglobin concentration were seen. δ-ALAD is one of the most sensitive and one of the first enzymes in the pathway for heme synthesis which is inhibited by lead. In the absence of effects on hemoglobin and hematocrit, it is an early biological indicator of subclinical lead poisoning in humans and birds (Goyer 1996).

The study by Eastin et al. (1983) suggests that used oil is a source of lead exposure and hematoxicity in mechanics (Clausen and Rastogi 1977). Other studies by Blakley and Brockman (1976), Osweiler

et al. (1973), and Sas (1989) also support the conclusion that lead poisoning is a probable hazard from exposure to used crankcase oils. High blood lead levels have been associated with anemia (see the ATSDR profile on lead [ATSDR 1993b]). Although used mineral-based crankcase oil was determined to be one source of lead exposure, other sources of lead exposure (exhaust gas, gasoline, gear oil) may also have contributed to the effects observed.

No studies were located regarding the hematological effects of used mineral-based crankcase oil in animals after inhalation exposure.

Hepatic Effects. Increased serum bilirubin and thymol reaction (indicative of increased serum alkaline phosphatase) were observed in 14% of the mechanics examined in a study of auto shops in Denmark (Clausen and Rastogi 1977). In addition, 11% of the mechanics had elevated serum alanine and aspartate aminotransferase and lactate dehydrogenase activities, suggesting hepatic damage. However, it is unclear whether dermal exposures, substances other than used mineral-based crankcase oil, or diseases unrelated to employment may have contributed to the effects observed. Also, it is unknown whether these effects were significantly increased relative to a control population.

No studies were located regarding hepatic effects in animals after inhalation exposure to used mineral-based crankcase oil.

Dermal Effects. Skin irritation has been reported in epidemiological and experimental studies of humans after exposure to used mineral-based crankcase oil (Clausen and Rastogi 1977; Dautrebande and Capps 1950). However, these effects were probably due to direct contact of aerosols or volatile components with these tissues and will be discussed under the section dealing with effects of dermal exposures.

No studies were located regarding dermal effects in animals after inhalation exposure to used mineral-based crankcase oil.

Ocular Effects. Eye irritation has been reported in epidemiological and experimental studies of humans after exposure to used mineral-based crankcase oil (Clausen and Rastogi 1977; Dautrebande and Capps 1950). However, these effects were probably due to direct contact of aerosols or volatile

components with these tissues and will be discussed under the section dealing with effects of dermal exposures.

No studies were located regarding ocular effects in animals after inhalation exposure to used mineral-based crankcase oil.

2.2.1.3 Immunological and Lymphoreticular Effects

No studies were located regarding immunological and lymphoreticular effects in humans or animals after inhalation exposure to used mineral-based crankcase oil.

2.2.1.4 Neurological Effects

Examination of workers from 10 auto shops in Denmark revealed headaches in the morning among 14% and headaches in the evening among 20% of the mechanics (Clausen and Rastogi 1977). Tremors in the hands were reported by 11% of the mechanics. However, as indicated above under Cardiovascular Effects, interpretation of these results is severely limited because of the absence of a comparison with a control population, the likelihood of dermal as well as inhalation exposures, the high probability of exposure to other causative substances, and the failure to consider other confounding factors.

Exposure of rats (head-only) to an aerosol of filtered used mineral-based crankcase oil (concentration not specified) for 7 hours resulted in no adverse effects on behavior during a 40-day postexposure observation period (DOT 1983). Similarly, no adverse effects were observed in chickens during or following a 7-hour exposure to an aerosol of used mineral-based crankcase oil (concentration not specified). The chickens were placed in either head-only exposure chambers or given whole body exposures. The motor oil tested was obtained from a turboprop aircraft (used for 165 hours). The study was limited as the actual dose in the breathing space and particle size was not measured, and only a small number of animals were tested. No control group was used. (DOT 1983).

No studies were located regarding the following health effects in humans or animals after inhalation exposure to used mineral-based crankcase oil.

2.2.1.5 Reproductive Effects

2.2.1.6 Developmental Effects

2.2.1.7 Genotoxic Effects

Genotoxicity studies are discussed in Section 2.5.

2.2.1.8 Cancer

An epidemiological study of the incidence of renal pelvic and bladder cancer among workers in Sweden between 1961 and 1979 showed no significant increases in relative risk among workers exposed to motor oil or oil mists (excluding mists from cutting oils or fluids) (Steineck et al. 1989). This study was limited as the time, dose, and route of exposure were not provided. Also confounding factors were not separated out, the substances that subjects were exposed to were not clearly defined, and the job descriptions of the workers may have been erroneous. No studies were located regarding the incidence of other cancer types among humans exposed by inhalation to used mineral-based crankcase oil.

No studies were located regarding cancer in animals after inhalation exposure to used mineral-based crankcase oil.

2.2.2 Oral Exposure

2.2.2.1 Death

No studies were located regarding death in humans after oral exposure to used mineral-based crankcase oil.

No deaths were observed in rats for up to 14 days after ingestion of 22,500 mg/kg of used mineral-based crankcase oil (API 1980b; Beck et al. 1984; Vernot et al. 1990). However, increased mortality was observed among cattle believed to have ingested discarded used mineral-based crankcase oil (Osweiler et al. 1973). Tissue lead levels were elevated in the cattle, and toxic symptoms observed in the affected cattle were attributed to ingestion of lead contained in the used oil. Fatalities among

cattle ingesting used mineral-based crankcase oil have also been attributed to molybdenum contained in the used oil (Sas 1989). It was suggested that the ingested molybdenum caused death as a result of central nervous system hypoxia.

2.2.2.2 Systemic Effects

No studies were located regarding respiratory, cardiovascular, musculoskeletal, renal, or ocular effects in humans or animals after oral exposure to used mineral-based crankcase oil. The systemic effects observed after oral exposure are discussed below. The highest NOAEL and LOAEL values for systemic effects in rats after an acute-duration oral exposure to used mineral-based crankcase oil are recorded in Table 2-2 and plotted in Figure 2-2.

Gastrointestinal Effects. No studies were located regarding gastrointestinal effects in humans after oral exposure to used mineral-based crankcase oil.

No effects were observed in rats for up to 14 days after ingestion of 22,500 mg/kg of used mineral-based crankcase oil, while a single oral dose of 9,000 mg/kg of used mineral-based crankcase oil resulted in oily diarrhea in exposed rats (API 1980b; Beck et al. 1984; Vemot et al. 1990). Diarrhea was also observed in some cattle that grazed for at least 2 weeks in a pasture contaminated with used mineral-based crankcase oil (Sas 1989). This effect is not unexpected because medicinal-grade mineral oil is used as a cathartic (Fingl 1980); however, other chemicals present in used mineral-based crankcase oil may have contributed to the laxative effect.

Hematological Effects. No studies were located regarding hematological effects in humans after oral exposure to used mineral-based crankcase oil.

Anemia was observed in cattle that had ingested an unknown amount of used mineral-based crankcase oil while grazing in a pasture (Sas 1989). The used oil had molybdenum bisulfide present as an additive. The anemia was attributed to increased molybdenum intake and molybdenum-induced copper deficiency. No effects on hematocrit or hemoglobin concentration were observed in ducks and pheasants given diets containing up to 4.5% used mineral-based crankcase oil (Eastin et al. 1983). However, δ-ALAD activity was significantly decreased in the ducks and pheasants in a dose-related manner at all dietary concentrations (0.5%, 1.5%, and 4.5%) (ANOVA and Tukey's HSD test,

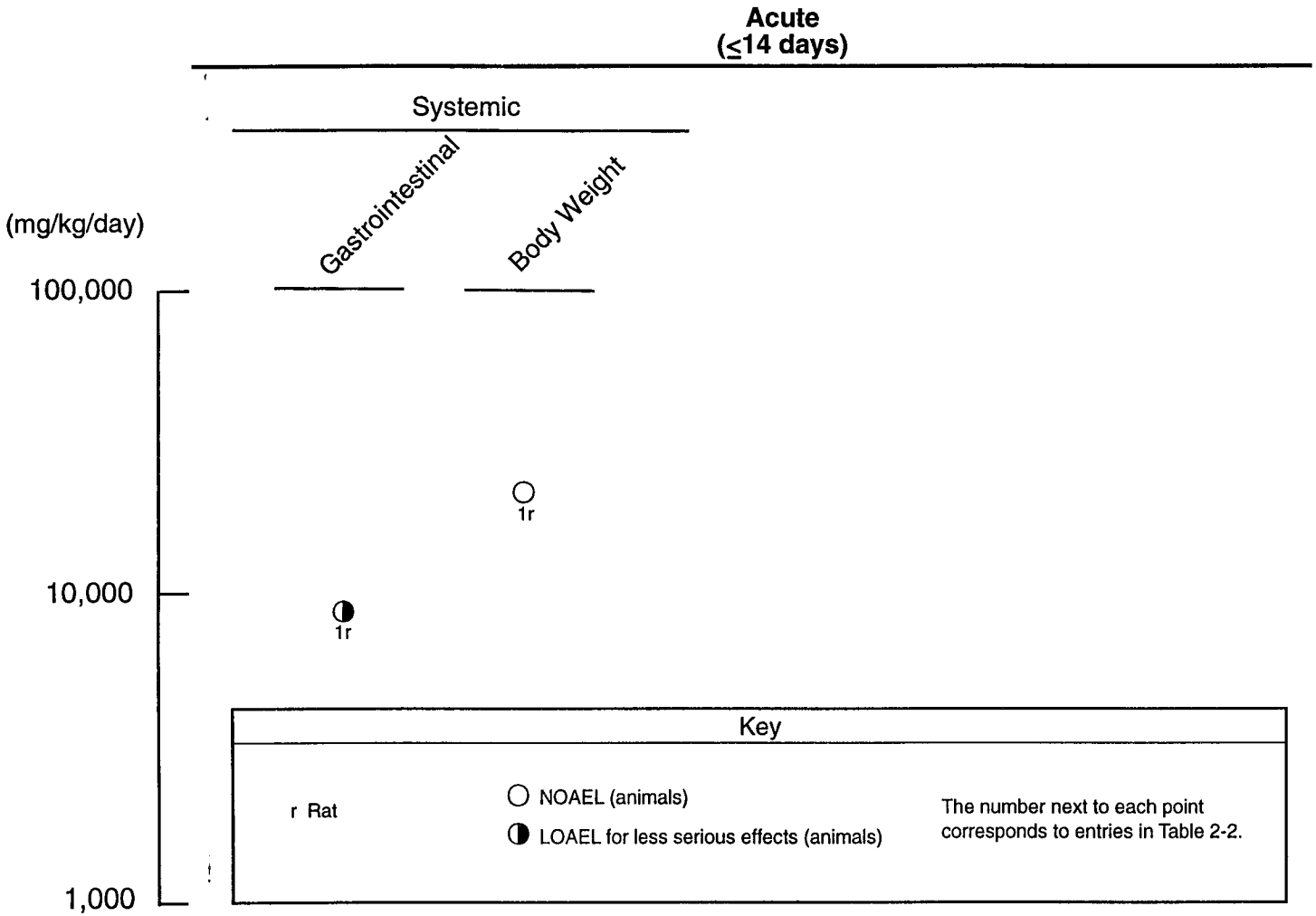
TABLE 2-2. Levels of Significant Exposure to Used Mineral-based Crankcase Oil - Oral

Key [a] to figure	Species (strain)	Exposure duration/ frequency (specific route)	System	NOAEL (mg/kg/day)	LOAEL (effect)			Reference
					Less serious (mg/kg/day)		Serious (mg/kg/day)	
ACUTE EXPOSURE								
Systemic								
1	Rat (Sprague Dawley)	once (G)	Gastro		9000	(oily diarrhea)		API 1980; Beck et al. 1984; Vernot et al. 1990
			Other	22500				

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

(G) = gavage; Gastro = gastrointestinal; LOAEL = lowest-observed-adverse-effect level; NOAEL = no-observed-adverse-effect level

Figure 2-2. Levels of Significant Exposure to Used Mineral-based Crankcase Oil - Oral



p <0.05). The decrease in δ-ALAD activity suggests an effect on heme synthesis, but in the absence of effects on hemoglobin concentration or hematocrit, the toxicological significance of this change is unclear. δ-ALAD is one of the most sensitive and one of the first enzymes in the pathway for heme synthesis which is inhibited by lead. In the absence of effects on hemoglobin and hematocrit, it serves as an early biological indicator of subclinical lead poisoning in humans and birds (Goyer 1996).

Hepatic Effects. No studies were located regarding hepatic effects in humans after oral exposure to used mineral-based crankcase oil.

The only information located regarding hepatic effects after oral exposure to used mineral-based crankcase oil was a study that showed a significant increase in serum aspartate aminotransferase activity in ducks after 3 weeks of dietary exposure at a dietary concentration of 4.5% (Eastin et al. 1983) (ANOVA and Tukey's HSD test, p <0.05). No effects were observed on the serum levels of alanine aminotransferase, uric acid, glucose, triglycerides, total protein, and cholesterol after l-3 weeks of dietary exposure of ducks and pheasants to dietary concentrations of used mineral-based crankcase oil as high as 4.5%.

Dermal Effects. No studies were located regarding dermal effects in humans after oral exposure to used mineral-based crankcase oil.

Cattle that ingested used mineral-based crankcase oil in a pasture for at least 2 weeks had decreased pigmentation in the hair around the eyes (Sas 1989). This effect may have been the result of a molybdenum-induced copper deficiency. No other information was located regarding dermal effects in animals after oral exposure to used mineral-based crankcase oil.

Body Weight Effects. No studies were located regarding body weight effects in humans after oral exposure to used mineral-based crankcase oil.

No effects on body weight gain or growth were observed in rats that received single doses of used mineral-based crankcase oil as high as 22,500 mg/kg (API 1980b; Beck et al. 1984; Vernot et al. 1990) or in ducks or pheasants that ingested an unspecified amount of used mineral-based crankcase oil in their diets for up to 3 weeks (Eastin et al. 1983). The lack of effects on body weight gain or

growth probably indicates that higher doses of used crankcase oil in the diet would have been tolerated.

2.2.2.3 Immunological and Lymphoreticular Effects

No studies were located regarding immunological or lymphoreticular effects in humans or animals after oral exposure to used mineral-based crankcase oil.

2.2.2.4 Neurological Effects

No studies were located regarding neurological effects in humans after oral exposure to used mineral-based crankcase oil.

Cattle that ingested an unspecified amount of used mineral-based crankcase oil as a result of grazing in contaminated pastures for approximately one year have shown a number of neurological disorders (Osweiler et al. 1973; Sas 1989). Blindness, muscle twitching, hyperirritability, depression, and convulsions were observed and attributed to lead poisoning (Osweiler et al. 1973). Muscle tremors and weakness were observed in another study (Sas 1989). These effects may be associated with molybdenum-induced copper deficiency.

No studies were located regarding the following health effects in humans or animals after oral exposure to used mineral-based crankcase oil.

2.2.2.5 Reproductive Effects

2.2.2.6 Developmental Effects

2.2.2.7 Genotoxic Effects

Genotoxicity studies are discussed in Section 2.5.

2.2.2.8 Cancer

No studies were located regarding cancer in humans or animals after oral exposure to used mineral-based crankcase oil.

2.2.3 Dermal Exposure

2.2.3.1 Death

No studies were located regarding death in humans after dermal exposure to used mineral-based crankcase oil.

Contact of 5 mL/kg (4,500 mg/kg) of used mineral-based crankcase oil with either abraded or intact sites on the backs of rabbits (approximately 30% of the total surface area of the rabbits) for 24 hours resulted in no mortality (API 1980b; Beck et al. 1984; Vemot et al. 1990). No increase in death rate was reported in a skin-painting study in mice in which 1,667 mg/kg was applied to the backs of male C3WHeJ mice (precise area of application was not specified) twice per week for 2 years (API 1983). Negative controls included a group of mice that were untreated and one group that was treated with toluene. Positive controls included a group that was treated with 0.05% or 0.15% benzo(a)pyrene (a known carcinogen) in toluene. Survival was slightly decreased in mice treated with used motor oil at weeks 91 (22% survival versus 36% in controls) and 104 (10% versus 22% in controls). The study is limited as only one dose was studied and only males were used (API 1983).

2.2.3.2 Systemic Effects

No studies were located regarding musculoskeletal effects in humans or animals after dermal exposure to used mineral-based crankcase oil. The systemic effects observed after dermal exposure are discussed below: The highest NOAEL and LOAEL values for each end point in each species and duration category after dermal exposure to used mineral-based crankcase oil are recorded in Table 2-3.

TABLE 2-3. Levels of Significant Exposure to Used Mineral-based Crankcase Oil - Dermal

Species (strain)	Exposure duration/ frequency (specific route)	System	NOAEL	LOAEL (effect) Less serious		Serious		Reference
ACUTE EXPOSURE								
Systemic								
Rabbit (New Zealand White)	2 wks 5d/wk 24hr/d	Hepatic	8 mL/kg					API 1980; Beck et al. 1984
		Renal	8 mL/kg					
		Dermal				8 mL/kg	(acanthosis, chronic inflammation, dermal congestion, edema, hyperkeratosis, hair loss)	
		Other		8 mL/kg	(decreased food consumption, 18% weight loss)			
Rabbit (New Zealand White)	24 hr	Dermal		5 mL	(slight erythema)			API 1980; Beck et al. 1984; Vernot et al. 1990
Rabbit (New Zealand White)	24 hr	Dermal		0.5 mL	(very slight erythema)			API 1980; Beck et al. 1984; Vernot et al. 1990

TABLE 2-3. Levels of Significant Exposure to Used Mineral-based Crankcase Oil - Dermal (continued)

Species (strain)	Exposure duration/ frequency (specific route)	System	NOAEL (mg/kg/day)	LOAEL (effect) Less serious (mg/kg/day)		Serious (mg/kg/day)		Reference
Rabbit (New Zealand White)	once	Ocular		0.1 mL				API 1980; Beck et al. 1984; Vernot et al. 1990
INTERMEDIATE EXPOSURE								
Systemic								
Gn pig (albino)	3.5 wks 3x/wk 6hr/d	Dermal		0.5 mL	(none-to-slight erythema)			Vernot et al. 1990
Immuno/Lymphor								
Gn pig (albino)	3.5 wks 3x/wk 6hr/d		0.5 mL					API 1980; Beck et al. 1984
CHRONIC EXPOSURE								
Systemic								
Mouse (C3H/HeJ)	104 wks 2d/wk	Dermal				1667 mg/kg	(acanthosis, hyperkeratosis, and fibrosis of skin)	API 1983
		Body wt	1667 mg/kg					

TABLE 2-3. Levels of Significant Exposure to Used Mineral-based Crankcase Oil - Dermal (continued)

Species (strain)	Exposure duration/ frequency (specific route)	System	NOAEL (mg/kg/day)	LOAEL (effect)			Reference
				Less serious (mg/kg/day)	Serious (mg/kg/day)		
Cancer							
Mouse (C3H/HeJ)	104 wks 2d/wk				1667 mg/kg	(CEL - dermal papillomas, keratocanthomas, squamous cell carcinomas, and hemangiosarcomas)	API 1983
Mouse (CFLP)	104 wks 2x/wk				60 mg/kg	(CEL - dermal papillomas and carcinomas)	Grimmer et al. 1982a, 1982b, 1983
Mouse (C3H/HeJ)	approx 79 wks 3x/wk				900 mg/kg	(CEL - dermal papillomas and carcinomas)	McKee and Plutnick 1989
Mouse (C3H/HeJ)	104 wk 2x/wk				1667 mg/kg	(CEL - skin)	Schreiner and Mackerer 1982; University of Cincinnati 1980

approx = approximately; Body wt = body weight; CEL = cancer effect level; d = day(s); Gn pig = guinea pig; hr = hour(s); Immuno/Lymphor = Immunological and Lymphoreticular; LOAEL = lowest-observed-adverse-effect level; NOAEL = no-observed-adverse-effect level; wk(s) = week(s); x = time(s)

Respiratory Effects. Volunteers, whose eyes were exposed to aerosols of used mineral-based crankcase oil at concentrations ranging from 42 to 84 mg/m^3 in specially designed goggles, reported chest tightness (Dautrebande et al. 1951). The method of exposure used in this study was designed to limit inhalation exposures, but it is unlikely that inhalation exposure was completely prevented. Thus, it is unclear whether the chest tightness represented an effect stemming directly from the ocular exposure or whether inhalation of the test material contributed to the feeling of chest tightness. Furthermore, the data were collected over 40 years ago, and formulations have changed considerably since that time.

No increase in the incidence of pneumonia was noted in mice that received dermal doses of 1,667 mg/kg/day, applied to their backs twice a week, for 2 years. However, this study is limited in that only half of the animals treated (n=50) were examined histologically (API 1983).

Cardiovascular Effects. High blood pressure was noted in 37% of the mechanics, 7% of the apprentice mechanics, and 18% of the miscellaneous workers examined in a study of workers from 10 auto shops in Denmark (Clausen and Rastogi 1977). However, interpretation of these results is severely limited because of the absence of comparison with a control population, the likelihood of inhalation as well as dermal exposures, the high probability of exposure to other causative substances, and the failure to consider other confounding factors such as diseases unrelated to exposure.

No studies were located regarding cardiovascular effects in animals after derrnal exposure to used mineral-based crankcase oil.

Gastrointestinal Effects. The incidence of gastrointestinal effects such as stomach pain, constipation, or anorexia was not increased in a group of mechanics and apprentice mechanics from auto shops in Denmark (Clausen and Rastogi 1977). However, interpretation of these results is severely limited-because of the absence of comparison with a control population, the likelihood of inhalation as well as dermal exposures, the high probability of exposure to other causative substances, and the failure to consider confounding factors.

No studies were located regarding gastrointestinal effects in animals after dermal exposure to used mineral-based crankcase oil.

Hematological Effects. Lower than normal hematocrit and mean corpuscular hemoglobin were observed in a number of mechanics and apprentice mechanics in a study of workers from 10 auto shops in Denmark (Clausen and Rastogi 1977). Elevated blood lead levels were observed in 52% of the mechanics when compared to controls. In several cases, the elevated lead levels were correlated with the decreases in hematocrit and mean corpuscular hemoglobin, suggesting that the effects may have been related to blood lead levels. High blood lead levels have been associated with anemia (see the ATSDR profile on lead [ATSDR 1993b]). Used mineral-based crankcase oil was determined to be one source of lead exposure, but other sources of lead exposure (exhaust gas, gasoline, gear oil) may have been contributors.

No studies were located regarding hematological effects in animals after dermal exposure to used mineral-based crankcase oil.

Hepatic Effects. Increased serum levels of bilirubin and thymol reaction (indicative of increased serum alkaline phosphatase) were observed in 14% of the mechanics examined in a study of workers from 10 auto shops in Denmark (Clausen and Rastogi 1977). Elevated serum alanine and aspartate aminotransferase and lactate dehydrogenase were also found in 11% of the mechanics. However, interpretation of these results is limited because of the absence of comparison with a control population, the likelihood of inhalation as well as dermal exposures, the high probability of exposure to other causative substances, and the failure to consider other confounding factors (such as diseases unrelated to employment).

Histopathological analysis of livers of rabbits dermally exposed to 8 mL/kg of used mineral-based crankcase oil on a 4-inch-square area of their backs for 5 days/week for 2 weeks showed no marked increase in adverse effects on the liver (API 1980b; Beck et al. 1984; Vemot et al. 1990). No studies regarding the hepatic effects of longer-term dermal exposure of animals to used mineral-based crankcase oil were located.

Renal Effects. No studies were located regarding renal effects in humans after dermal exposure to used mineral-based crankcase oil.

Histopathological analyses of kidneys and urinary bladders from rabbits given dermal applications of 8 mL/kg of used mineral-based crankcase oil on a 4-inch-square area of their backs 5 days/week for 2 weeks showed no adverse effects (API 1980b; Beck et al. 1984; Vernot et al. 1990). No increase in the incidence of renal necrosis or chronic interstitial nephritis was observed in mice receiving dermal doses of 1,667 mg/kg/day on their backs, twice a week, for 2 years (API 1983). However, this study is limited in that only half of the animals treated were examined histologically.

Dermal Effects. Rashes on the hands or arms were reported by 29% of the mechanics in a study of workers from 10 auto shops in Denmark (Clausen and Rastogi 1977). However, interpretation of this result is difficult because other chemical exposures may have contributed to the effects observed.

A single 5-mL dose of used mineral-based crankcase oil maintained in contact with abraded and intact skin on the backs of rabbits (approximately 30% of the total surface area) for 24 hours caused slight erythema (API 1980b; Beck et al. 1984; Vemot et al. 1990). At 0.5 mL, the amount of erythema noted in the l-inch-square area of application after a 24-hour exposure was very slight, and no redness was observed on either intact or abraded sites on rabbits by 72 hours postexposure. Daily application of 8 mL/kg of used mineral-based crankcase oil to a 4-inch-square area on the backs of rabbits for 24 hours/day, 5 days/week, for 2 weeks resulted in irritation at the application site and hair loss on adjacent tissues. Histopathological examination of the skin showed acanthosis, chronic inflammation, dermal congestion, edema, and hyperkeratosis (API 1980b; Beck et al. 1984). In contrast, daily application of 0.5 mL of used mineral-based crankcase oil to a l-inch-square area on the back of guinea pigs for 6 hours/day, 3 days/week, for 3.5 weeks resulted in only none-to-slight erythema (API 1980b; Beck et al. 1984; Vemot et al. 1990). Dermal application of 1,667 mg/kg (50 mg applied to an unspecified area on the backs of each animal) two times a week for 104 weeks resulted in a slightly increased incidence of acanthosis and hyperkeratosis and slightly increased severity of fibrosis in C3H/HeJ mice (API 1983).

Dermal exposure of rats to 125-167 mg/kg/day of used mineral-based crankcase oil for 3 days resulted in increased activity of microsomal enzyme activity of the skin (Rahimtula et al. 1982). Similar

effects were observed following a single dose of an unspecified amount of used mineral-based crankcase oil (Rahimtula et al. 1984). This is considered to be an adaptive effect.

Ocular Effects. Mild-to-moderate eye irritation was reported by volunteers exposed to aerosols of used mineral-based crankcase oil (Dautrebande and Capps 1950; Dautrebande et al. 1951). Although considerable variability in individual sensitivities was observed, mild eye irritation was observed in at least one of the subjects at exposure levels as low as 18.9 mg/m^3. It is likely that the eye irritation was due to direct contact of the aerosol with the eye.

Instillation of 0.1 mL of used mineral-based crankcase oil into the eyes of rabbits resulted in slight swelling and conjunctival redness in the eye of only one of the six rabbits tested (API 1980b; Beck et al. 1984; Vernot et al. 1990). Thus, the oil was classified as nonirritating.

Body Weight Effects. No studies were located regarding body weight effects in humans after dermal exposure to used mineral-based crankcase oil.

Exposure of a 4-inch-square area on the backs of rabbits to 8 mL/kg of used mineral-based crankcase oil for 24 hours/day, 5 days/week, for 2 weeks resulted in marked decrease in food consumption and weight loss (API 1980b; Beck et al. 1984; Vernot et al. 1990). The biological basis for the decreased food consumption was not identified. In contrast, application of a somewhat lower dose (1,667 mg/kg/day) to an unspecified area on the backs of mice 2 days/week for 2 years resulted in no effect on body weight gain (API 1983).

2.2.3.3 Immunological and Lymphoreticular Effects

No studies were located regarding immunological or lymphoreticular effects in humans after dermal exposure to used mineral-based crankcase oil.

No sensitization was observed in guinea pigs when challenged 2 weeks after a 3 day/week, 3.5-week exposure to 0.5 mL/day (API 1980b; Beck et al. 1984; Vernot et al. 1990). However, the positive control group also did not exhibit sensitization. Other studies examining the immunological effects of used mineral-based crankcase oil after dermal exposure were not located. The NOAEL for

sensitization after acute-duration dermal exposure to used mineral-based crankcase oil is recorded in Table 2-3.

2.2.3.4 Neurological Effects

Examination of workers from 10 auto shops in Denmark revealed headaches in the morning among 14% and headaches in the evening among 20% of the mechanics (Clausen and Rastogi 1977). Tremors of the hands were reported by 11% of the mechanics. It is unclear whether the incidence of these findings are elevated relative to those in a control population. Also, exposures to substances other than used mineral-based crankcase oil, as well as other confounding factors (such as lifestyle or preexisting diseases) may have contributed to the effects observed.

No studies were located regarding neurological effects in animals after dermal exposure to used mineral-based crankcase oil.

2.2.3.5 Reproductive Effects

No studies were located regarding reproductive effects in humans or animals after dermal exposure to used mineral-based crankcase oil.

2.2.3.6 Developmental Effects

No studies were located regarding developmental effects in humans or animals after dermal exposure to used mineral-based crankcase oil.

2.2.3.7 Genotoxic Effects

Genotoxic carcinogens exert their carcinogenic effects by damaging DNA. There are two types of genotoxic carcinogens: (1) direct acting carcinogens (or ultimate carcinogens), which bind directly to DNA and other cellular macromolecules, and (2) precarcinogens (or procarcinogens), which have to be bioactivated either directly or indirectly to ultimate carcinogens. These ultimate carcinogens, which are electrophilic in nature, are highly reactive. They bind directly to DNA to form DNA adducts, producing tumors. Polycyclic aromatic compounds (PAHs), for example, benzo[a]pyrene, are among

the various chemical carcinogens found in this second category. Bioactivation occurs mainly in the liver but also in the lung and skin. Most of the reactions in this process, which converts certain chemically stable compounds to chemically reactive metabolites, are catalyzed by the cytochrome P450 (CYP450)-dependent monooxygenase systems. Most aromatic compounds like the PAHs are converted to epoxides, which are extremely reactive metabolites, by the CYP450 system. These epoxides bind to DNA to form DNA adducts, resulting in necrosis and/or cancer (Lu 1991).

In humans, absorption of PAHs after therapeutic or occupational exposure occurs mainly through the skin. Inhalation exposure is less likely. No studies were located regarding mutagenic or clastogenic effects in humans following dermal exposure to used mineral-based crankcase oil. However, dermal application of used mineral-based crankcase oil to the shaved backs of mice produced DNA adducts in the skin and the lungs, indicating that used mineral-based crankcase oil was genotoxic (Carmichael et al. 1990; Schoket et al. 1989). A single application of about 50 mg of used gasoline or diesel engine crankcase oil to the shaved backs of male Parkes mice produced adducts in DNA isolated from the skin 24 hours after treatment. Analysis of the adducts by thin-layer chromatography showed that the number and variety of adducts found in the skin with oil from gasoline-powered engines was greater than that observed with either unused oil or oil from diesel engines. No adducts were observed in the lung following a single application, but lung adducts were observed after treatment on 4 consecutive days (Schoket et al. 1989). Similar to the effects in the skin, oil from gasoline-powered engines produced a response in the lungs, but the number and variety of adducts found in the lung were less than in the skin. The levels of skin ($p < 0.05$) and lung DNA ($p < 0.01$) adducts produced by gasoline engine oil correlated with an index of oil use (calculated as the product of miles since the last oil change and total engine mileage). The total PAH concentration in gasoline engine oil proved to be a good predictor of adduct levels. When adduct levels were compared with the concentrations of individual PAHs in the oil, the best correlation was with benz(a)anthracene; skin DNA, $\delta = 0.91$ ($p < 0.0l$) and lung DNA, $\delta = 0.96$ ($p < 0.01$). The correlation with benzo(a)pyrene was poor, and was not statistically significant (Carmichael et al. 1990).

To identify further the PAHs responsible for the major adducts, Carmichael et al. (1992) fractionated one of the samples of used gasoline engine oil studied by Carmichael et al. (1990). They then painted the fractions on the shaved backs of male Parkes mice (four/group), and identified the adducts formed in skin DNA. In addition, individual PAHs dissolved in unused motor oil at concentrations similar to those detected in the unfractionated used oil were evaluated in this assay. Based on this analysis, the

major adducts produced in mouse skin by used gasoline engine oil were attributed to reactive metabolites of the PAHs benzo(*b*)naphtho(1,2-*d*)thiophene, benzo(*c*)phenanthrene, benzo(*g,h,i*)-fluoranthene, chrysene, benzo(*a*)pyrene, and benzo(*g,h,i*)perylene, several of which are known genotoxins and carcinogens (ATSDR 1990c). The study authors did not provide any explanation for the different PAHs implicated in the two studies. However, the concentrations of benzo(*c*)phenanthrene, benzo(*g,h,i*)fluoranthene and chrysene were significantly correlated with adduct levels in the study by Carmichael et al. (1990).

Other genotoxicity studies are discussed in Section 2.5.

2.2.3.8 Cancer

An epidemiological study of the incidence of renal pelvic and bladder cancer among workers in Sweden between 1961 and 1979 showed no significant increases in relative risk among workers exposed to motor oil or oil mists other than mists from cutting oils or fluids (Steineck et al. 1989). This study was limited as the time, dose, and route of exposure were not provided. Also, confounding factors were not separated out, the substances subjects were exposed to were not clearly defined, and the job descriptions of the workers may have been erroneous. No studies were located regarding the incidence of other types of cancer among humans dermally exposed to used mineral-based crankcase oil.

Several studies have examined the dermal carcinogenicity of used mineral-based crankcase oil in mice (API 1983; Grimmer et al. 1982a, 1982b, 1983; McKee and Plutnick 1989). These studies have shown that the incidence of dermal papillomas and carcinomas among male C3H/HEJ mice (API 1983; McKee and Plutnick 1989) and female CFLP mice (Grimmer et al. 1982a, 1982b, 1983) are increased after chronic-duration dermal exposure to used mineral-based crankcase oil from gasoline-powered cars. The greatest tumor incidence was observed in mice exposed to oil from cars driven the longest distances prior to removing the oil (McKee and Plutnick 1989), and no tumors were observed in mice exposed to unused motor oil (API 1983; McKee and Plutnick 1989), indicating that carcinogens accumulated in the oil during its use. The increase in carcinogenicity was attributed to accumulation of PAHs in the oils because the tumor incidence correlated with PAH content of the oil (McKee and Plutnick 1989). Fractionation of the oil showed tumor induction only with the fraction containing PAHs with more than three rings (Grimmer et al. 1982a, 1982b, 1983). In contrast, a study by Ingram

et al. (1994) of the main mutagenic components of a carcinogenic oil by fractionation and testing in the modified Ames assay showed that the mutagenicity of PAHs with l-3 rings was greater than that of PAHs with 4-6 rings. The authors concluded that the 4-6 ring PAHs were not the main mutagenic components of the oil examined, and therefore the mutagenic components may be different from the carcinogenic components. In contrast to used mineral-based crankcase oil from gasoline-powered automobiles, used mineral-based crankcase oil from diesel-powered automobiles showed no increase in tumor incidence, even when the diesel-powered automobiles were driven extremely long distances prior to removal of the oil (McKee and Plutnick 1989). The CELs for dermal tumors observed in mice after chronic-duration exposure to used mineral-based crankcase oil are recorded in Table 2-3.

In the past, increased mutagenicity and cancer mortality have been associated with exposure to the PAHs present in complex mixtures such as skin oils of roofing workers (Wolff et al. 1982), used metalworking cutting oils or mineral oils (Apostoli et al. 1993; Bingham 1988; Cruickshank and Squire 1950; Eyres 1981; Roy et al. 1988), diesel soot particles (Vogl and Elstner 1989), coal tar (Clonfero et al. 1986; Jongeneelen et al. 1988c; Wheeler et al. 1981), petroleum products (Witschi et al. 1987), cigarette smoke, diesel exhausts, and air pollution (Bond et al. 1988; Gallagher et al 1990; Iyer et al. 1990; Schenker et al. 1984; Siemiatycki et al. 1988; Woskie et al. 1988). The carcinogenic potential of these complex mixtures is related to their PAH content. Benzo[a]pyrene (BaP), a PAH, is mainly associated with the carcinogenicity of complex mixtures. However, a study by Warshawsky et al. (1993) showed that BaP alone was not responsible for the observed potency of these mixtures. The presence of other low carcinogenic compounds, e.g., methylbenz[a]anthracenes and straight chain aliphatics, can also contribute to carcinogenicity. It is possible that these compounds may play a role in the formation of tumors following exposure to used mineral-based crankcase oils.

2.3 TOXICOKINETICS

Used mineral-based crankcase oil is a complex mixture of PAHs and metals. Consequently, it is difficult to determine its toxicokinetics because of the extensive variability in its composition and also because of a lack of definitive data for either humans or animals. However, a brief discussion of the toxicokinetics of the toxic components of used crankcase oil e.g., heavy metals such as lead, cadmium, chromium amongst others, and PAHs is provided. Additional information regarding the toxicokinetics of individual components of used mineral-based crankcase oil can be found in ATSDR profiles for the components.

No human studies were located regarding the toxicokinetics of used mineral-based crankcase oil. Only a very limited number of animal studies were located. Data based on studies of poisoning in cattle indicate that lead and other metals in used mineral-based crankcase oil may be absorbed and distributed to various tissues following oral exposure and that the feces is a significant path of excretion (Blakely and Brockman 1976; Osweiler et al. 1973). Limited rodent data indicate that, following oral exposure, used mineral-based crankcase oil is absorbed and excreted in the feces. PAHs found in used mineral-based crankcase oil are absorbed and distributed to various tissues as indicated by the presence of PAH-DNA adducts in the skin and lungs of male mice that were dermally exposed to used mineral-based crankcase oil. As PAHs are lipophilic compounds, they are mainly stored in adipose tissue and secreted in milk (Lu 1991). In both humans and animals, lead is stored in the skeletal and soft tissue pool, cadmium is accumulated in the kidneys, while molybdenum is stored somewhat in the liver and rapidly excreted in the urine and in the bile. The half-life of cadmium is 30 years, and hence, is excreted very slowly. The kidney is the primary target organ of cadmium. It damages the renal proximal tubules, forming lesions and causing urinary excretion of small-molecule proteins, amino acids and glucose. Chromium also damages the proximal tubules (Lu 1991). No other information regarding the toxicokinetics of used mineral-based crankcase oil was located.

2.3.1 Absorption

2.3.1.1 Inhalation Exposure

No studies were located regarding the absorption of used mineral-based crankcase oil in humans or animals after inhalation. Insufficient information was available from inhalation toxicity studies to determine whether absorption through the respiratory tract may have occurred.

2.3.1.2 Oral Exposure

No studies were located regarding the absorption of used mineral-based crankcase oil in-humans after oral exposure.

The few animal studies available indicate that lead and other metals in used mineral-based crankcase oil may be absorbed following ingestion (Blakley and Brockman 1976; Osweiler et al. 1973; Sas 1989). Ingestion of used mineral-based crankcase oil was determined to be the source of elevated

tissue lead levels in 22 cases of lead toxicosis in cattle (Blakley and Brockman 1976). Blood lead levels (0.98 ppm) from cattle with lead poisoning from all sources (oil, batteries, paint, chemical, and unknown) averaged 13-fold higher than in controls. A mean blood lead level of 0.78 ppm was measured in another study of cattle (80 cases) with lead poisoning (Osweiler et al. 1973). The source of the lead was determined to be ingestion of used mineral-based crankcase oil in 29% of the cases. In neither study was information provided on the dose, duration of exposure, or absorption rates. Molybdenum levels were elevated by two orders of magnitude in the livers and kidneys of cattle that ingested used mineral-based crankcase oil known to contain molybdenum bisulfide as an additive (Sas 1989).

2.3.1.3 Dermal Exposure

No studies were located regarding the absorption of used mineral-based crankcase oil in humans after dermal exposure.

The few studies available indicate that PAHs found in used mineral-based crankcase oil can penetrate the outer layer of skin as shown by the finding of PAH-DNA adducts in the skin of male mice receiving a single dermal application or four daily applications of used mineral-based crankcase oil (≈40-50 mg) (Carmichael et al. 1990; Schoket et al. 1989). PAH-DNA adducts were also found in the lungs, suggesting that PAHs found in used mineral-based crankcase oil or metabolites may be systemically available following dermal application (Carmichael et al. 1990; Schoket et al. 1989).

2.3.2 Distribution

Petroleum hydrocarbons are lipophilic. Therefore, they would tend to distribute in fatty tissue (Rozman and Klaassen 1996). Among the metals found in used mineral-based crankcase oil, lead is stored in skeletal tissue, cadmium accumulates in the kidneys, and molybdenum tends to accumulate in the liver (Goyer-1996). Specific data regarding the distribution of components of used mineral-based crankcase oil following different routes of exposure can be found in compound specific ATSDR Profiles.

2.3.2.1 Inhalation Exposure

No studies were located regarding the distribution of used mineral-based crankcase oil in humans or animals after inhalation exposure.

2.3.2.2 Oral Exposure

No studies were located regarding the distribution of used mineral-based crankcase oil in humans after oral exposure.

Studies of poisoning in cattle indicate that metals found in used mineral-based crankcase oil are distributed to various tissues (Blakley and Brockman 1976; Osweiler et al. 1973; Sas 1989). In studies examining the distribution of lead, the kidneys appear to be the major site of lead accumulation (Blakley and Brockman 1976; Osweiler et al. 1973). Lead levels were elevated ≈100-fold, 24-fold, 4-fold, and 460-fold in the kidney, liver, brain, and rumen contents, respectively. The respective values were statistically different ($p<0.05$) for all tissues except the brain. The rumen contents exhibited a high degree of variability. The source of the lead was determined to be ingestion of used mineral-based crankcase oil in 22 cases (Blakley and Brockman 1976). In another study of cattle (80 cases) with lead poisoning, mean tissue levels of lead were 29.7 and 57.7 ppm in the liver and kidney, respectively (Osweiler et al. 1973). The source of the lead was determined to be ingestion of used mineral-based crankcase oil in 29% of the cases. Molybdenum concentrations in the livers and kidneys of cows that ingested used mineral-based crankcase oil known to contain molybdenum bisulfide as an additive were found to exceed normal physiological concentrations by two orders of magnitude (Sas 1989). Very limited information on the dose and duration of exposure was provided in these studies.

2.3.2.3 Dermal Exposure

No data were located regarding the distribution of used mineral-based crankcase oil in humans after dermal exposure.

Animal data indicate that PAHs found in used mineral-based crankcase oil are found in tissues other than the skin of dermally exposed mice (Carmichael et al. 1990; Schoket et al. 1989). PAH-DNA

adducts were found in both the skin and lungs of male mice receiving either one or four daily applications of used mineral-based crankcase oil (≈1,333-1,780 mg/kg) (Carmichael et al. 1990; Schoket et al. 1989).

2.3.3 Metabolism

No studies were located regarding the metabolism of used mineral-based crankcase oil in humans or animals.

2.3.4 Excretion

2.3.4.1 Inhalation Exposure

No studies were located regarding the excretion of used mineral-based crankcase oil in humans or animals after inhalation exposure.

2.3.4.2 Oral Exposure

No studies were located regarding the excretion of used mineral-based crankcase oil in humans after oral exposure.

Very limited animal data, based on a study of lead poisoning in cattle (90 cases), indicate that the feces is a significant path of excretion (Blakley and Brockman 1976). Lead levels in the feces were >160-fold the normal level. The source of the lead was determined to be ingestion of used mineral-based crankcase oil in 22 cases (Blakley and Brockman 1976). No data on dose, duration of exposure, or excretion rates were provided. Rats given a single oral dose (9,000 or 22,500 mg/kg) of used mineral-based crankcase oil excreted the oil in the feces as evidenced by dose-related increases in oily diarrhea (API 1980b; Beck et al. 1984; Vernot et al. 1990). However, it is unclear whether the oil excreted in the feces represented mainly unabsorbed oil.

2.3.4.3 Dermal Exposure

No studies were located regarding the excretion of used mineral-based crankcase oil in humans or animals after dermal exposure.

2.4 MECHANISMS OF ACTION

Although the toxicity of used mineral-based crankcase oil has not been fully investigated, the toxicity of used oil is attributed to additives and contaminants in the oil such as PAHs, lead, molybdenum, chromium, zinc, cadmium, copper, and silicon. New (unused) mineral-based crankcase oil and refinery streams are, by comparison, relatively nontoxic (API 1979, 1980a, 1983; Beck et al. 1984). In contrast, systemic toxicity and death in cattle that ingested used mineral-based crankcase oil had been attributed in one instance to lead accumulation in the oil (Osweiler et al. 1973), and in another to molybdenum present as an additive in the oil that was ingested (Sas 1989). The toxicity in the report by Sas (1989) was attributed to molybdenum present in the used oil. The molybdenum was suggested to cause hypochromic microcytic anemia and death as a result of molybdenum-induced copper deficiency. The molybdenum was suggested to have caused the formation of copper-thiomolybdate complexes in both the rumen and in the blood, thereby decreasing the copper available for absorption or for use by copper-requiring enzymes. The mechanism of the lead-induced toxicity was not specified in the report by Osweiler et al. (1973), but detailed information describing the fundamental mechanisms for lead-mediated effects may be found in the ATSDR profile on lead (ATSDR 1993b).

One of the target organs of lead is the hematopoietic system. It inhibits δ-ALAD, a zinc-dependent enzyme, by displacing zinc and hence, the synthesis of heme. Heme is the main component of hemoglobin (Lu 1991). The nervous system is also a target of lead. Young, developing children and unborn fetuses are extremely susceptible to lead toxicity. High blood levels of lead results in encephalopathy, ataxia, stupor, coma and convulsions, peripheral neuropathy, decreased attention span and mental retardation. Chronic lead exposure induces inclusion bodies in the nuclei ofrenal proximal tubular cells. Lead also results in renal adenocarcinoma in animals (Lu 1991). However, it should be noted that current lead levels in fuels, oils and additives are considerably lower due to stringent rules and regulations. Hence, the risk of toxic effects from lead exposure has been reduced.

Acute exposure to cadmium results in local irritation. Exposure via inhalation to cadmium results in pulmonary edema and chemical pneumonitis. Oral exposure results in nausea, vomiting and abdominal pain (Lu 1991). Cadmium is a probable human carcinogen. Like lead, cadmium also accumulates in the lysosomes of the renal proximal tubular cells as cadmium-metallothionein complex, where the cadmium complex degrades and releases Cd^{2+}. The cadmium ion (Cd^{2+}) inhibits the proteolytic enzymes in the lysosomes and damages the cell (Lu 1991). Chromium is a known human carcinogen. Occupational exposure to chromium induces lung cancer, which is thought to be caused by hexavalent chromium. Hexavalent chromium is rapidly taken up by cells and converted to trivalent chromium intracellularly. The trivalent chromium ion is extremely reactive and binds to nucleic acid, initiating the carcinogenic process. Hexavalent chromium is extremely corrosive and causes nasal and skin ulcers (Lu 1991).

PAHs are potent inducers of microsomal enzymes. Hence, they can enhance the metabolism of steroids such as estradiol and androsterone which affect the reproductive system (Lu 1991). Because of their effects on enzymes, PAHs can affect the toxicity of other chemicals and promote carcinogenesis in the liver, induce immunosuppression (depressed immune system) and adversely affect reproductive functions (Lu 1991).

The carcinogenicity of used mineral-based crankcase oil has been attributed to accumulations of PAHs, lead, molybdenum, chromium, zinc, cadmium, copper, and silicon in the oil (Grimmer et al. 1982a, 1982b, 1983; McKee and Plutnick 1989) based on the correlation of tumor incidence with the PAH content of the oil and the isolation and testing of PAH-containing fractions of the oil for carcinogenicity. The concentration of PAHs was increased in used oil, and PAHs containing more than three rings contributed to 70% of the total carcinogenicity in used oil. In several studies, benzolalpyrene present in automobile exhaust condensate, crankcase oil (used oil), and smoke condensate contributed to 10, 18, and 5-8% respectively of the total carcinogenicity observed (Grimmer et al. 1982a, 1982b, 1983; McKee and Plutnick 1989).

In contrast, a study by Ingram et al. (1994) of the main mutagenic components of a carcinogenic oil by fractionation and testing in the modified Ames assay showed that the mutagenicity of PAHs with l-3 rings was greater than that of PAHs with 4-6 rings. The authors concluded that the 4-6 ring PAHs were not the main mutagenic components of the oil examined, and therefore the mutagenic components may be different from the carcinogenic components. However, the information on the

carcinogenicity of used mineral-based crankcase oil in humans is very limited. Genotoxicity studies with used mineral-based crankcase oil support the conclusion that PAHs contained in the used oil may be responsible for the carcinogenicity that has been observed (Carmichael et al. 1990, 1991, 1992; Hermann et al. 1980b; Peake and Parker 1980; Schoket et al. 1989; Schreiner and Mackerer 1982). Further information on the mutagenicity and carcinogenicity of PAHs may be found in the ATSDR profile on PAHs (ATSDR 1990c).

2.5 RELEVANCE TO PUBLIC HEALTH

Persons in the vicinity of hazardous waste sites are likely to be exposed to used mineral-based crankcase oil primarily via skin contact or ingestion of contaminated soil. Used mineral-based crankcase oil may also be found in surface water as a result of runoff. Because of its poor volatility, inhalation exposure is unlikely unless the oil is aerosolized. Its poor solubility suggests that drinking water exposures are also unlikely. However, chemical constituents (especially metals) of used mineral-based crankcase oil may be released from the oil into the environment, and significant exposure to toxic constituents may occur in the drinking water or as the result of bioaccumulation in foods.

Relatively little is known about the toxicity of used mineral-based crankcase oil. Few studies have been reported which examined the toxicity of used mineral-based crankcase oil. Studies examining the toxicity of the naphthenic and paraffinic base stocks used to formulate mineral-based crankcase oil indicate that these base stocks are relatively nontoxic (API 1983; Beck et al. 1984). Thus, the toxicity associated with exposure to used mineral-based crankcase oil does not appear to be due to the oil present in used mineral-based crankcase oil. Rather, the toxicity that has been observed has been attributed to additives present in the oil (up to 25% of mineral-based crankcase oil formulations) or to decomposition products or contaminants that build up in the oil with use. Thus, results of studies are likely to be specific for the particular additive composition or use characteristics of oil that was used and may not be able to be generalized to all exposures to used mineral-based crankcase oil. For example, lead contamination of oil is expected to be substantially greater in oil from engines using leaded gasolines than from engines powered with unleaded gasolines.

Few well-conducted studies exist that have examined the toxicity of used mineral-based crankcase oil. Furthermore, correlations between exposure and effects in humans are extremely tenuous. For example, a study of mechanics and other auto workers occupationally exposed to used mineral-based

crankcase oil (as well as a variety of other potentially causative substances) suggests that the skin, blood, liver, nervous system, and cardiovascular system may be target organs. However, strong correlations between effects in these organs and exposure to used mineral-based crankcase oil were not established. Inhalation studies in humans suggest that used mineral-based crankcase oil is minimally irritating to the tissues of the respiratory tract, but exposure levels were not well described. Animal data are somewhat more reliable. However, some of the information comes from case reports in species, such as cattle, in which extrapolation to human effects is more difficult. Acute inhalation studies in guinea pigs support the human data indicating that used mineral-based crankcase oil is only minimally irritating to the tissues of the respiratory tract.

Reports of toxicity associated with ingestion of used mineral-based crankcase oil have shown that large oral doses with low levels of metal contamination are tolerated by rats without death or overt toxic signs other than diarrhea. However, cattle ingesting used oil in contaminated pastures have died and shown marked hematological toxicity and neurotoxicity. Cows that ingested used mineral-based crankcase oil during grazing developed clinical lead toxicosis from used crankcase oil or lead containing paint from paint buckets or from sides of buildings or fences with flaking or peeling paint (Osweiler et al. 1973). Oil, paint, grease, trash piles, and lead storage batteries were frequent sources of lead poisoning in 67% of the cases. Of the 67%, oil accounted for 29% of all bovine lead poisoning cases. The source of oil was primarily used crankcase oil (Osweiler et al. 1973). The death of cows in central Hungary was due to molybdenum-induced secondary copper deficiency. The origin of environmental molybdenum was used motor oil containing molybdenum bisulfide as an additive, which had polluted the cows' pasture. The animals had been grazing on the contaminated area for 2 weeks before their illnesses and deaths (Sas 1989). Acute-duration dermal and ocular exposures in rodents have generally resulted in negligible eye irritation and only low-grade dermal irritation. Repeated dermal applications in rabbits have resulted in weight loss of an unknown etiology, suggesting that toxicity may not be limited to the skin.

Carcinogenicity and genotoxicity studies have shown that used mineral-based crankcase oil is carcinogenic to the skin after long-term dermal exposures. The carcinogenicity is most likely due to PAHs found in the oil. Although neoplasia and skin cancer are a possible hazard from repeated dermal exposure to used crankcase oil, this has not been conclusively established, even though used motor oil is genotoxic using the Ames test. Respiratory and ocular discomfort and irritation are possible from short-term exposure to high concentrations of oil mist, aerosol, or exhaust from autos

with inadequate piston rings. At hazardous waste sites, respiratory, cardiovascular, or gastrointestinal effects are unlikely. Hematological effects appear possible with repeated inhalation or dermal contact with oil contaminated with lead, but would probably not occur at hazardous waste sites.

The potential toxicity of lead and other heavy metals in the oil, such as copper, molybdenum, and cadmium, etc. has been noted and would occur upon sufficient exposure to oil or by ingestion of contaminated groundwater. Hepatic, renal, immunological, or neurological effects on fetal development also appear likely. The dose, route, and frequency of exposure are very important in predicting systemic effects from used motor oil. The amount of potential exposure to used motor oil at hazardous waste sites appears to be minimal. The hazard associated with such exposures is anticipated to be low, but adequate data for a comprehensive risk assessment are not available.

Acute effects associated with ingestion of used motor oil are unlikely for humans, but not impossible. Children could accidentally ingest used motor oil. Used crankcase oil stored in homes of do-it-yourself mechanics would be the source of such oil.

Repeated dermal exposure by mechanics and others, respiratory and ocular exposure in closed spaces by mechanics working with motorized vehicles, and oral exposure of children is likely. Exposure to water soluble components, e.g., metals, in contaminated drinking water is also possible.

As noted above, most of the toxicity of used mineral-based crankcase oil has been attributed to additives present in the oil or contaminants that have accumulated in the oil with use. Elements commonly found as additives or contaminants in used mineral-based crankcase oil include lead, zinc, calcium, barium, magnesium, iron, sodium, cadmium, copper, aluminum, chromium, manganese, potassium, nickel, tin, silicon, boron, and molybdenum (Vazquez-Duhalt 1989). In addition, PAHs increase in oil with use (Vazquez-Duhalt 1989). In some cases, observed toxicity has been correlated with individual constituents of the used oil, but this aspect of toxicity with used mineral-based crankcase oil has not been extensively studied. For information on the toxicities associated with exposures to specific contaminants or additives in used mineral-based crankcase oil, see the ATSDR profiles on PAHs (ATSDR 1990c), lead (ATSDR 1993b), zinc (ATSDR 1989b), cadmium (ATSDR 1992d), copper (ATSDR 1990b), chromium (ATSDR 1993a), nickel (ATSDR 1992f), barium (ATSDR 1992b), boron (ATSDR 1992c), manganese (ATSDR 1992e), tin (ATSDR 1992g), and aluminum (ATSDR 1992a).

Minimal Risk Levels for Used Mineral-based Crankcase Oil

As indicated in the introduction to Chapter 2, the composition of used mineral-based crankcase oil is expected to vary depending on the specific additives present in the oil, the type of fuel used, the mechanical condition of the engine, and how long the oil was used. Thus, the NOAEL and LOAEL values presented in Section 2.2 must be considered only as extremely rough estimates of exposure levels associated with the effects observed. Because of the substantial amount of uncertainty regarding these values, no meaningful MRL values could be derived.

Death. No studies were located that reported deaths in humans attributable to exposure to used mineral-based crankcase oil. Studies in rats have shown that ingestion of large doses of used mineral-based crankcase oil over a short period may be well tolerated (API 1980b; Beck et al. 1984; Vemot et al. 1990), but ingestion by cattle while grazing in contaminated pastures resulted in the deaths of several animals (Osweiler et al. 1973; Sas 1989). The deaths of the cattle were attributed to lead and molybdenum in the oil. It is unknown whether the rats and cattle ingested used mineral-based crankcase oils with similar metal contamination. Acute-duration dermal exposures of rabbits (API 1980b; Beck et al. 1984; Vernot et al. 1990) and chronic-duration dermal exposures of mice to used mineral-based crankcase oil (API 1983) did not result in increased mortality. Thus, it is unlikely that persons exposed through skin contact with used mineral-based crankcase oil are at risk of death. However, it is possible that ingestion of sufficient amounts of oil may result in death among susceptible groups in the population depending on the specific contaminant content of the oil. For additional information on death associated with known contaminants of used mineral-based crankcase oil, see the other ATSDR profiles noted above in the introduction to Section 2.5.

Systemic Effects

Respiratory Effects. Inhalation exposure of volunteers to aerosols of used mineral-based crankcase oil for a few minutes has resulted in nose and throat irritation (Dautrebande and Capps 1956) and a feeling of chest tightness (Dautrebande et al. 1951). Animal studies have not, however, shown effects of acute-duration exposure to used mineral-based crankcase oil on pulmonary function. Guinea pigs exposed for up to 1 hour at concentrations as high as 222 mg/m^3 showed no evidence of pulmonary irritation (Costa and Amdur 1979b). Animal studies examining the effects of *unused* mineral-based crankcase oil have shown infectious focal pneumonia and interstitial inflammation in monkeys after

chronic-duration inhalation exposure (Lushbaugh et al. 1950). Thus, the possibility exists that chronic-duration exposure to used mineral-based crankcase oil may also result in similar effects. In addition, exposure to minerals oils present in laxatives and various aerolized industrial materials by inhalation or aspiration causes exogenous lipoid or lipid pneumonia (oil droplets in the lungs) and proliferative fibrosis of the lung (Fan and Graham 1994; Spickard and Hirschmann 1994). However, lipid pneumonia has not been widely observed after inhalation of unused mineral-based crankcase oil (Lushbaugh et al. 1950; Shoshkes et al. 1950) except when aspiration of the material was induced (Gerarde 1963). Because it is unlikely that persons at hazardous waste sites would be exposed to aerosols of used mineral-based crankcase oil, and inhalation is an unlikely route of exposure, respiratory effects from exposures at hazardous waste sites are also highly unlikely to occur.

Cardiovascular Effects. Elevated blood pressure has been reported in a number of workers with occupational exposure to used mineral-based crankcase oil (Clausen and Rastogi 1977). However, it is unclear whether other environmental, genetic, pathophysiological, or behavioral factors may have caused the increase in blood pressure. Systematic studies of whether a significant correlation exists between elevated blood pressure and exposure to used mineral-based crankcase oil have not been performed. Thus, insufficient information exists to determine whether persons exposed to used mineral-based crankcase oil at hazardous waste sites may be at an increased risk of elevated blood pressure.

Gastrointestinal Effects. No increase in clinical signs of gastrointestinal toxicity (stomach pain, constipation, anorexia) was found in workers occupationally exposed to used mineral-based crankcase oil (Clausen and Rastogi 1977). However, diarrhea has been observed in rats (API 1980b; Beck et al. 1984; Vernot et al. 1990) and cattle (Osweiler et al. 1973) that ingested used mineral-based crankcase oil. Medicinal-grade mineral oil is used therapeutically as an emollient laxative acting to soften the stool by retarding reabsorption of water from the gastrointestinal tract (Fingl 1980). Thus, it is likely that used mineral-based crankcase oil is acting at least in part by the same mechanism. A slight irritant effect on-the-gastrointestinal tract by additives or contaminants cannot be eliminated based on the stomach irritation observed in monkeys. Hyperplastic gastritis has been observed in monkeys that were believed to have swallowed significant amounts of inhaled aerosols of *unused* mineral-based crankcase oil (Lushbaugh et al. 1950). While the component of *unused* oil responsible for the gastritis was not identified, it is possible that such a component is also present in used mineral-based crankcase

oil. Thus, the possibility exists that ingestion of large amounts of used mineral-based crankcase oil may result in diarrhea or stomach damage.

Hematological Effects. Lower than normal hematocrit and mean corpuscular hemoglobin have been observed in several workers with occupational exposure to used mineral-based crankcase oil (Clausen and Rastogi 1977). In several cases, the hematological effects correlated with elevated blood lead levels suggesting a role of lead in the effects observed. However, sources of lead exposure other than from used mineral-based crankcase oil and other causes for the blood effects were not eliminated. Ingestion of used mineral-based crankcase oil by ducks and pheasants resulted in dose-related decreases in δ-ALAD activity (Eastin et al. 1983). Since δ-ALAD is involved in heme synthesis and is sensitive to inhibition by lead, this study supports the possibility that lead in used mineral-based crankcase oil may have contributed to the anemia observed in the auto workers. Ingestion of used mineral-based crankcase oil containing high levels of molybdenum resulted in anemia in cattle (Sas 1989). Thus, the possibility exists that anemia may occur in persons exposed to lead or molybdenum in used mineral-based crankcase oil.

Hepatic Effects. Limited epidemiological data show that a small percentage of workers with occupational exposure to used mineral-based crankcase oil had elevated clinical chemistry values indicative of liver damage (increased serum bilirubin, alanine and aspartate aminotransferases, lactate dehydrogenase, and thymol reaction) (Clausen and Rastogi 1977). However, other environmental or behavioral factors were not eliminated as potential causes. Animal data have not shown biochemical evidence after intermediate-duration oral exposure (Eastin et al. 1983) or histopathological evidence of hepatic toxicity after acute-duration dermal exposure (API 1980b; Beck et al. 1984; Vemot et al. 1990). Insufficient information exists to predict whether persons exposed to used mineral-based crankcase oil at hazardous waste sites will exhibit hepatotoxicity.

Renal Effects. No studies were located that reported renal effects in humans attributable to exposure to used mineral-based crankcase oil. The only information regarding renal effects in animals after exposure to used mineral-based crankcase oil came from a 2-week dermal study in rabbits that indicated no histopathological evidence of toxicity of the kidneys and urinary bladder (API 1980b; Beck et al. 1984; Vemot et al. 1990). Insufficient information was located to determine whether renal toxicity may result from exposure to used mineral-based crankcase oil at hazardous waste sites.

Dermal Effects. Skin rashes on the hands and arms were reported by a number of workers exposed to used mineral-based crankcase oil (Clausen and Rastogi 1977). However, it was not established whether used mineral-based crankcase oil was the causative agent. Studies in rabbits (API 1980b; Beck et al. 1984; Vemot et al. 1990) and mice (API 1983) have shown that dermal exposure to used mineral-based crankcase oil results in mild-to-moderate skin irritation. In rabbits, multiple exposures also resulted in hair loss adjacent to the application site (API 1980b; Beck et al. 1984; Vemot et al. 1990). Monkeys chronically exposed (whole-body) to aerosols of *unused* mineral-based crankcase oil also experienced extensive hair loss (Lushbaugh et al. 1950). It is unknown whether the underlying cause for the hair loss is similar in these two studies, but the data suggest that components present in both unused and used mineral-based crankcase oil may result in some degree of hair loss.

Ocular Effects. Volunteers exposed to aerosols of used mineral-based crankcase oil have reported eye irritation (Dautrebande and Capps 1950; Dautrebande et al. 1951). In contrast, negligible irritation was noted in rabbits when 0.1 mL was directly instilled into the eye (API 1980b; Beck et al. 1984; Vernot et al. 1990). This apparent discrepancy may be due to the differing compositions of the samples of the used mineral-based crankcase oil that were tested. It is unlikely that persons at hazardous waste sites would be exposed to aerosols of used mineral-based crankcase oil. However, dermal exposure is possible and eye irritation from used mineral-based crankcase oils may occur.

Body Weight Effects. No studies were located regarding body weight effects in humans after exposure to used mineral-based crankcase oil. No effects on body weight gain or growth were observed in rats that received single doses or used mineral-based crankcase as high as 22,500 mg/kg (API 1980b; Beck et al. 1984; Vemot et al. 1990) nor in mice (API 1983).

In contrast, 8 mL/kg of used mineral-based crankcase applied on the backs of rabbits 24 hours/day, 5 days/week, for 2 weeks resulted in a marked decrease in food consumption and weight loss (API 1980a; Beck et al. 1984; Vemot et al. 1990). The biological basis for the decreased food consumption was not identified.

Immunological and Lymphoreticular Effects. Extremely little information was located regarding immunological effects of used mineral-based crankcase oil in humans or animals. A dermal sensitization study conducted with guinea pigs showed no sensitization potential (API 1980b; Beck et al. 1984; Vemot et al. 1990), but the positive control also failed to show sensitization. Other aspects

of immune function were not assessed. Thus, some uncertainty exists regarding the likelihood of contact dermatitis following exposure to used mineral-based crankcase oil near hazardous waste sites.

Neurological Effects. Limited information was located regarding neurological effects in humans or animals after exposure to used mineral-based crankcase oil. A study of a group of auto workers showed headaches and/or tremors of the hands in a small number of the exposed workers (Clausen and Rastogi 1977). However, it was not established whether the used mineral-based crankcase oil was the causative agent. Studies in rodents have not reported overt neurotoxicity following inhalation, oral, or dermal exposure to used mineral-based crankcase oil, but cattle that have ingested used mineral-based crankcase oil have shown muscle tremors, weakness, blindness, muscle twitching, hyperirritability, depression, and convulsions (Osweiler et al. 1973; Sas 1989). The neurological effects in the study by Osweiler et al. (1973) were attributed to lead poisoning, and those in the study by Sas (1989) were attributed to molybdenum poisoning. Insufficient information exists to determine whether persons exposed to used mineral-based crankcase oil at hazardous waste sites may experience adverse neurological effects.

Reproductive Effects. No information was located regarding reproductive effects in humans or animals after exposure to used mineral-based crankcase oil.

Developmental Effects. No information was located regarding developmental effects in humans or animals after inhalation, oral, or dermal exposure to used mineral-based crankcase oil. However, a study by Hoffmann and Albers (1984) investigated the potential embryotoxicity and teratogenicity of automotive virgin crankcase oil and waste crankcase oil in mallard *(Anus platyrhynchos)* eggs. The oil was applied to upright fertile eggs with a microliter pipet by streaking the surface just below the air space and allowing the oil to spread freely (Hoffmann and Albers 1984). Application of waste crankcase oil significantly affected embryonic survival. It had an LD_{50} of 5.3 µL/egg (=90 µg/g egg) compared to that of virgin crankcase oil, which was 23.5 µL/egg. At doses below the LD_{50}, waste crankcase oil also resulted in more survivors with severe abnormalities, including defects of the brain (anencephaly and exencephaly) and of the eye (microphthalmia), than with virgin crankcase oil (Hoffmann and Albers 1984).

An egg-painting study using quail and ducks showed decreased growth and survival in both quail and duck fetuses, edema, incomplete ossification, and exencephaly in quail; and hemorrhages, edema,

incomplete ossification, anencephaly, and microphthalmia in ducks (Hoffman et al. 1982a). In addition, quail and duck embryos and/or hatchlings showed low hemoglobin and decreased blood and liver δ-ALAD. The toxicity was attributed to the lead and aromatic hydrocarbon content of the used oil rather than to asphyxiation since higher volumes of other oils were not toxic. The presence of heavy metals in the oil may be a significant factor in its toxicity. Automotive waste oil contained 4,600 ppm lead, which caused a significant depression of red blood cell δ-ALAD activity and decreased hemoglobin concentration in mallard and bobwhite embryos (Hoffmann and Albers 1984; Hoffman et al. 1982a). It is unknown whether comparable developmental toxicity would be observed in persons exposed to used mineral-based crankcase oil. It should be noted here that painting or injecting bird eggs with used crankcase oil is very different from performing reproductive toxicity tests using mammalian species. In the mammalian situation, the pregnant animal is treated or administered the substance. In addition, there are fundamental maternal, circulatory, excretory, and metabolic influences which determine exposure to the fetus, qualitatively and quantitatively. Hence, the relevance to humans of bird egg toxicity after exposure to used mineral-based crankcase oil is remote.

Genotoxic Effects. No standard *in vivo* or *in vitro* genotoxicity studies of used crankcase oil in mammalian test systems were located, but several studies (Carmichael et al. 1990, 1991, 1992; Schoket et al. 1989) identified DNA adducts formed by acute-duration dermal application of used mineral-based crankcase oils and suggested that specific DNA adducts may be attributed to the reaction of specific PAHs with DNA. PAHs, like benzo[a]pyrene, induce certain drug-metabolizing enzymes, mainly the cytochrome P4501A subfamily (CYPIA), which is also known as CYP448 and aryl hydrocarbon hydroxylase (AHH). The CYPIA enzymes metabolize PAHs into epoxides, which are then hydroxylated to dihydrodiols and are noncarcinogenic. However, further epoxidation of the dihydrodiols by the CYPIA enzymes generates reactive “electrophilic” species which are capable of binding and damaging DNA by forming DNA adducts, resulting in necrosis and/or cancer. However, while these experiments clearly show that PAHs in used motor oil interacted with DNA, most adducts would be repaired, and few would result in genetic lesions. Thus, the rate of repair and the final outcome of the DNA damage is of considerable interest.

Similarly, DNA adducts were formed when human skin cultures were exposed to used oils (Carmichael et al. 1991). Application of diesel engine oil resulted in fewer adducts than did gasoline engine oil in both *in vivo* and *in vitro* test systems. Qualitative differences in the adducts formed in the human and mouse skin culture systems were consistent with the metabolism of certain PAHs by

species-specific pathways. Based on the comparison of DNA-adducts formed by fractionated oils with those formed by individual PAHs, Carmichael et al. (1992) attributed the major adducts produced in mouse skin by used gasoline engine oil to reactive metabolites of benzo(*b*)naphtho(l,2-*d*)thiophene, benzo(*c*)phenanthrene, benzo(*g,h,i*)fluoranthene, chrysene, benzo(*a*)pyrene, and benzo(*g,h,i*)perylene. Used mineral-based crankcase oil also contains compounds that induce aryl hydrocarbon hydroxylases (AHHs) (Rahimtula et al. 1982). As with the adducts observed in the dermal studies, only unrepaired adducts would result in genetic lesions. Furthermore, it is not known how well the skin cell culture system simulates dermal exposure *in vivo*.

Used mineral-based crankcase oil from gasoline-powered automobiles is consistently positive in *Salmonella typhimurium*/mammalian microsome mutagenicity assays in the presence or absence of S9 activation, but the revertant yield is higher with the S9-activated material (Blackbum et al. 1983; Granella and Clonfero 1991; Pasquini and Monarca 1983; Payne et al. 1978; Peake and Parker 1980; Rahimtula et al. 1984; Schreiner and Mackerer 1982). The strongest mutagenic responses are generally observed in strains TA98 and TA100. *Unused* mineral-based crankcase oil is generally negative in this assay in both the presence and absence of S9 activation (Granella and Clonfero 1991; Pasquini and Monarca 1983; Schreiner and Mackerer 1982). Key genotoxicity studies using *S. typhimurium* are presented in Table 2-4.

The mutagenicity of oil appears to increase as a function of the mileage since the last oil change. An increase in the number of revertants/mg oil, induced with or without S9, was observed in repeated sampling of the motor oil in one car (EPA 1980d). The level of skin and lung adducts produced by dermal application of used engine oil to male mice correlated with an index of oil use calculated as the product of the number of miles since the last oil change and the total engine mileage (Carmichael et al. 1990). Not all studies have shown similar results, but possible methodological differences may have accounted for the differing results. In a comparison of used oils from eight different cars, no correlation was observed between mutagenicity and engine age or miles since the last oil change (Dutcher et al. l-986), but no analysis was conducted to determine whether there was a correlation with engine age *and* miles since the last oil change. Such a correlation could have been missed because the newer cars tended to have higher mileage since the last oil change. The water-soluble fraction of used mineral-based crankcase oil was not mutagenic with or without S9 activation, but this fraction was obtained in a highly diluted form, and toxic levels were not tested (Vandermeulen et al. 1985).

TABLE 2-4. Genotoxicity of Used Mineral-based Crankcase Oil *In Vitro*

Species (test system)	End point	Results: With activation	Results: Without activation	Reference
Prokaryotic organisms:				
Gasoline engine oil				
Salmonella typhimurium	Gene mutation	+	+	Abdelnasser et al. 1986
S. typhimurium	Gene mutation	+	+	Blackburn et al. 1983
S. typhimurium	Gene mutation	+	+	Dutcher et al. 1986
S. typhimurium	Gene mutation	+	+	Granella and Clonfero 1991
S. typhimurium	Gene mutation	+	ND	Hermann et al. 1980a
S. typhimurium	Gene mutation	+	ND	Hermann et al. 1980b
S. typhimurium	Gene mutation	+	+	EPA 1980d
S. typhimurium	Gene mutation	+	+	Manabe et al. 1984
S. typhimurium	Gene mutation	+	+	Pasquini and Monarca 1983
S. typhimurium	Gene mutation	+	–	Payne et al. 1978
S. typhimurium	Gene mutation	+	+	Peake and Parker 1980
S. typhimurium	Gene mutation	+	+	Rahimtula et al. 1984
S. typhimurium	Gene mutation	+	+	Schreiner and Mackerer 1982
S. typhimurium	Gene mutation	+	+	Vandermeulen et al. 1985
Diesel engine oil				
S. typhimurium	Gene mutation	+	+	Dutcher and Clark 1981
S. typhimurium	Gene mutation	+	+	Dutcher et al. 1986
S. typhimurium	Gene mutation	ND	+	Manabe et al. 1984

+ = positive result; – = negative result; ND = not determined

Data regarding the mutagenicity of used oil from diesel engines are much more limited than those regarding used gasoline engine oil. However, there is evidence that nonactivated and S9-activated diesel engine oil is mutagenic in *S. typhimurium* strain TA98 (Dutcher and Clark 1981; Dutcher et al. 1986). In another study, extracts of diesel engine oils were reported to cause a small increase in TA98 mutants (EPA 1980d). Diesel engine oil was less potent than gasoline engine crankcase oil in the induction of DNA adducts following dermal application to mice *in vivo* (Carmichael et al. 1990, 1991; Schoket et al. 1989) and application to mouse or human skin cultures *in vitro* (Carmichael et al. 1991). The lower mutagenicity of diesel engine oils may be attributable to the lower mileage since the last oil change, compared to oil from gasoline-powered engines. However, this would not be expected to affect the human health risk since diesel engine oil is changed more frequently than oil in gasoline-powered engines.

The *Salmonella* assay has also been used by several investigators to compare the mutagenicity of different samples of used mineral-based crankcase oil and to identify the mutagenic components in the complex chemical mixture found in used mineral-based crankcase oil. The data regarding the specific PAHs that account for the mutagenicity of used crankcase oil are contradictory. As discussed above, some inconsistencies may arise from variations in the oil used. In an early experiment, the mutagenic activity of used oil largely did not migrate together with benzopyrene or benzanthracene (isomers not reported) on thin-layer chromatography plates (Payne et al. 1978). Most of the mutagenicity of another oil sample was attributed to PAHs containing four, five, or six fused aromatic rings (Hermann et al. 1980b). The PAH with the largest contribution to the mutagenicity was benzo(a)pyrene. Most of the adducts found following dermal treatment of mice with used mineral-based crankcase oil were attributed to metabolites of PAHs with four or fewer aromatic rings. These data are too limited and contradictory to determine which compounds are responsible for much of the mutagenic activity of used mineral-based crankcase oil.

Results from the controlled “aging” of new oil under defined conditions suggest that nitrated PAHs or other compounds generated by reaction of the oil with nitrogen dioxide account for much of the direct-acting mutagenic activity of used oil. Exposure to nitrogen dioxide markedly increased the yield of revertants induced by fresh crankcase oil in *S. typhimurium* strain TA98 with or without S9 activation. By contrast, heating the oil to 100°C for 100 hours, or exposing it to air, sulfur dioxide, or hydrogen sulfide did not result in the production of mutagenic material (Abdelnasser et al. 1986). Thin-layer chromatographic fractionation revealed that the composition of mutagenic compounds in used oil was

similar to that in fresh oil treated with heat and nitrogen dioxide. The study authors suggested that these mutagenic species are nitrated PAHs. Other authors have also implicated reaction with nitrogen dioxide in the production of mutagenic compounds (Naughton and Jespersen 1991).

Genetic analysis has also indicated that a major portion of the mutagenic activity of used mineralbased crankcase oil in the absence of S9 activation is due to nitroaromatic compounds (nitroPAHs). The yield of revertants induced in TA98 by a dimethyl sulfoxide extract of used mineral-based crankcase oil in the absence of S9 was reduced by about 65% in the nitroreductase-deficient strain TA98NR (Blackburn et al. 1983), a finding confirmed by Manabe et al. (1984). 1-Nitropyrene (1-NP) and 1,6-dinitropyrene (diNP) were tentatively identified as direct-acting mutagenic compounds in used mineral-based crankcase oil. Thus, the direct-acting mutagenic activity of used mineral-based crankcase oil is largely attributed to nitroaromatic compounds, while the mutagenic activity that requires S9 activation is attributed largely to PAH metabolites. Nitro-PAHs can be metabolized to aromatic amine derivatives, some of which are capable of inducing urinary bladder cancer in humans (Iyer et al. 1990). 1-NP is also capable of generating oxidants or reactive oxygen species such as the superoxide radical (Nachtman 1986), which can react with and damage cellular molecules (DNA, lipids, proteins) or activate genotoxic substances such as PAHs, which can eventually lead to cancer (Sun 1990). These oxidants are also generated in the body by white blood cells as a defense mechanism during inflammation (Sun 1990). Reactive oxygen species are thought to play a role in the toxicity and mutagenicity of diesel soot (Vogl and Elstner 1989) and in the metabolic activation of PAHs in mouse skin (Kensler et al. 1987). 1-NP, diNP, and trinitropyrene were shown to be potent lung DNA binding agents in the absence of the inducing agent, BaP, but were extremely potent after pretreatment with BaP (Howard et al 1986). Hence, it is possible that 1-NP and diNP may play a role in the toxicity of used mineral-based crankcase oil by generating superoxide radicals which can damage cells and lead to the development of cancer.

Although no genotoxicity data were located regarding used mineral-based crankcase oil in mammalian systems, the evidence suggests that it can be mutagenic to humans. Crankcase oil accumulates PAHs, many of which are known genotoxins and fractionate together with mutagenic activity in the oil. Oil from gasoline engines would presumably be more mutagenic than oil from diesel engines. No possible explanations were located for the higher mutagenic potency of oil from gasoline engines. Used oil is mutagenic in the *Salmonella* mutation assay, the mutagenic activity is stronger in the presence of S9 activation, and DNA adducts are found in cutaneous DNA following application of used oil to mice *in*

vivo or to samples of human or mouse skin *in vitro*. Thus, the genotoxicity data support the data from animal studies indicating that dermal exposure to used mineral-based crankcase oil can cause cancer. The genotoxicity data also suggest that used mineral-based crankcase oil may be a developmental toxicant. Therefore, dermal exposure to used mineral-based crankcase oil may pose a human health hazard.

Cancer. Limited information was located regarding the carcinogenicity of used mineral-based crankcase oil in humans. An epidemiological study of workers in Sweden indicated that the risk of developing renal pelvic and bladder cancer was not increased in workers exposed to motor oil (Steineck et al. 1989), but no information was located regarding the development of other types of cancer. Studies in mice demonstrate that chronic-duration dermal exposure to used mineral-based crankcase oil results in an increased incidence of dermal papillomas and carcinomas (API 1983; Grimmer et al. 1982a, 1982b, 1983; McKee and Plutnick 1989). The tumor incidence correlated with the PAH content of the oil (Grimmer et al. 1982a, 1982b, 1983; McKee and Plutnick 1989). DNA adducts were identified in both the skin and lungs of mice that had received dermal treatments for l-4 days (Carmichael et al. 1990; Schoket et al. 1989), suggesting that tumor development in organs other than the skin may be possible.

Based on the information from the mouse studies, IARC has determined that one sample of used mineral-based crankcase oil showed sufficient evidence for carcinogenicity in animals; however, data are inadequate to evaluate used mineral-based crankcase oil as a class “since the possible carcinogenic activity of individual products is dependent upon the quality of the base oils used, the nature and concentration of additives and contaminants, and the conditions of use” (IARC 1984). EPA and the Department of Health and Human Services have not classified used mineral-based crankcase oil as to its carcinogenicity. The IARC cancer classification reflects the uncertainty regarding the composition of different batches of used mineral-based crankcase oil.

2.6 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).

Due to a nascent understanding of the use and interpretation of biomarkers, implementation of biomarkers as tools of exposure in the general population is very limited. A biomarker of exposure is a xenobiotic substance or 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. However, 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 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 used mineral-based crankcase oil are discussed in Section 2.6.1.

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 not often substance specific. They also may not be directly adverse, but can indicate potential health impairment (e.g., DNA adducts). Biomarkers of effects caused by used mineral-based crankcase oil are discussed in Section 2.6.2.

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 increasein absorbed dose, a decrease in the biologically effective dose, or a target tissue response. If biomarkers of susceptibility exist, they are discussed in Section 2.8, Populations That Are Unusually Susceptible.

2.6.1 Biomarkers Used to Identify or Quantify Exposure to Used Mineral-based Crankcase Oil

No information was located regarding biomarkers that could be used to identify exposure to used mineral-based crankcase oil. However, biomarkers for PAHs such as DNA adduct formation (Carmichael et al. 1990; Schoket et al. 1989) or biomarkers for metals such as metal content of the blood (Clausen and Rastogi 1977; Osweiler et al. 1973; Sas 1989) or decreased δ-aminolevulinic acid dehydratase in blood (Clausen and Rastogi 1977; Eastin et al. 1983; Hoffman et al. 1982a) have been identified as possible indicators of exposure to chemicals found in used mineral-based crankcase oil. It should be noted that these biomarkers are not specific for exposure to used mineral-based crankcase oil itself but may be specific for toxic chemicals found in the oil. Any substance containing PAHs and/or heavy metals could be the source of exposure. Although there are valid tests to measure biomarkers of exposure, high blood lead for instance, the presence of a biomarker does not necessarily imply exposure to used motor oil. Hence, evidence of exposure to used mineral-based crankcase oil must first be determined before biomarkers of exposure to metals or PAHs are used to help confirm exposure to used mineral-based crankcase oil. Biomarkers for exposure to metals and aromatic hydrocarbons are discussed in more detail in ATSDR profiles on the following substances: PAHs (ATSDR 1990c), lead (ATSDR 1993b), zinc (ATSDR 1989b), cadmium (ATSDR 1992d), copper (ATSDR 1990b), chromium (ATSDR 1993a), nickel (ATSDR 1992f), barium (ATSDR 1992b), boron (ATSDR 1992c), manganese (ATSDR 1992e), tin (ATSDR 1992g), and aluminum (ATSDR 1992a).

2.6.2 Biomarkers Used to Characterize Effects Caused by Used Mineral-based Crankcase Oil

While the health effects of exposure to used mineral-based crankcase oil have not been fully described, some indicators of toxicity have been identified that may be useful in characterizing potential effects. Hematological analyses for hemoglobin or hematocrit could be useful for predicting anemia (Clausen and Rastogi 1977). Analyses for serum copper and ceruloplasmin levels may be useful in detecting copper deficiency (Sas 1989), and examination of skin for the formation of elevated levels of DNA adducts (Carmichael et al. 1990, 1991, 1992; Schoket et al. 1989) may be indicative of potentially genotoxic exposures. The biomarkers mentioned are specific for toxic materials which may be components of used motor oil, but are not specific for used motor oil itself. For further descriptions of the utility of these biomarkers in characterizing effects of individual chemicals potentially found in used mineral-based crankcase oil, see the ATSDR profiles on PAHs (ATSDR 1990c), lead (ATSDR

1993b), zinc (ATSDR 1989b), cadmium (ATSDR 1992d), copper (ATSDR 1990b), chromium (ATSDR 1993a), nickel (ATSDR 1992f), barium (ATSDR 1992b), boron (ATSDR 1992c), manganese (ATSDR 1992e), tin (ATSDR 1992g), and aluminum (ATSDR 1992a).

2.7 INTERACTIONS WITH OTHER SUBSTANCES

Extremely limited and conflicting information was located regarding interactions of used mineral-based crankcase oil with other chemicals. As very little is known about this subject, the discussion is necessarily quite limited. While a decrease in the pulmonary irritancy of sulfur dioxide in guinea pigs was reported by Costa and Amdur (1979a) when aerosols of used mineral-based crankcase oil were administered simultaneously with sulfur dioxide, human subjects experienced an increase in eye, nose, and throat irritation when exposed to aerosols containing both used mineral-based crankcase oil and sulfur dioxide (Dautrebande and Capps 1950). The differences in the results may be due to differences in the end points examined or in the amount or composition of the samples of used mineral-based crankcase oil that were employed, but they do not appear to be attributable to differences in the concentration of sulfur dioxide. Other interactions reported included less-than-additive eye, nose, and throat irritation reported by human subjects exposed to aerosols of combinations of used mineral-based crankcase oil with either carbon black, a combination of sulfur dioxide and carbon black, or a combination of sulfur trioxide and carbon black (Dautrebande and Capps 1950).

2.8 POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE

A susceptible population will exhibit a different or enhanced response to used mineral-based crankcase oil than will most persons exposed to the same level of used mineral-based crankcase oil in the environment. Reasons include genetic make-up, developmental stage, age, health and nutritional status (including dietary habits that may increase susceptibility, such as inconsistent diets or nutritional deficiencies), and substance exposure history (including smoking). These parameters may result in decreased function of the detoxification and excretory processes (mainly hepatic, renal, and respiratory) or the pre-existing compromised function of target organs (including effects or clearance rates and any resulting end-product metabolites). 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.

Review of the literature on toxic effects of used mineral-based crankcase oil did not identify specific populations that are known to be unusually susceptible to its effects. Depending on the additive and contaminant composition of individual oils, various populations may be expected to be particularly susceptible. The groups with high susceptibility would be expected to vary with the constituents of the oil. It is beyond the scope of this profile to detail all possible exposure scenarios and the respective sensitive groups. However, certain populations with high susceptibility to PAH or lead toxicity will be summarized below. For additional information on the populations that may be expected to be susceptible to the toxicities of PAHs, lead, and other potential constituents, see the ATSDR profiles on PAHs (ATSDR 1990c), lead (ATSDR 1993b), zinc (ATSDR 1989b), cadmium (ATSDR 1992d), copper (ATSDR 1990b), chromium (ATSDR 1993a), nickel (ATSDR 1992f), barium (ATSDR 1992b), boron (ATSDR 1992c), manganese (ATSDR 1992e), tin (ATSDR 1992g), and aluminum (ATSDR 1992a).

Persons repeatedly exposed to used motor oil would potentially be a very susceptible group (see Section 5.6). Populations expected to show an increased susceptibility to toxic effects of PAHs include persons with induced aryl hydrocarbon hydroxylase activity (the enzyme responsible for transformation of PAHs to reactive intermediates), those with nutritional deficiencies in vitamins A, C, and D, iron, and riboflavin (an increased cancer incidence has been seen under these conditions), and persons with genetic diseases affecting DNA repair capabilities (ATSDR 1990c). Assuming used mineral-based crankcase oil has carcinogenic components, persons who smoke and those with excessive sun exposure may show increased carcinogenicity because particulates in cigarette smoke and ultraviolet radiation potentiate the carcinogenic response to PAH exposure. Also, the very young and persons without fully functional immune systems may be impaired in their ability to resist PAH-induced toxicity (ATSDR 1990c). The very young and the very old, for reasons of physiology and metabolism, would also be expected to be more susceptible than the “normal” adult population to any adverse effect of exposure to used motor oil, as would be persons with a Vitamin A, C, or D deficiency. Deficiencies in Vitamins C and D enhances lead and cadmium toxicity.

Populations that may be more susceptible to the toxic effects of lead exposure include young children (<5 years old), pregnant women, the elderly, smokers, alcoholics, and people with genetic diseases

affecting heme synthesis, and nutritional deficiencies (ATSDR 1993b). Children have a greater gastrointestinal absorption of lead and lower blood thresholds for hematological and neurological effects induced by lead exposure. Dietary deficiencies in calcium and iron potentiate the toxicity of lead. Pregnant women and the elderly may have increased blood lead levels because of greater mobilization from bone. Persons who consume large amounts of alcohol may be at increased risk of hematological, neurological, and hepatotoxic effects of lead because of alcohol's synergistic effects on these tissues (ATSDR 1993b).

2.9 METHODS FOR REDUCING TOXIC EFFECTS

This section describes clinical practice and research concerning methods for reducing toxic effects of exposure to used mineral-based crankcase oil. 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 used mineral-based crankcase oil. When specific exposures have occurred, poison control centers and medical toxicologists should be consulted for medical advice.

2.9.1 Reducing Peak Absorption Following Exposure

Treatment recommendations are sufficiently general for virtually any mineral oil exposure. There are no specific methods for used mineral-based crankcase oil. Because the volatility of used mineralbased crankcase oil is expected to be negligible and inhalation exposure is a concern only in situations where aerosols may be generated, exposure to used mineral-based crankcase oil is expected to occur mainly via the oral and dermal routes. Effects of dermal exposures may be limited by washing exposed skin with mild green (lipophilic) soap (Ellenhorn and Barceloux 1988). Care should be taken not to abrade the skin, since dermal abrasions may facilitate the absorption of toxic chemicals found in the oil. Virtually nothing is known about the absorption of used mineral-based crankcase oil from the gastrointestinal tract, but toxicity studies suggest that absorption of chemicals (contaminants or additives) found in the oil may be toxicologically significant (Osweiler et al. 1973; Sas 1989). Therefore, it may be desirable to take steps to reduce absorption of chemicals found in the oil. This is especially true for those chemicals whose absorption is augmented in the presence of oil. In general, steps taken to reduce the gastrointestinal absorption of chemicals found in the oil should be tailored to practices known to be effective for the constituents of concern (Ellenhorn and Barceloux 1988; Goldfrank et al. 1990; Haddad and Winchester 1990). Since the risk of aspiration of used mineral-

based crankcase oil is not as great as with many hydrocarbons (Gerarde 1963; Goldfrank et al. 1990), induction of emesis may be a viable means of removing the oil from the gastrointestinal tract. Gastric lavage has been recommended only in unconscious patients with the use of a cuffed endotracheal tube to limit aspiration that could result in induction of lipid pneumonia (Gerarde 1963; Goldfrank et al. 1990). Activated charcoal has not been shown to be effective in adsorbing petroleum distillates (Goldfrank et al. 1990) but may be effective in limiting the absorption of other chemicals found in the oil. Cathartics may help speed removal from the gastrointestinal tract; however, the used mineralbased crankcase oil may itself act as a cathartic if sufficiently large amounts are ingested. If additional cathartics are required, osmotic cathartics such as sorbitol are preferable to mineral oil cathartics because of the possibility of aspiration of the oil (Ellenhorn and Barceloux 1988; Haddad and Winchester 1990).

2.9.2 Reducing Body Burden

Available information indicates that the toxicity of used mineral-based crankcase oil is primarily a function of the toxicities of additives or contaminants found in the oil. Therefore, steps used to reduce the body burden should be tailored to the specific additives or contaminants of concern. It is beyond the scope of this profile to present the methods associated with decreasing body levels of all of the potential chemicals of concern. However, a brief discussion of lead and PAHs is provided as they are the primary hazardous constituents associated with used motor oil. In adults and children, approximately 94% and 73% of the total body burden of lead is found in bones, respectively (ATSDR 1993b). Currently, chelating agents are used to reduce the body burden of lead and hence, its toxicity. All of the chelating agents bind inorganic lead and facilitate the transfer of lead from soft tissues to the circulation where it can be excreted. Since excretion of chelated lead is via the kidney, caution should be used in patients with renal failure. The standard chelating agents currently used are dimercaprol (British Anti-Lewisite or BAL) and ethylenediaminetetraacetic acid (EDTA). Both these agents are administered parenterally (ATSDR 1993b).

Currently, there are no known methods available for reducing the body burden of PAHs. PAHs are rapidly metabolized and conjugated to form water-soluble metabolites that are completely eliminated in the urine and feces within a matter of days (ATSDR 1990c). No data are available on the kinetics of PAHs following chronic exposure, so it is not known if PAHs or their metabolites bioaccumulate in these exposure situations. Given the relatively rapid and complete excretion following short-term

exposures, it is unlikely that PAHs will bioaccumulate to a large extent. However, since PAHs are lipophilic, it is possible that the unmetabolized parent compound could accumulate in fat tissue (ATSDR 1990c). It is possible that high-fat diets may favor accumulation of parent PAHs in lipids so that they are not metabolized to reactive intermediates or water-soluble conjugates. Therefore, modulating body fat content may reduce the body burden of PAHs by hastening their metabolism to water-soluble conjugates. However, this may result in increased toxicity due to the increased metabolism to reactive metabolites (ATSDR 1990c).

For substance-specific information on methods that may be effective in reducing levels of other individual chemicals potentially found in used mineral-based crankcase oil, see the ATSDR profiles on PAHs (ATSDR 1990c), lead (ATSDR 1993b), zinc (ATSDR 1989b), cadmium (ATSDR 1992d), copper (ATSDR 1990b), chromium (ATSDR 1993a), nickel (ATSDR 1992f), barium (ATSDR 1992b), boron (ATSDR 1992c), manganese (ATSDR 1992e), tin (ATSDR 1992g), and aluminum (ATSDR 1992a).

2.9.3 Interfering with the Mechanism of Action for Toxic Effects

As indicated above in the section on mechanisms of action, the toxicity of used mineral-based crankcase oil has been attributed primarily to additives or contaminants found in the oil. As both the additive composition and contaminants are likely to vary depending on the brand of oil used and its use characteristics, steps taken to interfere with mechanisms underlying toxic responses should be tailored to the individual chemicals of concern. It is beyond the scope of this profile to address actions that may be taken to interfere with the mechanisms of action of all potential toxic additives or contaminants. However, a brief discussion of lead and PAHs is provided as they are the primary hazardous constituents associated with used motor oil. Chelating agents are used to prevent the toxicity of lead by reducing the body burden. All of the chelating agents bind inorganic lead and facilitate the transfer of lead from soft tissues to the circulation where it can be excreted. BAL chelates both intracellular and extracellular stores of lead. BAL-lead chelates are excreted mainly in the bile and some in the urine. BAL is used in individuals with renal impairment (ATSDR 1993b). For adults that are symptomatic or have blood lead levels >70 μg/dL and for children (symptomatic or asymptomatic) with blood lead levels >70 μg/dL, therapy with BAL is followed by EDTA. For asymptomatic children with blood lead levels >45-69 μg/dL, EDTA chelation therapy is used. 2,3-Dimercaptoseccinia acid (DSMA) is an orally administered chelating agent approved by the FDA

for treating children with blood levels >45 μg/dL. DSMA is also being used to treat lead poisoning in adults (ATSDR 1993b).

The toxic and carcinogenic effects of PAHs are thought to be mediated by reactive diol-epoxide intermediates that interact directly with DNA and RNA, producing adducts. These adducts result in neoplastic transformation and interfere with the normal functioning of rapidly proliferating tissues. The reactive intermediates are formed when PAHs are biotransformed by the cytochrome P450 enzymes (ATSDR 1990c). Interference with these metabolic pathways, by inactivation of the activated diol epoxides, or a reduction in tissue levels of cytochrome P450 enzymes responsible for forming the reactive intermediates, could reduce the toxic and carcinogenic effects of PAHs. Various drugs, such as cobaltous chloride, SKF-535A and 6-nitro-1,2,3-benzothiadiazole can inhibit the cytochrome P450 enzymes. Because PAHs are detoxified by conjugation with substances such as glutathione, sufficient glutathione stores in the body may reduce the chances of toxic effects following acute exposure to PAHs.

For additional chemical-specific information see the ATSDR profiles on PAHs (ATSDR 1990c), lead (ATSDR 1993b), zinc (ATSDR 1989b), cadmium (ATSDR 1992d), copper (ATSDR 1990b), chromium (ATSDR 1993a), nickel (ATSDR 1992f), barium (ATSDR 1992b), boron (ATSDR 1992c), manganese (ATSDR 1992e), tin (ATSDR 1992g), and aluminum (ATSDR 1992a).

2.10 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 used mineral-based crankcase oil 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 usedmineral-based crankcase oil.

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 the uncertainties of human health assessment. This definition should not be interpreted to mean

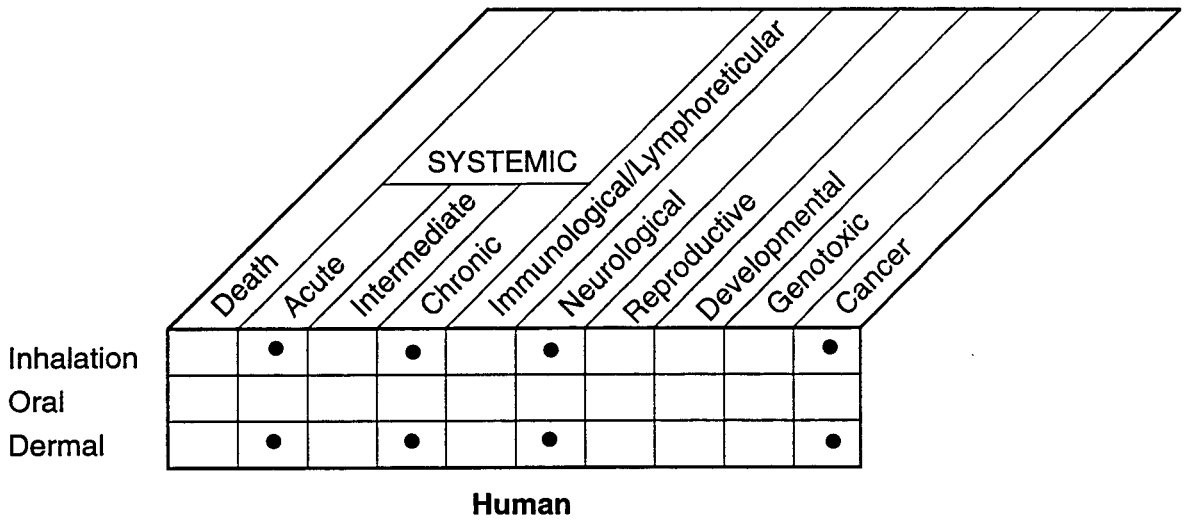
that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

2.10.1 Existing Information on Health Effects of Used Mineral-based Crankcase Oil

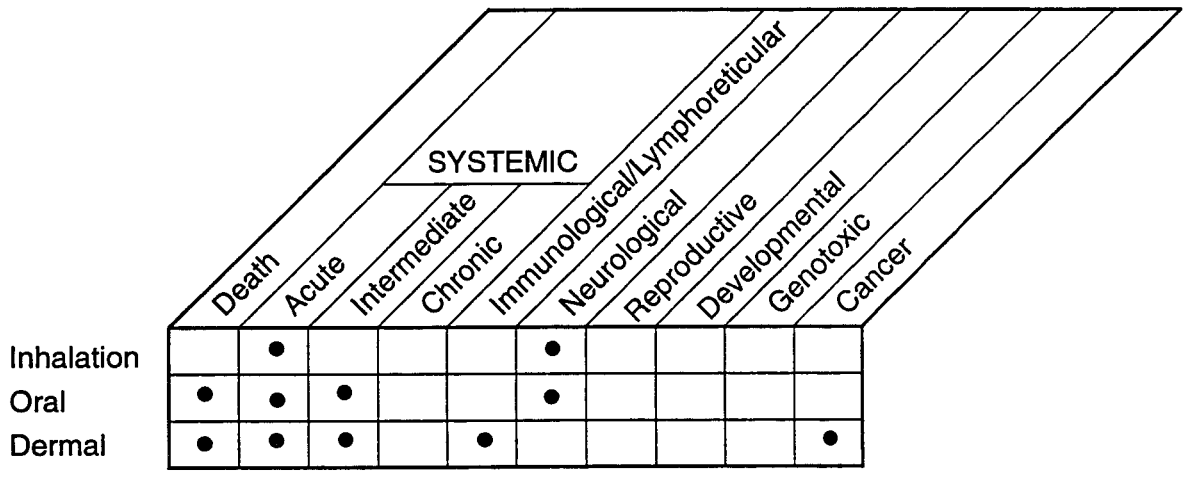
The existing data on health effects of inhalation, oral, and dermal exposure of humans and animals to used mineral-based crankcase oil are summarized in Figure 2-3. The purpose of this figure is to illustrate the existing information concerning the health effects of used mineral-based crankcase oil. Each dot in the figure indicates that one or more studies provide information associated with that particular effect. The dot does not necessarily imply anything about the quality of the study or studies, nor should missing information in this figure be interpreted as a "data need." A data need, as defined in ATSDR's Decision Guide for Identifying Substance-Specific Data Needs Related to Toxicological Profiles (ATSDR 1989a), is substance-specific information necessary to conduct comprehensive public health assessments. Generally, ATSDR defines a data gap more broadly as any substance-specific information missing from the scientific literature.

As seen in Figure 2-3, a large number of gaps exist in current knowledge of the health effects of used mineral-based crankcase oil in humans and animals. Data from humans are limited to two studies in which volunteers were exposed briefly to aerosols of used mineral-based crankcase oil (Dautrebande and Capps 1950; Dautrebande et al. 1951) and two epidemiology studies of persons exposed occupationally to used mineral-based crankcase oil (Clausen and Rastogi 1977; Steineck et al. 1989). Animal data are also limited, consisting of a series of acute oral and dermal toxicity and irritation tests in rodents (API 1980b; Beck et al. 1984; Vemot et al. 1990), two acute-duration inhalation toxicity studies in rodents (Costa and Amdur 1979b; DOT 1983), an acute-to-intermediate-duration dietary study in ducks and pheasants (Eastin et al. 1983), two case reports of poisonings in cattle that grazed in contaminated pastures (Osweiler et al. 1973; Sas 1989), and a number of skin-painting oncogenicity studies in mice (API 1983; Grimmer et al. 1982a, 1982b, 1983; McKee and Plutnick 1989).

FIGURE 2-3. Existing Information on Health Effects of Used Mineral-based Crankcase Oil



Human



Animal

● Existing Studies

2.10.2 Identification of Data Needs

Acute-Duration Exposure. Studies exist that have examined the effects of acute-duration inhalation, oral, and dermal exposures to used mineral-based crankcase oil in humans and/or animals (API 1980b; Beck et al. 1984; Costa and Amdur 1979b; Dautrebande and Capps 1950; Dautrebande et al. 1951; DOT 1983; Eastin et al. 1983; Vemot et al. 1990). However, no MRLs were derived for acute-duration inhalation or oral exposure to used mineral-based crankcase oil. This was due in part to the lack of reliable dose-response data and in part to the variability in the composition of batches of used mineral-based crankcase oil. Inhalation studies in humans identified mild eye, nose, and throat irritation as toxic end points (Dautrebande and Capps 1950; Dautrebande et al. 1951), but a study with guinea pigs showed no effect on pulmonary function (Costa and Amdur 1979a). In addition, an acute-duration inhalation study in rats showed no overt effects on either behavior or on pathology at necropsy (DOT 1983). An acute oral LD_{50} study in rats showed no lethality, but oily diarrhea was observed following administration of large oral doses (API 1980b; Beck et al. 1984; Vemot et al. 1990). Acute-duration dermal studies in rabbits identified slight-to-moderate skin irritation, but only negligible eye irritation was reported (API 1980b; Beck et al. 1984; Vemot et al. 1990). Repeated dermal doses in rabbits resulted in emaciation, but no specific systemic cause for the weight loss was identified (API 1980b; Beck et al. 1984; Vemot et al. 1990). This suggests that toxicity other than mild eye and skin irritation is possible with acute-duration exposures. The emaciation is probably due to reduced food intake, effects on intestinal absorption, general systemic toxicity or stress which alters metabolism, or a combination of these factors. What is required is a study in which food intake is carefully measured, histopathology and clinical chemistry is performed, and metal content of major organs is determined. Additional acute-duration inhalation studies are not warranted at this time since inhalation exposure to used mineral-based crankcase oil is unlikely.

Intermediate-Duration Exposure. No intermediate-duration inhalation or oral MRLs were derived for used mineral-based crankcase oil because of the absence of reliable dose-response data and variability in the composition of different batches of used mineral-based crankcase oil. No intermediate-duration inhalation studies were located, but such studies do not appear necessary at this time since inhalation exposures are not expected to occur unless the oil becomes aerosolized. Case reports of intermediate-duration oral exposures in cattle suggest that effects associated with metal toxicity may occur (Osweiler et al. 1973; Sas 1989). In addition, a study examining the health effects

associated with intermediate-duration exposure of monkeys to *unused* mineral-based crankcase oil indicated that severe gastrointestinal damage may occur with ingestion (Lushbaugh et al. 1950). Since used mineral-based crankcase oil may contain the same chemical that was responsible for the effects of exposure to the unused oil, similar effects may occur with intermediate-duration ingestion of used oil. These effects have not been thoroughly studied, and a well-conducted 90-day oral study in animals could provide valuable information about target organs in humans and exposure levels associated with toxicity. The only intermediate-duration dermal study that was located was a dermal sensitization study in guinea pigs that reported only effects on the skin (API 1980b; Beck et al. 1984; Vemot et al. 1990). As indicated in the repeated-dose, acute-duration study described above, target organs other than the skin are possible. Therefore, a 21- or 90-day dermal toxicity study in animals could provide valuable information regarding effects likely to occur in persons with intermediate-duration dermal exposures. The variations in used oil composition must be adequately dealt with for such studies to be meaningful. Pooled composite samples or possibly several pooled samples from vehicles driven a range of mileages would be required. A dermal study could provide information on potential systemic effects of used oil applied to the skin. Four consecutive daily applications of used motor oil to the skin of mice produced adducts in the DNA of the lung (Schoket et al. 1989). Evaluation of DNA adducts in the major organs, assays for red blood cell δ-ALAD to detect lead poisoning, measurement of concentrations of specific metals in serum and selected organs, and histopathologic examination of major organs should be performed in such a study.

Chronic-Duration Exposure and Cancer. No chronic-duration inhalation or oral MRLs were derived for used mineral-based crankcase oil because of the absence of reliable dose-response data and variability in the composition of different batches of used mineral-based crankcase oil. The only information located regarding the effects of chronic-duration exposure to used mineral-based crankcase oil in humans was a report of health effects experienced by a number of mechanics and other auto workers from 10 auto shops in Denmark (Clausen and Rastogi 1977). Additional studies examining health effects of chronic oral and dermal exposures among exposed populations would provide valuable information regarding the potential for such effects in similarly exposed populations. Chronic-duration animal studies with used mineral-based crankcase oil are limited to studies whose intent was to identify dermal carcinogenicity, and therefore other types of toxicity were not examined (Grimmer et al. 1982a, 1982b, 1983; McKee and Plutnick 1989) or were examined incompletely (API 1983). Additional animal studies examining the health effects of chronic-duration oral and dermal exposures to used mineral-based crankcase oil, as well as water-soluble constituents of the used oil are

necessary to identify health effects that may be associated with such exposures. For the chronic oral study, daily administration of the water soluble components of used motor oil may be most useful for assessing risk around hazardous waste sites, as these components may leach into drinking water.

The only information located regarding carcinogenicity in humans after exposure to used mineral-based crankcase oil was a study that showed that persons with occupational exposure are not at an increased risk of renal pelvic or bladder cancer (Steineck et al. 1989). No information was located regarding the occurrence of other types of cancer in humans exposed to used mineral-based crankcase oil. Studies in mice have demonstrated that chronic-duration cutaneous exposure to used mineralbased crankcase oil increases the incidence of skin tumors (API 1983; Grimmer et al. 1982a, 1982b, 1983; McKee and Plutnick 1989). Thus, similar tumors may occur in exposed human populations. Additional studies directed at determining the cancer incidence in persons with chronic-duration dermal exposure to used mineral-based crankcase oil (occupational studies) could provide information on whether similar effects may occur in chronically exposed populations. However, epidemiology studies in which the effects of chronic dermal exposure to used motor oil are evaluated are often not very practical. The individuals who have repeated contact with used oil in service stations and garages do not, as a group, remain on the job for extended periods of time; hence a suitable population is almost impossible to find. A study examining the carcinogenicity of chronic-duration oral exposures to used mineral-based crankcase oil in rodents may provide information on whether persons exposed similarly might expect an increased risk of cancer.

Genotoxicity. No genotoxicity data were located regarding used mineral-based crankcase oil in mammalian systems. However, results from Salmonella mutation assays (Dutcher et al. 1986; Granella and Clonfero 1991; EPA 1980d; Manabe et al. 1984; Pasquini and Monarca 1983; Payne et al. 1978; Peake and Parker 1980; Schreiner and Mackerer 1982) and the formation of DNA adducts in the skin following cutaneous application of used mineral-based crankcase oil (Carmichael et al. 1990, 1992; Schoket et al. 1989) suggest that this substance may be mutagenic in humans. Additional *in vitro* mutagenicity tests in mammalian cells and *in vivo* analyses such as tests for chromosomal aberrations or micronucleus tests could provide more conclusive evidence for the genotoxicity of used mineral-based crankcase oil in humans. In addition, tests assessing DNA adducts in tissues of animals exposed experimentally could provide useful information about the distribution of such changes in the body.

Reproductive Toxicity. No information was located regarding the reproductive toxicity of used mineral-based crankcase oil in humans or animals. As the oral and dermal routes are the most likely routes of exposure to used mineral-based crankcase oil, intermediate-duration studies in animals using these routes of exposure and examining the effects on reproductive organs would be a useful first step in assessing the reproductive toxicity of this substance.

Developmental Toxicity. The only information located regarding developmental toxicity came from an egg-painting study using ducks and quail (Hoffman et al. 1982a) and another study in which used and virgin crankcase oil was injected into mallard eggs (Hoffmann and Albers 1984). These studies suggest that the developing bird fetus may be at risk when exposed to chemicals found in used mineral-based crankcase oil. Studies in mammals designed to assess the toxicity of used mineralbased crankcase oil following oral or dermal exposure during gestation could provide valuable information regarding the likelihood of developmental toxicity with maternal exposure. Toxicokinetic studies of known contaminants such as lead or PAHs in pregnant animals when administered in a mineral oil carrier could also provide information on the likelihood of fetal exposure.

Immunotoxicity. The only information located regarding immunological effects of used mineralbased crankcase oil came from a dermal sensitization study using guinea pigs that showed no sensitization after intermediate-duration exposure (API 1980b; Beck et al. 1984; Vernot et al. 1990). However, the positive control group also failed to show a potentiation of the dermal response in this study. Thus, the results are inconclusive. Since workers with exposure to used mineral-based crankcase oil report a moderate incidence of skin rashes on the hands and arms (not specified whether the rashes were allergic in nature) (Clausen and Rastogi 1977), an additional skin sensitization study could provide valuable information. In addition, if a subchronic oral or dermal exposure study in animals were conducted, examination of effects on lymphoid tissue and leukocyte content could also provide information indicating whether the immune system may be adversely affected. A test of bacterial infectivity could also provide information regarding immune function. Also, measurement of blood levels of immunoglobulins may provide subtle, but useful, information.

Neurotoxicity. Information regarding the neurotoxic potential of used mineral-based crankcase oil is largely anecdotal in nature. Limited information from a report of health effects in mechanics and other auto workers suggests that neurotoxic effects may be associated with occupational exposure to used mineral-based crankcase oil (Clausen and Rastogi 1977). In addition, cattle that ingested used

mineral-based crankcase oil showed marked neurotoxicity (Osweiler et al. 1973; Sas 1989). Additional experimental studies in animals examining the potential for neurotoxicity after oral and dermal exposures under controlled conditions could provide valuable information as to whether persons exposed to used mineral-based crankcase oil under such conditions may be expected to experience adverse neurological effects. Neurotoxicity of crankcase oils can be evaluated as part of the subchronic oral or dermal studies.

Epidemiological and Human Dosimetry Studies. Limited information was located regarding epidemiological analyses of exposed populations. Renal, pelvic, and bladder cancers among workers exposed to motor oil was examined in a study assessing the incidence of these tumor types among different occupational groups (Steineck et al. 1989), and health effects and blood lead levels were examined among workers in a small number of auto shops in Denmark (Clausen and Rastogi 1977). Assessing the health effects among various groups of workers occupationally exposed to used mineral-based crankcase oil could be valuable in establishing cause/effect relationships that would be helpful in monitoring populations living near hazardous waste sites.

Biomarkers of Exposure and Effect

Exposure. No information was located regarding biomarkers that are specific for exposure to used mineral-based crankcase oil. The available literature on health effects of used mineral-based crankcase oil indicates a few biomarkers of exposure for specific chemicals found in used mineral-based crankcase oil such as metal content of the blood (Clausen and Rastogi 1977; Osweiler et al. 1973; Sas 1989) or DNA-adduct formation with PAHs (Carmichael et al. 1990; Schoket et al. 1989) . However, since the toxicity of used mineral-based crankcase oil is expected to vary with chemical content, effective biomarkers for exposure would also be expected to vary with chemical content of the oil. Thus, additional research regarding biomarkers of exposure should be addressed on a chemical-specific basis.

Effect. As discussed above for biomarkers of exposure, no information was located regarding biomarkers that were specific for effects of used mineral-based crankcase oil. Examination of the literature on health effects of used mineral-based crankcase oil indicated that DNA-adduct formation by PAHs may be an indicator of the genotoxic potential of used mineral-based crankcase oil. However, the chemical content of used mineral-based crankcase oil is expected to vary greatly, and

additional research regarding other biomarkers of effect should be addressed on a chemical-specific basis.

Absorption, Distribution, Metabolism, and Excretion. No information was located regarding the metabolism of used mineral-based crankcase oil, and only very limited animal data were located regarding absorption, distribution, and excretion following oral and dermal exposures to used mineral-based crankcase oil (API 1980b; Beck et al. 1984; Blakley and Brockman 1976; Carmichael et al. 1990; Osweiler et al. 1973; Sas 1989; Schoket et al. 1989; Vemot et al. 1990). No other quantitative data were located regarding the absorption, distribution, metabolism, and excretion of used mineral-based crankcase oil following inhalation, oral, or dermal exposure in humans or animals. Therefore, acute, intermediate, and chronic exposure data are needed in order to assess the relative rates and extent of absorption, distribution, metabolism, and excretion with respect to all three routes of exposure, as well as with respect to time and dose for the primary chemical constituents of concern. A definitive pharmacokinetics study of absorption of PAHs, lead, and other potentially toxic materials administered in mineral oil through the intact skin of animals would be useful. Once that information is available, the actual hazard associated with dermal exposure can be estimated. In addition, animal and human data are needed to identify target organs among multiple species.

Comparative Toxicokinetics. No studies were located regarding comparative toxicokinetics of used mineral-based crankcase oil. Human and animal data are needed in order to examine toxicokinetics across species, i.e., in humans and animals of multiple species. This information is needed in order to identify similar target organs and to adequately assess which animals can serve as the best models for humans.

Methods for Reducing Toxic Effects. Although the mechanisms of absorption of used mineralbased crankcase oil from the gastrointestinal tract and skin have not been studied, methods have been identified that may be useful for reducing absorption of used mineral-based crankcase oil from these areas. For example, washing with mild lipophilic soap has been recommended for removal from exposed skin (Ellenhorn and Barceloux 1988), and induction of emesis, gastric lavage, and administration of activated charcoal and/or osmotic cathartics have been suggested for decreasing gastrointestinal absorption (Ellenhom and Barceloux 1988; Goldfrank et al. 1990; Haddad and Winchester 1990). Additional research into the mechanisms of absorption of used mineral-based crankcase oil and/or chemicals contained in the oil through the skin or the gastrointestinal tract could

provide a basis for developing more specific methods for reducing absorption. No specific methods were identified for reducing the body burden or for interfering with the mechanisms of action of used mineral-based crankcase oil. Such mitigation strategies would best be approached on a substance-specific basis depending on the additives or contaminants in the oil that are believed to be of toxicological significance. Until specific toxic effects are clearly defined, methods for reducing toxic effects must remain generalized.

2.10.3 On-going Studies

No information was located regarding on-going studies evaluating either the health effects or toxicokinetics of used mineral-based crankcase oil.

3. CHEMICAL AND PHYSICAL INFORMATION

3.1 CHEMICAL IDENTITY

Used mineral-based crankcase oil is a complex mixture of low and high (C_{15}-C_{50}) molecular weight aliphatic and aromatic hydrocarbons, lubrication additives, metals, and various organic and inorganic compounds. The chemical composition of used mineral-based crankcase oil varies widely and depends on the original crude oil, the processes used during refining, the efficiency and type of engine the oil is lubricating, the gasoline combustion products, the additives added to the fuel and to the original oil, and the length of time that the oil remains in the engine. The oil is typically 73-80% weight/weight aliphatic hydrocarbons (primarily alkanes and cycloalkanes with l-6 rings); 11-15% monoaromatic hydrocarbons; 2-5% diaromatic hydrocarbons; and 4-8% polyaromatic hydrocarbons (Vasquez-Duhalt 1989). The lubrication additives, which are approximately 20% of the oil, consist primarily of zinc diaryl, molybdenum disulfide, zinc dithiophosphate, metal soaps, and other organometallic compounds. Detergents and dispersants constitute 2-15% of the additives (Vasquez-Duhalt 1989).

Other compounds found in used mineral-based crankcase oil as a result of oil additives include barium, phosphorus, zinc, and some chlorine and bromine compounds (Vermont Agency of Natural Resources 1994). Additional organic and inorganic compounds found in used mineral-based crankcase oil are sulfur, aluminum, arsenic, calcium, chromium, copper, iron, magnesium, manganese, potassium, silicon, sodium, tin, toluene, benzene, xylene, ethylbenzene, and nitrogen, although most organic compounds are destroyed during combustion of used mineral-based crankcase oil as fuel (Canadian Environmental Protection Agency 1994; Vermont Agency of Natural Resources 1994).

Small amounts of PCBs were in the past incorporated into transmission fluids to control swelling of rubber seals (Mueller Associates 1987). PCBs were also detected in 4 of 24 used oil samples at concentrations of 7, 13, 18, and 65 ppm (Suprenant 1983). It should be noted that PCBs have never been used in lubricant additives or in the creation of new motor oils. Because dioxins can form from PCBs, which has severe health implications, production of PCBs for commercial purposes has almost ceased (Hewstone 1994b).

Information regarding the chemical identity of used mineral-based crankcase oil is located in Table 3-l.

3.2 PHYSICAL AND CHEMICAL PROPERTIES

Information regarding the physical and chemical properties of used mineral-based crankcase oil is located in Table 3-2.

TABLE 3-1. Chemical Identity of Used Mineral-based Crankcase Oil *In Vitro*

Characteristic	Information	Reference
Chemical name	Mineral-based crankcase oil	DOE 1989
Synonym(s)	API 79-7; API service classification SAE 30 automotive motor oil; monograde automotive engine oil; multigrade automotive engine oil; marine engine oil; base engine oil; monograde diesel oil; railway diesel oil; marine diesel oil	American Petroleum Institute 1980; DOE 1989
Registered trade name(s)	No data	
Chemical formula	No data	
Chemical composition	Branched alkanes; cycloalkanes; benzenes and alkylbenzenes; naphthalenes; polynuclear aromatic hydrocarbons; linear alkanes; additives; decomposition products; and contaminants	DOE 1989
Identification numbers:		
CAS registry	8002-05-9	IARC 1984
NIOSH RTECS	No data	
EPA hazardous waste	No data	
OHM/TADS	No data	
DOT/UN/NA/IMCO shipping	No data	
HSDB	No data	
NCI	No data	

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

TABLE 3-2. Physical and Chemical Properties of Used Mineral-based Crankcase Oil

Property	Information	Reference
Molecular weight	No data	
Color	Yellow brown to black	DOE 1989
Physical state	Liquid, oily	DOE 1989
Melting point	-34.4°C	DOE 1989
Boiling point	360.0°C	DOE 1989
Density	Not applicable	
Viscosity:		
at 25°C	Variable	
Odor	Lube oil odor	DOE 1989
Odor threshold	No data	
Solubility:	No data	
Water at 20°C	Insoluble	DOE 1989
Organic solvent(s)	No data	
Partition coefficients:		
Log K_{ow}	No data	
Log K_{oc}	No data	
Vapor pressure at 20°C	No data	
Henry's law constant	No data	
Autoignition temperature	≥135°C	DOE 1989
Flashpoint	≥163°C	DOE 1989
Flammability limits	No data	
Conversion factors	No data	
Explosive limits	No data	

4. PRODUCTION, IMPORT, USE, AND DISPOSAL

4.1 PRODUCTION

Used mineral-based crankcase oil is a complex mixture of low and high molecular weight (C_{15}-C_{50}) aliphatic (paraffins, olefins, and acetylene hydrocarbons and their derivatives), aromatic, and naphthenic hydrocarbons, lubrication additives, sulfur, oxygen and nitrogen compounds, and metals. Aliphatic hydrocarbons contain a molecule with carbon atoms in simple or branched chains. Aromatic hydrocarbons are compounds with unsaturated carbons in six-membered rings and properties similar to benzene. Polycyclic aromatic hydrocarbons (PAHs) are complex organic compounds containing 3 or more aromatic rings. The chemical composition of new or used mineral-based crankcase oil varies widely and depends on the original crude oil and the processes used during refining.

Used mineral-based crankcase oil is produced when new mineral-based crankcase oil is subjected to high temperatures and high mechanical strains. The lubricating oil is altered by nitration, cracking of polymers, oxidation, and decomposition of organometallic compounds. Therefore, fuel (gasoline or diesel), water, metals, metal oxides, and other combustion products accumulate in the oil, resulting in used mineral-based crankcase oil (Vasquez-Duhalt 1989). Used mineral-based crankcase oil has been determined to be a source of lead exposure, but other sources like exhaust gas, gasoline, and gear oil can also contribute to lead exposure. Lead emissions are a direct result of lead added to fuel as an antiknock agent in order to increase fuel efficiency. However, because of stringent emissions controls in the late 1980s, atmospheric and human lead levels have dropped drastically with the introduction of unleaded gasoline and the reduction of lead levels in leaded gasoline (Sawyer 1993). Other factors such as the efficiency and type of engine the oil is lubricating, the gasoline combustion products, and the interval between oil changes and mileage driven also alter the chemical composition of used mineral-based crankcase oil.

The production of used mineral-based crankcase oil is expected to be equivalent to the sales of new mineral-based crankcase oil. An estimated 65-68% of the oil is recovered when engine oil is changed; the remaining motor oil is released into the environment while the engine is operating (Tanacredi 1977).

In 1972, EPA estimated that 500 million gallons of used mineral-based crankcase oil was produced in the United States. The American Petroleum Institute (API) estimated that 616 million gallons of used mineral-based crankcase oil were generated in 1974; 81% was collected by automotive shops, and the remaining oil was obtained when automobile owners changed their own oil at home. Production increased to 720 million gallons in 1975, and in 1980, more than 9 million tons were produced. In 1982, 1,350 million gallons of used mineral-based crankcase oil were produced in the United States (API 1974; Brinkman et al. 1982; Maugh 1976; Vasquez-Duhalt 1989; Weinstein 1974).

4.2 IMPORT/EXPORT

No information on the import or export of used mineral-based crankcase oil was located.

4.3 USE

Used mineral-based crankcase oil is burned as a supplemental fuel in industrial steam boilers, domestic oil burners, utility steam boilers, waste disposal incinerators, and rotary cement kilns. It is also used as a component in asphalt and as a dust suppressant on rural roads. Used mineral-based crankcase oil is also re-refined to make lubricating oils (EPA 1974b; NATO 1981; Tanacredi 1977; Weinstein 1974).

In 1974, 31.6-50% of used mineral-based crankcase oil (195-308 million gallons) was utilized as supplemental fuel; 11-16.7% (67-103 million gallons) was incorporated into asphalt for roads; 23.2% (143 million gallons) was used for road oiling; 3.3-9.2% (20-57 million gallons) was re-refined; and 23.0-30.0% (142-185 million gallons) was released into the environment (API 1974; EPA 1972; NATO 1981; Stehouwer 1980; Tanacredi 1977). In 1989, a total of 750 million liters (198 million gallons) of used mineral-based crankcase oil was used as road oil or incorporated into asphalt for roads (DOE 1989).

4.4 DISPOSAL

In 1974, automobile owners who changed their own oil disposed of 69 million gallons of used mineral-based crankcase oil directly onto the ground or into landfills, storm sewers, toilets, and sinks; 37 million gallons were returned to service stations for disposal. A national telephone survey of 4,805

U.S. households found that the disposal methods for the automobile owners who changed their own oil were as follows: oil poured onto the ground-40%; oil placed in trash to be collected-21%; oil taken to gas station or recycled-14%; miscellaneous uses-25% (i.e., used as a lubricant on machinery, stored, used as an insecticide). In rural areas, the most common way to dispose of oil was pouring onto gravel roads and driveways. A Wisconsin survey of automobile owners who changed their own oil found the following breakdown of disposal methods: oil dumped on property-33%; oil taken to service station-15%; oil taken to public dump-11%; oil dumped in storm sewer-11%; oil dumped in garbage can-10%; oil dumped in empty lot-3%; and other-17% (API 1974; Brinkman et al. 1982).

In a Wisconsin survey of service stations, the used mineral-based crankcase oil was disposed of in the following manner: re-refined to lubricating oil-33.8%; re-used as fuel oil-37.2%; re-used for road application-7.4%; reused on a farm-19.3%; dumped on the ground-2.1%; and not known-0.3% (API 1974).

The two main methods for recycling used mineral-based crankcase oil are burning as fuel and rerefining. Used mineral-based crankcase oil can easily be re-processed by settling and filtration to remove water and solids followed by blending with diesel fuel, coal, or residual oil; it can then be burned as a supplemental fuel. Used mineral-based crankcase oil may also be re-refined into a lubricant blending stock. In 1980, 11.4% of used mineral-based crankcase oil was re-refined. Both recycling methods have problems: high levels of metals are released into the atmosphere when used mineral-based crankcase oil is burned, and re-refining processes produce wastes that cause disposal problems. Additionally, the quality of re-refined motor oil is inferior to that of new motor oil because there is no way to assure batch-batch quality of the re-refined base stocks (Stehouwer 1980).

5. POTENTIAL FOR HUMAN EXPOSURE

5.1 OVERVIEW

Used mineral-based crankcase oil is a complex mixture of low and high (C_{15}-C_{50}) molecular weight aliphatic and aromatic hydrocarbons, lubrication additives, metals, and various organic and inorganic compounds. The chemical composition of used mineral-based crankcase oil varies widely and depends on the original crude oil, the processes used during refining, the efficiency and type of engine the oil is lubricating, the gasoline combustion products, the additives added to the fuel and to the original oil, and the length of time that the oil remains in the engine. The oil is typically 73-80% weight/weight aliphatic hydrocarbons (primarily alkanes and cycloalkanes with l-6 rings); 11-15% monoaromatic hydrocarbons; 2-5% diaromatic hydrocarbons; and 4-8% polyaromatic hydrocarbons (Vasquez-Duhalt 1989). The lubrication additives, which are approximately 20% of the oil, consist primarily of zinc diaryl, molybdenum disulfide, zinc dithiophosphate, metal soaps, and other organometallic compounds. Detergents and dispersants constitute 2-15% of the additives (Vasquez-Duhalt 1989).

Other compounds found in used mineral-based crankcase oil as a result of oil additives include barium, phosphorus, zinc, and some chlorine and bromine compounds (Vermont Agency of Natural Resources 1994). Average metal concentrations found in used gasoline engine oil in a 1994 study are barium at 2.73 ppm, beryllium at <0.02 ppm, cadmium at <1.51 ppm, chromium at 3.19 ppm, lead at 47.23 ppm, nickel at <1.40 ppm, and zinc at 1,161 ppm (Vermont Agency of Natural Resources 1994). Levels of barium have decreased dramatically in recent years because barium is no longer is a component in lubricant dispersants. However, zinc concentrations have remained steady because of its use in lubricant detergents (Entropy 1994). Generally, metal levels in automotive-generated oils are higher than the levels found in industrial-generated oils (Mueller Associates 1987). The highest concentrations of metal levels are found in the combustion effluents of automotive-generated used oils rather than in truck-generated oils (Hall et al. 1983a). Prior to the introduction of unleaded gasoline, lead levels in used mineral-based crankcase oil were approximately 13,000 μg/g (Vasquez-Duhalt 1989). In 1983, used oil lead levels had dropped to an average of 1,100 ppm for the 24 samples of used oil tested (Suprenant et al. 1983). In a 1994 study, a lead level of 47.23 ppm was found in used gasoline engine oil (Vermont Agency of Natural Resources 1994). Lead content in used mineral-based

crankcase oil is predominantly a result of piston blow-by of the combustion products of leaded gasoline (Mueller Associates 1987; Vermont Agency of Natural Resources 1994).

Additional organic and inorganic compounds found in used mineral-based crankcase oil are sulfur, aluminum, arsenic, calcium, chromium, copper, iron, magnesium, manganese, potassium, silicon, sodium, tin, toluene, benzene, xylene, ethylbenzene, and nitrogen, although most organic compounds are destroyed during combustion of used mineral-based crankcase oil as fuel (Canadian Environmental Protection Agency 1994; Vermont Agency of Natural Resources 1994). The toxicity of chromium is dependent upon its structural form, as it exists in eight different oxidation states. The most stable forms are the trivalent and hexavalent forms, with the hexavalent form being the most toxic of the various states (Vermont Agency of Natural Resources 1994).

Small amounts of PCBs were in the past incorporated into transmission fluids to control swelling of rubber seals (Mueller Associates 1987). PCBs were also detected in 4 of 24 used oil samples at concentrations of 7, 13, 18, and 65 ppm (Suprenant 1983). It should be noted that PCBs have never been used in lubricant additives or in the creation of new motor oils. Because dioxins can form from PCBs, which has severe health implications, production of PCBs for commercial purposes has almost ceased (Hewstone 1994b). The 24 samples in the Suprenant et al. (1983) study also contained an average concentration of 20 ppm of benz(a)anthracene with benzo(a)pyrene below the detection limit of 5 ppm. Both of these polynuclear aromatics (PNAs) are considered hazardous and carcinogenic to humans (Suprenant 1983).

Used mineral-based crankcase oil may be released directly into the air, water, or soil through fuel burning, re-refining, engine oil leaks, automobile or truck exhaust, uncontrolled disposal into sewers or landfills, and to a lesser extent via environmental spills. Used mineral-based crankcase oil can also be released into the environment as a dust suppressant on rural roads (Canadian Environmental Protection Act 1994). However, in most states strict regulations now prohibit or limit the use of used oil as a dust suppressanf (Mueller Associates 1987). In addition, metal particulates may be released into the atmosphere, depending upon their volatility, through engine exhaust or when used mineral-based crankcase oil is used as supplemental fuel in boilers and incinerators.

In 1983, approximately 699 million gallons of automotive used oil were created. The primary sources of this oil were do-it-yourself oil changers, which accounted for 34.2% of all automotive oil generated.

Sources such as service stations and repair shops accounted for approximately 214.8 million gallons of used automotive oils. The total used oil, including industrial and automotive oils, consisted of 1.2 billion gallons in 1983. Burning was the primary use of used oils, consuming 48.9% or 590.1 million gallons of the total used oil generated in 1983 (Mueller Associates 1987).

The environmental fate of used mineral-based crankcase oil is dependent on its individual components. See other ATSDR profiles for the environmental fate of specific components such as PAHs, lead, cadmium, and zinc (ATSDR 1990c, 1993b, 1992d, 1989b). Chemical and biological processes such as microbial degradation and oxidation that normally degrade hydrocarbons are less effective on used mineral-based crankcase oil because the high molecular weight hydrocarbons and less-soluble aromatics are less susceptible to these environmental processes. Most high molecular weight hydrocarbons will sorb to organic matter in the soil or particulates in the water column and will eventually accumulate in the sediment. Microbial degradation is dependent on the hydrocarbon fractions present in the used mineral-based crankcase oil, the number of microorganisms present in the soil, sediment, or water, and the environmental conditions, for example, temperature. During microbial degradation, metals may be released from the oil phase into the aqueous phase, which increases the persistence, mobility, and toxicity of the metals. Heavy metals may then accumulate in plants, soil, sediments, surface water, and groundwater.

Minimal exposure to the general population may occur when an individual adds engine oil or checks the engine oil level in a vehicle. Dermal exposure appears to be the primary route of occupational exposure for individuals employed in the automotive industry and for individuals who change their oil or otherwise handle engine components contaminated with used mineral-based crankcase oil. However, non-occupational exposure is expected to occur only a few days per year. Other populations potentially exposed either dermally or by inhalation to used mineral-based crankcase oil include individuals living or working in the vicinity of service stations that improperly dispose of used mineral-based crankcase oil, active or inactive oil recycling or re-refining facilities, or roads that are treated with used mineral-based crankcase oil for dust suppression. Individuals who live& work near facilities that bum used mineral-based crankcase oil as a supplemental fuel for boilers or waste disposal incinerators may be exposed to high levels of metal particulates.

Used mineral-based crankcase oil has been found in at least 85 of the 1,430 current or former EPA National Priorities List (NPL) hazardous waste sites (HAZDAT 1996). However, the number of sites

evaluated for used mineral-based crankcase oil is not known. Of these sites, all 85 are located in the United States, and none are located in the Commonwealth of Puerto Rico.

The frequency of these sites within the United States can be seen in Figure 5-l.

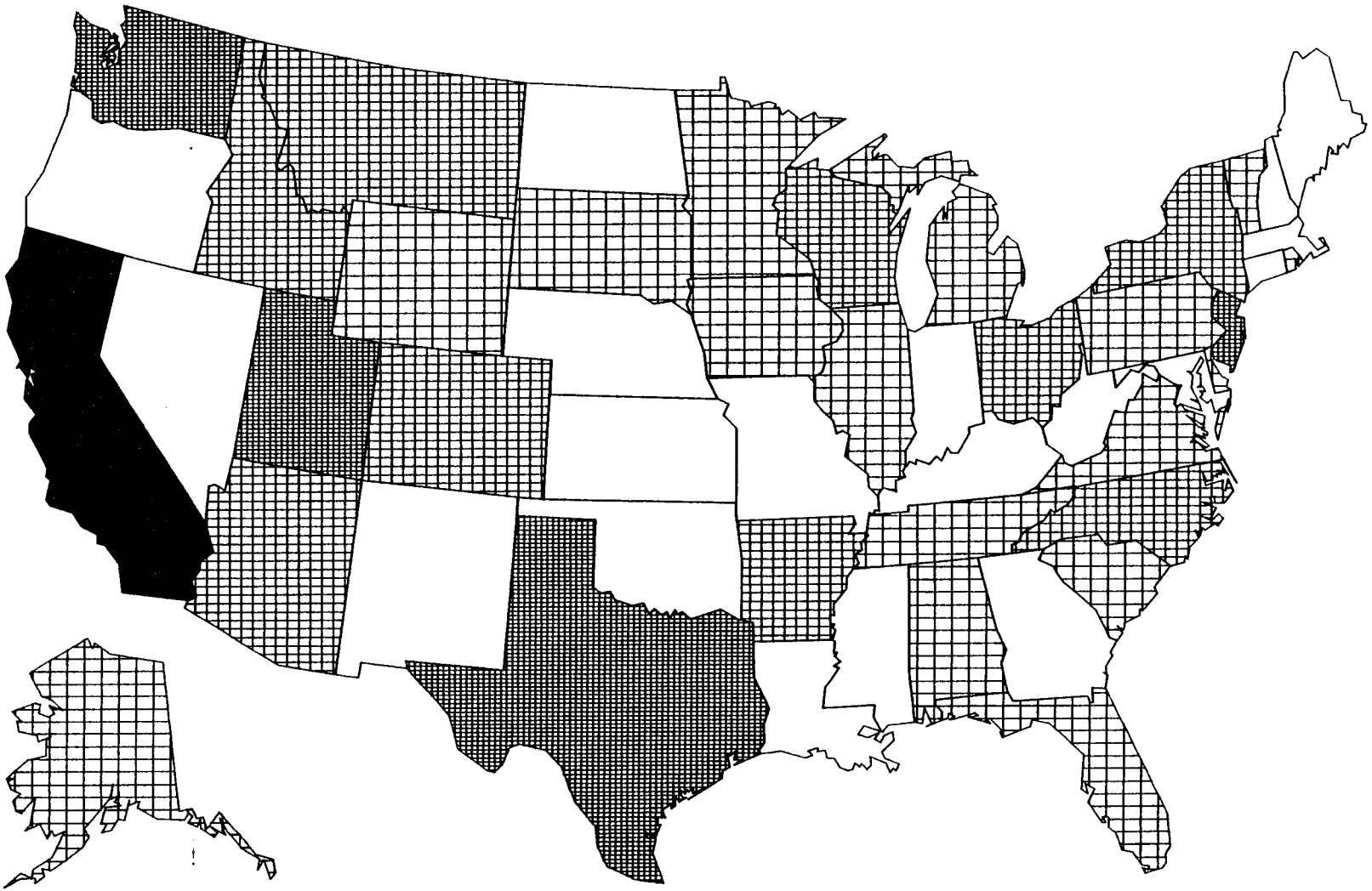
5.2 RELEASES TO THE ENVIRONMENT

Releases of used mineral-based crankcase oil are not required to be reported under SARA Section 313. Consequently, there are no data for this compound in the 1993 Toxics Release Inventory (TRI) (TR193 1995).

5.2.1 Air

The combustion products of used mineral-based crankcase oil may be released to the atmosphere when the oil is used as primary or supplemental fuel in industrial steam boilers, domestic oil burners, utility steam boilers, rotary cement kilns, or waste disposal incinerators. Combustion products of used mineral-based crankcase oil include lead, zinc, chromium, aluminum, nickel, copper, other metal particles, sulfur, nitro-compounds, sulfur dioxide, phosphorus, calcium, hydrochloric acid, and nitrogen oxides (AEC 1974; Canadian Environmental Protection Act 1994; Vasquez-Duhalt 1989). These products may also be released during soot blowing at large boilers or during the cleaning of furnaces or boilers (Mueller Associates 1987). Due to the release of these combustion products, used fuel oil is regulated by its contaminant concentrations and physical characteristics. Used fuel oil which exceeds these maximum concentrations is considered off-specification and is subject to additional regulations regarding the combustion processes (Entropy 1994). According to Entropy (1994), industrial consumers of used oil bum a combined total of 800 million gallons per year for fuel and release 28.06 tons of lead emissions per year including vapor and uncollected particles. It is used in space heaters, commercial boilers, industrial boilers, marine boilers, utility boilers, asphalt plants, cement plants, and pulp-mills. Asphalt plants consume the largest amounts of used oil per year-at 313.4 million gallons, with total lead emissions per year of 9.52 tons. Industrial and utility boilers are the second and third largest consumers of used oil with estimates of 101.4 million gallons and 92.3 million gallons burned per year respectively. Particulate collection efficiency for the different types ranged from 0% efficiency to 95% efficiency for cement plants and steel mills (Entropy 1994). Lead removal efficiency in large boilers >25x106 Btu/hr) averages around 70% as a result of destruction

FIGURE 5-1. FREQUENCY OF NPL SITES WITH USED MINERAL-BASED CRANKCASE OIL CONTAMINATION *



*Derived from HazDat 1995

during combustion. Small boilers emit approximately <50% of the lead in the fuel as emissions (Suprenant et al. 1983). A 1983 study reported 590.1 million gallons of used oil was disposed of by incineration (Mueller Associates 1987).

Prior to the introduction of unleaded gasoline, the combustion of used mineral-based crankcase oil in power plants resulted in high levels of lead emissions (5.0 $\mu g/m^3$) for 24-hour periods. The combustion of used mineral-based crankcase oil originating from automobile engines, collected from service stations, released 20.9 $\mu g/m^3$ respirable particulates despite the use of an electrostatic precipitator. No recent data are available for lead emissions from similar combustion sources (Cooper 1977; Mumford et al. 1986). Oil from crankcase-lubricated engines may also be emitted directly with automobile exhaust at concentrations of 0.1-0.25 L/1,000 km or together with particulate emissions in exhaust (Van Donkelaar 1990). Releases of nitrosamines (animal carcinogens and possible human carcinogens) from heavy duty diesel crankcase emissions were reported when using fresh oil. The amount of nitrosamines released is dependant on the type of oil used and the flow rate of NO_x in the crankcase. Limited evidence exists for a decrease in nitrosamine release as the age of the oil increases. Other sources are automobile interiors and possibly diesel exhaust (Baines 1981). Vapors from the application of used mineral-based crankcase oil to rural roads for dust suppression may also be released to the atmosphere.

5.2.2 Water

Releases of used mineral-based crankcase oil in urban stormwater runoff have been reported. Used mineral-based crankcase oil from engine leaks accumulates on parking lots, in garages, and in the central area of roadway lanes. Stormwater runoff from heavily traveled roadways and bridges is often contaminated with used mineral-based crankcase oil, with aliphatic hydrocarbon concentrations ranging from 0.20 to 24 mg/L (200-24,000 $\mu g/L$) (Hunter et al. 1979; MacKenzie and Hunter 1979; Van Donkelaar 1990; Wakeham 1977). The contaminated runoff generally enters a sanitary, storm, or combined sewer system and may bypass treatment facilities during periods of heavy rainfall (Tanacredi 1977). When more communities separate storm sewers and sanitary sewers, stormwater treatment plants may be able to remove a portion of the used mineral-based crankcase oil from the stormwater. Direct dumping of used mineral-based crankcase oil into storm drains is a result of motorists changing their own engine oil and not having a convenient collection facility available for disposal. An estimated 7 million gallons out of 340 million gallons of oil drained from automobiles per year are

dumped into sewers (Suprenant et al. 1983). Contaminated water ultimately reaches streams, rivers, lakes, and sediments because most of the associated hydrocarbons and metals are resistant to traditional waste water treatment methods (Falahi-Ardakani 1984; Farrington and Quinn 1973; Freestone 1979; Hunter et al. 1979; Latimer et al. 1990; MacKenzie and Hunter 1979; Tanacredi 1977; Van Donkelaar 1990; Wakeham 1977).

Hydrocarbons sampled from urban runoff at commercial, residential, and interstate highway sites displayed the same chromatographic characteristics and PAH distribution as used mineral-based crankcase oil. Stormwater runoff from an industrial site in Rhode Island used by oil distributors, scrap metal dealers, and metal finishers contained primarily hydrocarbons (96%) associated with used mineral-based crankcase oil (Latimer et al. 1990). Runoff from roads treated for dust suppression has been hypothesized as a major mechanism of transport for oil leaving the road surface from the months of September through June on a road sampled in New Jersey. Soil samples taken at the base of a drain pipe carrying water away from the treated road showed a lead concentration of 87 mg/kg. A stream receiving runoff from the treated road showed two oil patches which, when analyzed, contained hydrocarbons similar to those found in waste crankcase oil (Freestone 1972). From the data, it is clear that hydrocarbons and heavy metals from used mineral-based crankcase oil are distributed widely in the environment, especially where industrial activity and motor vehicles are found, or where used oil is incinerated, otherwise utilized, or dumped. Surface water, especially runoff from roads, streets, bridges, and sediments in streams and lakes, contains considerable amounts of these materials. Hence, the potential risk to human health and to the environment is significant.

5.2.3 Soil

In 1983, an estimated 50-80 million gallons of used oil was used as a dust suppressant for dirt roads in the United States. At the time of this estimate, road oiling was banned in 8 states and regulated in 13 additional states (Suprenant et al. 1983). This oil can be a source of nearby soil and water contamination because of runoff and leaching. The rates of removal are highly variable ranging from 3-5% removal to as high as 99% removal (Canadian Environmental Protection Act 1994; Freestone 1972). The rate of removal varies as a result of the environmental conditions the road is subjected to and the condition and materials of the road itself. Additional removal of used mineral-based crankcase oil is facilitated by other processes such as adhesion to tires and chemical and biological

degradation (Freestone 1972; Raymond et al 1976; Rudolph 1980; Mueller Associates 1987). The majority of the oil does not penetrate the road and remains close to the surface.

A simulation of oil application on a roadbed showed that the oil did not penetrate to depths deeper than 1 cm, with the largest concentration being within the top few millimeters of the soil. Lead concentrations in soil in a drainage ditch near (exact distance not stated) an oil-treated road ranged from 50 to 136 mg/kg. Concentrations of lead in soil samples taken from a wheat field 47.7 meters from the oil-treated road averaged 24 mg/kg, which is higher than expected for the lead background levels near a rural road (Falahi-Ardakani 1984; Freestone 1979). Other chemicals detected in runoff include zinc, aluminum, sodium, calcium, and organic compounds such as phenol and chlorophenol (Canadian Environmental Protection Act 1994). Solid wastes containing metals, chlorinated solvents, PAHs, and other organics that are created during re-refining are often disposed of in landfills or lagoons or incorporated into road building materials (Mueller Associates 1987; Weinstein et al. 1982). Lead levels in soil near a waste oil re-refinery in Oklahoma City, Oklahoma, which is on the NPL, have been measured as high as 4,810 mg/kg (ATSDR 1990a).

5.3 ENVIRONMENTAL FATE

5.3.1 Transport and Partitioning

Used mineral-based crankcase oil is a complex mixture of aliphatic and aromatic hydrocarbons, metals, and other additives. Upon release to the environment, used mineral-based crankcase oil is not transported as a mixture, but rather, the various components partition to the atmosphere, water, or soil according to their different physical and chemical properties. Therefore, a mixture of used mineral-based crankcase oil is unlikely to be found intact in various media. However, small and moderate spills on soil may remain entrained in the soil near or on the surface. The partitioning behavior of metals is dependent on the form that the metals are in when released into the environment (DOE 1989). Metals released during combustion transform and partition into four different forms: solids that remain in the combustion process, flyash, solid phase product streams, and vapor phase materials (Entropy 1994). The fate of the oil that is directly released to the soil is determined by the nature and the organic content of the soil. Soils with high organic content will tend to absorb the oil and prevent its spread downward as opposed to permeable soil with little organic matter, which will allow the oil to migrate downward and possibly contaminate groundwater supplies (Raymond et al. 1976).

The majority of the hydrocarbons in used mineral-based crankcase oil are of high molecular weight and will volatilize more slowly because of low estimated vapor pressures (<0.001 mmHg at 25°C). Lower molecular weight hydrocarbons will volatilize quickly from the soil surface to the atmosphere. However, if used mineral-based crankcase oil is spilled on soil, volatilization may be the major loss mechanism over long periods of time because the oil is resistant to leaching and degradation and characteristically remains on the soil surface (DOE 1989). Abiotic degradation was measured in a laboratory weathering experiment by placing a mixture of 80% used mineral-based crankcase oil and 20% No. 6 sludge (a mixture typically applied to roads) into a shallow pan. The mixture was subjected to simulated weathering stresses (wind and sunlight for 12 days); a 16-18% loss of weight was attributed to volatilization (Freestone 1979).

Hydrocarbons, such as highly branched alkanes and PAHs with three or more rings, are transported in soil runoff to surface water and settle out of the water cohunn into the sediment where they may persist for many years (Brown et al. 1985; Wakeham and Carpenter 1976). Most hydrocarbons in used mineral-based crankcase oil have estimated water solubilities of less than 1 mg/L and soil partition coefficients of 10,000-l,000,000. These values indicate that used mineral-based crankcase oil will strongly adsorb to soils. Low molecular weight nitrogen- and sulfur-containing molecules, various additives, and solubilized metals in the mixture may be more mobile (DOE 1989; Vasquez-Duhalt and Greppin 1986).

When exposed to water, the various water-soluble fractions of used mineral-based crankcase oil will separate with their concentrations increasing with increasing agitation. Concentrations released from a 1:10 oil-to-water mixture have ranged from 54.4 μg/L to 198 μg/L for benzene, 516 μg/L to 781 μg/L for toluene, a nondeductible level to 0.17 μg/L for ethylbenzene, and 172 μg/L to 339 μg/L for xylenes. The ranges were affected by the length of agitation with the lower concentration being measured after 0.5 hours and the higher concentration being measured after 24 hours of agitation. These ranges provide the upper ranges of concentrations that can be found in groundwater where the soil is contaminated'with used motor oil. Other water soluble fractions of used motor oil include napthalene, 1-methylnapthalene, 2-methylnapthalene, acenaphthylene, phenanthrene, anthracene, fluoranthene, aenapthene, pyrene, and chrysene. Additional compounds detected in the water soluble fractions include napthalene, 1-methylnapthalene, 2-methylnapthalene, phenanthrene, and acenaphthene. In the previous study, iron, copper, cadmium, lead, nickel, and zinc were also detected in the water soluble fractions (Chen et al. 1994).

Studies have shown that used lubricating oil when applied to soil degrades without significant contamination of the surrounding soil and groundwater. This degradation is due to bacteria and fungi which can degrade the components of used lubricating oil (Neal et al. 1977; Rittmann and Johnson 1989). The degradation process is faster when the oil is applied to soil that has been previously exposed to oil due to the increased numbers of the degrading bacteria (Rittmann and Johnson 1989). To increase the degrading ability of the soil, oil-degrading bacteria that have been cultured in a lab can be applied to the soil. The speed of the degradation process decreases after a period of time due to the increase in concentrations of oil components that are harder to degrade (Rittmann and Johnson 1989). Another method of increasing the degradation of oil involves the addition of nitrogen and phosphorus to the soil (Neal et al. 1977; Rittmann and Johnson 1989). An oil decomposition rate of approximately 1 pound per cubic foot of soil per month was recorded in one study when commercial fertilisers were added to the soil (Neal et al. 1977). Additional increases in the rate of degradation can be obtained by tilling the soil to provide aeration (Elsavage and Sexstone 1989; Raymond et al. 1976; Rittmann and Johnson 1989). A study of soil degradation of waste oil emulsion used as a coolant showed a 96% decrease in the concentration of pristane and hexadecane. This decrease was not seen in sterilized soil. Soil with a higher rate of hexadecane biodegradation also showed a corresponding higher rate of respiration. The fatty acids contained in the waste emulsion were broken down within 28 days. Plants were observed to grow on the soil immediately after cessation of the oil application, although it must be noted that in this study the waste oil did not contain the metals found in waste crankcase oil. In soil cores, the majority of the applied compounds remained within the top 48 cm, and the components were not detected in significant concentrations in the groundwater below the site (Elsavage and Sexstone 1989).

Used mineral-based crankcase oil applied to rural roads for dust suppression can be transported to the air, water, and soil by several mechanisms: volatilization, runoff, capillary action, adhesion to dust particles, or adhesion to passing vehicles (Freestone 1972, 1979). Factors affecting the transport of used mineral-based crankcase oil include its viscosity, volatility, presence of surface-active agents, soil porosity, weather conditions, time to first rain after oil application, presence of oil-consuming microorganisms, and road conditions. Only 1% of the estimated amount of used mineral-based crankcase oil applied to two rural roads in New Jersey remained on the top 1 inch of the road surface material (Freestone 1979). This estimate was based on calculations using measurements of oil in the surface of the road, and assumptions that two applications were completed per year for 12-20 years at an application rate of 0.05 gallon/foot^2 for each application. The majority of oil transport occurred via

runoff after the first few rainfalls following the oil application. During dry periods, the primary forms of transport were volatilization and dust transport. Dust particles may be carried by wind and contaminate crops located near the oiled roads. Samples were taken from oil on the banks of a stream near a road treated with used mineral-based crankcase oil and from the treated road. Fluorescent spectral analysis displayed similar hydrocarbon properties from each sample, indicating that the oil was reaching the stream and was deposited on the banks during a period of high stream flow (Freestone 1979).

Hydrocarbons from used mineral-based crankcase oil in stormwater were associated primarily with particulates (86.4%), indicating that adsorption to particles is an integral method of transport (Freestone 1979; Hunter et al. 1979). Once used mineral-based crankcase oil becomes adsorbed to particulate matter, diaromatics are lost through natural weathering (MacKenzie and Hunter 1979). During a storm, the particulates are washed into the sewers and are eventually deposited in the sediments of the receiving water, especially if combined sewers are utilized and the treatment facility is bypassed (Freestone 1979; Hunter et al. 1979; MacKenzie and Hunter 1979). In one laboratory simulation of oil transport by runoff from oil-treated sand roads, the primary mechanism of transport was flotation of oil-wet sand particles. After two simulated monthly rainfalls, 20% of the oil-wet sand particles were transported by runoff from the road surface, and the applied used mineral-based crankcase oil had leached 1 mm into the clay surface of the road (Freestone 1979).

Used mineral-based crankcase oil can affect marine organisms in several different ways. These effects are categorized as lethal and sublethal toxicity by contact, physical coating by the oil, incorporation of the oil into tissues, and habitat alteration (Moore and Dwyer 1974). The effects of used mineral-based crankcase oil on marine organisms depends upon the nature of the oil to which the organism is exposed. The PAHs present in oil are known carcinogens and are found throughout the ocean in the residual fractions of crude oil and can concentrate in animal tissue (Hyland and Schneider 1976). Uptake of PAHs in marine fish and shellfish occurs via absorption of hydrocarbons on the skin, via dissolved hydrocarbons from the water diffusing through the skin, by transport across the gill membranes, and through ingestion of hydrocarbon-contaminated food (Connell and Miller 1981). This uptake is due primarily to the fact that aquatic organisms reach chemical homeostasis with the surrounding water (Moore and Dwyer 1974).

In Williams et al., lobsters (*Homarus americanus*) were exposed to a surface slick of 25 mL diesel oil. PAH accumulations were observed in hepatopancreas tissue and tail muscle at days 3 and 4 of the experiment. The route of exposure for the lobsters was determined to be via absorption of dissolved hydrocarbons through the gills (Williams et al 1989b). Nine PAHs commonly detected in used mineral-based crankcase oil were found to bioconcentrate in bivalve mollusks (*Mercenaria mercenariu*) within 48 hours of initial exposure (*in vitro* exposure for 48 hours). Uptake of the PAHs was directly related to the weight of the mollusks. PAHs were maintained at detectable levels (the average total PAH concentration was 1522.55 nglg wet tissue weight; the detection limit not reported) at the end of a 45-day depuration period, suggesting that mollusks may not be able to metabolize PAHs into water-soluble metabolites that are easily secreted (Tanacredi and Cardenas 1991). Laboratory studies on *Melitu nitidu* Smith, an estuarine amphipod, showed decreased survivability among the juveniles when exposed to 100 ppm waste oil in sediments. The females' ability to reproduce decreased as the sediment concentration of waste oil increased. This was due to morphological abnormalities in the reproductive structures (Borowsky et al. 1993).

Studies on exposure of fish to used mineral-based crankcase oil have shown that rainbow trout *(Oncorhynchus mykiss)* exposed to waste crankcase oil exhibited an increase in liver, kidney, and heart mixed-function oxidase (MFO) activity along with a corresponding increase in bile metabolites. Maximum levels were reached in days 1-4 and returned to basal levels by day 12. The threshold level for increased liver activity was 3 mg of PACs (polycyclic aromatic compounds) per kilogram of fish (Upshall et al. 1993). Carp *(Cyprinus carpio)* exposed to 50 ppb diesel 2 oil within 3 days exhibited an increase of bile fluorescence to a level 5.8-fold higher than unexposed carp. A saturation level representing a 12-fold increase in bile fluorescence was reached after 12 days of exposure (Britvic et al. 1992). Used mineral-based crankcase oil has also been shown to have negative effects on the growth of tadpoles *(Hyla cinerea)* as compared to the control groups (Mahaney 1994).

Earthworms found in the soil 10 meters from a roadway were soil-purged and found to accumulate up to 670 ppm cadmium and 331.4 ppm lead, attributed to used mineral-based crankcase oils deposited on road surfaces. These metal levels may be lethal to earthworm predators (such as amphibians, reptiles, birds, and mammals) and may have a detrimental effect on the food chain (Gish and Christensen 1973). Metals can also accumulate in roadside plants through foliar absorption or root uptake (Falahi-Ardakani 1984).

5.3.2 Transformation and Degradation

5.3.2.1 Air

The major removal processes of particles in the air, including aerosols of used mineral-based crankcase oil, are coagulation, which leads to larger particle sizes that sediment out of the atmosphere and scavenging by precipitation. Hydrocarbons are removed from the air by chemical and photochemical reactions. Many of these reactions lead to the formation of free radicals. When photosensitized, metal oxides may promote the oxidation of hydrocarbons (Manahan 1979). Photochemical transformation has been considered to be the most important method of atmospheric decomposition of PAH in both the gas phase and as particulates (Baek et al. 1991).

When used mineral-based crankcase oil is burned, metals, organics, sulfur dioxide, nitrogen dioxide, acid halides, and particulates are released into the atmosphere. Metals such as lead and zinc, in particular, are emitted in substantial quantities. The majority of organic compounds in used mineral-based crankcase oil are destroyed during combustion. When used mineral-based crankcase oil is applied to roads as a dust suppressant, volatilization can occur which results in its components being released into the atmosphere (Canadian Environmental Protection Act 1994). Moderately volatile metals such as arsenic, cadmium, lead, zinc, and nickel compounds will nucleate to form particles in the submicron size range. Metals of low volatility such as barium, chromium, nickel, and tin will either vaporize incompletely and nucleate onto solids emitted during combustion or form large-sized particles (Entropy 1994).

5.3.2.2 Water

When automotive lubricating oil comes into contact with water, a partitioning of various volatile organic compounds, polynuclear aromatic hydrocarbons, and metals takes place. The hydrocarbons found in motor oil consist mainly of napthenes and aromatic compounds with smaller amounts of paraffins. The ratio of these constituents determines the physical properties of the oil such as viscosity and viscosity-temperature-pressure characteristics. In a study by Chen et al. (1994), the partition coefficients of aromatic compounds from motor oil to water were estimated using Raoult's law to determine equilibrium conditions for the water-to-oil positioning. Compounds detected in the water-soluble fractions of used motor oil include naphthalene, 1-methylnaphthalene, 2-methylnaphthalene,

acenaphthalene, phenanthrene, anthracene, fluoranthene, acenaphthene, pyrene, and chrysene. No phenols were detected in the water-soluble fractions. The metals detected in the water-soluble fractions were iron, copper, lead, nickel, and zinc. The concentrations of the solutes introduced into the water increased with increasing agitation of the oil-water mixture (Chen et a1.1994).

The various constituents of used mineral-based crankcase oil undergo several weathering processes following a spill. These include volatilization, sinking, emulsification, agglomeration, photodegradation, and biodegradation, depending on the chemical and physical nature of the compounds (Canadian Environmental Protection Act 1994).

Metals found in used mineral-based crankcase oil did not inhibit the growth of *Pseudomonas fluorescent* bacteria, possibly because the metals were not in a form that the bacteria could assimilate. However, when the metabolic rate exceeded the growth rate during bacterial biodegradation, metals were transferred from the oil phase to the aqueous phase. A fraction of the metals in the aqueous phase was immobilized by adsorption, precipitation, or assimilation during bacterial growth. The amount of metals solubilized was dependent on the equilibrium between growth and metabolism. The transfer from the oil phase to the aqueous phase involved an increase in metal persistence, mobility, and toxicity (Vasquez-Duhalt and Greppin 1986).

5.3.2.3 Sediment and Soil

Used mineral-based crankcase oil released to the soil surface is subject to weathering stresses because the two primary loss mechanisms, volatilization and aerobic microbial degradation, are very slow. Microbial degradation of hydrocarbons is highly dependent on the chemical and physical properties of the hydrocarbon fractions present and on environmental conditions. When used mineral-based crankcase oil penetrates through the soil to the soil/groundwater interface, microbial degradation and hydrolysis processes, which are effective in degrading typical hydrocarbons, are not effective in degrading the less soluble aromatic and high molecular weight aliphatic constituents. Used mineral-based crankcase oil is also resistant to oxidation because anti-oxidants are added to crankcase oil when originally formulated (DOE 1989). Initial resistance to biodegradation may be attributed to the inability of the large hydrocarbon molecules to pass through the cell walls to be degraded within the bacteria. Consequently, the bacteria use extracellular enzymes, and therefore biodegradation is delayed during the adaptation period (DOE 1989). In soils that were previously exposed to oil, the rate of

degradation increased because of the maintenance of a microbial population with the capacity to degrade hydrocarbons (Elsavage and Sexstone 1989; Neal et al. 1977).

Sediments in the Delaware River near the outfall of a sewage treatment plant were sampled, and the sulfur profile of the hydrocarbons was found to be analogous to the hydrocarbon profile of used mineral-based crankcase oil and stormwater runoff. The presence of high-boiling aromatic sulfur compounds and other hydrocarbons suggested that degradation did not occur in the sediments (MacKenzie and Hunter 1979).

A single application of 2.98 L/m^2 of used mineral-based crankcase oil from automobiles and trucks to field soil plots in Pennsylvania (silt loam), Oklahoma (sandy loam), and Texas (clay loam) was degraded by 55-82% (oil from automobiles) and 24-53% (oil from trucks) over 1 year (Raymond et al. 1976). Concentrations in fertilized fields of used mineral-based crankcase oil from automobiles and trucks decreased by 71-82% and 46-77%, respectively. The maximum rate of degradation was 0.60 L/m^2 each month. Cold ambient temperatures appeared to decrease the rate of degradation, and fertilizer application had little effect on the degradation until 50% of the oil was degraded. Following 50% degradation of the applied oil, the fertilizer-treated plots exhibited degradation rates 20% faster in Pennsylvania and 4% faster in Texas, compared to the unfertilized plots. The fertilizer-treated plots did not have significantly more used mineral-based crankcase oil degrading microorganisms compared to the unfertilized plots. Most of the degradation activity occurred in the top 10 cm of the soil, suggesting aerobic degradation. Most seeds germinated in treated and control plots; however, few plants survived and developed normally in both plots. Analyses suggested that an accumulation of lead affected the plants (Raymond et al. 1976).

Biodegradation of used mineral-based crankcase oil on road surfaces is minimal compared to losses from volatilization and runoff. Nevertheless, the degradation rate of used mineral-based crankcase oil on road surface material depends on the presence of hydrocarbon-consuming microorganisms, moisture content of soil, concentration of nitrogen and phosphorus, and ambient temperatures (Freestone 1979).

5.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

5.4.1 Air

Used mineral-based crankcase oil is not monitored in the air as a mixture but rather by its individual components, such as lead particulates and total hydrocarbons. The individual components may enter the atmosphere when used mineral-based crankcase oil is used as a fuel for boilers or waste disposal incinerators (API 1975; Cooper 1977; EPA 1974a; Mumford et al. 1986; NATO 1981), or it may be emitted directly with auto exhaust (Williams et al. 1989a). Models of emissions of a re-refinery in Buffalo, New York, produced estimates of hydrocarbons ranging from 4.87 to 10.56 $\mu g/m^3$ depending on atmospheric conditions at a distance of 150 meters from the source (Booth et al. 1983). The combustion of used mineral-based crankcase oil as l-15% of the total fuel in power plants and other boilers resulted in measured ambient lead concentrations less than 5.0 $\mu g/m^3$ (0.005 mg/m^3) for 24-hour periods. The detection limit was not reported (Cooper 1977). Average concentrations were measured at 0.0001 $\mu g/m^3$ at a distance of 10,000 meters from the stack (API 1975).

Used mineral-based automotive crankcase oil was utilized as fuel in two waste oil heaters, an atomizing oil burner (AOB) and a vaporizing oil burner (VOB). The authors did not state if leaded or unleaded gasoline was used in the engines which produced the used mineral-based crankcase oil. In two separate test bums, the AOB emitted 20.9 and 224.45 mg/m^3 of particles $\leq$1 μm in diameter consisting primarily of antimony, chromium, copper, lead, magnesium, manganese, nickel, and zinc. The VOB emitted 0.22 and 18.2 mg/m^3 particles $\leq$3.0 μm in diameter primarily consisting of sulfur, zinc, aluminum, silicon, and lead (detection limits were not reported). Lead was present at concentrations up to 7.5% in the AOB emission particulates. The concentration of lead in the used mineral-based crankcase oil prior to combustion was 1,075 $\mu g/g$. An electrostatic precipitator was used for the two tests which emitted 20.9 and 0.22 $\mu g/m^3$ particulates. Combustion of used mineral-based automotive crankcase oil emitted greater levels of inorganic compounds compared to used mineral-based truck crankcase oil. Cadmium, cobalt, copper, iron, lead, phosphorus, and zinc exceeded the threshold limit values (time-weighted averages) in both of the waste oil heaters (Hall et al. 1983b; Mumford et al. 1986). In a separate study of emissions from a commercial boiler, a vaporizing pot burner, and an AOB, zinc concentrations in the stack were 34.8 mg/m^3, 0.04 mg/m^3, and 55.7 mg/m^3, respectively. Emissions of other metals were reported to follow trends similar to those seen in the zinc concentrations (Canadian Environmental Protection Act 1994).

A study comparing emissions of used crankcase oil with those of No. 2 fuel oil in air atomizing burners showed significantly higher emissions of hydrochloric acid, particulates, and lead from the used crankcase oil. Lead emissions from the used oil were 74 times higher than lead emission levels from the burning of No. 2 fuel oil. Results are from an average of tests from five different air atomizing burners with used oil from gasoline engines sampled in four of the tests and used oil from diesel engines sampled in the remaining test. The average concentrations were 346.25 mg/minute of HCl, 466.0 mg/minute of particulates, 1.6455 mg/minute of chromium, 20.8314 mg/minute of lead, and nondetectable levels of <0.2679 and <0.7428 mg/minute for arsenic and cadmium, respectively. Used crankcase oil emissions were still below applicable ambient standards (Vermont Agency of Natural Resources 1994).

The contribution of crankcase oil to exhaust particulate matter during a simulated urban drive cycle was analyzed by using ^{13}C-labelled lubricating oil. A C_{19} alkane was synthesized with a ^{13}C-labelled tracer and used in two vehicles initially registered in 1978 and 1981. Sampling was executed over a transient urban drive cycle with speeds ranging from 0 to 91 km/hour, with a mean value of 31.7 km/hour. The ^{13}C-labelled tracer demonstrated that 15% weight/weight of particulate matter emissions were attributable to crankcase oil. The percentage of particles ≤1 μm in diameter ranged from 70% to 90%, with a mean value of 85% (Williams et al. 1989a).

5.4.2 Water

The mean background concentration for total hydrocarbons is 2 μg/L for ocean water (Tanacredi 1977). Ultraviolet-fluorescence and gas chromatography were used to determine the relationship between waste-water effluents in Jamaica Bay, New York, and used mineral-based crankcase oil. Although specific sources for waste petroleum products were not established, samples taken from Jamaica Bay displayed chromatographs and ultraviolet-fluorescence responses similar to those of used mineral-based crankcase oil (Tanacredi 1977).

Waste water from a waste oil refinery that was released into the Buffalo Sewer Authority System contained 44.8 and 78.0 mg/L of what was categorically referred to as “Hydrocarbon Oil & Grease” (Booth et al. 1983). Surface water samples from on-site ponds at an NPL site, the Double Eagle Refinery, an inactive waste-oil re-refining facility in Oklahoma City, Oklahoma, were analyzed for chromium, lead, toluene, xylene, and unspecified hydrocarbons. Only chromium, lead, and xylene

levels were precisely measured and found to be 10, 118, and 14 μg/L, respectively (ATSDR 1990a). Another waste oil refinery was tested for inorganic and organic concentrations found in the waste water effluent. Inorganic concentrations measured were 0.34 mg/L for cadmium, 271 mg/L for lead, 3.4 mg/L for arsenic, 10 mg/L for chromium, 80 mg/L for barium, and 250 mg/L for zinc. Organic concentrations measured were 1,306 mg/L for toluene, 283 mg/L for napthalene, 364 mg/L for benzene, and 309-666 mg/L for several unnamed chlorinated solvents. Benzo(a)anthracene and benzo(a)pyrene concentrations were below detectable limits (0.02mg/L) (Canadian Environmental Protection Act 1994).

Total extractable hydrocarbon levels from Jamaica Bay surface water ranged from 0.5 to 3.1 mg/L in 1973 and from 0.88 to 5.1 mg/L in 1974. Gas chromatographic sulfur fingerprints of the aromatic fraction of stormwater runoff in Delaware indicated that used mineral-based crankcase oil was the likely source of hydrocarbons. Concentrations of particles containing aromatics in the stormwater ranged from 0.99 to 1.65 mg/L (990-1,650 μg/L), and soluble aromatics ranged from 0.04 to 0.07 mg/L (40-70 μg/L). Detection limits were not reported (MacKenzie and Hunter 1979).

An estimated 7 million gallons out of 340 million gallons of oil drained from automobiles per year are dumped into sewers (Suprenant et al. 1983). Hydrocarbon concentrations were 2,037±127 μg/L (Brown et al. 1985) and 8 mg/L (8,000 μg/L) (Hunter et al. 1979) in stormwater at the beginning of a storm, indicating a flushing effect, since the total flow-weighted hydrocarbon concentrations over storms ranged from 618 μg/L (Brown et al. 1985) to 5.30 mg/L (5,300 μg/L) (Hunter et al. 1979).

The concentrations of aliphatic hydrocarbons in runoff from two bridges in Seattle ranged from 6,000 to 24,000 μg/L, with a mean of 12,000 μg/L (Wakeham 1977). Only 1% of the hydrocarbons (25-250 μg/L; mean 100 μg/L) were n-paraffins. Gas chromatography suggested that the source of the hydrocarbons was used mineral-based crankcase oil. Crankcase oils are dewaxed and generally have few paraffins. Urban stormwater runoff had lower concentrations of aliphatic hydrocarbons, (200-7,500 μg/L; mean 1,200 μg/L) and similar to the bridge runoff, low levels of n-paraffins (4-350 μg/L; mean 13 μg/L). The chromatographic responses of used mineral-based crankcase oil and the hydrocarbons in the bridge runoff were very similar. The concentration of aliphatic hydrocarbons in the rivers and streams in the Seattle area varied with the season. During the rainy season, the hydrocarbon level was slightly higher; however, the paraffin concentrations remained low, ranging

from 0.1 to 1 μg/L, indicating the presence of hydrocarbons from used mineral-based crankcase oil (Wakeham 1977).

Total flow-weighted hydrocarbon concentrations in particulates from urban runoff in Rhode Island ranged from 16,400 to 34,000 μg/g (mean 24,800 ± 6570 μg/g) in commercial areas, from 61,900 to 507,000 μg/g (mean 211,000 ± 189,000 μg/g) in industrial areas, from 8,750 to 51,800 μg/g (mean 24,800 ± 19,900 μg/g) near interstate highways, and from 15,700 to 59,800 μg/g (mean 42,000 ± 23,200) near residential areas. A PAH signature of the runoff analysis was performed to confirm that the hydrocarbons originated from used mineral-based crankcase oil (Latimer et al. 1990).

5.4.3 Sediment and Soil

Aliphatic hydrocarbon levels in deep sediments (40-42 cm) samples (n=20) from Lake Washington in Seattle, Washington, ranged from 10 to 160 μg/g with an average of 32 μg/g for the entire lake. Surface sediments samples (n=20) ranged from 280 to 1,700 μg/g, with a mean of 1,400 μg/g. Surface samples were collected from areas near suspected hydrocarbon sources. Gas chromatographic analyses of the surface sediments indicate that the hydrocarbons were primarily from lubricating oils and pyrolysis products from automobiles that were transported by stormwater. Samples collected from two cores 94 cm deep showed an increase in hydrocarbon concentrations after 1880, which correlated with the industrial development of the area near the lake (Wakeham and Carpenter 1976). An analysis of sediment hydrocarbons from the Hillsborough Reservoir in Tampa, Florida, indicated that runoff from stormwater contaminated by used mineral-based crankcase oil was the likely source of the hydrocarbons. Aliphatic hydrocarbon concentrations ranged from 62 to 396 μg/g dry sediment, and aromatic hydrocarbon concentrations ranged from 15 to 89 μg/g dry sediment (Brown et al. 1985). Used mineral-based crankcase oil, determined by a lead concentration of 640 ppm in sediment samples, was detected 100 feet from an NPL site that was formerly a waste oil recycling facility in Moira, New York.

Chromium, lead, and unspecified hydrocarbons were detected in the sediments of on-site ponds at an inactive waste oil re-refining facility, which is also an NPL site. Only chromium was accurately measured on-site (89,000 μg/kg). Waste solids were reported to have been dumped 850 feet south of the facility, and the maximum concentrations of chromium and lead in the contaminated soil were 220,000 and 4,810,000 μg/kg, respectively (ATSDR 1990a).

Background concentrations of used mineral-based crankcase oil hydrocarbons in roadside soil were 856 μg/g in industrial areas, 265 μg/g near highways, 38.9 μg/g in residential areas, and 138 μg/g in commercial areas. Metals such as cadmium, copper, lead, manganese, and zinc were also measured in roadside source materials. Concentration ranges were 0.02-3.40 μg/g for cadmium, 10.4-228 μg/g for copper, 123-1,410 μg/g for lead, and 47.8-655 μg/g for zinc (Latimer et al. 1990). Soil samples taken 47.7 meters from an oil-treated road in Hunterdon County, New Jersey, contained an average lead concentration of 24 mg/kg. Two soil samples taken from a drainage culvert that carried runoff had lead concentrations of 78 and 96 mg/kg (Freestone 1979). Lead deposition from auto exhaust is typically limited to within 100 feet of a road (Motto 1970); therefore, the high lead concentrations in soil samples near the oil-treated road in Hunterdon County were most likely caused by the application of used mineral-based crankcase oil originating from engines burning leaded fuel. As leaded gasoline was a significant source of lead in soils, the use of unleaded gasoline should result in a reduction of lead in used mineral-based crank case oils, and a stabilization or reduction of lead in soil near roadsides.

5.4.4 Other Environmental Media

Background concentrations of lead on road surfaces averaged 39 mg/kg, while lead levels on dirt roads treated with used mineral-based crankcase oil averaged 209.25 mg/kg. Hydrocarbon concentrations at the surface of crankcase-oil-treated roads ranged from 5,880.24 to 13,441.25 mg/kg with a mean of 8,169.63 mg/kg. Hydrocarbon concentrations 6 inches below the road surface ranged from 7.65 to 67.63 mg/kg with a mean of 30.82 mg/kg (Freestone 1979).

Ambient hydrocarbon concentrations were measured in street dust and roadside vegetation in Rhode Island (Latimer et al. 1990). The PAH distribution in the hydrocarbons was similar to that in used mineral-based crankcase oil, suggesting that used mineral-based crankcase oil was the source of the hydrocarbons. Concentrations of hydrocarbons in street dust were 157 μg/g in commercial areas, 3,490 μg/g in industrial areas, 1,680 μg/g near highways, and 353 μg/g in residential areas. In roadside vegetation, the concentrations ranged from 40.6 to 46.1 μg/g in commercial, highway, and residential areas; however, in industrial areas the concentration was 290 μg/g. (Latimer et al. 1990).

Cadmium and lead at concentrations of 620 ppm and 331 ppm, respectively, associated with used mineral-based crankcase oil, were found in earthworms near a highway. The levels in the soil-purged

earthworms were positively associated with traffic volume and negatively associated with distance from the highway (Gish and Christensen 1973).

Plants sampled from a field 150 feet from a road where used oil had been applied for dust suppression contained an average lead concentration of 33.5 mg/kg. The lead concentration in the soil of the field was 24.0 mg/kg. A possible mechanism of transport to the field was dust containing lead or oil from the road. The average lead concentration in the oiled section of the road was 209.25 mg/kg as compared to a lead concentration of 39.0 mg/kg in the un-oiled section of the road. Aquatic organisms living in a stream running perpendicular to the road showed no real trend in their lead concentrations from areas upstream of the road and areas downstream of the road (Freestone 1972).

The fluorescence analyses of marine benthic organism (*Mya arenaria*) extracts from Jamaica Bay, New York, and used mineral-based crankcase oil were almost identical; however, no concentrations were reported (Tanacredi 1977).

5.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

The general population may be exposed to used mineral-based crankcase oil when checking the engine oil level of a motor vehicle; however, the extent of exposure to used mineral-based crankcase oil is minimal under these conditions (Kahsnitz et al. 1982). Exposure may also occur to the general population from the prevalent use of used mineral-based crankcase oil as a chain lubricant on household machinery such as chain saws, sewing machines, hedge trimmers, and lawn mowers (Brinkman et al. 1982). Several incidents of lead poisoning in cattle have been attributed to the ingestion of used mineral-based crankcase oil drained in pastures (Osweiler et al. 1973). Individuals who eat meat or dairy products from contaminated cattle may be exposed to excessive lead concentrations.

Occupational exposure to used mineral-based crankcase oil exists for engine mechanics who change crankcase oil or work with automobile parts contaminated with used mineral-based crankcase oil. However, exposure is likely to be low because most automobile repair shops have implemented procedures to reduce worker exposure (Kahsnitz et al. 1982).

Transport of used mineral-based crankcase oil through the soil from a contaminated site and into groundwater may result in human exposure via ingestion of contaminated drinking water, or dermal absorption if the water is used for recreational purposes (ATSDR 1988, 1990a; DOE 1989).

5.6 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Motorists who maintain and service their own automobiles may increase their likelihood of exposure if they spend long periods of time in contact with used mineral-based crankcase oil contaminated auto parts or do not practice good personal hygiene (DOE 1989; Kahsnitz et al. 1982).

Workers involved in the remediation of environmental contamination from used oil re-refining or recycling facilities may inhale contaminated particulates or volatiles, or be dermally exposed to contaminated soil, sludge, sediment, or sorbent pads (ATSDR 1988, 1990a). Workers involved in collecting used mineral-based crankcase oil for recycling or re-refining may also be dermally exposed.

Individuals who work or live in buildings that burn used mineral-based crankcase oil for heating fuel may be exposed to high levels of metal particulates in the respirable range (Cooper 1977; Hall et al. 1983b; Mumford et al. 1986).

Individuals who live near roads that have been treated with used mineral-based crankcase oil as a dust suppressant may be exposed to dust particles which are contaminated with used oil.

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 used mineral-based crankcase oil is available. Where adequate information is not available, ATSDR, in conjunction with the 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 used mineral-based crankcase oil.

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 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 will be proposed.

5.7.1 Identification of Data Needs

Physical and Chemical Properties. The physical and chemical properties of used mineral-based crankcase oil as a mixture are not well defined, and data should be gathered in order to estimate the fate of this oil and its components in the environment. Data needs associated with some specific compounds that are components of used mineral-based crankcase oil (e.g., PAHs, lead, cadmium, and zinc) are presented in the ATSDR profiles for these chemicals (ATSDR 1990c, 1993b, 1992d, 1989b, respectively).

Production, Import/Export, Use, Release, and Disposal. In 1972, 500 million gallons of used mineral-based crankcase oil were produced in the United States, and in 1982, production increased to 1,350 million gallons (API 1974; Brinkman et al. 1982; Maugh 1976; Vasquez-Duhalt 1989; Weinstein 1974). More recent data on the production of used mineral-based crankcase oil are needed. Used mineral-based crankcase oil has been used as a supplemental fuel for boilers, waste disposal incinerators, and cement kilns (API 1975; Cooper 1977; EPA 1974a; Hall et al. 1983a; Mumford et al. 1986; NATO 1981). Most releases of used mineral-based crankcase oil are the result of illegal disposal and runoff from highways and rural roads. The environmental media most likely to be contaminated by used mineral-based crankcase oil are water, soil, and sediment. Current disposal methods are not satisfactory, since a national survey of disposal methods indicated that 40% of used mineral-based crankcase oil is poured onto the ground, 21% is placed in the trash to be collected, 14% is recycled, and 25% is used for other purposes (Brinkman et al. 1982). Used mineral-based crankcase oil is regulated by EPA and by the individual states. All states except West Virginia recognize road oiling for dust suppression as an important route of exposure and regulate for this release. Further regulations for used mineral-based crankcase oil are expected in the future.

According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit substance release and off-site transfer information to the EPA. The Toxics Release Inventory (TRI), which contains this information for 1993, became available in

May of 1995. This database will be updated yearly and should provide a list of industrial production facilities and emissions. However, no data for used mineral-based crankcase oil are included in TRI.

Environmental Fate. Used mineral-based crankcase oil partitions to various environmental media depending on the physical and chemical properties of its individual components. Major fate processes include slow volatilization of lower weight aliphatic and aromatic hydrocarbons, and sorption of higher molecular weight hydrocarbons to soil (DOE 1989). Slow microbial degradation is the primary loss mechanism for hydrocarbon fractions not lost through volatilization (DOE 1989; Raymond et al. 1976). Metals may be solubilized during microbial degradation, consequently increasing their mobility and toxicity (Vasquez-Duhalt and Greppin 1986). Used mineral-based crankcase oil is usually transported to surface water by stormwater runoff. Hydrocarbons that settle out of the water column into the sediment are persistent (Brown et al. 1985; Wakeham and Carpenter 1976). The half-life of used mineral-based crankcase oil in soil and water has not been established; therefore, further studies are required. The environmental fate of some of the individual components has been studied (see ATSDR profiles for PAHs, cadmium, lead, and zinc [ATSDR 1990c, 1992d, 1993b, 1989b]). The environmental fate and behavior of used mineral-based crankcase oil have not been well characterized. Consequently, further data is needed on movement and persistence of the components of used mineral-based crankcase oil in all environmental media.

Bioavailability from Environmental Media. There are limited animal data on the absorption of used mineral-based crankcase oil by the inhalation, oral, or dermal routes. However, several of the components of used mineral-based crankcase oil are known to be absorbed. For more information on absorption of the individual components (e.g., lead, cadmium) see the ATSDR toxicological profiles on these compounds (ATSDR 1993b, 1992d). Limited animal data regarding lead poisoning related to the ingestion of used mineral-based crankcase oil indicate that lead from this source is distributed to various tissues (Osweiler et al. 1973). Limited data exist on absorption of lead in plants grown in contaminated soil (Raymond et al. 1976) and absorption of PAHs by marine organisms in contaminated water (Tanacredi 1977; Tanacredi and Cardenas 1991); however, more data are needed to determine whether the components of used mineral-based crankcase oil are likely to enter the food chain. Additional data on rates and extent of absorption through inhalation, oral, and dermal routes may be helpful in determining bioavailability from environmental media.

Food Chain Bioaccumulation. Used mineral-based crankcase oil, as a mixture, does not bioconcentrate in the food chain. However, the individual components may bioconcentrate depending on their physical and chemical properties. Metals, especially lead, have been found to bioconcentrate in crops and cause abnormal growth (Falahi-Ardakani 1984; Raymond et al. 1976). Limited information was also located on the bioconcentration of used mineral-based crankcase oil components in terrestrial animals and aquatic organisms (Osweiler et al. 1973; Tanacredi 1977; Tanacredi and Cardenas 1991). Research on the biomagnification of used mineral-based crankcase oil would not be useful because the oil is not available to the food chain as a mixture. However, further research on the biomagnification potential of the individual components of used mineral-based crankcase oil may be helpful in assessing the risk associated with eating foods grown in contaminated soil.

Exposure Levels in Environmental Media. Most monitoring studies assessed the concentration of used mineral-based crankcase oil in stormwater runoff and sediments, although limited data exist on hydrocarbon and metal concentrations in surface water, air, marine benthic organisms, roadside plants, and cattle (Brown et al. 1985; Falahi-Ardakani 1984; Freestone 1979; Gish and Christensen 1973; Hunter et al. 1979; Latimer et al. 1990; MacKenzie and Hunter 1979; Osweiler et al. 1973; Tanacredi 1977; Van Donkelaar 1990; Wakeham 1977). Limited data are available from monitoring studies near hazardous waste sites. Lead was detected in sediments and groundwater near a hazardous waste site (ATSDR 1988), and chromium and lead were detected in soil near another hazardous waste site (ATSDR 1990a). However, the sampling at each site was not comprehensive, and the validity of the results could not be verified. Estimates should also be developed for human intake of used mineral-based crankcase oil from environmental media. Recent data on levels of used mineral-based crankcase oil and its components in environmental media, especially groundwater, are needed to further assess potential risk from likely sources of exposure. There are no known populations with extraordinarily high exposure to used mineral-based crankcase oil.

Reliable monitoring data for the levels of used mineral-based crankcase oil in contaminated media at hazardous waste-sites are needed so that the information obtained on levels of used mineral-based crankcase oil in the environment can be used in combination with the known body burden of used mineral-based crankcase oil to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

Exposure Levels in Humans. This information is necessary for assessing the need to conduct health studies on these populations. There is no specific biomarker of exposure to used mineral-based crankcase oil in humans; therefore, biological monitoring studies are not recommended. See other ATSDR profiles (PAHs, lead, zinc, and cadmium) for levels of used mineral-based crankcase oil components detected in human tissue (ATSDR 1990c, 1992d, 1993b, 1989b, respectively).

Exposure Registries. No exposure registries for used mineral-based crankcase oil were located. This substance is not currently one of the compounds for which a subregistry has been established in the National Exposure Registry. The substance 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 exposure to this substance.

5.7.2 On-going Studies

No on-going studies on the exposure or environmental fate of used mineral-based crankcase oil 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/or monitoring used mineral-based crankcase oil, its metabolites, and other biomarkers of exposure and effect to used mineral-based crankcase oil. The intent is not to provide an exhaustive list of analytical methods. Rather, the intention is to identify well-established methods that are used as the standard methods of analysis. Many of the analytical methods used for environmental samples are the methods approved by federal agencies and 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 modify previously used methods to obtain lower detection limits, and/or to improve accuracy and precision.

6.1 BIOLOGICAL MATERIALS

Used mineral-based crankcase oil is a complex mixture of hydrocarbons, metals, and additives (Vazquez-Duhalt 1989). It contains high levels of metals, such as lead, zinc, copper, chromium, nickel, and cadmium. It also contains alkanes, cycloalkanes, monoaromatics, diaromatics, PAHs, and aromatic compounds which contain sulfur, nitrogen, or oxygen. Atomic absorption spectrophotometry (AAS) is the most commonly used method for detecting the metal components of used mineral-based crankcase oil in biological materials. Biological materials include the skin, liver, lung, serum, urine, and milk. Analytical methods for measuring both biological materials and environmental samples are described in this chapter. The ^{32}P-postlabeling assay has been used to detect the presence of potentially carcinogenic PAH adducts bound covalently to macromolecules (e.g., DNA) in skin and lung tissues. See Table 6-l for a summary of the analytical methods most commonly used to determine the various components of used mineral-based crankcase oil in biological materials and environmental samples. For more information regarding the analytical methods used for detecting the various components such as PAHs and lead in biological tissues, see the ATSDR profiles for these substances (ATSDR 1990c, 1993b).

TABLE 6-1. Analytical Methods for Determining Used Mineral-based Crankcase Oil in Biological Materials

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Skin (PAHs)	Isolate epidermal DNA from human and mouse skin treated with used oil; digest DNA enzymatically with micrococcal nuclease, spleen phosphodiesterase, and nuclease P_1; label DNA adduct by ^{32}P-post-labeling technique involving incubation with carrier-free [γ-^{32}P]ATP and polynucleotide kinase	^{32}P-postlabeling followed by TLC and autoradiography	0.20–0.57 fmol adducts/μg DNA	No data	Carmichael et al. 1990, 1991, 1992
Skin (PAHs)	Digest DNA samples with micrococcal nuclease, spleen phosphodieterase, and nuclease P_1; incubate; digest with [γ-^{32}P]ATP and polynucleotide kinase	^{32}P-postlabeling followed by TLC and autoradiography	0.05–0.13 fmol adducts/μg DNA	No data	Phillips et al. 1990

TABLE 6-1. Analytical Methods for Determining Used Mineral-based Crankcase Oil in Biological Materials (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Skin and lungs (PAHs)	Digest DNA samples with micrococcal nuclease and spleen phosphodiesterase; extract twice with water-saturated butanol; combine organic phases and back extract twice with water; neutralize with Tris and evaporate; label DNA adduct by ^{32}P-postlabeling technique involving incubation with carrier-free [γ-^{32}P]ATP and polynucleotide kinase	^{32}P-postlabeling followed by TLC and autoradiography	40–150 amol total adducts/µg DNA	No data	Schoket et al. 1989
Liver, kidney, and rumen contents (molybdenum)	Dry ash samples; add pyrogallol red and ethyltrimethyl-ammonium bromide dissolved in acetate buffer	UV-VIS spectro-photometer	No data	No data	Sas 1989
Blood (lead)	No data	AAS	No data	No data	Clausen and Rastogi 1977

TABLE 6-1. Analytical Methods for Determining Used Mineral-based Crankcase Oil in Biological Materials (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Urine (1-pyrenol)	Urine samples immediately frozen at -20°C and kept in the dark until analysis; over-diluted samples (<0.6 g creatinine/liter) were discarded and fresh samples taken	HPLC/fluorescence detector; separation using methanol:water linear gradient	0.02 µg/L (signal/noise ratio >3); mean coefficient of variance and analysis 10%	No data	Granella and Clonfero 1993

AAS = atomic absorption spectrophotometry; amol = attomole; DNA = deoxyribonucleic acid; fmol = femtomole; HPLC = high-performance liquid chromatography; P_i = phosphorus$_i$; ^{32}P = radio-labeled phosphorus; [γ-^{32}P]ATP = [gamma-^{32}phosphorus]adenosine triphosphate; PAHs = polycyclic aromatic hydrocarbons; TLC = thin-layer chromatography; UV-VIS = ultraviolet-visible

AAS has been used to measure lead compounds from used mineral-based crankcase oil in blood (Clausen and Rastogi 1977). AAS is the most common detector used to measure lead compounds in blood, since AAS is a lead-specific detector. The sample preparation procedure, limit of detection, accuracy, and precision of this method were not reported. Molybdenum has been determined in rumen contents and in liver and kidney samples using ultraviolet-visible (UV-VIS) spectrophotometry; however, no information was provided regarding the sample preparation, detection limit, accuracy, or precision of this method (Sas 1989).

The ^{32}P-postlabelling assay is a highly sensitive and specific method for detecting PAH-DNA adducts in the skin and lungs of humans and animals (Carmichael et al. 1990, 1991, 1992; Phillips et al. 1990; Schoket et al. 1989). The technique generally involves isolating epidermal DNA from human and mouse skin treated with used oil, and then digesting the DNA enzymatically with micrococcal nuclease, spleen phosphodiesterase, and nuclease Pl before labeling the DNA adduct by ^{32}P-postlabelling. Detection limits ranging from 0.05 to 0.57 femtomole (fmol) adducts/μg DNA (Carmichael et al. 1990, 1991, 1992; Phillips et al. 1990) and from 40 to 150 attomole (amol) total adducts/μg DNA (Schoket et al. 1989) have been achieved. Recovery and precision data were not reported.

The urinary concentration of 1-pyrenol, a pyrene metabolite, can also be used as a biological indicator of exposure to PAHs in automotive repair workers. Granella and Clonfero (1993) evaluated the skin absorption of PAHs in automotive repair workers whose skin was exposed to used engine oils from cars and trucks and compared the results to a control group. For each worker, data were obtained on smoking habits, use of medicines, and hobbies. Subjects were also instructed not to eat foods with high concentrations of PAHs, such as grilled or barbecued meat, 48 hours before the urine samplings. The control group was placed on control diets and not occupationally exposed to PAHs. This group had a higher percentage of smokers, but among smokers in both the control and the exposed groups, there was no difference in the daily consumption of cigarettes (Granella and Clonfero 1993). The levels of 1-pyrenol were determined using high-performance liquid chromatography (HPLC) with a fluorescence detector. Urinary excretion of 1-pyrenol in this group of workers compared to occupationally exposed subjects (e.g., creosote, coke oven, and graphite electrode workers) indicated that exposure to PAHs present in used engine oil through the skin during automotive repair work is very low (Granella and Clonfero 1993). The urinary 1-pyrenol values were higher in both smoking (0.259 ± 0.201 umol/mol creatinine) and nonsmoking workers (0.154 ± 0.105 pmol/mol creatinine, as

compared to 0.083 ± 0.042 pmol/mol creatinine for the nonsmoking controls) (Granella and Clonfero 1993). However, according to the study authors, tobacco smoking and PAH-rich diets are confounding factors when monitoring this type of exposure, as they influence the urinary concentration of 1-pyrenol in the general population. Hence, smoking habits and diets should be verified accurately, including exposure to air pollution, in order to evaluate the specific effect of low-level PAH exposure, especially among repair workers. In used engine oils, the concentration of 1-pyrenol ranges from 32% (petrol engines) to 3% (diesel engines) (Granella and Clonfero 1993).

6.2 ENVIRONMENTAL SAMPLES

AAS is the most commonly used method for detecting the metal components of used mineral-based crankcase oil found in environmental samples. The methods most commonly used to detect or identify the major hydrocarbon components of used mineral-based crankcase oil in environmental samples include gas chromatography equipped with a flame ionization detector (GC/FID), flame photometric detector (GC/FPD), or mass spectrometer (GC/MS), and high-performance liquid chromatography with ultraviolet detection (HPLC/UV) or a fluorescence detector. Volatile organics have been detected using an ion trap mass spectrometer (EMS). Infrared spectroscopy (IR) is commonly used to characterize the major components found in used crankcase oil. See Table 6-2 for a summary of the analytical methods used to determine used mineral-based crankcase oil in environmental samples. For further information regarding the analytical methods for detecting the various components such as PAHs, lead, copper, nickel, and zinc in environmental samples see the ATSDR profiles for these substances (ATSDR 1989b, 1990b, 1990c, 1992e, 1993b).

Pyrene levels in cloths used by automotive repair workers were determined by HPLC with a UV-VIS detector. Pyrene contents in oily material taken from cloths used to clean the different types of engines ranged from 2.8 ± 0.4 ppm (mean ± SD) for matter from diesel truck engines to 9.3 ± 8.2 ppm for matter from petrol car engines. The recovery rate was 96%. These values were much lower than that found in coal tar, creosote, petroleum, coke, and quenching oils. However, petrol engines produced five times more pyrene than diesel engines (Granella and Clonfero 1993).

AAS is the most commonly used method to selectively detect and identify the metal components (lead, cadmium, manganese, copper, nickel, iron, and zinc) of used mineral-based crankcase oil in air (Clausen and Rastogi 1977), street or stormwater runoff (Latimer et al. 1990; Newton et al. 1974), soil

TABLE 6-2. Analytical Methods for Determining Used Mineral-based Crankcase Oil in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Air (lead)	Collect air sample through Whatman Glass filter paper	AAS	No data	No data	Clausen and Rastogi 1977
Air (engine oil)	Collect airborne mist of engine oil on membrane filter; extract with $C_2Cl_3F_3$	IR spectrophotometry	0.5 mg/m^3	98	NIOSH 1984
Air (oil mist)	Collect airborne mist of oil on membrane filter; extract with carbon tetrachloride	IR spectrophotometry	0.3 mg/m^3	No data	NIOSH 1978
Air (oil mist)	Collect airborne mist of oil on membrane filter; extract with chloroform	Fluorescence spectrophotometry	No data	97.7	NIOSH 1977
Air (volatile organics)	Direct injection with no sample preparation (direct air sampling)	ITMS	1 ppb	No data	Buchanan et al. 1990
Air (volatile organics)	Preconcentrate on resin trap followed by direct thermal desorption	ITMS	ppt levels	No data	Buchanan et al. 1990
Crankcase oil	Heat sample to 300°C and use hydrogen as the carrier gas and nitrogen as the make-up gas	PID; FID	No data	≥97	Bemgård and Colmsjö 1992

TABLE 6-2. Analytical Methods for Determining Used Mineral-based Crankcase Oil in Environmental Samples (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Waste incineration by-products (crankcase oil; smelter particulates; composite waste particulates)	No preparation for samples on filter; solid samples are ground and mounted in bulk specimen. If there are insufficient quantities for bulk handling, samples are cast in a collodion film on glass slide	XRD; samples scanned with a diffractometer and the XRD data is reduced using JCPDS files	No data	No data	Briden 1984
Waste incineration by-products (crankcase oil; smelter particulates; composite waste particulates)	No preparation for samples on filter; solid samples are ground, mixed 1:3 with organic binder, pressed into a pellet	XRF; samples qualitatively scanned on sequential wave-length spectrometer	No data	No data	Briden 1984
Water (volatile organics)	Direct purge of sample with helium into the mass spectrometer sample interface	ITMS	ppb or lower	No data	Buchanan et al. 1990
Surface water	Add sulfuric acid and (CCl_4) to water sample; jet-air evaporate CCl_4 extract; concentrate; bring up to volume with hexane	GC/FID; UV fluorescence spectroscopy; GC/MS	No data	No data	Tanacredi 1977

TABLE 6-2. Analytical Methods for Determining Used Mineral-based Crankcase Oil in Environmental Samples (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Water (PAHs)	Extract water samples with methylene chloride in separatory funnels; evaporate; clean up on deactivated silica/alumina gel column; elute with benzene-methanol	GC/FID; GC/MS	No data	No data	Tanacredi and Cardenas 1991
Stormwater runoff (hydrocarbon analysis)	Filter runoff samples through glass fiber filter; add internal standards; extract filtrate with methylene chloride; solvent exchange to hexane; evaporate to 1 mL; clean up by adsorption column chromatography using silica gel; elute saturated hydrocarbons (normal, branched, and cyclic alkanes) with hexane; elute unsaturated hydrocarbons (PAHs) with hexane/methylene chloride	No data	No data	No data	Latimer et al. 1990

TABLE 6-2. Analytical Methods for Determining Used Mineral-based Crankcase Oil in Environmental Samples (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Stormwater runoff (hydrocarbons)	Fractionate water sample to separate aqueous and particulate phases; pass aqueous portion through activated carbon column, then dry; centrifuge particulate phase and dessicate; extract both fractions successively with hexane, benzene, and chloroform using Soxhlet extraction; clean up using silica gel column; elute with hexane (aliphatic hydrocarbons), benzene (aromatic hydrocarbons), and chloroform/methanol (oxy-polar compounds); evaporate to dryness and record weight	IR; GC/FID/FPD	No data	85–95	Hunter et al. 1979
Street runoff (lead)	Acid digest sample	AAS	No data	No data	Newton et al. 1974
Stormwater runoff and sediment	Extract and saponify by Soxhlet extraction using a benzene/potassium solvent system	GC/FID	No data	No data	Brown et al. 1985

TABLE 6-2. Analytical Methods for Determining Used Mineral-based Crankcase Oil in Environmental Samples (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Soil (volatile organics)	Add water to soil sample; purge	ITMS	ppb or lower	No data	Buchanan et al. 1990
Street dust, surface soil, vegetation, atmospheric deposition (hydrocarbon analysis)	Collect dust and particulates on filter; air dry and weigh all samples; reflux in KOH mixture in methanol; water; partition hydrocarbons against petroleum ether; evaporate extracts to 1 mL; clean up with adsorption column chromatography with silica gel; elute saturated hydrocarbons with hexane; elute unsaturated hydrocarbons including PAHs with hexane:methylene chloride	GC/FID	No data	No data	Latimer et al. 1990
Stormwater runoff, dust, soil, vegetation, atmospheric deposition (metals analysis [Pb, Cd, Mn, Cu, Fe, and Zn])	Leach samples for one week in 5% HNO_3 solution	AAS	No data	No data	Latimer et al. 1990

TABLE 6-2. Analytical Methods for Determining Used Mineral-based Crankcase Oil in Environmental Samples (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Stormwater runoff (aromatic hydrocarbons and sulfur compounds)	Separate aqueous and particulate phase by centrifugation; filter particulates and dry in oven; adsorb supernatant onto activated carbon column; Soxhlet extract both phases using hexane, benzene, and chloroform; extracts adsorbed to silica gel; elute with hexane to obtain aliphatic hydrocarbons; elute with benzene to obtain aromatic hydrocarbon and sulfur compounds; evaporate to dryness; weigh; resolubilize in methylene chloride; pass through copper column to eliminate free sulfur contamination	GC/FID/FPD; GC/MS	No data	≈70 (dibenzo-thiophene)	MacKenzie and Hunter 1979

TABLE 6-2. Analytical Methods for Determining Used Mineral-based Crankcase Oil in Environmental Samples (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Used engine oil (pyrene)	Carefully preweighed cloth left in workshops which a worker cleaned with his hands; cloth then weighed and oily matter in the cloth extracted with acetonitrile in ultrasonic bath thermostated at 40°C for 15 minutes; this operation repeated three times; concentration of pyrene in extracts then determined	HPLC/UV-VIS detector at 350 mm wavelength; 20 µL of extract eluated with acetonitrile water gradient for 15 minutes at 40°C, flow rate 1.0 mL/minute	No data	96	Cranella and Clonfero 1993
Wastewater (1-nitropyrene and 1,6-dinitropyrene)	Fractionate waste water into diethyl ether-soluble neutral, acidic, and basic fractions	HPLC/UV detector/ fluorescence detector; GC/MS	1.1×10^{-2} pmol (1-NP); 1.3×10^{-2} pmol (1,6-diNP)	No data	Manabe et al. 1984
Waste water (hydrocarbons)	Clean up sample on alumina or silica gel column; elute hydrocarbons with petroleum ether; concentrate	GC; IR; TLC	0.5 mg/L	No data	Farrington and Quinn 1973

TABLE 6-2. Analytical Methods for Determining Used Mineral-based Crankcase Oil in Environmental Samples (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Soil and vegetation (Cd, Ni, Pb, and Zn)	Agitate soil samples with HCl and centrifuge; digest grass samples with hot, concentrated HNO_3 and $HClO_4$	AAS	No data	No data	Lagerwerff and Specht 1970
Soil (heavy metals)	Dry soil samples; add alcoholic magnesium nitrate; heat to dryness; ash; add concentrated HCl to cooled sample; heat to dryness; add concentrated HCl and water to dried material; bring solution to boil; bring cooled solution up to volume; shake and filter	AAS	No data	99.4 (Cd); 102.5 (Ni); 98.8 (Pb); 95.25 (Zn)	Gish and Christensen 1973

TABLE 6-2. Analytical Methods for Determining Used Mineral-based Crankcase Oil in Environmental Samples (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Soil and plants	Air dry and grind samples; boil soil samples for 2 hours in perchloric acid; digest dried plant samples in 4:1 mixture of nitric and perchloric acids	AAS	No data	No data	Motto 1970
Soil (motor oil)	Dry soil sample; extract with methylene chloride and agitate; concentrate extract using low heat	TLC	100 ppb	70	Newborn and Preston 1991
Sediments (hydrocarbons)	Extract lipids from sediments by Soxhlet extraction with benzene and methanol; saponify to separate hydrocarbons from fatty acid esters; isolate hydrocarbons by column chromatography on alumina packed over silica beds; elute aliphatic hydrocarbons with pentane	GC/FID	No data	>75	Wakeham and Carpenter 1976
Clams (PAHs)	Homogenize tissue; Soxhlet extract in hexane for 8 hours	HPLC/UV detector	No data	No data	Tanacredi and Cardenas 1991

TABLE 6-2. Analytical Methods for Determining Used Mineral-based Crankcase Oil in Environmental Samples (*continued*)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Earthworms	Blend sample; mix with alcohol magnesium nitrate; dry, char, and ash sample; wet cooled ash with nitric acid; dry; add concentrated HCl and heat to dryness; add concentrated HCl and water and bring to a boil; bring cool solution up to volume	AAS	No data	89.25 (Cd); 106.5 (Ni); 93.25 (Pb); 89.15 (Zn)	Gish and Christensen 1973

AAS = atomic absorption spectrophotometry; CCl_4 = carbon tetrachloride; $C_2Cl_3F_3$ = trichlorotrifluoroethane; Cd = cadmium; Cu = copper; Fe = iron; FID = flame ionization detector; FPD = flame photometric detector; GC = gas chromatography; HCl = hydrochloric acid; $HClO_4$ = perchloric acid; HNO_3 = nitric acid; HPLC = high-performance liquid chromatography; IR = infrared; ITMS = ion trap mass spectrometer; JCPDS = Joint Committee on Powder Diffraction Standards; KOH = potassium hydroxide; Mn = manganese; MS = mass spectrometry; Ni = nickel; PAHs - polycyclic aromatic hydrocarbons; Pb = lead; PID = photoionization detector; TLC = thin-layer chromatography; UV = ultraviolet; VIS = visible; XRF = x-ray fluorescence; XRD = x-ray diffraction; Zn = zinc

(Gish and Christensen 1973; Lagerwerff and Specht 1970; Motto 1970), dust, vegetation, atmospheric deposition, and earthworms (Gish and Christensen 1973; Lagerwerff and Specht 1970; Latimer et al. 1990). Air samples are collected on filter paper (Clausen and Rastogi 1977). The other matrices (runoff, soil, vegetation, and earthworm samples) are usually subjected to an acid digestion procedure. Although sensitivity data were not reported, ppm-to-ppb levels can be detected based on the measured samples. Recovery data for measuring metals in air and runoff samples were not reported. Recoveries for soil samples (>95%) and earthworms (>89%) were very good (Gish and Christensen 1973). Precision data were not reported.

GC/FID and GUMS are methods used for detecting the hydrocarbon components of used crankcase oil in surface water (Tanacredi 1977), stormwater runoff and waste water (Brown et al. 1985; Farrington and Quinn 1973; Latimer et al. 1990), and sediments (Wakeham and Carpenter 1976). Sample preparation methods generally include solvent extraction followed by a cleanup step using adsorption chromatography. Although detection limits were not reported for these methods, sensitivity is in the ppm-to-ppb range based on data reported. Recovery for hydrocarbons in sediment using GC/FID was >75% (Wakeham and Carpenter 1976). Precision data were not reported.

Petroleum-derived aromatic and aliphatic hydrocarbons and associated sulfur compounds in urban stormwater runoff and sediment samples were characterized using GC equipped with a flame ionization/sulfur specific flame photometric detector system (Hunter et al. 1979; MacKenzie and Hunter 1979). These detectors allowed simultaneous detection of both hydrocarbon and sulfur-containing materials. Although sulfur compounds represent only about 4% of a petroleum oil, sulfur fingerprints have been shown to be a valuable aid in oil characterization and source correlation (MacKenzie and Hunter 1979). The sulfur aromatics in petroleum oil are predominantly thiophenes (benzothiophene, dibenzothiophene, naphthobenzothiophene, and their alkyl derivatives). Aromatic sulfur compounds have been detected in petroleum oils from stormwater runoff. Dibenzothiophene and phenanthrene and/or anthracene were present in all samples (both used crankcase oil and stormwater particulates). An average of approximately 70% of the dibenzothiophene was recovered in stormwater by the analytical method and separation scheme. Recoveries for the aliphatic and aromatic fractions ranged from 85% to 95% (Hunter et al. 1979). Although detection limits were not reported, ppm-to-ppt concentrations can be measured based on levels reported (Hunter et al. 1979; MacKenzie and Hunter 1979). Precision data were not reported.

In Manabe et al. (1984), mutagenic studies (using *S. typhimurium* strains) of crankcase oil from a gasoline engine, fractionated by Soxhlet extraction, indicated that used engine oil contained 1-nitropyrene (1-NP) and dinitropyrenes (diNP). Crankcase oils from gasoline and diesel engines contained 138 and 349 ng of 1-NP and 2 and 31 ng 1,6-diNP, respectively, per mL of oil. In this study, the 1-NP fraction was collected, analyzed, and quantified by GC/MS and HPLC to prove that waste water contained 1-NP. Since many unknown compounds in the waste water showed the same retention time as 1-NP using HPLC, 1-NP was not quantitated precisely by use of an ultraviolet detector and HPLC. Since the reduced product of 1-NP, 1-aminopyrine (l-AP), is fluorescent and easily detectable with the fluorescence detector of HPLC, 1-NP was reduced with nitroreductase and l-AP was then measured. The reaction mixture was incubated at 37°C for 5 or 15 hours without shaking. An ethyl acetate extract of the mixture was then evaporated, redissolved in methanol, and injected into the chromatograph. Detection limits for 1-NP and diNP were $1.1x10^{-2}$ and $1.3x10^{-2}$ pmol, respectively. Recovery and precision data were not reported. 1,6-diNP (31 ng) accounted for 12% of the total mutagenicity of the neutral fraction in the assay system (Manabe et al. 1984). According to the study authors, diNPs showed higher mutagenic activity than 1-NP, and 1,6-diNP was shown to be carcinogenic in mice.

X-ray diffraction (XRD) phase analysis in conjunction with x-ray florescence (XRF) elemental analysis is an extremely effective method for characterizing waste incineration by-products, particularly crankcase oil incineration residues. XRD is a useful technique for determining the phase of the compounds in a sample. The technique involves scanning the sample with a diffractometer and interpreting the data according to the Joint Committee on Powder Diffraction Standard (JCPDS) files. To determine which elements are present in the sample, XRF is conducted using minimal sample preparation and analysis time. This method analyzes the samples for elements ranging from sodium to uranium by quantitatively scanning with a sequential wavelength dispersive spectrometer. In the past, using XRD phase analysis, together with XRF elemental analysis techniques, has produced accurate percentages of the elements present in the emissions from used mineral-based crankcase oil commercial waste incinerators and from lead and copper smelters (Briden 1984).

Volatile organics found in used mineral-based crankcase oil have been measured in air, water, and soil using an ITMS (Buchanan et al. 1990). The ITMS operates at a higher pressure than conventional mass spectrometers, making it amenable to accommodating higher gas loads, thereby allowing the direct introduction of gaseous analytes into the ITMS with little or no sample preparation. Because no

chromatography is used, sample turn around is less than 5 minutes. This method yields excellent detection limits (ppb-to-ppt levels) and is reproducible for all media. Recovery and precision data were not reported.

Thin-layer chromatography (TLC) has been used as a screening tool for on-site environmental analysis of used mineral-based crankcase oil in soil (Newborn and Preston 1991). A detection limit of 100 ppm and a recovery of 70% were achieved for motor oil. TLC is a cost-effective tool for field screening of samples, especially soils, when low detection limits (i.e., the sensitivity of gas chromatography) are not required.

IR and fluorescence spectrophotometry have been used to detect mineral oil in air (NIOSH 1977, 1978, 1984). For IR, sensitivity is in the low-ppm range (NIOSH 1978, 1984). For both methods, recovery (98%) and precision (5% relative standard deviation; 6.5% coefficient of variation) are good (NIOSH 1977, 1978, 1984). Sensitivity was not reported for fluorescence spectrophotometry. IR, fluorescence spectrophotometry, and ultraviolet spectroscopy have been used to detect and identify the main components of oils in sea water (Adler et al. 1990; Zieba 1985).

A photoionization detector (PID) in conjunction with capillary chromatography has been used to detect and identify PAHs. This is done by comparing the PID response with that of a flame ionization detector (FID) which gives a substance-specific response ratio. This method is limited in its usefulness because the obtained ratios are specific to the particular system being used. Ratios for the same compound can be off by as much as 20% for the same compound in different systems (Berngird and Colmsjo 1992).

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 used mineral-based crankcase oil is available. Where adequate information is not available, ATSDR, in conjunction with the 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 used mineral-based crankcase oil.

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 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 will be proposed.

6.3.1 Identification of Data Needs

Methods for Determining Biomarkers of Exposure and Effect

Exposure. Although specific biomarkers for exposure to used mineral-based crankcase oil have not been identified, methods exist to measure the PAH and metal components of used mineral-based crankcase oil in tissues (skin, lung, liver, kidney) and blood (Carmichael et al. 1990, 1991, 1992; Phillips et al. 1990; Sas 1989; Schoket et al. 1989). Biomarkers such as metal content of the blood (Clausen and Rastogi 1977; Sas 1989) or DNA adduct formation (Carmichael et al. 1990, 1991, 1992; Kurelec and Gupta 1993; Schoket et al. 1989) have been identified as possible indicators of exposure to chemicals found in used mineral-based crankcase oil. These biomarkers are not specific for exposure to used mineral-based crankcase oil but may be specific for chemicals found in the oil. Caution should be used to avoid misinterpretation when using DNA adducts as biomarkers in lower invertebrates such as sea urchins and sponges due to their inability to form DNA adducts when exposed to certain compounds such as PAHs. Another consideration with respect to using DNA adducts as biomarkers is that DNA modifications are found in varying levels in certain aquatic organisms depending on the season (Kurelec and Gupta 1993). Biomarkers of exposure to metals and aromatic hydrocarbons are discussed in more detail in other ATSDR profiles (ATSDR 1989b, 1990b, 1990c, 1992e, 1993b). The existing methods are sensitive enough to measure background levels in the population and levels at which biological effects occur. Recovery and precision data are needed for measuring PAH-DNA adduct levels in tissues and levels of metals in blood and tissue. Detection limit data are also needed for measuring metals in blood and tissues. These data will help to improve the reliability and reproducibility of the methods and will be useful in monitoring populations exposed to used mineral-based crankcase oil.

Effect. No information was located regarding biomarkers of effect that were specific for used mineral-based crankcase oil. The biomarkers of effect are similar to the biomarkers of exposure listed above.

The methods used to measure biomarkers of exposure are the same as those used to measure biomarkers of effect and data needs for biomarkers of effect are also similar to those mentioned for biomarkers of exposure.

Methods for Determining Parent Compounds and Degradation Products in Environmental Media. Methods exist to detect the various components of used mineral-based crankcase oil, such as metals (Clausen and Rastogi 1977; Gish and Christensen 1973; Lagerwerff and Specht 1970; Latimer et al. 1990; Motto 1970; Newton et al. 1974), as well as hydrocarbons and additives (Adler et al. 1990; Brown et al. 1985; Buchanan et al. 1990; Farrington and Quinn 1973; Hunter et al. 1979; Latimer et al. 1990; MacKenzie and Hunter 1979; Manabe et al. 1984; NIOSH 1977, 1978, 1984; Tanacredi 1977; Wakeham and Carpenter 1976; Zieba 1985). These methods detect components in air, water (including runoff), soil, sediments, and some aquatic and terrestrial organisms. They include AAS, GC/FID, GC/MS, GC/FID/FPD, ITMS, HPLC/UV, HPLC/fluorescence detector, TLC, IR, fluorescence spectrophotometry, and ultraviolet spectroscopy. These methods are relatively sensitive and selective and can be used to detect the levels of used mineral-based crankcase oil components found in the environment and the levels at which health effects could occur. However, recovery, sensitivity, and detection limit data are needed for measuring the components found in all media. Recovery and precision data will help to assess and improve the reproducibility of the methods. Detection limit data will aid in comparison of sensitivity between methods and indicate where improvements in sensitivity are needed. This information will be useful in monitoring used mineral-based crankcase oil contamination in the environment. So far, degradation products of used mineral-based crankcase oil have not been detected.

6.3.2 On-going Studies

No on-going analytical methods studies were located.

7. REGULATIONS AND ADVISORIES

National and state regulations and guidelines pertinent to human exposure to used mineral-based crankcase oil are summarized in Table 7-1. No international regulations were found.

MRLs have not been derived. Reference dose (RfD) and reference concentration (RfC) data were not found for used mineral-based crankcase oil.

The information in the following table indicates that used crankcase oil regulations are still being revised and created. Few water quality standards were found for used mineral-based crankcase oil. This may be because few states recognize or define used mineral-based crankcase oil as a hazardous waste.

The primary state regulations listed in Table 7-l are adaptations of the federal regulations. It is important to note that Colorado, the District of Columbia, and West Virginia have no or very few regulations for used mineral-based crankcase oil. Also, all states, with the exception of West Virginia, recognize road oiling and dust suppression as the primary route of environmental exposure and have enacted regulations to address this release. Further regulations are expected as the category of used mineral-based crankcase oil becomes more clearly defined.

TABLE 7-1. Regulations and Guidelines Applicable to Used Mineral-based Crankcase Oil

Agency	Description	Information	Reference
INTERNATIONAL			
IARC	Carcinogenic classification	NS[a]	IARC 1984
NATIONAL			
Regulations:			
a. Air:			
EPA	Used oil burned for energy recovery is subject to regulation if the allowable levels of any of the following constituents are exceeded:	Yes	EPA 1992a (40 CFR 279.11); EPA 1992a (40 CFR 279 Subpart G)
	Arsenic-maximum	5 ppm	
	Cadmium-maximum	2 ppm	
	Chromium-maximum	10 ppm	
	Lead-maximum	100 ppm	
	Flashpoint-maximum	100°F	
	Total halogens-maximum	4000 ppm	
b. Water:			
EPA	EPA water quality criteria for human health and aquatic life (mineral-based crankcase oils)	No	EPA 1989 (FR 45:79318)
EPA	Priority pollutant listing	No	EPA 1989 (FR 45:79318)
c. Other:			
EPA	Disposal regulations for nonhazardous used oil	No	EPA 1990 (40 CFR 261.21); EPA 1992b (40 CFR 261.4)
EPA	Standard for used oil application in dust suppression and the disposal of used oil	Yes	EPA 1992a (40 CFR 279 Subpart I)
EPA	Standards for used oil collection centers and aggregation points	Yes	EPA 1992a (40 CFR 279 Subpart D)
EPA	Standards for used oil processors and re-refining including facility standards, rebuttable presumption, notification requirements, management requirements, tracking, operating standards and offsite shipment standards	Yes	EPA 1992a (40 CFR 279 Subpart F)
EPA	Standard for used oil transport and transfer facilities including restrictions on nonprocessing transporters, notification requirements under RCRA, storage, transport, and tracking	Yes	EPA 1992a (40 CFR 279 Subpart E)
EPA	Used oil regulated as a hazardous waste if it contains greater than 1,000 ppm total halogens and/or is mixed with a hazardous waste	Yes	EPA 1980a (40 CFR 261.33); EPA 1992a (40 CFR 279.10)

TABLE 7-1. Regulations and Guidelines Applicable to Used Mineral-based Crankcase Oil (*continued*)

Agency	Description	Information	Reference
STATE			
Regulations and Guidelines			
a. Air:			
	Regulations on the burning and blending of used oil for the purpose of energy recovery (end-use regulation):		
Alabama		Yes[b]	State of Alabama 1991
Alaska		Yes[b]	State of Alaska 1992
Arizona		Yes	State of Arizona 1992
Arkansas		Yes[b]	State of Arkansas 1992
California		Yes	State of California 1992
Colorado		No	State of Colorado 1992
Connecticut		Yes[b]	State of Connecticut 1991
Delaware		Yes[b]	State of Delaware 1992
Washington, DC		No	District of Columbia 1992
Florida		Yes[b]	State of Florida 1992
Georgia		Yes[b]	State of Georgia 1992
Hawaii		Yes[b]	State of Hawaii 1992
Idaho		Yes[b]	State of Idaho 1992
Illinois		Yes[b]	State of Illinois 1992
Indiana		Yes[b]	State of Indiana 1992
Iowa		Yes[b]	State of Iowa 1992
Kansas		Yes[b]	State of Kansas 1992
Kentucky		Yes[b]	Commonwealth of Kentucky 1992
Louisiana		Yes	State of Louisiana 1992
Maine		Yes	State of Maine 1992
Maryland		Yes[b]	State of Maryland 1992
Massachusetts		Yes	Commonwealth of Massachusetts 1992
Michigan		Yes[b]	State of Michigan 1992
Minnesota		Yes[b]	State of Minnesota 1992
Mississippi		Yes[b]	State of Mississippi 1992
Missouri		Yes	State of Missouri 1992
Montana		Yes	State of Montana 1992
Nebraska		Yes[b]	State of Nebraska 1992
Nevada		Yes[b]	State of Nevada 1992
New Hampshire		Yes[b]	State of New Hampshire 1992
New Jersey		Yes	State of New Jersey 1992
New Mexico		Yes[b]	State of New Mexico 1991
New York		Yes	State of New York 1992
North Carolina		Yes[b]	State of North Carolina 1992
North Dakota		No	State of North Dakota 1992
Ohio		Yes[b]	State of Ohio 1992
Oklahoma		Yes[b]	State of Oklahoma 1991
Oregon		Yes[b]	State of Oregon 1992
Pennsylvania		Yes[b]	Commonwealth of Pennsylvania 1992
Rhode Island		Yes[b]	State of Rhode Island 1992
South Carolina		Yes[b]	State of South Carolina 1992
South Dakota		Yes[b]	State of South Dakota 1992
Tennessee		No	State of Tennessee 1992
Texas		No	State of Texas 1992
Utah		Yes[b]	State of Utah 1992

TABLE 7-1. Regulations and Guidelines Applicable to Used Mineral-based Crankcase Oil (*continued*)

Agency	Description	Information	Reference
STATE (Cont.)			
Vermont		Yes	State of Vermont 1992
Virginia		Yes	Commonwealth of Virginia 1992
Washington		Yes[b]	State of Washington 1992
West Virginia		No	State of West Virginia 1992
Wisconsin		Yes[b]	State of Wisconsin 1992
Wyoming		No	State of Wyoming 1992
b. Other:			
Connecticut	The use of reasonably available control technologies to limit the discharge of used oil (VOC)	Yes	CELDS 1993
	Regulations affecting marketers, generators, storers, and disposers of used oil and used oil burned for energy recovery (recycling collection/storage):		
Alabama		Yes	State of Alabama 1991
Alaska		Yes	State of Alaska 1992
Arizona		No	State of Arizona 1992
Arkansas		Yes	State of Arkansas 1992
California		Yes	State of California 1992
Colorado		No	State of Colorado 1992
Connecticut		Yes	State of Connecticut 1991
Delaware		Yes	State of Delaware 1992
Washington, DC		No	District of Columbia 1992
Florida		Yes	State of Florida 1992
Georgia		Yes	State of Georgia 1992
Hawaii		No	State of Hawaii 1992
Idaho		Yes	State of Idaho 1992
Illinois		Yes	State of Illinois 1992
Indiana		Yes	State of Indiana 1992
Iowa		Yes	State of Iowa 1992
Kansas		Yes	State of Kansas 1992
Kentucky		Yes	Commonwealth of Kentucky 1992
Louisiana		Yes	State of Louisiana 1992
Maine		Yes	State of Maine 1992
Maryland		Yes	State of Maryland 1992
Massachusetts		Yes	Commonwealth of Massachusetts 1992
Michigan		Yes	State of Michigan 1992
Minnesota		Yes	State of Minnesota 1992
Mississippi		Yes	State of Mississippi 1992
Missouri		Yes	State of Missouri 1992
Montana		Yes	State of Montana 1992
Nebraska		Yes	State of Nebraska 1992
Nevada		Yes	State of Nevada 1992
New Hampshire		Yes	State of New Hampshire 1992
New Jersey		Yes	State of New Jersey 1992
New Mexico		Yes	State of New Mexico 1991
New York		Yes	State of New York 1992
North Carolina		Yes	State of North Carolina 1992

TABLE 7-1. Regulations and Guidelines Applicable to Used Mineral-based Crankcase Oil (*continued*)

Agency	Description	Information	Reference
STATE (Cont.)			
North Dakota		No	State of North Dakota 1992
Ohio		Yes	State of Ohio 1992
Oklahoma		Yes	State of Oklahoma 1991
Oregon		Yes	State of Oregon 1992
Pennsylvania		Yes	Commonwealth of Pennsylvania 1992
Rhode Island		Yes	State of Rhode Island 1992
South Carolina		Yes	State of South Carolina 1992
South Dakota		Yes	State of South Dakota 1992
Tennessee		No	State of Tennessee 1992
Texas		Yes	State of Texas 1992
Utah		Yes	State of Utah 1992
Vermont		Yes	State of Vermont 1992
Virginia		Yes	Commonwealth of Virginia 1992
Washington		Yes	State of Washington 1992
West Virginia		No	State of West Virginia 1992
Wisconsin		Yes	State of Wisconsin 1992
Wyoming		Yes	State of Wyoming 1992
	Labeling requirement regulations (recycling)		
Alabama		No	State of Alabama 1991
Alaska		No	State of Alaska 1992
Arizona		No	State of Arizona 1992
Arkansas		No	State of Arkansas 1992
California		Yes	State of California 1992
Colorado		No	State of Colorado 1992
Connecticut		No	State of Connecticut 1991
Delaware		No	State of Delaware 1992
Washington, DC		No	District of Columbia 1992
Florida		No	State of Florida 1992
Georgia		No	State of Georgia 1992
Hawaii		No	State of Hawaii 1992
Idaho		Yes	State of Idaho 1992
Illinois		No	State of Illinois 1992
Indiana		Yes	State of Indiana 1992
Iowa		No	State of Iowa 1992
Kansas		No	State of Kansas 1992
Kentucky		Yes (guideline)	Commonwealth of Kentucky 1992
Louisiana		No	State of Louisiana 1992
Maine		No	State of Maine 1992
Maryland		Yes (guideline)	State of Maryland 1992
Massachusetts		No	Commonwealth of Massachusetts 1992
Michigan		No	State of Michigan 1992
Minnesota		No	State of Minnesota 1992
Mississippi		No	State of Mississippi 1992
Missouri		Yes	State of Missouri 1992
Montana		No	State of Montana 1992
Nebraska		No	State of Nebraska 1992
Nevada		No	State of Nevada 1992
New Hampshire		Yes	State of New Hampshire 1992

TABLE 7-1. Regulations and Guidelines Applicable to Used Mineral-based Crankcase Oil (*continued*)

Agency	Description	Information	Reference
STATE (Cont.)			
New Jersey		Yes (hazardous waste)	State of New Jersey 1992
New Mexico		No	State of New Mexico 1991
New York		Yes	State of New York 1992
North Carolina		No	State of North Carolina 1992
North Dakota		No	State of North Dakota 1992
Ohio		No	State of Ohio 1992
Oklahoma		No	State of Oklahoma 1991
Oregon		No	State of Oregon 1992
Pennsylvania		Yes	Commonwealth of Pennsylvania 1992
Rhode Island		Yes	State of Rhode Island 1992
South Carolina		Yes	State of South Carolina 1992
South Dakota		No	State of South Dakota 1992
Tennessee		No	State of Tennessee 1992
Texas		No	State of Texas 1992
Utah		No	State of Utah 1992
Vermont		No	State of Vermont 1992
Virginia		No	Commonwealth of Virginia 1992
Washington		No	State of Washington 1992
West Virginia		No	State of West Virginia 1992
Wisconsin		Yes	State of Wisconsin 1992
Wyoming		Yes	State of Wyoming 1992
	Liability protection statute for collectors (recycling)		
Alabama		No[c]	State of Alabama 1991
Alaska		No[c]	State of Alaska 1992
Arizona		Yes	State of Arizona 1992
Arkansas		No[c]	State of Arkansas 1992
California		Yes	State of California 1992
Colorado		No[c]	State of Colorado 1992
Connecticut		No[c]	State of Connecticut 1991
Delaware		Yes	State of Delaware 1992
Washington, DC		No[c]	District of Columbia 1992
Florida		Yes	State of Florida 1992
Georgia		No[c]	State of Georgia 1992
Hawaii		No[c]	State of Hawaii 1992
Idaho		No[c]	State of Idaho 1992
Illinois		No[c]	State of Illinois 1992
Indiana		No[c]	State of Indiana 1992
Iowa		No[c]	State of Iowa 1992
Kansas		No[c]	State of Kansas 1992
Kentucky		No[c]	Commonwealth of Kentucky 1992
Louisiana		Yes	State of Louisiana 1992
Maine		No[c]	State of Maine 1992
Maryland		Yes (guideline)	State of Maryland 1992
Massachusetts		No[c]	Commonwealth of Massachusetts 1992
Michigan		No[c]	State of Michigan 1992
Minnesota		No[c]	State of Minnesota 1992
Mississippi		Yes	State of Mississippi 1992

TABLE 7-1. Regulations and Guidelines Applicable to Used Mineral-based Crankcase Oil (*continued*)

Agency	Description	Information	Reference
STATE (Cont.)			
Missouri		No[c]	State of Missouri 1992
Montana		No[c]	State of Montana 1992
Nebraska		No[c]	State of Nebraska 1992
Nevada		No[c]	State of Nevada 1992
New Hampshire		No[c]	State of New Hampshire 1992
New Jersey		No[c]	State of New Jersey 1992
New Mexico		No[c]	State of New Mexico 1991
New York		Yes	State of New York 1992
North Carolina		No[c]	State of North Carolina 1992
North Dakota		No[c]	State of North Dakota 1992
Ohio		No[c]	State of Ohio 1992
Oklahoma		No[c]	State of Oklahoma 1991
Oregon		No[c]	State of Oregon 1992
Pennsylvania		Yes	Commonwealth of Pennsylvania 1992
Rhode Island		No[c]	State of Rhode Island 1992
South Carolina		Yes	State of South Carolina 1992
South Dakota		No[c]	State of South Dakota 1992
Tennessee		No[c]	State of Tennessee 1992
Texas		Yes	State of Texas 1992
Utah		No[c]	State of Utah 1992
Vermont		No[c]	State of Vermont 1992
Virginia		No[c]	Commonwealth of Virginia 1992
Washington		Yes	State of Washington 1992
West Virginia		No[c]	State of West Virginia 1992
Wisconsin		No[c]	State of Wisconsin 1992
Wyoming		No[c]	State of Wyoming 1992
	Transportation requirements for used oil burned for energy recovery (recycling)		
Alabama		Yes	State of Alabama 1991
Alaska		Yes	State of Alaska 1992
Arizona		Yes	State of Arizona 1992
Arkansas		Yes	State of Arkansas 1992
California		Yes	State of California 1992
Colorado		No	State of Colorado 1992
Connecticut		Yes	State of Connecticut 1991
Delaware		Yes	State of Delaware 1992
Washington, DC		No	District of Columbia 1992
Florida		Yes	State of Florida 1992
Georgia		Yes	State of Georgia 1992
Hawaii		Yes	State of Hawaii 1992
Idaho		Yes	State of Idaho 1992
Illinois		Yes	State of Illinois 1992
Indiana		Yes	State of Indiana 1992
Iowa		No	State of Iowa 1992
Kansas		Yes	State of Kansas 1992
Kentucky		Yes	Commonwealth of Kentucky 1992
Louisiana		Yes	State of Louisiana 1992
Maine		Yes	State of Maine 1992

TABLE 7-1. Regulations and Guidelines Applicable to Used Mineral-based Crankcase Oil (*continued*)

Agency	Description	Information	Reference
STATE (Cont.)			
Maryland		Yes	State of Maryland 1992
Massachusetts		Yes	Commonwealth of Massachusetts 1992
Michigan		Yes	State of Michigan 1992
Minnesota		Yes	State of Minnesota 1992
Mississippi		Yes	State of Mississippi 1992
Missouri		Yes	State of Missouri 1992
Montana		Yes	State of Montana 1992
Nebraska		Yes	State of Nebraska 1992
Nevada		Yes	State of Nevada 1992
New Hampshire		Yes	State of New Hampshire 1992
New Jersey		Yes	State of New Jersey 1992
New Mexico		Yes	State of New Mexico 1991
New York		Yes	State of New York 1992
North Carolina		Yes	State of North Carolina 1992
North Dakota		Yes	State of North Dakota 1992
Ohio		Yes	State of Ohio 1992
Oklahoma		Yes	State of Oklahoma 1991
Oregon		Yes	State of Oregon 1992
Pennsylvania		Yes	Commonwealth of Pennsylvania 1992
Rhode Island		Yes	State of Rhode Island 1992
South Carolina		Yes	State of South Carolina 1992
South Dakota		Yes	State of South Dakota 1992
Tennessee		No	State of Tennessee 1992
Texas		Yes	State of Texas 1992
Utah		Yes	State of Utah 1992
Vermont		Yes	State of Vermont 1992
Virginia		Yes	Commonwealth of Virginia 1992
Washington		Yes	State of Washington 1992
West Virginia		No	State of West Virginia 1992
Wisconsin		Yes	State of Wisconsin 1992
Wyoming		Yes	State of Wyoming 1992
	Used oil landfill disposal regulations (disposal)		
Alabama		No	State of Alabama 1991
Alaska		No	State of Alaska 1992
Arizona		Yes	State of Arizona 1992
Arkansas		No	State of Arkansas 1992
California		Yes	State of California 1992
Colorado		Yes (liquid waste)	State of Colorado 1992
Connecticut		Yes	State of Connecticut 1991
Delaware		No	State of Delaware 1992
Washington, DC		No	District of Columbia 1992
Florida		Yes	State of Florida 1992
Georgia		Yes	State of Georgia 1992
Hawaii		Yes	State of Hawaii 1992
Idaho		Yes	State of Idaho 1992
Illinois		Yes	State of Illinois 1992
Indiana		Yes	State of Indiana 1992
Iowa		Yes	State of Iowa 1992

TABLE 7-1. Regulations and Guidelines Applicable to Used Mineral-based Crankcase Oil (*continued*)

Agency	Description	Information	Reference
STATE (Cont.)			
Kansas		Yes	State of Kansas 1992
Kentucky		Yes	Commonwealth of Kentucky 1992
Louisiana		Yes	State of Louisiana 1992
Maine		Yes	State of Maine 1992
Maryland		Yes	State of Maryland 1992
Massachusetts		Yes	Commonwealth of Massachusetts 1992
Michigan		Yes	State of Michigan 1992
Minnesota		Yes	State of Minnesota 1992
Mississippi		Yes (liquid waste)	State of Mississippi 1992
Missouri		Yes	State of Missouri 1992
Montana		No	State of Montana 1992
Nebraska		No	State of Nebraska 1992
Nevada		Yes	State of Nevada 1992
New Hampshire		Yes	State of New Hampshire 1992
New Jersey		Yes	State of New Jersey 1992
New Mexico		No	State of New Mexico 1991
New York		Yes	State of New York 1992
North Carolina		Yes	State of North Carolina 1992
North Dakota		Yes	State of North Dakota 1992
Ohio		No	State of Ohio 1992
Oklahoma		No	State of Oklahoma 1991
Oregon		Yes	State of Oregon 1992
Pennsylvania		Yes	Commonwealth of Pennsylvania 1992
Rhode Island		Yes	State of Rhode Island 1992
South Carolina		Yes	State of South Carolina 1992
South Dakota		No	State of South Dakota 1992
Tennessee		No	State of Tennessee 1992
Texas		Yes	State of Texas 1992
Utah		Yes	State of Utah 1992
Vermont		Yes	State of Vermont 1992
Virginia		Yes (free liquid)	Commonwealth of Virginia 1992
Washington		Yes	State of Washington 1992
West Virginia		Yes (free liquid)	State of West Virginia 1992
Wisconsin		Yes	State of Wisconsin 1992
Wyoming		Yes	State of Wyoming 1992
	Regulations on used oil as a dust suppressant and as used for road oiling (disposal)		
Alabama		Yes	State of Alabama 1991
Alaska		Yes	State of Alaska 1992
Arizona		Yes	State of Arizona 1992
Arkansas		Yes	State of Arkansas 1992
California		Yes	State of California 1992
Colorado		Yes	State of Colorado 1992
Connecticut		Yes	State of Connecticut 1991
Delaware		Yes	State of Delaware 1992
Washington, DC		Yes	District of Columbia 1992

TABLE 7-1. Regulations and Guidelines Applicable to Used Mineral-based Crankcase Oil (*continued*)

Agency	Description	Information	Reference
STATE (Cont.)			
Florida		Yes	State of Florida 1992
Georgia		Yes	State of Georgia 1992
Hawaii		Yes	State of Hawaii 1992
Idaho		Yes	State of Idaho 1992
Illinois		Yes	State of Illinois 1992
Indiana		Yes	State of Indiana 1992
Iowa		Yes	State of Iowa 1992
Kansas		Yes	State of Kansas 1992
Kentucky		Yes	Commonwealth of Kentucky 1992
Louisiana		Yes	State of Louisiana 1992
Maine		Yes	State of Maine 1992
Maryland		Yes	State of Maryland 1992
Massachusetts		Yes	Commonwealth of Massachusetts 1992
Michigan		Yes	State of Michigan 1992
Minnesota		Yes	State of Minnesota 1992
Mississippi		Yes	State of Mississippi 1992
Missouri		Yes	State of Missouri 1992
Montana		Yes	State of Montana 1992
Nebraska		Yes	State of Nebraska 1992
Nevada		Yes	State of Nevada 1992
New Hampshire		Yes	State of New Hampshire 1992
New Jersey		Yes	State of New Jersey 1992
New Mexico		Yes	State of New Mexico 1991
New York		Yes	State of New York 1992
North Carolina		Yes	State of North Carolina 1992
North Dakota		Yes	State of North Dakota 1992
Ohio		Yes	State of Ohio 1992
Oklahoma		Yes	State of Oklahoma 1991
Oregon		Yes	State of Oregon 1992
Pennsylvania		Yes	Commonwealth of Pennsylvania 1992
Rhode Island		Yes	State of Rhode Island 1992
South Carolina		Yes	State of South Carolina 1992
South Dakota		Yes	State of South Dakota 1992
Tennessee		Yes	State of Tennessee 1992
Texas		Yes	State of Texas 1992
Utah		Yes	State of Utah 1992
Vermont		Yes	State of Vermont 1992
Virginia		Yes	Commonwealth of Virginia 1992
Washington		Yes	State of Washington 1992
West Virginia		No	State of West Virginia 1992
Wisconsin		Yes	State of Wisconsin 1992
Wyoming		Yes	State of Wyoming 1992
	Used oil classification as a hazardous waste by the state		
Alabama		No	State of Alabama 1991
Alaska		No	State of Alaska 1992
Arizona		No	State of Arizona 1992
Arkansas		No	State of Arkansas 1992
California		Yes[d]	State of California 1992

TABLE 7-1. Regulations and Guidelines Applicable to Used Mineral-based Crankcase Oil (*continued*)

Agency	Description	Information	Reference
STATE (Cont.)			
Colorado		No	State of Colorado 1992
Connecticut		No	State of Connecticut 1991
Delaware		No	State of Delaware 1992
Washington, DC		No	District of Columbia 1992
Florida		Yes[d]	State of Florida 1992
Georgia		No	State of Georgia 1992
Hawaii		No	State of Hawaii 1992
Idaho		No	State of Idaho 1992
Illinois		Yes[d]	State of Illinois 1992
Indiana		No	State of Indiana 1992
Iowa		No	State of Iowa 1992
Kansas		No	State of Kansas 1992
Kentucky		No	Commonwealth of Kentucky 1992
Louisiana		No	State of Louisiana 1992
Maine		No	State of Maine 1992
Maryland		No	State of Maryland 1992
Massachusetts		Yes	Commonwealth of Massachusetts 1992
Michigan		No	State of Michigan 1992
Minnesota		Yes (non-recycled)	State of Minnesota 1992
Mississippi		No	State of Mississippi 1992
Missouri		Yes	State of Missouri 1992
Montana		No	State of Montana 1992
Nebraska		No	State of Nebraska 1992
Nevada		No	State of Nevada 1992
New Hampshire		Yes	State of New Hampshire 1992
New Jersey		Yes	State of New Jersey 1992
New Mexico		No	State of New Mexico 1991
New York		No	State of New York 1992
North Carolina		No	State of North Carolina 1992
North Dakota		No	State of North Dakota 1992
Ohio		No	State of Ohio 1992
Oklahoma		No	State of Oklahoma 1991
Oregon		No	State of Oregon 1992
Pennsylvania		No	Commonwealth of Pennsylvania 1992
Rhode Island		Yes	State of Rhode Island 1992
South Carolina		No	State of South Carolina 1992
South Dakota		No	State of South Dakota 1992
Tennessee		No	State of Tennessee 1992
Texas		No	State of Texas 1992
Utah		No	State of Utah 1992
Vermont		Yes	State of Vermont 1992
Virginia		No	Commonwealth of Virginia 1992
Washington		No	State of Washington 1992
West Virginia		No	State of West Virginia 1992
Wisconsin		No	State of Wisconsin 1992
Wyoming		Yes	State of Wyoming 1992

TABLE 7-1. Regulations and Guidelines Applicable to Used Mineral-based Crankcase Oil (*continued*)

Agency	Description	Information	Reference
STATE (Cont.)			
	State regulation concerning the tracking of used oil (end-use regulation)		
Alabama		Yes	State of Alabama 1991
Alaska		Yes	State of Alaska 1992
Arizona		Yes	State of Arizona 1992
Arkansas		Yes	State of Arkansas 1992
California		Yes	State of California 1992
Colorado		No	State of Colorado 1992
Connecticut		Yes	State of Connecticut 1991
Delaware		Yes	State of Delaware 1992
Washington, DC		No	District of Columbia 1992
Florida		Yes	State of Florida 1992
Georgia		Yes	State of Georgia 1992
Hawaii		Yes	State of Hawaii 1992
Idaho		No	State of Idaho 1992
Illinois		Yes	State of Illinois 1992
Indiana		Yes	State of Indiana 1992
Iowa		No	State of Iowa 1992
Kansas		Yes	State of Kansas 1992
Kentucky		Yes	Commonwealth of Kentucky 1992
Louisiana		Yes	State of Louisiana 1992
Maine		Yes	State of Maine 1992
Maryland		Yes	State of Maryland 1992
Massachusetts		Yes	Commonwealth of Massachusetts 1992
Michigan		Yes	State of Michigan 1992
Minnesota		Yes	State of Minnesota 1992
Mississippi		Yes	State of Mississippi 1992
Missouri		Yes	State of Missouri 1992
Montana		Yes	State of Montana 1992
Nebraska		Yes	State of Nebraska 1992
Nevada		Yes	State of Nevada 1992
New Hampshire		Yes	State of New Hampshire 1992
New Jersey		Yes	State of New Jersey 1992
New Mexico		Yes	State of New Mexico 1991
New York		Yes	State of New York 1992
North Carolina		Yes	State of North Carolina 1992
North Dakota		No	State of North Dakota 1992
Ohio		Yes	State of Ohio 1992
Oklahoma		Yes	State of Oklahoma 1991
Oregon		Yes	State of Oregon 1992
Pennsylvania		Yes	Commonwealth of Pennsylvania 1992
Rhode Island		Yes	State of Rhode Island 1992
South Carolina		Yes	State of South Carolina 1992
South Dakota		Yes	State of South Dakota 1992
Tennessee		No	State of Tennessee 1992
Texas		Yes	State of Texas 1992
Utah		Yes	State of Utah 1992
Vermont		Yes	State of Vermont 1992
Virginia		Yes	Commonwealth of Virginia 1992

TABLE 7-1. Regulations and Guidelines Applicable to Used Mineral-based Crankcase Oil (*continued*)

Agency	Description	Information	Reference
STATE (Cont.)			
Washington		Yes	State of Washington 1992
West Virginia		No	State of West Virginia 1992
Wisconsin		Yes	State of Wisconsin 1992
Wyoming		No	State of Wyoming 1992

[a]There is sufficient evidence for the carcinogenicity of one sample of used gasoline-engine oil [class 7.2] and limited evidence for the carcinogenicity of some cutting oils [classes 7.1 and 7.2] to experimental animals. There is sufficient evidence from studies in humans that mineral oils (containing various additives and impurities) which have been used in occupations such a mulespinning, metal machining and jute processing are carcinogenic to humans.
[b]The state has adopted the Federal Regulation found in 40 CFR 266.30-266.44.
[c]The "No" indicates that the state does not specifically recognize used oil, so the liability regulations may or may not apply.
[d]Used crankcase oil is not a hazardous waste, but is regulated as a hazardous waste.

EPA = Environmental Protection Agency; IARC = International Agency for Research on Cancer; NS = not specified; RCRA = Resource Conservation and Recovery Act; VOC = Volatile Organic Compound

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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.

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 15364 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 doserbut 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 $\mu g/m^3$ 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

ATSDR MINIMAL RISK LEVEL

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) [42 U.S.C. 9601 et seq.], as amended by the Superfund Amendments and Reauthorization Act (SARA) [Pub. L. 99-4991, requires that the Agency for Toxic Substances and Disease Registry (ATSDR) develop jointly with the U.S. Environmental Protection Agency (EPA), in order of priority, a list of hazardous substances most commonly found at facilities on the CERCLA National Priorities List (NPL); prepare toxicological profiles for each substance included on the priority list of hazardous substances; and assure the initiation of a research program to fill identified data needs associated with the substances.

The toxicological profiles include an examination, summary, and interpretation of available toxicological information and epidemiologic evaluations of a hazardous substance. During the development of toxicological profiles, Minimal Risk Levels (MRLs) are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given route of exposure. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure. MRLs are based on noncancer health effects only and are not based on a consideration of cancer effects. These substance-specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors to identify contaminants and potential health effects that may be of concern at hazardous waste sites. It is important to note that MRLs are not intended to define clean-up or action levels.

MRLs are derived for hazardous substances using the no-observed-adverse-effect level/uncertainty factor approach. They are below levels that might cause adverse health effects in the people most sensitive to such chemical-induced effects. MRLs are derived for acute (1-14 days), intermediate (15-364 days), and chronic (365 days and longer) durations and for the oral and inhalation routes of exposure. Currently, MRLs for the dermal route of exposure are not derived because ATSDR has not yet identified a method suitable for this route of exposure. MRLs are generally based on the most sensitive chemical-induced end point considered to be of relevance to humans. Serious health effects (such as irreparable damage to the liver or kidneys, or birth defects) are not used as a basis for establishing MRLs. Exposure to a level above the MRL does not mean that adverse health effects will occur.

APPENDIX A

MRLs are intended only to serve as a screening tool to help public health professionals decide where to look more closely. They may also be viewed as a mechanism to identify those hazardous waste sites that are not expected to cause adverse health effects. Most MRLs contain a degree of uncertainty because of the lack of precise toxicological information on the people who might be most sensitive (e.g., infants, elderly, nutritionally or immunologically compromised) to the effects of hazardous substances. ATSDR uses a conservative (i.e., protective) approach to address this uncertainty consistent with the public health principle of prevention. Although human data are preferred, MRLs often must be based on animal studies because relevant human studies are lacking. In the absence of evidence to the contrary, ATSDR assumes that humans are more sensitive to the effects of hazardous substance than animals and that certain persons may be particularly sensitive. Thus, the resulting MRL may be as much as a hundredfold below levels that have been shown to be nontoxic in laboratory animals.

Proposed MRLs undergo a rigorous review process: Health Effects/MRL Workgroup reviews within the Division of Toxicology, expert panel peer reviews, and agencywide MRL Workgroup reviews, with participation from other federal agencies and comments from the public. They are subject to change as new information becomes available concomitant with updating the toxicological profiles. Thus, MRLs in the most recent toxicological profiles supersede previously published levels. For additional information regarding MRLs, please contact the Division of Toxicology, Agency for Toxic Substances and Disease Registry, 1600 Clifton Road, Mailstop E-29, Atlanta, Georgia 30333.

APPENDIX A

MINIMAL RISK LEVEL WORKSHEETS

Data were not sufficient for the derivation of Minimal Risk Levels (MRLs). Therefore, there are no MRL worksheets for used mineral-based crankcase oil.

APPENDIX B
USER'S GUIDE

Chapter 1

Public Health Statement

This chapter of the profile is a health effects summary written in non-technical language. Its intended audience is the general public especially people living in the vicinity of a hazardous waste site or chemical 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 chemical.

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-l) 2-2, and 2-3) and figures (2-l and 2-2) are used to summarize health effects and illustrate graphically levels of exposure associated with those effects. These levels cover health effects observed at increasing dose concentrations and durations, differences in response by species, minimal risk levels (MRLs) to humans for noncancer end points, and EPA's estimated range associated with an upperbound individual lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. Use the LSE tables and figures 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. 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), or Cancer Effect Levels (CELs).

The legends presented below demonstrate the application of these tables and figures. Representative examples 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 exists, 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. Not all substances will have data on each route of exposure and will not therefore have all five of the tables and figures.

(2) Exposure Period Three exposure periods - acute (less than 15 days), intermediate (15-364 days), and chronic (365 days or more) are presented within each relevant route of exposure. In this

example, an inhalation study of intermediate exposure duratoin is reported. For quick reference to health effects occuring from a known length of exposure, locate the applicable exposure period with the LSE table and figure.

(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 (see key number 18).

(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 derive a NOAEL and a Less Serious LOAEL (also see the 2 "18r" data points in Figure 2-l).

(5) Species The test species, whether animal or human, are identified in this column. Section 2.5, "Relevance to Public Health," covers the relevance of animal data to human toxicity and Section 2.3, "Toxicokinetics," contains any available information on comparative Toxicokinetics. Although NOAELs and LOAELs are species specific, the levels are extrapolated to equivalent human doses to derive an MRL.

(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 IS), rats were exposed to 1,1,2,2-tetrachloroethane via inhalation for 6 hours per day, 5 days per week, for 3 weeks. For a more complete review of the dosing regimen refer to the appropriate sections of the text or the original reference paper, i.e., Nitschke et al. 1981.

(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, 1 systemic effect (respiratory) was investigated.

(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 dose 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 endpoint used to quantify the adverse effect accompanies the LOAEL. The respiratory effect reported in key number 18 (hyperplasia) is a Less serious LOAEL of 10 ppm. MRLs are not derived from Serious LOAELs.

(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 epidemiologic studies. CELs are always considered serious

effects. The LSE tables and figures do not contain NOAELs for cancer, but the text may report doses not causing measurable cancer increases.

(11) 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 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 concentrations for particular exposure periods.

(13) Exposure Period 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) Health Effect These are the categories of health effects for which reliable quantitative data exists. The same health effects appear in the LSE table.

(15) Levels of Exposure concentrations or doses for each health effect in the LSE tables are graphically displayed in the LSE figures. Exposure concentration or dose is measured on the log scale "y" axis. Inhalation exposure is reported in mglm' or ppm and oral exposure is reported in mg/kg/day.

(16) NOAEL In this example, 18r NOAEL is the critical endpoint for which an intermediate inhalation exposure MRL is based. As you can see from the LSE figure key, the open-circle symbol indicates to 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) CEL Key number 38r is 1 of 3 studies for which Cancer Effect Levels were derived. The diamond symbol refers to a Cancer Effect Level for the test species-mouse. The number 38 corresponds to the entry in the LSE table.

(18) Estimated Upper-Bound Human Cancer Risk Levels 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 the EPA's Human Health Assessment Group's upper-bound estimates of the slope of the cancer dose response curve at low dose levels (q_1*).

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

SAMPLE

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) Less serious (ppm)	LOAEL (effect) Serious (ppm)	Reference
2 → INTERMEDIATE EXPOSURE							
	5	6	7	8	9		10
3 → Systemic	↓	↓	↓	↓	↓		↓
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

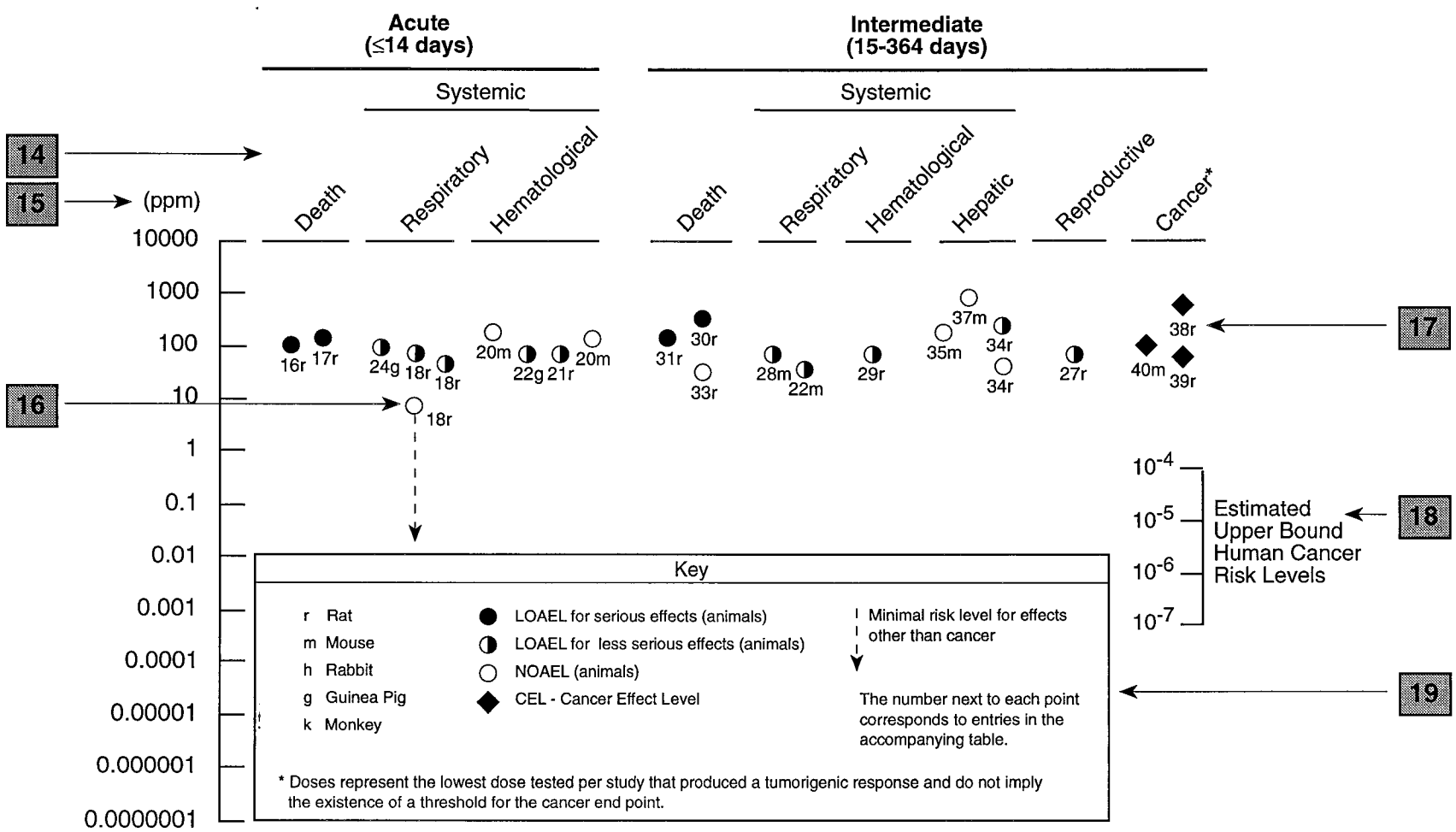
[a] The number corresponds to entries in Figure 2-1.

12 → [b] Used to derive an intermediate inhalation Minimal Risk Level (MRL) of 5 x 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 = days(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

13 → **Figure 2-1. Levels of Significant Exposure to [Chemical X] – Inhalation**



Chapter 2 (Section 2.5)

Relevance to Public Health

The Relevance to Public Health section provides a health effects summary based on evaluations of existing toxicologic, epidemiologic, 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 covers end points in the same order they appear within the Discussion of Health Effects by Route of Exposure section, by route (inhalation, oral, dermal) and within route by effect. Human data are presented first, then animal data. Both are organized by duration (acute, intermediate, 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. Minimal risk levels (MRLs) for noncancer end points (if derived) and the end points from which they were derived are indicated and discussed.

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

Interpretation of Minimal Risk Levels

Where sufficient toxicologic information is available, we have derived minimal risk levels (MRLs) for inhalation and oral routes of entry at each duration of exposure (acute, intermediate, and 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 chemical emission, given the concentration of a contaminant in air or the estimated daily dose in water. MRLs are based largely on toxicological studies in animals and on reports of human occupational exposure.

MRL users should be familiar with the toxicologic information on which the number is based. Chapter 2.5, “Relevance to Public Health,” contains basic information known about the substance. Other sections such as 2.7, “Interactions with Other Substances,” and 2.8, “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 the Environmental Protection Agency (EPA) provides (Barnes and Dourson 1988) to determine reference doses for lifetime exposure (RfDs).

To derive an MRL, ATSDR generally selects the most sensitive endpoint 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 systemic, neurological, and developmental effects. If this information and reliable quantitative data on the chosen endpoint 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. When a NOAEL is not available, a lowest-observed-adverse-effect level (LOAEL) can be used to derive an MRL, and an uncertainty factor (UF) of 10 must be employed. Additional uncertainty factors of 10 must be used both for human variability to protect sensitive subpopulations (people who are most susceptible to the health effects caused by the substance) and for interspecies variability (extrapolation from animals to humans). In deriving an MRL, these individual uncertainty factors are multiplied together. The product is then divided into the 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 C

ACRONYMS, ABBREVIATIONS, AND SYMBOLS

ACGIH	American Conference of Governmental Industrial Hygienists
ADME	Absorption, Distribution, Metabolism, and Excretion
AML	acute myeloid leukemia
atm	atmosphere
ATSDR	Agency for Toxic Substances and Disease Registry
BCF	bioconcentration factor
BEI	Biological Exposure Index
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
Ci	curie
CLP	Contract Laboratory Program
cm	centimeter
CML	chronic myeloid leukemia
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

APPENDIX C

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
MA	trans,trans-muconic acid
mCi	millicurie
mg	milligram
min	minute
mL	milliliter
mm	millimeter
mm Hg	millimeters of mercury
mmol	millimole
mo	month
mppcf	millions of particles per cubic foot
MRL	Minimal Risk Level
MS	mass spectrometry
NCE	normochromatic erythrocytes
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
PCE	polychromatic erythrocytes
pg	picogram
pmol	picomole
PHS	Public Health Service
PMR	proportionate mortality ratio
ppb	parts per billion
ppm	parts per million

APPENDIX C

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
UMDNJ	University of Medicine and Dentistry New Jersey
U.S.	United States
UF	uncertainty factor
yr	year
WHO	World Health Organization
wk	week

>	greater than
≥	greater than or equal to
=	equal to
<	less than
≤	less than or equal to
%	percent
α	alpha
β	beta
δ	delta
γ	gamma
μm	micrometer
μg	microgram