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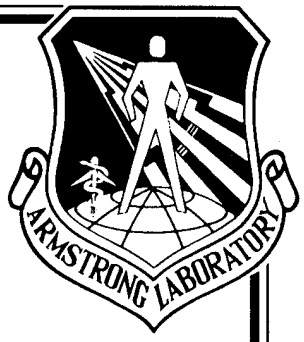

ARMSTRONG LABORATORY

DEVELOPMENT OF A HUMAN HEALTH ORAL RISK FACTOR FOR LONG CHAIN PETROLEUM HYDROCARBONS

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October 1994

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AL/OE-TR-1995-0007

The experiments reported herein were conducted according to the "Guide for the Care and Use of Laboratory Animals," Institute of Laboratory Animal Resources, National Research Council.

This report has been reviewed by the Office of Public Affairs (PA) and is releasable to the National Technical Information Service (NTIS). At NTIS, it will be available to the general public, including foreign nations.

This technical report has been reviewed and is approved for publication.

FOR THE COMMANDER

TERRY A. CHILDRESS, Lt Col, USAF, BSC
Director, Toxicology Division
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The primary objective of the research presented herein was to develop oral risk factors for long chain petroleum hydrocarbons (LCPHs). Literature searches were conducted on petroleum products and specific LCPHs. Over 5,000 references were identified and over 100 references were reviewed. Information was collected on the chemical composition and analysis of total petroleum hydrocarbons (TPH); the environmental regulation of TPH in soils; the weathering of TPH in soils; risk factors previously developed for neat petroleum products and four LCPHs; and on the toxicity of LCPHs. Oral reference doses were developed for nonane, decane, C_{10}-C_{11} isoparaffinic hydrocarbon dearomatized white spirit C_{11}-C_{12}, mineral oil, and petroleum wax. Gaps in the data necessary for the development of risk factors for LCPHs were identified and suggestions were made for future research to elucidate risk assessment at petroleum contamination sites. In addition, four DoD sites were identified for potential demonstration of risk assessment and risk-based cleanup versus cleanup based on regulatory standards for soils.

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PREFACE

The goal of the research presented in this technical report was to develop oral risk factors for long chain petroleum hydrocarbons (LCPHs). Oral reference doses were developed for nonane; decane; C_{10}-C_{11} isoparaffinic hydrocarbon; dearomatized white spirit C_{11}-C_{12}; mineral oil; and petroleum wax. Areas of future research were suggested that would fill data gaps and provide a more reliable basis for oral risk factor development for LCPHs.

This work was supported by the Depart of Defense's Small Business Innovative Research Program and the U.S. Air Force. The contract was awarded May 15, 1994, and the technical contract monitor was Jeffrey Fisher, Ph.D., Toxicology Division, Armstrong Laboratory, Wright-Patterson AFB, OH.

No animals were used in this effort.

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EXECUTIVE SUMMARY

The primary objective of the research presented herein was to develop oral risk factors for long chain petroleum hydrocarbons (LCPHs). Literature searches were conducted on petroleum products and specific LCPHs. Over 5,000 references were identified and over 100 references were reviewed. Information was collected on the chemical composition and analysis of total petroleum hydrocarbons (TPH); the environmental regulation of TPH in soils; the weathering of TPH in soils; risk factors previously developed for neat petroleum products and for LCPHs; and on the toxicity of LCPHs. Oral reference doses were developed for nonane, decane, C_{10}-C_{11} isoparaffinic hydrocarbon, dearomatized white spirit C_{11}-C_{12}, mineral oil, and petroleum wax. Gaps in the data necessary for the development of risk factors for LCPHs were identified and suggestions were made for future research to elucidate risk assessment at petroleum contamination sites. In addition, four DoD sites were identified for potential demonstration of risk assessment and risk-based cleanup versus cleanup based on regulatory standards for soils.

1.0 INTRODUCTION

In January of 1994, Staats Creative Sciences (SCS) responded to Air Force (AF) Topic Number AF94-016 entitled Human Health Standards for Groundwater Contaminants in Solicitation 94.1 of the Department of Defense (DOD) request for proposals in the Small Business Innovation Research (SBIR) Program. The proposal submitted by SCS for a Phase I research project was entitled "Development of a Human Health Risk Factor for Long Chain Petroleum Hydrocarbons". Contract number F41624-94-C-9009 was awarded on May 15, 1994 to SCS for the proposed services. The information contained within this report represents the substantive fulfillment of SCS's contractual obligation to the Air Force and the SBIR program.

Section 1.0 of this report provides introductory and background information on the problem addressed by the research presented. Also, the objectives of the research are defined and a brief description of the technical approach for the research is presented. Section 2.0 describes the literature searches conducted and the types of information collected. Background information on the definition and regulation of Total Petroleum Hydrocarbons (TPH) is provided. A brief discussion of the degradation pattern of TPH and possible locations for a future risk assessment demonstration site are presented. Section 3.0 discusses risk factor development for long chain petroleum hydrocarbons (LCPHs) as well as other TPH components and neat fuels. Risk factors develop previously are discussed along with the risk factor developed herein by SCS. Additional data needed to refine risk factor development are identified in Section 4.0 and future research efforts are proposed that would fill these data gaps. Section 5.0 summarizes the information presented in this report and draws conclusions based on these data. Reference materials are cited in Section 6.0.

1.1 Technical Abstract

The following is the technical abstract submitted in SCS's Phase I research proposal:

Contamination of soil and groundwater by petroleum products is a common problem at DoD facilities. Site-specific human health risk assessments of petroleum contamination, particularly at weathered sites, may save millions of dollars in cleanup costs. However, many risk factors necessary to conduct such risk assessments are not available. Specifically, the risk factor does not exist for LCPHs which are components of petroleum products, primarily diesel fuels. This proposal focuses on development of a risk factor for LCPHs in order to facilitate future site-specific health risk assessments at petroleum sites. In addition, a petroleum contamination site at an AF installation would be identified for a demonstration study of cleanup costs based on non-risk based regulatory cleanup standards versus risk-based cleanup standards.

1.2 Identification and Significance of Problem

Contamination of soil and groundwater with petroleum products is the most common environmental problem at AF bases and other DoD facilities. At the over 4,000 groundwater contamination sites belonging to the AF, 60 percent involve some type of petroleum product. Petroleum products include gasoline, diesel fuel, and jet propulsion (JP) fuel. Millions of dollars are spent each year to assess and remediate petroleum contamination. However, much of this remediation may not be necessary. Site-specific evaluation of the risk to human health from petroleum contamination could save millions of dollars in unnecessary cleanup costs.

Although petroleum products are a complex mixture of hydrocarbons, cleanup of these contaminants are often regulated by a single numerical standard for TPH. The soil cleanup standard for TPH varies between states from 10 ppm to 1,000 ppm. Most states have TPH soil standards below 100 ppm. Unfortunately, most state regulators cannot provide the

scientific and/or technical basis for the TPH standards. In fact, these standards may have originated as arbitrary values set for specific sites.

The various chemicals in petroleum products each have different toxicity and leaching characteristics. Benzene, toluene, ethylbenzene, and xylene (BTEX) are the most toxic and the most mobile components. These chemicals are often regulated individually. Because of their volatility, leaching characteristics, and biodegradation, these chemicals often are not present at old contamination sites. After weathering, petroleum components that do not migrate or biodegrade rapidly, such as LCPH or polynuclear aromatic hydrocarbons (PAHs), remain in the soils. Cleanup of these remaining chemicals to the TPH standards of most states is probably not necessary from a health risk perspective.

For example, at an old diesel fuel spill, the soil concentration of BTEXs is probably quite low but the LCPHs remain. The TPH standard could be exceeded by the LCPHs, even though the more toxic BTEX are virtually gone. Therefore, cleanup would be required by the regulatory agency without regard to the actual health risk associated with the site. The toxicity of LCPH is thought to be quite low, although toxicological data on these chemicals are limited. Also, LCPHs readily adsorb to soils so migration into groundwater and to a receptor is unlikely. A site-specific health risk assessment could show that the cleanup is not necessary at all or is minimal.

In order to perform a quantitative risk assessment for exposure to LCPHs, a chemical-specific risk factor for LCPHs must be available. However, a risk factor for LCPHs has not been developed, primarily due to the limited amount of toxicological data for these compounds. The ultimate goal of the research presented herein is to develop a risk factor for oral exposure of humans to LCPHs. Because LCPHs are a group of many compounds, more than one risk factor may be necessary; however, it is impractical to develop a risk factor for every LCPH because of the analytical costs and because many compounds have not yet been isolated and identified.

1.3 Objectives of Research

The three major technical objectives of this research are enumerated below along with a brief discussion of their attainment by SCS:

OBJECTIVE #1: To Identify and Collect Toxicological and Regulatory Data on LCPHs and Other Chemically Similar Compounds.

These data have been used as the basis for risk factor development. Additionally, deficiencies in the toxicological database necessary for risk factor development have been identified and future areas of research are suggested.

OBJECTIVE #2: To Develop a Risk Factor(s) for LCPHs.

Previous approaches to risk factor development for TPH is presented as well as risk factors developed by SCS.

OBJECTIVE #3: To Identify an AF Site for a Future Demonstration Project.

Four weathered petroleum sites have been identified that could serve as future risk assessment demonstration sites.

1.4 Technical Approach

The technical approach for achieving the above objectives included the following steps:

1. Literature Search and Retrieval - Information on the toxicology and environmental regulation of TPH, LCPHs and other chemically related compounds were obtained through computer literature searches and from Air Force personnel and other members of the TPH Working Group. Once references were obtained via computer, the reference titles were reviewed, and specific articles were identified and collected.

2. Literature Review - First, the information collected was categorized in an effort to streamline the review process. Then, the information was reviewed and the most relevant and appropriate information was identified for inclusion in this research.

3. Development of Risk Factor(s) - First, risk factors which had been developed previously for TPH-related compounds were identified and the methodology was evaluated. These data focused on risk factors for neat fuel products and on TPH representative compounds, rather than on the risk factors previously developed by United States Environmental Protection Agency (USEPA) for such compounds as BTEX and PAHs. Then, the data identified in Step 2 and the previous work was combined into the development of risk factors for LCPHs.

4. Identification of Future Research Requirements - During the performance of Steps 2 and 3, gaps in the data necessary to develop risk factors for TPH-related compounds became evident. Based on these identified gaps, suggestions were developed for future research which would enhance and solidify risk factor development for TPH-related compounds.

5. Identification of Demonstration Site(s) - During communications with Air Force personnel, several sites that have undergone weathering or bioventing were identified.

2.0 DATA COLLECTION

The first step in the research process was to identify and collect information germane to the following topics:

- general definition of TPH
- toxicity of LCPHs and neat fuel products
- governmental regulation of TPH in soils
- general patterns of weathering and degradation of petroleum products in soils
- identification of AF sites for possible risk assessment demonstration

In order to refine the literature search strategy for collecting toxicological information on TPH, a preliminary search was performed to generally define TPH.

2.1 Defining TPH

The term TPH, as the name "total petroleum hydrocarbons" infers, should be the sum total concentration of all hydrocarbons derived from petroleum. However, because of the complexity of the petroleum hydrocarbon mixture, no analytical method is capable of measuring all petroleum hydrocarbons and no analytical method can selectively measure only petroleum-derived hydrocarbons. In fact, hydrocarbons from naturally occurring organic matter or soil type can cause falsely high TPH levels in soils. This interference is particularly a problem with the infrared (IR) analytical methods for TPH. Another problem with the IR Modified EPA #418.1 method is the use of the ozone depleting chemical, freon (Schwerko, 1993). This method is required by many states.

Although the actual range of carbon chain lengths in TPH compounds from petroleum products and refinery streams may be from C_1 - C_{85}, compounds less than C_6 (due to their high volatility) or larger than C_{36} can not be measured with common TPH analytical methodology. In addition, quantitation of C_{24}

C_{36} compounds with standard TPH methodologies is not extremely accurate. Therefore, realistically TPH is defined by the particular analytical method used to measure it (Schwerko, 1993).

The TPH mixture is primarily comprised of the following classes of chemical compounds:

Heterocyclics - hydrocarbon rings with one or more non-carbon atoms; present in small amounts in refined petroleum products (example - pyrrole);

Saturated Aliphatics or Alicyclics - (1) straight or branched (called paraffins, alkanes or methanes; example: ethane) or ring structured (called naphthenes or cycloparaffins; example: cyclohexane) hydrocarbon chains with only single carbon bonds;

Unsaturated Aliphatics or Alicyclics - hydrocarbons with one or more double or triple carbon bonds; includes: (1) alkenes or olefins - straight, branched or cyclic hydrocarbon chains with one or more double carbon bonds (example - ethylene); (2) alkynes or acetylenes - straight or branched hydrocarbon chains with one or more triple carbon bonds (example - 2-butyne);

Aromatics or Arenes - cyclic hydrocarbons with three double carbon bonds in a ring which results in delocalization of electrons in the pi shell around the ring; includes: (1) the single aromatic ring, benzene, which is the basic building block of the other aromatics; (2) alkylbenzenes - functional groups are attached to the benzene ring (examples - TEX); and (3) polynuclear aromatic hydrocarbons (PAHs) - multiple aromatic rings joined together (example - naphthalene) (Schwerko, 1993).

Petroleum hydrocarbons are often classified according to (1) the number of carbons in the molecules or (2) their boiling point. In the refining process, atmospheric and vacuum distillation is used to separate crude oil into three primary fractions, (1) the naphthas (boiling range 120 - 350 °F); (2) the middle distillates (MD) (boiling range 350 - 700 °F); and lubricating oils (boiling range 700 - 1070 °F). Higher boiling fractions are called vacuum residuum (King et al., 1984). The end-use MDs are usually fractionated from 350 - 500 °F and contain C_5 - C_{16} carbon length compounds. Petroleum products produced from the MD fraction include kerosene, jet fuels, diesel fuels, solvents and fuel oils (Sandmeyer, 1981). Gasoline is produced from the naphtha fraction. The major classes of petroleum products, along with their boiling points and range of carbon length compounds are listed in Table 2.1. The percentage by weight of the alkanes in gasoline and diesel fuel are presented in Table 2.2.

Although various petroleum products may have similar boiling points and ranges of carbon chain lengths, the relative concentration of the various compounds may vary greatly. Figure 2.1 depicts the gas chromatograms of gasoline and jet fuels JP-4, JP-7, and JP-8 and exemplifies the variability in concentrations of the petroleum components. For instance, note the increasing concentration of C-11 compounds from gasoline to JP-4 to JP-8 to JP-7. Also note that although JP-4 like gasoline contains compounds below the C_8 range, such as the BTEX, JP-8 and JP-7 do not. This suggests that a TPH cleanup standard based on the toxicity of benzene is probably not appropriate for JP-8 and JP-7.

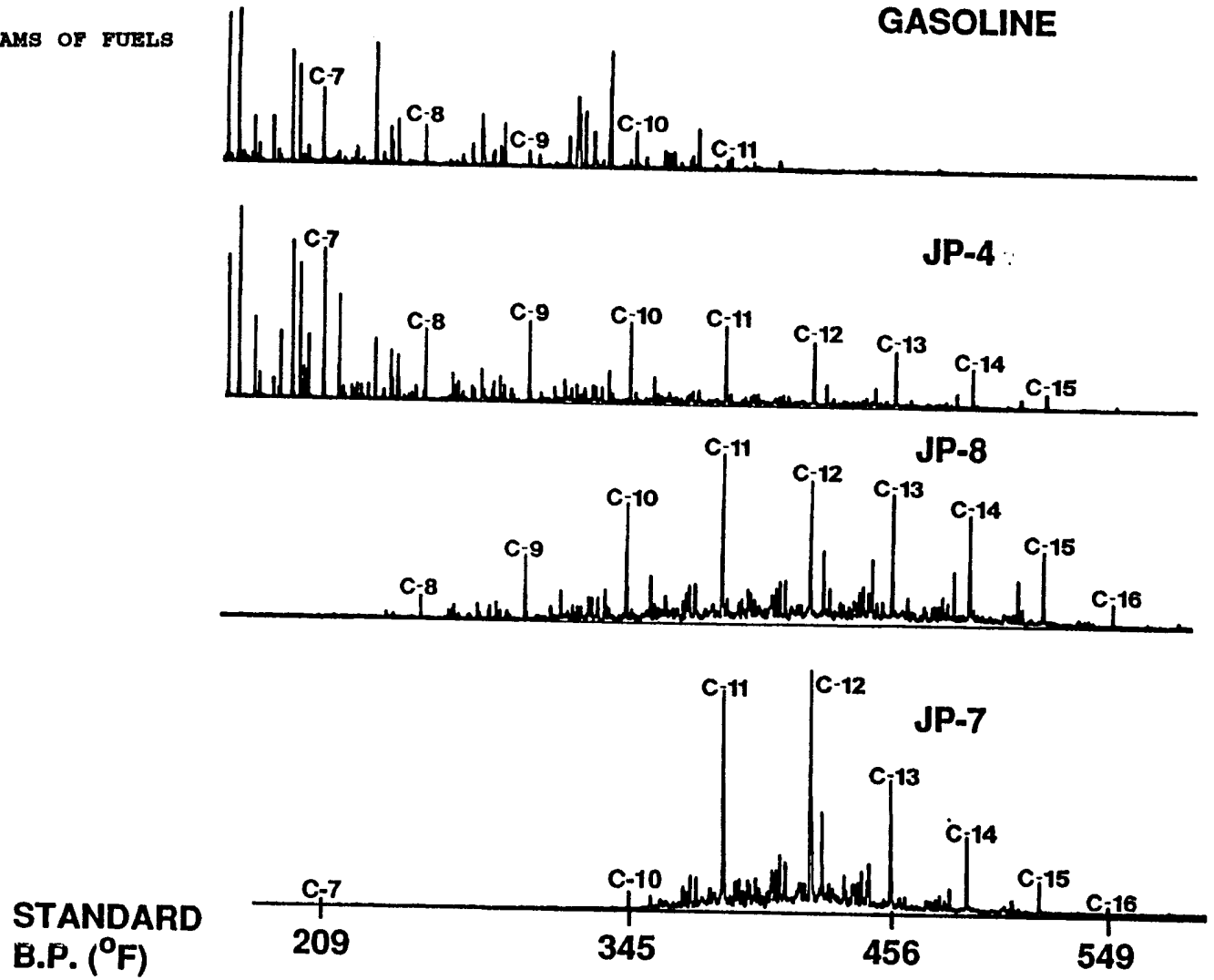
2.2 Literature Search and Retrieval on the Toxicology of TPH

Because of the vast number of articles on TPH and related compounds, the search and collection of information was focused primarily on LCPHs or alkanes such as nonane, decane, dodecane, and tridecane; and on neat petroleum products such as gasoline, kerosine, diesel fuel, or jet fuel. A potential surrogate mixture for TPH, mineral oil, was included as a search term also.

TABLE 2.1 MAJOR CLASSES OF PETROLEUM PRODUCTS		
COMMON NAME	**BOILING POINT (°F)**	**NUMBER OF CARBONS**
Liquefied Petroleum Gases (primarily propane and butane)	>30	C_3 - C_5
Gasolines	90 - 390	C_6 - C_{12}
Kerosines	300 - 480	C_8 - C_{17}
Jet Fuels	300 - 480	C_7 - C_{16}
Diesel Fuels	480 - 660	C_8 - C_{24}
Fuel Oils	390 - 840	C_{12} - C_{30}
Lubricating Oils	570 - 1,000	C_{20} - C_{40}
Adapted from: Jercinovic, 1985; and Schwerko, 1993.		

TABLE 2.2 COMPOSITION OF STRAIGHT CHAIN ALKANES IN GASOLINE AND DIESEL FUEL		
COMPOUND	NUMBER OF CARBONS	CONCENTRATION (weight percent)
GASOLINE		
Propane	3	0.01 - 0.14
n-Butane	4	3.93 - 4.70
n-Pentane	5	5.75 - 10.92
n-Hexane	6	0.24 - 3.50
n-Heptane	7	0.31 - 1.96
n-Octane	8	0.36 - 1.43
n-Nonane	9	0.07 - 0.83
n-Decane	10	0.04 - 0.50
n-Undecane	11	0.05 - 0.22
n-Dodecane	12	0.04 - 0.09
DIESEL		
n-Nonane	9	0.1
n-Decane	10	0.5 - 2
n-Undecane	11	0.98 - 9
n-Dodecane	12	0.96 - 11
n-Tridecane	13	1.1 - 10
n-Tetradecane	14	1.1 - 9
n-Pentadecane	15	1.0 - 7
n-Hexadecane	16	1.2 - 6
n-Heptadecane	17	1.2 - 6
n-Octadecane	18	0.82 - 5
n-Nonadecane	19	0.53 - 4
n-Eicosane	20	0.23 - 3
n-Heneicosane	21	1
n-Docosane	22	<0.2
CLUFTTF, 1989.		

FIGURE 2.1 GAS CHROMATOGRAMS OF FUELS



Adapted from: Fuels and Lubricants Laboratory, Wright-Patterson AFB.

Initial computer literature searches were performed via modem on the National Library of Medicine's Medlars System. The specific searches and the number of references retrieved per search are listed in Table 2.3. A total of over 5,000 references were retrieved; however, some of these references are duplicated between searches. After reviewing the titles and abstracts, over 100 pertinent papers were identified and collected.

Diskettes containing the retrieved references for each search as well as the collected articles were forwarded to Air Force Captain Wade Weisman, chairman of the TPH Working Group. This information will be included in the TPH database and repository that is under development by this group. The TPH Working Group is comprised of representatives from the Air Force, USEPA, various state environmental regulatory agencies, numerous corporations in the petroleum industry, and other individuals interested in the issue of petroleum contamination and cleanup. This database is also a deliverable to Dr. Jeff Fisher, the contract technical monitor for this project.

Unpublished as well as published literature also was retrieved from researchers at the Air Force's Armstrong Laboratories at Wright-Patterson AFB and Tyndall AFB. Other information was collected from the American Petroleum Institute (API) and from other members of the TPH Working Group. Non-copyrighted information also was forwarded to the TPH Working Group for inclusion in the TPH database and repository.

The focus of this work is to develop oral risk factors. Therefore, the literature search focused primarily on finding oral exposure data. However, so little oral data is available that inhalation exposure data on neat products and LCPHs was also collected. In addition, some dermal exposure data was collected also. The inhalation and dermal data on neat products and LCPHs also adds supporting evidence for systemic toxic effects. The identified toxicology literature germane to development of risk factors for TPH is discussed below in Section 3.3.

2.3 Regulation of TPH in the Environment

The second category of data identified deals with the regulation of TPH in the environment by governmental agencies. A federal regulatory standard for TPH in soil has not been set by the U.S. USEPA. However, many states have cleanup guidelines or standards for TPH in soil; these are listed in Table 2.4. The standard ranges from 10 to 10,000 ppm and most commonly is 100 ppm. Many other states allow site-specific cleanup criteria for TPH in soils. Most of states have additional soil cleanup guidelines for some of the specific chemical components of TPH, such as BTEX.

Several states allow consideration of site-specific geological characteristics and health risk in the derivation of the TPH soil cleanup level. The methodologies of some of these states are discussed below.

California has developed a phased approach to leaking underground fuel tank site investigation and cleanup of petroleum contamination that is tailored to the severity of each specific site (CLUFTTF, 1989). Sites are categorized based on the extent of contamination. A scoring system was developed for estimating the concentrations of TPH and BTEX that can be left in place without threatening groundwater. Sites are scored numerically based on the following characteristics: depth to groundwater; fractures in subsurface; average annual rainfall; man-made conduits; unique site features such as recharge area, coarse soil, and nearby wells, etc. The scores correspond to categories of low, medium, or high leaching potential and maximum allowable BTEX and TPH levels for sites in each category. These allowable levels for soil TPH from gasoline and diesel, respectively, are: low leachability - 1000/10,000 ppm; medium leachability - 100/1,000 ppm; and high leachability - 10/100 ppm. Levels were set for each of the BTEX for sites in each leaching category.

The allowable levels in soil were determined by using two mathematical models in tandem: the SESOIL (seasonal soil compartment) model which simulates movement of pollutants from the vadose zone into groundwater; and the AT123D model which is

TABLE 2.3 LITERATURE SEARCHES ON NATIONAL LIBRARY OF MEDICINE MEDLARS SYSTEM		
SEARCH	**DATABASE**	**NO. OF REFERENCES**
nonane or decane or dodecane or tridecane or alkanes with: English only	Toxline	1-500 501-1,133
petroleum or gasoline or kerosine or diesel or jet-fuel with: English only	Toxline65	1-1,000 1,001-2,000 2,001-3,061
mineral-oil or paraffin or white-oil or wax with: toxic; English only	Toxline	274
mineral-oil or paraffin or white-oil or wax with: toxic; English only	Toxline65	197
petroleum or gasoline or kerosine or diesel or jet-fuel with: biodegradation; English only	Toxline	370
mineral-oil or paraffin or white-oil or wax with: food additives or laxatives; English only	Medline 1966-1994	28
mineral-oil or paraffin or white-oil or wax with: toxic; English only	Medline 1966-1994	104

TABLE 2.4 CLEANUP GUIDANCE FOR TPH IN SOILS PER STATE[1]

STATE	STANDARD[2] (ppm)	STATE	STANDARD[2] (ppm)
Alabama	100	Montana	100
Alaska	50 (g)[3] 100 (d)[3]	Nevada	100
Arizona	50 (g) 100 (d)	New Hampshire	10 (g)
Arkansas	100	New Mexico	100 (d)
California	10 to 1,000 (g) 100 to 10,000 (d)	North Dakota	100
Colorado	100 - 500	Oklahoma	50 (g)
Delaware	100 (g) 1,000 (d)	Oregon	40 - 130 (g) 100 - 1,000 (d)
Florida	10 - 100	Pennsylvania	10 (g)
Georgia	100	Rhode Island	300
Idaho	40 - 200 (g) 100 (d)	South Dakota	10 - 100
Iowa	100	Tennessee	100
Kansas	100	Texas	100
Maryland	100	Utah	30 - 300 (g) 100 - 500 (d)
Michigan	100 (d)	Washington	100 (g) 200 (d)
Mississippi	100 (d)	Wyoming	30 - 100
Missouri	50 - 500 (g)		

[1] Adapted from: Marencik, 1992.
[2] Action level or recommended cleanup level.
[3] g = gasoline; d = diesel
Note: Many states allow site-specific TPH cleanup levels.

an analytical transient one-, two-, or three- dimensional model designed to estimate the rate of pollutant transport/ transformation in a groundwater system.

If the concentrations of TPH and BTEX in soil do not exceed these allowable levels and there are no anticipated risks or evidence of risks to other resources, such as groundwater, then the site investigation can end and no remedial action is required. If the allowable levels in soil are exceeded, a more sophisticated leaching potential analysis, the general risk appraisal, can be performed. The volume of contaminated soil is considered and the concentrations of BTEX for each 5-feet interval must be summed progressively with vertical distance. This sum is the cumulative contamination level. Tables of acceptable cumulative contamination levels have been developed through modeling for each of the BTEX. Also, an alternate risk appraisal can be performed in which the leaching and transport models are set to site-specific parameter values.

A similar scoring approach has been taken in Oregon (ODEQ, 1994a). Numeric Soil Cleanup Standards have been developed for a scoring system or matrix which evaluates the following parameters: depth to groundwater; mean annual precipitation; native soil or rock type; sensitivity (usability or potability) of the uppermost aquifer; distance to the nearest receptor; and the number of receptors potentially at risk. The numerical scores are divided into three categories or levels. The allowable levels for TPH in soils for gasoline and diesel sites, respectively, are: Level 1 - 40/100 ppm; Level 2 - 80/500 ppm; and Level 3 - 130/1,000 ppm.

Also, Oregon has developed soil cleanup levels in industrial and in residential settings for 76 hazardous chemicals based on risk (ODEQ, 1994b). Acceptable leachate concentrations have been determined using the SESOIL and AT123D models in tandem. The exposure pathways considered were: leaching into groundwater and subsequent ingestion of groundwater; inhalation of volatiles; incidental ingestion by child and by an adult. This is an excellent attempt to incorporate risk into the determination of soil cleanup levels, both direct contact exposures and migration

into groundwater was considered, as well as industrial and residential exposure scenarios.

Washington also has developed a rating matrix procedure (WDE, 1992) that generates a petroleum-contaminated soil cleanup level that is protective of groundwater. This matrix assigns numerical point values to each of five site-specific parameters and the points are summed to determine a final score. The five parameters assessed in the matrix are: depth to groundwater; rainfall; soil type; distance to receptors; and area of contaminated soil. The possible scores have been divided into four to five categories with corresponding cleanup levels for each of the BTEX and for TPH. The cleanup levels were determined based on leachability and transport modeling using the SESOIL and AT123D models. Soil TPH cleanup levels for gasoline and diesel sites, respectively, range from a low of 100/200 ppm to a high of 600/800 ppm.

2.4 Weathering and Degradation of Petroleum Products in Soils

The health risks associated with a weathered site are directly dependent on the type and concentrations of chemical components remaining in the soils after natural degradation; therefore, SCS attempted to locate this type of information in the scientific literature. Very little site-specific information of this type was found. Degradation studies primarily focus on spills of crude oil. However, some general information on the degradation of petroleum products was obtained through personal communications with chemists who age-date petroleum products in soils (Peterson, 1994). In general, normal paraffins are the first class of compounds to degrade. Within ten years, the concentration of normal paraffins should be approximately one-third of their original concentration; and should be completely degraded within 20 years. The rates of degradation of the other components in descending sequence is: branched paraffins, cycloparaffins, and aromatics. This pattern of degradation has been demonstrated very recently by Huesemann (1994) in crude oils and diesel fuel. It is theorized that the olefins are converted into paraffins over time; however, the degradation rates for

olefins has not been studied. Of course, site-specific conditions can significantly alter this pattern of degradation.

Further information may be found in the literature on biodegradation. A literature search was performed by SCS on biodegradation with petroleum or gasoline or kerosine or diesel or jet fuel (see Table 2.2); 370 references were recovered. However, a complete review of these data is not within the scope of this Phase I project. This database is provided to the AF as supplemental information for potential future research.

2.5 Identification of Potential Demonstration Sites

Due to the lack of information on the exact type of petroleum chemicals which remain in soils after weathering, the AF has expressed an interest in conducting research to collect this information. In collaboration with AF personnel, several AF sites have been identified as good sources of weathered petroleum contaminated soils; these are:

(1) Travers City, MI: U.S. Coast Guard Site with weathered jet fuel;
(2) Eglin AFB, extremely aged gasoline spill;
(3) Kelly AFB, two weather JP-4 sites;
(4) WPAFB, a weather JP-4 site now undergoing remediation.

Once the remaining petroleum components at these sites have been identified and quantified and risk factors have been developed, a risk assessment could be performed to demonstrate the cost effectiveness of remediation based on risk rather than on the TPH regulatory requirement.

3.0 RISK FACTOR DEVELOPMENT

Risk factors can be developed for threshold toxicological effects, i.e. Reference Dose (RfD), or for non-threshold carcinogenic effects, i.e. Cancer Slope Factor (CSF). The RfD is defined by the USEPA (1989) as an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. The CSF is defined by the USEPA (1989) as a plausible upper-bound estimate of the probability of a response per unit intake of a chemical over a lifetime. The slope factor is used to estimate an upper-bound probability of an individual developing cancer as a result of a lifetime of exposure to a particular level of a potential carcinogen.

Development of risk factors for petroleum products can be approached by several methods including:

(1) development of risk factors for specific neat petroleum product such as gasoline, diesel fuel, or jet fuels;
(2) development of risk factors for each chemical component of the petroleum mixture;
(3) development of risk factors for petroleum components which are chemically and toxicologically representative of other components;
(4) development of risk factors for a surrogate mixture which is chemically and toxicologically similar to the petroleum hydrocarbon mixture.

There are serious drawbacks to each of these methods. First, risk factors for neat petroleum products may be appropriate for risk assessment at fresh spill sites. However, after weathering many of the most toxic compounds are no longer present in the soils; therefore using these factors for the neat product to determine risk and cleanup levels would not be appropriate at weathered sites.

The identity of each specific petroleum component has not been fully elucidated, and attempting to perform such an analyses at every petroleum contamination site would be quite costly and time consuming. Therefore, method 2 is impractical.

The major shortcomings inherent in methods 3 and 4 is the lack of certainty in choosing the representative compound or the surrogate mixture. The representative or the surrogate may not truly reflect the toxicity of the compounds it represents.

Finally, the toxicological data necessary to support oral risk factor development utilizing any of the above methods is very limited. The toxicological data pertinent to oral risk factor development obtained in the literature search is discussed below.

3.1 Previous Development of Risk Factors for TPHs

Even though toxicological information on petroleum products and components is scant, oral risk factor development for neat petroleum products and for representative compounds has been attempted recently. Estimates of uncertainty have been included in the risk factor calculations to account for the lack of data. These previously developed risk factors are presented below.

3.1.1 USEPA

Recently the USEPA (1992) attempted to develop risk factors for three neat petroleum products: gasoline, JP-4, and JP-5. Table 3.1 presents these provisional risk factors which have not undergone full review by USEPA and are not listed in the USEPA's Integrated Risk Information System (IRIS) database. A major problem with these oral risk factors is that they were derived based on inhalation data rather than oral data; it was assumed that the absorption and the toxicity for both routes are the same. Confidence in these risk factors is low primarily because no chronic oral toxicity data was available for their development. The reference studies used as the basis for risk factor development also is presented in Table 3.1

TABLE 3.1 ORAL RISK FACTORS FOR NEAT PETROLEUM PRODUCTS[1]

CARCINOGENS

COMPOUND	CANCER SLOPE FACTOR (mg/kg/day)$^{-1}$	WEIGHT OF EVIDENCE CLASS	CANCER TYPE	REFERENCE STUDY
Gasoline	1.7 E-3	C	liver tumors	McFarland et al., 1984a; chronic inhalation; female mice; inhalation unit risk 2.1 E-3 ppm^{-1} or 4.75 E-4 (mg/m^3)$^{-1}$ ÷ adult human reference inhalation rate of 20 m^3/day * mouse body weight of 0.0246 kg

NON-CARCINOGENS

COMPOUND	CHRONIC ORAL REFERENCE DOSE (mg/kg/day)	CONFIDENCE LEVEL/ UNCERTAINTY OR MODIFYING FACTORS	CRITICAL EFFECT OR TARGET ORGAN	REFERENCE STUDY
Gasoline	2.0 E-1	low/ 1,000 H,A,D[2]	decreased body weight gain	MacFarland et al., 1984b; chronic inhalation; female rat; NOAEL 292 ppm or 230 mg/m^3 ÷ rat body weight of 0.229 kg * rat inhalation rate of 0.24 m^3/day ÷ 1,000 uncertainty factor
JP-4	8.0 E-2	low/ 10,000 H,A,S,L,D[2]	fatty changes in liver	MacNaughton and Uddin, 1984; MacEwen and Vernot, 1985; subchronic inhalation; female mice; LOAEL 500 mg/m^3 ÷ mouse body weight of 0.0246 * mouse inhalation rate of 0.04 m^3/day ÷ 10,000 uncertainty factor
JP-5	2.0 E-2	low/ 10,000 H,A,S,L,D[2]	fatty changes & vacuolization in liver	MacEwen and Vernot, 1985; subchronic inhalation; female mice; LOAEL 150 mg/m^3 ÷ mouse body weight of 0.0246 * mouse inhalation rate of 0.04 m^3/day ÷ 10,000 uncertainty factor

[1] USEPA, 1992.
[2] An uncertainty factor of 10 was included in the RfD development for each of the following uncertainties (up to a maximum of 10,000):
- Variation in human sensitivity (H);
- Extrapolation from animal data to humans (A);
- Extrapolation from subchronic to chronic NOAEL (S);
- Extrapolation from LOAEL to NOAEL (L);
- Study deficiency or incomplete data (D).

3.1.2 Massachusetts

Very recently the Massachusetts Department of Environmental Protection (MDEP, 1994) Bureau of Waste Site Cleanup published an excellent attempt at developing risk factors for representative petroleum components. Table 3.2 presents the proposed alternate RfDs developed for these compounds which were chosen to represent three classes of alkanes. The class definition was based on carbon chain lengths. Also, pyrene was selected as the representative compound for C_9 - C_{32} aromatics and alkenes. The RfD previously developed for pyrene by the USEPA was identified as the risk factor for this class of compounds.

The RfD chosen for hexane is that developed by the USEPA (1994); however, it must be noted that although this risk factor is listed in USEPA's Health Effects Assessment Summary Tables (HEAST), it has not undergone full USEPA review and therefore, is not available on IRIS. The RfD for nonane was developed by MDEP by estimating the relative toxicity of nonane as one-tenth that of hexane, so the RfD was estimated as one-tenth the RfD for hexane. Although eicosane was chosen as the representative compound, little data on it's toxicity; thus the surrogate mixture mineral oil provided the toxicological basis for alkanes and cycloalkanes for chemicals with carbon chain lengths from 19 to 32.

3.1.3 Diesel Fuel No. 2

Millner, et al., 1992, developed a CSF for diesel fuel no. 2 based on six dermal carcinogenicity studies in mice. The geometric mean of the slope of the dose-response curves or the maximum likelihood estimate (q_1) (MLE) of cancer potency for these studies was 6.7×10^{-4}. The upper 95% confidence limit (at low doses) of the MLE, the q_1^*, was 1.09×10^{-3}. Mechanistic studies suggest that diesel fuel is an epigenetic carcinogen in that it does not directly cause DNA mutations; therefore, it may have a threshold dose below which it is not carcinogenic. Thus, the non-threshold linearized multistage model of carcinogenesis used to develop CSFs would constitute an extremely conservative approach to risk assessment for diesel fuel.

TABLE 3.2 ORAL RISK FACTORS FOR REPRESENTATIVE COMPOUNDS

REPRESENTATIVE COMPOUND	CLASS OF COMPOUNDS	ORAL REFERENCE DOSE (mg/kg/day)	CRITICAL EFFECT/ TARGET ORGAN	REFERENCE STUDY(S)
ALKANES/CYCLOALKANES				
n-Hexane	C_5 - C_8	0.06	neurotoxicity	Dunnick et al., 1989; subchronic inhalation; mice; NOAEL 500
n-Nonane	C_9 - C_{18}	0.6	neurotoxicity	Carpenter et al., 1978; subchronic inhalation; rat; NOAEL 590 ppm
Eicosane[2]	C_{19} - C_{32}	6.0	irritation/ functional changes	API, 1992; chronic oral; male rat; mineral oil; NOAEL 6,000 mg/kg/day ÷ 1,000 uncertainty factor

[1] MDEP, 1994.
[2] Although eicosane was chosen as the representative compound, toxicological data was insufficient to develop a risk factor for this compound. Instead a risk factor was developed based on oral exposure data for mineral oil.

3.2 Development of Risk Factors for LCPHs

Very little oral exposure data is available on LCPHs or neat petroleum products. Subchronic oral exposure data of sufficient quality to support oral risk factor development was identified for only four compounds: the neat product JP-8; a possible representative chemical, n-hexane; and two possible surrogate mixtures, mineral oil and petroleum wax. In addition, subchronic inhalation exposure data sufficient for oral risk factor development for LCPHs was identified for the following compounds and surrogate mixtures: nonane, decane, dearomatized white spirit (DAWS), and C_{10} - C_{11} isoparaffinic hydrocarbon (IPH). Risk factor development and its toxicological basis for each of these compounds are discussed below.

3.2.1 Methodology for RfD Development

The methodology used by USEPA (1992) to develop RfDs for neat petroleum products was used by SCS to develop RfDs for the above-stated compounds. RfDs were developed from the following type of data:

- oral exposure, if available;
- inhalation exposure when oral data was not available;
- subchronic or chronic exposure;
- numerous toxicological indices were evaluated, such as behavior, hematology, gross anatomical changes, and histopathological changes.

The exposure concentrations were mathematically adjusted to result in final units of mg/kg body weight/day. These adjustments included:

- converting an exposure concentration given in units of ppm to mg/m^3 by multiplying ppm by the molecular weight of the chemical by a constant of 24.45;
- converting intermittent exposure to continuous exposure, for example: an exposure of 6 hr/day for 5 days/week would required multiplying the exposure concentration by 6/24 hr and by 5/7 days.

In addition, mathematical manipulations were made to convert inhalation into oral exposure (assuming the same absorbance and toxicity for these routes) and to account for body weight. For example: the continuous exposure concentration would be multiplied by 1/body weight in kg and by the inhalation rate of the animal which is 0.24 m^3/day for rats.

Finally the daily oral dose was divided by an uncertainty factor. A factor of ten is applied for each of the following: extrapolation from animals to humans (A); extrapolation from subchronic to chronic exposure (S); variation in human sensitivity (H); extrapolation from a Lowest Observable Adverse Effect Level (LOAEL) to a No Observable Adverse Effect Level (NOAEL) (L); and insufficiency in the database (D). A maximum uncertainty factor of 10,000 is used by USEPA. The LOAEL was used as the basis for RfD development; if no LOAEL was available, then the highest NOAEL was used.

Data that indicated that the target organ was the male rat kidney and the effect was related to hyaline droplets and $alpha_{2\mu}$-globulin were not considered appropriate as the basis for RfD development because this effect is not applicable to humans (USEPA, 1991). Also, in several of the studies that served as the basis for RfD development, a change in body weight or in the organ weight to body weight ratio was the only observed effect with no significant behavioral, anatomical, hematological, or histopathological effects noted. It is questionable whether this effect, even though it may be statistically significant, is actually adverse. Therefore, SCS adopted the position that these effects are not significantly adverse and the exposure concentrations in these studies would be considered NOAELs, even if the author considered them LOAELs.

The final oral RfDs as well as information concerning their development are listed in Table 3.3. The reference studies chosen as the basis for risk factor development are briefly discussed below.

3.2.2 Hexane

A chronic oral RfD for hexane is listed in HEAST (USEPA, 1994) as 0.06 mg/kg/day; however, this RfD is not listed in IRIS (USEPA, 1994) which indicates that it has not undergone complete review by USEPA. This RfD is based on a LOAEL of 570 mg/kg/day from a 90-day gavage study in rats (Krasavage et al., 1980); neuropathy and testicular atrophy are listed as the critical effects. A maximum uncertainty factor of 10,000 was applied in RfD development. The neurotoxicity of hexane has been observed in many other studies including an epidemiological study conducted by Sanagai (1980) on workers exposed to hexane for 1 - 12 years. A time-weighted average exposure concentration of 73 mg/m^3 was determined and sensorimotor polyneuropathy was the primary effect. This study serves as the basis for the Reference Concentration (RfC) developed by USEPA and listed on IRIS.

Other studies suggest that hexane is more neurotoxic than other short-chain alkanes; for example in rats exposed to 3000 ppm of n-pentane, n-hexane, or n-heptane for 12 hours/day for 16 weeks, hexane disturbed the conduction velocity of the motor nerve and the mixed nerve and prolonged the distal latency in the rat's tail, but the other two alkanes did not (Takeuchi, et al., 1980) Interestingly, the uptake rate of hydrocarbons during inhalation generally increases with increasing carbon chain length (Dahl, et al., 1988); however, the rate of uptake of heptane was less than that of hexane. The rates of uptake for the straight chain alkanes in this study were butane < pentane < heptane < hexane < nonane. Also, the uptake rates of the isoalkanes generally was less than their normal alkane counterparts; for example: isobutane < n-butane; isopentane and neopentane < n-pentane; isohexane < n-hexane. Zahlsen et al., (1992) found that generally uptake of n-alkanes increases with increasing carbon chain length; for example: in rat brain tissue, after a 12 hour inhalation exposure to 100 ppm of alkane with a 12 hour recovery period, alkane concentrations increased in the following manner: hexane > heptane < octane < nonane < decane. A similar pattern was observed in blood, liver, and fat tissues. Nilsen et al., (1988) noted gross ataxia, general and focal seizure, and spasms in rats n-nonane for 8 hours at air concentrations of 3560 ppm

and above. No toxic effects were noted in rats exposed to nonane at 2414 ppm or below or in rats exposed to decane, undecane, dodecane, or tridecane; however, the exposure concentrations of the other alkanes were significantly lower than those concentrations of nonane at which an effect was observed. Kristiansen and Nielsen (1988) found that the potency for depressing respiratory rates in mice increased with increasing carbon chain length, i.e. heptane < octane < nonane < decane < undecane. All these studies involve inhalation exposure. The relative toxicity and absorption of the alkanes may be quite different for oral exposures.

3.2.3 Nonane

A chronic oral RfD for n-nonane was developed by SCS based on the work of Carpenter et al. (1978). Rats were exposed via inhalation to 1600, 590, or 360 ppm of nonane for 6 hours per day, 5 days per week, for 63 days. A suppression of body weight gain was observed at 1600 ppm, no effects were seen in the other treatment groups. A NOAEL of 1600 ppm was mathematically adjusted to an equivalent oral dose (EOD) of 1,017.6 mg/kg/day. An average body weight during exposure of the 1600 ppm group was calculated as 0.353 kg. The molecular weight of nonane is 128.26 g/mole (Lide, 1991). The EOD was divided by an uncertainty factor of 1000 (HAS). Therefore, the chronic oral RfD for nonane is 1.017 mg/kg/day.

3.2.4 Decane

A chronic oral RfD for n-decane was developed by SCS based on inhalation exposure at 540 ppm over 91 days for approximately 17 hours per day (1,501 hours out of 2,184 hours) in rats. Although statistical comparisons are not evident, the author states that mean body weights of treated animals were increased compared to controls after 91 days of exposure; however, the means are within one standard deviation of each other. Also, the authors states that the mean white blood cell count (WBC) was decreased at 57 days after treatment and increased at 91 days after treatment in

comparison to the mean WBC of the controls; however, again these corresponding treated and control means are within one standard deviation. The statistical significance of these effects are questionable. The experiment was continued for 123 days; no other effects were noted. The average body weight during exposure was 0.389 kg. The molecular weight of decane is 142.28 g/mole (Lide, 1991). Based on a NOAEL of 540 ppm, the EOD was calculated to be 1,332.1 mg/kg/day. With an uncertainty factor of 1000 (HAS), the oral RfD is 1.332 mg/kg/day.

3.2.5 IPH and DAWS

IPH contains 100% isoparaffins, primarily in the C_{10} - C_{11} range. DAWS contains <0.5% aromatics, 58% paraffins and 42% naphthenes, primarily in the C_{11} - C_{12} range. Chronic oral RfDs were developed for IPH and for DAWS based on the work of Phillips and Egan (1984). Rats were exposed via inhalation 6 hours/day, 5 days/week for 12 weeks. Increases in liver weight during this study were observed in male and female rats exposed to either DAWS or IPH primarily in the high exposure groups, 890 and 922 ppm, respectively. Increases in total organ weight and in the organ weight to body weight were scattered throughout the sacrifice intervals for liver, kidney, adrenals, gonads, brain, and lungs. However, only the relative weight of liver was increased at all sacrifice intervals in both males and females. Relative kidney weight was increased at all intervals in only the male rat. The authors conclude that these changes were not relevant to the toxicity of the test materials. Male rats exhibited renal effects which were later associated with $alpha_{2\mu}$-globulin. The average body weights during exposure were 0.254 kg. The mean molecular weights of the mixtures were 154 and 149 g/mole for DAWS and IPH, respectively. The EODs were 946.2 and 947.9 mg/kg/day for DAWS and IPH, respectively. With an uncertainty factor of 1000 (HAS), the oral RfDs for DAWS and IPH are 0946 and 0.948 mg/kg/day, respectively.

3.2.6 JP-8

Rats were exposed to neat JP-8 by gavage for 90 days (Mattie, et al., 1994). At the highest dose, 750 mg/kg/day, only a decrease in total body weight and an increase in the brain weight to body weight ratio was observed. With an uncertainty factor of 1000 (HAS), the oral RfD for JP-8 is 0.75 mg/kg/day. In a developmental toxicity study, a LOEL of 500 mg/kg/day was determined for the pregnant rat (Cooper and Mattie, 1994)

3.2.7 Mineral Oil

Many studies have demonstrated the low toxicity of mineral oil. Two 90-day oral gavage studies using a medicinal white oil (PRIMOL 185) in male and female rats conducted by Exxon showed a NOEL at 4,350 mg/kg/day (Exxon Biomedical Sciences, Inc., 1984 and 1985). Using an uncertainty factor of 1,000 (HAS), the oral RfD for mineral oil is 4.35 mg/kg/day. White mineral oil consists of saturated hydrocarbons having carbon numbers in the range of C_{15} - C_{50} (API, 1992). Some toxicity has been demonstrated in studies conducted by Shell Oil; however, these studies are in conflict with all other oral studies on mineral oil toxicity and probable contamination of the feed with hexane has been cited as the mostly likely cause for the discrepancies.

3.2.8 Petroleum Wax

A two-year feeding study on five petroleum waxes showed a NOEL of 6,000 mg/kg/day in male and female rats (Shubik et al., 1962). With an uncertainty factor of 100 (HA), the RfD for petroleum wax is 60 mg/kg/day. Other studies have shown that these waxes are not absorbed from the gastrointestinal tract (API, 1992). Petroleum waxes are any of a range of relatively high-molecular weight hydrocarbons (approximately C_{16} - C_{50}), solid at room temperature, derived from the higher-boiling petroleum fractions.

3.2.9 Summary of Risk Factors

Table 3.3 presents a summary of the risk factors identified or developed by SCS. Interestingly, the RfDs for the C_9 - C_{12} alkane compounds listed up to mineral oil are similar to each other and to that for JP-8. These alkanes comprise the majority of JP-8. Note that the JP-8 reference study involved oral dosing while the reference studies for the C_9 - C_{12} alkane compounds were inhalation exposures. Deriving an oral risk factor based on inhalation studies introduces a great deal of uncertainty because one must assume that the internal dose and the systemic toxicity are the same regardless of exposure route. Also, any local effects peculiar to the route of entry are not considered. For example, C_7 - C_{11} normal alkanes are respiratory irritants which can cause decreased respiration rates in rats; thus less chemical enters the body. This phenomenon results in additional uncertainty in estimating the internal dose of the chemical. Therefore, oral risk factor development is most appropriately based on oral exposure data.

The oral risk factors for the C_9 - C_{12} alkane compounds and JP-8 are approximately an order of magnitude above that for hexane, and below that for mineral oil. This relative toxicity is similar to that assumed by Massachusetts (1994). The toxicity of the very long chain hydrocarbons such as in white mineral oil and petroleum wax is very low.

TABLE 3.3 ORAL RISK FACTORS IDENTIFIED OR DEVELOPED BY SCS

COMPOUND	CRITICAL EFFECT or TARGET ORGAN	EOD[1] (mg/kg/d)	NOAEL[2] or LOAEL[3]	UNCERTAINTY FACTOR[4]	RfD (mg/kg/day)	REFERENCE STUDY
n-Hexane	neuropathy or testicular atrophy	570	570[3] mg/kg/d	10,000 (HASL)	0.06	Krasavage et al., 1980; oral; rat; 90 d; EPA, 1994.
n-Nonane	decreased body weight	1,017.6	1,600[2] ppm	1,000 (HAS)	1.017	Carpenter et al., 1978; inhalation; rat; 6hr/d, 5 d/week, 63 d.
n-Decane	increased body weight? & changes in white blood cell count ?	1,332.1	540[2] ppm	1,000 (HAS)	1.332	Nau et al., 1966; inhalation rat; 17 hr/d, 91 d.
DAWS[5]	increased liver weight?	946.2	890[2] ppm	1,000 (HAS)	0.946	Phillips & Egan, 1984; inhalation; rat; 6 hr/d, 5 d/week, 12 weeks.
IPH[6]	increased liver weight?	947.9	922[2] ppm	1,000 (HAS)	0.948	Phillips & Egan, 1984; inhalation; rat; 6 hr/d, 5 d/week, 12 weeks.
JP-8	decreased body weight	750	750[2] mg/kg/d	1,000 (HAS)	0.75	Mattie et al., 1994; oral; female rats; 90d.
Mineral Oil	none observed	4,350	4,350[2] mg/kg/d	1,000 (HAS)	4.35	Exxon 1984 & 1985; oral; male & female rats; 90d.
Petroleum Wax	none observed	6,000	6,000[2] mg/kg/d	100 (HA)	60	Shubik et al., 1962; oral; male & female rats; 2 yrs.

[1] EOD = Equivalent Oral Dose
[2] NOAEL = No Observable Adverse Effect Level
[3] LOAEL = Lowest Observable Adverse Effect Level
[4] An uncertainty factor of 10 was included in the RfD development for each of the following uncertainties (up to a maximum of 10,000):
- Variation in human sensitivity (H);
- Extrapolation from animal data to humans (A);
- Extrapolation from subchronic to chronic NOAEL (S);
- Extrapolation from LOAEL to NOAEL (L);
- Study deficiency or incomplete data (D).

[5] DAWS = Dearomatized White Spirit
[6] IPH = C_{10} - C_{11} Isoparaffinic Hydrocarbon

4.0 FUTURE RESEARCH

In the process of conducting the work presented above, gaps in the data on TPH and LCPHs became apparent. These gaps are discussed below and suggestions are presented for future research to obtain more reliable and appropriate data necessary to conduct scientifically defensible risk assessments at petroleum contamination sites. A schematic overview of the suggested research is provided in Figure 4.1.

First, little information is available on the types of chemicals remaining in petroleum contaminated soils after weathering. This information would contribute significantly to the determination of appropriate representative compounds for LCPHs. Secondly, no particular analytical method emerged as the most appropriate for determining BTEX, PAHs, paraffins, naphthenes, and olefins. In fact, each method implies its own definition of TPH. Therefore, the first research effort should focus on identifying the petroleum components remaining in soils after weathering. Various types of soils should be tested as well as various types of petroleum products. The development of analytical methodology most likely will be necessary in order to accomplish this task. A method which is relatively simple, cost effective, and timely that could be translated into the private sector is most desirable.

The majority of available toxicity data on the LCPHs involved the inhalation route of exposure. Extrapolation across dose routes incorporates uncertainties in risk factor development. Toxicity data from oral exposure studies is imperative for oral risk factor development. Acute oral toxicity studies should be conducted on the n-alkanes from C_5 through C_{20} to determine their relative toxicity. Based on this information, on data from the literature concerning their relative toxicity, and on the results of the weathered soil analysis, representative compounds should be chosen. Then, 90-day, subchronic oral exposure studies should be conducted on the chosen representative compounds. Subchronic oral toxicity studies can be conducted also using neat petroleum

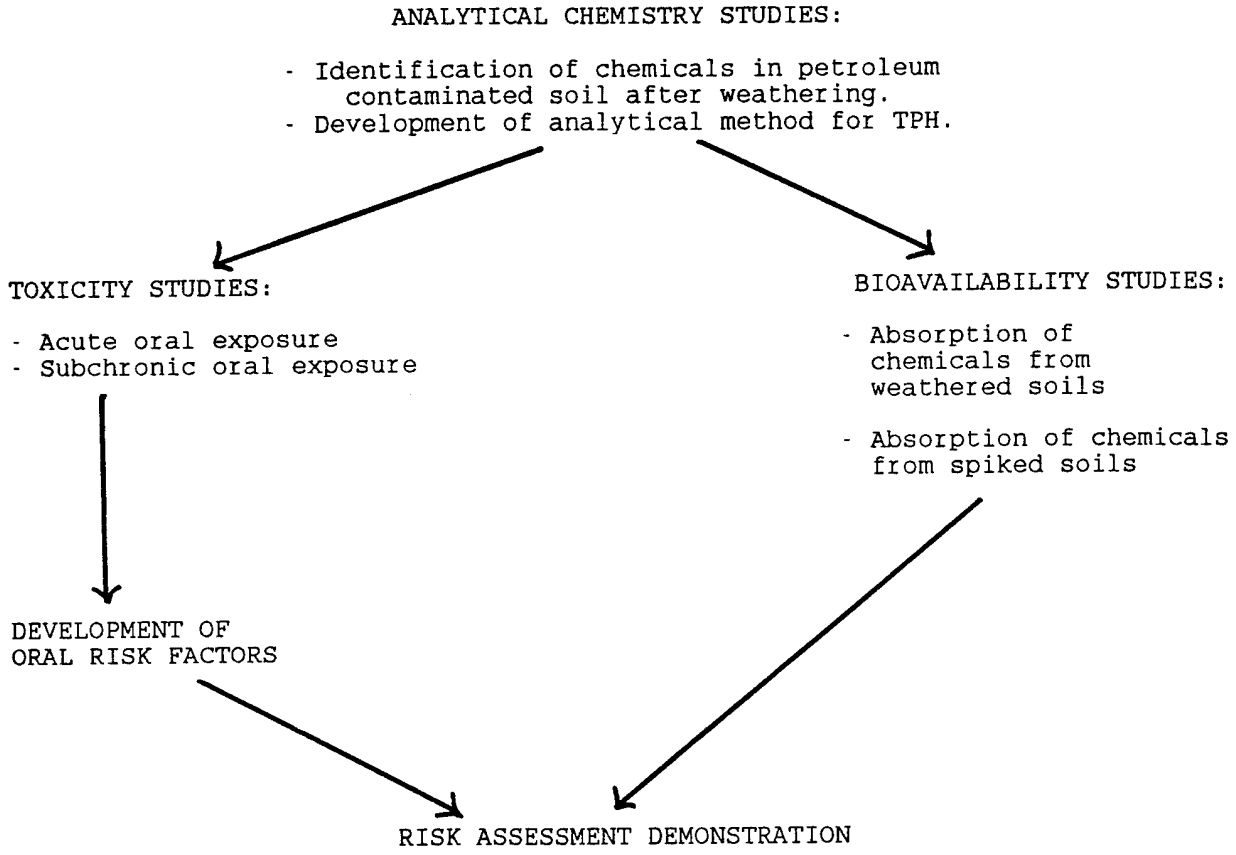
products. Oral risk factors can be derived based on the data from the subchronic studies. The risk factors for neat products would be used most appropriately at fresh spill sites; even then, the assessment of risk from BTEX and PAHs probably would be required also. Therefore, focusing research on the representative compounds would be most cost effective because their risk factors could be used at both fresh and weathered sites.

A factor for the absorption of chemical into the body across the gastrointestinal (GI) tract is incorporated into the exposure estimation process. This factor is assumed to be 1 or 100% meaning that the entire dose is absorbed by the body. However, this may be a gross overestimation, particularly when the chemical is adsorbed onto soils. Therefore, studies on the GI absorption of petroleum chemicals in weathered soils and of representative alkanes spiked onto soils should be conducted.

The chemical analysis, the oral risk factors developed for the representative compounds and their factors for absorption from soil can be incorporated into a site-specific risk assessment for one of the four demonstration sites. Site-specific, risk-based cleanup levels can be determined. Then, a comparison of the cost of remediation based on risk and based on regulatory guidelines can be performed.

The suggested research focuses on the oral route of exposure which is most appropriate for a residential exposure scenario. However, a worker exposure scenario would be more appropriate for sites that are not closed. The dermal route of exposure may be more reasonable for the worker scenario. Additional future research should be directed at developing risk factors for dermal exposure. Dermal toxicity studies of both neat products and representative compounds would be required to determine the necessary risk factors.

FIGURE 4.1 OVERVIEW OF FUTURE RESEARCH



5.0 CONCLUSIONS

A risk-based approach to cleanup of petroleum contamination may prove to be more cost effective and scientifically rational than cleanup based on arbitrary regulatory TPH standards. This approach may be particularly beneficial at weathered sites where BTEX are virtually gone and TPH levels in soil exceed regulatory standards because of paraffins and naphthenes. In addition, sites contaminated with jet fuels that do not contain BTEX, such as JP-7 and JP-8, also may be appropriately remediated with a risk-based approach.

In order to develop risk-based cleanup levels, risk factors must be available for the recalcitrant petroleum components in soil. The risk factors developed herein for LCPHs can be incorporated into an overall risk assessment methodology, such as that suggested by Massachusetts. These risk factors can serve as representatives or surrogates for other paraffinic or naphthenic compounds in the risk assessment process. However, several of these oral risk factors were derived from inhalation exposure studies which introduces uncertainties. Oral exposure studies should be conducted on the representative compounds or surrogate mixtures in an effort to develop more reliable risk factors for LCPHs.

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