INTERIM FINAL PETROLEUM REPORT: DEVELOPMENT OF HEALTH-BASED ALTERNATIVE TO THE TOTAL PETROLEUM HYDROCARBON (TPH) PARAMETER

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EXECUTIVE SUMMARY

This document describes an alternative approach to the analysis and interpretation of the "TPH" parameter used at oil contaminated waste sites. The alternative can be used to perform site-specific risk assessments or to develop health-based cleanup standards for petroleum hydrocarbons. Rather than quantifying the entire range of petroleum hydrocarbons as one mass, a technique is used which divides the broad chemical classes of petroleum hydrocarbons (alkanes, cycloalkanes, alkenes and aromatics) into subgroups of compounds based on numbers of carbon atoms in the compounds in each subgroup and translates the masses of compounds in each specific segment into discrete estimates of health risk for specified exposure scenarios.

Oil products are complex mixtures of hundreds of chemicals, with each compound having its own toxicity characteristics. There are many difficulties associated with assessing the health effects of such complex mixtures with regard to hazardous waste site remediation. For many sites the identity of the fuel product spilled is unknown. While health information is available on the toxicities of pure products, once a petroleum product is released to the environment, changes in composition occur as a result of weathering. These compositional changes may result in changes in the toxicities of the products.

One approach for assessing the toxicity of oil products is to use toxicity information from studies conducted on the whole product. A second approach is to identify and quantify all component chemicals and then consider their toxicities. A third approach is to use some estimate of total petroleum hydrocarbons (TPH) from component chemical groups and toxicity measures specific to the chemical fractions analyzed in the TPH measure. TPH is a loosely defined parameter which can be quantified using a number of different analyses. It should be an estimate of the total concentration of petroleum hydrocarbons in a sample. Depending on the analytical method used to quantify TPH, the TPH concentration may represent the sum of concentrations of a limited number of compounds (for instance benzene, toluene, ethylbenzene and xylenes), groups of compounds (e.g. primarily aliphatics), or the entire range of petroleum hydrocarbons. Shortcomings of current approaches include incomplete accounting for all compounds present and their potential toxicities and failure to account for the effects of differential weathering of some compounds in the environment.

The health hazard evaluation process described in this document consists of the identification of a "reference compound" for each range of compounds, usually chosen because its toxicity is relatively well characterized. For each reference compound, a U. S. Environmental Protection Agency (USEPA) published oral reference dose value (RfD) or cancer slope factor (SF) is identified or, for those "reference compounds" without USEPA published values, an oral dose-response value has been developed from available toxicity information. Inhalation dose-response values are not presented in this document, but will be developed in a later document.

The mass of petroleum hydrocarbons in each segment of a chromatogram is quantified and converted to a medium-specific concentration which is then entered into standard intake equations (such as ingestion of soil by a child) to arrive at an intake of petroleum hydrocarbon per kilogram body weight per day (a "dose"). This dose is then used with the toxicity value identified for the particular segment of the chromatogram to derive an excess lifetime cancer risk or hazard quotient. Cancer risks or hazard quotients are summed across the hydrocarbon fractions to arrive at a total hazard index or cancer risk for the exposure.

Compounds which have been adequately evaluated are used as representative "reference" compounds. The reference compounds were also used to derive alternate RfDs for structurally similar compounds. The alkanes and cycloalkanes are divided into groups based on number of carbons and known structure-activity relationships. These classifications are used to develop alternate RfD values when information on individual chemicals is not available.

A USEPA RfD is available for only one alkane, n-hexane. No RfDs are available for other alkanes, nor for any cycloalkane or alkene. For this assessment, alkanes and cycloalkanes are treated similarly because of the limited information on toxic effects associated with exposure to the cycloalkanes, and the fact that available literature indicates similar toxic effects for alkanes and cycloalkanes. Compounds in the C1-C4 category are not considered further because of their high volatility. This volatility makes chronic exposure at sites unlikely. With limited information available on other toxic endpoints, relative potency of neurotoxicity was used to derive alternate RfDs for the smaller alkanes. The toxic effects associated with the larger alkanes (C19-C32) include irritation and functional changes at the cellular level. Cycloalkanes are expected to exhibit similar effects as the comparable alkane. Reference compounds identified for each group are as follows:

- n-hexane - for C5 through C8
- n-nonane - for C9 through C18
- eicosane - for C19 through C32

Compounds with five through eight carbons are grouped with n-hexane and assigned the same RfD of 0.06 mg/kg/day. C9 through C18 hydrocarbons are grouped with n-nonane and assigned an alternate RfD of 0.6 mg/kg/day. The compounds in the third group are assigned an RfD of 6 mg/kg/d.

In the analytical scheme accompanying this methodology, aromatics and alkenes will be separated from the alkanes and cycloalkanes. Alkenes are therefore evaluated similarly to aromatics for methodological convenience: a decision which can also be supported by toxicological considerations.

The USEPA has published chronic oral RfDs for several of the lower molecular weight aromatic hydrocarbons covering a range of carbon numbers from C9 to C15. These values are all quite similar, ranging (with the exception of anthracene), from 0.03 to 0.06 mg/kg/day. The RfD for anthracene is 0.3 mg/kg/day. Because of the similarities in the RfD level, in metabolism, and effects, one alternate RfD is assigned for the entire range of C9 through C32 aromatics. The alternate RfD is the lowest of those developed by USEPA for the noncarcinogenic aromatics: 0.03 mg/kg/day for pyrene. A compound-specific approach is being used for BTEX and carcinogenic PAHs. Cancer slope factors are available for two carcinogenic aromatic petroleum compounds: benzene, a single-ringed compound; and BaP, a large five-ringed polycyclic aromatic hydrocarbon (PAH). The assessment methodology for carcinogenic PAHs is currently under development in DEP's Office of Research and Standards.

The analytical approach which will provide the information required for conducting the health risk assessments uses two high resolution capillary gas chromatography methods: one for analyzing volatile petroleum hydrocarbons (VPH) and one for analyzing extractable petroleum hydrocarbons (EPH). Gasoline-range volatile hydrocarbons in soil and water are extracted and captured with a purge and trap concentrating system and analyzed by a gas chromatograph equipped with flame ionization (FID) and photoionization (PID) detectors in series. Benzene, toluene, ethylbenzene, and total xylenes are identified and quantitated as individual compounds. The remaining portions of the chromatograms for alkanes/cycloalkanes and aromatics/alkenes are then divided into ranges based upon the number of carbon atoms in each compound and the areas under each curve in each range are determined to provide masses of hydrocarbons in each range for the sample being analyzed.

Extractable petroleum hydrocarbons (EPH) are extracted with methylene chloride and analyzed with a GC equipped with FID and PID detectors in series. The identification and

quantitation for the EPH procedure is similar to the VPH procedure. Polycyclic aromatic hydrocarbons are identified and quantitated individually from the EPH extract. This method can measure extractable hydrocarbons in soil and water corresponding to carbon number ranges of approximately C10 to C40.

In site-specific risk assessments, the approach presented here will be used in conjunction with a compound-specific risk assessment. Aromatic compounds with fewer than nine carbons (such as benzene, toluene, ethylbenzene and xylenes) will be evaluated on a compound-specific basis. These compounds are, therefore, not included in this "alternate RfD" approach. Because of the compound-specific approach employed for aromatics with less than nine carbon atoms, C5 through C8 alkenes are not evaluated in this approach. This approach also does not address additives to fuel products (such as methyl tertiary-butyl ether, ethylene dibromide, etc). The evaluation of additives to fuel products is discussed in other Massachusetts petroleum policy documents.

Implementation guidance for the petroleum evaluation procedure in the context of the Massachusetts Contingency Plan (MCP) for hazardous waste sites is currently being developed. Issues which will be covered by that guidance and the MCP include the relationship of the single number Method 1 "TPH" Standard now in place to the new evaluation process, the exposure component of the risk assessment, conditions under which a whole product toxicity evaluation may be employed, and data format reporting requirements.

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1.0 INTRODUCTION

1.1 BACKGROUND

Contamination of the environment by petroleum hydrocarbons is both widespread and frequent. Crude oils and refined oil products are accidentally released from oil tanker accidents, road transport tanker truck accidents, leaks from storage tanks, and during transfer of these products. Intentional releases also occur. Petroleum products account for a large fraction of the contamination at hazardous waste sites. Complete assessment of the human health and ecological risks posed by these complex mixtures of organic compounds has traditionally been hampered by limitations imposed by the costs and capabilities of analytical instrumentation, by the complexity of the organisms and ecosystems affected and by incomplete information on the toxicology of all the component compounds in these mixtures.

In Massachusetts, the identification, evaluation and remediation of hazardous waste sites is accomplished under a specific state statute referred to as the Massachusetts Oil and Hazardous Material Release Prevention and Response Act (MGL Chapter 21E). The Massachusetts Department of Environmental Protection Bureau of Waste Site Cleanup (BWSC) is charged with implementing this act. Petroleum-only cases make up about half of the state's hazardous waste sites, and another 10 percent have petroleum constituents as well as other contaminants. To more efficiently address this significant category of sites, BWSC is developing a "Policy for the Investigation, Assessment and Remediation of Petroleum Releases", or the "Petroleum Policy".

The Petroleum Policy is an ongoing project to provide guidance for the assessment and cleanup of sites contaminated by petroleum products. Three policies have already been adopted by the BWSC as part of the Petroleum Policy "package":

- Interim Site Investigation Protocol (WSC#-401-91)
- Interim Remediation Waste Management Policy for Petroleum Contaminated Soils (WSC#-94-400)
- Off-gas Treatment of Point-source Remedial Air Emissions (WSC-94-150)

In addition, two draft documents are undergoing review:

- Interim Laboratory Guidance Manual for Petroleum Contaminated Media
- Remedial Action Design Document

A key component of the evaluation of petroleum contaminated sites is the assessment of potential human health risks. These evaluations can be performed either by comparing concentrations of chemicals to guidance values or through a risk assessment. To allow for the most streamlined evaluation of some sites, health-based guidance values have been developed: Reportable Concentrations (RCs) and "Method 1 Standards" (M1Ss). RCs are the concentration of oil or hazardous material (OHM) in soil or groundwater which requires notification to the Department of Environmental Protection (DEP). M1Ss are groundwater and soil standards that have been developed for the Department considering a defined set of exposures and toxicity values.

The performance of a risk assessment on petroleum products and development of RCs and M1Ss requires the use of appropriate toxicity values for petroleum products or component chemicals. These values are not currently available for petroleum products and component chemicals. In addition, some of the analytical techniques that are commonly used at many petroleum sites employ a parameter, Total Petroleum Hydrocarbons (TPH), which is not particularly useful in health risk assessment. This document addresses these shortcomings and describes a method to improve upon the presently used approaches for the evaluation of health hazards posed by complex mixtures of petroleum hydrocarbons.

1.2 PURPOSE

The purpose of this document is to identify an alternative to the TPH parameter which can be used to develop health-based cleanup standards or used in the conduct of site-specific risk assessments. This document also identifies dose-response values to be used with the evaluation methodology.

This approach is applicable to all petroleum products. The most commonly encountered at 21E sites are:

- Gasoline
- No.2 Fuel/Diesel Fuel
- No.6 Fuel
- Jet Fuel
- Kerosene
- Crankcase Oil
- Waste Oil

Waste oil and used crankcase oil present special concerns with regard to their evaluation. The used product is contaminated with combustion products in addition to additives and inorganics present in the fresh product. The potential toxicity of these contaminants is not addressed in this policy, but should be considered in the site evaluation.

Each of these products is a complex mixture of hundreds of chemicals, each with its individual toxic effect. There are many difficulties associated with assessing the health effects of such complex mixtures with regard to site remediation. First, for many sites the identity of the fuel product spilled is unknown. Second, while health information is available on the toxicities of pure products (ATSDR, 1993 a,b,c; IARC, 1989 a,b,c,d; USEPA, 1992a; Millner *et al.*, 1992), once a petroleum product is released to the environment, changes in composition occur as a result of weathering. Weathering involves a number of processes including volatilization, hydrolysis, photolysis, biodegradation, biotransformation, physical breakup and dissolution. The relative importance and rates of each of these processes varies from situation to situation. Therefore, it becomes very difficult to accurately predict the composition of weathered products from knowledge of the composition of fresh products and to equate potential toxicities of the weathered products from knowledge of the toxicities of the fresh products.

The variability in weathering rates of a variety of crude oils and their derivatives is illustrated by data from marine field and laboratory studies shown in Table 1. While data from freshwater and terrestrial systems would be more applicable to the situations addressed most often by hazardous waste site programs, these more readily available data are presented to demonstrate the relative magnitudes and importance of differential weathering of oils in the environment. Losses of from 30-100% of total aromatic compounds present in fresh product can occur over several weeks in marine sediments

TABLE 1
Weathering Rates of Various Oils in Marine Environments

TYPE	GROUP	LOSS	PERIOD	MEDIUM
#5 Fuel Oil (a)	Total Aromatics Naphthalene Phenanthrene Fluorene	58-98% 88-99% 75% 72.5% 86%	2 wks 5 wks 2 wks 2 wks 2 wks	Suspended Sediments
Murban Crude(b)	Aliphatics	50%	2.3-7 wks	Sediments
Prudhoe Bay Crude (c)	Aromatics & Saturates Docosane Naphthalene Phenanthrene	 50-100% 50% 97.9% 99% 97.4%	 290d (coarse) 13-47d (fine) 270d 270d	 Sediments Sediments
Alaskan Crude (d)	Total Aromatics	30%	30d	Sediments
#2 Fuel Oil(e) Bunker C S. Louisiana Crude Kuwait Crude	Naphthalenes	56-94%	96h	Water-soluble Fraction

Source:

(a) Boehm et al., 1982
(b) Page et al., 1983
(c) Anderson et al., 1978
(d) McCain et al., 1978
(e) Rossi et al., 1976

oiled with fresh product. Degradation rates for different petroleum hydrocarbon classes vary, with rates decreasing as molecular mass and degree of molecular branching or substitution increases (Whittle *et al.*, 1982). For instance, naphthalene in water and sediments weathers under natural conditions at rates up to several orders of magnitude greater than does benzo(a)pyrene (Whittle *et al.*, 1982).

One approach for assessing the toxicity of oil products is to use toxicity information from studies conducted on the whole product. Since most knowledge of the toxicity of oil products is based upon fresh products and most of the material at waste sites has undergone varying degrees of differential weathering, the difficulty and imprecision in predicting toxicity or health risks from exposures to those weathered substances using data for fresh substances should be apparent. Those compounds responsible for recorded toxicities of fresh product may no longer be present or are present at reduced concentrations in weathered samples.

A second approach for assessing the potential toxicity of complex mixtures of hydrocarbons is to identify and quantify all component chemicals. This approach produces data which theoretically could be compared to the known toxicity of each compound. The impracticality of this approach stems from its high analytical cost and the absence of toxicity data for many of the component chemicals found in hydrocarbon mixtures.

A third approach to the assessment of petroleum-contaminated sites is the use of the Total Petroleum Hydrocarbon (TPH) parameter. TPH is a loosely defined parameter which can be quantified using a number of different analyses. The TPH parameter is an estimate of the total concentration of petroleum hydrocarbons in a sample. Depending on the analytical method used to quantify TPH, the TPH concentration may represent the sum of concentrations of a limited number of compounds (for instance benzene, toluene, ethylbenzene and xylenes), groups of compounds (e.g. primarily aliphatics), or the entire range of petroleum hydrocarbons from C_4 to C_{32}, and petrogenic as well as phytogenic hydrocarbons. Because the TPH parameter includes a number of compounds of differing toxicities, the health effects associated with exposure to particular concentrations of TPH cannot be determined. However, many states including Massachusetts, have identified clean-up levels based on specific levels of TPH. In most states, these levels are not health-based. The TPH values that are presently listed in the MCP as MCP Method 1 standards are health-based and were calculated using conservative assumptions as to the composition of the spilled fuel products and ceiling values derived based on welfare considerations. Guidance will be provided by BWSC for using the approach presented in this document in the context of the MCP Method 1 standards.

1.3 APPROACH

This document presents an alternative to the TPH parameter. Rather than quantifying the entire range of petroleum hydrocarbons as one mass, an analytical technique is proposed which divides petroleum hydrocarbons into subgroups of compounds based on numbers of carbon atoms in the molecules in each subgroup.

For each subgroup of compounds, a "reference compound" is identified, usually chosen because its toxicity is relatively well characterized. For each reference compound, a USEPA-published oral reference dose value (RfD) or cancer slope factor (SF) is identified or, for those "reference compounds" without USEPA published values, an oral dose-response value is proposed based on available toxicity information. Inhalation dose-response values are not presented in this document, but will be developed in a later document.

The approach described above requires the development of an analytical method that can quantify the mass of petroleum hydrocarbons in each specific segment of a chromatogram for which a reference compound has been identified.

The mass of petroleum hydrocarbons quantified in each segment of a chromatogram is converted to a medium-specific concentration which is then entered into standard intake equations (such as ingestion of soil by a child) to arrive at an intake of petroleum hydrocarbon per kilogram body weight per day. This intake is then combined with the toxicity value identified for the particular segment of the chromatogram to arrive at an excess lifetime cancer risk or hazard quotient. Cancer risks or hazard quotients are summed across the hydrocarbon fractions to arrive at a total hazard index or cancer risk for the exposure. This approach is in keeping with USEPA's "Guidelines for the Health Risk Assessment of Chemical Mixtures" (USEPA, 1986). This guidance states that if sufficient data are not available on the effects of the chemical mixture of concern, the approved approach is to assume additivity of risks of the components of the mixture.

This approach does not address additives to fuel products (such as MTBE, EDB, etc). The contribution of these compounds is subtracted from the total mass of hydrocarbons quantified in the chromatogram. The evaluation of additives to fuel products is discussed in other petroleum policy documents.

This alternative approach should result in a more comprehensive evaluation of sites. Currently, the TPH analysis is used as a screening tool for evaluating a site. In order to conduct a risk assessment, additional samples must be collected and compound-specific analytical data obtained for use in the risk assessment. With this approach, the initial data can be used in the risk assessment. This approach is being applied at this point only to oral and dermal exposures to petroleum hydrocarbons. It is anticipated that in the future, inhalation exposures will also be evaluated.

As an alternative to the approach described in this document, the use of whole product toxicity values to represent the toxicity of the fuel products found at 21E sites was considered. Because 1) at many sites the origin and type of the fuel product is unknown and, 2) at many sites the spills are older and the spilled product has weathered, BWSC believes that use of a whole product toxicity value adds additional uncertainty to the risk estimate and use of a component approach is preferable. The described component approach is applicable to all fuel types and to eithered weathered or unweathered product. Such flexibility is a significant benefit to evaluations at 21E sites.

The remainder of this document is divided into five principal sections. In Section 2, the demonstrated toxic effects of petroleum hydrocarbons are summarized; Section 3 presents the development of toxicity values for components of petroleum products. Chemical analytical requirements and an analytical approach are described in Section 4. In Section 5, application of the approach is described. Uncertainties inherent in the approach are discussed in Section 6, as well as validation exercises that have been performed.

2.0 HAZARD IDENTIFICATION FOR PETROLEUM HYDROCARBONS

2.1 COMPOSITION OF PETROLEUM PRODUCTS

Petroleum products are derived from crude oil. Crude oil and its derivatives are complex mixtures of hundreds of compounds primarily composed of carbon and hydrogen. Compounds containing only carbon and hydrogen atoms are called hydrocarbons and comprise between 50 and 98 percent of most petroleum products. Sulfur, nitrogen and oxygen are important minor constituents of petroleum that are incorporated with carbon and hydrogen to form heterocyclic compounds.

Petroleum hydrocarbons are comprised of four major groups: alkanes, alkenes, cycloalkanes and aromatics. The relative percentages of these components in any one petroleum fraction can vary greatly. Alkanes are also referred to as paraffins or saturated aliphatics. They can be straight chain molecules (normal paraffins) or branched chain alkanes (isoparaffins). As the chains increase in carbon number, the molecular weights and consequently the boiling points of the compounds increase.

Alkenes are also called unsaturated aliphatics or olefins. They are similar to the alkanes except that they contain one or several double bonds and can be either straight chain or branched compounds. The double bond decreases the flexibility of the molecule and increases its molecular reactivity. Alkenes are minor components of petroleum products.

Cycloalkanes are saturated cyclic compounds and are also referred to as cycloparaffins or naphthenes. The cycloalkanes can be very complex depending on the number of rings in the compound and the extent of side chain substitution.

Aromatic compounds are cyclic, unsaturated hydrocarbons characterized by the presence of at least one benzene ring. Benzene is the first and least complex of this very large group of compounds. Additions of aliphatic side chains or other saturated or non-saturated ring structures to the benzene ring result in hundreds of complex aromatic structures. For example, the addition of methyl groups to benzene produces toluene or the three xylene positional isomers. Addition of alkanes to the aromatic ring results in ethylbenzene and other alkylbenzenes. Two attached benzene rings result in naphthalene. Several benzene rings can be attached in many different ways to form the class of compounds known as the polycyclic aromatic hydrocarbons (PAHs). The positions of the aromatic rings relative to

each other are responsible for the vastly different physical properties and different biological activities associated with aromatic compounds.

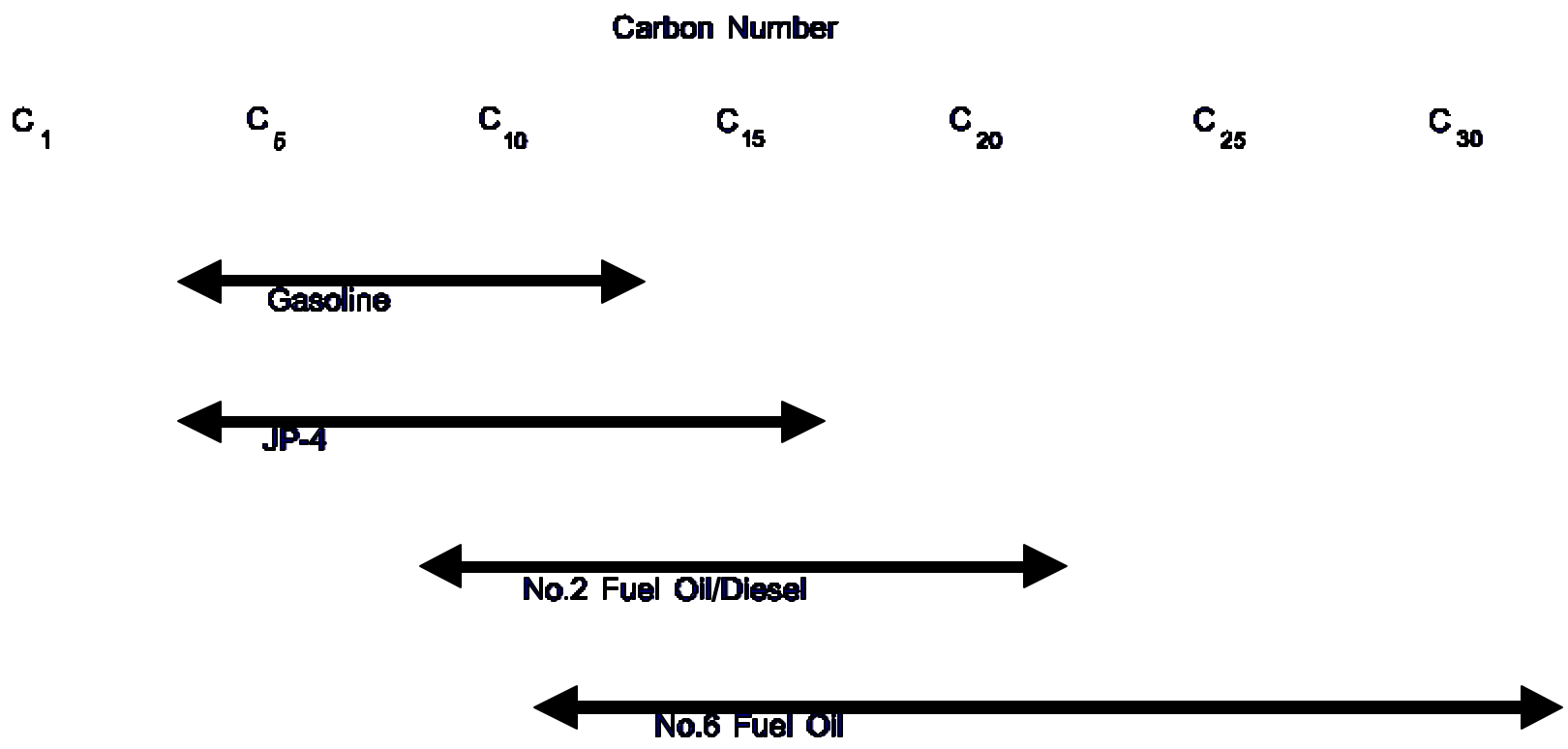
Crude oil is distilled into a series of fractions which are characterized by distillation temperature ranges and vapor pressures. In general, the lighter fractions (with lower boiling points) are gasoline-range hydrocarbons. The intermediate or middle distillate fractions are feedstock for diesel fuel, jet fuels and "light" heating oils. The residual heavier fractions are used as fuels in industrial boilers. Figure 1 presents carbon number ranges for some products. Other refinery processes beyond distillation are also used to achieve required characteristics. For instance, gasolines are blended products which may also contain additives to improve performance. A brief description of the composition of important fuel products follows. Appendix A presents detailed composition data for some products.

- Gasoline - Gasoline is a fuel product blended from several refinery process streams, including any of the various naphtha streams. Hydrocarbons are predominantly in the range of C4 through C12, with a boiling range of 50 to 200°C (IARC, 1989a). The concentration of BTEX compounds (benzene, toluene, ethyl benzene and xylene) in gasoline varies dependent on the feed stock and refinery process, but is in the range of 10-20% of total hydrocarbons. Other aromatics may account for up to another 39% and aliphatics about 49-62%. Gasoline also contains additives to improve performance. These compounds include anti-knock agents, antioxidants, metal deactivators, lead scavengers, anti-rust agents, anti-icing agents, upper cylinder lubricants, detergents, and dyes (IARC, 1989a).

- Fuel Oil No. 2 and Diesel Fuel No. 2 - Fuel oil No. 2 and diesel fuel No. 2 have similar compositions and are both obtained from distilled process streams. Both are less volatile than gasoline and consist of hydrocarbons having carbon numbers in the range of approximately C9 (No. 2 diesel fuel) or C11 (No. 2 fuel oil) through C20. Diesel fuel No. 2 has a boiling range of approximately 163 to 357°C (IARC, 1989c) or 282 to 338°C (CHRIS, 1991). The boiling point of No. 2 fuel oil is similar to that for diesel (CHRIS, 1991). Aliphatic hydrocarbons may account for about 64% of the total hydrocarbon content of No. 2 fuel oil, alkenes for about 1-2% and aromatics for about 35%

Figure 1

Carbon Ranges
For Fuel Products



(ABB-ES, 1990). Small amounts of n-hexane (less than 0.1%), benzene (below 0.02%), toluene, xylenes and ethyl benzene (0.25 to 0.5%) may also be found in these products (IARC, 1989c). Distillate fuels may also contain additives that serve as antioxidants, dispersants and corrosion inhibitors (IARC, 1989c).

- No. 6 Fuel Oil - No. 6 fuel oil is a residual oil, obtained from the residues remaining after distillation or cracking, and blends of these residues with distillates (IARC, 1989c). The specific composition of residual oils is difficult to describe because they are such complex mixtures (ATSDR, 1993b). They have a boiling point of 212 to greater than 588°C (CHRIS, 1991). Depending on the stocks used to create the fuel, the percentage of three- to seven-ring PAHs can range from 6 to 8 % to greater than 20% (IARC, 1989c). Paraffins may make up about 20% of the total hydrocarbons in a No. 6 fuel oil and aromatics about 34%, with benzenes about 2% of the total (Table A-3, Appendix A). Additives to residual fuels are based mostly on oil-soluble compounds of calcium, iron and manganese (IARC, 1989c).

- Kerosene - Kerosene (No. 1 fuel oil) is a straight-run distillate with a boiling range of 193 to 293°C (CHRIS, 1991). It consists of hydrocarbon compounds with carbon numbers ranging from C9 through C16 (IARC, 1989b). The majority (about 80%) of compounds in kerosene are n-alkenes, isoalkenes and cycloalkenes. Aromatics may account for approximately 5-20% of the hydrocarbons, the majority of them being alkylbenzene (Table A-2, Appendix A; ABB-ES, 1990).

- Jet Fuel - Jet fuels are similar in composition to kerosene, although in some fuels (wide-cut fuels) lower boiling streams are added to increase volatile hydrocarbons (IARC, 1989b). These fuels consist of hydrocarbons with carbon numbers generally in the range of C9 through C16 (C4 through C16 for wide-cut fuels). JP-4 fuel may contain up to 80% paraffins, and 20% aromatics (ABB-ES, 1990). BTEX compounds made up about 5% of one JP-4 sample (Air Force 1981 via ATSDR, 1993c). Jet fuels have a boiling range of 150 to 300°C. A variety of additives may also be used.

- Crankcase Oil - Crankcase oils can be either mineral-based or synthetic. The mineral-based oils are most widely used in automotive and other engines and are described here. These oils consist of hydrocarbons with carbon numbers generally in the range of C15 to C50 (IRP, 1991) and a boiling range of 300 to 600°C. While new oil contains only trace levels of PAHs, used oils may contain higher PAH concentrations as well as a variety of other impurities from engine operation (for example, heavy metals and breakdown products) (IRP, 1991).

2.2 TOXIC EFFECTS OF WHOLE PRODUCTS

The following text presents a brief summary of toxicological information on some whole products: gasoline; diesel fuel, middle weight fuel oils and jet propulsion (JP) fuel; and, No. 6 fuel oil. This information is presented not as a comprehensive review of the literature, but to provide insight into the types of effects associated with petroleum products. Toxicity information is divided into threshold and non-threshold (carcinogenic) effects for each product. Table 2 presents provisional oral RfDs and slope factors calculated by USEPA (1992a) for some whole products. Provisional values have not undergone full review by USEPA and are not listed in the USEPA Integrated Risk Information System (IRIS) data base or Health Effects Assessment Summary Tables (HEAST; USEPA, 1993a,b). Since the release of these provisional dose response values, USEPA has received comments on their development. The comments have resulted in the withdrawal of the RfD for marine diesel fuel and the classification of jet fuels as Group C carcinogens. The validity of the extrapolation from inhalation exposure to oral exposure was also questioned. Thus, it is possible that these values may be revised. The dose-response values listed in Table 2 are for pure products. Their applicability to weathered product often encountered at petroleum sites is questionable.

2.2.1 Gasoline

Gasoline is the most studied of the petroleum products and a substantial amount of toxicity data is available. Most data, however, are based on inhalation exposures. A draft toxicological profile has been prepared by ATSDR on automotive gasoline (ATSDR, 1993a) and recently a summary of Health Effects of Gasoline has been published as an Environmental Health Perspectives Supplement (December, 1993).

TABLE 2
ORAL DOSE/RESPONSE VALUES FOR WHOLE PETROLEUM PRODUCTS

CARCINOGENS

COMPOUND	SLOPE FACTOR (mg/kg/day)-1	SOURCE	DATE (1)	STUDY TYPE	WEIGHT OF EVIDENCE	TEST SPECIES	CANCER TYPE
Gasoline	1.70e-03	USEPA	1992	Inhalation	C	Mouse	Liver tumors

NONCARCINOGENS

COMPOUND	CHRONIC RfD mg/kg/day	SOURCE	DATE (1)	STUDY TYPE	CONFIDENCE LEVEL	CRITICAL EFFECT	TEST ANIMAL	UNCERTAINTY AND MODIFYING FACTORS (2)
Gasoline	2.0E-01	USEPA	1992	Inhalation	Low	Lowered Body Weight Gain	Rat	UF = 1000 H,A,D
JP-4	8.0E-02	USEPA	1992	Inhalation	Low	Liver	Mouse	UF = 10,000 H,A,S,L,D
JP-5	2.0E-02	USEPA	1992	Inhalation	Low	Liver	Mouse	UF = 10,000 H,A,S,L,D
Marine Diesel	Withdrawn							

(1) USEPA,1992 = United States Environmental Protection Agency, Master List of Responses for 2QTR 1992.
Superfund Health Risk Technical Support Center Chemical Specific Risk Assessment Issue Papers
Office of Research and Development, ECAO, Cincinnati, OH

(2) H = variation in human sensitivity
A = animal to human extrapolation
S = extrapoloation from subchronic to chronic NOAEL
L = extrapolation from LOAEL to NOAEL
D = study deficiency or incomplete data

2.2.1.1 Threshold Effects. Acute inhalation exposure to gasoline at 500 ppm results in central nervous system effects including headache, dizziness, nausea and drowsiness; at 1000 ppm to 5000 ppm for 15 to 60 minutes, effects such as anesthesia, loss of reflexes, convulsions and delirium may occur; and at greater than 5000 ppm, unconsciousness, coma and death may occur (Anonymous, 1989).

MacFarland *et al*. (1984) conducted an inhalation study which examined mice and rats exposed for two years to wholly vaporized gasoline. This exposure methodology is very different from what would occur at an actual site in that pure product is aerosolized. Thus, both volatile and nonvolatile compounds are inhaled. The investigators identified reduction in body weight gain in both rats and mice (at an exposure level of 2056 ppm), and nephrotoxicity in the male rat. The nephrotoxicity observed has been associated with a protein unique to the male rat and is not applicable to human health risk assessment (USEPA, 1991a).

One developmental study (Litton Bionetics, 1978) located by USEPA (1992a) was inconclusive. Mated female Charles River rats were exposed to 0, 400, or 1600 ppm in air of unleaded gasoline on days 6 through 15 of gestation (6 hrs/day). There was an apparent increase in the incidence of skeletal abnormalities in the high dose group when the fetus was used as the unit of comparison. This increase was not significant when the litter was used as the unit of comparison. USEPA considers the increase to be a possible indication of developmental toxicity.

A provisional oral RfD was developed by the USEPA, Environmental Criteria and Assessment Office (ECAO), Superfund Health Risk Technical Support Center (USEPA, 1992a) based on the MacFarland *et al*. (1984) inhalation study and using route-to-route extrapolation. A NOAEL (based on reduced body weight gain) of 292 ppm (230 mg/m^3) was identified, which was translated into an equivalent oral dose, and, with an uncertainty factor of 1000 (10 for intraspecies variation, 10 for interspecies variation and 10 for deficiencies in the data base) into a provisional RfD of 0.2 mg/kg/day.

2.2.1.2 Non-threshold Effects. Ames Salmonella assays, mouse lymphoma assays, and rat bone marrow cytogenetics tests have been nonpositive in unleaded gasoline studies (Weaver, 1988). However, dose-related increases in unscheduled DNA synthesis were observed in rat hepatocytes (Loury *et al*., 1986).

MacFarland *et al*. (1984) reported that in addition to increased incidence of renal tumors in male rats, there was a renal sarcoma in one intermediate-dose female rat. In mice, there was an increased incidence of hepatocellular tumors (adenomas and carcinomas) in high dose females. Two renal tumors (adenoma and adenocarcinoma) were observed in high-dose female mice.

IARC (1989a) concluded that there is inadequate evidence for carcinogenicity of unleaded gasoline in humans and limited evidence of carcinogenicity in experimental animals, and classifies gasoline as Group 2B, possibly carcinogenic in humans. USEPA had previously classified gasoline to Group B2, a probable human carcinogen (USEPA, 1987). However, that classification predates the USEPA (1991a) conclusion that male rat kidney tumors produced by gasoline are not predictive for humans and, therefore, should not contribute to the weight-of-evidence assessment. USEPA (1992a) assigns gasoline to Group C, possible human carcinogen.

The inhalation unit risk value of 2.1×10^{-3} per ppm is based on the incidence of hepatocellular adenomas/carcinomas in female mice (USEPA, 1987). This was converted by USEPA (1992a) to a provisional oral slope factor of 1.7×10^{-3} $(mg/kg/day)^{-1}$.

2.2.2 Diesel, Light Weight Fuel Oils and Jet Fuel

Diesel fuels, light weight fuel oils and jet fuel are all middle distillate fractions of crude oil. Because of the similarities in the composition of these fuels, they are discussed together. ATSDR has published draft toxicological profiles on fuel oils (ATSDR, 1993b) and jet fuels (ATSDR, 1993c).

2.2.2.1 Threshold Effects. Acute inhalation of jet fuel vapors causes dizziness, headache, nausea and fatigue in exposed workers (IARC, 1989b). Chronic inhalation may also induce neurasthenic symptoms (e.g. fatigue, anxiety, mood changes and memory difficulties) in exposed workers (Knave *et al*., 1978, 1979). Liver effects were seen in rats and mice following exposure via inhalation for 90 days to JP-5 at 150 and 750 mg/m^3 (Gaworski *et al*., 1984) and JP-4 at 500 to 1000 mg/m^3 (MacEwen and Vernot, 1984, 1985). Other observed effects of JP-5 inhalation exposures were slightly reduced red blood cell count, hematocrit, and hemoglobin in both rats and dogs, and mild nasal inflammatory changes and moderately decreased body weight gains in rats (Gaworski *et al*., 1984). Single oral doses of JP-5 at 1 ml/kg produced behavioral effects in rats (Bogo *et al*., 1984).

Female rats inhaling up to 400 ppm of Jet Fuel A (similar to JP-5) on days 6 through 15 of gestation had no embryotoxic, fetotoxic, or teratogenic effect in offspring (IARC, 1989b). An unspecified diesel fuel was also found to be without embryotoxic effects (IARC, 1989b).

Both JP-5 and marine diesel fuel produced lesions in the kidneys of C3Hf/Bd mice treated dermally with undiluted fuel for 60 weeks (Easely *et al.*, 1982). Kidney lesions were not observed in a second dermal study in which mice were treated with up to 500 mg/kg of JP-5 or marine diesel fuel diluted in acetone for 103 weeks (NTP, 1986a). Body weight gain was decreased by week 30 in all dose groups receiving marine diesel fuel and in the high-dose group (500 mg/kg) of mice receiving JP-5. Animals in the high-dose group for both fuels were sacrificed early due to excessive irritation and ulceration at the site of application.

Provisional oral RfDs have been developed by the USEPA, Environmental Criteria and Assessment Office (ECAO), Superfund Health Risk Technical Support Center (USEPA, 1992a) for JP-4 and JP-5 based on subchronic inhalation studies and using route-to-route extrapolation. For JP-4 a LOAELs (based on hepatocellular fatty change and vacuolization in mice) of 813 mg/m^3 was identified (MacEwen and Vernot, 1985; MacNaughton and Uddin, 1984). The LOAEL was translated into an equivalent oral dose, and, with an uncertainty factor of 10,000 (10 for intraspecies and interspecies variation, 10 for use of a LOAEL, 10 for extrapolation to chronic duration and 10 for deficiencies in the data base) into a provisional RfD of 0.08 mg/kg/day.

For JP-5, a LOAEL (based on hepatocellular fatty change and vacuolization in mice) of 150 mg/m^3 was identified (Gaworski *et al.*, 1984), which was translated into an equivalent oral dose, and, with an uncertainty factor of 10,000 (similar to that for JP-4) into a provisional RfD of 0.02 mg/kg/day.

2.2.2.2 Non-threshold Effects. Studies of the genotoxicity of JP-4 (Brusick and Matheson, 1978a) and JP-8 (Brusick and Matheson, 1978b) in Ames Salmonella and Saccharomyces cerevisiae tests, and in TK mouse lymphoma cell assays were generally nonpositive. Benz and Beltz (1980) exposed beagles via inhalation to JP-4, JP-5 or JP-10 and observed no increase in sister chromatid exchanges or micronuclei in peripheral lymphocytes. Studies with diesel fuel and kerosene (similar to JP-5 in composition) were negative for Ames Salmonella tests and mouse lymphoma assays. Positive results were seen with *in vivo* rat bone marrow cytogenetics tests with diesel fuel (Millner *et al.*, 1992). No. 2 fuel oil was positive in mouse lymphoma assays and *in vivo* rat bone marrow cytogenetics tests (Millner *et al.*, 1992).

In a 103 week NTP dermal study with B6C3F1 mice (NTP, 1986), JP-5 failed to produce skin tumors or other neoplasms. Marine diesel fuel produced slight, but significant dose-related increases in the incidence of squamous cell neoplasms of the skin. JP-4 has also been reported to produce skin tumors following chronic dermal treatment of mice (Clark *et al.*, 1988). No. 2 diesel fuel did not produce tumors by itself, but promoted the development of skin tumors initiated by other compounds (Slaga *et al.*, 1986). Tumor promotion and complete carcinogenesis of middle distillates, including jet and diesel fuels, is possibly due to chronic irritation and hyperplasia produced by these chemicals (USEPA, 1992a; McKee *et al.*, 1989).

Millner *et al.* (1992) calculated an oral cancer slope factor for diesel fuel No. 2 based on dermal studies. It should be noted that USEPA has not developed a SF for jet fuels but has assigned them to Group C, possible human carcinogen (M. Dourson, USEPA, ORD, ECAO, letter to M. Hutcheson, MADEP, November 30, 1993).

IARC (1989b,c) has classified marine diesel fuel as possibly carcinogenic to humans (Group 2B), while light diesel fuels and jet fuels were Group 3, not classifiable as to their carcinogenicity in humans.

2.2.3 No. 6 Fuel Oil

Little information is available on the toxicity of the residual fuels, including No. 6 fuel, also called Bunker oil. The following presents a summary of available information.

2.2.3.1 Threshold Effects. In acute studies, heavy No. 6 fuel oil applied dermally to rabbits caused toxicity at 5 g/kg, including severe dermal irritation, weight loss, anorexia, ataxia and lethargy. Necropsy revealed acute toxic hepatitis, gastrointestinal irritation and congested lungs. Other grades of No. 6 fuel produced mild to moderate irritation but no systemic signs of toxicity (Beck *et al.*, 1984). No. 6 fuel oil applied dermally to rabbits at doses ranging from 1 to 10 ml/kg for two consecutive five day periods, separated by a two day rest period, produced 75% mortality at a dose of 2.5 ml/kg. Effects included severe weight loss, anorexia, dermal irritation, hemorrhagic gastroenteritis and liver necrosis.
Little information is available on the effects of No. 6 fuel on reproduction and developmental toxicity. Bunker fuel was reported to reduce duck egg hatchability at unspecified dosages (Szaro, 1979). However IARC (1989d) notes that the avian model is a

highly sensitive model for embryotoxic effects and results should be interpreted with caution.

2.2.3.2 Non-threshold Effects. Bunker fuel was not mutagenic in Salmonella assays (Vandermeulen *et al.*, 1985; Farrow *et al.*, 1983). It did not induce forward mutation in Chlamydomonas reinhardtii (Vandermeulen and Lee, 1986). Neither did it induce sister chromatid exchange in cultured Chinese hamster ovary cells nor mutations in cultured mouse L5178Y TK +/- lymphoma cells (Farrow *et al.*, 1983). B-class residual oil (containing many PAHs and nitrogen-containing chemicals such as aza-arenes) induced an increase in the frequency of chromosomal aberrations in cultured Chinese hamster lung cells at a concentration of 2.0 mg/ml in the presence of an exogenous metabolic system from rat liver (Matsuoka *et al.*, 1982).

In a study of unspecified duration described by IARC (1989d), C3H mice received 20 or 50 mg of dermally applied cracked bunker fuel or a West Texas uncracked residue (Bingham *et al.*, 1980). Cracked bunker fuel produced malignant and benign skin tumors at both dose levels. The addition of various amounts of cracked residue resulted in an increase in tumor frequency and a decrease in latency.

IARC (1989d) concludes that there is sufficient evidence of carcinogenicity of heavy fuel oils in experimental animals and classifies residual (heavy) fuel oils as Group 2B, possibly carcinogenic to humans. USEPA has not assigned No. 6 fuel oils to a weight-of-evidence class.

2.3 TOXIC EFFECTS OF SELECTED PETROLEUM COMPONENT COMPOUNDS

In the following section, brief summaries of toxic effects of compounds for which USEPA has developed dose-response values are presented. Summaries of toxic effects of other petroleum compounds are described in Section 2.3.2.

2.3.1 Compounds with USEPA Approved Dose-Response Values

The toxicities of specific petroleum-derived hydrocarbons (specifically those for which USEPA has developed dose-response values, listed in Table 3) have been described in detail. The following summaries are not intended as comprehensive, in-depth reviews, but rather to present a general overview of effects. More detailed summaries of health effects for these compounds can be found in their respective Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profiles.

2.3.1.1 Threshold Effects

ALIPHATIC COMPOUNDS

n-Hexane

Through epidemiological studies on n-hexane-exposed workers, it has been observed that sensorimotor polyneuropathy is the main toxic effect of long-term exposure. Other effects noted include cranial neuropathy, blurred vision, and abnormal color vision. The onset of symptoms may be delayed for several months to a year after the beginning of exposure. Affected individuals may recover completely, but in severe cases may retain some sensorimotor deficits. The best conducted study with respect to establishing actual exposure levels is by Sanagai *et al.* (1980). They conducted a study of factory workers exposed to n-hexane vapors. The average exposure duration was 6.2 years. There were no neurological abnormalities noted. Neurophysiological tests, however, showed significant differences between exposed workers and a control group in motor nerve conduction velocities and residual latency of motor nerve conduction. These observed differences are consistent with n-hexane-induced peripheral neuropathy observed in other studies on both humans and animals. The Sanagai *et al.* study supports the designation of 58 ppm as an inhalation Lowest Observed Adverse Effect Level (LOAEL).

There are numerous animal studies which document n-hexane's neuropathic effects. The characteristic pathological sign of n-hexane-produced degeneration is paranodal thickening of peripheral nerves and giant swellings of axons in the CNS. IRIS (1994) lists an inhalation No Observed Adverse Effect Level (NOAEL) of 500 ppm for n-hexane. The study referenced (Dunnick *et al.*, 1989) examined neuropathological, respiratory and nasal turbinate abnormalities in mice exposed to levels ranging from 500-10,000 ppm. The 500 ppm exposure group is more appropriately considered a LOAEL based on mild lesions of the nasal turbinates of mice (Dunnick *et al.*, 1989).

TABLE 3
ORAL DOSE/RESPONSE VALUES FOR PETROLEUM HYDROCARBONS

CARCINOGENS

COMPOUND	SLOPE FACTOR $(mg/kg/day)^{-1}$	SOURCE	DATE (1)	STUDY TYPE	WEIGHT OF EVIDENCE	TEST SPECIES	CANCER TYPE
Benzene	2.90e-02	IRIS	3/94	Occup.	A	Human	Leukemia
Benzo(a)pyrene	7.30e+00	IRIS	3/94	Diet	B2	Mouse	Forestomach

Occup. = occupational

NONCARCINOGENS

COMPOUND	CHRONIC RfD mg/kg/day	SOURCE	DATE (1)	STUDY TYPE	CONFIDENCE LEVEL	CRITICAL EFFECT	TEST ANIMAL	UNCERTAINTY AND MODIFYING FACTORS
Acenaphthene	6.00e-02	IRIS	3/94	Gavage	Low	Hepatotoxicity	Mouse	UF = 3000 H,A,S,D
Anthracene	3.00e-01	IRIS	3/94	Gavage	Low	No observed effects	Mouse	UF = 3000 H,A,S,D
Benzene	5.00e-03	MADEP	3/94	Inhalation	NS	Hematologic	Rat	UF = 1000 H,A,S
1,1-Biphenyl	5.00e-02	IRIS	3/94	Diet	Medium	Kidney damage	Rat	UF = 100 H,A; MF = 10
Cumene	4.00e-02	IRIS	3/94	Gavage	Low	Incr. avge. kidney wt.	Rat	UF = 3000 H,A,S,D
Ethylbenzene	1.00e-01	IRIS	3/94	Gavage	Low	Liver & kidney toxicity	Rat	UF = 1000 H,A,S
Fluoranthene	4.00e-02	IRIS	3/94	Gavage	Low	Nephropathy, increased liver weights	Mouse	UF = 3000 H,A,S,D
Fluorene	4.00e-02	IRIS	3/94	Gavage	Low	Decreased RBC	Mouse	UF = 3000 H,A,S,D
Hexane, N-	6.00e-02	HEAST	3/94	Gavage		Nervous system neuropathy, testis atrophy, decreased wt. gain,	Rat	UF = 10000
Naphthalene	4.00e-02	ECAO	3/94	Gavage		Decreased weight	Rat	UF = 1000
Pyrene	3.00e-02	IRIS	3/94	Gavage	Low	Kidney effects	Mouse	UF = 3000 H,A,S,D
Toluene	2.00e-01	IRIS	3/94	Gavage	Medium	Changes in liver and kidney weights	Rat	UF = 1000 H,A,S,D
Xylene(s)	2.00e+00	IRIS	3/94	Gavage	Medium	Hyperactivity, decr. body wt., increased mortality (males)	Rat	UF = 100 H,A

(1) Date last verified
IRIS = Integrated Risk Information System.
HEAST = Health Effects Assessment Summary Tables. Annual Update (1993a) and Supplement No. 1 (1993b).
MADEP = Massachusetts Department of Environmental Protection, Documentation for the Risk Assessment Shortform, 1992
ECAO = USEPA at ECAO Cincinnati, Superfund Health Risk Technical Support Center.
NS = Not specified
H = variation in human sensitivity
A = animal to human extrapolation
S = extrapoloation from subchronic to chronic NOAEL
D = study deficiency or incomplete data

* Under review

Neuropathological effects were not examined in the 500 ppm exposure group. Therefore, it is impossible to rule out the presence of mild neuropathological effects paralleling the detected nasal lesions, since these two effects occur with increasing frequency and severity as higher exposure levels. A study by Cavender *et al.* (1984) in rats demonstrated that n-hexane-induced neuropathies appear sooner with continuous exposure rather than intermittent exposure. Several animal studies have shown no teratologic effects from n-hexane exposure. n-Hexane's oral RfD is based on a 90 day gavage study with rats (Krasavage *et al.*, 1980), in which neuropathy was the main toxic effect observed, along with atrophy of the testis and decreased weight gain. In this study, a LOAEL of 570 mg/kg/day was identified.

AROMATIC COMPOUNDS

Acenaphthene

Adverse effects on the lungs, glands, and blood were observed in rats after administration of 12 mg/m^3 acenaphthene aerosol for a duration of five months (USEPA, 1981). Acenaphthene's RfD is based on hepatotoxic effects in mice following oral exposure. Both mutagenicity and carcinogenicity tests for acenaphthene were negative.

Anthracene

Anthracene is a skin irritant and allergen. Occupational exposure in humans has resulted in skin disorders (Clement, 1985). Exposure to anthracene in humans has been associated with gastrointestinal and hemopoietic toxicity; however the usefulness of this information is limited by confounding factors. A study in which anthracene was administered to mice by gavage for 90 days found no effects at doses up to 1000 mg/kg/day (USEPA, 1989a). The RfD for anthracene is based on a gavage study in mice in which no toxic effects were observed at the administered doses. The majority of mutagenicity tests for anthracene have been negative. The USEPA considers carcinogenicity data on anthracene to be inadequate.

Benzene

Most toxicity information for benzene exposure is a result of inhalation exposures of both animals and humans, although some oral studies in animals are reported. Effects of acute inhalation exposure to high concentrations of benzene (300 to 3,000 ppm) include drowsiness, dizziness, headache, vertigo, tremor, delirium, and loss of consciousness.

Exposure to higher concentrations (20,000 ppm) results in death due to asphyxiation, respiratory arrest, central nervous system depression or cardiac collapse (ATSDR, 1993d).

Inhalation exposure to benzene for several months to several years results in a reduction in the number of all three major types of blood cells (erythrocytes, thrombocytes and leukocytes). Continued exposure may also result in aplastic anemia which may develop into leukemia (ATSDR, 1993d). Gavage doses of 25 mg/kg for two years resulted in leukopenia in both rats and mice (NTP, 1986b).

Both animal and human studies indicate that benzene damages both humoral and cellular immunity. Exposure to benzene at 10 ppm and above for 6 days reduced the ability of bone marrow cells to produce mature B-lymphocytes in C57BL/6 mice (Rozen *et al.*, 1984). Blastogenesis of B- and T-lymphocytes was depressed at 10 ppm and above. Continued exposure for 6 and 23 weeks at 300 ppm showed continued decreases in the number of mature B- and T-lymphocytes produced in the bone marrow, spleen and thymus (Rozen and Snyder, 1985).

Chronic occupational exposure to benzene (possibly in combination with other solvents) results in neurological effects. Exposure to 210 ppm or higher induces effects on the nervous system involving peripheral nerves and/or spinal cord (ATSDR, 1993d).

The available human data on the developmental effects of benzene after inhalation exposure are inconclusive (ATSDR, 1993d). Benzene crosses the human placenta and is present in the cord blood in amounts equal to or greater than those in maternal blood (Dowty *et al.*, 1976). No animal inhalation studies indicate that benzene is teratogenic even at levels that induce maternal and fetal toxicity (ATSDR, 1993d). Fetotoxicity was evidenced by decreased body weight and by increased skeletal variants. Alterations in hematopoiesis have also been observed in the fetuses and offspring of pregnant mice exposed to low levels (20 ppm) of benzene (Keller and Snyder, 1986; 1988).

Studies suggest that occupational exposure to benzene may impair fertility in women, but are inconclusive because of inadequacies in the study design (ATSDR, 1993d). Rabbits exposed to 313 ppm of benzene for 13 days during gestation exhibited a decrease in weight gain and a loss in the number of fetuses, while fetuses had minor internal anomalies and decreased body weight (Ungvary and Tatrai, 1985). Male and female CD-1 mice exposed intermittently to benzene vapor (300 ppm) for 13 weeks evidenced histopathological changes in ovaries and testes (Ward *et al.*, 1985).

The chronic oral reference dose developed by MADEP is based on a study by Deichman (1963) who exposed rats to benzene via inhalation. Hematological effects were noted, including leukopenia. A NOAEL was observed at 31 ppm and is the basis for the oral RfD.

Biphenyl

Chronic exposure to biphenyl is characterized by central nervous system effects, fatigue, headache, tremor, insomnia and sensory impairment, accompanied by clinical findings of cardiac and hepatic impairment, irregularities of the peripheral and central nervous system, and possibly some brain lesions (Sandmeyer, 1981). Rats fed levels of biphenyl 0.5% or greater exhibited kidney damage, reduced hemoglobin levels, decreased food intake and decreased longevity (Ambrose *et al.*, 1960). An unpublished study (SRI, 1960, cited in Ambrose *et al.*, 1960) reported a NOAEL of 0.1% biphenyl in the diet, both in a subchronic rat feeding study and a three generation rat reproduction study. The oral RfD is based on the NOAEL of 0.1% in the diet.

Cumene (Isopropylbenzene)

Cumene is a potent narcotic and is also a primary skin and eye irritant. It is absorbed through the intact skin more rapidly than toluene, xylene or ethylbenzene. In some short term high dose experiments, animals exhibited damage to the spleen and fatty changes to the liver, but no renal or pulmonary irritancy (Sandmeyer, 1981). Rats administered cumene by gavage over a 194 day period showed no effects at 154 mg/kg/day (Wolf *et al.*, 1956). At 462 mg/kg/day, a slight but significant increase in kidney weight occurred. The oral RfD is based on the NOAEL of 154 mg/kg/day.

Ethylbenzene

Humans exposed to low levels of ethylbenzene through inhalation experience eye and throat irritation. Exposure to higher concentrations can cause effects such as central nervous system (CNS) depression, dizziness, and more severe mucous membrane irritation. There is no available information that indicates ethylbenzene produces toxicity in other organs of humans after short-term or prolonged exposure (ATSDR, 1990). Animal studies indicate primary symptoms from acute exposures are neurological and respiratory depression. Other studies indicate that target organs of ethylbenzene toxicity are the liver, kidney, and hemopoietic system (ATSDR, 1990). One study demonstrated that inhalation exposure of pregnant rats produced fetotoxic effects at levels that also induced maternal toxicity (Andrew *et al.*, 1981).

The RfD for ethylbenzene is based on hepatotoxic and nephrotoxic effects in rats via dietary exposure. Genotoxicity tests on ethylbenzene are generally negative. It has, however, caused mutagenic effects in mouse lymphoma cells and a significant increase in sister chromatid exchange in human lymphocytes. These studies indicate that ethylbenzene may cause an increased potential for genotoxicity in humans (ATSDR, 1990). A chronic bioassay for carcinogenicity in animals produced inconclusive results via oral exposure (Maltoni *et al.*, 1985).

Fluoranthene

Information on fluoranthene's effects on humans, separate from other PAHs, is limited. Effects of PAH mixtures include skin lesions and non-cancer lung diseases such as bronchitis. A 13 week subchronic study in which mice were gavaged with up to 500 mg/kg/day of fluoranthene produced clinical effects, nephropathy, increased liver weights, and hematological alterations (USEPA, 1988). Fluoranthene's RfD is based on a gavage study in mice in which nephrotoxic and hematological effects were observed. Chronic dermal application to the backs of mice did not induce skin tumors. There is some evidence that fluoranthene is genotoxic; however, it is not a complete carcinogen (ATSDR, 1993b).

Fluorene

Information on fluorene's toxicity is limited to the effects of PAH mixtures. Effects attributed to exposure include skin lesions and non-cancer lung diseases such as bronchitis. One animal study indicated that mice exposed by gavage with up to 500 mg/kg/day showed hypoactivity, decreases in red blood cell count, packed cell volume, and hemoglobin, and increases in liver, spleen, and kidney weights (USEPA, 1989b). The RfD for fluorene is based on a gavage study in mice in which hematological effects were observed. Limited studies have provided no evidence that fluorene is genotoxic. Fluorene is not reported to be a complete skin carcinogen (ATSDR, 1993e) and was inactive as a tumor initiator (LaVoie, 1980).

Naphthalene

Humans exposed to naphthalene through inhalation have experienced vomiting, abdominal pain, and anemia. The primary site of toxicity from inhalation exposure to naphthalene is the erythrocyte, resulting in hemolytic anemia. Rats and mice appear to be relatively resistant to red cell hemolysis compared to humans and dogs (ATSDR, 1993f). Exposures of humans through all routes have caused jaundice and liver enlargement (ATSDR, 1993f).

Oral doses result in effects on the kidney in humans. Renal effects have not been reported in animal studies (ATSDR, 1993f). The provisional RfD for naphthalene (USEPA, 1994) is based on decreased body weight in rats as a result of gavage exposure. There are no identified studies of genotoxic effects. There is no human epidemiological evidence for naphthalene exposure being correlated with increased cancer rates and inconclusive evidence of carcinogenicity in rats or mice.

Pyrene

Pyrene is reported to be a human skin irritant (Sax, 1984). Subchronic dietary exposure of rats to pyrene resulted in enlarged and fatty appearing livers (ATSDR, 1993e). A 13 week gavage study in which mice were exposed to 125 mg/kg/day of pyrene reported nephropathy and decreased kidney weights (USEPA, 1989b). The RfD for pyrene is based on a gavage study in mice in which nephrotoxic effects were noted. The majority of genotoxic tests of pyrene are negative. Bioassays involving mouse skin painting indicate that pyrene is neither a complete skin carcinogen nor an initiator.

Toluene

Exposure of humans to toluene primarily results in effects to the CNS. Acute effects include CNS depression, neurological dysfunction, and narcosis. Chronic exposures have resulted in permanent effects such as ataxia, tremors, and impaired speech, vision, and hearing. Toluene vapors cause respiratory tract irritation and chronic exposures may produce cardiac arrhythmias (Anderson *et al.*, 1982). Toluene exposure in animals produces CNS damage such as impaired motor abilities, narcosis, tremors, and changes in EEG activity. Reported hepatic effects are increases in liver weights and minor ultrastructural changes. In utero exposures have resulted in skeletal anomalies, retarded skeletal growth, and decreased fetal weights (Ungvary, 1985). The RfD for toluene is based on a gavage study in rats in which liver and kidney weight changes were observed. *In vivo* and *in vitro* studies suggest that toluene is not genotoxic.

Xylene(s)

Human exposures by any route to xylenes result in primarily CNS effects that may include headaches, nausea, mental confusion, dizziness, tremors, unconsciousness, and coma, depending on dose and length of exposure. Inhalation exposures cause respiratory tract irritation and pulmonary edema. In animals, the CNS is also the primary target of xylene exposure. Limited evidence of xylene's effects on animals include cardiac arrhythmias,

atrial fibrillation, hepatic enzyme induction and ultrastructural alterations, renal atrophy, and tubular alterations in the kidney. Animal studies suggest that xylenes may produce developmental effects including increased fetal death, decreased fetal weight, delayed skeletal development and gross anomalies (Marks *et al.*, 1982; Ungvary *et al.*, 1980). Xylene's RfD is based on hyperactivity in rats via gavage exposure. Genotoxicity tests for xylene have been negative. There is no evidence of carcinogenicity in either humans or laboratory animals (ATSDR, 1993f).

2.3.1.2 Non-threshold Effects

Benzene

In vivo and *in vitro* data from both humans and animals indicate that benzene and/or its metabolites are genotoxic (ATSDR, 1993d). Both gavage and inhalation exposures of rodents to benzene have resulted in development of neoplasia. Epidemiological and case studies correlate benzene exposure with leukemia (ATSDR, 1993d). USEPA has classified benzene as Group A, human carcinogen via oral and inhalation routes (IRIS, 1994). This classification is based on several studies indicating increased incidence of nonlymphocytic leukemia from occupational exposure, as well as increased incidence of neoplasia in rats and mice. Both the oral and inhalation slope factors derived by USEPA are based on pooled data from a number of occupational exposure studies which found an increased incidence of nonlymphocytic leukemia resulting from inhalation exposures (IRIS, 1994).

Benzo(a)pyrene

Information on human benzo(a)pyrene (BaP) toxicity is limited to the effects of PAH mixtures. Epidemiologic studies have shown increased mortality due to lung cancer in humans exposed to coke-oven emissions, roofing tar emissions and cigarette smoke, which all contain mixtures of BaP as well as other carcinogenic and noncarcinogenic PAHs (ATSDR, 1993e). BaP acts as a carcinogen in numerous animal species via many routes of exposure (ATSDR, 1993e). Organs in which tumors have been produced include the forestomach, pulmonary system, and alimentary tract. Single oral doses of 200 and 100 mg/kg produced mammary tumors in 88 percent and 77 percent of female rats, respectively (Huggins and Yang, 1962; McCormick, 1981). BaP has been classified by USEPA as a B2, probable human carcinogen. BaP is a potent genotoxic agent when metabolically activated in both *in vitro* and *in vivo* tests. The oral slope factor for BaP is the geometric mean of slope factors based on two different studies (Neal and Rigdon, 1967; Brune *et al.*, 1981) using both rats and mice.

2.3.2 Other Petroleum Hydrocarbons

Chronic toxicity test results are the preferred starting point for development of dose-response values for evaluating chronic exposures. If these data are not available, shorter duration test data could be used to estimate chronic toxicity. Toxicity information based on the oral route of exposure, is also preferred for estimating oral RfDs. Likewise, for inhalation RfDs, toxicity information based on inhalation exposures is preferred.

2.3.2.1 Availability of Chronic Toxicity Information. Available toxicity data have been used by the USEPA to develop dose-response values for a number of aromatic compounds and for one aliphatic compound, n-hexane. These values were presented in Table 3. A number of different approaches were taken in this document to identify and obtain chronic toxicity information for the many other petroleum hydrocarbons for which no dose-response values are available. The composition of petroleum products has been summarized (ABB-ES, 1990). From this summary, a list of the major component petroleum hydrocarbons found in gasoline, Nos. 2, 4, and 6 fuel oil, jet fuel and kerosene was prepared. Computer searches of the National Technical Information Service (NTIS), and the National Library of Medicine's (NLM's) TOXLINE and TOXLIT data bases (1981 to 1991) were performed to identify available toxicity literature on petroleum hydrocarbons and fuel products. A search of the NLM MEDLINE database for 1989 - 1993 was also conducted for the following petroleum hydrocarbons: n-alkanes, octane, nonane, decane, undecane, dodecane, tridecane, tetradecane, nonadecane, isoalkanes, cycloalkanes, and alkenes.

In addition, a number of American petroleum companies and the American Petroleum Institute (API) were contacted to find out if any compound-specific (as opposed to product-specific) test data were available. Petroleum organizations in Germany, United Kingdom, Canada, Norway and Belgium were also contacted.

While none of the American petroleum companies nor the European petroleum organizations supplied any compound-specific data (although there is a substantial amount of whole product toxicity data), API has conducted some individual compound subchronic testing. These data (API, 1985a and b, 1986), as well as much of the data described in the literature, focus on one particular endpoint, the accumulation of $alpha_{2u}$-globulin in the renal tubule of male rats. The accumulation of this protein is followed by kidney disease (nephropathy) and an increased incidence of kidney tumors. The USEPA (USEPA, 1991a)

has determined that the response of male rats is unlike that of other laboratory species and that non-mutagenic animal carcinogens that produce only male rat kidney tumors through an alpha$_{2u}$-globulin-mediated mechanism are probably not carcinogenic in humans. The testing by API did not identify NOAELs or LOAELs.

For toxicological purposes, the petroleum hydrocarbons can be divided into two very broad classes: the alkane/cycloalkane and the aromatic/alkene compounds. A brief overview of the toxic effects of these classes of compounds is presented below.

Alkane/Cycloalkane. The largest body of toxicity information for this group of compounds is available for the alkanes. The studies on alkanes are short-term studies which primarily focus on their relative effectiveness in causing mucous membrane irritation or disruption of the CNS. CNS effects are commonly associated with exposure to the lower molecular weight compounds (C5 through C9). The mechanism of toxicity is thought to involve the interaction of the lipid-soluble hydrocarbon with the lipid membrane of the nerve cell. The potency may be a function of lipid solubility and, therefore, a function of carbon chain length (Clement Assoc., 1989; Casarett and Doull, 1986). Animal studies indicate that narcotic activity within the C5-C8 range increases as a function of carbon chain length (Swann *et al.*, 1974; ACGIH, 1986). The narcotic potency decreases beyond C9 (Crisp *et al.*, 1967). Evidence of cerebellar dysfunction and damage to cerebellar neurons suggests that the CNS is a target organ for the toxic effects of n-nonane (Nilsen *et al.*, 1988). Exposure of Harlan-Wistar rats to 1,500 ppm of n-nonane for 65 days, six hours/day, five days/week (Carpenter *et al.*, 1978) resulted in mild tremors, slight coordination loss, and low irritation of the eyes and extremities. A no ill-effect level was reported at 590 ppm (3,095 mg/m^3).

The American Conference of Governmental Industrial Hygienists (ACGIH) has identified workplace exposure limits for a number of the alkanes (pentane, n-hexane, heptane, octane and nonane) as have the National Institute for Occupational Safety and Health (NIOSH) and the Occupational Safety and Health Administration (OSHA) (Table 4). While these values are applicable to workplace exposures only, they provide an indication of these agencies' opinions of the *relative* toxicity of the various alkanes. n-Hexane is the most toxic of these alkanes. As described in Section 2.3.1, peripheral neuropathy has been observed in workers exposed to n-hexane. There is some question as to whether pentane, heptane and octane are also associated with this effect. Experimental data has demonstrated that a metabolite of n-hexane, 2,5-hexanedione, is responsible for the neurotoxicity (Couri *et al.*, 1978). The potential for a compound to be metabolized to a (-diketone appears to be essential to development of peripheral neuropathy (Couri and Milks, 1985). It has been

suggested that heptane and octane may be metabolized to neurotoxic products (Spencer and Schaumburg, 1977). In addition, ACGIH (1986) cites a study by Gaultier *et al.* (1973) in which employees at a belt manufacturing shop were exposed to a solvent containing 80% pentane, 14% heptane and 5% hexane. Three of five cases exhibited symptoms which included peripheral nerve changes and paresthesis. However, one study has shown that among the aliphatic hydrocarbons n-pentane, n-hexane and n-heptane, only n-hexane is neurotoxic (Takeuchi *et al.* 1980). The ACGIH has established the threshold limit value (TLV) for n-hexane at about an order of magnitude lower than the other alkanes.

The TLVs for pentane, heptane and octane are generally based on effects associated with acute inhalation exposure to these compounds, primarily narcosis and mucus membrane irritation. For nonane, little information on workplace exposures is available and the TLV is established based on the lethal concentration for inhalation of nonane compared to the smaller (heptane, octane) alkanes. While neurotoxic effects were seen upon exposure to nonane, no pathological changes were observed in animals exposed to a series of compounds C10-C13 via inhalation for 8 hours and observed for the following 14 days (Nilsen *et al.*, 1988). In a study using the mouse ear edema model, dodecane (C12) was nonirritating, while tridecane (C13) only

TABLE 4
Workplace Exposure Limits for Selected Alkanes/Cycloalkanes

COMPOUND	ACGIH TLV[a] (mg/m3)	NIOSH PEL[b] (mg/m3)	OSHA PEL[b] (mg/m3)
ALKANES:			
Pentane	1,770	350	1800
n-Hexane	176	180	180
Other Hexane Isomers	1760	NA	NA
Heptane	1,640	350	1600
Octane	1,400	350	1450
Nonane	1,050	NA	NA
CYCLOALKANES:			
Cyclopentane	1,720	-	-
Cyclohexane	1,030	1,050	1,050
Methyl cyclohexane	1,600	1,600	1,600

Source:

(a) TLV = Threshold Limit Value; ACGIH, 1992
(b) PEL = Permissible Exposure Limit; NIOSH, 1990

exhibited a response after 96 hours (Moloney and Teal, 1988). Tetradecane (C14) was the strongest irritant, and hexadecane (C16), octadecane (C18) and eicosane (C20) exhibited progressively decreasing activity.

Similar to the straight chain hydrocarbons, the cycloalkanes are dermal irritants and also affect the central nervous system. They have a similar level of activity as the aliphatic hydrocarbons. ACGIH, NIOSH and OSHA have established workplace standards for cyclopentane, cylcohexane and methyl cyclohexane that are of similar magnitudes as the non-n-hexane alkanes (Table 4). The somewhat lower TLV for cyclohexane is based on a 1943 study of inhalation exposure of rabbits for 50 periods of 6 hours each. This study found no toxic effects in tissues at a concentration of 434 ppm. A TLV was set at 300 ppm (1030 mg/m^3).

One additional source of toxicity information for the higher molecular weight alkanes, cycloalkanes and isoalkanes is available in the study of white mineral oils (API, 1992). White mineral oil is a heavily refined petroleum product consisting almost entirely of straight chain, branched or cyclic alkanes, having carbon numbers in the range of C15 to C50. They may have residual aromatic compound contents of several percent, dependent upon the degree of refining of the feedstock. These oils are used as laxatives and in pharmaceutical and cosmetic applications. They are also used commercially in bakery products, as a dust control agent for grain, as defoamers in foods, on raw fruits and vegetables, and in the manufacture of candy (API, 1992).

A number of 90-day subchronic dietary or oral gavage toxicity studies of white mineral oil have been conducted by Exxon Biomedical Sciences, Inc., Atlantic Richfield Company (ARCO), and Shell Oil Company. The Exxon studies included four white mineral oils tested in rats and dogs and one medicinal grade mineral oil tested in rats only. No toxicity was observed with any oil in any species and NOAELs exceeded 125 mg/kg/day for the white mineral oils, and 4350 mg/kg/day for the medicinal grade oil. Lifetime dietary feeding studies in rats also failed to establish any chronic or carcinogenic effects, and NOAELs exceeded 1200 to 6000 mg/kg/day in these studies (API, 1992).

ARCO conducted a 90-day feeding study in rats of a technical (medicinal) grade oil and found no toxic effects at dietary concentrations up to 10,000 ppm (unpublished study, reported in API, 1992).

Shell Oil Company (Hernandez, 1989) tested two white mineral oils which were treated by two different distillation processes, one by oleum treatment and one by hydrotreating. Male

and female rats were exposed at concentrations ranging from 5000 ppm to 20,000 ppm in the diet. In a second study of only female rats, exposures ranged from 10 ppm to 20,000 ppm in the diet (0.6 to 1150 mg/kg/day). In the first study, hyperphagocytic granulomas were detected in the livers of female rats at dosages of 5000 ppm or above, with greater incidences in the oleum treated mineral oil group. Male rats showed Kupffer-cell hypertrophy and very slight multifocal granulomas at 20,000 ppm. In the second study, one female rat had frequent granulomatous macrophage syncytia at 100 ppm and no lesions were present at 10 ppm. Three of five rats given 5000 ppm presented this lesion.

The cause of the hepatic lesions in these studies is probably related to the absorption of high molecular weight hydrocarbons. Once absorbed, the presence of the relatively inert hydrocarbon in the liver results in a local inflammatory reaction due to difficulties in the metabolism or excretion of the inert material.

The findings of the Shell study were not supported by the studies conducted by Exxon Biomedical or ARCO (API 1992) and may be the result of the crude type of oil used or contamination of the tested oil. Chemical speciation was not performed, so it is impossible to determine whether more toxic constituents (eg. aromatics) were present in these oils and contributed to the effects seen. All of the investigations listed above were complete and evaluated similar parameters.

Studies of human subjects who had prolonged or excessive exposure to white mineral oil revealed structural and functional changes in the cells of the liver, lung, spleen and mesenteric lymph nodes which are minor in nature and not considered to be of significance. Accumulation of mineral oil hydrocarbons in human liver, spleen and lymph nodes has been documented, although it has not been considered harmful (Hernandez, 1989).

There is no evidence that alkanes are complete carcinogens. However, higher alkanes (decane and larger) appear to act as cocarcinogens or promoters of carcinogenic effects. Horton and Christian (1974) report that cocarcinogenic activity (which may be the result of the solvent properties of these compounds) may be common to many C12 to C30 aliphatic hydrocarbons. Decane, dodecane and tetradecane when applied to the backs of mice enhance the rate of tumor development following exposure to UV light (Bingham and Nord, 1977). n-Decane exhibits potent cocarcinogenic activity and tetradecane exhibits weak to moderate activity on mouse skin initiated with benzo(a)pyrene (Van Duuren and Goldschmidt, 1976). Dodecane is a potentiator of benzo(a)pyrene and benzo(a)anthracene carcinogenicity when applied to the backs of mice (Bingham and Falk, 1969; Horton *et al.*, 1976). Dodecane and tetradecane promote papilloma growth on the skin of Swiss mice

treated with dimethylbenzanthracene (DMBA), and also produce severe dermal irritation (Baxter and Miller, 1987; Sice, 1966).

Aromatic/Alkene Compounds. The aromatic compounds can be divided into benzene, and its alkyl derivatives; phenylic compounds; and, the polycyclic aromatic hydrocarbons (PAHs). The toxic effects of benzene, and some of its alkyl derivatives (toluene--methyl benzene, xylene--dimethyl benzene, ethyl benzene and cumene--isopropyl benzene) were discussed previously. No dose-response values have been established for other, larger alkyl benzenes. In general alkyl benzenes are CNS depressants due to their affinity for nerve tissue (Sandmeyer, 1981a), and may also produce kidney and liver effects. Toxic effects of biphenyl were also discussed previously and include effects to the central and peripheral nervous systems, cardiac, hepatic and renal systems.

The health effects of chronic exposure to a number of PAHs for which USEPA has established dose-response values were discussed previously. Common threshold effects associated with this class of compounds includes dermal irritation, blood toxicities and kidney and/or liver effects.

Alkenes are not considered to be particularly toxicologically active (Sandmeyer, 1981b) and do not exhibit neurotoxic properties. Repeated exposure to high concentrations of the smaller alkenes have produced hepatic damage and hyperplasia of the bone marrow in animals. No corresponding effects have been recorded in humans (Sandmeyer, 1981b).

2.3.2.2 Availability of Acute Toxicity Information. In the absence of a relatively comprehensive chronic toxicity database for use in this project, the acute toxicity literature was examined. The objective was to identify relative magnitudes of toxicity between petroleum hydrocarbon compounds based on LD_{50} or LC_{50} values. With this relative scale, and selected chronic toxicity values for some compounds, relative chronic toxicity values could be assigned to other compounds, based on intercompound variation in acute toxicity values.

Acute toxicity data were obtained from the following references: Dangerous Properties of Industrial Materials (Sax, 1989); Patty's Industrial Hygiene (Clayton and Clayton, 1981); Installation Restoration Program Toxicology Guide (IRP, 1991); Occupational Health Services MSDS on Disk (OHS, 1992); and, Registry of Toxic Effects of Chemical Substances Data Base (Micromedex, 1992). While these were secondary compendia of information, they provided rapid access to a large database for the purposes of this review. One shortcoming associated with using these types of sources is incorrectly reported

toxicity values. Our analysis included one method for detecting such errors, which is described below.

A summary of the acute toxicity data obtained is presented in Appendix A. Also added to the data file were a limited number of NOAEL values from animal exposure studies cited. These doses had been adjusted to continuous exposure doses from primarily subchronic duration intermittent exposures. While a substantial amount of acute toxicity data (i.e., LD_{50} or LC_{50}) was identified, these data represented different exposure routes in several species. In addition, the secondary sources reviewed did not present specifics on the testing procedures (which would allow a screening of the data quality). Attempts to correlate acute toxicity data with chronic RfDs did not reveal significant relationships between the two toxicity indicators for exposures of different duration. Also compiled for each compound were its chemical and physical characteristics: carbon number, molecular weight, water solubility, vapor pressure, Henry's Law Constant, octanol: water partition coefficient (K_{ow}), and soil adsorption coefficient (K_{oc}). A preliminary data analysis was conducted with the objective of identifying any underlying relationships between the physical and chemical attributes of the chemicals (treated as independent variables) and the indicators of lethal and sublethal toxicity (treated as dependent variables).

Statistical analyses were performed in an attempt to identify a relationship between chemical/physical characteristics and acute toxicity dose-response values. Pearson-product moment correlations were determined between all possible pairs of variables. Bivariate scattergrams of all pairs of variables were also used to aid in the identification of these relationships. Visual examination of the scattergrams also permitted the detection of any obvious outliers in the data which might have been due to incorrectly reported values in the data compendia or errors in transcription. The check did not permit detection of slight errors in the data. Since the objective of this exercise was to identify overall trends in a large data set, the effect of any such undetected errors was judged to be relatively insignificant. These analyses were initially performed on all aggregated data and then for specific petroleum hydrocarbon subgroups (classified by carbon number ranges, structural groups, exposure routes, and animal species).

Stepwise multiple regression analyses of toxicity indicator values versus independent variables were performed. The correlation analysis was first used to identify any cross-correlations between variables and only one of highly correlated pairs of independent variables was used in the regression analysis.

This quantitative approach to understanding possible determinants of petroleum hydrocarbon compound toxicity was generally unproductive. Correlations between either LC50 values or NOAEL values and chemical/physical characteristics were quite low and insignificant. The independent variables never predicted more than a few percent of the variation in the toxicity values for any of the data subsets analyzed. It was therefore concluded from the analysis that acute toxicity information could not be used to assign relative chronic toxicity values, nor could these toxicity indicator values be predicted by knowledge of the physical and chemical characteristics of the compounds.

2.3.3 Summary of Available Toxicity Information

Toxicity data were identified for whole product, and for chronic and acute effects of component hydrocarbons. Whole product testing is conducted on pure, fresh product. Its applicability to the weathered product encountered at petroleum sites is questionable, because the compositions of weathered petroleum products differ substantially from that of pure products. For this reason, it was decided that whole product toxicity data would not be used in this assessment.

Adequate chronic toxicity information is available for a limited number of compounds. USEPA has used these data to develop RfDs and SFs. A search for chronic toxicity data for other petroleum hydrocarbons revealed that sufficient chronic toxicity information was not available for individual petroleum compounds to allow the development of chronic toxicity values. Acute toxicity data were also identified in the hope that these data could be correlated with available chronic toxicity data and used to infer chronic toxicity dose response relationships for additional petroleum hydrocarbons. Evaluation of these data did not reveal any significant relationships that would allow the estimation of additional dose-response values.

3.0 ASSIGNMENT OF TOXICITY INDICATOR VALUES FOR COMPONENTS OF PETROLEUM PRODUCTS

As described previously, neither adequate acute nor chronic chemical-specific toxicity data were identified for petroleum hydrocarbons components other than those for which USEPA has established RfDs or slope factors. The following analysis was conducted to facilitate the assignment of toxicity values for individual compounds, or classes of compounds, derived from petroleum products.

The components of petroleum can be generally divided into broad chemical classes: alkanes, cycloalkanes, alkenes, and aromatics. A review of Table 3 reveals that a USEPA RfD is available for only one alkane, n-hexane. No RfDs are available for other alkanes, nor for any cycloalkane or alkene. For this assessment, because of the limited information on toxic effects associated with exposure to the cycloalkanes, and the fact that available literature indicates similar toxic effects for alkanes and cycloalkanes; alkanes and cycloalkanes are treated similarly.

Alkenes are evaluated similarly to aromatics for methodological convenience, which incidentally can be supported by technical rationalizations. In the analytical scheme accompanying this methodology, aromatics and alkenes will separate out together from the alkanes and cycloalkanes (Section 4.2.4). Rather than requiring additional, more expensive analytical steps to separate the aromatics and alkenes, they are treated together for the following reasons. First, both aromatics and alkenes are metabolized by conversion to epoxides of varying reactivity (Casarett and Doull, 1991). Additionally, since alkenes (olefins) are found in gasoline at 3 to 5% (IARC, 1989a), at less than 1% in aviation fuels (IARC, 1989b); and at 1-2% in No. 2 fuel oils (NAS, 1976), the assumption that alkene toxicity is of the same magnitude as aromatics should not overly bias or underestimate risk estimates.

RfDs have been developed by USEPA for a number of noncarcinogenic aromatic petroleum compounds ranging from toluene (methylbenzene) to fluoranthene and pyrene (multiringed structures with 16 carbons). Slope factors are available for two carcinogenic aromatic petroleum compounds: benzene, a single-ringed compound; and BaP, a large five-ringed polycyclic aromatic hydrocarbon (PAH).

Compounds which have been adequately evaluated are used as representative "reference" compounds. The reference compounds were also used to derive alternate RfDs for structurally similar compounds.

3.1 ALKANES AND CYCLOALKANES

The alkanes and cycloalkanes are divided into groups based on number of carbons and the structure-activity relationships previously described. These classifications are used to develop alternate RfD values when information on individual chemicals is not available. Compounds in the C1-C4 category were not considered further because of their high volatility. This volatility makes chronic exposure at sites unlikely. With limited information available on other toxic endpoints, relative potency of neurotoxicity was used to derive alternate RfDs for the smaller alkanes. Cycloalkanes are expected to exhibit similar effects as the comparable alkane. Reference compounds identified for each group are as follows:

- n-hexane - for C5 through C8
- n-nonane - for C9 through C18
- eicosane - for C19 through C32

Table 5 presents proposed alternate RfDs for petroleum hydrocarbons. Compounds with five through eight carbons were grouped with n-hexane and assigned the same RfD of 0.06 mg/kg/day. This is a health protective approach because n-pentane, n-heptane and n-octane may be associated with peripheral neuropathies, but most likely to a lesser extent than n-hexane.

C9 through C18 hydrocarbons were grouped with n-nonane. This is because while decreasing neurotoxic effects are anticipated with increasing chain length, higher levels of dermal irritation are found from C13 through C18. Compounds in this group were assigned an alternate RfD of 0.6 mg/kg/day, ten times that for n-hexane. The rationale for this value is two-fold.

First, to characterize the relative potency of n-nonane compared to n-hexane, results of two subchronic inhalation studies were reviewed (Carpenter *et al.*, 1978; Dunnick *et al.*, 1989). No oral exposure studies are available for n-nonane. Each inhalation study exposed animals (rats for n-nonane, mice for n-hexane) to similar air concentrations for 6 hours/day, 5 days/week for 13 weeks. Carpenter *et al.* reported a NOAEL of 590 ppm for n-nonane. Dunnick *et al*. found minimal effects to the olfactory epithelium of two of ten female mice at 500 ppm, the lowest concentration to which animals were exposed. Thus, for n-hexane the LOAEL is 500 ppm. As discussed previously (Section 2.3.1.1), IRIS (1994) identified 500 ppm as a NOAEL. This selection is judged to be inappropriate based on the presence of mild nasal

TABLE 5
Alternate Oral Reference Dose Values
for Petroleum Related Hydrocarbons
Based on Chemical Classification.

	Reference Compounds	**Toxic Effect**	**Proposed Alternate RfD mg/kg/day**
ALKANES/CYCLOAKANES			
C5-C8	n-Hexane	Neurotoxicity	0.06
C9-C18	n-Nonane	Neurotoxicity	0.6
C19 - C32	Eicosane	Irritation/ functional changes	6.0
AROMATICS/ALKENES			
C9-C32	Pyrene	Nephrotoxic	0.03

lesions at this exposure level. The presence of nasal lesions at this exposure level may further indicate that neurological lesions, not examined in the 500 ppm group, may be occurring since these two endpoints parallel one another in frequency and severity at higher exposure levels.

A NOAEL for n-hexane can be estimated using the standard assumption that a 10- fold uncertainty factor is appropriate to convert a LOAEL to a NOAEL. It thus appears that n-hexane and n-nonane differ in potency by approximately one order of magnitude. While this comparison of toxicity is based on inhalation exposures and the exposure under consideration is oral, it is believed that the relative ability of these compounds to produce toxic effects is similar whether the exposure occurs via the inhalation or oral routes. Even though the absorption of these compounds will differ dependent on the route of exposure, once the compound is present in the blood stream, the distribution to the target organ and therefore, relative potency should be similar.

The second reason for assigning n-nonane an RfD that is ten times that for n-hexane is that a review of TLVs established for some petroleum hydrocarbons (Table 4) reveals that the TLV for n-nonane is approximately an order of magnitude greater than that for n-hexane, and n-nonane is not associated with peripheral neuropathies. The TLV for n-nonane is based on a comparison of lethal concentrations for n-nonane versus those for smaller alkanes.

Another approach to estimating an oral RfD for n-nonane would be to utilize route-to-route extrapolation, adjusting inhalation toxicity values to oral values based on relative absorption efficiencies of the two routes in question.

For volatile or semi-volatile molecules possessing lipophilicity, absorption is largely dependent on the establishment of a chemical equilibrium between the environmental levels and the bloodstream. The initial absorption rate tends to be large, but rapidly decreases as equilibrium is approached and achieved. Once equilibrium is established, absorption is dependent on clearance mechanisms which remove the chemical from the bloodstream (distribution to peripheral tissues, storage in fat, metabolism and excretion). The chemical will be absorbed only as rapidly as clearance occurs. This relationship maintains the equilibrium condition, as long as the air concentration is constant. When the air concentration is altered, a new equilibrium is rapidly established as clearance mechanisms adjust to the change in blood concentration. It is not possible to determine an absorption efficiency for the inhalation route which is constant at all environmental concentrations. A more appropriate measure of inhalation absorption would be a rate (ug/kg/min/ppm in air)

dependent on air concentration and biological processes. This rate could be large or small, depending on the environmental concentration.

Since it is not possible to derive a meaningful absorption efficiency for the inhalation route, extrapolation of inhalation toxicity values to oral values is not possible based on this simplistic approach. However, it may be possible to perform a route-to-route extrapolation with the use of physiologically-based pharmacokinetic (PBPK) modeling. PBPK modeling is a mathematical tool that has been used to increase the understanding of extrapolations performed in risk assessment. The USEPA has used this pharmocokinetic approach for the extrapolation of inhalation toxicity data to oral RfDs for acetonitrile, carbon disulfide, and nitrobenzene. PBPK models reconstruct the uptake, distribution, metabolism, storage and elimination of chemicals within the body, taking into account specific chemical and physiological parameters (chemical-specific partitioning coefficients, tissue blood flows and metabolic rates). Differential equations are used to describe the rate of change of chemical concentrations within these physiologically realistic compartments. The principle advantage offered by PBPK models is that they can relate an external airborne concentration or applied oral dose to an estimation of internal dose to a toxicological target. This allows the comparison of chemical exposures, by different routes and in different species, and extrapolation based on relevant information.

PBPK modeling may be used to extrapolate the inhalation toxicity information for n-nonane into an estimate of oral toxicity. However, the input parameters required for the model, though available for n-hexane, are not available for n-nonane. Therefore, the inhalation toxicity data will serve as an indicator of relative potency of n-nonane to n-hexane rather than as the basis for the derivation of oral toxicity values.

Alkanes C19 and longer were grouped together, with eicosane identified as a reference compound. These alkanes cause little neurotoxicity (Clement Assoc., 1989). Compounds in this group were assigned an RfD of 6.0 mg/kg/day. No quantitative toxicity data are available for any particular one of these larger alkanes. However, toxicity testing has been conducted on white mineral oil, a complex mixture of C15 to C50 saturated hydrocarbons. With the exception of a subchronic toxicity study of two types of mineral oil conducted by Shell Oil (Hernandez, 1989), this product has been shown to be of low toxicity (API, 1992). LOAELs have not been identified (other than the Shell Oil study) and NOAELs up to 6000 mg/kg/day are reported (API, 1992). The Shell Oil study described the formation of hyperphagocytic granulomas in the livers of rats, resulting from the absorption of limited amounts of large molecular weight petroleum hydrocarbons and a subsequent localized inflammatory reaction resulting from the presence of the inert hydrocarbons in the liver.

Because of the large body of evidence in animal studies, as well as the human use of mineral oil, which has not resulted in pathological effects, it is concluded that the Shell Oil study is not representative of the toxicity of most white mineral oils. Since no LOAELS were identified in the studies of white mineral oil, the highest NOAEL can be used as a basis for the derivation of an RfD. The highest NOAEL reported is 6,000 mg/kg/day in a lifetime dietary feeding study. A 1000-fold uncertainty factor is appropriate for use in this case (10 for subchronic exposure, 10 to account for differences between species, and 10 to protect sensitive human subpopulations), resulting in an RfD of 6 mg/kg/day.

3.2 AROMATICS AND ALKENES

3.2.1 Threshold Effects

The USEPA has published chronic oral RfDs for several of the lower molecular weight aromatic hydrocarbons (Table 3) covering a range of carbon numbers from C9 to C15. These values are all quite similar, ranging (with the exception of anthracene), from 0.03 to 0.06 mg/kg/day. The RfD for anthracene is 0.3 mg/kg/day. The limited systemic toxicity data on these compounds (summarized in ATSDR, 1993e) indicates that their structural similarity results in similarities in their metabolism. Their toxic effects are also exerted on similar organ systems; primarily the blood, kidney, and liver.

Because of the similarities in the RfD level, in metabolism, and effect, one alternate RfD is assigned for the entire range of C9 through C32 PAHs. The alternate RfD is the lowest of those developed by USEPA for the noncarcinogenic PAHs: 0.03 mg/kg/day for pyrene. This approach results in an overestimate of risk for anthracene, which has a USEPA RfD of 0.3 mg/kg/day. However, results of analyses of weathered No. 6 fuel oil, a mixture of weathered No. 2 diesel fuel and fuel oil, No. 2 fuel oil, No. 4 fuel oil, a mixture of weathered gasoline and diesel fuel, and Bunker fuel oil No. 6, reveals that anthracene makes up less than 1 percent of the total TPH for these fuel products. Thus, applying the lower RfD to this compound should not significantly affect the total risk estimate.

The selection of one RfD for the C9 through C32 PAHs is considered to be appropriate for a number of reasons:

- Use of one RfD appropriately reflects the uncertainty inherent in the estimation of the toxicity of the numerous PAHs with nine to 32 carbon atoms.

- It results in the simplification of analysis.

An alternate approach would be to quantify those PAHs for which a USEPA RfD is available and evaluate their toxicity separately from the remaining mass of PAHs (for which one RfD is assigned). Because of the similarity in the level and type of toxic effects exerted by these compounds, this approach does not seem to be worth the added cost or effort.

In site-specific risk assessments, the approach presented here will be used in conjunction with a compound-specific risk assessment approach. Aromatic compounds with fewer than nine carbons (such as benzene, toluene, ethylbenzene and xylenes) will be evaluated on a compound-specific basis. These compounds are, therefore, not included in this "alternate RfD" approach. Because of the compound-specific approach employed for aromatics with less than nine carbon atoms, C5 through C8 alkenes are not evaluated in this approach. Because all alkenes in petroleum products comprise from 1 to 5 percent of total hydrocarbons, it is not anticipated that this approach will excessively bias or underestimate the risk estimate.

3.2.2 Non-threshold Effects

USEPA cancer slope factors are identified for only two aromatic petroleum hydrocarbons: benzene and BaP. For site-specific risk assessments, cancer risk of exposure to benzene will be evaluated using a compound-specific risk assessment approach. While a slope factor is available only for BaP, USEPA considers the following PAHs to be carcinogenic: benzo(a)anthracene, indeno(1,2,3-cd)pyrene, dibenz(a,h)anthracene, chrysene, benzo(b)fluoranthene, and benzo(k)fluoranthene.

An "Estimated Order of Potential Potency" for these carcinogenic PAHs relative to BaP has been published by USEPA in "Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons" (USEPA, 1993c). BWSC is developing guidance on the relative potency of carcinogenic PAHs and has prepared a "Draft Proposal for the Assessment of PAH Carcinogenicity" (MADEP, 1994). Carcinogenic PAHs will be quantified individually and a compound-specific risk assessment will be performed.

4.0 DEVELOPMENT OF ALTERNATIVE ANALYTICAL TECHNIQUE

4.1 INTRODUCTION

In this section proposed analytical requirements for the identification and quantitation of individual compounds and particular molecular weight ranges of petroleum hydrocarbons are described. These requirements were developed in response to the risk assessment approaches described earlier in this paper for addressing petroleum contaminated media.

Throughout this section, TPH refers to the identification and quantitation of petroleum compounds from C5 to C32, including fractions and individual compounds, and never refers to a particular analytical method.

4.2 CURRENTLY AVAILABLE METHODS

Several methods are available for the analysis of petroleum hydrocarbons. The methods vary in several ways including analysis cost, the range of compounds detected, detection limits and the availability of quantitation. Table 6 summarizes some of the advantages and disadvantages of the different petroleum analysis methods. The methods range in compound specificity from screening to fully quantitative methods. A brief discussion of the most commonly used methods and their usefulness for developing health risk assessments is presented below.

4.2.1 Gravimetric Methods

Gravimetric analysis is considered to be a general screening procedure which detects a wide range of compounds including hydrocarbons from anthropogenic and non-anthropogenic sources. The method will not discriminate between simple classes of hydrocarbons. This procedure represents the most basic level of analysis and is not recommended for health risk assessment purposes.

TABLE 6
Comparison of Petroleum Hydrocarbon Analysis Methods

METHOD	APPROXIMATE DETECTION LIMITS	APPROXIMATE COST PER SAMPLE (DOLLARS)	ADVANTAGE	DISADVANTAGES
Gravimetric	10 ppm	50	1	7,8
Infra-red	1 to 10 ppm	50 - 80	1	7,8
Ultra-violet Fluorescence	10 ppb	50	1	7
GC/FID Screen	10 ppm	100 - 150	2,3	7,8
GC with FID/PID	1 ppb	150 - 200	2,3,4	9
GC/FID w/ clean-up	100 ppb	200 - 300	2,3,4	10
GC/MS SIMS	10 ppb 0.1 ppb for PAHs	600 - 800	2,3,4,5,6	10

Advantages

1. Inexpensive
2. Will identify products
3. Will identify non-target compounds
4. Will separate aliphatics and aromatics
5. Quantitative
6. Very low detection limits

Disadvantages

7. Is a general screening method
8. High detection limits
9. Not widely used
10. Expensive

4.2.2 Infra-Red and Ultra-Violet Methods

Infra-red (IR) and ultra-violet fluorescence (UV) methods are very similar in the types of information that they provide. Both techniques are considered screening methods. Ultra-violet methods rely on the emitted fluorescence of energy from primarily aromatic compounds which are excited at discrete wavelengths or over a range of wavelengths. These methods have been used extensively in marine oil work but are not often used when generating data for use in health risk assessments at hazardous waste sites or for drinking water or groundwater monitoring.

USEPA Method 418.1, the most widely used IR method, uses a single wavelength of 2930 cm^{-1} to detect the carbon-hydrogen stretch present in aliphatic and aromatic hydrocarbons. This technique does not provide product identification if it is performed as outlined in the EPA method.

There are several problems with analyzing petroleum hydrocarbons by IR. Volatile organic compounds (VOC) are lost in the Freon 113 extraction step and it is therefore necessary to do a specific volatile organic test, either by gas chromatography (GC) or gas chromatography/mass spectrometry (GC/MS) if VOCs are a concern. Five and six membered ring compounds are removed during the silica gel clean-up stage of the analysis and the results may be artificially high or low. Natural products (e.g, plant waxes, containing odd numbered alkanes from n-C25 to n-C31) and siloxane's are sometimes measured as part of an IR method for petroleum hydrocarbons. The standard calibration methods are not applicable for quantitating these compounds. This method is not recommended for generating data used in health risk assessments.

4.2.3 Gas Chromatography Methods

There are several gas chromatography methods available for the analysis of petroleum hydrocarbons. High resolution capillary column chromatography (HRCC) is generally used for the separation of the complex mixture, which makes up petroleum, into fractions and in some cases, individual components. The methods have been adapted for many different applications including sample screening, product identification, detection limit improvement and quantitation of specific compounds.

Fingerprinting methods are refined GC techniques that separate, and many times quantitate, the complex mixture of chemicals present in petroleum into product fractions based on

molecular weight and boiling point ranges. The term fingerprinting is a general term that can represent several different analysis techniques. Fingerprinting methods are often referred to as product identification methods. Different fractions of petroleum produce unique patterns on a chromatogram. Pattern recognition is used to identify the products. High resolution capillary chromatography is widely used for the separation of petroleum since it is the only generally available separation technique that has the power necessary for dealing with a complex mixture.

4.2.4 Methods for Separating Alkanes and Cycloalkanes from Alkenes and Aromatics

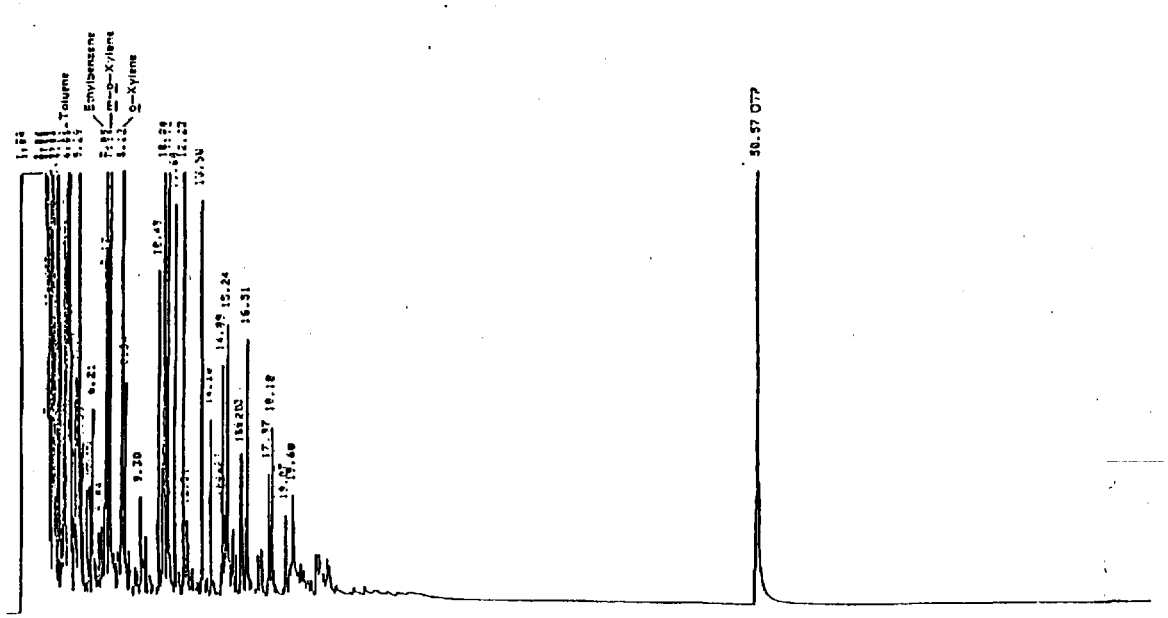
Separate fractions, one consisting of alkanes and cycloalkanes and the other containing alkenes and aromatic compounds, can be isolated from the mixture of petroleum hydrocarbons by two common processes. The first method is a column clean-up using silica or alumina gel chromatography or high performance liquid chromatography (HPLC). Two fractions are isolated: one is the aliphatic (saturated) hydrocarbons, the other the aromatic and unsaturated hydrocarbons. The aliphatic fraction is analyzed using a flame ionization detector (FID) and the aromatic/unsaturated fraction is analyzed using a photoionization detector (PID). Solid phase extraction is another column clean-up technique that is being used more routinely. Figure 2 presents a FID chromatogram for gasoline.

second way to differentiate components of petroleum hydrocarbons is by using detectors that respond preferentially to unique structural features of the compounds of interest. By exploiting the double bond contained in the unsaturated and aromatic hydrocarbons, the PID can be used to identify aromatic compounds and alkenes present in petroleum.

4.2.5 Mass Spectrometry

Analytical mass spectrometry (MS) is well suited for the analysis of complex mixtures of chemicals as are found in petroleum products. Mass spectrometry, especially when coupled with gas chromatography (GC/MS), has inherent advantages over other

Figure 2
Example of a FID Chromatogram for Gasoline



detection methods often used for the analysis of petroleum. Electron impact (EI) ionization is the most commonly used method of ionizing molecules used to detect organic compounds in environmental analysis. Structural information from the ionization, and subsequent fragmentation, of molecules in the mass spectrometer provides confirmatory or unequivocal identification of target and non-target analytes which may be important in many site investigations. When capillary GC columns are attached to a MS, each eluated peak is scanned several times every tenth of a second over a predetermined mass range. The mass range is typically from 35 to 500 amu (atomic mass units) depending on the molecular weights of the compounds of interest. Scanning several points across a GC peak provides several spectra which can be evaluated for homogeneity. Chromatographic peaks that overlap can be identified or resolved.

Selected ion monitoring (SIM) is a refinement of mass scanning that results in greater analyte specificity and lower sample detection limits. It is not uncommon to achieve detection limits of 100 ng/l in water.

4.3 RECOMMENDED ALTERNATIVE ANALYTICAL TECHNIQUE

4.3.1 Introduction

Several commonly used methods for characterizing petroleum hydrocarbons in water and soil have been described. Some methods that have traditionally been used for petroleum hydrocarbon analysis lack the specificity and sensitivity necessary for the objectives of risk assessment. Even though the gravimetric, UV and IR methods are not recommended for risk assessment purposes, they are well adapted to other investigation needs, for example as a screening tool and for mapping plume size and movement. In most cases, gas chromatography is the method of choice for the qualitative and quantitative analysis of environmental samples for petroleum hydrocarbons.

ORS is presenting an analytical approach that uses two high resolution capillary gas chromatography methods: one for analyzing volatile petroleum hydrocarbons (VPH) and one for analyzing extractable petroleum hydrocarbons (EPH). There are other methods available for analyzing petroleum but the approaches that are outlined in the following sections produce results which are necessary for the purposes of this project, namely to generate data on the identity and quantity of petroleum hydrocarbons in water and soil samples that can be used in health risk assessments.

From a technical stand point, mass spectrometry coupled with high resolution gas chromatography is the preferred method for analyzing mixtures of petroleum hydrocarbons because it offers the advantages of target analyte confirmation and non-target analyte identification by compound fragmentation. However, it is not the recommended method for several reasons.

Mass spectrometers are not as common as FID or PID in environmental laboratories primarily due to cost. Sample analysis by mass spectrometry is generally more expensive than analysis by PID or FID because of initial capital outlay and continued maintenance costs. Mass spectrometers are generally not available for use in environmental analysis as separate detectors. They are part of packaged units consisting of a gas chromatograph, vacuum system, ionization chamber, and a mass analyzer which are much more expensive than PID or FID.

A second major reason that MS is not as widely available as other GC detection techniques is the higher degree of training necessary for instrument operators. The daily maintenance of an MS requires operators that are skilled in electronics and instrument repair. The instrument must be tuned at least daily to ensure the integrity of the mass spectra that are generated. Also, the interpretation of the spectra resulting from sample analysis requires a thorough knowledge of organic chemistry to recognize nuances in fragmentation patterns.

Advances in instrument and computer technology within the last few years have made the mass spectrometer more affordable and easier to use. Mass spectrometers are smaller and less expensive. One-piece ionization source and quadrupole units have replaced the older multi-component units that were a major source of maintenance time. Instead of spending an entire day on repair or maintenance, a mass spectrometer can be cleaned or repaired often within hours. Significant improvements in computer generated data acquisition and mass spectral database searching have decreased the requirement for some of the very high level of training previously necessary of mass spectrometrists.

The sample extraction and concentration procedures and chromatographic run conditions are identical for the detection of compounds by MS, PID or FID. Analysis by PID and FID in series was chosen as the recommended method because of the greater availability of these detectors over mass spectrometers. There is no objection for a lab to submit data from mass spectrometry analysis as long as it can be demonstrated that equal performance with the recommended method detection limits and linearity has been achieved. Data generated

from methods other than the recommended methods will be evaluated on a case-by-case basis. The validation of MS methods by DEP is being considered.

The procedures outlined in sections 4.3.2 and 4.3.3 are based primarily on a 1990 draft document jointly issued by the Underground Storage Tank Work Group of the USEPA, the American Petroleum Institute, the Midwest Research Institute and Enseco Incorporated entitled: Measurement of Petroleum Hydrocarbons: Report on Activities to Develop a Manual (USEPA, 1990.). This draft document is based on specific methods described in the USEPA SW-846, Test Methods for Evaluating Solid Wastes (SW-846) 3rd ed: methods 3510, 3520, 3540, 3550, 8000 and 8100. Other states (California, New Jersey, and Wisconsin) have adapted this method to address analytical and regulatory requirements within their states.

The protocols presented in the following sections are currently being validated by the Wall Experiment Station which is the analytical laboratory for the Massachusetts Department of Environmental Protection. The validation procedure is a rigorous approach to determining precision, accuracy and detection limits for a given analytical protocol. Once the protocols have met the QA/QC requirements of the validation procedure, non-laboratory samples can be analyzed to show that the protocols are applicable to "real-world" samples. Protocols that have met all the QA/QC criteria for method validation and applicability to actual samples are usually called methods.

Alternative methods for the analysis of petroleum hydrocarbons may be submitted for consideration by the Department. The method must generate analytical data that are appropriate for use in health risk assessments. The method validation requirements of the proposed methods must be at least as stringent as the validation procedures used by the Department. The final decision regarding the acceptability of an alternative analytical method for generating data to be used for evaluating health risk from exposure to petroleum hydrocarbons in Massachusetts rests with the DEP.

4.3.2 Volatile Petroleum Hydrocarbon (VPH) Method

Gasoline-range volatile hydrocarbons in soil and water are analyzed by gas chromatography coupled to a purge and trap concentrating system. The method is capable of detecting hydrocarbons with a molecular weight range of approximately C_5 to C_{11}. The detectable molecular weight range can be changed by adjusting GC run conditions. Detection is achieved by using an PID in series with a FID. Quantitation is done by comparing the area under the chromatogram from the appropriate FID or PID response to the corresponding response of a volatile petroleum product standard.

The specific compounds and carbon number ranges which need to be identified and quantitated by VPH analysis for the proposed risk assessment methods are classified as aliphatic and aromatic fractions and are presented in Table 7.

Petroleum hydrocarbons are identified by comparing the environmental sample FID or PID chromatogram to the FID or PID chromatogram of an appropriate volatile hydrocarbon standard chromatogram. The standard chromatogram is generated by analyzing a laboratory water sample that has been spiked with a mixture of petroleum associated components at known concentrations. Benzene, toluene, ethylbenzene, and total xylenes are identified and quantitated as individual compounds. Quantitation is achieved by comparing the total area under the retention time range corresponding to a particular molecular weight range of a sample chromatogram to the corresponding total area for a standard chromatogram. This is applicable to both aliphatic and aromatic compounds. The components of the standard can be varied depending on the application of the final analytical results. Figure 3 presents a graphic depiction of the VPH analytical procedure.

4.3.3 Extractable Petroleum Hydrocarbon (EPH) Method

This method can measure extractable hydrocarbons in soil and water corresponding to carbon number ranges of approximately C10 to C32. Samples are spiked with a surrogate compound and extracted with methylene chloride. Surrogate compounds are used to monitor extraction efficiency. The extract is dried and concentrated to a final volume of approximately 1 milliliter. Approximately 2 microliters of the extract is injected onto a GC equipped with an FID and a PID in series.

TABLE 7
Required Analytical Parameters
Detected by VPH Analysis

ALKANES/CYCLOALKANES	AROMATICS/ALKENES
C_5 TO C_8	Benzene
	Toluene
	Ethylbenzene
	Xylenes (total)
	C_9 to C_{11}

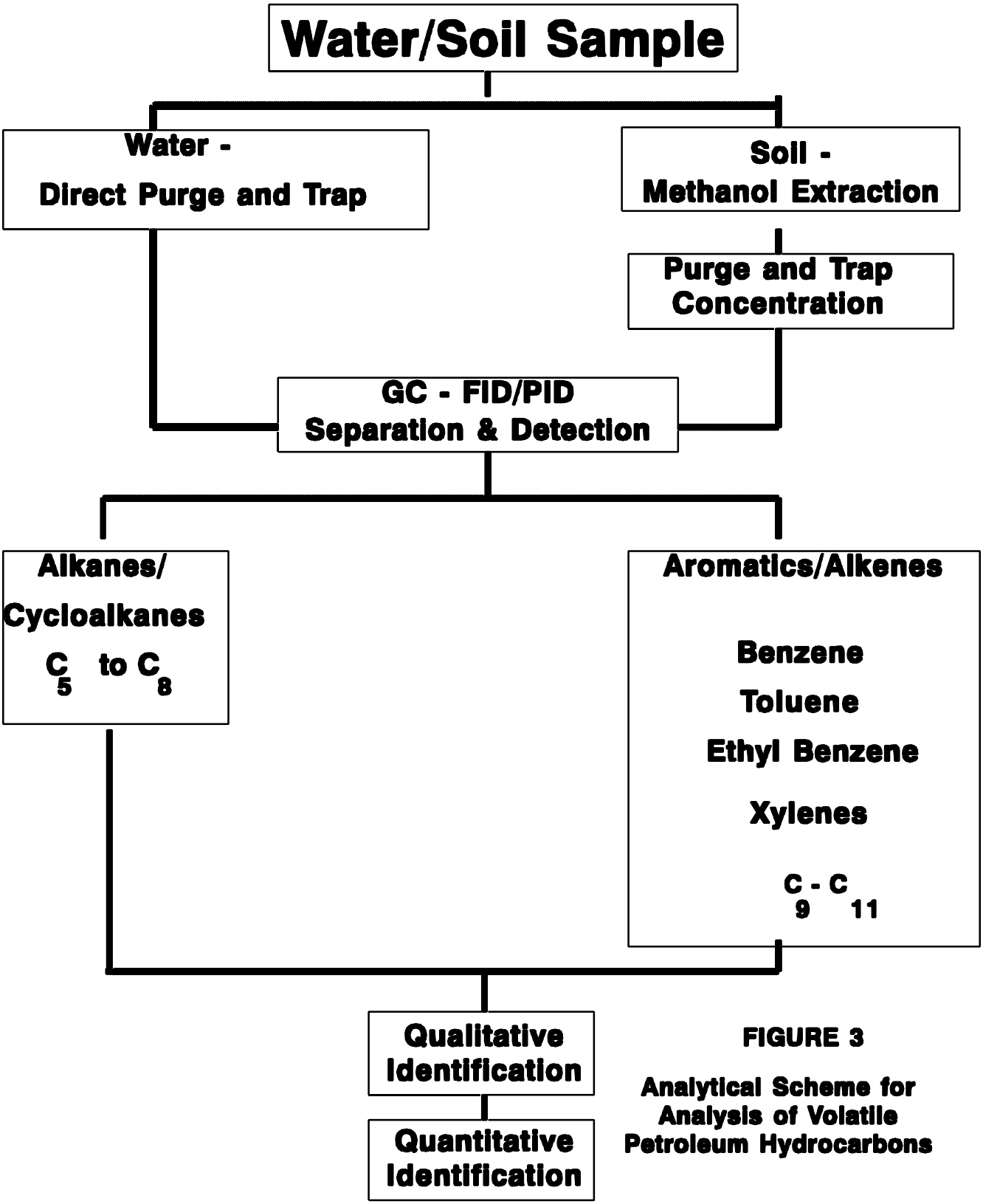


FIGURE 3

Analytical Scheme for Analysis of Volatile Petroleum Hydrocarbons

Quantitation is done by comparing the area under the chromatogram from the appropriate FID or PID response of a sample to the corresponding response of a standard mixture containing the compounds of interest.

The identification and quantitation for the EPH procedure is similar to the VPH procedure. The total area under the retention time range corresponding to a particular molecular weight range of a sample chromatogram is compared to the corresponding total area of a standard chromatogram. Polyaromatic hydrocarbons are identified and quantitated individually from the EPH extract. The compound ranges and specific compounds required for use in health risk assessments are presented in Table 8. Figure 4 presents a graphic depiction of the EPH analytical procedure.

Table 8
Required Analytical Parameters
Detected by EPH Analysis

ALKANES/CYCLOALKANES	AROMATICS/ALKENES
C_9 to C_{18}	C_{12} to C_{32}
C_{19} to C_{32}	cPAHs*

* Requirements for individual carcinogenic PAHs will be specified on a case-by-case basis.

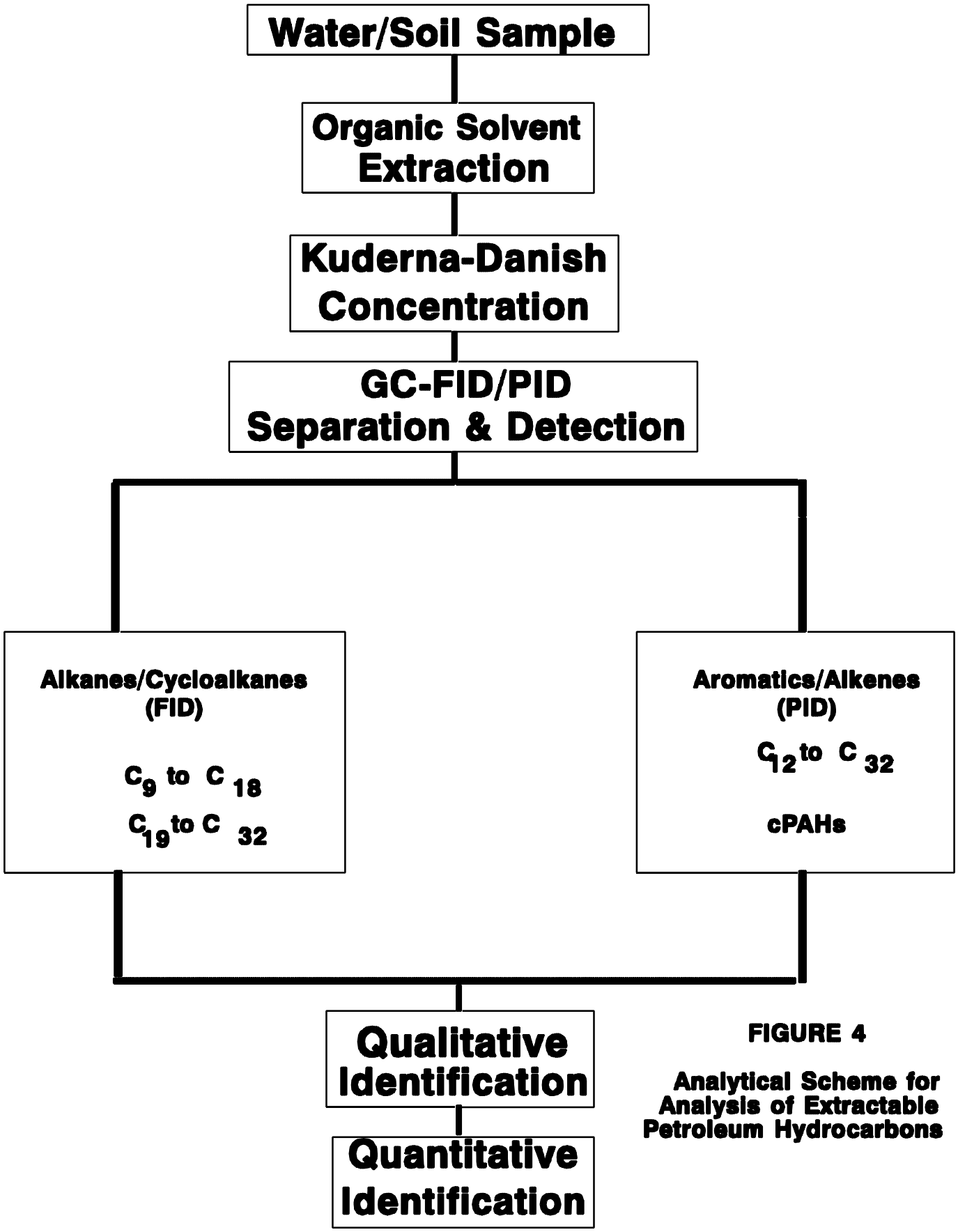


FIGURE 4

Analytical Scheme for Analysis of Extractable Petroleum Hydrocarbons

5.0 APPLICATION OF THE APPROACH

Figure 5 presents a hypothetical illustration of the approach for one petroleum hydrocarbon fraction, the alkane/cycloalkane portion of a soil analysis. A similar approach could be used for the aromatic/alkene fraction, in other media and for exposure routes other than ingestion. BTEX compounds and carcinogenic PAHs would be subtracted from the chromatogram and evaluated separately using their specific RfDs or SFs.

The following example uses simple point estimates for exposure parameters as inputs to the dosage calculations. Alternatively, a probabilistic analysis can be performed. The cumulative cancer and noncancer risks associated with the 95th percentile estimate of exposure should be presented as specified in the MCP (30 CMR Subpart I, 40.0993 (5) (c)). Use of probability analyses in risk characterization under the MCP is discussed in "Guidance for Disposal Site Risk Characterization" (MADEP, 1994).

Using the analytical technique described in the previous section, the mass of alkanes and cycloalkanes in each specified region of the chromatogram is quantified. That mass is converted to a concentration in soil (Line 1 of Figure 5). For each region, the concentration of petroleum hydrocarbon is entered into the appropriate exposure equation. In this example, we have selected a child soil ingestion exposure scenario. Thus, the concentration of petroleum hydrocarbons, in units of mg/kg would be entered into the following equation:

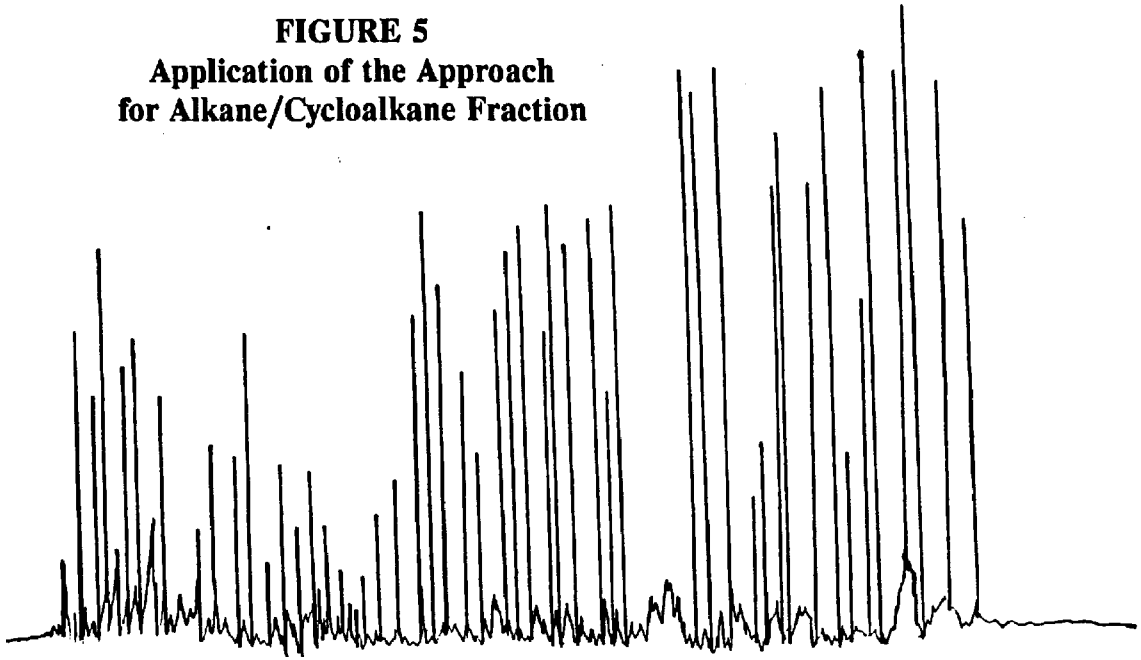
$$ADD_{soi} = \frac{[PHC]_{so} \ x \ I \ x \ BAF \ x \ D_1 \ x \ D_2 \ x \ F \ x \ C}{Bw_{avg} \ x \ AP}$$

Where:

$[PHC]_{so}$	-	Representative concentration of petroleum hydrocarbons in the soil at the exposure point during the period of exposure (mg/kg)
I	-	Daily soil ingestion rate on days exposed during the exposure period (Assume 200 mg/day).
BAF	-	Bioavailability Adjustment Factor (Assume 1)

FIGURE 5
Application of the Approach for Alkane/Cycloalkane Fraction

Hypothetical Gas Chromatogram for the Alkane/Cycloalkane Fraction



S))))))

D_1	-	Average duration of each exposure event (Assume 1 day/event)
D_2	-	Duration of the exposure period (Assume 7 years)
F	-	Number of exposure events during the exposure period divided by the number of days in the exposure period (Assume 1 event/day).
C	-	Appropriate units conversion factor(s)
BW_{avg}	-	Average body weight of the receptor of concern during the averaging period (Assume 16 kg).
AP	-	Averaging Period (Assume 7 years)

In this example, the intake of C5 through C8 hydrocarbons ingested by a child is calculated as 2.5×10^{-4} mg/kg/day (Line 2 of Figure 4). A hazard quotient is then calculated for each fraction of petroleum hydrocarbons by dividing the average daily dose (ADD_{soi}) for each fraction by the proposed alternate Reference Dose identified for that fraction (listed in Table 5 and Line 3 of Figure 4). The hazard quotients (Line 4) are summed together (Line 5) to arrive at a hazard index for the petroleum hydrocarbons detected in the chromatograph.

$$\text{Hazard Index} = ADD_1/ARfD_1 + ADD_2/ARfD_2 + ... + ADD_i/ARfD_i$$

Where:

$ARfD_i$	-	The alternate reference dose for exposure to fraction i.
ADD_i	-	The daily dose of fraction i via the particular exposure route

The hazard index may be summed with hazard indices from other relevant exposure pathways. The total hazard index is then compared to the target hazard index identified under the MCP. If the hazard index is found to exceed one, then the hazard indices should be separated by effect and mechanism of action. For example, while the effects of the C5-C8 and C9-C18 alkanes/cycloalkanes are associated with neurotoxicity, those compounds larger than C18 are not associated with neurotoxic effect, so that two hazard index can be considered separately.

BWSC is considering, as an alternative to the reporting approach suggested above, requiring laboratories to report one TPH value that would incorporate the relative toxicity of the various fractions. BTEX compounds, MTBE, and the carcinogenic PAHs would not be included in this TPH value, and would be evaluated separately. Thus, the noncarcinogenic PAHs and alkenes (C9 through C32), having the lowest RfD, would be assigned a relative

toxicity of one. Alkanes and cycloalkanes C5 through C8 would be assigned a relative toxicity of 0.5; C9 through C18, 0.05; and C19 through C32, 0.005.

This total weighted TPH value would be run through the risk calculations as described above.

The following presents an example of the application of the use of a "total weighted TPH value".

TPH Range	Concentration in Soil (mg/kg)	Toxicity Weighting Factor	Toxicity-weighted TPH (mg/kg)
Alkanes/Cycloalkanes			
C5 - C8	200	0.5	100
C9 - C18	5000	0.05	250
C19 - C32	1500	0.005	7.5
Alkenes/Aromatics			
C9 - C32	1200	1	1200
Total	7900	-	1557.5

The total toxicity-weighted TPH value (1557.5 mg/kg) is used in risk calculations with the reference dose value for noncarcinogenic PAHs (0.03 mg/kg/day). For example, using the exposure assumptions described in Figure 4 and the toxicity-weighted TPH value of 1558 mg/kg calculated above, a child's dosage as a result of soil ingestion of 0.02 mg/kg/day of TPH is calculated. Comparison of this dosage to the RfD results in a hazard index of 0.65.

6.0 UNCERTAINTY ANALYSIS

The approach described in this document is based on numerous assumptions. Each of these assumptions is associated with some degree of uncertainty. These uncertainties may result in either an over- or under-estimation of actual risks at specific sites. A discussion of the significant uncertainties incorporated in this approach are described below.

Uncertainties in Sampling and Analysis - Analytical requirements are only preliminarily described in this document. Uncertainties can result from error inherent in the sampling and analysis procedures, from a failure to take an adequate number of samples, from mistakes on the part of the sampler, from heterogeneity of the matrix being sampled, or from intentional bias in sample collection at each site. Interpretation of the chromatogram is also difficult.

The methodology presented in this document was designed to overcome the interpretive difficulties posed by weathering of oil in the environment and to minimize the effects of multi-product contamination on the interpretation of petroleum hydrocarbon analyses. By dividing the gas chromatogram into sections that correspond to empirically derived carbon number ranges, the necessity to know the identity of the original product, or how many different petroleum products are contributing to the total concentration of an individual chemical or range, is eliminated. However, there is some uncertainty in deciding the exact point of demarcation between compounds within a carbon number range. For example, there may be some overlap between the retention time of a straight chain hydrocarbon and a branched hydrocarbon that differ by only one carbon. However, the analytical methodology will be developed to minimize these potential difficulties. The contribution of an individual compound to the total concentration of a range of compounds is likely small and therefore should not make a significant difference in calculating the concentration of hydrocarbons within a particular carbon number range. The information needed from the petroleum analysis for the purposes of health risk assessment is the concentration of hydrocarbons in each range and is available from these methods.

Use of Ranges of Compounds, Rather Than Whole Product - Sufficient testing has been performed on some whole products (gasoline, and Nos. 4 and 5 jet fuel) to allow USEPA to develop provisional reference dose values and slope factors. One approach to assessing petroleum site risks is to apply whole product toxicity values to weathered products. Such an approach does not recognize the changes in composition that occur once a product is released to the environment. Those compounds that are responsible for the measured

toxicity of the whole product may no longer be present or are likely at reduced concentrations in weathered samples. By identifying the mass of hydrocarbons in smaller portions of the chromatograph and estimating the toxicity of these smaller groups of compounds, the changes in the composition of a product are acknowledged and reflected in the toxicity evaluation. Thus, this approach should reduce uncertainty in comparison to a whole product approach.

In keeping with USEPA guidance (USEPA, 1986), the proposed approach adds hazard indices and cancer risks across chemicals and media for each receptor. This is to say, if an individual is exposed to petroleum hydrocarbons by ingestion of and dermal contact with soils, and by the ingestion of water contaminated with petroleum hydrocarbons, the hazard indices or cancer risks are added together to obtain a total hazard index or risk for a receptor. Thus, a total hazard index or cancer risk is obtained which reflects the toxic effect of the entire weathered product to a receptor. This additive approach assumes independence of action and if incorrect, could result in over- or under-estimation of the actual risk.

Grouping of Alkenes with Aromatics. In this approach, alkenes are evaluated similarly to aromatics. However, since a compound-specific approach is being used for C6 through C8 aromatics, C6 through C8 alkenes and cycloalkenes will therefore not be quantified or evaluated. This approach therefore underestimates risk for the C6 through C8 alkenes. Alkenes larger than C8 are assumed to have a level of toxicity of similar magnitude to the aromatics. Because all alkenes in refined petroleum products make up from 1 to 5 percent of total hydrocarbons, it is not anticipated that this approach will excessively bias the risk estimate.

Use of Reference Compounds - Reference compounds were identified for particular groups of compounds. The toxicity of the other compounds within the group is assumed to be the same as the reference compound. It is possible that the toxicity of other compounds within a group is of greater or lesser toxicity than the reference compound and, therefore, risk would be under- or over-estimated. A review of oral RfDs established by USEPA for some petroleum hydrocarbons (Table 2), as well as the provisional RfDs developed for whole products such as gasoline and jet fuel (Table 3) reveals that the levels of toxicity of many petroleum hydrocarbons, as well as whole products, are of a similar order of magnitude. In addition, the compound-specific approach suggested for the carcinogenic compounds (benzene and the carcinogenic PAHs) should account for the most toxic constituents. Thus, it does not appear that this approach will significantly under-estimate risk.

It has been suggested that for the alkanes/cycloalkanes, an individual alternate RfD should be established for compounds with the same carbon number (e.g. C6, C7, C8 etc.), based on the apparent linear decrease in toxicity suggested by the toxicity data (as evidenced by the reference compound RfDs of 0.06, 0.6 and 6 mg/kg/day). BWSC believes that the division of the alkanes/cycloalkanes into three groups appropriately reflects the degree of certainty in these values, as applied to such a wide range of compounds. Further division, without additional toxicological information, would imply a greater degree of certainty than is suggested by the available data.

To confirm the validity of the approach described in this document, an exercise was performed for a hypothetical exposure scenario to obtain risk estimates using the new approach and using whole product toxicity information for gasoline. This validation exercise is presented in Appendix C. Hazard indices obtained using the new approach were approximately three times higher than those obtained using the USEPA provisional RfD for gasoline. This difference may be explained in part by the fact that the gasoline RfD is based on an inhalation study that exposed animals to aerosolized gasoline, while the component approach used oral studies.

The cancer risk estimates are two and one-half to five times lower than those predicted using the gasoline SF. The discrepancy in the cancer risk estimate may be explained by the fact that the new approach is driven solely by the toxicity of benzene. The cancer slope factor for benzene is based on human exposures, while the gasoline slope factor is based on an animal study. In the IRIS file for benzene, USEPA comments that toxicity values based on animal gavage studies for benzene are about five times higher than those derived from human data. The agreement in risk estimates (within an order of magnitude) obtained in this exercise is considered acceptable.

Compound specific composition data on other products having toxicity values is currently being sought. Since it is impossible to quantify the numerous individual hydrocarbons which occur in a product and also impossible to determine the toxicity of each of these compounds, use of reference compounds to represent the toxicity of a group is preferable to not evaluating a group of compounds at all, or of applying a criterion with no health basis (such as the TPH parameter).

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APPENDIX A

WHOLE PRODUCT COMPOSITION

TABLE A-1
COMPOSITION DATA FOR VARIOUS GASOLINES[a]

Hydrocarbon	Leaded [b]	Unleaded [b]	Super Unleaded [b]
n-Alkanes			
C_5	2.2	3.0	1.9
C_6	11.0	11.6	12.9
C_7	2.3	1.2	0.2
C_9	0.8	0.7	0.4
C_{10}-C_{13}	0.6	0.8	0.2
Branched Alkanes			
C_4	1.6	2.2	1.2
C_5	17.3	15.1	8.6
C_6	9.7	8.0	6.2
C_7	2.7	1.9	1.4
C_8	2.0	1.8	8.7
C_9	2.7	2.1	1.2
C_{10}-C_{13}	0.5	1.0	1.1
Cycloalkanes			
C_6	3.9	3.0	3.0
C_7	1.0	1.4	0.2
C_8	0.6	0.6	0.2
Olefins			
C_6	1.1	1.8	1.0
Aromatics			
Benzene	3.9	3.2	4.4
Toluene	4.5	4.8	6.0
Xylenes	5.6	6.6	7.4
Ethylbenzene	1.2	1.4	1.4
C_3-benzenes	3.4	4.2	5.7
C_4-benzenes	5.6	7.6	5.8
Others	2.0	2.7	1.6
Unknowns	7.8	6.6	13.8

a) Source: Hoag et al. 1984 via IRP, 1991
b) Percent by weight

TABLE A-2
COMPOSITION DATA FOR FUEL OILS [a]

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

a) Source: IARC, 1989d via ATSDR, 1993b
b) Percent by volume

TABLE A-3
COMPOSITION DATA FOR NO. 6 FUEL [a]

Hydrocarbon	Percent Compositon [b]
Saturated Compounds	21.1
n-paraffins	1.73
C_{13}	0.07
C_{14}	0.11
C_{15}	0.12
C_{16}	0.14
C_{17}	0.15
C_{18}	0.12
C_{19}	0.14
C_{20}	0.12
C_{21}	0.11
C_{22}	0.10
C_{23}	0.09
C_{24}	0.08
C_{25}	0.07
C_{26}	0.05
C_{27}	0.04
C_{28}	0.05
C_{29}	0.04
C_{30}	0.04
C_{31}	0.04
C_{32} plus	0.05
Isoparaffins	5.0
1-ring cycloparaffins	3.9
2-ring cycloparaffins	3.4
3-ring cycloparaffins	2.9
4-ring cycloparaffins	2.7
5-ring cycloparaffins	1.9
6-ring cycloparaffins	0.4
Aromatics	34.2
Benzenes	1.9
Indans and tetralins	2.1
Dinaphthenobenzenes	2.0

TABLE A-3
COMPOSITION DATA FOR NO. 6 FUEL [a]

Hydrocarbon	Percent Compositon [b]
Other	
Methylnaphthalenes	2.6
Acenaphthenes	3.1
Acenaphthalenes	7.0
Phenanthrenes	11.6
Pyrenes	1.7
Benzothiophenes	1.5
Dibenzothiophenes	0.7
Metals	
Nickel (ppm)	89
Vanadium (ppm)	73

[a]Source: Pancirov et al, 1980
[b]Percent by weight

TABLE A-4
COMPOSITION DATA FOR JP-4 [a]

Hydrocarbon	Percent Composition [b]	Hydrocarbon	Percent Composition[b]
Alkanes		Cycloalkanes	
n-butane	0.12	methylclopentante	1.16
isobutane	0.66	trans-2,3-dimethylcylopentane	0.36
2,2,3,3-tetramethylbutane	0.24	cic-1,3-dimethylcylopentane	0.34
n-pentane	1.06	cic-1,2-dimethylcylopentane	0.54
2,2-dimethylbutane	0.10	ethylcyclopentane	0.26
2-methylpentane	1.28	1,2,4-trimethylcyclopentane	0.25
3-methylpentane	0.89	1,2,3-trimethylcyclopentane	0.25
n-hexane	2.21	cyclohexane	1.24
2,2-dimethylpentane	0.25	methylcyclohexane	2.27
2-methylhexane	2.35	dimethylcylohexane	0.43
3-methylhexane	1.97	cis-1,3-dimethylcyclohexane	0.42
2,2-dimethylhexane	0.71	1-methyl-2-ethylcyclohexane	0.39
2,5-dimethylhexane	0.37	1-methyl-3-ethylcyclohexane	0.17
2,4-dimethylhexane	0.58	1-methyl-4-ethylcyclohexane	0.48
3,3-dimethylhexane	0.26	1,3,5-trimethycyclohexane	0.99
n-heptane	3.67	1,1,3-trimethycyclohexane	0.48
2-methylheptane	2.70	n-butylcyclohexane	0.70
3-methylheptane	3.04	Total of major cycloalkanes	10.73
4-methylheptane	0.92		
2,5-dimethylheptane	0.52	Alkylbenzenes	
3,4-dimethylheptane	0.43	benzene	0.50
4-ethylheptane	0.18	toluene	1.33
n-octane	3.80	ethylbenzene	0.37
2-methyloctane	0.88	o-xylene	1.01
3-methyloctane	0.79	m-xylene	0.96
4-methyloctane	0.86	p-xylene	0.35
n-nonane	2.25	1,2,4-trimethylbenzene	1.01
n-decane	2.16	1,3,5-trimethylbenzene	0.42
n-undecane	2.32	1-methyl-2-ethylbenzene	0.23
2-methylundecane	0.64	1-methyl-3-ethylbenzene	0.49
2,6-dimethylundecane	0.71	1-methyl-4-ethylbenzene	0.43
n-dodecane	2.00	isopropylbenze	0.30
n-tridecane	1.52	n-propylbenzene	0.71

TABLE A-4
COMPOSITION DATA FOR JP-4 [a]

Hydrocarbon	Percent Composition [b]	Hydrocarbon	Percent Composition[b]
n-tetradecane	0.73	1,3-diethylbenzene	0.46
Total of major alkanes	43.17	1,3-dimethyl-5-ethylbenzene	0.61
		1-methyl-4-propylbenzene	0.40
Naphthalenes		1-methyl-2-isopropylbenzene	0.29
naphthalene	0.50	1,2-dimethyl-4-ethylbenzene	0.77
1-methylnaphthalene	0.78	1,4-dimethyl-2-ethylbenzene	0.70
2-methylnaphthalene	0.56	1,2,3,4-tetramethylbenzene	0.75
2,6-dimethylnaphthalene	0.25	Total of major alkylbenzenes	12.09
Total of major naphthalenes	2.09		
Other identified components			
dicycloparaffins			
indans			
tetralins			
olefins			

[a]Source: Air Force, 1981 via ATSDR, 1993c
[b]Percent by weight

APPENDIX B

ACUTE TOXICITY DATABASE

TABLE B-1
ACUTE TOXICITY DATA BASE

COMPONENT	TEST ANIMAL	TEST	DURATION	CONCENTRATION	UNITS	REFERENCE
C-4						
n-Butane	Rat	LC50-ihl	4 hour	658	g/m3	Sax, 1984
	Mouse	LC50-ihl	2 hour	680	g/m3	RTECS, 1992
Isobutane	mouse	LC50-ihl	1 hour	52	mg/kg	Patty, 1981
Isobutuene	rat	LC50-ihl	4 hour	620	g/m3	Sax, 1984
	mouse	LC50-ihl	2 hour	415	g/m3	Sax, 1984
C-5						
n-Pentane	Mouse	LD50-ivn	ns	446	mg/kg	Sax, 1984
Isopentane	mouse	LC50-ihl	ns	1000	mg/L	IRP
C-6						
Benzene	Rat	LD50-orl	ns	3800	mg/kg	Sax, 1984
	Rat	LD50-orl	ns	5600	mg/kg	Patty, 1981
	Rat	LD50-orl	ns	930	mg/kg	RTECS, 1992
	Rat	LD50-ipl	ns	2.89	mg/kg	RTECS, 1992
	Rat	LC50-ihl	7 hours	10000	ppm	Sax, 1984
	Rat	LC50-ihl	4 hours	16000	ppm	Patty, 1981
	Mouse	LD50-orl	ns	4700	mg/kg	Sax, 1984
	Mouse	LC50-ihl	ns	9980	ppm	Sax, 1984
	Mouse	LD50-ipr	ns	990	ug/kg	Sax, 1984
	Mouse	LD50-ipr	ns	340	mg/kg	RTECS, 1992
n-Hexane	Rat	LD50-orl	ns	28.7	mg/kg	Sax, 1984
	Rat	LD50-oral	ns	49	mL/kg	IRP
	Rat	LC50-ihl	4 hour	33000	ppm	IRP
2-Methylpentane	Rabbit	LD50-oral	ns	5.5	g/kg	IRP
2-Methylpentene	Rat	LC50-ihl	4 hour	115	g/m3	Sax, 1989
	Mouse	LC50-ihl	2 hour	127	g/m3	Sax, 1989
Cyclohexane	Rat	LD50-orl	ns	29820	mg/kg	Sax, 1984
	Rat	LD50-orl	ns	12705	mg/kg	RTECS, 1992
	Mouse	LD50-orl	ns	813	mg/kg	RTECS, 1992
Methylcyclopentane	Mouse	LCLo-ihl	ns	95000	mg/m3	RTECS, 1992
C-7						
n-Heptane	Mouse	LD50-ivn	ns	222	mg/kg	Sax, 1984
	Mouse	LC50-ihl	2 hour	75	g/m3	RTECS, 1992
Methylcyclohexane	rat	LD50-orl	ns	2250	mg/kg	IRP
	Mouse	LC50-ihl	2 hours	41500	mg/m3	RTECS, 1992

TABLE B-1
ACUTE TOXICITY DATA BASE

COMPONENT	TEST ANIMAL	TEST	DURATION	CONCENTRATION	UNITS	REFERENCE
Toluene	Rat	LD50-orl	ns	5000	mg/kg	Sax, 1984
	Rat	LD50-orl	ns	636	mg/kg	RTECS, 1992
	Rat	LD50-orl	ns	6.4	ml/kg	Patty, 1981
	Rat	LD50-orl	ns	7	mg/kg	Patty, 1981
	Rat	LD50-orl	ns	7.4	mg/kg	Patty, 1981
	Rat	LD50-orl	ns	7.53	ml/kg	Patty, 1981
	Rat	LD50-ipr	ns	800	mg/kg	Patty, 1981
	Rat	LD50-ipr	ns	1640	mg/kg	Patty, 1981
	Rat	LD50-ipr	ns	1332	mg/kg	RTECS, 1992
	Rat	LD50-inv	ns	1960	mg/kg	RTECS, 1992
	Rat	LC50-ihl	4 hours	8000	ppm	Patty, 1981
	Rat	LC50-ihl	4 hours	8800	ppm	Patty, 1981
	Mouse	LD50-ipr	ns	1.12	mg/kg	Sax, 1984
	Mouse	LD50-ipr	ns	59	mg/kg	RTECS, 1992
	Mouse	LC50-ihl	8 hours	5320	ppm	Sax, 1984
	Mouse	LC50-ihl	24 hours	400	ppm	RTECS, 1992
	rabbit	LD50-skn	ns	14	g/kg	Sax, 1984
	rat	LD50-unk	ns	6900	mg/kg	Sax, 1984
	mouse	LD50-unk	ns	2000	mg/kg	Sax, 1984
	rabbit	LD50-skn	ns	12124	mg/kg	IRP
C-8						
Xylenes	Rat	LD50-orl	ns	4300	mg/kg	Sax, 1984
	Rat	LD50-orl	ns	10	ml/kg	Patty, 1981
	Rat	scu-LD50	ns	1700	mg/kg	Sax, 1984
	Rat	LD50-ipl	ns	2459	mg/kg	RTECS, 1992
	Mouse	LD50-ipr	ns	1.57	mg/kg	Sax, 1984
	Mouse	LD50-ipl	ns	1548	mg/kg	RTECS, 1992
	Rat	LC50-ihl	4 hours	5000	ppm	Sax, 1984
o-Xylene	Rat	LC50-ihl	4 hours	6350	ppm	Patty, 1981
	Rat	LC50-ihl	4 hours	6700	ppm	Patty, 1981
	Mouse	LD50-ipl	ns	1364	mg/kg	RTECS, 1992
m-Xylene	Rat	LD50-orl	ns	5000	mg/kg	Sax, 1984
	Mouse	LD50-ipl	ns	1739	mg/kg	RTECS, 1992
p-Xylene	Rat	LD50-orl	ns	5000	mg/kg	Sax, 1984
	Rat	LD50-ipl	ns	3810	mg/kg	RTECS, 1992

TABLE B-1
ACUTE TOXICITY DATA BASE

COMPONENT	TEST ANIMAL	TEST	DURATION	CONCENTRATION	UNITS	REFERENCE
	Rat	LC50-ihl	4 hours	4550	ppm	RTECS, 1992
	Mouse	LD50-ipl	ns	2110	mg/kg	RTECS, 1992
Ethylbenzene	rat	LD50-orl	ns	3500	mg/kg	Sax, 1984
	rat	LD50-orl	ns	5.46	ml/kg	Patty, 1981
	Mouse	LD50-ipl	ns	2272	mg/kg	RTECS, 1992
	rabbit	LD50-skn	ns	5000	mg/kg	Sax, 1984
	rabbit	LD50-skn	ns	17.8	ml/kg	Patty, 1981
Octane	Mouse	LDlo-ivn	ns	428	mg/kg	OHS MSDS, 1992
C-9						
n-Nonane	Mouse	LD50-ivn	ns	218	mg/kg	Sax, 1984
	Rat	LC50-ihl	4 hour	3200	ppm	Patty, 1981
	Rat	LC50-ihl	8 hour	4467	ppm	Nilsen et al,1988
1,2,4-Trimethylbenzene	rat	LC50-ihl	4 hour	18	g/m3	RTECS, 1992
	rat	LD50-orl	ns	5000	mg/kg	RTECS, 1992
1,3,5-Trimethylbenzene	rat	LC50-ihl	4 hours	24	g/m3	RTECS, 1992
Trimethylbenzene	Rat	LD50-orl	ns	8970	mg/kg	OHS MSDS, 1992
Isopropylbenzene	rat	LD50-orl	ns	1.4	g/kg	Patty, 1981
	rat	LC50-ihl	4 hours	8000	ppm	Patty, 1981
	Mouse	LD50-orl	ns	12750	mg/kg	RTECS, 1992
1-ethyl 2-methyl toluene	Mouse	LC50-ihl	4 hours	54	g/m3	Sax, 1989
	Cat	LC50-ihl	2 hours	50	g/kg	Sax, 1989
C-10						
n-Decane	Mouse	TDLo-skn	52 wk/Int	25	g/kg	Sax, 1984
	Rat	LC50-ihl	8 hour	>1369	ppm	Nilsen,1988
	Mouse	LC50-ihl	2 hour	72300	mg/m3	OHS MSDS, 1992
1,2,3,4-Tetramethlybenzene	rat	LD50-orl	ns	6408	mg/kg	Sax, 1984
1,2,3,5-Tetramethlybenzene	rat	LD50-orl	ns	5157	mg/kg	Sax, 1984
1,2,4,5-Tetramethlybenzene	rat	LD50-orl	ns	6989	mg/kg	Sax, 1984
Naphthalene	rat	LD50-orl	ns	1780	mg/kg	Sax, 1984
	Rat	LD50-orl	ns	490	mg/kg	RTECS, 1992
	Mouse	LD50-orl	ns	533	mg/kg	RTECS, 1992
	Mouse	LD50-ipr	ns	150	mg/kg	Sax, 1984
	Mouse	LD50-scu	ns	969	mg/kg	Sax, 1984
	Mouse	LD50-scu	ns	5.1	g/kg	Patty, 1981
	Mouse	LD50-ivn	ns	100	mg/kg	Sax, 1984

TABLE B-1
ACUTE TOXICITY DATA BASE

COMPONENT	TEST ANIMAL	TEST	DURATION	CONCENTRATION	UNITS	REFERENCE
	Mouse	LD50-gav	8 days	354	mg/kg	ATSDR, 1990
	Guinea Pig	LD50-orl	ns	1200	mg/kg	RTECS, 1992
C-11						
n-Undecane	Mouse	LD50-ivn	ns	517	mg/kg	Sax, 1984
1-Methylnaphthalene	rat	LDLo-orl	ns	5000	mg/kg	Sax, 1984
	rat	LD50-orl	ns	1840	mg/kg	IRP
2-Methylnaphthalene	rat	LDLo-orl	ns	5000	mg/kg	Sax, 1984
	rat	LD50-orl	ns	1630	mg/kg	IRP
C-12						
n-Dodecane	Mouse	TDLo-skn	22 wk/Int	11	g/kg	Sax, 1984
Triethylbenzene	rat	LDLo-orl	ns	5000	mg/kg	Sax, 1984
Acenapthene	Rat	LD50-ipl	ns	600	mg/kg	OHS MSDS, 1992
Acenapthylene	Rat	LD50-ipl	ns	1700	mg/kg	RTECS, 1992
C-13						
n-Tridecane	Mouse	LD50-ivn	ns	1161	mg/kg	Sax, 1984
	Rat	LC50-ihl	8 hour	>41	ppm	Nilsen et al, 1988
Fluorene	Mouse	LD50-ipl	ns	2000	mg/kg	OHS MSDS, 1992
C-14						
Tetradecane	Mouse	LDlo-ivn	ns	5800	mg/kg	RTECS, 1992
Anthracene	rat	TDLo-orl	79 weeks-I	20	g/kg	Sax, 1984
	Mouse	LD50-ipl	ns	430	mg/kg	OHS MSDS, 1992
Phenanthrene	Mouse	LD50-orl	ns	700	mg/kg	Sax, 1984
	Mouse	LD50-ivn	ns	56	mg/kg	Sax, 1984
C-15						
Pentadecane	Mouse	LD50-ivn	ns	3494	mg/kg	Sax, 1989
C-16						
Pyrene	Rat	LD50-orl	ns	2700	mg/kg	Sax, 1989
	Mouse	LD50-orl	ns	800	mg/kg	OHS MSDS, 1992
	Mouse	LD50-ipl		514	mg/kg	OHS MSDS, 1992
	Rat	LC50-ihl	ns	170	mg/m3	OHS MSDS, 1992
Fluoranthene	Rat	LD50-orl	ns	2000	mg/kg	Sax, 1989
	Mouse	LD50-ivn	ns	100	mg/kg	Sax, 1989
	Rabbit	LD50-skin	ns	3180	mg/kg	Sax, 1989
Hexadecane	Mouse	LDlo-ivn	ns	9821	mg/kg	RTECS, 1992

TABLE B-1
ACUTE TOXICITY DATA BASE

COMPONENT	TEST ANIMAL	TEST	DURATION	CONCENTRATION	UNITS	REFERENCE
C-17						
Heptadecane	Mouse	LDlo-ivn	ns	9821	mg/kg	RTECS, 1992
C-20						
Benzo(a)pyrene	Rat	LD50-sbcu	ns	50	mg/kg	OHS MSDS, 1992

Notes:
gav = gavage exposure
ihl = inhalation exposure
ivn = intravenous exposure
ipl = intraperitoneal exposure
orl = oral exposure
sbcu = subcutaneous exposure
skn = dermal exposure
ns = not specified

APPENDIX C

VALIDATION BY COMPARISON WITH WHOLE PRODUCT TOXICITY

1.0 INTRODUCTION

A validation exercise was conducted to determine whether the components approach described in this document to estimate the toxicity of petroleum hydrocarbon mixtures (TPH) appropriately predicts the toxicity of a known hydrocarbon product, eg; gasoline. In this exercise, risk estimates for a hypothetical exposure scenario are calculated for ranges of petroleum hydrocarbons in gasoline using the toxicity values and the approach developed in Section 3 of this document. These risk estimates are compared to estimates calculated using whole product toxicity values (the provisional RfD and SF for gasoline developed by USEPA, 1992a) and a total TPH value for gasoline.

Data are not yet available using the analytical technique described in Section 4 of this document to obtain the mass of hydrocarbons in each of the ranges of TPH in environmental samples (i.e., soil, water, etc.). However, data are available on the weight percent of component petroleum hydrocarbons in whole, virgin gasoline (API, 1994). This information can be used to estimate the concentration of petroleum hydrocarbons in the carbon chain ranges (e.g., C5 - C8 alkanes/cycloalkanes) identified in this document.

2.0 METHOD

As a preliminary validation exercise, individual component hydrocarbons in two unleaded gasoline products (API PS-6 and API 91-1) were separated into the ranges of hydrocarbons defined in Section 3 of the attached document. Detailed composition data for these gasolines are presented as Attachment C-1 to this appendix. It was assumed that these gasolines were each newly spilled onto soil resulting in 100 mg/kg TPH, with the identical composition of the virgin fuel. Consistent with the described approach, benzene, toluene, ethylbenzene and xylene are quantified and evaluated individually. Table C-1 presents the composition of the gasoline, divided into the groups developed in this document.

As can be seen, the total concentration for neither gasoline product adds up to 100 mg/kg. This is because:

- C5 - C8 alkenes/cycloalkenes, which are not accounted for in this approach, make up 13.7% and 9.8%, respectively of PS-6 and 91-1 gasolines.

TABLE C-1
Composition of Two Gasoline Products,
Assuming a Total TPH of 100 mg/kg

Petroleum Hydrocarbon Carbon Ranges and Groups	Concentration in API PS-6[a] (mg/kg)	Concentration in API 91-1[a] (mg/kg)
C5 - C8 Alkanes/Cycloalkanes	41.006	38.78
C9 - C18 Alkanes/Cycloalkanes	8.725	3.16
C19 - C32 Alkanes/Cycloalkanes	ND[b]	ND[b]
C9 - C32 Aromatics/Alkenes	15.81	18.51
Benzene	2.12	1.22
Toluene	3.97	6.94
Ethyl Benzene	1.87	3.42
Xylene	5.945	10.19
Total	79.446	82.22

Notes:
[a)] Gasoline compositional data supplied by API (1994)
[b)] None detected.

- The analysis of the fuel could not identify all individual compounds. For PS-6 and 91-1 gasolines, 6.3% and 8%, respectively were identified by the laboratory as, for example, olefin, unidentified C8's, or paraffin. These unknowns were classified as miscellaneous and were not included in this exercise. When the proposed analytical method is used to analyze samples, these miscellaneous compounds will be quantified as part of the ranges of compounds. Thus, this validation exercise underestimates the risks that the new method will predict.

The concentrations were entered into risk calculations, using the same exposure assumptions as were used in Section 5 of this document, Application of the Proposed Approach. The spreadsheets that were used to perform these calculations are presented in Attachment C-2 as Tables 1 and 2 for PS-6 and 91-1 gasolines, respectively. Pathway 1 in each table presents the risk calculations using the toxicity values assigned to the various ranges of compounds developed in this document. Pathway 2 uses the total 100 mg/kg TPH and the provisional toxicity values for gasoline developed by USEPA (USEPA, 1992a).

3.0 RESULTS

Table C-2 presents the results of the risk evaluation. Hazard indices calculated using the new approach are approximately three times higher than those calculated using the whole product provisional RfD. This difference may be explained in part by the fact that the gasoline RfD is based on an inhalation study that exposed animals to aerosolized gasoline, while the component approach used oral studies.

The cancer risk estimates are two and one-half to five times lower than those predicted using the gasoline SF. The discrepancy in the cancer risk estimate may be explained by the fact that the new approach is driven solely by the toxicity of benzene. The cancer slope factor for benzene is based on human exposures, while the gasoline slope factor is based on an animal study. In the IRIS file for benzene, USEPA comments that toxicity values based on animal gavage studies for benzene are about five times higher than those derived from human data.

The values predicted by the two methodologies are within an order of magnitude (10-fold) of one another which is considered to be acceptable agreement.

Table C-2
Risk Estimates for Ingestion Exposure
to 100 mg/kg TPH in Soil[a]

	Hazard Index		Cancer Risk	
	New Approach	Whole Product	New Approach	Whole Product
API PS-6	0.02	0.006	8×10^{-8}	2×10^{-7}
API 91-1	0.02	0.006	4×10^{-8}	2×10^{-7}

Notes:
[a)] Assumes a 16 kg child consumes 200 mg soil per day, 365 days per year

ATTACHMENT C-1

COMPOSITION DATA FOR TWO GASOLINE PRODUCTS

CONSCI

American Petroleum Institute
1220 L Street Northwest
Washington D.C., 20005

January 13, 1994

Attn: Chris Sexsmith

Invoice #: 31207009

Certificate #: 31214002
Sample ID: PS-6 Gasoline
Date Received: December 14, 1993

CERTIFICATE OF ANALYSIS

Component Name	Wt. %	LV %
Propane	0.02	0.03
Isobutane	0.60	0.79
Isobutylene + 1-Butene	0.09	0.11
n-Butane	3.05	3.82
trans-2-Butene	0.16	0.19
cis-2-Butene	0.1[illegible]	0.20
3-Methyl-1-butene	0[illegible]	0.07
Isopentane	[illegible].5	9.40
1-Pentene	[illegible].18	0.21
2-Methyl-1-butene	0.40	0.45
n-Pentane	3.29	3.85
trans-2-Pentene	0.51	0.58
3,3-Dimethyl-1-butene	0.84	0.94
cis-2-Pentene	0.29	0.32
2,2-Dimethylbutane + Cyclopentadiene + cis-1,3-Pentadiene	0.00	0.00
Cyclopentene	0.08	0.08
4-Methyl-1-pentene	0.03	0.03
3-Methyl-1-pentene	0.05	0.06
Cyclopentane	0.24	0.24
2,3-Dimethylbutane	1.84	2.04
2,3-Dimethyl-1-butene	0.09	0.10
4-Methyl-cis-2-pentene	0.09	0.10
2-Methylpentane	4.04	4.54

CERTIFICATE OF ANALYSIS

Component Name	Wt. %	LV %
trans-3-Hexene	0.07	0.08
trans-2-Hexene	0.36	0.40
2-Methyl-2-pentene	0.33	0.36
3-Methylcyclopentene	0.08	0.08
3-Methyl-cis-2-pentene	0.36	0.39
4-Methylcyclopentene	0.05	0.05
3-Methyl-trans-2-pentene	0.21	0.22
cis-2-Hexene	0.42	0.46
2,2-Dimethylpentane	0.08	0.09
Methylcyclopentane	1.09	1.08
2,4-Dimethylpentane	0.68	0.75
C7 Olefins + C7 Cyclo-olefin/diolefins	0.11	0.11
1-Methylcyclopentene	0.38	0.36
Benzene	1.22	1.03
3,3-Dimethylpentane	0.12	0.13
Cyclohexane	0.22	0.21
C7 Olefin	0.02	0.02
C7 Olefin	0.06	0.06
C7 Olefin	0.02	0.02
C7 Olefin	0.05	0.05
2-Methylhexane	1.78	1.95
2,3-Dimethylpentane	1.31	1.40
1,1-Dimethylcyclopentane	0.03	0.03
C7 Olefin	0.04	0.04
3-Methylhexane	1.76	1.91
C7 Olefin	0.04	0.04
t-1,3-Dimethylcyclopentane	0.32	0.32
c-1,3-Dimethylcyclopentane	0.28	0.28
3-Ethylpentane + C7 olefin	0.24	0.26
t-1,2-Dimethylcyclopentane	0.26	0.26
2,2,4-Trimethylpentane + C7 olefin	1.87	2.01
C7 Olefin	0.06	0.06
C7 Olefin	0.14	0.15

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Component Name	Wt. %	LV %
n-Heptane	1.36	1.48
C7 Olefin	0.14	0.15
C7 Olefin	0.25	0.26
C7 Olefin	0.11	0.12
C7 Olefin	0.13	0.14
C7 Olefin	0.06	0.06
C7 Olefin	0.13	0.14
Olefin	0.14	0.15
Olefin	0.07	0.07
Olefin	0.06	0.06
Olefin	0.01	0.01
Olefin	0.01	0.01
c-1,2-Dimethylcyclopentane	0.16	0.15
Methylcyclohexane	0.44	0.43
2,2-Dimethylhexane	0.06	0.06
1,1,3-Trimethylcyclopentane	0.07	0.07
Olefin	0.01	0.01
Olefin	0.03	0.03
Ethylcyclopentane + 2,5-Dimethylhexane	0.49	0.50
2,2,3-Trimethylpentane + 2,4-Dimethylhexane	0.57	0.60
1,2,4-Trimethylcyclopentane	0.13	0.13
3,3-Dimethylhexane	0.06	0.06
2,3,4-Trimethylpentane	0.79	0.82
2,3,3-Trimethylpentane	0.87	0.90
Toluene	6.94	5.97
2,3-Dimethylhexane + C8 olefin	0.41	0.43
C8 Olefin	0.11	0.11
C8 Cyclo-olefin/diolefin	0.06	0.06
2-Methylheptane	0.88	0.94
4-Methylheptane	0.38	0.40
3-Methyl-3-ethylpentane	0.14	0.14
3-Methylheptane	0.96	1.01
3-Ethylhexane + C8 olefin	0.13	0.14

CERTIFICATE OF ANALYSIS

Component Name	Wt. %	LV %
c-1,4-Dimethylcyclohexane	0.04	0.04
t-1,4-Dimethylcyclohexane	0.03	0.03
2,2,4-Trimethylhexane	0.27	0.28
C8 Naphthene	0.17	0.17
C8 Naphthene	0.12	0.12
Unidentified C8's	0.65	0.68
n-Octane	0.71	0.75
t-1,2-Dimethylcyclohexane	0.14	0.13
1,2,3-Trimethylcyclopentane	0.05	0.05
Isopropylcyclopentane + C8 Cyclo-olefin/Diolefin	0.14	0.13
C9 Paraffin	0.05	0.05
2,5 + 3,5-Dimethylheptane	0.11	0.11
C9 Paraffin	0.18	0.19
C9 Paraffin	0.23	0.24
Ethylbenzene	3.42	2.94
C9 Naphthene	0.04	0.04
m-Xylene	5.37	4.64
p-Xylene	2.18	1.89
3,4-Dimethylheptane	0.05	0.05
2-Methyloctane	0.30	0.31
4-Methyloctane	0.35	0.36
3-Ethylhexane	0.06	0.05
3-Methyloctane	0.36	0.37
3,3-Diethylpentane	0.02	0.02
C9 Naphthene	0.01	0.01
C9 Naphthene	0.03	0.03
o-Xylene	2.64	2.24
C10 Naphthene	0.04	0.04
C9 Naphthene + C9 olefin	0.05	0.05
C10 Naphthene + C9 olefin	0.08	0.07
C9 Naphthene	0.07	0.07
C10 Naphthene + C9 olefin	0.03	0.03
C9 Olefin	0.03	0.03

CERTIFICATE OF ANALYSIS

Component Name	Wt. %	LV %
Unidentified C9's	0.71	0.72
n-Nonane	0.24	0.25
C9 Olefin	0.03	0.03
C9 Olefin	0.03	0.03
C9 Naphthene	0.02	0.02
C9 Olefin	0.03	0.03
C9 Naphthene	0.02	0.02
C9 Olefin	0.01	0.01
Isopropylbenzene	0.16	0.14
Paraffin	0.05	0.05
Paraffin	0.04	0.04
Paraffin	0.03	0.03
Paraffin	0.07	0.07
n-Propylbenzene	0.77	0.67
1-Methyl-3-ethylbenzene	2.26	1.95
1-Methyl-4-ethylbenzene	1.02	0.88
C10 Paraffin	0.03	0.03
1,3,5-Trimethylbenzene	1.04	0.90
4-Methylnonane	0.05	0.05
2-Methylnonane	0.10	0.10
Paraffin	0.11	0.11
1-Methyl-2-ethylbenzene	0.66	0.56
3-Methylnonane	0.13	0.13
1,2,4-Trimethylbenzene	3.04	2.59
Unidentified C10's	0.19	0.19
n-Decane	0.05	0.05
iso-Butylbenzene	0.14	0.12
1,2,3-Trimethylbenzene	0.62	0.52
Indane	0.40	0.31
C11 Paraffin	0.02	0.02
Indene	0.04	0.03
Naphthene	0.02	0.02
C11 Paraffin	0.04	0.04
1,3-Diethylbenzene	0.22	0.19
1-Methyl-3-n-propylbenzene	0.50	0.43

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Component Name	Wt. %	LV %
1-Methyl-4-n-propylbenzene	0.30	0.26
n-Butylbenzene	0.16	0.14
1,2-Diethylbenzene	0.49	0.42
1,3-Dimethyl-5-ethylbenzene + 1,4-Diethylbenzene	0.05	0.04
1-Methyl-2-n-propylbenzene	0.20	0.17
C11 Paraffin	0.06	0.06
Paraffin	0.08	0.08
1,4-Dimethyl-2-ethylbenzene	0.31	0.26
1,3-Dimethyl-4-ethylbenzene + C10 Indane	0.34	0.29
C10 Indane	0.07	0.06
1,2-Dimethyl-4-ethylbenzene	0.66	0.56
1,3-Dimethyl-2-ethylbenzene + C10 Indane	0.03	0.03
Unidentified C11's	0.23	0.23
Alkylbenzene	0.11	0.09
n-Undecane	0.07	0.07
1,2,4,5-Tetramethylbenzene	0.27	0.23
1,2,3,5-Tetramethylbenzene + C11 Cyclo-diolefin	0.39	0.33
C12 Paraffins	0.15	0.15
C11 Aromatics	1.19	1.04
C10 Indanes	0.67	0.55
Naphthalene	0.63	0.40
Unidentified C12's	0.28	0.26
C11 Indanes	1.10	0.91
C13 Paraffins	0.26	0.25
C12 Aromatics	0.15	0.13
C12 Indanes	0.13	0.11
Methylnaphthalenes	0.93	0.69
Unidentified Heavies	0.37	0.29
Totals	100.00	100.00

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CERTIFICATE OF ANALYSIS

	Wt. %	LV %
Paraffins	43.90	49.41
Naphthenes	4.35	4.28
Aromatics	40.74	34.64
Olefins	9.44	10.18
Unknowns	1.56	1.47
Oxygenates	< 0.01	< 0.01
Totals	100.00	99.99

Paul A. Radenheimer

PAR/ar

CONSCI

American Petroleum Institute
1220 L Street Northwest
Washington, D.C. 20005

January 13, 1994

Attn: Chris Sexsmith

Invoice #: 31207009

Certificate #: 31207009
Sample ID: API-91-1
Date Received: December 7, 1993

CERTIFICATE OF ANALYSIS

Bromine number	36.80
Reid Vapor Pressure, psi	8.28
RON (Research Octane Number)	91.80
MON (Motor Octane Number)	82.10
(R+M)/2	87.00
Carbon, wt.%	86.40
Hydrogen, wt.%	13.60
Nitrogen, ppm	51.00
Sulfur, ppm	350.00
Oxygen, wt.%	<0.10
Molecular Weight	101.00
	Vol.%
MTBE	<0.10
Methanol	<0.10
Benzene	1.03

CERTIFICATE OF ANALYSIS

Distillation, vol%/deg F @760mm	
IBP/5	95116
10/20	131/154
30/40	176/198
50/60	222/246
70/80	270/297
90/95	339/377
EP	420
Rec/Res	98.0/1.0

Paul A. Radenheimer

PAR/ar

CONSCI

American Petroleum Institute
1220 L Street Northwest
Washington, D.C. 20005

January 13, 1994

Attn: Chris Sexsmith

Invoice #: 31207009

Certificate #: 31214002
Sample ID: PS-6 Gasoline
Date Received: December 14, 1993

CERTIFICATE OF ANALYSIS

Bromine number	20.40
Reid Vapor Pressure, psi	8.92
RON (Research Octane Number)	91.70
MON (Motor Octane Number)	83.80
(R+M)/2	87.80
Carbon, wt.%	86.49
Hydrogen, wt.%	13.51
Nitrogen, ppm	10.00
Sulfur, ppm	200.00
Oxygen, wt.%	<0.10
Molecular Weight	103.00
	Vol.%
MTBE	<0.10
Methanol	<0.10
Benzene	1.77

CERTIFICATE OF ANALYSIS

Component Name	Wt. %	LV %
4-Methyl-trans-2-pentene	2.37	2.60
2-Methyl-1-pentene	0.12	0.13
1-Hexene	0.12	0.13
n-Hexane + 2-Ethyl-1-butene	1.71	1.90
cis-3-Hexene	0.10	0.11
trans-3-Hexene	0.04	0.04
trans-2-Hexene	0.21	0.23
2-Methyl-2-pentene	0.26	0.28
3-Methyl-cis-2-pentene	0.21	0.22
4-Methylcyclopentene	0.02	0.02
3-Methyl-trans-2-pentene	0.12	0.13
cis-2-Hexene	0.26	0.28
2,2-Dimethylpentane	0.05	0.05
Methylcyclopentane	1.12	1.10
2,4-Dimethylpentane	1.01	1.10
2,2,3-Trimethyl-1-butene	0.01	0.01
2,2,3-Trimethylbutane	0.07	0.07
1-Methylcyclopentene	0.16	0.15
C7 Olefin	0.02	0.02
Benzene	2.12	1.77
3,3-Dimethylpentane + C7 olefin	0.07	0.07
Cyclohexane + C7 olefin	0.14	0.13
C7 Cyclo-olefin/diolefin	0.02	0.02
C7 Olefin	0.03	0.03
2-Methylhexane	1.56	1.69
2,3-Dimethylpentane + C7 olefin	0.84	0.89
1,1-Dimethylcyclopentane	0.02	0.02
3-Methylhexane	1.50	1.60
C7 Olefin	0.03	0.03
t-1,3-Dimethylcyclopentane	0.31	0.30
c-1,3-Dimethylcyclopentane	0.27	0.27
3-Ethylpentane + C7 olefin	0.15	0.16
t-1,2-Dimethylcyclopentane	0.19	0.19
2,2,4-Trimethylpentane	5.73	6.08

CERTIFICATE OF ANALYSIS

Component Name	Wt. %	LV %
C7 Olefin	0.04	0.04
C7 Olefin	0.07	0.07
n-Heptane	0.62	0.67
C7 Olefin	0.06	0.06
C7 Olefin	0.14	0.15
C7 Olefin	0.06	0.06
C7 Olefin	0.06	0.06
C7 Olefin	0.04	0.04
C7 Olefin	0.08	0.08
C8 Olefin	0.04	0.04
C8 Olefin	0.04	0.04
C8 Olefin	0.03	0.03
C8 Olefin	0.02	0.02
c-1,2-Dimethylcyclopentane	0.16	0.15
Methylcyclohexane	0.35	0.33
2,2-Dimethylhexane	0.03	0.03
C8 Cyclo-olefin/diolefin	0.06	0.06
C8 Cyclo-olefin/diolefin	0.01	0.01
Ethylcyclopentane + 2,5-Dimethylhexane	1.20	1.21
2,2,3-Trimethylpentane + C8 olefin + 2,4-Dimethylhexane	1.24	1.29
1,2,4-Trimethylcyclopentane	0.11	0.11
3,3-Dimethylhexane	0.03	0.03
C8 Olefin	0.01	0.01
1,2,3-Trimethylcyclopentane + C8 Cyclo-olefin/diolefin	0.04	0.04
2,3,4-Trimethylpentane + C8 olefin	3.40	3.47
2,3,3-Trimethylpentane + C8 olefin	3.57	3.61
Toluene	3.97	3.36
2,3-Dimethylhexane + C8 olefin	0.90	0.93
1-Methyl-1-ethylpentane	0.08	0.08
C8 Olefin	0.02	0.02
2-Methylheptane	0.68	0.72

CERTIFICATE OF ANALYSIS

Component Name	Wt. %	LV %
4-Methylheptane	0.27	0.28
3-Methyl-3-ethylpentane + C8 olefin	0.18	0.18
3-Methylheptane	0.81	0.84
3-Ethylhexane + C8 olefin	0.13	0.13
c-1,4-Dimethylcyclohexane	0.08	0.08
t-1,4-Dimethylcyclohexane	0.06	0.06
C8 Naphthene + C8 olefin	0.05	0.05
C8 Naphthene + C8 olefin	0.01	0.01
C8 Naphthene + C8 olefin	0.06	0.06
C8 Naphthene + C8 olefin	0.02	0.02
2,2,4-Trimethylhexane	0.74	0.76
C8 Naphthene	0.12	0.12
C8 Naphthene	0.09	0.09
Unidentified C8's	0.12	0.12
n-Octane	0.37	0.39
t-1,2-Dimethylcyclohexane	0.01	0.01
1,2,3-Trimethylcyclopentane	0.10	0.09
C8 Olefin	0.02	0.02
C8 Olefin	0.01	0.01
C8 Olefin + C9 olefin	0.04	0.04
C9 Naphthene	0.01	0.01
C9 Paraffin + C8 olefin	0.13	0.13
Diolefin	0.06	0.06
C8 Olefin	0.01	0.01
C8 Olefin	0.02	0.02
C9 Paraffin	0.09	0.09
Olefin	0.04	0.04
c-1,2-Dimethylcyclohexane + C9 olefin	0.04	0.04
C9 Paraffin	0.16	0.16
Ethylcyclohexane	0.02	0.02
C9 Paraffin	0.22	0.22
C9 Paraffin	0.01	0.01
1,3,5-Trimethylcyclohexane + C9 olefin	0.01	0.01
C9 Naphthene	0.01	0.01

CERTIFICATE OF ANALYSIS

Component Name	Wt. %	LV %
C9 Naphthene + C9 olefin	0.01	0.01
Ethylbenzene	1.87	1.58
C9 Olefin	0.04	0.04
m-Xylene + C9 paraffin	5.03	4.28
p-Xylene + C9 paraffin	1.96	1.67
3,4-Dimethylheptane	0.04	0.04
2-Methyloctane	0.22	0.23
4-Methyloctane	0.31	0.32
3-Ethyloctane	0.20	0.16
3-Methyloctane	0.31	0.32
3,3-Diethylpentane	0.07	0.07
o-Xylene	2.45	2.05
C10 Naphthene	0.11	0.10
Naphthene	0.02	0.02
Naphthene	0.05	0.05
Paraffin	0.09	0.08
Naphthene	0.01	0.01
Unidentified C9's	0.07	0.07
n-Nonane	0.19	0.19
C9 Naphthene	0.05	0.05
Isopropylbenzene	0.11	0.09
C9 Naphthene	0.02	0.02
Paraffin	0.04	0.04
C10 Paraffin	0.04	0.04
C10 Paraffin	0.01	0.01
C10 Paraffin	0.05	0.05
C10 Paraffin	0.01	0.01
C10 Naphthene	0.02	0.02
C10 Paraffin	0.00	0.00
3,3-Dimethyloctane	0.07	0.07
n-Propylbenzene	0.54	0.46
1-Methyl-3-ethylbenzene	2.02	1.72
1-Methyl-4-ethylbenzene	0.89	0.76
C10 Paraffin	0.02	0.02

CERTIFICATE OF ANALYSIS

Component Name	Wt. %	LV %
1,3,5-Trimethylbenzene	1.13	0.96
4-Methylnonane	0.15	0.15
2-Methylnonane	0.12	0.12
Paraffin	0.23	0.23
1-Methyl-2-ethylbenzene	0.62	0.52
3-Methylnonane	0.13	0.13
Paraffin	0.05	0.04
Paraffin	0.02	0.02
1,2,4-Trimethylbenzene	3.36	2.82
Naphthene + olefin	0.03	0.03
Paraffin	0.02	0.02
C10 Aromatic	0.04	0.03
Unidentified C10's	0.11	0.11
n-Decane	0.18	0.18
iso-Butylbenzene	0.02	0.02
Paraffin	0.01	0.01
1,2,3-Trimethylbenzene + C10 Styrene	0.59	0.49
C11 Paraffin	0.02	0.02
C11 Paraffin	0.07	0.06
Indane	0.28	0.21
C11 Paraffin	0.14	0.14
Paraffin	0.18	0.17
C11 Paraffin	0.37	0.36
1,3-Diethylbenzene	0.14	0.12
1-Methyl-3-n-propylbenzene	0.25	0.21
1-Methyl-4-n-propylbenzene	0.22	0.19
n-Butylbenzene	0.08	0.07
1,2-Diethylbenzene	0.40	0.33
1,3-Dimethyl-5-ethylbenzene + 1,4-Diethylbenzene	0.01	0.01
C11 Paraffin	0.04	0.04
C11 Paraffin	0.30	0.30
C11 Paraffin	0.01	0.01
Paraffin	0.12	0.10

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CERTIFICATE OF ANALYSIS

Component Name	Wt. %	LV %
C11 Paraffin	0.14	0.14
1,3-Dimethyl-4-ethylbenzene + Indane	0.36	0.30
Paraffin	0.15	0.12
1,2-Dimethyl-4-ethylbenzene + Indane	0.52	0.44
Paraffin	0.16	0.13
Paraffin	0.01	0.01
Paraffin	0.02	0.02
C11 Aromatic	0.02	0.02
Unidentified C11's	0.07	0.07
1,2-Dimethyl-3-ethylbenzene	0.13	0.11
n-Undecane	0.15	0.15
Aromatic	0.01	0.01
1,2,4,5-Tetramethylbenzene	0.26	0.22
1,2,3,5-Tetramethylbenzene	0.37	0.31
Unidentified C12's	0.09	0.09
Dodecane	0.21	0.20
C12 Paraffins	0.17	0.17
C10 Indanes	0.44	0.36
Naphthalene	0.39	0.24
C11 Indanes	0.61	0.50
C13 Paraffins	0.25	0.24
Tridecane	0.24	0.23
C12 Aromatics	1.00	0.86
C12 Indanes	0.05	0.04
C14+ Paraffins	0.23	0.22
Methylnaphthalenes	0.83	0.61
C13+ Aromatics	0.21	0.18
Dimethylnaphthalenes	0.22	0.16
Unidentified Heavies	0.00	0.00
Totals	100.00	100.00

ATTACHMENT C-2

VALIDATION CALCULATIONS

VALIDATION CALCULATION SOIL INGESTION PATHWAYS-CHILD
ALTERNATE TPH - GASOLINE
API PS-6 GASOLINE

TABLE 1

EXPOSURE PARAMETERS

PARAMETER	SYMBOL	PATHWAY 1	PATHWAY 2	UNITS	SOURCE
RECEPTOR		CHILD	CHILD		
CONCENTRATION IN SOIL	[OHM]			mg/kg	
INGESTION RATE	I	200	200	mg/day	DEP, 1989
BIOAVAILABILITY FACTOR	BAF			unitless	
SURFACE AREA EXPOSED	SA	NA	NA	cm^2/day	DEP, 1989
MASS SOIL ADHERED TO SKIN	MS	NA	NA	mg/cm^2	DEP, 1989
CONVERSION FACTOR	CF	0.000001	0.000001	kg/mg	
BODY WEIGHT	BW	16	16	kg	DEP, 1989
EXPOSURE FREQUENCY	F	1	1	events/days	Assumption
DURATION OF EVENT	D1	1	1	days/event	Assumption
DURATION OF EXPOSURE					
CANCER	D2	7	7	years	Assumption
NONCANCER	D2	7	7	years	Assumption
AVERAGING PERIOD					
CANCER	AP	70	70	years	DEP, 1989
NONCANCER	AP	7	7	years	Assumption

PATHWAY 1: INCIDENTAL INGESTION OF SOIL BY A CHILD - USING NEW APPROACH

VALIDATION CALCULATION SOIL INGESTION PATHWAYS-CHILD
ALTERNATE TPH - GASOLINE
API PS-6 GASOLINE

TABLE 1

COMPOUND	AVERAGE [OHM] (mg/kg)	MAXIMUM [OHM] (mg/kg)	BAF INGESTION	BAF DERMAL	LADD AVERAGE (mg/kg-day)	ADD AVERAGE (mg/kg-day)
C5-C8 ALKA/CYCLO	41.606		1		5.2e-05	5.2e-04
C9-C18 ALKA/CYCLO	8.725		1		1.1e-05	1.1e-04
C9-C32 AROM/ALKEN	15.81		1		2.0e-05	2.0e-04
BENZENE	2.12		1		2.7e-06	2.7e-05
TOLUENE	3.97		1		5.0e-06	5.0e-05
ETHYLBENZENE	1.87		1		2.3e-06	2.3e-05
XYLENE	5.945		1		7.4e-06	7.4e-05

PATHWAY 2: INCIDENTAL INGESTION OF SOIL BY A CHILD - USING WHOLE PRODUCT TOXICITY VALUES

COMPOUND	AVERAGE [OHM] (mg/kg)	MAXIMUM [OHM] (mg/kg)	BAF INGESTION	BAF DERMAL	LADD AVERAGE (mg/kg-day)	ADD AVERAGE (mg/kg-day)
TOTAL GAS	100		1	NA	1.3e-04	1.3e-03

VALIDATION CALCULATION SOIL INGESTION PATHWAYS-CHILD
ALTERNATE TPH - GASOLINE
API PS-6 GASOLINE

TABLE 1

PATHWAY 1: INCIDENTAL INGESTION OF SOIL BY A CHILD - USING NEW APPROACH

COMPOUND	CANCER SF $(mg/kg\text{-}day)^{-1}$	ELCR AVERAGE	ELCR MAXIMUM	ORAL RFD (mg/kg-day)	HI AVERAGE	HI MAXIMUM
C5-C8 ALKA/CYCLO	NA			0.06	8.7e-03	0.0
C9-C18 ALKA/CYCLO	NA			0.6	1.8e-04	0.0
C9-C32 AROM/ALKEN	NA			0.03	6.6e-03	0.0
BENZENE	0.029	7.7e-08	0.0	0.005	5.3e-03	0.0
TOLUENE	NA			0.2	2.5e-04	0.0
ETHYLBENZENE	NA			0.1	2.3e-04	0.0
XYLENE	NA			2	3.7e-05	0.0
	TOTAL ELCR	7.7e-08	0.0	TOTAL HI	2.1e-02	0.0

VALIDATION CALCULATION SOIL INGESTION PATHWAYS-CHILD
ALTERNATE TPH - GASOLINE
API PS-6 GASOLINE

TABLE 1

PATHWAY 2: INCIDENTAL INGESTION OF SOIL
BY A CHILD - USING WHOLE PRODUCT TOXICITY VALUES

COMPOUND	CANCER SF $(mg/kg\text{-}day)^{-1}$	ELCR AVERAGE	ELCR MAXIMUM
TOTAL GAS	0.0017	2.1e-07	0.0
	TOTAL ELCR	2.1e-07	0.0

ORAL RFD (mg/kg-day)	HI AVERAGE	HI MAXIMUM
0.2	6.3e-03	0.0
TOTAL HI	6.3e-03	0.0

EXPOSURE AND RISK CALCULATION DOCUMENTATION
SOIL INGESTION PATHWAYS-CHILD
ALTERNATE TPH - GASOLINE
API 91-1 GASOLINE

TABLE 2

EXPOSURE PARAMETERS

PARAMETER	SYMBOL	PATHWAY 1	PATHWAY 2	UNITS	SOURCE
RECEPTOR		CHILD	CHILD		
CONCENTRATION IN SOIL	[OHM]			mg/kg	
INGESTION RATE	I	200	200	mg/day	DEP, 1989
BIOAVAILABILITY FACTOR	BAF			unitless	
SURFACE AREA EXPOSED	SA	NA	NA	cm^2/day	DEP, 1989
MASS SOIL ADHERED TO SKIN	MS	NA	NA	mg/cm^2	DEP, 1989
CONVERSION FACTOR	CF	0.000001	0.000001	kg/mg	
BODY WEIGHT	BW	16	16	kg	DEP, 1989
EXPOSURE FREQUENCY	F	1	1	events/days	Assumption
DURATION OF EVENT	D1	1	1	days/event	Assumption
DURATION OF EXPOSURE					
CANCER	D2	7	7	years	Assumption
NONCANCER	D2	7	7	years	Assumption
AVERAGING PERIOD					
CANCER	AP	70	70	years	DEP, 1989
NONCANCER	AP	7	7	years	Assumption

EXPOSURE AND RISK CALCULATION DOCUMENTATION
SOIL INGESTION PATHWAYS-CHILD
ALTERNATE TPH - GASOLINE
API 91-1 GASOLINE

TABLE 2

PATHWAY 1: INCIDENTAL INGESTION OF SOIL BY A CHILD - USING NEW APPROACH

COMPOUND	AVERAGE [OHM] (mg/kg)	MAXIMUM [OHM] (mg/kg)	BAF INGESTION	BAF DERMAL	LADD AVERAGE (mg/kg-day)	ADD AVERAGE (mg/kg-day)
C5-C8 ALKA/CYCLO	38.78		1		4.8e-05	4.8e-04
C9-C18 ALKA/CYCLO	3.16		1		4.0e-06	4.0e-05
C9-C32 AROM/ALKEN	18.51		1		2.3e-05	2.3e-04
BENZENE	1.22		1		1.5e-06	1.5e-05
TOLUENE	6.94		1		8.7e-06	8.7e-05
ETHYLBENZENE	3.42		1		4.3e-06	4.3e-05
XYLENE	10.19		1		1.3e-05	1.3e-04

PATHWAY 2: INCIDENTAL INGESTION OF SOIL BY A CHILD - USING WHOLE PRODUCT TOXICITY VALUES

COMPOUND	AVERAGE [OHM] (mg/kg)	MAXIMUM [OHM] (mg/kg)	BAF INGESTION	BAF DERMAL	LADD AVERAGE (mg/kg-day)	ADD AVERAGE (mg/kg-day)
TOTAL GAS	100		1	NA	1.3e-04	1.3e-03

EXPOSURE AND RISK CALCULATION DOCUMENTATION
SOIL INGESTION PATHWAYS-CHILD
ALTERNATE TPH - GASOLINE
API 91-1 GASOLINE

TABLE 2

PATHWAY 1: INCIDENTAL INGESTION OF SOIL BY A CHILD - USING NEW APPROACH

COMPOUND	CANCER SF (mg/kg-day)$^{-1}$	ELCR AVERAGE	ELCR MAXIMUM	ORAL RFD (mg/kg-day)	HI AVERAGE	HI MAXIMUM
C5-C8 ALKA/CYCLO	NA			0.06	8.1e-03	0.0
C9-C18 ALKA/CYCLO	NA			0.6	6.6e-05	0.0
C9-C32 AROM/ALKEN	NA			0.03	7.7e-03	0.0
BENZENE	0.029	4.4e-08	0.0	0.005	3.1e-03	0.0
TOLUENE	NA			0.2	4.3e-04	0.0
ETHYLBENZENE	NA			0.1	4.3e-04	0.0
XYLENE	NA			2	6.4e-05	0.0
	TOTAL ELCR	4.4e-08	0.0	TOTAL HI	2.0e-02	0.0

PATHWAY 2: INCIDENTAL INGESTION OF SOIL BY A CHILD - USING WHOLE PRODUCT TOXICITY VALUES

COMPOUND	CANCER SF (mg/kg-day)$^{-1}$	ELCR AVERAGE	ELCR MAXIMUM	ORAL RFD (mg/kg-day)	HI AVERAGE	HI MAXIMUM
TOTAL GAS	0.0017	2.1e-07	0.0	0.2	6.3e-03	0.0
	TOTAL ELCR	2.1e-07	0.0	TOTAL HI	6.3e-03	0.0