American Petroleum Institute

Chemical Human Health Hazards Associated With Oil Spill Response

Regulatory Analysis and Scientific Affairs

PUBLICATION NUMBER 4689
AUGUST 2001

CHEMICAL HUMAN HEALTH HAZARDS ASSOCIATED WITH OIL SPILL RESPONSE

Prepared for the
American Petroleum Institute
1220 L Street, NW
Washington, DC 20005

Prepared by

Evan C. Thayer
Anita George-Ares
Robert T Plutnick
Ruth A. Kaufman

Exxon Biomedical Sciences, Inc.
Mettlers Rd. CN 2350
East Millstone, NJ 08875-2350

August 2001

FOREWORD

API PUBLICATIONS NECESSARILY ADDRESS PROBLEMS OF A GENERAL NATURE. WITH RESPECT TO PARTICULAR CIRCUMSTANCES, LOCAL, STATE, AND FEDERAL LAWS AND REGULATIONS SHOULD BE REVIEWED.

API IS NOT UNDERTAKING TO MEET THE DUTIES OF EMPLOYERS, MANUFACTURERS, OR SUPPLIERS TO WARN AND PROPERLY TRAIN AND EQUIP THEIR EMPLOYEES, AND OTHERS EXPOSED, CONCERNING HEALTH AND SAFETY RISKS AND PRECAUTIONS, NOR UNDERTAKING THEIR OBLIGATIONS UNDER LOCAL, STATE, OR FEDERAL LAWS.

NOTHING CONTAINED IN ANY API PUBLICATION IS TO BE CONSTRUED AS GRANTING ANY RIGHT, BY IMPLICATION OR OTHERWISE, FOR THE MANUFACTURE, SALE, OR USE OF ANY METHOD, APPARATUS, OR PRODUCT COVERED BY LETTERS PATENT. NEITHER SHOULD ANYTHING CONTAINED IN THE PUBLICATION BE CONSTRUED AS INSURING ANYONE AGAINST LIABILITY FOR INFRINGEMENT OF LETTERS PATENT.

ACKNOWLEDGMENTS

The following people are recognized for their contributions of time and expertise during the preparation of this report:

API STAFF CONTACT

Thomas Purcell, Regulatory Analysis and Scientific Affairs

MEMBERS OF THE OIL SPILL SCIENCE AND TECHNOLOGY WORK GROUP

David Fritz, Chairperson, BP
Dan Allen, Chevron North America E&P Company
Ken Bitting, US Coast Guard R&D Center
Bon Britton, US Fish and Wildlife Service
Michael Carter, US Maritime Administration
Jim Clow, Equiva Services
Bill Dahl, ExxonMobil Research & Engineering Co.
Donald Erickson, Bay West, Inc.
Ronald Goodman, Imperial Oil Ltd.
Brad Hahn, State of Alaska
Bela James, Equilon Enterprises, LLC
Robin Jamail, Texas General Land Office
Roger LaFerriere, US Coast Guard
Jerry Langley, Williams Pipe Line Company
Stephen Lehamann, NOAA
Richard Lessard, ExxonMobil Research and Engineering Company
Dan Leubecker, US Maritime Administration
Edwin Levine, NOAA
Jason Maddox, NOAA
Joseph Mullin, Minerals Management Service
William Nichols, U.S. EPA
Douglas O'Donovan, Marine Spill Response Corporation
W. Michael Pittman, US Coast Guard
Ninette Sadusky, US Navy SUPSALV
Jim Sanders, CITGO Pipeline Company
Dana Slade, Lakehead Pipe Line Company
Jean Snider, NOAA
Alexis Steen, ENTRIX Corp.
Robert Urban, PCCI
Carol Voigt, CITGO Petroleum Corporation

The American Petroleum Institute recognizes Woodward Clyde Consultants and ENSR Consulting and Engineering for their assistance in the completion of this project. LCDR Roger LaFerriere and LCDR David Stalfort, both from the US Coast Guard, are also recognized for their technical review and creative contributions to the final preparation of this project.

TABLE OF CONTENTS

LIST OF FIGURES

Figures

LIST OF TABLES

Table

EXECUTIVE SUMMARY

This report provides an overview of potential human health hazards encountered by personnel involved with petroleum product spills or leaks. Widely distributed products are covered, including crude oil, gasolines, various middle distillates (e.g., kerosene, jet fuel, diesel fuel, and home heating oil), heavy fuel oil, and asphalt. The main objective is to define basic components and products of concern based on their inherent toxicity and potential risks to oil spill workers. In addition, environmental factors that may affect the risk of exposure to the various components are discussed. A brief summary of important exposure considerations is included (Section 4).

In order to discuss the potential human health hazards associated with these petroleum hydrocarbon products, it is helpful to break down the products into the various components of concern and understand the toxicology of those components. Table 2-1 links the various products to their associated components of concern. The main components of health concern in Table 2-1 include benzene, hydrogen sulfide, n-hexane, naphthalene, toluene, trimethyl benzene, polycyclic aromatic hydrocarbons (PAHs) and, in specific instances, organic lead compounds.

Acute health hazards associated with many components of concern include depression of the central nervous system, and irritation to the skin, eyes, and respiratory tract. Chronic exposure to some components, such as benzene and polycyclic aromatic hydrocarbons, has been associated with cancer risks.

Other than the toxicological properties of the components and products of concern, other factors affect the nature of health hazards during spill response activities. These include environmental conditions, the physical characteristics of the products, and the type of spill or leak scenario and time factors. The environmental fate of a spilled material is determined by various mechanisms including evaporation, spreading, dissolution, drift, dispersion, and emulsification.

Many of the more toxic components evaporate rapidly, so risk of exposure to airborne contaminants diminishes greatly with time. This is especially true for one of the most inherently toxic components, hydrogen sulfide. Benzene and other very light hydrocarbons may evaporate

within the first 6-8 hours after a spill. As the material continues to weather, physical contact becomes the main route of exposure. Chemical hazards relating to oil treating agents such as dispersants or chemical cleaners are not evaluated in this report.

Section 1

INTRODUCTION

Oil spills can originate from many sources. Many spill incidents are attributed to short-term, high volume releases from pipeline breaks, vessel casualties such as sinking, grounding, and collision, and from facilities during fuel or cargo transfers. The potential human health hazards associated with long-term chronic exposure to petroleum fuels during petroleum *production* operations have been reviewed and summarized (CONCAWE, 1985); however, the short-term health hazards to human health from petroleum *spills* have not been discussed in detail (API, 1997).

It is important to understand potential hazards associated with an oil spill so that efforts can be made to reduce acute health impacts, particularly during emergency response. The potential health hazards from a petroleum spill depend on many factors, the most important are the:

- Chemical and physical properties and composition of the petroleum product;
- Environmental conditions both during and after the release;
- Location and types of tasks performed by the oil spill workers;
- Control measures used to minimize worker exposure.

The objective of the report is to identify the potential chemical health hazards and provide information on exposure to spilled petroleum products. The report is not intended to be used as a field guide during cleanup operations following a spill. Physical hazards, such as slips, trips, falls, fire, and explosion are important, but are not discussed in this report.

Hazard identification is the first step in the process of linking scientific information about hazards (risk assessment) to the decision-making process (risk management) during which these hazards are mitigated. Figure 1-1 shows the elements of risk assessment and risk management and highlights the risk assessment process covered in this report.

Hazard identification is defined as the process of determining whether exposure to an agent can cause an increase in the incidence of a health condition (EPA, 1992). In order to identify the overall health hazards, the toxicological properties of each petroleum constituent were reviewed

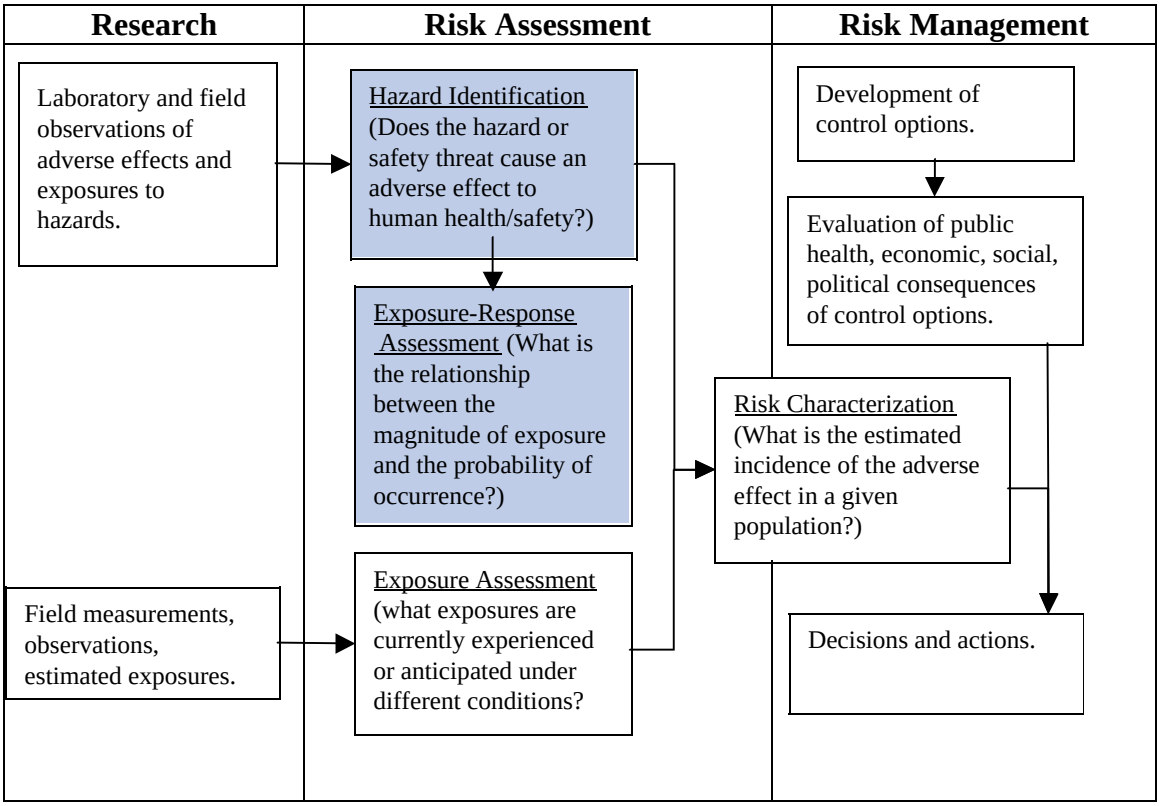

Figure 1-1. Elements of Risk Assessment and Risk Management (modified from NRC 1993).

due to the poor health hazard information available on oils as a whole. Information used to compile this summary includes toxicology databases, case histories of oil spill drills and actual incidents, as well as model data. Little relevant information on exposure to oil spill responders is available in the literature. Factors evaluated include chronic and acute toxicity by the two major routes of exposure (dermal and inhalation), and the component concentrations in the common petroleum products. These factors are summarized in Table 2-1.

The following topics are discussed:

- Identification of the most commonly spilled petroleum products;
- Review of their overall potential health hazards as well as those associated with their components of concern;
- Summarization of the relevant environmental fate of petroleum products;
- Additional considerations related to health hazard assessment.

This document is intended to provide general guidance for oil spill response planning. Therefore, conclusions reached may not apply to all situations. Actual health concerns may vary

depending upon product composition, environmental conditions, worker training, and other factors. Where potential exposures are uncertain, air monitoring and/or conservative protective measures are recommended.

Section 2

ENVIRONMENTAL FATE AND EXPOSURE CONSIDERATIONS

This section describes the environmental fate processes and provides a brief overview of some considerations that need to be included in discussions about potential exposure. The information found in this section is based on referenced literature (Fingas, 1994, 1995; GESAMP, 1993; Koons and Jahns, 1992; Mielke, 1990; NRC, 1985, 1989). A more complete description of the environmental fate and effect processes are found in the *Fate and Environmental Effects of Oil Spills in Freshwater Environments* (API, 1999).

ENVIRONMENTAL FATE

Introduction

When oil is released into the environment, numerous processes begin to affect the environmental fate of oil components and the chemical-physical properties of the oil. The composition and physical/chemical properties of crude oil and refined products are variable; therefore, no two crude or refined oils will behave exactly the same in the environment. In addition, meteorological and oceanographic conditions will affect the movement and weathering of oil.

Spreading, drift, evaporation, dissolution, and dispersion are processes that begin immediately once oil is spilled. Figure 2-1 illustrates these processes. One process can occur at a greater relative magnitude than another process. For example, in the early phases of an oil spill, spreading occurs at a greater rate than oil drift. Dispersion of oil occurs at a greater magnitude than dissolution since only small amounts of oil components are soluble in water. Emulsification, sedimentation, biodegradation, and photooxidation can occur within the first day of a spill, but these processes are not as predominant as spreading, evaporation, and dispersion.

Some of the fate processes, for example, evaporation, may be most relevant to human health exposure. Other processes, such as photooxidation and sedimentation, are not important determinants of human exposure. The major processes are discussed here to provide a general understanding of the environmental fate of spilled oil.

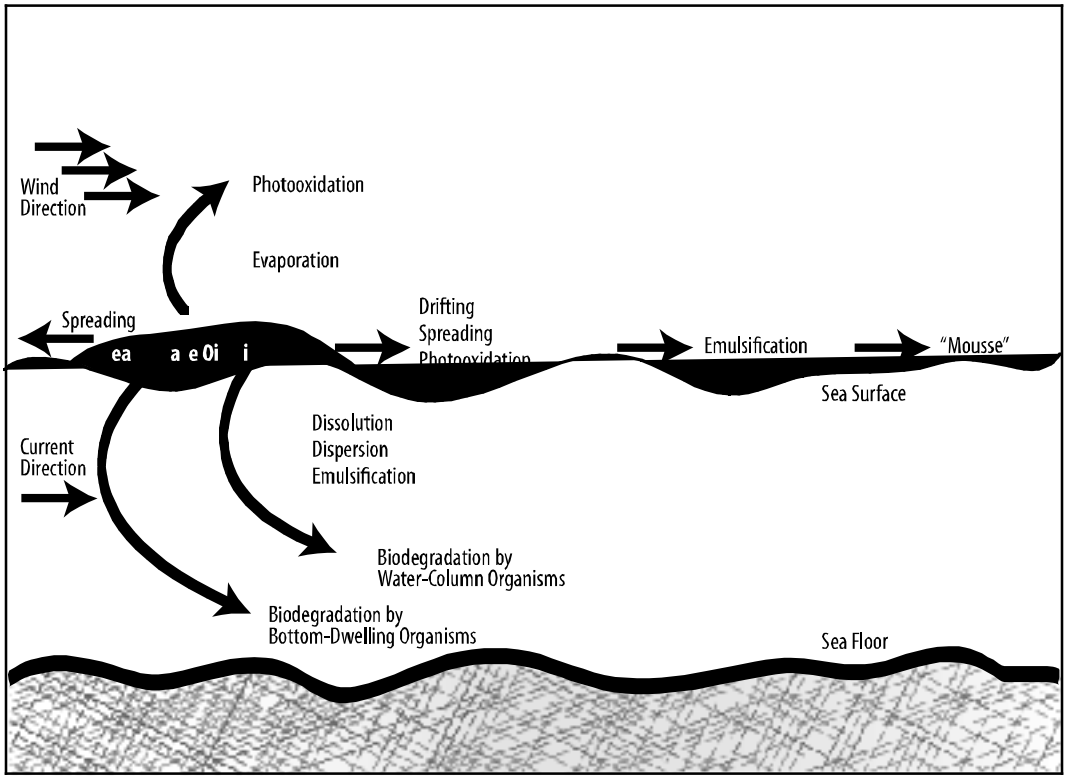

Figure 2-1. Processes Acting on Spilled Oil (Exxon, 1985)

Spreading

Spreading is variable, depending on the type of oil spilled. If a light oil is spilled, it begins to spread immediately and rapidly. In the hours following a spill, as the layer of oil thins, the rate of spreading decreases. Some oils, generally those with a specific gravity greater than 1.0 and those with a pour point greater than the ambient water temperature, tend to form lumps and may sink rather than spread.

Spreading is controlled by oil volume, viscosity, and surface tension. Surface tension and viscosity vary with temperature and type of oil. As surface tension increases, spreading becomes less rapid.

Weathering diminishes spreading by reducing oil volume and increasing oil density and viscosity. High viscosity oils spread slowly while low viscosity oils spread more rapidly. The type of oil determines the rate at which cold water temperatures increase oil viscosity and, thus, slow the spreading of oil as well as the rate at which higher water temperatures enhance oil spreading.

However, relative to other controlling factors, the effects of ambient temperature on spreading are usually negligible for oils, except for high viscosity oils such as Bunker C.

An oil slick contains areas of variable thickness. Under relatively calm conditions, oil slicks typically may have a thick (≥ 1 mm) area surrounded by a more extensive thin (< 10 μm) area. The thick portion contains the greatest volume of oil (approximately 90%) while the thin portion of the slick contains the balance. The thin area, however, represents 80% to 90% of the total slick area.

Local physical factors make predictions of spreading difficult. Wave and wind action modify oil spreading. Natural surface convergences (areas where currents meet) or divergences (areas where adjacent currents move in different directions) may cause accumulations or separations of oil. As a result, windrows (streaks) and oil patches may occur. Spreading is an important determinant for rates of dissolution, dispersion, and photooxidation of oil.

Drift

The combined action of wind, surface currents, and waves causes the oil slick to drift. Prediction of slick drift is necessary for spill response planning and operations. Slick drift is largely independent of spill volume, spreading, or weathering. However, a "thick" slick drifts faster than a "thin" slick, because wind interaction is increased with thicker oil in comparison to thinner oil. Therefore, the thicker portions of the slick often form the leading edge of an advancing spill. Local conditions such as river outflow, nearshore structures, or the presence of surface debris can also affect slick drift.

Evaporation

Evaporation is one of the primary weathering processes involved in the removal of oil from the sea. Evaporation begins immediately upon release of oil and is especially dominant during the first few days of a spill. During evaporation, volatile hydrocarbon components from the oil slick escape to the atmosphere. In the first few days following a spill, light crude oils can lose up to 75% of their volume due to evaporation. Medium crude oils and heavy crude oils may lose up to 40% and 10%, respectively, of their volume through evaporation.

Crude oil is a complex mixture containing numerous components with different volatilities. Lower molecular weight components are generally the most volatile. These components, such as benzene and toluene, evaporate rapidly, typically in less than one day. As evaporation proceeds, the oil becomes enriched in non-volatile components, higher molecular weight hydrocarbons, and complex nitrogen-sulfur-oxygen compounds.

As low molecular weight components evaporate, the physical properties of the remaining oil change. For example, specific gravity and viscosity of the oil will increase. These changes will tend to retard the rate of spreading and evaporation.

Dissolution

Dissolution is the transfer of soluble hydrocarbons from a slick or dispersed oil into solution in water. Dissolution begins immediately after a spill.

The concentration of soluble components in crude oil is low, about 10 to 30 ppm. Low molecular weight components tend to be more soluble than higher molecular weight components. The more polar aromatic components (benzene, toluene, xylenes) tend to be more soluble than alkanes (up to C5). In addition, some of the sulfur-containing components and salts present in crude oil are water soluble.

Since only a very small fraction of an oil slick dissolves, it is unlikely that dissolution significantly affects oil weathering. Although evaporation occurs more rapidly than dissolution, dissolution is an important process under conditions when evaporation is minimal. For example, evaporation is not likely to occur when oil droplets are dispersed or when oil is trapped under or in ice.

Dispersion

Oil can enter water naturally, without chemical enhancement, as dispersed droplets. The diameter of dispersed droplets generally ranges from 10 to 100 μm. Dispersion makes it difficult to interpret oil concentrations in water (e.g., soluble oil) measured beneath slicks. The difficulty lies in separating water-soluble components of oil from neat oil droplets prior to analysis. Most oil in the

water column under a slick is believed to be dispersed droplets rather than truly dissolved hydrocarbons.

Oil composition, slick thickness, oil-water interfacial tension, and turbulence are factors affecting oil dispersion. Crude oils and other petroleum products contain nitrogen, sulfur, and oxygen-containing compounds that act as surface active agents (surfactants). Natural surfactants in petroleum tend to reduce the oil-water interfacial tension allowing the oil to disperse more readily.

Turbulence enhances oil dispersion by increasing the interaction between the oil and water layers; therefore, dispersion is greater where there is high wave energy. Breaking waves may entrain fragments of the slick into the upper water column. The larger, more buoyant oil droplets may surface and coalesce with the slick, while smaller oil droplets are incorporated in the water column.

Dispersion also occurs in the absence of breaking waves. Oil droplets in the water column will continue to disperse under conditions of turbulent mixing and currents. Use of chemical dispersants during spill response operations allows dispersion to occur at much lower energy levels and minimizes recoalescence of dispersed droplets.

Both natural and chemical dispersion accelerate oil degradation. Dispersion increases the surface area of oil relative to its volume. Therefore, dissolution and biodegradation may proceed more rapidly.

Emulsification

Water-in-oil emulsification is the incorporation of water droplets (1 to 15 μm diameters) into oil. These emulsions, also referred to as "mousse", may contain from 20 to 80 percent water.

The factors that affect the formation of emulsions are not completely understood. Emulsion formation and stability are affected by temperature and the amount of asphaltenes, waxes, and surface-active materials in oil. Data are available for the emulsion-forming properties of various oils. Oils having high viscosity and high specific gravity are more likely to form mousse than are oils with lower viscosity and specific gravity.

Water-in-oil emulsions slow the natural degradation of oil. These emulsions may persist for months or years following a spill, depending upon local conditions. Depending on the oil type, water in oil emulsification can significantly increase the volume and viscosity of the oil/water mixture. An emulsion containing 70 to 80% water is 3 to 4 times larger than the initial volume of spilled oil. Mousse viscosity is typically far greater than that of the parent oil.

If mousse strands on shore, it may accumulate debris and sediment. Following the evaporation of water from stranded mousse, a tarry residue may remain and slowly weather over time.

Sedimentation

Sedimentation occurs when the specific gravity of oil becomes greater than that of water. The specific gravity of floating or dispersed oil can increase due to

1) weathering (evaporation, dissolution, and emulsification),
2) adhesion or sorption onto suspended particles or,
3) zooplankton ingestion of oil with subsequent incorporation into fecal pellets.

Heavy oil, highly weathered oil, and tar may accumulate debris and sink, becoming incorporated in sediments. Oil may become incorporated into nearshore sediments as oil comes in contact with shorelines.

The composition and concentration of particles are important factors affecting sedimentation. Oil can sorb onto plankton or sediment. Fine-grained sediments such as silts and clays often contain charged particles that can bind to hydrocarbons. Consequently, hydrocarbons tend to accumulate in fine-grained sediments. Sedimentation will vary with location, depending on the concentration and type of suspended particles in the water column. Sedimentation is an important process in rivers, deltas, and estuaries where suspended particle levels are high.

Biodegradation

Marine and freshwater environments contain populations of bacteria and fungi that can metabolize and degrade hydrocarbons (a carbon source). Carbon, in conjunction with other nutrients, aids survival, growth, and reproduction of certain microorganisms.

The rate and extent of oil biodegradation depend on numerous factors such as:

1) Abundance and type of petroleum-degrading species,
2) Available oxygen and nutrients,
3) Temperature,
4) Oil composition,
5) Degree of oil weathering, and
6) Whether or not chemical dispersants are used.

Oil biodegradation in arctic regions may occur slowly due to lower temperatures and the slow loss of volatile components. In coastal waters, terrestrial runoff and sediment resuspension add to the nutrient supply of local waters and may enhance biodegradation. In offshore waters, where available nutrients are not increased by terrestrial runoff, oil biodegradation may occur more slowly. Wave and current action may enhance biodegradation by increasing the supply of available oxygen.

Different species of microorganisms degrade different components of oil. Petroleum-degrading microbes are widespread and abundant in rivers, coastal waters, and streams chronically polluted by hydrocarbons. Dispersed oil can be readily biodegraded because of the increased surface area available for microbial action. Mousse degrades slowly since exchange of water, nutrients, and oxygen is limited.

Photooxidation

Volatilized hydrocarbons can be oxidized by solar radiation in a process known as photooxidation. Dissolved aromatic and polar compounds at the surface of the water column may also undergo photooxidation.

Products of photooxidation tend to be soluble in seawater and are removed from the slick. The oxidation process is minimal in the overall removal of oil; however, oxidation does alter the chemical characteristics of remaining oil allowing other, more significant, degradative processes to operate.

The physical properties of oil and oil thickness govern oxidation rates. Thin slicks oxidize more rapidly than thick oil or mousse. Photooxidation is not an important degradative process for tar balls, since only the surface is actively oxidized. Oil incorporated in sediments or deep in the water column are not exposed to photooxidation.

Fate of Oil Onshore

Dispersion, dissolution, and emulsification can occur in nearshore waters, depending on waves, currents, tides, and oil properties. Breaking waves create a high-energy environment, leading to greater dispersion and emulsification rates. As wave and tidal action remove and replace shoreline substrate, oil moves from the shoreline to nearshore waters. On medium to high energy shorelines, oil is removed more rapidly than from sheltered, low energy shorelines.

Shorelines act as thermal insulators and stranded oil exposed to sun can attain greater temperatures than oil on water. Oil heated by the sun or by warm terrestrial surfaces may spread farther and more rapidly than oil on water. This increases the oil surface area exposed to sunlight, raising the oil temperature even further. Therefore, the evaporation rate for stranded oil may be greater than for oil on water. When stranded oil is exposed to solar energy, some emulsions may break down and release water, which will reduce oil volume. Biodegradation rates of onshore oil depend on numerous factors, including microbial species, temperature, oil properties, and available nutrients (nitrogen, phosphorus).

The degree of oil penetration on a shoreline depends on the substrate and oil type. For example, oil readily penetrates pebble and cobble beaches resulting in isolation of oil from the surface environment. Consequently, weathering and biodegradation are decreased if the oil reaches deeper zones with restricted oxygen, nutrients, or flushing. Low viscosity oils will penetrate beaches more readily than high viscosity oils. Depending on the nature of the sediment/substrate, subsurface oil can persist for months to several years. If oil adheres to sheltered rocks onshore, it may not be easily removed by wave and tidal action. Oil adhesion varies with oil type and weathered state, as well as characteristics of the rock surface (wetness, presence of organisms).

Fate Summary

An improved understanding of oil fate aids decision-making and response at the time of a spill. The fate of spilled crude oil and refined products is governed by a complex interaction of physical, chemical, and biological processes. The magnitude of each process varies over time based upon the type of oil, and local sea and weather conditions.

Spilled oil is physically transported via spreading, drifting, natural dispersion, and sedimentation. These processes affect the distribution of oil at the sea surface, in the water column, and in sediments. In general, these processes act on whole oil without changing its composition or physical properties.

Weathering processes include evaporation, dissolution, and emulsification, which affect the composition and physical properties (density, viscosity) of spilled oil. These processes vary based upon the physical and chemical properties of petroleum and site-specific conditions such as wind, temperature, and sea state. Evaporation results in the transfer of the volatile components of oil to the atmosphere. In dissolution, soluble components of oil dissolve in the water column, while emulsification is the incorporation of water into the oil. Degradative processes such as photooxidation and biodegradation alter the chemical structure of oil components. Degradation rates vary according to a number of local factors.

EXPOSURE CONSIDERATIONS

Besides toxicity of the products and components, other things that need to be considered in a complete health hazard assessment include time factors, location of personnel, tasks being performed, nature of products and spill scenario, and regulatory factors.

Time Factors

Little data is available describing actual chemical exposures immediately following a crude oil spill. Typically, the time of highest expected airborne contaminants of concern is immediately following the spill. As time passes, the potential for exposure to volatile contaminants, including benzene and hydrogen sulfide, diminishes.

Thayer and Tell (1999) reviewed available benzene exposure data from oil spills as well as modeling data conducted at Exxon Biomedical Sciences. The combined data indicate that initial benzene concentrations may exceed 1 ppm (the OSHA and U.S. Coast Guard Permissible Exposure Limit), but levels decrease to less than 1 ppm within about 6 hours. Similarly, model data of simulated sour crude oil spills show that initial concentrations of hydrogen sulfide may be considered immediately dangerous to workers in the area. However, modeled exposures drop to safe levels in about 4 minutes. Based on this data, it appears that exposure to benzene and hydrogen sulfide is a consideration for personnel at the spill site, (e.g., ship or terminal personnel) within the first 6-8 hours after fresh oil is no longer being spilled. The potential for benzene and hydrogen sulfide exposure to oil spill cleanup workers arriving one or more days after the spill would be lower.

As more time elapses, evaporation of volatile components continues and airborne contaminant levels continue to decrease. Payne, *et al.,* (1991) report that with water temperatures of 11-14ºC (52-57ºF), all compounds with boiling points <400°F were lost within the first nine days. Between two weeks and seven months, there was an additional loss of compounds between n-C11 and n-C-13 (boiling points <450°F). Therefore, exposure to airborne contaminants is less of a concern after weathering occurs.

Nature of Products, Spill Scenario and Tasks

The nature of the product and the spill scenario also affect the nature of the potential health hazards. Heavy products are affected little by evaporation, but may sink or be washed ashore as tar balls or mousse. Since there is little evaporation occurring, exposure to airborne contaminants would not be a concern unless the material is aerosolized (e.g., via high pressure water washing the shore). For those heavy products that contain PAHs, skin contact may be the primary concern for oil spill workers, depending on the nature of the cleanup tasks.

Products such as gasoline, middle distillates, and most crude oils contain volatile components that could generate high concentrations of light hydrocarbons, especially in enclosed spaces. Again, depending on the spill scenario and tasks required of cleanup workers, risk of

fire/explosion, oxygen deficiency, and mixed hydrocarbon exposure may need to be considered in these situations. When two or more hazardous substances which act upon the same organ system are present, there is a concern of additive effects (ACGIH, Appendix C, 1998). With mixed volatile hydrocarbons in enclosed spaces, this could mean greater potential (relative to that in well-ventilated areas) for central nervous system effects leading to increased risk of falls, e.g., dizziness or narcotic effects or poor judgment (Holliday and Park, 1993).

In some spill scenarios, such as a punctured hull, underwater wellhead or pipeline leaks, oil continues to be spilled over a long period. In these scenarios, evaporation of volatile components and, thus, airborne contamination continues as long as leakage is occurring. Weathering, in these cases, may not be as effective in reducing levels of volatile components. Also, unusual tasks may be necessary to stop or control the spill, and the nature of these tasks could affect the health risks to workers.

All of the above information is very generic in nature and is only intended to provide rough guidance regarding the components of concern for the various products. If there is any doubt regarding the potential exposures to oil spill workers, there are two main courses of action: monitoring (direct reading) can be conducted to determine actual exposures or, if this is not feasible, workers can be provided with sufficient protective gear to protect against worst-case exposure scenarios. Whenever feasible, monitoring and interpretation of results should be conducted by, or in consultation with, industrial hygienists or adequately trained personnel.

The spill scenario timing, nature of the products, and cleanup tasks need to be considered when assessing and controlling health hazards. During periods when air-borne contaminant concentrations is high, respiratory and eye protection may be needed. Skin protection is an important consideration for tasks where workers may come in direct contact with spilled oil.

Regulatory Issues

Another issue to consider with respect to health hazards and their evaluation and control is the existence of national, state, and local occupational health regulations applicable to oil spill response activities for exposure and training. Examples are those issued by the U.S.

Occupational Safety and Health Administration (OSHA) for spills connected to land (e.g., reaching shoreline or docks), and the U.S. Coast Guard for spills offshore. Activities associated with some hazardous components such as benzene may be heavily regulated. Detailed discussion of regulatory issues is outside the scope of this document, but exposure limits set by OSHA are listed in Table 2-1 along with exposure limits established by the American Conference of Governmental Industrial Hygienists (ACGIH).

One commonly misunderstood factor affecting oil spill worker exposures relates to the length of exposure. Health standards that are typically compared to worker exposure monitoring results are time-weighted averages (TWAs). As an example, ACGIH defines this as "the time-weighted average concentration for a conventional 8-hour workday and a 40-hour workweek, to which it is believed that nearly all workers may be repeatedly exposed, day after day, without adverse effect." In developing such standards, toxicologists commonly assume that workers could be exposed to these levels for a working lifetime to ensure a standard that encompasses worst-case scenarios. However, oil-spill cleanup workers are most often temporarily employed from the local work force, and would commonly only be exposed for one day to a week. Although regulators overseeing cleanup activities are correct to compare cleanup exposure results to TWAs instead of the alternative 15-minute short-term limits, it should be recognized that this adds an extra safety factor to the worker's overall risk.

TABLE 2-1

Exposure Limits for Components of Concern in Crude Oils

Chemical Name	1998 ACGIH TLV's*			OSHA Permissible Exposure Limit (PEL)		
	8-Hr TWA (ppm unless specified)	**STEL/C**	**Notations, TLV Basis-Critical Effects**	**8-Hr TWA** (ppm unless specified)	**STEL/C**	**Comments**
Benzene	0.5	2.5	Skin, A1, Cancer	1	5	
Toluene	50	-	Skin, A4, CNS	200	C300	500 ppm for 10 min peak per 8-hr shift
n-Hexane	50	-	Skin, Neuropathy, CNS, Irritation	500	-	
Naphthalene	10	15	A4, Irritation, CNS, Blood	-	-	
Hydrogen Sulfide	(10) [5]	(15)	Sudden death, Irritation, CNS	-	C20	50 ppm for 10 min peak per 8-hr shift
Benzo[a]pyrene	NA	NA	A2, Cancer	0.2 mg/m^3	-	Polycyclic Aromatic Hydrocarbons
Benz[a]anthracene	NA	NA	A2, Cancer	-	-	Polycyclic Aromatic Hydrocarbons
Trimethyl benzene	25	-	Irritation, CNS, Blood	-	-	
Gasoline	300	500	A3, Irritation, CNS	-	-	
Kerosene (Diesel fuel/kerosene)	[100 mg/m^3]	-	Skin, A3, Irritation, Dermatitis, Lung	-	-	
Tetraethyl Lead	0.1 mg/m^3	-	Skin, A4, CNS	0.075 mg/m^3	-	Skin
Tetramethyl Lead	0.15 mg/m^3	-	Skin, CNS	0.075 mg/m^3	-	Skin
Oil Mist	5 mg/m^3	-	Lung	5 mg/m^3	-	

A1 Confirmed Human Carcinogen
A2 Suspected Human Carcinogen
A3 Confirmed Animal Carcinogen with Unknown Relevance to Humans
A4 Not classifiable as a Human Carcinogen
CNS Central Nervous System
Skin Potential for exposure via skin absorption
( ) Current limit shown but change has been proposed (for proposed H_2S TWA = 5 ppm)
[ ] Proposed value listed in Notice of Intended Changes
C Ceiling Limit - concentration should not be exceeded during any part of the working shift
NA Not Applicable
Source (ACGIH, 1998) (US OSHA, 1997)

Section 3

HEALTH HAZARDS OF PRODUCTS AND COMPONENTS OF CONCERN

The horizontal axis in Table 3-1 lists the primary petroleum products associated with spills, and the various components of concern, which are listed on the vertical axis. The body of the table provides some generic guidance regarding the relative health concern associated with the various components for each product. These estimates of relative risk may vary considerably depending on the specific spill circumstances. The specific toxicity and health aspects of petroleum products are discussed in this section.

Table 3-1. Relative Potential Health Concern for Components of Petroleum Products[1]

Components of Potential Concern	Crude Oil	Gasoline	Middle Distillates:	Kerosene	Jet Fuel	Diesel/ Heating Oil	Heavy Fuel Oil	Asphalt
Benzene	✚	✚		✓	✓	✓	✓	✓
n-Hexane	✶	✶		✓	✓	✓	✓	✓
Hydrogen Sulfide	✚[2]	✓		✓	✓	✓	✶	✶
Naphthalene	✓	✓		✶	✶	✶	✓	✓
PNAs	✚	✓		✓	✓	✓	✚	✶
TEL/TML[3]	✓	✓[4]✶[5]		✓	✓	✓	✓	✓
Toluene	✶	✶		✓	✶	✓	✓	✓
Total Hydrocarbons	✶	✚		✶	✶	✶	✓	✓
Trimethyl Benzene	✶	✶		✶	✶	✶	✓	✓

Key:

✚ - Component of concern

✶ - Component of low concern

✓ - Component of negligible or no concern

[1]Ratings are relative to specific conditions and may vary considerably

[2]Sweet crude oils do not contain hydrogen sulfide

[3]Tetraethyl lead/tetramethyl lead

[4]Ratings may be higher for motor gasoline outside the USA

[5]Rating for aviation gasoline

IDENTIFICATION OF PRODUCTS AND COMPONENTS OF CONCERN

Table 3-1 links petroleum products with the relevant components of concern. The following describes basic human toxicology information available on each component, and summarizes that information for each product. The products and components are listed in alphabetical order below, and include Chemical Abstracts Service Registry Numbers (CAS No.). CAS Numbers are unique numerical identifiers assigned to chemical substances and recorded in the CAS Chemical Registry System which provide a concise and unique means of substance identification. For reference purposes, Table 2-1 lists current exposure limits established by ACGIH (1998) and the OSHA (1997). A detailed discussion of these exposure limits is outside the scope of this document.

Products of Concern

Crude Oil (CAS No. 8002-05-9). There are many different formulations of crude oil, which vary based on the parent material, properties, geographic location of the well, and formulations. Crude oil is composed of highly complex and variable mixtures of paraffinic (alkane), cycloparaffinic (naphthenic), and aromatic hydrocarbons of different molecular weight and structure. Crude oil may also contain low percentages of sulfur and trace amounts of nitrogen and oxygen compounds, as well as metals such as nickel and vanadium and other elements. Vapor pressure varies widely based on composition.

Toxicity/Health Aspects: The health effects associated with exposure to crude oil are related to the individual components that make up the crude and their respective concentrations. Generally, crude oil has a low order of acute oral and dermal toxicity and may cause slight to moderate skin irritation (Smith, *et al.,* 1981). Ingested crude oil may be aspirated into the lungs, causing severe chemical pneumonitis, which can be fatal (Holliday and Park, 1993). Inhalation of the vapors of aromatic and paraffinic components may cause dizziness, incoordination, nausea, headache, and fatigue at concentrations above 100 to 200 ppm (Holliday and Park, 1993). In crude oils with sulfur compounds (sour crudes), hydrogen sulfide and mercaptans may be released in early stages of a spill or if the material is overheated. Hydrogen sulfide may cause

irritation, respiratory failure, coma, or death depending on the concentration of the gas and the duration of exposure (Yant, 1930). Benzene exposure may also be a health concern in the early stages of a crude spill. Benzene is a known human carcinogen, and long-term exposure can cause anemia and other blood diseases, including leukemia (Department of Labor, 1977). Mists and aerosols generated during spill cleanup provide a route for the inhalation of nonvolatile components of crude oil, such as polycyclic aromatic hydrocarbons (PAHs). Mists and aerosols containing certain PAHs are, in principle, a cancer risk, but it is not clear whether such particles could be formed in sufficient quantities or of the appropriate size during spill cleanup operations to make the risk significant (Holliday and Park, 1993). The heavy molecular weight fraction of crude oil contains PAHs, some of which are carcinogenic to the skin after repeated, prolonged exposure if proper decontamination techniques are not used (Holliday and Park, 1993). The International Agency for Research on Cancer (IARC) determined that crude oil is not classifiable as to its carcinogenicity to humans. The IARC evaluation states that there is inadequate evidence for the carcinogenicity of crude oil in humans and experimental animals, respectively (International Agency for Research on Cancer, 1989).

Gasoline (CAS No. 86290-81-5). Complex combination of volatile, highly flammable hydrocarbons of carbon number C_4 to C_{10} with boiling points between 30 and 260°C and with a flash point < -40°C (CONCAWE, 1992c). Vapor pressure range @20°C = 38-300 mmHg.

Toxicity/Health Aspects: The main health hazard associated with the ingestion of gasoline is the possibility of severe, potentially fatal damage to lung tissue, which can occur following aspiration of even small amounts of gasoline into the lungs (Sciences International Inc., 1995); (CONCAWE, 1992c); (CONCAWE, 1997). Gasolines are unlikely to cause systemic toxicity following accidental ingestion or skin exposure. Skin contact may cause irritation and, if contact is frequent or prolonged, skin reactions may be severe. Gasolines also have a defatting action on the skin which can result in drying, cracking, and dermatitis. Accidental splashes entering the eye may cause irritation and discomfort. Such effects usually are transient and reversible (Sciences International, 1995b); (CONCAWE, 1992c); (CONCAWE, 1997).

Short-term exposure to high concentrations of vapor (>500 ppm) may cause irritation of the eyes, nose, and respiratory tract and possibly signs of central nervous system depression e.g., headache, dizziness, and mental confusion. Inhalation of 20,000 ppm gasoline vapor for five minutes has been shown to be fatal (Sciences International, 1995b). No studies are available which indicate that gasolines cause adverse reproductive effects in humans.

Marketed gasolines contain proprietary chemical additives, e.g., detergents, dyes, and antiknock compounds. In general, these additives are present at low concentrations (<0.1%) and are unlikely to influence the toxicity of gasoline product. Alkyl lead compounds, such as tetraethyl and tetramethyl lead, are toxic; however, they are present at low concentrations (typically with a maximum of 1.12 g/l lead in certain aviation gasolines). Unleaded gasolines commonly contain oxygenates, e.g., up to 10% ethanol, 3% methanol, or 15% methyl tertiary butyl ether. In terms of acute toxicity, only the presence of methanol causes concern (CONCAWE, 1997).

The International Agency for Research on Cancer (IARC) concluded that limited evidence exists for the carcinogenicity of unleaded gasoline in animals. The studies were inadequate in demonstrating increased carcinogenic risk in humans. The IARC Working Group did find that some components of gasoline, especially benzene, are carcinogenic in humans, and concluded that gasoline is possibly carcinogenic in humans (International Agency for Research on Cancer, 1989); (Hathaway, *et al.,* 1991).

Middle Distillate Fuels. Petroleum materials boiling between 145 and 450°C, which include all fractions derived from crude oil between naphtha and residuum are referred to as middle distillate fuels. These include kerosenes (aviation turbine fuels), diesel fuels, and heating oils (CONCAWE, 1995); (CONCAWE, 1994). Vapor pressure depends on composition, e.g., Kerosene @ 20°C = 0.48 mmHg.

Toxicity/Health Aspects: It is reported that inhalation of middle distillate fuel aerosols and vapors causes respiratory irritation, headaches, dizziness, mental confusion, and unconsciousness (CONCAWE, 1994); (CONCAWE, 1997). Middle distillate liquids and mists can cause transient eye irritation, while prolonged exposure can cause conjunctivitis (CONCAWE, 1994).

Middle distillate fuels can defat the skin which may lead to skin irritation following prolonged and repeated exposures (American Petroleum Institute, 1967); (Gerarde, 1960). Although of a low order of oral toxicity, middle distillate fuels, if ingested, may be aspirated into the lungs causing severe chemical pneumonitis, which can be fatal (Swanson, 1985); (CONCAWE, 1997).

Some straight-run fuels (kerosenes and gas oils) in this class exhibit weak carcinogenic potential when repeatedly applied to the skin of animals over the course of their lifetime (CONCAWE, 1994); (CONCAWE, 1995). These products contain very low concentrations of 3-7 ring polycyclic aromatic hydrocarbons (PAHs) which are generally regarded as being responsible for the dermal carcinogenicity of petroleum products (CONCAWE, 1994). Recent toxicological data suggest that the carcinogenic potential of these materials is dependent on a mechanism involving chronic irritation caused by repeated application to the skin (Freeman, *et al.,* 1993). Some diesel fuels and heating oils are composed of heavier atmospheric, vacuum, or cracked gas oil components, which can contain carcinogenic PAHs. Some of these components have been found to cause skin cancer in laboratory animals following repeated and prolonged exposures (CONCAWE, 1995). Middle distillate fuels represent a negligible skin cancer risk, provided excessive skin contact is avoided (CONCAWE, 1994). No information is available on the developmental or reproductive effects of middle distillates in humans.

Middle distillate fuels may contain chemical additives such as antioxidants, flow/combustion improvers, detergents, or corrosion inhibitors. These additives are usually present at concentrations that do not add to the toxicity of the fuel itself (CONCAWE, 1997).

Heavy Fuel Oil. The residue from refinery distillation or cracking processes constitutes heavy fuel oil. It contains saturated, aromatic, and olefinic hydrocarbons mainly in the range C_9-C_{50}, boiling in the range 160-600°C. (CONCAWE, 1998); (CONCAWE, 1997). Vapor pressure @20°C = negligible.

Toxicity/Health Aspects: Significant concentrations of hydrogen sulfide can accumulate in storage tanks which contain heavy fuel oils (CONCAWE, 1997). Appropriate precautions should be exercised to prevent exposure to this hazardous gas. Accidental exposure to heavy fuel oil does not present a systemic toxicity hazard following a single oral, dermal, or inhalation exposure. However, ingestion may result in transient gastrointestinal effects. Physical contact with the eyes may produce temporary irritation and discomfort. Heavy fuel oils are unlikely to be irritating to the skin following a single exposure, but repeated or prolonged contact may cause dermatitis and possibly skin cancer due to the presence of carcinogenic polycyclic aromatic hydrocarbons (PAHs) (CONCAWE, 1997); (CONCAWE, 1998). The low vapor pressure of heavy fuel oil prevents the formation of significant vapor concentrations at ambient temperatures. Elevated temperatures and mechanical action may lead to the formation of vapors and mists, which can cause eye, nose, and respiratory irritation.

Asphalt (CAS No. 8052-42-4). Asphalt is a complex combination of high molecular weight organic compounds containing a relatively high proportion of saturated and aromatic hydrocarbons having carbon numbers greater than C25. Asphalt may be mixed with volatile petroleum diluents or oxidized to modify its physical or chemical properties (CONCAWE, 1997). Vapor pressure @ 20°C is usually negligible but depends on composition.

Toxicity/Health Aspects: The greatest acute hazard is burns from contact with hot asphalt (American Conference of Government Industrial Hygienists, 1997). At ambient temperature, asphalt is non-volatile. Reports of reduced appetite, fatigue, laryngeal/pharyngeal irritation and cough following exposure to asphalt may be due to the presence of solvents added to the asphalt. Contact with asphalt has been reported to cause dermatitis (CONCAWE, 1997). Generally, the potential for undiluted asphalt to cause skin and eye irritation is considered to be low (CONCAWE, 1992b). The acute toxic health concerns for asphalt are also considered to be low. Asphalt may contain traces of highly toxic hydrogen sulfide which may accumulate during storage. Appropriate precautions should be exercised to prevent exposure to hydrogen sulfide. The International Agency for Research on Cancer (IARC) stated that there was inadequate evidence that bitumens (asphalt) alone are carcinogenic to humans, but there was sufficient evidence of animal carcinogenicity of certain asphalt extracts (International Agency for Research on Cancer, 1985).

Components of Concern

Benzene (CAS No. 71-43-2). Colorless liquid. Vapor Pressure @ 20°C = 76 mmHg.

Inhalation/Contact with Vapor or Fumes: Acute benzene exposure causes central nervous system depression. Human exposure to approximately 20,000 ppm is fatal in 5 to 10 minutes. Convulsive movements followed by unconsciousness follow severe exposures (Hathaway, *et al.,* 1991). Brief exposures to concentrations in excess of 3,000 ppm are irritating to the eyes and respiratory tract. Continued exposure may cause euphoria, nausea, a staggering gait, and coma (Gerarde, 1960). Exposures to lower concentrations (250 to 500 ppm) are reported to cause vertigo, drowsiness, headache, and nausea (Department of Labor, 1977).

Prolonged and repeated exposure to low concentrations (above 1 ppm) of benzene has been found to adversely affect the blood-forming system (Collins, *et al.,* 1991); (Tsai, *et al.,* 1983); (Yardley-Jones, *et al.,* 1988). Long term exposure causes a reduction in the numbers of red cells, white cells, and/or platelets produced by the bone marrow (anemia, leukopenia, and thrombocytopenia, respectively) (Department of Labor, 1977). Aplastic anemia, the depression of all three blood cell types as well as bone marrow necrosis, may also develop following prolonged exposure (Goldstein, 1977). Typical symptoms of aplastic anemia include light-headedness, headache, loss of appetite, abdominal discomfort, weakness, blurring of vision, labored breathing, pale mucous membranes, and easy bruising (Committee on Toxicology of the National Research Council, 1976). Eventually, aplastic anemia may lead to death due to infection or hemorrhage. The International Agency for Research on Cancer (IARC) classified benzene as a proven human carcinogen, based on studies of leukemias (International Agency for Research on Cancer, 1982). Evidence linking benzene to other types of cancers is contradictory and inconsistent. Benzene does not appear to induce significant adverse reproductive effects, based on the results of laboratory animal studies (Schwetz, 1983).

Dermal: Direct contact with the skin may cause erythema and vesiculation. Prolonged or repeated exposure to benzene may lead to dermatitis, which may lead to secondary infections.

Benzene is absorbed through the skin and may contribute somewhat to systemic toxicity (Susten, 1985). The permeability coefficient of benzene in human skin (K_p) is $1x10^{-1}$ cm/hr (Environmental Protection Agency, 1992).

n-Hexane. (CAS No. 110-54-3). Colorless liquid.

Inhalation/Contact with Vapor: n-Hexane is an upper respiratory irritant and causes central nervous system depression. Exposure to greater than 1,000 ppm is reported to cause dizziness, nausea, headache, numbness of the limbs, and confusion; no effects were seen below 500 ppm (Cavender, 1998); (Hathaway, *et al.,* 1991). An exposure of 880 ppm for 15 minutes can cause eye and respiratory tract irritation (Cavender, 1998).

Repeated exposure to 400-600 ppm n-hexane has been linked to damage to the peripheral nerves which eventually reverses following cessation of exposure. Symptoms include muscle weakness, sensation disturbances, pain in the extremities, and atrophy. (National Institute for Occupational Safety and Health, 1977); (Jorgensen and Cohr, 1981); (Spencer, *et al.,* 1980). Based on animal studies, n-hexane is not expected to cause birth defects in humans. There have been no reports of sterility or other adverse effects on the human reproductive system due to n-hexane exposure (Research Triangle Institute, 1997). n-Hexane is not categorized as a carcinogen by the US Environmental Protection Agency or the International Agency for Research on Cancer.

Dermal: n-Hexane contact causes skin irritation (National Institute for Occupational Safety and Health, 1977). n-Hexane is absorbed into the skin; however, the rate of skin resorption is 100-fold lower for n-hexane than for benzene (Research Triangle Institute, 1997). (Permeability coefficient K_p value is not available for n-hexane).

Hydrogen Sulfide (CAS No. 7783-06-4). Colorless gas. Vapor pressure @ 20° C = 13,376 mmHg.

Inhalation: Hydrogen sulfide is a highly toxic gas with an offensive odor that is immediately lethal in concentrations greater than 2,000 ppm. Inhalation of 1 or 2 breaths of air containing 5,000 ppm hydrogen sulfide causes unconsciousness (Environmental Protection Agency, 1993). At concentrations of 500 to 1,000 ppm, respiratory paralysis is preceded by a period of rapid breathing, and death will result unless the victim is removed from the contaminated area and given artificial ventilation (Yant, 1930). Lethality appears to be due to a lack of oxygen in brain and heart tissues, through a mechanism similar to that of cyanide (Environmental Protection Agency, 1993). Hydrogen sulfide produces irritation of the mucous membranes, particularly those of the respiratory tract and eyes. Due to its low water solubility, hydrogen sulfide penetrates deeply into the respiratory tract, rendering it capable of causing lung alveolar injury leading to acute pulmonary edema (excessive accumulation of fluids in the lung) (Guidotti, 1994). Pulmonary edema occurs at sublethal concentrations (250 to 500 ppm). Pulmonary edema has also been reported after long-term exposure to levels as low as 50 ppm. Concentrations of 150 ppm or less are reported to produce incoherence and to affect the ability to think logically (Environmental Protection Agency, 1993). Prolonged exposure to hydrogen sulfide at concentrations above 50 ppm causes dryness and inflammation of the epithelia of the respiratory tract. The epithelia of the eye, especially the conjunctiva and cornea, are similarly affected. Ocular effects resulting from hydrogen sulfide exposure include inflammation, tearing, changes in visual acuity, perception of blue or rainbow colors around lights, and possibly ulceration of the cornea leading to permanent scarring (Luck and Kaye, 1989). Other symptoms reported following subacute exposure include headache, fatigue, irritability, vertigo, loss of libido, and gastrointestinal disturbances such as nausea, abdominal cramping, vomiting, and severe diarrhea (Environmental Protection Agency, 1993); (Ahlborg G, 1951); (Hathaway, *et al.,* 1991).

The lower limit for detection of odor for hydrogen sulfide is reported to be 0.003-0.02 ppm. However, olfactory sensation is lost at 150 to 200 ppm; hence, the characteristic rotten egg odor of hydrogen sulfide is not sufficient to warn of lethal exposure. Similarly, it should not be assumed that pain from the irritant effect, especially in the eyes, will warn of dangerous exposure since the gas anesthetizes the nerve endings in the mucous membranes (Environmental Protection Agency, 1993).

Hydrogen sulfide is not considered to be a cumulative poison since it is rapidly oxidized to sulfates and excreted by the kidneys (Curtis, *et al.,* 1972). Some permanent or persistent neurological effects have been reported following acute high level exposures (Guidotti, 1994); (Wasch, *et al.,* 1989). The extent to which oxygen deprivation plays a role in these effects is unclear. Chronic exposure to hydrogen sulfide below 50 ppm may cause fatigue, headache, dizziness, and irritability (Hathaway, *et al.,* 1991). There are insufficient data to determine if hydrogen sulfide causes mutagenic, carcinogenic, or reproductive effects.

Dermal: Absorption through the skin appears to be minimal, but absorption through the nasal and lung mucosa occurs readily (Environmental Protection Agency, 1993).

Naphthalene. (CAS No. 91-20-3). White crystalline solid. Vapor pressure @ 20°C = 0.08 mmHg.

Inhalation/Contact with Vapor or Fumes: Naphthalene vapor is reported to cause headache, nausea, and confusion, although occupational poisoning from vapor exposure is rare (Gosselin, *et al.,* 1984); (Hathaway, *et al.,* 1991). Concentrations of 15 ppm cause eye irritation. Cataracts have been reported in workers exposed to high concentrations of fume or vapors for 5 years (Grant, 1986). Although ingestion of naphthalene causes damage to the red blood cells (hemolysis) leading to anemia and other blood disorders, this effect is only seen by inhalation at very high vapor concentrations or in individuals with a specific genetic defect (Sciences International, 1995a).

Dermal: Skin contact may cause skin irritation and, in a sensitized person, severe dermatitis. Absorption through the skin may be enhanced if naphthalene is mixed with oil (Sciences International, 1995). The predicted Permeation Coefficient (K_p) for naphthalene is 6.9×10^{-2} cm/hr (Environmental Protection Agency, 1992).

The evidence available does not indicate that naphthalene is a human carcinogen. No studies are available which indicate that naphthalene causes reproductive or developmental effects in humans following inhalation or dermal exposure.

Polycyclic Aromatic Hydrocarbons. Polycyclic aromatic hydrocarbons (PAHs) are a group of organic compounds composed of three or more fused aromatic rings, containing only carbon and hydrogen atoms (Stony Brook Laboratories Inc., 1995); (Agency for Toxic Substances and Disease Registry, 1993). They are a subset of broader chemical groups known as polynuclear aromatic compounds (PNA) and polyaromatic compounds (PAC). PAHs occur naturally in crude oil and may become concentrated in certain high boiling (>340°C) petroleum products during refining. Vapor pressure is negligible at 20°C.

Carcinogenic PAHs: Not all PAHs contained in petroleum products are carcinogens. Certain PAHs with four to seven rings have been demonstrated to be carcinogens, while PAHs with fewer than four, or more than seven, rings generally have not shown carcinogenicity. Benzo[a]pyrene, benz[a]anthracene, and dibenz[a,h]anthracene are examples of PAHs found in certain high boiling petroleum products that are classified as probably carcinogenic to humans (Stony Brook Laboratories Inc., 1995); (Agency for Toxic Substances and Disease Registry, 1993).

Inhalation: Exposure to PAHs is possible through the inhalation of smoke and fumes generated by the combustion of petroleum products. However, due to the low volatility of PAHs, dermal contact, not inhalation, is the major route of exposure to those dealing with spills of PAH-containing petroleum products. Under conditions where an oil mist is formed, inhalation may contribute to PAH toxicity (CONCAWE, 1992a). Studies in animals indicate that repeated inhalation exposure to carcinogenic PAHs causes lung and respiratory tract cancer (Schulte, *et al.,* 1993); (Thyssen, *et al.,* 1981).

Dermal: Prolonged and repeated contact with petroleum products containing significant concentrations of PAHs has been shown to cause skin cancer. Tumors generally occur at the site

of contact, but when metabolites of the PAHs are transported to other sites in the body, tumors may also occur elsewhere (Stony Brook Laboratories Inc., 1995).

Absorption of benzo[a]pyrene into the skin is expected to be rapid (Research Triangle Institute, 1995); however, substantial metabolism and binding in the skin limits what is available to enter the systemic circulation (Research Triangle Institute, 1995). The permeability coefficient for benzo[a]pyrene is estimated to be 1.2 cm/hr (Environmental Protection Agency, 1992).

There is insufficient information available to determine if PAH exposure causes reproductive or developmental effects in humans.

Tetramethyl and Tetraethyl Lead. (CAS Nos. 75-74-Land, 78-00-2). Colorless liquids. Vapor pressure at 20°C = 23 mmHg and 0.2 mmHg.

Inhalation: Tetramethyl lead (TML) is believed to produce effects identical to tetraethyl lead (TEL), but of less intensity (ILO, 1983). TML and TEL affect the central nervous system and cause mental aberrations, including psychosis, mania, and convulsions. Intoxication occurs following brief exposures to high levels of TEL (100 mg/m^3 for 1 hour) or prolonged exposures at lower concentrations (American Conference of Governmental Industrial Hygienists, 1992a); (Kehoe, 1983). Initial symptoms include weakness, fatigue, headache, nausea, vomiting, and diarrhea. These signs are often followed by insomnia, incoordination, tremors, slowed heartbeat, and hypothermia. Severe intoxication causes recurrent or nearly continuous episodes of disorientation, irritability, hallucinations, facial contortions, intense hyperactivity, and may result in coma and death. If death does not occur, recovery may take weeks to months, and complete recovery may never occur (American Conference of Governmental Industrial Hygienists, 1992a); (American Conference of Governmental Industrial Hygienists, 1992b); (Grandjean, 1983); (Hathaway, *et al.,* 1991). Psychological relapses during recovery are common (Hathaway, *et al.,* 1991).

Dermal: TML and TEL can be readily absorbed through the skin in amounts that cause systemic poisoning, although TEL is thought to be absorbed more quickly than TML in humans

(American Conference of Governmental Industrial Hygienists, 1992a); (Hathaway, *et al.,* 1991). A single exposure to TEL can be fatal (Grandjean, 1983). As such, TML and TEL absorbed through the skin are a human developmental or reproductive hazard.

The International Agency for Research on Cancer (IARC) concluded that there is inadequate evidence for carcinogenicity for organolead compounds in humans (International Agency for Research on Cancer, 1987).

Toluene. (CAS No. 108-88-3). Colorless liquid. Vapor pressure at 20°C = 22 mmHg.

Inhalation/Contact with Vapor: The primary effect of toluene is on the nervous system. Exposures to extremely high concentrations of toluene (15,000-30,000 ppm) may cause mental confusion, loss of coordination, and unconsciousness within a few minutes (Hathaway, *et al.,* 1991). Concentrations of 800 ppm or higher have been shown to produce rapid eye and respiratory tract irritation, nasal discharge, drowsiness, ataxia, and dizziness. Toluene concentrations from 100 to 600 ppm have been reported to produce headache, eye irritation, lacrimation, mild irritation of the upper respiratory tract, lassitude, and nausea (Gosselin, *et al.,* 1984).

Workers repeatedly exposed to 50 to 200 ppm toluene over 1 to 3 weeks reported headache, lassitude, and loss of appetite while those repeatedly exposed to 200-500 ppm exhibited impaired coordination, increased reaction time, and transient memory loss. Repeated exposures to toluene concentrations above 500 ppm also caused muscular fatigue, nervousness, palpitations, and insomnia (National Institute for Occupational Safety and Health, 1973); (Department of Labor, 1975); (von Oettingen, *et al.,* 1942); (Low, *et al.,* 1988); (Wilson, 1943). There is no conclusive evidence that toluene exposure leads to irreversible damage to the nervous system.

There is no evidence that toluene causes developmental toxicity at current occupational exposure limits. Chronic inhalation abuse of toluene during pregnancy has been associated with birth defects (Hersh, 1989). There is no evidence that toluene is carcinogenic.

Dermal: Toluene is a slight to moderate dermal irritant. Repeated or prolonged skin contact removes lipids from the skin and may cause dryness, fissures, and contact dermatitis. (Browning, 1965); (Gerarde, 1960). Accidental contact of liquid toluene with the eyes has produced transient injury to the eyes with no loss of vision. There is no evidence that toluene causes skin sensitization. Toluene is absorbed through the skin and may contribute to systemic toxicity. Permeability coefficient of toluene in human skin is (K_p) cm/hr (Environmental Protection Agency, 1992).

Trimethyl Benzene: **Mesitylene** (1,3,5-trimethylbenzene) (CAS No. 108-67-8), **Pseudocumene** (1,2,4-trimethylbenzene) (CAS No. 95-63-6), and **Hemimelitene** (1,2,3-trimethylbenzene) (CAS No. 526-73-8) **Mixed Trimethyl Benzene** (CAS No. 25551-13-7). Colorless liquid composed of three isomers, mesitylene (1,3,5-trimethylbenzene), pseudocumene (1,2,4-trimethylbenzene), and hemimellitene (1,2,3-trimethylbenzene). Vapor pressure @ 20°C ranged 1-2 mmHg.

Inhalation or Contact with Vapor: Exposure to trimethyl benzene at up to 60 ppm was reported to cause impairment of the respiratory, nervous, and blood-forming systems. Symptoms included headaches, drowsiness, and anemia (American Conference of Governmental Industrial Hygienists, 1992); (Hathaway, *et al.,* 1991). In animals, concentrations of greater than 7,000 ppm mesitylene and pseudocumene for 2 hours resulted in depression of the central nervous system. Trimethyl benzene is a skin and respiratory tract irritant and presumably an eye irritant (American Conference of Governmental Industrial Hygienists, 1992); (Hazardous Substances Data Bank, 1999).

Dermal: Repeated dermal exposure is expected to cause drying and cracking of the skin (Browning 1987). A permeability coefficient value (K_p) is not available for trimethyl benzene.

The evidence available does not indicate that trimethyl benzene is a human carcinogen. No studies are available which indicate that trimethyl benzene causes reproductive or developmental effects in humans following inhalation or dermal exposure.

Section 4

SUMMARY AND CONCLUSIONS

In order to understand the possible health hazards encountered during oil spill response activities, it is necessary to identify and review health hazards of the key components of concern. Environmental factors, the identity of the product spilled, and the nature of the spill or leak also affect the potential human health risks.

- The most toxic constituents naturally found in crude oils and petroleum products include benzene, hydrogen sulfide, n-hexane, trimethylbenzene, toluene, naphthalene, polycyclic aromatic hydrocarbons (PAHs), and may include organic lead additions in specific products. The components of concern vary depending on their concentrations in the specific oil or product.

- Environmental mechanisms such as evaporation, spreading, dissolution, dispersion, etc. and the chemical characteristics of the spilled material will affect potential exposure risks to oil spill cleanup workers.

- Time also greatly affects the potential exposure risks with the initial period having the highest potential for airborne contaminant exposure as volatile components begin to evaporate from the surface. After weathering, skin exposure hazards are the most significant.

- All of the above factors were considered in constructing Table 3-1, which generically estimates broad levels of concern for each constituent of each product. It is meant to provide rough guidance, and modification based on the specific spill situation will need to be considered.

This report provides an overview of the possible human health hazards associated with exposure to petroleum and petroleum products during spill response operations. Assessment and control of these health hazards may be covered in subsequent reports.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). 1993. Polycyclic Aromatic Hydrocarbon Toxicity. *American Family Physician*. pp. 47623-628.

Ahlborg G. 1951. Hydrogen Sulphide Poisoning in Shale Oil Industry. *AMA Archives of Industrial Hygiene and Occupational Medicine*. pp. 3247-266.

American Conference of Governmental Industrial Hygienists (ACGIH). 1992. Trimethyl Benzene Isomers, In *Documentation of the Threshold Limit Values and Biological Exposure Indices.* Cincinnati, OH. pp. 1648-1649

American Conference of Governmental Industrial Hygienists (ACGIH). 1992a. Tetraethyl lead, In *Documentation of the Threshold Limit Values and Biological Exposure Indices.* Cincinnati, OH. pp. 1513-1516.

American Conference of Governmental Industrial Hygienists (ACGIH). 1992b. Trimethyl Benzene Isomers, In *Documentation of the Threshold Limit Values and Biological Exposure Indices.* Cincinnati, OH. pp. 1648-1649.

American Conference of Governmental Industrial Hygienists (ACGIH). 1997. Asphalt (Petroleum)(Bitumen) Fumes, *In Documentation of the Threshold Limit Values and Biological Exposure Indices*. Cincinnati, OH.

American Conference of Governmental Industrial Hygienists (ACGIH). 1998. *Guide to Occupational Exposure Values - 1998,* Compiled by the American Conference of Governmental Industrial Hygienists. Cincinnati, OH.

American Petroleum Institute (API). 1967. API *Toxicological Review of Kerosene*, New York, NY.

American Petroleum Institute (API). 1997. *Petroleum in the Freshwater Environment. An Annotated Bibliography 1946-1997.* API Publication Number 4640. American Petroleum Institute. Washington, D.C.

American Petroleum Institute (API). 1999. *Fate and Environmental Effects of Oil Spills in Freshwater Environments*. API Publication Number 4675, American Petroleum Institute. Washington, D.C.

Browning, E. 1965. *Toxicity and Metabolism of Industrial Solvents.* Elsevier, NY.

Browning, E.C. 1987. *Hydrocarbons.* Elsevier, NY.

Cavender, F. 1998. Aliphatic Hydrocarbons, In *Patty's Industrial Hygiene and Toxicology*-CD-ROM, Volume 2B (Ed, Clayton, G.D.) John Wiley & Sons. Chapter 19, p. 1233.

Collins, J.J., Conner, P., Friedlander, B.R., Easterday, P.A., Nair, R.S. and Braun, J. 1991. A Study of the Haematologic Effects of Chronic Low-Level Exposure to Benzene. *Journal of Occupational Medicine*. pp. 33619-625.

Committee on Toxicology of the National Research Council. 1976. *Health Effects of Benzene - A Review*. PB-254 388. Washington, D.C.

CONCAWE. 1985. *Health Effects of Petroleum Fuels-General Principles*. Den Haag, The Netherlands.

CONCAWE. 1992a. *Aromatic Extracts*. 92/01. Brussels, Belgium

CONCAWE. 1992b. *Bitumens and Bitumen Derivatives*. 92/104. Brussels, Belgium

CONCAWE. 1992c. *Gasolines*. 92/103. Brussels, Belgium

CONCAWE. 1994. *Kerosenes/jet*. 94/106. Brussels, Belgium

CONCAWE. 1995. *Gas Oils (diesel fuels/heating oils)*. 95/107. Brussels, Belgium.

CONCAWE. 1997. *Petroleum Products-First Aid Emergency and Medical Advice*. 1/97. Brussels, Belgium.

CONCAWE. 1998. *Heavy Fuel Oils*. 98/109. Brussels, Belgium

Curtis, C., Bartholomew, T.C., Rose, F.A. and Dodgson, K.S. 1972. Detoxication of Sodium 35S-Sulphide in the Rat. *Biochemical Pharmacology*. pp. 212313-2321.

Department of Labor. 1975. Occupational Exposure to Toluene. Volume 40 *Federal Register*. pp. 46206-46219.

Department of Labor. 1977. Occupational Exposure to Benzene. Volume 42 *Federal Register*. pp. 22516-22529.

Environmental Protection Agency (EPA). 1992. *Dermal Exposure Assessment Principles and Applications*. EPA/600/8-91/011B. Washington, D.C.

Environmental Protection Agency (EPA). 1993. *Health Assessment Document for Hydrogen Sulfide*. EPA600.08.86.026F. Research Triangle Park, NC.

Exxon Corporation. 1985. *Fate and Effects of Oil in the Seas*. Exxon Background Series. New York, NY.

Freeman, J. J., Federici, T.M. and McKee, R.H. 1993. Evaluation of the Contribution of Chronic Skin Irritation and Selected Compositional Parameters to the Tumorigenicity of Petroleum Middle Distillates in Mouse Skin. *Toxicology*. pp. 81103-112.

Fingas, M. 1994. Studies on the Evaporation of Oil Spills. pp. 189-212. In *Proceedings,* 17th Arctic and Marine Oil Spill Program. June 8-10, 1994. Vancouver, British Columbia.

Fingas, M. 1995. The Evaporation of Oil Spills. pp. 43-60 in *Proceedings,* 18th Arctic and Marine Oil Spill Program. June 14-16, 1995. Edmonton, Alberta.

GESAMP (Joint Group of Experts on the Scientific Aspects of Marine Pollution). 1993. Impact of Oil and Related Chemicals and Wastes on the Marine Environment. *GESAMP Reports and Studies.* No. 50. 180 pp.

Gerarde, J.W. 1960. *Toxicology and Biochemistry of Aromatic Hydrocarbons*. Elsevier, NY.

Goldstein, B.D. 1977. Hematotoxicity in Humans, In *Journal of Toxicology and Environmental Health Supplement*, Volume 2 (Editors: Laskin, S. and Goldstein, B.D.). pp. 69-105.

Gosselin, R.E., Smith, R.P., and Hodge, H.C. 1984. *Clinical Toxicology of Commercial Products.* Williams and Wilkins. Baltimore, MD.

Grandjean, P. 1983. Organolead Exposures and Intoxications, In *Biological Effects of Organolead Compounds* (Editor, Grandjean, P.), CRC Press. Boca Raton, FL. pp. 1-278.

Grant, W.M. 1986. *Toxicology of the Eye*. Charles C. Thomas. Springfield, IL.

Guidotti, T.L. 1994. Occupational Exposure to Hydrogen Sulfide in the Sour Gas Industry, Some Unresolved Issues. *Archives of Occupational and Environmental Health*. pp. 66153-160.

Hathaway, G.J., Proctor, N.H., Hughes, J.P., and Fischman, M.L. 1991. *Proctor and Hughes' Chemical Hazards of the Workplace*. Van Nostrand Reinhold, New York, NY.

Hazardous Substances Data Bank. 1999. *Trimethyl Benzene*, (Editor, National Library of Medicine). MICROMEDEX. Englewood, CO.

Hersh, J.H. 1989. Toluene Embryopathy: Two New Cases. *Journal of Medical Genetics*. pp. 26333-337.

Holliday, M.G. and Park, J.M. 1993. *Occupational Health Implications of Crude Oil Exposure: Literature Review and Research Needs.* Marine Spill Response Corporation (MSRC). Technical Report Series. pp. 93-007, Washington, D.C.

ILO. 1983. *Encyclopedia of Occupational Health and Safety.* International Labour Office. Geneva, Switzerland.

International Agency for Research on Cancer (IARC). 1982. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, In *Some Industrial Chemicals and Dyestuffs*. Volume 29. Lyon, France. pp. 93-148.

International Agency for Research on Cancer (IARC). 1985. Bitumens, Coal-Tars and Derived Products, Shale Oils and Soots, In *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans*. Volume 35. Lyon, France.

International Agency for Research on Cancer (IRAC). 1987. *Monographs on the Evaluation of Carcinogenic Risks to Humans, Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs*. In Supplement. 7, Volume 1 to 42. IARC. Lyon, France. pp. 230-232.

International Agency for Research on Cancer. (IRAC) 1989. Crude Oil, In *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*. Volume 45. IARC. Lyon, France. pp. 119-158.

Jorgensen, N.K. and Cohr, K.H. 1981. n-Hexane and its Toxicological Effects. *Scandinavian Journal of Work, Environment & Health*. pp. 7157-168.

Kehoe, R. 1983. Lead, Alkyl Compounds, In *Encyclopedia of Occupational Health and Safety*. Vol. II. ILO. Geneva, Switzerland. pp. 1197-1199.

Koons, C.B. and Jahns, H.O. 1992. The Fate of Oil from the Exxon Valdez-A Perspective. *Marine Technological Society*. 26:61-69.

Low, L.K., Meeks, J.R. and Mackerer, C.R. 1988. Health Effects of the Alkylbenzenes, *Toxicology and Industrial Health*. pp. 449-75.

Luck, J. and Kaye, S.B. 1989. An Unrecognised Form of Hydrogen Sulphide Keratoconjunctivitis, *British Journal of Industrial Medicine*. pp. 46748-749.

Mielke, J.E. 1990. *Oil in the Ocean The Short- and Long-Term Impacts of a Spill*. Congressional Research Service. 90-356 SPR. The Library of Congress. Washington, D.C. 34 pp.

National Research Council (NRC). 1993. *Issues in Risk Assessment: A Paradigm for Ecological Risk Assessment.* National Academy Press. Washington, D.C.

National Research Council (NRC). 1985. Oil in the Sea. *Inputs, Fates, and Effects*. National Academy Press. Washington, D.C. 601 pp.

National Research Council (NRC). 1989. *Using Oil Spill Dispersants on the Sea*. National Academy Press. Washington, D.C. 335 pp.

National Institute for Occupational Safety and Health (NIOSH). 1973. *Criteria for a Recommended Standard-Occupational Exposure to Toluene,* NIOSH. Pub. # 73-11023. Washington, D.C.

National Institute for Occupational Safety and Health (NIOSH). 1977. *Criteria for a Recommended Standard - Occupational Exposure to Alkanes* (C5-C8). pp. 77-151. Washington D.C.

Payne, J.R., Claytown, J. R., McNabb, Jr., G.D. and Kirstein, B. 1991. *Exxon Valdez Oil Weathering Fate and Behavior: Model Predictions and Field Observations.* International Oil Spill Conference. San Diego, CA.

Research Triangle Institute. 1995. *Polycyclic Aromatic Hydrocarbons.* Agency for Toxic Substances and Disease Registry (ATSDR). Atlanta, GA.

Research Triangle Institute. 1997. *Toxicological Profile for n-Hexane, Draft Toxicological Profile.* Agency for Toxic Substances and Disease Registry (ASTDR). Atlanta, GA.

Sciences International, Inc. 1995a. *Toxicological Profile for Naphthalene, 1-Methylnaphthalene, and 2-Methylnaphthalene,* Agency for Toxic Substances and Disease Registry (ATSDR). Atlanta, GA.

Sciences International, Inc. 1995b. *Toxicological Profile for Gasoline.* Agency for Toxic Substances and Disease Registry (ATSDR). Atlanta, GA.

Schulte, A., Ernst, H. and Peters, L. 1993. Introduction of Squamous Cell Carcinomas in the Mouse Lung After Long-Term Inhalation Of Polycyclic Aromatic-Rich Exhausts. *Experimental and Toxicologic Pathology.* pp. 45415-421.

Schwetz, B.A. 1983. Carcinogenicity and Toxicity of Benzene., In *Advances in Modern Environmental Toxicology*, Volume IV (Editor, Mehlman, M. A.). Princeton Scientific, Princeton, NJ. pp. 17-21.

Smith, L. H., Haschek, W.M. and Witschi, H. 1981. Acute Toxicity of Selected Crude and Refined Shale Oil- and Petroleum-Derived Substances., *In Health Effects Investigation of Oil Shale Development* (Editors, Griest, W. H., Guerin, M. R. and Coffin, D. L.). Ann Arbor Science. Ann Arbor, MI. pp. 141-160.

Spencer, P.S., *et al.* 1980. The Enlarging View of Hexacarbon and Neurotoxicity. *CRC Critical Reviews in Toxicology.* pp. 7279-356.

Stony Brook Laboratories, Inc. 1995. *Health Effects of Polycyclic Aromatic Hydrocarbons.* CONCAWE. Brussels, Belgium.

Susten, A.S. 1985. Percutaneous Penetration of Benzene in Hairless Mice: An Estimate of Dermal Absorption During Tire Building Operations. *American Journal of Industrial Medicine.* pp. 7323-335.

Swanson, S.M. 1985. API Final Report of the Ad Hoc Committee on Middle Distillate Fuels. New York, NY.

Thayer, E.C. and Tell, J.G. 1999. Modeled Exposures to Freshly Spilled Crude Oil. *Proceedings of the International Oil Spill Conference.* Seattle, WA.

Thyssen, J., Althoff, J. and Mohr, U. 1981. Inhalation Studies with Benzo[a]pyrene in Syrian Golden Hamsters. *Journal of National Cancer Institute.* pp. 66575-577.

Tsai, S.P., Wen, C.P., Weiss, N.S., Wong, O., McClellan, W.A. and Gibson, R.L. 1983. Retrospective Mortality and Medical Surveillance Studies of Workers in Benzene Area of Refineries. *Journal of Occupational Medicine.* pp. 25685-692.

United States Occupational Safety and Health Administration (U.S. OSHA). 1997. Title 29 *Code of Federal Regulations* §1910.1000, 1028, 1200.

von Oettingen, W.F., Neal, P.A. and Donahue, D.D. 1942. The Toxicity and Potential Dangers of Toluene - Preliminary Report. *Journal of the American Medical Association.* pp. 113578-584.

Wasch, H.H., Estrin, W.J. and Yip, P. 1989. Prolongation of the P-300 Latency Associated with Hydrogen Sulfide Exposure. *Archives of Neurology.* 46:8.

Wilson, R.H. 1943. Toluene Poisoning. *Journal of the American Medical Association.* pp. 1231106-1108.

Yant, W.P. 1930. Hydrogen Sulphide in Industry: Occurrence, Effects, and Treatment. *American Journal of Public Health.* pp. 20598-606.

Yardley-Jones, A., Anderson, D., Jenkinson, P.C., Lovell, D. P., Blowers, S. D. and Davies, M. J. 1988. Genotoxic Effects in Peripheral Blood and Urine of Workers Exposed to Low Level Benzene. *British Journal of Industrial Medicine.* pp. 45694-700.

Additional copies are available through Global Engineering Documents at (800) 854-7179 or (303) 397-7956

Information about API Publications, Programs and Services is available on the World Wide Web at: http://www.api.org

American Petroleum Institute

1220 L Street, Northwest
Washington, D.C. 20005-4070
202-682-8000

Product No. I46890