Albers 2003

CHAPTER 14

Petroleum and Individual Polycyclic Aromatic Hydrocarbons

Peter H. Albers

CONTENTS

14.1 INTRODUCTION

Crude petroleum, refined petroleum products, and individual polycyclic aromatic hydrocarbons (PAHs) contained within petroleum are found throughout the world. Their presence has been detected in living and nonliving components of ecosystems. Petroleum can be an environmental hazard for all organisms. Individual PAHs can be toxic to organisms, but they are most commonly associated with illnesses in humans. Because petroleum is a major environmental source of these PAHs, petroleum and PAHs are jointly presented in this chapter. Composition, sources, environmental fate, and toxic effects on all organisms of aquatic and terrestrial environments are addressed.

Petroleum spills raised some environmental concern during the early twentieth century when ocean transport of large volumes of crude oil began.[1] World War I caused a large number of oil spills that had a noticeably adverse effect on marine birds. The subsequent conversion of the economy of the world from coal to oil, followed by World War II, greatly increased the petroleum threat to marine life. Efforts to deal with a growing number of oil spills and intentional oil discharges at sea continued during the 1950s and 1960s.[1] The wreck of the *Torrey Canyon* off the coast of England in 1967 produced worldwide concern about the consequences of massive oil spills in the marine environment. Research on the environmental fate and biological effects of spilled petroleum increased dramatically during the 1970s. The *Exxon Valdez* oil spill in Prince William Sound, Alaska, in 1989, and the massive releases of crude oil into the Arabian Gulf during the 1991 Gulf War again captured international attention and resulted in an increase in environmental research. Despite considerable progress in developing methods to clean up spills, the adoption of numerous national and international controls on shipping practices, and high public concern (e.g., passage of the Oil Pollution Act of 1990 [33 USCA Sec. 2701-2761] in the United States), petroleum continues to be a widespread environmental hazard.

The association between skin cancer in chimney sweeps and exposure to contaminants in soot was made in England during the late eighteenth century. By the early twentieth century, soot, coal tar, and pitch were all known to be carcinogenic to humans. In 1918 benzo(a)pyrene was identified as a major carcinogenic agent; other PAHs have since been identified as carcinogenic or tumorigenic. Many toxic and carcinogenic effects of PAHs on humans, laboratory animals, and wildlife have been described in numerous reviews.[2]

14.2 COMPOSITION AND CHARACTERISTICS

14.2.1 Petroleum

Petroleum consists of crude oils and a wide variety of refined oil products. Crude oils vary in chemical composition, color, viscosity, specific gravity, and other physical properties. Color ranges from light yellow-brown to black. Viscosity varies from free flowing to a substance that will barely pour. Specific gravity of most crude oils varies between 0.73 and 0.95.[3] Refined oil products most often spilled in large quantities are aviation fuel, gasoline, No. 2 fuel oil (diesel fuel), and No. 6 fuel oil (bunker C). Fuel oils (Nos. 1 to 6) become increasingly dense and viscous and contain increasingly fewer volatile compounds as their numeric classification proceeds from one to six.

Crude oil is a complex mixture of thousands of hydrocarbon and nonhydrocarbon compounds. Hydrocarbons comprise more than 75% of most crude and refined oils; heavy crude oils can contain more than 50% nonhydrocarbons.[3,4] Hydrocarbons in petroleum are divided into four major classes: (1) straight-chain alkanes (n-alkanes or n-paraffins), (2) branched alkanes (isoalkanes or isoparaffins), (3) cycloalkanes (cycloparaffins), and (4) aromatics (Figure 14.1). Alkenes occur in crude oil, but they are rare. A variety of combinations of the different types of compounds occur. Low-molecular-weight members of each class predominate in crude oils. Aliphatic hydro-

Figure 14.1 Type
omitt

carbons have t
hydrocarbons h
containing six c
(except alkenes)
do not have ma
between carbon
sulfur (S), nitro
and can range f
and cycloalkane
carbons are the
Crude oils
gravity determi
partitioned into
in various categ

14.2.2 PAHs

Polycyclic
rings that have
from naphthale
oils contain 0.
increases with
melting and bo
increase, and
biological effe
omatic rings, o
hydrogen (Fig

hydrocarbons
nce has been
environmental
ost commonly
ource of these
rces, environ-
are addressed.
century when
number of oil
ersion of the
the petroleum
oil discharges
f the coast of
il spills in the
led petroleum
illiam Sound,
the 1991 Gulf
ntal research.
of numerous
g., passage of
um continues

inants in soot
ry, soot, coal
was identified
tumorigenic.
wildlife have

e oils vary in
Color ranges
at will barely
roducts most
l), and No. 6
and contain
one to six.
compounds.
ude oils can
led into four
kanes (isoal-
4.1). Alkenes
f compounds
hatic hydro-

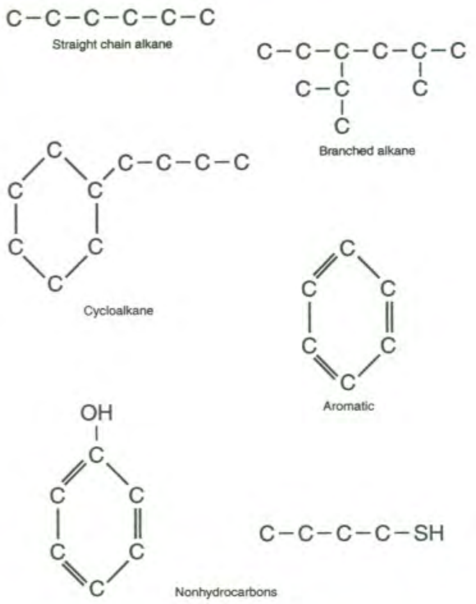

Figure 14.1 Types of molecular structures found in petroleum.[3] Hydrogen atoms bonded to carbon atoms are omitted.

carbons have the carbon atoms arranged in an open chain (straight or branched). Aromatic hydrocarbons have the carbon atoms arranged in ring structures (up to six), with each ring containing six carbon atoms with alternating single and double bonds. The aliphatic hydrocarbons (except alkenes) have maximum hydrogen content (saturated), whereas the aromatic hydrocarbons do not have maximum hydrogen content (unsaturated) because of the alternating double bonding between carbon atoms. Nonhydrocarbons in petroleum are compounds containing oxygen (O), sulfur (S), nitrogen (N) (Figures 14.1 and 14.3), or metals, in addition to hydrogen and carbon, and can range from simple open-chain molecules to molecules containing 10 to 20 fused aromatic and cycloalkane carbon rings with aliphatic side chains. The largest and most complex nonhydrocarbons are the resins and asphaltenes.

Crude oils are classified according to physical properties or chemical composition.[3,5] Specific gravity determines whether oil is classified as light, medium, or heavy. Crude oils also can be partitioned into chemical fractions according to boiling point. The relative amounts of compounds in various categories are sometimes used to classify oil (e.g., paraffinic, napthenic, high or low sulfur).

14.2.2 PAHs

Polycyclic aromatic hydrocarbons are aromatic hydrocarbons with two or more fused carbon rings that have hydrogen or an alkyl (Cn H2n+1) group attached to each carbon. Compounds range from naphthalene (C10 H8, two rings) to coronene (C24 H12, seven rings) (Figure 14.2). Crude oils contain 0.2 to 7% PAHs, with configurations ranging from two to six rings; PAH content increases with the specific gravity of the oil.[6–8] In general, PAHs have low solubility in water, high melting and boiling points, and low vapor pressure. Solubility decreases, melting and boiling points increase, and vapor pressure decreases with increasing molecular volume. Investigators assessing biological effects sometimes group true PAHs with compounds consisting of aromatic and nonaromatic rings, or compounds with N, S, or O within the ring (heterocycle) or substituted for attached hydrogen (Figure 14.3).

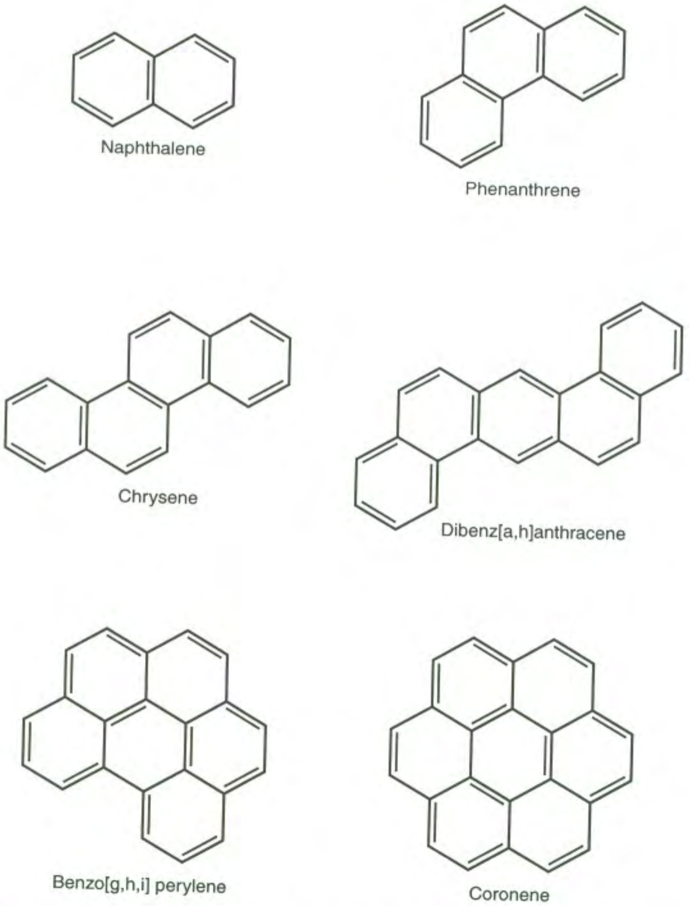

Figure 14.2 Examples of PAH compounds without attached alkyl groups.[16] Hydrogen atoms bonded to carbon atoms are omitted.

14.3 SOURCES

14.3.1 Petroleum

During the early 1970s about 35% of the petroleum hydrocarbons in the marine environment came from spills and discharges related to marine transportation; the remainder came from offshore oil and gas production, industrial and municipal discharges, stormwater discharges, river runoff, atmospheric deposition, and natural seeps[9,10] (Figure 14.4). Transportation spills and discharges probably accounted for less than 35% of the total oil discharged onto land and freshwater environments.[10] Estimates for the late 1970s indicated that about 45% of the petroleum hydrocarbons in the marine environment came from spills and discharges related to marine transportation.[11] In heavily used urban estuaries, the contribution of transportation spills and discharges to total petroleum hydrocarbon input can be 10% or less.[12,13] By contrast, the largest source of petroleum in coastal or inland areas removed from urban or industrial centers is petroleum transportation. In the 1980s and 1990s, war, terrorism, vandalism, and theft became additional, and sometimes major, causes of petroleum discharges into water and land environments.[14,15]

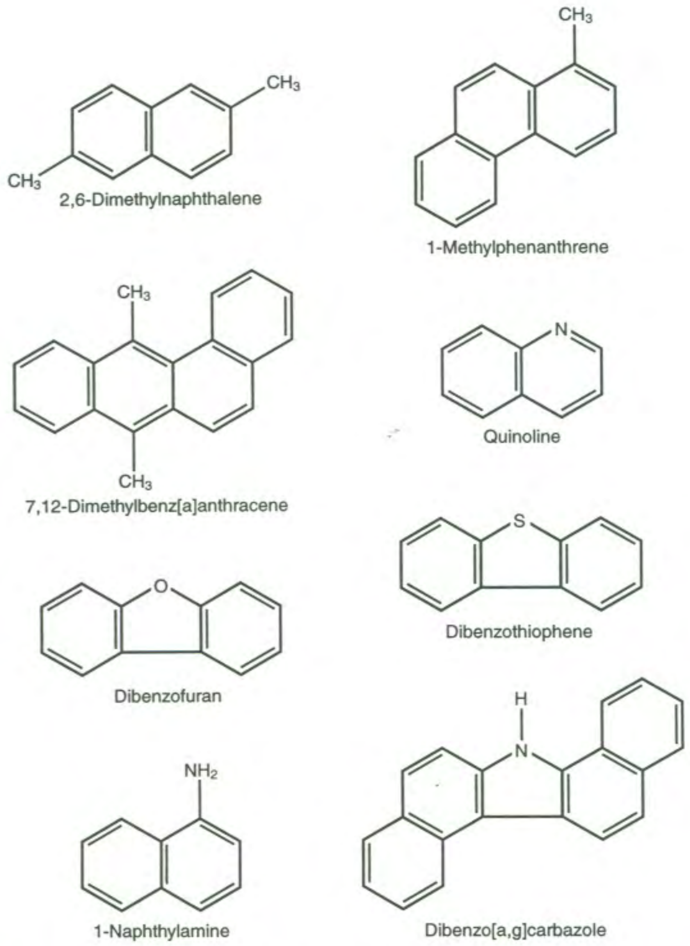

Figure 14.3 Examples of PAH compounds with attached alkyl groups and aromatic compounds with nitrogen, sulfur, or oxygen.[8] Except for the alkyl groups, hydrogen atoms bonded to carbon atoms are omitted.

14.3.2 PAHs

Most PAHs are formed by a process of thermal decomposition (pyrolysis) and subsequent recombination (pyrosynthesis) of organic molecules.[16] Incomplete combustion of organic matter produces PAHs in a high-temperature (500 to 800°C) environment. All forms of combustion, except flammable gases well mixed with air, produce some PAHs. Subjection of organic material in sediments to a low-temperature (100 to 300°C) environment for long periods of time produces PAHs as coal and oil deposits within sedimentary rock formations (a.k.a. diagenesis).[7,17] Although the PAH compounds formed by high- and low-temperature processes are similar, the occurrence of alkylated PAHs is greater in the low-temperature process.[14] Biological formation of PAHs occurs in chlorophyllous plants, fungi, and bacteria.[2,8]

Natural sources of PAHs include forest and grass fires, oil seeps, volcanoes, plants, fungi, and bacteria. Anthropogenic sources of PAHs include petroleum, electric power generation, refuse incineration, and home heating; production of coke, carbon black, coal tar, and asphalt; and internal combustion engines.[2,7] The primary mechanism for atmospheric contamination by PAHs is incom-

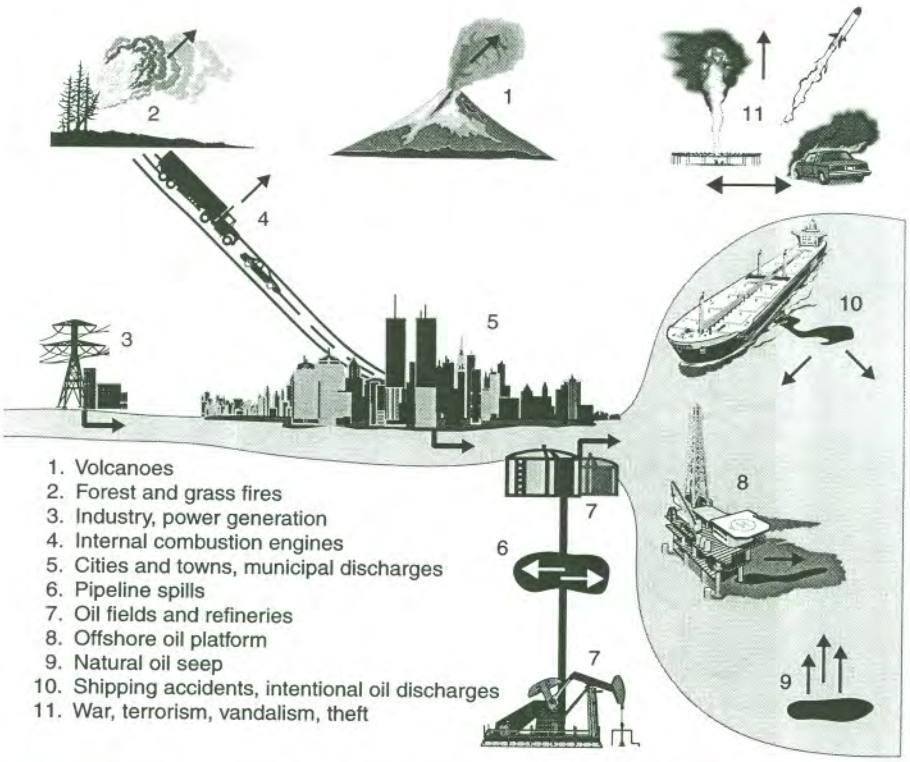

Figure 14.4 Sources of petroleum and PAHs in the environment. Arrows indicate the initial movement of PAH and petroleum into the air, water, and soil.

plete combustion of organic matter from previously mentioned sources.[15] Aquatic contamination by PAHs is caused by petroleum spills, discharges, and seepages; industrial and municipal wastewater; urban and suburban surface runoff; and atmospheric deposition.[2] Land contamination by PAHs is caused by petroleum spills and discharges, forest and grass fires, volcanoes, industrial and municipal solid waste, and atmospheric deposition.

14.4 ENVIRONMENTAL FATE

14.4.1 General Considerations

Petroleum is monitored as a liquid composed of a diverse array of thousands of compounds, but PAHs are monitored as a group of individual compounds with similar molecular structures. Polycyclic aromatic hydrocarbons from low- or high-temperature pyrolysis and pyrosynthesis of organic molecules have similar fates in the environment. Whereas PAHs from crude and refined oils and coal originate from a concentrated hydrocarbon source, PAHs produced by high temperature (combustion or industrial processes) are dispersed in the air, scattered on the ground, or included as a component of liquid waste and municipal sewage discharges.

14.4.2 Physical and Chemical

Petroleum discharged on water spreads quickly to cover large areas with a layer of oil varying from micrometers to centimeters thick. Some oils, especially heavy crudes and refined products,

---

Figure 14.5 Chem

sink and move b
currents mix th
emulsion. The fo
oil emulsions ha
“chocolate mous
eventually forms
into small pieces
Polycyclic a
airborne organic
eventually transp
Crude and r
sunlight[3] (Figure
oration varies fro
oil) to as much
5% of a crude o
drocarbons) diss
to polar oxidized
occurs when oxy
bons are particu
hydrocarbons ca
in the atmospher

14.4.3 Biolog

The moveme
exposes the mol

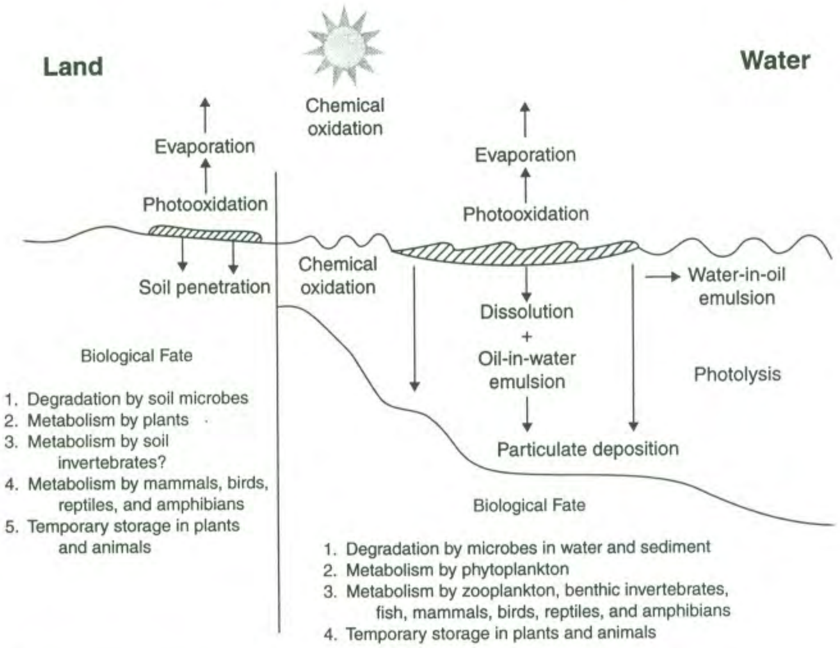

Figure 14.5 Chemical and biological fate of petroleum and PAHs in water and on land.

sink and move below the surface or along the bottom of the water body. Wave action and water currents mix the oil and water and produce either an oil-in-water emulsion or a water-in-oil emulsion. The former increasingly disperses with time, but the latter resists dispersion. Water-in-oil emulsions have 10 to 80% water content; 50 to 80% of emulsions are often described as "chocolate mousse" because of the thick, viscous, brown appearance. Oil remaining on the water eventually forms tar balls or pancake-shaped patches of surface oil that drift ashore or break up into small pieces and sink to the bottom.

Polycyclic aromatic hydrocarbons released into the atmosphere have a strong affinity for airborne organic particles and can be moved great distances by air currents. The molecules are eventually transported to earth as wet or dry particulate deposition.[18]

Crude and refined oil products begin to change composition on exposure to air, water, or sunlight[3] (Figure 14.5). Low-molecular-weight components evaporate readily; the amount of evaporation varies from about 10% of the spilled oil for heavy crudes and refined products (No. 6 fuel oil) to as much as 75% for light crudes and refined products (No. 2 fuel oil, gasoline). Less than 5% of a crude oil or refined product (primarily low-molecular-weight aromatics and polar nonhydrocarbons) dissolves in water. Hydrocarbons exposed to sunlight, in air or water, can be converted to polar oxidized compounds (photooxidation). Degradation of hydrocarbons in water by photolysis occurs when oxygen is insufficient for photooxidation; high-molecular-weight aromatic hydrocarbons are particularly likely to be altered by this mechanism.[7] Chemical oxidation of aromatic hydrocarbons can result from water and wastewater treatment operations[7] and chemical reactions in the atmosphere.[18]

14.4.3 Biological

The movement of oil from the water surface into the water column by dissolution and emulsion exposes the molecules and particles of oil to degradation and transport by organisms. Microbes (bacteria, yeast, filamentous fungi) in the water metabolize the light and structurally simple hydrocarbons and nonhydrocarbons.[3,19] Heavy and complex compounds are more resistant to microbial degradation and eventually move into bottom sediments. Oil particles and individual hydrocarbons (petroleum or recent pyrosynthesis) also adhere to particles (detritus, clay, microbes, phytoplankton) in the water and settle to the bottom, where a variety of microbes metabolize the light and structurally simple compounds. About 40 to 80% of a crude oil can be degraded by microbial action.[3]

Oil and anthropogenic PAHs are ingested by a variety of invertebrate and vertebrate organisms, in addition to microbes. Mammals, birds, fish, and many invertebrates (crustaceans, polychaetes, echinoderms, insects) metabolize and excrete some of the hydrocarbons ingested during feeding, grooming, and respiration.[2,20–24] Although bivalve mollusks and some zooplankton are either unable or marginally able to metabolize oil, they can transport and temporarily store it. Terrestrial plants and aquatic algae can assimilate and metabolize hydrocarbons.[2,25,26] Some soil invertebrates could be capable of metabolizing oil and anthropogenic PAHs, but evidence is absent from the literature.

Accumulation of hydrocarbons is usually inversely related to the ability of the organism to metabolize hydrocarbons.[2,6] For example, bivalve mollusks have a poorly developed mixed function oxygenase (MFO) capability and accumulate hydrocarbons rapidly. After acute exposure, depuration of light for structurally simple hydrocarbons, especially aliphatic hydrocarbons, is more rapid than for heavy hydrocarbons, especially aromatic hydrocarbons.[2,8,27,28] Hydrocarbons accumulated by bivalves through chronic exposure are depurated slowly.[8,29] Organisms, such as fish and some crustaceans, that possess a well-developed MFO system (microsomal monooxygenases) are capable of metabolizing hydrocarbons and only accumulate hydrocarbons in heavily polluted areas.[2] Aquatic environmental factors that reduce the potential for hydrocarbon uptake and retention include high levels of dissolved or suspended organic material and warm water temperatures. Increases in PAH accumulation have not been observed in the trophic levels of aquatic ecosystems.[8,30] Aquatic and terrestrial mammals, birds, reptiles, and amphibians have well-developed MFO systems, but enzyme induction by hydrocarbons in these species is not as well described as in fish and some aquatic invertebrates.[31] Phytoplankton can accumulate aromatic hydrocarbons from water.[2] Terrestrial plants are poor accumulators of soil PAHs, presumably because PAHs strongly adsorb to soil organic material. Most of the PAHs detected in terrestrial plants appear to be derived from the atmosphere; PAHs adhere to or are absorbed by plant tissue. Following the death of vegetation, PAHs are deposited into the surface litter of soil.[2,27,32]

Organisms with high lipid content, a poor MFO system, and that exhibit activity patterns or distributions that coincide with the location of hydrocarbon pollution are most likely to accumulate hydrocarbons.[2,8] Once assimilated, heavy aromatic hydrocarbons (four or more benzene rings) are the most difficult group of hydrocarbons to excrete, regardless of MFO capability.[2,33]

14.4.4 Residence Time

Residence time for petroleum in water is usually less than 6 months. Residence time for petroleum deposited on nearshore sediments and intertidal substrate is determined by the characteristics of the sediment and substrate. Oil-retention times for coastal environments range from a few days for rock cliffs to as much as 20 years for cobble beaches, sheltered tidal flats, and wetlands.[34–38] Microbes in the sediment and on the shoreline metabolize petroleum compounds; microbial degradation is reduced considerably by anaerobic conditions.[39,40] In cold climates ice, low wave energy, and decreased chemical and biological activity cause oil to remain in sediments or on the shore longer than in temperate or tropical climates; sheltered tidal flats and wetlands can be expected to retain oil for long periods of time.[41,42] Total petroleum hydrocarbons or individual PAHs in undisturbed estuarine subtidal sediments can remain as identifiable deposits for many decades.[43]

Oil spilled on
lakes and streams
the ocean becaus
evaporation, phot
soils are less we
(substrate type, o
in intertidal sedin
cold or temperate
in the number of

14.4.5 Spill R

Petroleum spi
initiated when ec
include mechanic
addition of chen
modify the expec
the case of dispe
biodegradation p
the stimulation o
or phosphorus to
in Prince Willia
environmental ef
and restoration n
in references[47–50]

14.5.1 Gener

Petroleum ca
modification (alt
Large discharge
habitat modifica
of the toxic effe

Polycyclic a
toxicity is repor
ciated with the n
in animals is th
PAHs to bind to
lead to mutation
PAHs have great
of alkyl groups
carcinogenic a
mutagenic with
can be increase
plasms have be
cerous neoplasn
tivity to PAH-in

Oil spilled on land has little time to change before it penetrates into the soil. Oil spilled on lakes and streams usually has less opportunity to change before drifting ashore than oil spilled on the ocean because the water bodies are smaller. Petroleum spilled directly on land is degraded by evaporation, photooxidation, and microbial action. Residence times for hydrocarbons in terrestrial soils are less well documented than for sediments but are determined by the same conditions (substrate type, oxygen availability, temperature, surface disruption) that determine residence times in intertidal sediments. Total petroleum hydrocarbons and individual PAHs can persist in soils of cold or temperate regions for at least 20 to 40 years; persistence of PAHs increases with increase in the number of benzene rings.[32,44–46]

14.4.5 Spill Response

Petroleum spills are sometimes left to disperse and degrade naturally,[47] but cleanup efforts are initiated when economically or biologically important areas are threatened. Oil-response actions include mechanical removal, chemical treatment, enhanced biodegradation, and restoration.[48,49] The addition of chemicals to floating oil for purposes of gelling, herding, or dispersing the oil can modify the expected effects of the oil. The chemicals themselves often have some toxicity, and in the case of dispersants, movement of oil into the water column is greatly accelerated. Enhanced biodegradation practices are evolving through experimentation, but the most common practice is the stimulation of indigenous microbial populations through application of supplemental nitrogen or phosphorus to oil on land or water. The addition of nitrogen and phosphorus to oiled shorelines in Prince William Sound, Alaska, accelerated biodegradation and did not increase toxicity or environmental effects of the oil.[50] The literature on oil-spill response procedures, cleanup methods, and restoration methods is extensive; the interested reader is encouraged to consult literature cited in references[47–50] and other sources for a more comprehensive treatment of this topic.

14.5 EFFECTS ON ORGANISMS

14.5.1 General

Petroleum can adversely affect organisms by physical action (smothering, reduced light), habitat modification (altered pH, decreased dissolved oxygen, decreased food availability), and toxic action. Large discharges of petroleum are most likely to produce notable effects from physical action and habitat modification. The aromatic fraction of petroleum is considered to be responsible for most of the toxic effects.

Polycyclic aromatic hydrocarbons affect organisms through toxic action. The mechanism of toxicity is reported to be interference with cellular membrane function and enzyme systems associated with the membrane.[7] Although unmetabolized PAHs can have toxic effects, a major concern in animals is the ability of the reactive metabolites, such as epoxides and dihydrodiols, of some PAHs to bind to cellular proteins and DNA. The resulting biochemical disruptions and cell damage lead to mutations, developmental malformations, tumors, and cancer.[2,6,51] Four-, five-, and six-ring PAHs have greater carcinogenic potential than the two-, three-, and seven-ring PAHs.[2,7] The addition of alkyl groups to the base PAH structure often produces carcinogenicity or enhances existing carcinogenic activity (e.g., 7,12-dimethylbenz[a]anthracene). Some halogenated PAHs are mutagenic without metabolic activation,[52] and the toxicity and possibly the carcinogenicity of PAHs can be increased by exposure to solar ultraviolet radiation.[53,54] Cancerous and precancerous neoplasms have been induced in aquatic organisms in laboratory studies, and cancerous and noncancerous neoplasms have been found in feral fish from heavily polluted sites.[2,7,55,56] However, sensitivity to PAH-induced carcinogenesis differs substantially among animals.[2,7]

In water the toxicity of individual PAHs to plants and animals increases as molecular weight (MW) increases up to MW 202 (fluoranthene, pyrene). Beyond MW 202 a rapid decline in solubility reduces PAH concentrations to less than lethal levels, regardless of their intrinsic toxicity. However, sublethal effects can result from exposure to these very low concentrations of high MW compounds.[7] Except for the vicinity of chemical or petroleum spills, environmental concentrations of PAHs in water are usually several orders of magnitude below levels that are acutely toxic to aquatic organisms. Sediment PAH concentrations can be much higher than water concentrations, but the limited bioavailability of these PAHs greatly reduces their toxic potential.[2] In general, caution should be employed when assessing the aquatic "toxicity" of biogenic or anthropogenic PAHs because bioavailability (solubility, sediment sequestration, mechanism of exposure) and chemical modification determine how much toxicity is realized.

14.5.2 Plants and Microbes

Reports of the effects of petroleum spills or discharges on plants and microbes contain accounts of injury or death to mangroves,[57,58] seagrasses,[59] and large intertidal algae;[60,61] severe and long-lasting (> 2 years) destruction of salt marsh vegetation[38,62–64] and freshwater wetland vegetation;[65,66] enhanced or reduced biomass and photosynthetic activity of phytoplankton communities;[67,68] genetic effects in mangroves and terrestrial plants;[69,70] and microbial community changes and increases in numbers of microbes (Table 14.1).[71–73] Differences in species sensitivity to petroleum are responsible for the wide variation in community response for phytoplankton and microbes.

Recovery from the effects of oil spills on most local populations of nonwoody plants can require from several weeks to 5 years, depending on the type of oil, circumstances of the spill, and species affected. Mechanical removal of petroleum in wetlands can markedly increase the recovery time.[74,75] Complete recovery by mangrove forests could require up to 20 years.[76] Phytoplankton and microbes in the water column of large bodies of water return to prespill conditions faster than phytoplankton and microbes in small bodies of water because of greater pollutant dilution and greater availability of colonizers in nearby waters.[77] Lethal and sublethal effects are caused by contact with oil or dissolved oil, systemic uptake of oil compounds, blockage of air exchange through surface pores, and possibly by chemical and physical alteration of soil and water, such as depletion of oxygen and nitrogen, pH change, and decreased light penetration.

The effects of petroleum on marine plants, such as mangroves, sea grasses, saltmarsh grasses, and micro- and macroalgae, and microbes have been studied with laboratory bioassays, experimental ecosystems, and field experiments and surveys (Table 14.1).[78–87] Petroleum caused death, reduced growth, and impaired reproduction in the large plants. Microalgae were either stimulated or inhibited, depending on the species and the type and amount of oil; response was expressed as changes in biomass, photosynthetic activity, and community structure. In response to petroleum exposure, community composition of indigenous bacteria was altered and total biomass increased.

The effects of petroleum on freshwater phytoplankton, periphyton, and microbes have been studied with laboratory bioassays and field experiments.[88–93] Petroleum induced effects similar to those described for marine microalgae and bacteria. Domestic and wild plants have been exposed to oiled soil in laboratory experiments[94–96] and tundra vegetation has been subjected to an experimental spill of crude oil.[97] Inhibition of seed germination, plant growth, and fungal colonization of roots was demonstrated in the laboratory bioassays. Death of herbaceous and woody plants and long-term community alteration were caused by the experimental tundra spill.

Individual PAHs, mostly two- and three-ring compounds, at low concentrations (5 to 100 ppb) can stimulate or inhibit growth and cell division in aquatic bacteria and algae. At high concentrations (0.2 to 10 ppm) the same PAHs interfere with cell division of bacteria and cell division and photosynthesis of algae and macrophytes; they can also cause death.[2,7]

Table 14.1

Death
Impaired re
Reduced gr
Altered rate
Altered DN
Malformatio
Tumors or l
Cancer
Impaired im
Altered end
Altered beh
Blood disor
Liver and ki
Hypotherm
Inflammatio
Altered resp
Impaired sa
Gill hyperpl
Fin erosion

Local popul
Altered com
Biomass ch

[a] Some effe
in laborato
[b] Includes a
[c] Population
in the pre
vertebrate

14.5.3 Inv

Reports
accounts of
for marine
coastal man
of petroleun
contact by a
oil or indivi
depletion ar
invertebrate

Recover
as a week fo
Uncertainty
mangrove b
of year, wea
from natura
hydrocarbo

Table 14.1 Effects of Petroleum or Individual PAHs on Organisms

	Type of Organism					
Effect[a]	Plant or Microbe	Invertebrate	Fish	Reptile or Amphibian	Bird	Mammal[b]
	Individual Organisms					
Death	X	X	X	X	X	X
Impaired reproduction	X	X	X	X	X	X
Reduced growth and development	X	X	X	X	X	
Altered rate of photosynthesis	X					
Altered DNA	X	X	X	X	X	X
Malformations			X		X	
Tumors or lesions		X	X	X		X
Cancer			X	X		X
Impaired immune system			X		X	X
Altered endocrine function			X		X	
Altered behavior		X	X	X	X	X
Blood disorders		X	X	X	X	X
Liver and kidney disorders			X		X	X
Hypothermia					X	X
Inflammation of epithelial tissue				X	X	X
Altered respiration or heart rate		X	X	X		
Impaired salt gland function				X	X	
Gill hyperplasia			X			
Fin erosion			X			
	Groups of Organisms[c]					
Local population change	X	X	X		X	X
Altered community structure	X	X	X		X	X
Biomass change	X	X	X			

[a] Some effects have been observed in the wild and in the laboratory, whereas others have only been induced in laboratory experiments or are population changes estimated from measures of reproduction and survival.

[b] Includes a sampling of literature involving laboratory and domestic animals.

[c] Populations of microalgae, microbes, soil invertebrates, and parasitic invertebrates can increase or decrease in the presence of petroleum, whereas populations of other plants and invertebrates and populations of vertebrates decrease.

14.5.3 Invertebrates

Reports of the effects of aquatic oil spills or discharges and oil-based drill cuttings often contain accounts of temporary debilitation, death, population change, or invertebrate community change for marine water column,[98,99] deepwater benthic,[100,101] nearshore subtidal,[102–105] intertidal,[64,106,107] coastal mangrove organisms,[102,108] and stream[109,110] and lake[111] organisms. For a review of the effects of petroleum on marine invertebrates, see Suchanek.[112] Observed effects are caused by smothering; contact by adults, juveniles, larvae, eggs, and sperm with whole or dissolved oil; ingestion of whole oil or individual compounds; and possibly by chemical changes in the water, including oxygen depletion and pH change. Accounts of the effects of petroleum spills or discharges on terrestrial invertebrates have not been published.

Recovery from the effects of oil spills on local populations of invertebrates can require as little as a week for pelagic zooplankton or as much as 10 years, or more, for nearshore subtidal meiofauna. Uncertainty of recovery time is particularly evident for intertidal, nearshore subtidal, and coastal mangrove biota; the prognosis for recovery is a function of the size of the spill, type of oil, season of year, weather, characteristics of affected habitat, and species affected. Chronic input of petroleum from natural seeps or anthropogenic sources produces communities of biota that are adapted to the hydrocarbon challenge; preexisting or continuing chronic input can complicate estimates of invertebrate recovery from ephemeral input. Aggressive cleaning of shorelines can retard recovery.[113] Zooplankton in large bodies of water return to prespill conditions faster than zooplankton in small bodies of water (isolated estuaries, lakes, stream headwaters) because of greater pollutant dilution and greater availability of colonizers in nearby waters.[77]

Much work has been done on the effects of petroleum on marine invertebrates with laboratory bioassays, mesocosms, enclosed ecosystems, and field experiments and surveys.[82,114–124] Less work has been conducted on freshwater invertebrates with laboratory bioassays and field experiments.[88,125–129] Among the effects reported are reduced survival, altered physiological function, soft-tissue abnormalities, inhibited reproduction, altered behavior, and changes in species populations and community composition.

Soil contaminated with petrochemicals has been associated with a reduced number of species and reduced species abundance of rodent parasites.[130] In contrast, petrochemical contamination of soil increased the abundance of isopods,[131] and chronic exposure to low concentrations of PAHs appeared to stimulate populations of several groups of soil invertebrates.[132] In soil remediation studies earthworms have been found to be a sensitive indicator of soil quality.[133]

In short-term exposure trials (24 to 96 h) on selected aquatic invertebrates, individual PAH compounds had LC_{50} values in water ranging from 0.1 to 5.6 ppm.[2,7] Eggs and larvae are more sensitive than juveniles or adults to dissolved PAHs. Larvae with a high tissue burden of PAHs from maternal transfer are likely to be at risk for increased toxicity from ultraviolet radiation.[134] Sublethal effects include reduced reproduction, inhibited development of embryos and larvae, delayed larval emergence, decreased respiration and heart rate, abnormal blood chemistry, and lesions.[2,7]

14.5.4 Fish

Adult and juvenile fish, larvae, and eggs are exposed to petroleum through contact with whole oil, dissolved hydrocarbons, particles of oil dispersed in the water column, or ingestion of contaminated food and water.[135,136]

Death of fish in natural habitat usually requires a heavy exposure to petroleum. Consequently, it is unlikely that large numbers of adult fish inhabiting large bodies of water would be killed by the toxic effects of petroleum. Fish kills usually are caused by large amounts of oil moving rapidly into shallow waters.[137,138] However, fresh and weathered crude oils and refined products vary considerably in their composition and toxicity, and the sensitivity of fish to petroleum differs among species. Petroleum concentrations (total petroleum hydrocarbons) in water of less than 0.5 ppm during long-term exposure[139] or higher concentrations (several to more than 100 ppm) in moderate- or short-term exposures can be lethal.[140–143] Sublethal effects begin at concentrations of less than 0.5 ppm and include changes in heart and respiratory rates, gill structural damage, enlarged liver, reduced growth, fin erosion, corticosteriod stress response, immunosuppression, impaired reproduction, increased external and decreased internal parasite burdens, behavioral responses, and a variety of biochemical, blood, and cellular changes.[136,142,144–154]

Eggs and larvae can suffer adverse effects, including death, when exposed to concentrations of petroleum in water ranging from less than 1 ppb (total PAHs) up to 500 ppb (total PAHs or total petroleum hydrocarbons).[155–158] Eggs, larvae, and early juveniles are generally more vulnerable than adults to oil spills and discharges because they have limited or no ability to avoid the oil, and they reside in locations that receive the most severe exposures (near the water surface or in shallow nearshore areas and streams). Effects of oil on eggs and larvae include death of embryos and larvae, abnormal development, reduced growth, premature and delayed hatching of eggs, DNA alterations, and other cellular abnormalities.[136,156–161] Evidence of continued adverse effects on the viability of pink salmon (*Oncorhynchus gorbuscha*) embryos for 4 years after the *Exxon Valdez* oil spill in Prince William Sound, Alaska, presented the possibility of gametic damage to exposed fish.[162]

Assessing the effect of petroleum spills and discharges on fish populations has been attempted with a variety of approaches, including taking fish samples from oiled and control areas after a

---

spill,[163] monit
after spill even
using a life-hi
munity compo
of deleterious
lation change.

Also, the
an extensive r
States.[167,168] U
as Atlantic c
(*Clupea hare*
direct field ob
year class fr
recruitment m

In genera
populations i
modeling ap
graphic scale

In short-t
had LC_{50} val
is the liver. S
previously de
but with grea

Induction
of lesions an
inated with P
of cancer in
Washington,
had cancero
external abn
and 50 to 10

There is
communities
biological m
an industrial
nonindustria
ture reveale
Rivers, Ohic

Metabol
is poorly st
effect and t
metabolism
efforts to ge

14.5.5 Re

The res
species of r
River (E.S.
report). Sea
could have

spill,[163] monitoring postspill fish harvest and recruitment,[138] aerial surveys of fish stocks before and after spill events,[164] translating abundance of benthic prey into estimates of demersal fish biomass,[104] using a life-history model to estimate effects on a species,[165] measuring differences in fish community composition above and below a discharge site on a stream,[149] and evaluating the potential of deleterious heritable mutations induced by a one-time spill event to produce measurable population change.[166] Several of these attempts revealed likely effects of petroleum on fish populations.

Also, the potential effects of oil spills on regional pelagic fish populations were evaluated with an extensive modeling effort of the Georges Bank fishery off the northeastern coast of the United States.[167,168] Unfortunately, normal variation in natural mortality of eggs and larvae for such species as Atlantic cod (*Gadus morhua*), haddock (*Melanogrammus aeglefinus*), and Atlantic herring (*Clupea harengus*) is often larger than mortality estimates for the largest of spills. Consequently, direct field observations would not be able to distinguish the effect of a major oil spill on a single year class from natural variation in recruitment. The authors concluded that a comprehensive recruitment model was needed to separate the effect of spilled oil from expected natural mortality.

In general, it is difficult to determine the effect of an ephemeral petroleum discharge on fish populations in large bodies of water. It is beneficial to have before-and-after data, and some modeling appears necessary to identify effects that are difficult to measure. Also, time and geographic scale are important considerations in any population assessment.

In short-term exposure trials (24 to 96 h) on selected species of fish, individual PAH compounds had LC_{50} values in water ranging from 1.3 to 3400 ppb.[2] The primary target organ for toxic action is the liver. Sublethal effects on eggs, larvae, juveniles, and adult fish[2] are generally similar to those previously described for exposure to fresh or weathered petroleum and separate aromatic fractions but with greater emphasis on neoplasm induction and DNA alteration.

Induction of precancerous cellular changes in laboratory studies with PAHs and high frequencies of lesions and cancerous and noncancerous neoplasms in bottom-dwelling fish from areas contaminated with PAHs provide support for a causal relation between PAHs in sediment and the presence of cancer in several species of bottom-dwelling marine fish.[7,33,55,56,169–171] Fish from Puget Sound, Washington,[172] tributaries of southern Lake Erie, Ohio,[55,173,174] and the Elizabeth River, Virginia,[175] had cancerous and noncancerous skin and liver neoplasms, fin erosion, and a variety of other external abnormalities. Concentrations of total PAHs in the sediment were sometimes > 100 ppm and 50 to 10,000 times greater than in reference areas.[2,55,173,175]

There is evidence that exposure to high concentrations of PAHs can affect fish populations and communities. Lesion frequency, overall health assessment, and population age structure were useful biological measures for differentiating a population of brown bullheads (*Ameiurus nebulosus*) in an industrialized urban river (Schuylkill River, Philadelphia, Pennsylvania) from a population in a nonindustrialized suburban pond (Haddonfield, New Jersey).[176] Analysis of fish community structure revealed lower species diversity for contaminated Lake Erie tributaries (Black and Cuyahoga Rivers, Ohio) than for a reference stream (Huron River, Ohio).[174]

Metabolism of PAHs by feral fish in areas chronically contaminated with multiple pollutants is poorly studied; studies such as van der Oost[177] are rare. Also lacking is information on dose-effect and temporal aspects of *in situ* exposure to carcinogens.[33,55] Species differences in PAH metabolism and incidence of neoplasms, even among closely related species,[33] further complicate efforts to generalize about findings from individual studies.

14.5.5 Reptiles and Amphibians

The response of reptiles and amphibians to petroleum exposure is not well characterized. Various species of reptiles and amphibians were killed by a spill of bunker C fuel oil in the St. Lawrence River (E.S. Smith, New York Department of Environmental Conservation, Albany, NY, unpublished report). Sea snakes were presumed to have been killed by crude oil in the Arabian Gulf.[178] Petroleum could have been a contributing factor in the deaths of sea turtles off the coast of Florida,[179] in the

Gulf of Mexico following the *Ixtoc I* oil spill,[180] and in the Arabian Gulf after the Gulf War oil spills,[181] but the cause of death was not determined. Ingestion of oil and plastic objects has been reported for green (*Chelonia mydas*), loggerhead (*Caretta caretta*), and Atlantic Ridley (*Lepidochelys kempi*) turtles.[179,180,182]

Experimental exposure of juvenile loggerhead turtles to crude oil slicks revealed effects on respiration, skin, blood characteristics and chemistry, and salt gland function.[183] Juvenile loggerhead turtles were exposed to artificially weathered crude oil for 4 days followed by an 18-day recovery period; blood abnormalities and severe skin and mucosal changes from exposure were reversed during the recovery period.[184] Atlantic Ridley and loggerhead embryos died or developed abnormally when the eggs were exposed to oiled sand; weathered oil was less harmful to the embryos than fresh oil (T.H. Fritts and M.A. McGehee, Fish and Wildlife Service, Denver Wildlife Research Center, Denver, CO, unpublished report).

Bullfrog tadpoles (*Rana catesbeiana*) were exposed to amounts of No. 6 fuel oil that could be expected in shallow waters following oil spills; death was most common in tadpoles that were in the late stages of development, and sublethal effects included grossly inflated lungs, fatty livers, and abnormal behavior.[185] Larvae of the wood frog (*Rana sylvatica*), the spotted salamander (*Ambystoma maculatum*), and two species of fish were exposed to several fuel oils and crude oils in static and flow-through tests; sensitivity of the amphibian larvae to oil was slightly less than that of the two species of fish.[140] Exposure of green treefrog (*Hyla cinerea*) embryos and larvae and larval mole salamanders (*Ambystoma opacum* and *A. tigrinum*) to used motor oil in natural ponds, field enclosures, or laboratory containers caused reduced growth or reduced food (algae) densities and prevented metamorphosis of green frogs at high exposure.[186,187]

The injection or implantation in amphibians of perylene or crystals of benzo(a)pyrene and 3-methylcholanthrene induced cancerous and noncancerous tissue changes.[2] It has been suggested that amphibians are more resistant to PAH carcinogenesis than mammals because of the demonstrated inability of the hepatic microsomes of the tiger salamander (*A. tigrinum*) to produce mutagenic metabolites.[188] The toxicity and genotoxicity of benzo(a)pyrene and refinery effluent to larval newts (*Pleurodeles waltlii*), in the presence and absence of ultraviolet radiation, was established in a series of experiments by Fernandez and l'Haridon.[189]

14.5.6 Birds

Birds can be affected by petroleum through external oiling, ingestion, egg oiling, and habitat changes. External oiling disrupts feather structure, causes matting of feathers, and produces eye and skin irritation. Death often results from hypothermia and drowning.[1,190–192] Bird losses in excess of 5000 individuals are common for moderate to large petroleum spills. Birds that spend much of their time in the water, such as alcids (*Alcidae*), waterfowl (*Anatidae*), and penguins (*Spheniscidae*), are the most vulnerable to surface oil.

Petroleum can be ingested through feather preening, consumption of contaminated food or water, and inhalation of fumes from evaporating oil. Ingestion of oil is seldom lethal, but it can cause many debilitating sublethal effects that promote death from other causes, including starvation, disease, and predation. Effects include gastrointestinal irritation, pneumonia, dehydration, red blood cell damage, impaired osmoregulation, immune system suppression, hormonal imbalance, inhibited reproduction, retarded growth, and abnormal parental behavior.[192–203]

Bird embryos are highly sensitive to petroleum. Contaminated nest material and oiled plumage are mechanisms for transferring oil to the shell surface. Small quantities (1 to 20 μL) of some types of oil (light fuel oils, certain crude oils) are sufficient to cause death, particularly during the early stages of incubation.[204,205] Eggshell applications of petroleum weathered for several weeks or longer are less toxic to bird embryos than fresh or slightly weathered petroleum.[206,207]

Petroleum spilled in avian habitats can have immediate and long-term effects on birds. Fumes from evaporating oil, a shortage of food, and cleanup activities can reduce use of an affected area.[208,209]

Long-term eff
are likely to h
The direct
level. Death f
availability, a
catastrophic e
(*Haliaeetus l*
March 1989 f
years seabird
to losses from
Effects of
measures of s
to local or reg
the loss of a
described for
Sound and ex
assessments
reported after
oil spill.[222] M
detectable by
and Symens
The cons
been estimate
in survival of
decrease in r
the most diffi
population of
small, but su
ductive poten
Much of
effects of pet
oils is respon
Further, the t
of the PAHs.
the chicken (
chos), and c
species and
most toxic of
of embryos,
receptor.[231] N
eider eggs ar
greatest con
fractionated
eggs and fou
of five- and
increased in
Studies w
Male mallar
of 10 alkane
higher testes
gulls (*Larus*

fter the Gulf War oil
stic objects has been
ntic Ridley (*Lepido-*

revealed effects on
Juvenile loggerhead
an 18-day recovery
osure were reversed
or developed abnor-
nful to the embryos
er Wildlife Research

uel oil that could be
dpoles that were in
l lungs, fatty livers,
spotted salamander
oils and crude oils
ightly less than that
yos and larvae and
il in natural ponds,
od (algae) densities

zo(a)pyrene and 3-
as been suggested
use of the demon-
rinum) to produce
refinery effluent to
diation, was estab-

oiling, and habitat
and produces eye
rd losses in excess
at spend much of
ns (*Spheniscidae*),

aminated food or
lethal, but it can
luding starvation,
dration, red blood
balance, inhibited

nd oiled plumage
L) of some types
during the early
l weeks or longer

on birds. Fumes
ffected area.[208,209]

Long-term effects are more difficult to document, but severely oiled wetlands and tidal mud flats are likely to have altered plant and animal communities for many years after a major spill.[62,82]

The direct and indirect effects of oil spills are difficult to quantify at the regional or species level. Death from natural causes and activities of humans (e.g., commercial fishing), weather, food availability, and movement of birds within the region can obscure the effects of a single or periodic catastrophic event. Regional population assessments in the northern Gulf of Alaska for bald eagles (*Haliaeetus leucocephalus*) and common murres (*Uria aalge*) after the *Exxon Valdez* oil spill in March 1989 failed to identify population changes attributable to the spill.[210–212] During the last 50 years seabird populations of western Europe have increased or decreased without apparent relation to losses from oil spills.[213,214]

Effects of oil spills are most likely to be detected at the level of local populations. Also, measures of survival, reproduction, and habitat use of numerous individual birds can be extrapolated to local or regional populations. The *Exxon Valdez* oil spill in Prince William Sound, Alaska caused the loss of an estimated 250,000 to 375,000 birds.[215,216] Adverse effects of this spill have been described for individuals of several species of seabirds or wintering waterfowl in Prince William Sound and extrapolated to populations.[217–220] Wiens et al.[218] also combined the species effects into assessments of avian community composition. Marine bird population declines have also been reported after the 1996 *Sea Empress* spill off the coast of England[221] and the 1991 Arabian Gulf oil spill.[222] Most of the changes in performance measures or local population size were no longer detectable by 2 years after the spill; exceptions were described by Wiens et al.,[218] Esler et al.,[221] and Symens and Suhaibani.[222]

The consequences of direct and indirect effects of oil spills on seabird populations have also been estimated with simulation models.[223–226] Models have shown that (1) an occasional decrease in survival of breeding adults will have a greater effect on seabird populations than an occasional decrease in reproductive success, (2) long-lived seabirds with low reproductive potential will have the most difficulty recovering from a catastrophic oil spill, and (3) recovery of a seriously depleted population of long-lived seabirds will be greatly hindered if adult survival and reproduction show small, but sustained, decreases. The overall recovery potential for a species depends on the reproductive potential of the survivors and the immigration potential from surrounding areas.[227,228]

Much of the available information on effects of PAHs on birds was produced by studies of the effects of petroleum on eggs. Experiments have shown that the PAH fraction of crude and refined oils is responsible for the lethal and sublethal effects on bird embryos caused by eggshell oiling.[205] Further, the toxicity of PAHs to bird embryos is a function of the quantity and molecular structure of the PAHs.[229] Brunstrom et al.[230] injected a mixture of 18 PAHs (2.0 mg/kg of egg) into eggs of the chicken (*Gallus domesticus*), turkey (*Meleagris gallopavo*), domestic mallard (*Anas platyrhynchos*), and common eider (*Somateria mollissima*) and found the mallard to be the most sensitive species and benzo[k]fluoranthene (four rings) and indeno[1,2,3-cd]pyrene (five rings) to be the most toxic of the PAHs tested. The most toxic PAHs were found to have additive effects on death of embryos, and the cause of toxicity was proposed to be a mechanism controlled by the Ah receptor.[231] Naf et al.[232] injected a mixture of 16 PAHs (0.2 mg/kg of egg) into chicken and common eider eggs and reported that > 90% was metabolized by day 18 of incubation (chicken), with the greatest concentration of PAHs in the gall bladder of both species. Mayura et al.[233] injected fractionated PAH mixtures from coal tar (0.0625 to 2.0 mg/kg of egg) into the yolk of chicken eggs and found that death, liver lesions and discoloration, and edema increased as the proportion of five- and > five-ring aromatics, compared to the proportion of two- to four-ring aromatics, increased in the mixture.

Studies with adults and nestlings revealed a variety of sublethal toxic effects induced by PAHs. Male mallard ducks fed 6000 ppm of a mixture of 10 PAHs combined with 4000 ppm of a mixture of 10 alkanes for 7 months in a chronic ingestion study had greater hepatic stress responses and higher testes weights than male mallards fed 10,000 ppm of the alkane mixture.[234] Nestling herring gulls (*Larus argentatus*) were administered single doses (0.2 or 1.0 mL) of crude oils, their aromatic or aliphatic fractions, or a mixture of crude oil and dispersant.[235,236] Retardation of nestling weight gain and increased adrenal and nasal gland weights was attributed to the PAHs with four or more rings. Immune function and mixed-function oxidase activity of adult European starlings (*Sturnus vulgaris*) were altered by subcutaneous injections (25 mg/kg body weight) of 7,12-dimethylbenz[a]anthracene, a four-ring PAH, every other day for 10 days.[237] Oral doses in adult birds (25 mg/kg body weight) and nestlings (20 mg/kg body weight) also altered immune function. The coefficient of variation of nuclear DNA volume of red blood cells from wild lesser scaup (*Athya affinis*) was positively correlated with the concentration of total PAHs in scaup carcasses.[238]

14.5.7 Mammals

Marine mammals that rely primarily on fur for insulation, such as the sea otter (*Enhydra lutris*), polar bear (*Ursus maritimus*), Alaska fur seal (*Callorhinus ursinus*), and newborn hair seal pups (*Phocidae*), are the most likely to die after contact with spilled oil.[22,239,240] Oiled fur becomes matted and loses its ability to trap air or water, resulting in hypothermia. Adult hair seals, sea lions (*Eumetopias jubatus, Zalophus californianus*), and cetaceans (whales, porpoises, dolphins) depend primarily on layers of fat for insulation; thus, oiling causes much less heat loss. However, skin and eye irritation and interference with normal swimming can occur. Skin absorption of oil has been reported for seals and polar bears.

Oil ingested in large quantities can have acute effects on marine mammals. However, marine mammals, such as seals and cetaceans, are capable of rapid hydrocarbon metabolism and renal clearance.[239] Ingested oil can cause gastrointestinal tract hemorrhaging in the European otter (*Lutra lutra*);[241] renal failure, anemia, and dehydration in the polar bear;[242] pulmonary emphysema, centrilobular hepatic necrosis, hepatic and renal lipidosis, increased nuclear DNA mass, altered blood chemistry, and possible gastric erosion and hemorrhage in sea otters,[243–245] and altered blood chemistry and reduced body weight in river otters (*Lontra canadensis*).[246] Inhalation of evaporating oil is a potential respiratory problem for mammals near or in contact with large quantities of unweathered oil.[239,247] Some of the previously described disorders are thought to be caused by hypothermia, shock, and stress rather than direct toxic action; distinguishing between the two types of causes can be difficult.[248]

Effects of the *Exxon Valdez* oil spill on populations of sea otter, harbor seals (*Phoca vitulina*), Stellar sea lions, killer whales (*Orcinus orca*), and humpback whales (*Megaptera novaeangliae*) in Prince William Sound, Alaska, are summarized in the overview provided by Loughlin et al.[245] Sea otters and harbor seals were the most affected, with loss estimates in the thousands for otters and in the hundreds for seals. Age distributions of dead sea otters systematically collected from oiled areas of western Prince William Sound between 1976 and 1998 revealed a reduction in the survival rate of otters during the 9 years after the spill (1990–1998).[249] Aerial counts of harbor seals at seven sites affected by the oil spill and 18 unaffected sites in central and eastern Prince William Sound revealed a 28% population reduction during the period 1990–1997; however, harbor seal populations on the survey route were declining prior to the 1989 spill.[250] Effects of the Gulf War oil spills on cetaceans in the Arabian Gulf were thought to be minimal.[251] Overall, the consequences of local effects (acute, chronic, and indirect) of catastrophic oil spills on regional populations of marine mammals have proven difficult to determine because of a lack of prespill population information, movement of animals within the region, and natural fluctuations in survival and reproduction.

Documentation of the effects of oil spills on wild nonmarine mammals is less than for marine mammals. Large numbers of muskrats (*Ondatra zibethica*) were killed by a spill of bunker C fuel oil in the St. Lawrence River (E.S. Smith, New York Department of Environmental Conservation, Albany, NY, unpublished report). Giant kangaroo rats (*Dopodomys ingens*) in California were found dead after being oiled,[252] beaver (*Castor canadensis*) and muskrats were killed by an aviation kerosine spill in a Virginia river,[253] and rice rats (*Oryzomys palustris*) in a laboratory experiment

---

died after sw
affected bloo
tion age and s
characterized
assemblages
literature on e
recent examp
(systemic eff
dosed cattle),
The effect
altered diet, a
be most serio
The metal
and domestic
selected PAH
PAH toxic act
lymphoid, an
hence, accum
fully and part
greatest poter
tissues of lab
appears to be
exposure con
accurately ass
Although
carcinogenic
PAHs that are
investigations
and participa
described PAI
of potential m

14.5.8 The

The 1989
oil spills (GW
for different r
pollution liter
The EVOS
latitude, relati
discharges, th
massive oil-re
3/24/89 to 10
cost of $45 m
In the Uni
Compensation
tribal) of affec
natural resour
injury and sub
cooperation a
years.[268] Two l

died after swimming through oil-covered water.[254] Oil field pollution in wooded areas in Russia affected blood characteristics, organ indices, species composition, relative abundance, and population age and sex structure of small mammals.[255] Cotton rats occupying old petrochemical sites were characterized by increased cell apoptosis in the ovary and thymus,[256] and rodent populations and assemblages on the same types of sites were altered compared to nearby reference sites.[257] The literature on effects of crude or refined petroleum on laboratory and domestic animals is substantial; recent examples are Lee and Talaska (DNA adduct formation in mouse skin),[258] Feuston et al. (systemic effects of dermal application in rats),[259] Khan et al. (clinical and metabolic effects in dosed cattle),[260] and Mattie et al. (pathology and biochemistry of dosed rats).[261]

The effects of major oil spills on the environment of mammals could include food reduction, an altered diet, and changed use of habitat.[262,263] These effects could be short- or long-term and would be most serious during the breeding season, when movement of females and young is restricted.

The metabolism and effects of some PAHs have been well documented in laboratory rodents and domestic mammals but poorly documented in wild mammals. Acute oral LD_{50} values for selected PAHs in laboratory rodents range from 50 to 2000 mg/kg body weight.[2] Target organs for PAH toxic action are skin, small intestine, kidney, and mammary gland; tissues of the hematopoietic, lymphoid, and immune systems; and gametic tissue. Nonalkylated PAHs are rapidly metabolized; hence, accumulation is less likely than for alkylated PAHs.[2] Partially aromatic PAHs, alkylated fully and partially aromatic PAHs, and metabolites of nonalkylated, fully aromatic PAHs have the greatest potential to alter DNA and induce cancerous and noncancerous neoplasms in epithelial tissues of laboratory and domestic mammals.[2] Species differences in sensitivity to carcinogenesis appears to be a function of differences in levels of mixed-function oxidase activities. Background exposure concentrations of PAHs and studies on mixtures of PAHs in mammals are needed to accurately assess the potential hazard of exposure to multiple PAHs in polluted environments.[2]

Although PAHs can produce systemic effects, DNA alterations, and cancer, concerns about the carcinogenic and mutagenic potential predominate, especially for human health. Consequently, PAHs that are carcinogenic or mutagenic are most studied.[2] With regard to wild mammals, a few investigations have documented the presence of PAH adducts on DNA of marine mammals,[264–266] and participants of a recent workshop on marine mammals and persistent ocean contaminants[267] described PAHs as a "less widely recognized" contaminant that should be further studied because of potential mutagenic and genotoxic effects.

14.5.8 The *Exxon Valdez* and Arabian Gulf Oil Spills

The 1989 *Exxon Valdez* oil spill (EVOS) in Prince William Sound, Alaska and the Gulf War oil spills (GWOS) in the Arabian Gulf in 1991 are worthy of special comment. Each was significant for different reasons, and reports of the fate and effects of these spills dominated the petroleum pollution literature during the 1990s.

The EVOS consisted of 36,000 metric tons of crude oil released in just a few days into a high-latitude, relatively pristine marine ecosystem. Although many times smaller than the Gulf War oil discharges, the scientific and societal response was immediate and intense. The spill resulted in a massive oil-removal effort,[268] a thorough crude oil mass balance determination spanning the period 3/24/89 to 10/1/92[269] and the largest wildlife rescue and rehabilitation effort ever attempted (at a cost of $45 million).[270]

In the United States, the Clean Water Act and the Comprehensive Environmental Response, Compensation, and Liability Act determined the response requirements for trustees (federal, state, tribal) of affected natural resources. Trustees initiated research that sought evidence of "injury" to natural resources, and Exxon Corporation initiated research that sought to demonstrate minimal injury and subsequent restoration of affected resources. This adversarial legal process discouraged cooperation among scientists and delayed public access to the results of the studies for several years.[268] Two books containing studies sponsored mostly by Exxon[271] and Trustees[272] provide many examples of conflicting interpretations of the effects of the spill; Wiens[273] provides a discussion of the effects of advocacy on investigations of the effects of the spill on birds. The EVOS is rapidly becoming the most studied oil spill in history; a stream of scientific reports continues to be generated by scientists assessing the biological consequences.

The GWOS consisted of 240 million metric tons of crude oil released into the northern Arabian Gulf over a 6-month period; the Gulf had a previous history of petroleum pollution from oil production activities and warfare. The spillage was caused by acts of war, and scientific coverage of the progression of the spill and its biological effects was delayed until hostilities ended and munitions were cleared from areas affected by discharged oil. Investigators from a number of countries performed assessments of the effects of the oil, which appear to have been less severe on plants and animals of aquatic and coastal environments than the effects reported for the EVOS. In contrast, the contamination of terrestrial environments caused by destruction of oil wells and pipelines was severe and is likely to affect terrestrial plants and animals for many years to come.[274,275]

14.5.9 Conclusions

The effects of petroleum on organisms are as varied as the composition of petroleum and the environmental conditions accompanying its appearance. Petroleum can cause environmental harm by toxic action, physical contact, chemical and physical changes within the soil or water medium, and habitat alteration. Oil spills have caused major changes in local plant and invertebrate populations lasting from several weeks to many years. Effects of oil spills on populations of mobile vertebrate species, such as fish, birds, and mammals, have been difficult to determine beyond an accounting of immediate losses and short-term changes in local populations. Reptiles and amphibians need further study. Knowledge of the biological effects of petroleum in freshwater environments continues to increase but still lags behind comparable information for saltwater environments.

Concentrations of individual PAHs in air, soil, and water are usually insufficient to be acutely toxic, but numerous sublethal effects can be produced. The induction of lesions and neoplasms in laboratory animals by metabolites of PAHs and observations of lesions and neoplasms in fish from PAH-contaminated sites indicate potential health problems for animals with a strong MFO system capable of metabolizing PAHs. Although evidence linking environmental PAHs to the incidence of cancerous neoplasms in wild vertebrates is primarily limited to fish, the growing quantities of PAHs entering our environment are a cause for concern.

14.6 SUMMARY

Petroleum and individual PAHs from anthropogenic sources are found throughout the world in all components of ecosystems. Crude and refined oils are highly variable in composition and physical characteristics and consist of thousands of hydrocarbon and nonhydrocarbon compounds. Polycyclic aromatic hydrocarbons are aromatic hydrocarbons with two to seven fused benzene rings that can have alkyl groups attached to the rings. Less than half of the petroleum in the environment comes from spills and discharges associated with petroleum transportation. Most of the petroleum comes from industrial, municipal, and household discharges; motorized vehicles; natural oil seeps; and acts of war, terrorism, vandalism, and theft. Most PAHs are formed by a process of thermal decomposition and subsequent recombination of organic molecules (pyrolysis and pyrosynthesis). Low-temperature processes produce the PAHs in coal and oil. High-temperature processes can occur naturally (forest and grass fires, volcanoes) or can be caused by anthropogenic activities (oil, coal, and wood combustion; refuse incineration; industrial activity). Most high-temperature PAHs enter the environment as combustion emissions or components of liquid waste effluents from industrial sites and municipal sewage plants.

Crude and ref
exposure to air,
currents and mo
degraded by mic
metabolize inges
and chemical oxi
Organisms w
location of the h
to accumulate h
Residence time f
ments can retain
type of substrate
persist for long
Petroleum ca
of oil spills requ
wetland plants;
can induce positi
can lead to deat
Oil spills oft
require a week
diverse amount
invertebrates, ha
munity effects. I
low concentratio
Eggs, larvae
limited or no ab
a large quantity
been documente
determine becau
metabolism and
imply that PAH
Uncertainties ab
inadequate know
ogens complicat
Adult reptil
sublethally affe
sensitivity to pe
Birds are oft
that spend muc
oil can cause m
of local popula
investigators of
Population mod
have the most
function of the
site. Experimen
revealed a vari
effects attribute
Mammals t
likely to die fr
infrequently kil

Crude and refined petroleum spreads rapidly on water and begins to change composition upon exposure to air, water, or sunlight. Anthropogenic aromatic hydrocarbons are dispersed by air currents and movement of waters receiving wastewater effluents. Hydrocarbons are primarily degraded by microbial metabolism; mammals, birds, fish, and many macroinvertebrates can also metabolize ingested hydrocarbons. Hydrocarbons are also degraded by photooxidation, photolysis, and chemical oxidation.

Organisms with high lipid content, activity patterns or distributions that coincide with the location of the hydrocarbon source, and a poor mixed-function oxygenase system are most likely to accumulate hydrocarbons. Trophic-level increases in accumulation have not been observed. Residence time for petroleum in the water column is usually less than 6 months. Coastal environments can retain oil from several days to 20 years, depending on the configuration of the shoreline, type of substrate, and climate. High-molecular-weight hydrocarbons, particularly aromatics, can persist for long periods of time (> 20 years) in sediments and terrestrial soils.

Petroleum can have lethal or sublethal effects in plants and microbes. Recovery from the effects of oil spills requires as little as a few weeks for water column microalgae up to 5 years for most wetland plants; mangroves could require up to 20 years. Individual PAHs at low concentrations can induce positive or negative sublethal effects in aquatic bacteria and algae; high concentrations can lead to death.

Oil spills often have pronounced effects on local populations of invertebrates; recovery could require a week for zooplankton or 10 years for intertidal populations of mollusks. A large and diverse amount of experimental and survey research, performed with saltwater and freshwater invertebrates, has demonstrated lethal and many sublethal effects as well as population and community effects. Individual PAHs can be lethal at high concentrations and cause sublethal effects at low concentrations.

Eggs, larvae, and early juvenile stages of fish are more vulnerable to oil because they have limited or no ability to avoid it. Large losses of adult fish are usually limited to situations where a large quantity of oil rapidly moves into shallow water. Many sublethal effects of oil on fish have been documented. Effects of oil spills on fish populations in large bodies of water are difficult to determine because of large natural variation in annual recruitment. Laboratory studies of PAH metabolism and lesions and tumors in fish collected from areas heavily contaminated with PAHs imply that PAH exposure can cause cancerous and noncancerous tissue changes in feral fish. Uncertainties about the interactive effects of multiple pollutants at heavily contaminated sites and inadequate knowledge of dose-effect responses and temporal aspects of *in situ* exposure to carcinogens complicate efforts to link environmental PAHs to neoplasms or local population changes.

Adult reptiles and amphibians can be killed and their eggs and amphibian larvae killed or sublethally affected by petroleum. However, available information is inadequate to compare their sensitivity to petroleum or individual PAHs to that of other vertebrates.

Birds are often killed by oil spills, primarily because of plumage oiling and oil ingestion. Birds that spend much of their time on the water surface are the most vulnerable to spilled oil. Ingested oil can cause many sublethal effects. Effects of oil spills are more likely to be detected at the level of local populations than at the regional or species level. When the quantity of data is large, investigators often extrapolate responses of individual birds to local and regional populations. Population modeling studies have shown that long-lived birds with low reproductive success will have the most difficulty recovering from a major oil spill. Recovery potential for a species is a function of the reproductive potential of the survivors and the immigration potential at the spill site. Experiments with individual or groups of PAHs and bird eggs, nestlings, and adults have revealed a variety of toxic responses and shown that PAHs are responsible for most of the toxic effects attributed to petroleum exposure.

Mammals that rely on fur for insulation (polar bear, otters, fur seals, muskrat) are the most likely to die from oiling. Mammals that rely on layers of fat for insulation (seals, cetaceans) are infrequently killed by oil. Ingested oil is rapidly metabolized and cleared, but it can cause many sublethal effects. Effects of spilled oil on local and regional populations of marine mammals have proven difficult to determine because of a lack of prespill population information, movement of animals within the region, and natural fluctuations in survival and reproduction. Laboratory mammals, but not wild mammals, have been extensively used to study the toxic and carcinogenic potential of individual PAHs. Partially aromatic PAHs, alkylated, fully and partially aromatic PAHs, and metabolites of nonalkylated, fully aromatic PAHs have the greatest potential to cause neoplasms in tissues of laboratory and domestic mammals. Investigations involving mixtures of PAHs are especially needed.

In general, petroleum negatively affects living organisms through physical contact, toxic action, and habitat modification, whereas individual PAHs have toxic effects. Partially metabolized and alkylated PAHs can induce genetic damage, developmental abnormalities, and cancerous and noncancerous tissue changes. Evidence linking environmental concentrations of PAHs to induction of cancer in wild animals is strongest for fish. Although concentrations of individual PAHs in aquatic environments are usually much lower than concentrations that are acutely toxic to aquatic organisms, sublethal effects can be produced. Effects of spills on populations of mobile species have been difficult to determine beyond an accounting of immediate losses and, sometimes, short-term changes in local populations.

REFERENCES

1. Bourne, W. R. P., Oil pollution and bird populations, in *The Biological Effects of Oil Pollution on Littoral Communities*, McCarthy, J. D. and Arthur, D. R., Eds., Field Studies 2 (Suppl.), 1968, 99.
2. Eisler, R., Polycyclic aromatic hydrocarbons, in *Handbook of Chemical Risk Assessment*, Vol. 2, Lewis Publishers, Boca Raton, FL, 2000, Chap. 25.
3. Atlas, R. M. and Bartha R., Fate and effects of polluting petroleum in the marine environment, *Residue Rev.*, 49, 49, 1973.
4. Posthuma, J., The composition of petroleum, Rapp. P.-V., *Reun. Cons. Int. Explor. Mer.*, 171, 7, 1977.
5. Farrington, J. W., Analytical techniques for the determination of petroleum contamination in marine organisms, in *Background Information: Workshop on Inputs, Fates, and Effects of Petroleum in the Marine Environment*, Ocean Affairs Board, National Academy of Sciences, Washington, D.C., 1973, 157.
6. Santodonato, S., Howard, P., and Basu, D., Health and ecological assessment of polynuclear aromatic hydrocarbons, *J. Environ. Pathol. Toxicol.*, Special Issue, 5, 1981.
7. Neff, J. M., Polycyclic aromatic hydrocarbons, in *Fundamentals of Aquatic Toxicology, Rand*, G. M. and Petrocilli, S. R., Eds., Hemisphere, New York, 1985, Chap. 14.
8. McElroy, A. E., Farrington, J. W., and Teal, J. M., Bioavailability of polycyclic aromatic hydrocarbons in the aquatic environment, in *Metabolism of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment*, Varanasi, U., Ed., CRC Press, Boca Raton, FL, 1989, Chap. 1.
9. National Academy of Sciences, Petroleum in the Marine Environment. Workshop on Inputs, Fates and the Effects of Petroleum in the Marine Environment, Ocean Affairs Board, Washington, D.C., 1975, Chap. 1.
10. Grossling, B. F., An estimate of the amounts of oil entering the oceans, in *Sources, Effects and Sinks of Hydrocarbons in the Aquatic Environment*, American Institute of Biological Sciences, Arlington, VA, 1976, 6.
11. National Research Council, *Oil in the Sea. Inputs, Fates, and Effects*, Board on Ocean Science and Policy, Washington, D.C., 1985, Chap. 1.
12. Whipple, W., Jr. and Hunter, J. V., Petroleum hydrocarbons in urban runoff, *Water Resour. Bull.*, 15, 1096, 1979.
13. Connell, D. W., An approximate petroleum hydrocarbon budget for the Hudson Raritan estuary — New York, *Mar. Pollut. Bull.*, 13, 89, 1982.
14. Readman, J. W. et al., Oil and combustion-product contamination of the Gulf marine environment following the war, *Nature*, 358, 662, 1992.

---

15. Hosmer, A
caused by
Petroleum
16. Mastral, A
from ener
17. Grimmer,
CRC Pres
18. Baek, S.
behavior,
19. Colwell,
marine en
20. Lee, R. F.
Fish and
21. McEwan,
glaucous-
58, 723, 1
22. Engelhard
23. James, M.
Aromatic
1989, Ch
24. Jenssen,
on the mi
25. Lytle, J. S
Proc. 198
26. Simonich
carbons f
27. Clement,
intertidal
28. Soler, M.,
in mussel
18, 1809,
29. Boehm,
shell clan
30. Broman,
mation a
Mytilus
Environ.
31. Rattner,
contamin
32. Wild, S.
amended
33. Varanasi,
Metaboli
Press, B
34. Gundlach
vulnerab
35. Page, D.
1989 Oil
36. Corredor
intertidal
37. Vanderm
bunker C
38. Baker, J.
1993 Oil
39. Gundlach

nals have
ement of
ory mam-
cinogenic
tic PAHs,
eoplasms
PAHs are

xic action,
lized and
erous and
induction
PAHs in
to aquatic
le species
nes, short-

ollution on
1968, 99.
ol. 2, Lewis

ent, *Residue*

71, 7, 1977.
n in marine
leum in the
D.C., 1973,

ear aromatic

Rand, G. M.

ydrocarbons
the Aquatic

nputs, Fates
ngton, D.C.,

ts and Sinks
s, Arlington,

Science and

ur. Bull., 15,

n estuary —

environment

15. Hosmer, A. W., Stanton, C. E., and Beane, J. L., Intent to spill: Environmental effects of oil spills caused by war, terrorism, vandalism, and theft, in *Proc. 1997 Int. Oil Spill Conf.*, Publ. 4651, American Petroleum Institute, Washington, D.C., 1997, 157.
16. Mastral, A. M. and Callen, M. S., A review on polycyclic aromatic hydrocarbon (PAH) emissions from energy generation, *Environ. Sci. Technol.*, 34, 3051, 2000.
17. Grimmer, G., in *Environmental Carcinogens: Polycyclic Aromatic Hydrocarbons*, Grimmer, G., Ed., CRC Press, Boca Raton, FL, 1983, Chap. 2.
18. Baek, S. O. et al., A review of atmospheric polycyclic aromatic hydrocarbons, sources, fate and behavior, *Water Air Soil Pollut.*, 60, 279, 1991.
19. Colwell, R. R. and Walker, J. D., Ecological aspects of microbial degradation of petroleum in the marine environment, *Crit. Rev. Microbiol.*, 5, 423, 1977.
20. Lee, R. F., Fate of oil in the sea, in *Proc. 1977 Oil Spill Response Workshop*, Four, P. L., Ed., U.S. Fish and Wildlife Service FWS/OBS/77-24, 1977, 43.
21. McEwan, E. H. and Whitehead, P. M., Uptake and clearance of petroleum hydrocarbons by the glaucous-winged gull (*Larus glaucescens*) and the mallard duck (*Anas platyrhynchos*), *Can. J. Zool.*, 58, 723, 1980.
22. Engelhardt, F. R., Petroleum effects on marine mammals, *Aquat. Toxicol.*, 4, 199, 1983.
23. James, M. O., Microbial degradation of PAH in the aquatic environment, in *Metabolism of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment*, Varanasi, U., Ed., CRC Press, Boca Raton, FL, 1989, Chap. 3.
24. Jenssen, B. M., Ekker, M., and Zahlsen, K., Effects of ingested crude oil on thyroid hormones and on the mixed function oxidase system in ducks, *Comp. Biochem. Physiol.*, 95C, 213, 1990.
25. Lytle, J. S. and Lytle, T. F., The role of *Juncus roemerianus* in cleanup of oil-polluted sediments, in *Proc. 1987 Oil Spill Conf.,* Publ. 4452, American Petroleum Institute, Washington, D.C., 1987, 495.
26. Simonich, S. L. and Hites, R. A., Importance of vegetation in removing polycyclic aromatic hydrocarbons from the atmosphere, *Nature*, 370, 49, 1994.
27. Clement, L. E., Stekoll, M. S., and Shaw, D. G. Accumulation, fractionation and release of oil by the intertidal clam *Macoma balthica*, *Mar. Biol.*, 57, 41, 1980.
28. Soler, M., Grimalt, J. O., and Albaiges, J., Vertical distribution of aliphatic and aromatic hydrocarbons in mussels from the Amposta offshore oil production platform (western Mediterrean), *Chemosphere*, 18, 1809, 1989.
29. Boehm, P. D. and Quinn, J. G., The persistence of chronically accumulated hydrocarbons in the hard shell clam *Mercenaria mercenaria*, *Mar. Biol.*, 44, 227, 1977.
30. Broman, D., Naf, C., Lundbergh, I., and Zebuhr, Y., An *in situ* study on the distribution, biotransformation and flux of polycyclic aromatic hydrocarbons (PAHs) in an aquatic food chain (seston — *Mytilus edulis L. — Somateria mollissima L.*) from the Baltic: An ecotoxicological perspective, *Environ. Toxicol. Chem.*, 9, 429, 1990.
31. Rattner, B. A., Hoffman, D. J., and Marn, C. N., Use of mixed-function oxygenases to monitor contaminant exposure, *Environ. Toxicol. Chem.*, 8, 1093, 1989.
32. Wild, S. R. et al., Polynuclear aromatic hydrocarbons in crops from long-term field experiments amended with sewage sludge, *Environ. Pollut.*, 76, 25, 1992.
33. Varanasi, U., Stein, J. E., and Nishimoto, M., Biotransformation and disposition of PAH in fish, in *Metabolism of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment*, Varanasi, U., Ed., CRC Press, Boca Raton, FL, 1989, Chap. 4.
34. Gundlach, E. R. and Hayes, M. O., Classification of coastal environments in terms of potential vulnerability to oil spill damage, *Mar. Technol. Soc. J.*, 12, 18, 1978.
35. Page, D. S. et al., Long-term weathering of *Amoco Cadiz* oil in soft intertidal sediments, in *Proc. 1989 Oil Spill Conf.,* American Petroleum Institute Publ. 4479, Washington, D.C., 1989, 401.
36. Corredor, J. E., Morell, J. M., and Del Castillo, C. E., Persistence of spilled crude oil in a tropical intertidal environment, *Mar. Pollut. Bull.*, 21, 385, 1990.
37. Vandermeulen, J. H. and Singh, J. G., ARROW oil spill, 1970–90; persistence of 20-yr weathered bunker C fuel oil, *Can. J. Fish. Aquat. Sci.*, 51, 845, 1994.
38. Baker, J. M. et al., Long-term fate and effects of untreated thick oil deposits on salt marshes, in *Proc. 1993 Oil Spill Conf.,* Publ. 4580, American Petroleum Institute, 1993, 395.
39. Gundlach, E. R. et al., The fate of *Amoco Cadiz* oil, *Science*, 221, 122, 1983.

40. DeLaune, R. D. et al., Degradation of petroleum hydrocarbons in sediment receiving produced water discharge, *J. Environ. Sci. Health*, A35, 1, 2000.
41. Gundlach, E. R., Domeracki, D. D., and Thebeau, L. C., Persistence of Metula oil in the Strait of Magellan six and one-half years after the incident, *Oil Petrochem. Pollut.*, 1, 37, 1982.
42. Haines, J. R. and Atlas, R. M., *In situ* microbial degradation of Prudhoe Bay crude oil in Beaufort Sea sediments, *Mar. Environ. Res.*, 7, 91, 1982.
43. Huntley, S. L., Bonnevie, N. L., and Wenning, R. J., Polycyclic aromatic hydrocarbons and petroleum hydrocarbon contamination in sediment from the Newark Bay Estuary, New Jersey, *Arch. Environ. Contam. Toxicol.*, 28, 93, 1995.
44. Olivera, F. L. et al., Prepared bed land treatment of soils containing diesel and crude oil hydrocarbons, *J. Soil Contam.*, 7, 657, 1998.
45. Wang, Z. et al., Study of the 25-year-old Nipisi oil spill: Persistence of oil residues and comparisons between surface and subsurface sediments, *Environ. Sci. Technol.*, 32, 2222, 1998.
46. Aislabie, J. et al., Polycyclic aromatic hydrocarbons in fuel-oil contaminated soils, Antarctica, *Chemosphere*, 39, 2201, 1999.
47. Jahn, A. E. and Robilliard, G. A., Natural recovery: A practical natural resource restoration option following oil spills, in *Proc. 1997 Oil Spill Conf.*, Publ. 4651, American Petroleum Institute, Washington, D.C., 1997, 665.
48. American Petroleum Institute, Oil Spill Cleanup: Options for Minimizing Adverse Ecological Impacts, Publ. 4435, American Petroleum Institute, Washington, D.C., 1985, Chap. 2.
49. Vandermeulen, J. H. and Ross, C. W., Oil spill response in freshwater: Assessment of the impact of cleanup as a management tool, *J. Environ. Manage.*, 44, 297, 1995.
50. Atlas, R.M., Petroleum biodegradation and oil spill bioremediation, *Mar. Pollut. Bull.*, 31, 178, 1995.
51. Varanasi, U., Metabolic activation of PAH in subcellular fractions and cell cultures from aquatic and terrestrial species, in *Metabolism of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment*, Varanasi, U., Ed., CRC Press, Boca Raton, FL, 1989, Chap. 6.
52. Fu, P. P. et al., Halogenated-polycyclic aromatic hydrocarbons: A class of genotoxic environmental pollutants, *Environ. Carcino. Ecotox. Revs.*, C17, 71, 1999.
53. Ren, L. et al., Photoinduced toxicity of three polycyclic aromatic hydrocarbons (fluoranthene, pyrene, and naphthalene) to the duckweed *Lemna gibba L.* G-3, *Ecotox. Environ. Saf.*, 28, 160, 1994.
54. Arfsten, D. P., Schaeffer, D. J., and Mulveny, D. C., The effects of near ultraviolet radiation on the toxic effects of polycyclic aromatic hydrocarbons in animals and plants: A review, *Ecotox. Environ. Saf.*, 33, 1, 1996.
55. Baumann, P. C., PAH, metabolites, and neoplasia in feral fish populations, in *Metabolism of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment*, Varanasi, U., Ed., CRC Press, Boca Raton, FL, 1989, Chap. 8.
56. Chang, S., Zdanowicz, V. S., and Murchelano, R. A., Associations between liver lesions in winter flounder (*Pleuronectes americanus*) and sediment chemical contaminants from north-east United States estuaries, *ICES J. Mar. Sci.*, 55, 954, 1998.
57. Baker, J. M. et al., Tropical marine ecosystems and the oil industry; with a description of a post-oil survey in Indonesian mangroves, in *Proc. PETROMAR '80*, Graham and Trotman, London, 1980, 617.
58. Duke, N. C., Pinzon, Z. S., and Prada, T. M. C., Large-scale damage to mangrove forests following two large oil spills in Panama, *Biotropica*, 29, 2, 1997.
59. Jackson, J. B. C. et al., Ecological effects of a major oil spill on Panamanian coastal marine communities, *Science*, 243, 37, 1989.
60. Floc'h, J.-Y. and Diouris, M., Initial effects of *Amoco Cadiz* oil on intertidal algae, *Ambio*, 9, 284, 1980.
61. Thomas, M. L. H., Long term biological effects of Bunker C oil in the intertidal zone, in *Fate and Effects of Petroleum Hydrocarbons in Marine Organisms and Ecosystems*, Wolfe, D. A., Ed., Pergamon Press, New York, 1977, 238.
62. Baca, B. J., Lankford, T. E., and Gundlach, E. R., Recovery of Brittany coastal marshes in the eight years following the *Amoco Cadiz* incident, in *Proc.1987 Oil Spill Conf.*, Publ. 4452, American Petroleum Institute, Washington, D.C., 1987, 459.
63. Mendelssohn, I. A., Hester, M. W., and Hill, J. M., Assessing the recovery of coastal wetlands from oil spills, in *Proc. 1993 Int. Oil Spill Conf.*, Publ. 4580, American Petroleum Institute, Washington, D.C., 1993, 141.

---

64. Jones, D. A
the 1991 G
65. Burk, C. J.,
Ecol., 14, 5
66. Baca, B. J.,
cleanup, in
1985, 385.
67. Johansson,
Pollut. Bul
68. Shailaja, M
biomass, *M*
69. Klekowski
166, 1994.
70. Malallah, (
52, 61, 19
71. Heitkamp,
river, *Can.*
72. Braddock,
ganisms ir
Pollut. Bu
73. Megharaj,
activities i
74. Gilfillan, I
Amoco Cc
75. Houghton
in *Proc. I*
679.
76. Burns, K.
from cata
77. Davenpor
78. Griffiths,
sediment
79. Vargo G.
natural pl
Mar. Env
80. Getter, C
seven-yea
81. Parra-Par
Maracaib
D.C., 19
82. Ballou, T
untreatec
Panama,
Testing
83. Siron, R
microalg
84. Sjotun, I
Mar. Bic
85. Antrim,
122, 23,
86. Braddoc
Sound,
Fisherie
87. Pezeshk
macropl

64. Jones, D. A. et al., Long-term (1991–1995) monitoring of the intertidal biota of Saudi Arabia after the 1991 Gulf War oil spill, *Mar. Pollut. Bull.*, 36, 472, 1998.
65. Burk, C. J., A four year analysis of vegetation following an oil spill in a freshwater marsh, *J. Appl. Ecol.*, 14, 515, 1977.
66. Baca, B. J., Getter, C. D., and Lindstedt-Siva, J., Freshwater oil spill considerations: Protection and cleanup, in *Proc. 1985 Oil Spill Conf.*, Publ. 4385, American Petroleum Institute, Washington, D.C., 1985, 385.
67. Johansson, S., Larsson, U., and Boehm, P., The *Tsesis* oil spill. Impact on the pelagic ecosystem, *Mar. Pollut. Bull.*, 11, 284, 1980.
68. Shailaja, M. S., The influence of dissolved petroleum hydrocarbon residues on natural phytoplankton biomass, *Mar. Environ. Res.*, 25, 315, 1988.
69. Klekowski, E. J., Jr. et al., Petroleum pollution and mutation in mangroves, *Mar. Pollut. Bull.*, 28, 166, 1994.
70. Malallah, G. et al., Genotoxicity of oil pollution on some species of Kuwaiti flora, *Biologia*, Bratislava, 52, 61, 1997.
71. Heitkamp, M. A. and Johnson, B. T., Impact of an oil field effluent on microbial activities in a Wyoming river, *Can. J. Microbiol.*, 30, 786, 1984.
72. Braddock, J. F., Lindstrom, J. E., and Brown, E. J., Distribution of hydrocarbon-degrading microorganisms in sediments from Prince William Sound, Alaska, following the *Exxon Valdez* oil spill, *Mar. Pollut. Bull.*, 30, 125, 1995.
73. Megharaj, M. et al., Influence of petroleum hydrocarbon contamination on microalgae and microbial activities in a long-term contaminated soil, *Arch. Environ. Contam. Toxicol.*, 38, 439, 2000.
74. Gilfillan, E. S. et al., Use of remote sensing to document changes in marsh vegetation following the *Amoco Cadiz* oil spill (Brittany, France, 1978), *Mar. Pollut. Bull.*, 30, 780, 1995.
75. Houghton, J. P. et al., Prince William Sound intertidal biota seven years later: Has it recovered?, in *Proc. 1997 Oil Spill Conf.*, Publ. 4651, American Petroleum Institute, Washington, D.C., 1997, 679.
76. Burns, K. A., Garrity, S. D., and Levings, S. C., How many years until mangrove ecosystems recover from catastrophic oil spills?, *Mar. Pollut. Bull.*, 26, 239, 1993.
77. Davenport, J., Oil and planktonic ecosystems, *Phil. Trans. R. Soc. Lond.*, B297, 369, 1982.
78. Griffiths, R. P. et al., The long-term effects of crude oil on microbial processes in subarctic marine sediments, *Estuar. Coastal Shelf Sci.*, 15, 183, 1982.
79. Vargo G. A., Hutchins, M., and Almquist, G., The effect of low, chronic levels of No. 2 fuel oil on natural phytoplankton assemblages in microcosms: 1. Species composition and seasonal succession, *Mar. Environ. Res.*, 6, 245, 1982.
80. Getter, C. D., Ballou, T. G., and Koons, C. B., Effects of dispersed oil on mangroves. Synthesis of a seven-year study, *Mar. Pollut. Bull.*, 16, 318, 1985.
81. Parra-Pardi, G., Sutton, E. A., and Rincon, N. E., Effects of petroleum on algal blooms in Lake Maracaibo, in *Proc. 1985 Oil Spill Conf.*, Publ. 4385, American Petroleum Institute, Washington, D.C., 1985, 373.
82. Ballou, T. T. et al., Tropical oil pollution investigations in coastal systems (tropics): The effects of untreated and chemically dispersed Prudhoe Bay crude oil on mangroves, seagrasses, and corals in Panama, in *Oil Dispersants: New Ecological Approaches*, Flaherty, L. M., Ed., American Society for Testing and Materials STP 1018, Philadelphia, 1989, 229.
83. Siron, R. et al., Water-soluble petroleum compounds: Chemical aspects and effects on the growth of microalgae, *Sci. Total Environ.*, 104, 211, 1991.
84. Sjotun, K. and Lein, T. E., Experimental oil exposure of *Ascophyllum nodosum* (*L.*) Le Jolis, *J. Exp. Mar. Biol. Ecol.*, 170, 197, 1993.
85. Antrim, L. D. et al., Effects of petroleum products on bull kelp (*Nereocystis luetkeana*), *Mar. Biol.*, 122, 23, 1995.
86. Braddock, J. F. et al., Patterns of microbial activity in oiled and unoiled sediments in Prince William Sound, in *Proc. Exxon Valdez Oil Spill Symp.*, Rice, S. D. et al., Eds., Symposium 18, American Fisheries Society, Bethesda, 1996, 94.
87. Pezeshki, S. R. et al., The effects of oil spill and clean-up on dominant US Gulf coast marsh macrophytes: A review, *Environ. Pollut.*, 108, 129, 2000.

88. Snow, N. B. and Scott, B. F., The effect and fate of crude oil spilt on two Arctic lakes, in *Proc. 1975 Conf. Prevention Control Oil Pollut.*, American Petroleum Institute, Washington, D.C., 1975, 527.
89. Bott, T. L., Rogenmuser, K., and Thorne, P., Effects of No. 2 fuel oil, Nigerian crude oil, and used crankcase oil on benthic algal communities, *J. Environ. Sci. Health*, A13, 751, 1978.
90. Bott, T. L. and Rogenmuser, K., Effects of No. 2 fuel oil, Nigerian crude oil, and used crankcase oil on attached algal communities: Acute and chronic toxicity of water-soluble constituents, *Appl. Environ. Microbiol.*, 36, 673, 1978.
91. Federle, T. W. et al., Effects of Prudhoe Bay crude oil on primary production and zooplankton in Arctic tundra thaw ponds, *Mar. Environ. Res.*, 2, 3, 1979.
92. Baker, J. H. and Morita, R. Y., A note on the effects of crude oil on microbial activities in a stream sediment, *Environ. Pollut. (Ser. A)*, 31, 149, 1983.
93. El-Dib, M. A., Abou-Waly, H. F., and El-Naby, A. M. H., Impact of fuel oil on the freshwater alga *Selenastrum capricornutum*, *Bull. Environ. Contam. Toxicol.*, 59, 438, 1997.
94. Anoliefo, G. O. and Vwioko, D. E., Effects of spent lubricating oil on the growth of *Capsicum annum L.* and *Lycopersicon esculentum* Miller, *Environ. Pollut.*, 88, 361, 1995.
95. Chaineau, C. H., Morel, J. L., and Oudot, J., Phytotoxicity and plant uptake of fuel oil hydrocarbons, *J. Environ. Qual.*, 26, 1478, 1997.
96. Nicolotti, G. and Egli, S., Soil contamination by crude oil: Impact on the mycorrhizosphere and on the revegetation potential of forest trees, *Environ. Pollut.*, 99, 37, 1998.
97. Collins, C. M., Racine, C. H., and Walsh, M. E., The physical, chemical, and biological effects of crude oil spills after 15 years on a black spruce forest, interior Alaska, *Arctic*, 47, 164, 1994.
98. Johansson, S., Larsson, U., and Boehm, P., The Tsesis oil spill, *Mar. Pollut. Bull.*, 11, 284, 1980.
99. Batten, S. D., Allen, R. J. S., and Wotton, C. O. M., The effects of the *Sea Empress* oil spill on the plankton of the southern Irish Sea, *Mar. Pollut. Bull.*, 36, 764, 1998.
100. Kingston, P. F., Impact of offshore oil production installations on the benthos of the North Sea, *ICES J. Mar. Sci.*, 49, 45, 1992.
101. Daan, R. and Mulder, M., On the short-term and long-term impact of drilling activities in the Dutch sector of the North Sea, *ICES J. Mar. Sci.*, 53, 1036, 1996.
102. Jackson, J. B. C. et al., Ecological effects of a major oil spill on Panamanian coastal marine communities, *Science*, 243, 37, 1989.
103. Lee, R. F. and Page, D. S., Petroleum hydrocarbons and their effects in subtidal regions after major oil spills, *Mar. Pollut. Bull.*, 34, 928, 1997.
104. Dauvin, J.-C., The fine sand *Abra alba* community of the Bay of Morlaix twenty years after the *Amoco Cadiz* oil spill, *Mar. Pollut. Bull.*, 36, 669, 1998.
105. Jewett, S. C. et al., "*Exxon Valdez*" oil spill: Impacts and recovery in the soft-bottom benthic community in and adjacent to eelgrass beds, *Mar. Ecol. Prog. Ser.*, 185, 59, 1999.
106. Sanders, H. L. et al., Anatomy of an oil spill: Long-term effects from the grounding of the barge *Florida* off West Falmouth, Massachusetts, *J. Mar. Res.*, 38, 265, 1980.
107. Highsmith, R. C. et al., Impact of the *Exxon Valdez* oil spill on intertidal biota, in *Proc. Exxon Valdez Oil Spill Symp.*, Rice, S. D. et al., Eds., Symposium 18, American Fisheries Society, Bethesda, 1996, 212.
108. Levings, S. C., Garrity, S. D., and Burns, K. A., The *Galeta* oil spill. III. Chronic reoiling, long-term toxicity of hydrocarbon residues and effects on epibiota in the mangrove fringe, *Estuar. Coastal Shelf Sci.*, 38, 365, 1994.
109. Crunkilton, R. L. and Duchrow, R. M., Impact of a massive crude oil spill on the invertebrate fauna of a Missouri Ozark stream, *Environ. Pollut.*, 63, 13, 1990.
110. Poulton, B. C. et al., Effects of an oil spill on leafpack-inhabiting macroinvertebrates in the Chariton River, Missouri, *Environ. Pollut.*, 99, 115, 1998.
111. Neff, J. et al., An oil spill in an Illinois lake: Ecological and human health assessment, in *Proc. 1995 Int. Oil Spill Conf.*, Publ. 4620, American Petroleum Institute, Washington, D.C., 1995, 415.
112. Suchanek, T. H., Oil impacts on marine invertebrate populations and communities, *Am. Zool.*, 33, 510, 1993.
113. Driskell, W. B. et al., Recovery of Prince William Sound intertidal in fauna from *Exxon Valdez* oiling and shoreline treatments, 1989 through 1992, in *Proc. Exxon Valdez Oil Spill Symp.*, Rice, S. D. et al., Eds., Symposium 18, American Fisheries Society, Bethesda, 1996, 362.

---

114. Straugha
spill data
115. Davies, J
in an enc
Cons. Ini
116. Carr, R.
and oil d
117. Frithsen,
level add
118. Cross, W
oil on ar
119. Moore, M
effects a
Aquatic
120. Nance, J
gradient
121. Burger, J
(*Uca pu*
122. Brown, H
a #2 fuel
123. Shelton,
lated wat
124. Carman,
taminati
49, 255,
125. Rogerso
rotifer *A.*
Environ.
126. Woodwa
contamin
Toxicol.,
127. Miller, M
and a su
128. Bobra,
Daphnia
129. Calfee,
Environ.
130. Faulkne
hispidus
2000.
131. Faulkne
terrestri
86, 200
132. Erstfeld
inverteb
133. Dorn, P.
and plan
134. Pelletier
hydroca
19, 269
135. Bowma
Argo M
1978, 1
136. Malins,
effects,

akes, in *Proc. 1975*
D.C., 1975, 527.
crude oil, and used
978.
used crankcase oil
ents, *Appl. Environ.*
nd zooplankton in
tivities in a stream
he freshwater alga
f *Capsicum annum*
l oil hydrocarbons,
hizosphere and on
ological effects of
164, 1994.
. 11, 284, 1980.
ess oil spill on the
e North Sea, *ICES*
vities in the Dutch
al marine commu-
egions after major
rs after the *Amoco*
tom benthic com-
ding of the barge
roc. Exxon Valdez
, Bethesda, 1996,
oiling, long-term
uar. Coastal Shelf
nvertebrate fauna
s in the Chariton
nt, in *Proc. 1995*
995, 415.
s, *Am. Zool.*, 33,
xon Valdez oiling
p., Rice, S. D. et

114. Straughan, D. and Hadley, D., Experiments with *Littorina* species to determine the relevancy of oil spill data from southern California to the Gulf of Alaska, *Mar. Environ. Res.*, 1, 135, 1978.
115. Davies, J. M. et al., Some effects of oil-derived hydrocarbons on a pelagic food web from observations in an enclosed ecosystem and a consideration of their implications for monitoring, *Rapp. P.-v. Reun. Cons. Int. Explor. Mer.*, 179, 201, 1980.
116. Carr, R. S. and Linden, O., Bioenergetic responses of *Gammarus salinus* and *Mytilus edulis* to oil and oil dispersants in a model ecosystem, *Mar. Ecol.Prog. Ser.*, 19, 285, 1984.
117. Frithsen, J. B., Elmgren, R., and Rudnick, D. T., Responses of benthic meiofauna to long-term, low-level additions of No. 2 fuel oil, *Mar. Ecol. Prog. Ser.*, 23, 1, 1985.
118. Cross, W. E., Martin, C. M., and Thomson, D. H., Effects of experimental releases of oil and dispersed oil on arctic nearshore macrobenthos. II. Epibenthos, *Arctic*, 40 (Suppl. 1), 201, 1987.
119. Moore, M. N., Livingstone, D. R., and Widdows, J., Hydrocarbons in marine mollusks: Biological effects and ecological consequences, in *Metabolism of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment*, Varanasi, U., Ed., CRC Press, Boca Raton, FL, 1989, Chap. 9.
120. Nance, J. M., Effects of oil/gas field produced water on the macrobenthic community in a small gradient estuary, *Hydrobiologia*, 220, 189, 1991.
121. Burger, J., Brzorad, J., and Gochfeld, M., Immediate effects of an oil spill on behavior of fiddler crabs (*Uca pugnax*), *Arch. Environ. Contam. Toxicol.*, 20, 404, 1991.
122. Brown, R. P., Cristini, A., and Cooper, K. R., Histopathological alterations in *Mya arenaria* following a #2 fuel oil spill in the Arthur Kill, Elizabeth, New Jersey, *Mar. Environ. Res.*, 34, 65, 1992.
123. Shelton, M. E. et al., Degradation of weathered oil by mixed marine bacteria and the toxicity of accumulated water-soluble material to two marine crustacea, *Arch. Environ. Contam. Toxicol.*, 36, 13, 1999.
124. Carman, K. R., Fleeger, J. W., and Pomarico, S. M., Does historical exposure to hydrocarbon contamination alter the response of benthic communities to diesel contamination?, *Mar. Environ. Res.*, 49, 255, 2000.
125. Rogerson, A., Berger, J., and Grosso, C. M., Acute toxicity of ten crude oils on the survival of the rotifer *Asplanchna sieboldi* and sublethal effects on rates of prey consumption and neonate production, *Environ. Pollut. (Ser. A)*, 29, 179, 1982.
126. Woodward, D. F. and Riley, R. G., Petroleum hydrocarbon concentrations in a salmonid stream contaminated by oil field discharge water and effects on macrobenthos, *Arch. Environ. Contam. Toxicol.*, 12, 327, 1983.
127. Miller, M. C. and Stout, J. R., Effects of a controlled under-ice oil spill on invertebrates of an arctic and a subarctic stream, *Environ. Pollut. (Ser. A)*, 42, 99, 1986.
128. Bobra, A. M. et al., Acute toxicity of dispersed fresh and weathered crude oil and dispersants to *Daphnia magna*, *Chemosphere*, 19, 1199, 1989.
129. Calfee, R. D. et al., Photoenhanced toxicity of a weathered oil on *Ceriodaphnia dubia* reproduction, *Environ. Sci. Pollut. Res.*, 6, 207, 1999.
130. Faulkner, B. C. and Lochmiller, R. L., Ecotoxicity revealed in parasite communities of *Sigmodon hispidus* in terrestrial environments contaminated with petrochemicals, *Environ. Pollut.*, 110, 135, 2000.
131. Faulkner, B. C. and Lochmiller, R. L., Increased abundance of terrestrial isopod populations in terrestrial ecosystems contaminated with petrochemical wastes, *Arch. Environ. Contam. Toxicol.*, 39, 86, 2000.
132. Erstfeld, K. M. and Snow-Ashbrook, J., Effects of chronic low-level PAH contamination on soil invertebrate communities, *Chemosphere*, 39, 2117, 1999.
133. Dorn, P. B. et al., Assessment of the acute toxicity of crude oils in soils using earthworms, Microtox, and plants, *Chemosphere*, 37, 845, 1998.
134. Pelletier, M. C. et al., Importance of maternal transfer of the photoreactive polycyclic aromatic hydrocarbon fluoranthrene from benthic adult bivalves to their pelagic larvae, *Environ. Toxicol. Chem.*, 19, 2691, 2000.
135. Bowman, R. E. and Langton, R. W., Fish predation on oil-contaminated prey from the region of the Argo Merchant oil spill, in *In the Wake of the Argo Merchant*, University of Rhode Island, Kingston, 1978, 137.
136. Malins, D. C. and Hodgins, H. O., Petroleum and marine fishes: A review of uptake, disposition, and effects, *Environ. Sci. Technol.*, 15, 1272, 1981.

137. Hampson, G. R. and Sanders, H. L., Local oil spill, *Oceanus*, 15,8, 1969.
138. Teal, J. M. and Howarth, R. W., Oil spill studies: A review of ecological effects, *Environ. Manage.*, 8, 27, 1984.
139. Woodward, D. F., Riley, R. G., and Smith, C. E., Accumulation, sublethal effects, and safe concentration of a refined oil as evaluated with cutthroat trout, *Arch. Environ. Contam. Toxicol.*, 12, 455, 1983.
140. Hedtke, S. F. and Puglisi, F. A., Short-term toxicity of five oils to four freshwater species, *Arch. Environ. Contam. Toxicol.*, 11, 425, 1982.
141. Anderson, J. W. et al., Toxicity of dispersed and undispersed Prudhoe Bay crude oil fractions to shrimp and fish, in *Proc. 1987 Oil Spill Conf.*, Publ. 4452, American Petroleum Institute, Washington, D.C., 1987, 235.
142. Barnett, J. and Toews, D., The effects of crude oil and the dispersant, Oilsperse 43, on respiration and coughing rates in Atlantic salmon (*Salmo salar*), *Can. J. Zool.*, 56, 307, 1978.
143. Little, E. E. et al., Assessment of the photoenhanced toxicity of a weathered oil to the tidewater silverside, *Environ. Toxicol. Chem.*, 19, 926, 2000.
144. Chambers, J. E. et al., Enzyme activities following chronic exposure to crude oil in a simulated ecosystem, *Environ. Res.*, 20, 140, 1979.
145. Thomas, P., Woodin, B. R., and Neff, J. M., Biochemical responses of the striped mullet *Mugil cephalus* to oil exposure, I. Acute responses — interrenal activations and secondary stress responses, *Mar. Biol.*, 59, 141, 1980.
146. Fletcher, G. L., Kiceniuk, J. W., and Williams, U. P., Effects of oiled sediments on mortality, feeding and growth of winter flounder *Pseudopleuronectes americanus*, *Mar. Ecol. Prog. Ser.*, 4, 91, 1981.
147. Weber, D. D. et al., Avoidance reactions of migrating adult salmon to petroleum hydrocarbons, *Can. J. Fish. Aquat. Sci.*, 38, 779, 1981.
148. Thomas, P. and Budiantara, L., Reproductive life history stages sensitive to oil and naphthalene in Atlantic croaker, *Mar. Environ. Res.*, 39, 147, 1995.
149. Kuehn, R. L. et al., Relationships among petroleum refining, water and sediment contamination, and fish health, *J. Toxicol. Environ. Health*, 46, 101, 1995.
150. Willette, M., Impacts of the *Exxon Valdez* oil spill on the migration, growth, and survival of juvenile pink salmon in Prince William Sound, in *Proc. Exxon Valdez Oil Spill Symp.*, Symposium 18, American Fisheries Society, Bethesda, 1996, 533.
151. Gregg, J. C, Fleeger, J. W., and Carman, K. R., Effects of suspended, diesel-contaminated sediment on feeding rate in the darter goby, *Gobionellus boleosoma* (*Teleostei: Gobiidae*), *Mar. Pollut. Bull.*, 34, 269, 1997.
152. Moles, A. and Norcross, B. L., Effects of oil-laden sediments on growth and health of juvenile flatfishes, *Can. J. Fish. Aquat. Sci.*, 55, 605, 1998.
153. Carls, M. G. et al., Expression of viral hemorrhagic septicemia virus in prespawning Pacific herring (*Clupea pallasi*) exposed to weathered crude oil, *Can. J. Fish. Aquat. Sci.*, 55, 2300, 1998.
154. Khan, R. A., Study of pearl dace (*Margariscus margarita*) inhabiting a stillwater pond contaminated with diesel fuel, *Bull. Environ. Contam. Toxicol.*, 62, 638, 1999.
155. Tilseth, S., Soldberg, T. S., and Westheim, K., Sublethal effects of the water-soluble fraction of Ekofisk crude oil on the early larval stages of cod (*Gadus morhua L.*), *Mar. Environ. Res.*, 11, 1, 1984.
156. Marty, G. D. et al., Histopathology and cytogenetic evaluation of Pacific herring larvae exposed to petroleum hydrocarbons in the laboratory or in Prince William Sound, Alaska, after the *Exxon Valdez* oil spill, *Can. J. Fish. Aquat. Sci.*, 54, 1846, 1997.
157. Heintz, R. A., Short, J. W., and Rice, S. D., Sensitivity of fish embryos to weathered crude oil: Part II. Increased mortality of pink salmon (*Oncorhynchus gorbuscha*) embryos incubating downstream from weathered *Exxon Valdez* crude oil, *Environ. Toxicol. Chem.*, 18, 494, 1999.
158. Carls, M. G., Rice, S. D., and Hose, J. E., Sensitivity of fish embryos to weathered crude oil: Part I. Low-level exposure during incubation causes malformations, genetic damage, and mortality in larval Pacific herring (*Clupea pallasi*), *Environ. Toxicol. Chem.*, 18, 481, 1999.
159. Falk-Petersen, I. and Kjorsvik, E., Acute toxicity tests of the effects of oils and dispersants on marine fish embryos and larvae — A review, *Sarsia*, 72, 411, 1987.
160. Hughes, J. B., Cytological-cytogenetic analyses of winter flounder embryos collected from the benthos at the barge North Cape oil spill, *Mar. Pollut. Bull.*, 38, 30, 1999.

---

161. Brown, E. D. et
after the *Exxon*
Fisheries Societ
162. Bue, B. G., Sh
Prince William
127, 35, 1998.
163. Mankki, J. and
archipelago, *A*
164. Squire, J., Jr.,
fishery resourc
165. Geiger, H. J. et
caused by the
Fisheries Socie
166. Cronin, M. A.
to induce herit
167. Spaulding, M.
Bank fish spec
168. Reed, M. et a
Estuar. Coast
169. Williams, D.
cytochrome P
170. French, B. L.
(*Pleuronectes*
171. Myers, M. S.
and effects in
Bull., 37, 92,
172. Myers, M. S.
toxic chemic
173. Baumann, P.
hydrocarbon
matic Hydro
G. L., Eds.,
174. Smith, S. B.,
elevated inci
175. Roberts, M.
Leiostomus
176. Steyermark,
alized Schuy
177. Van der Oos
(*Anguilla an*
1994.
178. Pierce, V., T
Junge, R. E.
179. Witham, R.,
Ecolog. Imp
180. Hall, R. J.,
exposed to
181. Symens, P.
in the Arabi
van Vessem.
Slimbridge,
182. Gramentz,
the central
183. Vargo, S. e
Shelf Rep.

Environ. Manage.,

and safe concen-
ol., 12, 455, 1983.
ter species, Arch.

ractions to shrimp
Washington, D.C.,

43, on respiration

to the tidewater

il in a simulated

ed mullet Mugil
stress responses,

nortality, feeding
r., 4, 91, 1981.
drocarbons, Can.

d naphthalene in

ntamination, and

vival of juvenile
um 18, American

inated sediment
'ar. Pollut. Bull.,

alth of juvenile

Pacific herring
, 1998.
nd contaminated

ction of Ekofisk
1, 1, 1984.
rvae exposed to
ne Exxon Valdez

l crude oil: Part
ng downstream

rude oil: Part I.
rtality in larval

ants on marine

om the benthos

---

161. Brown, E. D. et al., Injury to the early life history stages of Pacific herring in Prince William Sound after the *Exxon Valdez* oil spill, in *Proc. Exxon Valdez Oil Spill Symp.*, Symposium 18, American Fisheries Society, Bethesda, 1996, 448.
162. Bue, B. G., Sharr, S., and Seeb, J. E., Evidence of damage to pink salmon populations inhabiting Prince William Sound, Alaska, two generations after the *Exxon Valdez* oil spill, *Trans. Am. Fish. Soc.*, 127, 35, 1998.
163. Mankki, J. and Vauras, J., Littoral fish populations after an oil tanker disaster in the Finnish SW archipelago, *Ann. Zool. Fennici*, 11, 120, 1974.
164. Squire, J., Jr., Effects of the Santa Barbara, Calif., oil spill on the apparent abundance of pelagic fishery resources, *Mar. Fish. Rev.*, 54, 7, 1992.
165. Geiger, H. J. et al., A life history approach to estimating damage to Prince William Sound pink salmon caused by the *Exxon Valdez* oil spill, in *Proc. Exxon Valdez Oil Spill Symp.*, Symposium 18, American Fisheries Society, Bethesda, 1996, 487.
166. Cronin, M. A. and Bickham, J. W., A population genetic analysis of the potential for a crude oil spill to induce heritable mutations and impact natural populations, *Ecotoxicology*, 7, 259, 1998.
167. Spaulding, M. L. et al., Oil-spill fishery impact assessment model: Application to selected Georges Bank fish species, *Estuar. Coastal Shelf Sci.*, 16, 511, 1983.
168. Reed, M. et al., Oil spill fishery impact assessment modeling: The fisheries recruitment problem, *Estuar. Coastal Shelf Sci.*, 19, 591, 1984.
169. Williams, D. E., Lech, J. J., and Buhler, D. R., Xenobiotics and xenoestrogens in fish: Modulation of cytochrome P450 and carcinogenesis, *Mutation Res.*, 399, 179, 1998.
170. French, B. L. et al., Accumulation and dose-response of hepatic DNA adducts in English sole (*Pleuronectes vetulus*) exposed to a gradient of contaminated sediments, *Aquat. Toxicol.*, 36, 1, 1996.
171. Myers, M. S. et al., Toxicopathic hepatic lesions as biomarkers of chemical contaminant exposure and effects in marine bottomfish species from the Northeast and Pacific Coasts, USA, *Mar. Pollut. Bull.*, 37, 92, 1998.
172. Myers, M. S. et al., Relationships between hepatic neoplasms and related lesions and exposure to toxic chemicals in marine fish from the U.S. west coast, *Environ. Health Perspect.*, 90, 7, 1991.
173. Baumann, P. C., Smith, W. D., and Ribick, M., Hepatic tumor rates and polynuclear aromatic hydrocarbon levels in two populations of brown bullhead (*Ictalurus nebulosus*), in *Polynuclear Aromatic Hydrocarbons: Physical and Biological Chemistry*, Cooke, M. W., Dennis, A. J., and Fisher, G. L., Eds., Battelle Press, Columbus, Ohio, 1982, 93.
174. Smith, S. B., Blouin, M. A., and Mac, M. J., Ecological comparisons of Lake Erie tributaries with elevated incidence of fish tumors, *J. Great Lakes Res.*, 20, 701, 1994.
175. Roberts, M. H., Jr. et al., Acute toxicity of PAH contaminated sediments to the estuarine fish, *Leiostomus xanthurus*, *Bull. Environ. Contam. Toxicol.*, 42, 142, 1989.
176. Steyermark, A. C. et al., Biomarkers indicate health problems in brown bullheads from the industrialized Schuylkill River, Philadelphia, *Trans. Am. Fish. Soc.*, 128, 328, 1999.
177. Van der Oost, R. et al., Bioaccumulation, biotransformation and DNA binding of PAHs in feral eel (*Anguilla anguilla*) exposed to polluted sediments: A field survey, *Environ. Toxicol. Chem.*, 13, 859, 1994.
178. Pierce, V., The effects of the Arabian Gulf oil spill on wildlife, in *1991 Proc. Am. Assoc. Zoo Veterin.*, Junge, R. E., Ed., Calgary, Canada, 1991, 370.
179. Witham, R., Does a problem exist relative to small sea turtles and oil spills?, in *Proc. Conf. Assess. Ecolog. Impacts Oil Spills*, American Institute of Biological Sciences, Keystone, Colorado, 1978, 630.
180. Hall, R. J., Belisle, A. A., and Sileo, L., Residues of petroleum hydrocarbons in tissues of sea turtles exposed to the *Ixtoc I* oil spill, *J. Wildl. Dis.*, 19, 106, 1983.
181. Symens, P. and Al Salamah, M. I., The impact of the Gulf War oil spills on wetlands and waterfowl in the Arabian Gulf, in *Wetland and Waterfowl Conservation in South and West Asia*, Moser, M. and van Vessem, J., Eds., Special Publ. No. 25, The International Waterfowl and Wetlands Research Bureau, Slimbridge, U.K., 1993, 24.
182. Gramentz, D., Involvement of loggerhead turtle with the plastic, metal, and hydrocarbon pollution in the central Mediterranean, *Mar. Pollut. Bull.*, 19, 11, 1988.
183. Vargo, S. et al., Effects of Oil on Marine Turtles, Minerals Management Service Outer Continental Shelf Rep. MMS 86-0070, Vienna, VA, 1986.

184. Lutcavage, M. E. et al., Physiologic and clinicopathologic effects of crude oil on loggerhead sea turtles, *Arch. Environ. Contam. Toxicol.*, 28, 417, 1995.
185. McGrath, E. A. and Alexander, M. M., Observations on the exposure of larval bullfrogs to fuel oil, *Trans. N.E. Sect. Wildl. Soc.*, 36, 45, 1979.
186. Mahaney, P. A., Effects of freshwater petroleum contamination on amphibian hatching and metamorphosis, *Environ. Toxicol. Chem.*, 13, 259, 1994.
187. Lefcort, H. et al., The effects of used motor oil, silt, and the water mold *Saprolegnia parasitica* on the growth and survival of mole salamanders (Genus *Ambystoma*), *Arch. Environ. Contam. Toxicol.*, 32, 383, 1997.
188. Anderson, R. S., Doos, J. E., and Rose, F. L., Differential ability of *Ambystoma tigrinum* hepatic microsomes to produce mutagenic metabolites from polycyclic aromatic hydrocarbons and aromatic amines, *Cancer Lett.*, 16, 33, 1982.
189. Fernandez, M. and l'Haridon, J., Effects of light on the cytotoxicity and genotoxicity of benzo(a)pyrene and an oil refinery effluent in the newt, *Environ. Molec. Mutagen.*, 24, 124, 1994.
190. Vermeer, K. and Vermeer, R., Oil threat to birds on the Canadian coast, *Can. Field-Nat.*, 89, 278, 1975.
191. Tseng, F. S., Care of oiled seabirds: A veterinary perspective, in *Proc. 1993 Int. Oil Spill Conf.*, Publ. 4580, American Petroleum Institute, Washington, D.C., 1993, 421.
192. Jenssen, B. M., Review article: Effects of oil pollution, chemically treated oil, and cleaning on the thermal balance of birds, *Environ. Pollut.*, 86, 207, 1994.
193. Hartung, R. and Hunt, G. S., Toxicity of some oils to waterfowl, *J. Wildl. Manage.*, 30, 564, 1966.
194. Miller, D. S., Peakall, D. B., and Kinter, W. B., Ingestion of crude oil: Sublethal effects in herring gull chicks, *Science*, 199, 15, 1978.
195. Szaro. R. C., Hensler, G., and Heinz, G. H., Effects of chronic ingestion of No. 2 fuel oil on mallard ducklings, *J. Toxicol. Environ. Health*, 7, 789, 1981.
196. Albers, P. H., Effects of oil on avian reproduction: A review and discussion, in *The Effects of Oil on Birds. A Multi-Discipline Symposium*, Tri-State Bird Rescue and Research, Inc., Wilmington, DE, 1983, 78.
197. Leighton, F. A., The toxicity of petroleum oils to birds: An overview, in *The Effects of Oil on Wildlife*, White, J. et al., Eds., The Sheridan Press, Hanover, PA, 1991, 43.
198. Burger, A. E. and Fry, D. M, Effects of oil pollution on seabirds in the northeast Pacific, in *The Status, Ecology, and Conservation of Marine Birds of the North Pacific*, Vermeer, K. et al., Eds., Canadian Wildlife Service Special Publication, Ottawa, 1993, 254.
199. Fry, D. M. et al., Reduced reproduction of wedge-tailed shearwaters exposed to weathered Santa Barbara crude oil, *Arch. Environ. Contam. Toxicol.*, 15, 453, 1986.
200. Eppley, Z. A., Assessing indirect effects of oil in the presence of natural variation: The problem of reproductive failure in south polar skuas during the Bahia Paraiso oil spill, *Mar. Pollut. Bull.*, 25, 307, 1992.
201. Fowler, G. S., Wingfield, J. C., and Goersma, P. D., Hormonal and reproductive effects of low levels of petroleum fouling in Magellanic penguins (*Spheniscus magellanicus*), *The Auk*, 112, 382, 1995.
202. Walton, P. et al., Sub-lethal effects of an oil pollution incident on breeding kittiwakes, *Rissa tridactyla*, *Mar. Ecol. Prog. Ser.*, 155, 261, 1997.
203. Briggs, K. T., Gershwin, M. E., and Anderson, D. W., Consequences of petrochemical ingestion and stress on the immune system of seabirds, *ICES J. Mar. Sci.*, 54, 718, 1997.
204. Parnell, J. F., Shields, M. A., and Frierson, D., Jr., Hatching success of brown pelican eggs after contamination with oil, *Colonial Waterbirds*, 7, 22, 1984.
205. Hoffman, D. J., Embryotoxicity and teratogenicity of environmental contaminants to bird eggs, *Rev. Environ. Contam. Toxicol.*, 115, 39, 1990.
206. Szaro, R. C., Coon, N. C., and Stout, W., Weathered petroleum: Effects on mallard egg hatchability, *J. Wildl. Manage.*, 44, 709, 1980.
207. Stubblefield, W. A. et al., Evaluation of the toxic properties of naturally weathered *Exxon Valdez* crude oil to surrogate wildlife species, in *Exxon Valdez Oil Spill: Fate and Effects in Alaskan Waters*, Wells, P. G., Butler, J. N., and Hughes, J. S., Eds., ASTM STP 1219, American Society for Testing and Materials, Philadelphia, 1995, 665.
208. Parsons, K. C., The Arthur Kill oil spills: Biological effects in birds, in *Before and After an Oil Spill: The Arthur Kill*, Burger, J., Ed., Rutgers University Press, Rutgers, NJ, 1994, 305.
209. Day, R. H. et al., Effects of the *Exxon Valdez* oil spill on habitat use by birds in Prince William Sound, Alaska, *Ecol. Appl.*, 7, 593, 1997.

---

210. Piatt, J. F. and
changes in the
18, American
211. Bowman, T.
after the *Exxo*
212. Piatt, J. F. an
1993, *Mar. Po*
213. Dunnet, G. M
214. Piatt, J. F., C
in *The Effects*
215. Piatt, J. F. an
Exxon Valdez
216. Ford, R. G. e
Valdez Oil Sp
217. Bernatowicz,
in 1989 and
18, America
218. Wiens, J. A.
Sound, Alas
219. Andres, B.
Manage., 61
220. Esler, D., W
the *Exxon V*
221. Parr, S. J.,
Pembrokesh
Institute, W
222. Symens, P.
northern Ar
223. Samuels, W
northern G
224. Samuels, V
in the mid-
225. Wiens, J.
and sensiti
Report Vol
226. Ford, R. G
and kittiw
227. Albers, P.
U.S. Coas
228. Cairns, D
growth ce
229. Matsumo
ambient a
230. Brunstro
in three c
common
231. Brunstro
by polyc
Chem.-B
232. Naf, C.,
carbons
mollissi
233. Mayura,
aromati

210. Piatt, J. F. and Anderson, P., Response of common murres to the *Exxon Valdez* oil spill and long-term changes in the Gulf of Alaska marine ecosystem, in *Proc. Exxon Valdez Oil Spill Symp.*, Symposium 18, American Fisheries Society, Bethesda, Maryland, 1996, 720.
211. Bowman, T. D., Schempf, P. F., and Hodges, J. I., Bald eagle population in Prince William Sound after the *Exxon Valdez* oil spill, *J. Wildl. Manage.*, 61, 962, 1997.
212. Piatt, J. F. and van Pelt, T. I., Mass-mortality of guillemots (*Uria aalge*) in the Gulf of Alaska in 1993, *Mar. Pollut. Bull.*, 34, 656, 1997.
213. Dunnet, G. M., Oil pollution and seabird populations, *Phil. Trans. R. Soc. Lond.*, B297, 413, 1982.
214. Piatt, J. F., Carter, H. R., and Nettleship, D. N., Effects of oil pollution on marine bird populations, in *The Effects of Oil on Wildlife*, White, J. et al., Eds., The Sheridan Press, Hanover, PA, 1991, 125.
215. Piatt, J. F. and Gord R. G., How many seabirds were killed by the *Exxon Valdez* oil spill?, in *Proc. Exxon Valdez Oil Spill Symp.*, Symposium 18, American Fisheries Society, Bethesda, 1996, 712.
216. Ford, R. G. et al., Total direct mortality of seabirds from the *Exxon Valdez* oil spill, in *Proc. Exxon Valdez Oil Spill Symp.*, Symposium 18, American Fisheries Society, Bethesda, 1996, 684.
217. Bernatowicz, J. A., Schempf, P. F., and Bowman, T. D., Bald eagle productivity in south-central Alaska in 1989 and 1990 after the *Exxon Valdez* oil spill, in *Proc. Exxon Valdez Oil Spill Symp.*, Symposium 18, American Fisheries Society, Bethesda, 1996, 785.
218. Wiens, J. A. et al., Effects of the *Exxon Valdez* oil spill on marine bird communities in Prince William Sound, Alaska, *Ecol. Appl.*, 6, 828, 1996.
219. Andres, B. A., The *Exxon Valdez* oil spill disrupted the breeding of black oystercatchers, *J. Wildl. Manage.*, 61, 1322, 1997.
220. Esler, D., Winter survival of adult female harlequin ducks in relation to history of contamination by the *Exxon Valdez* oil spill, *J. Wildl. Manage.*, 64, 839, 2000.
221. Parr, S. J., Haycock, R. J., and Smith, M. E., The impact of the *Sea Empress* oil spill on birds of the Pembrokeshire coast and islands, in *Proc. 1997 Int. Oil Spill Conf.*, Publ. 4651, American Petroleum Institute, Washington, D.C., 1997, 217.
222. Symens, P. and Suhaibani, A., The impact of the 1991 Gulf War oil spill on bird populations in the northern Arabian Gulf — A review, *Courier Forsch.-Inst. Senckenberg*, 166, 47, 1994.
223. Samuels, W. B. and Lanfear, K. J., Simulations of seabird damage and recovery from oilspills in the northern Gulf of Alaska, *J. Environ. Manage.*, 15, 169, 1982.
224. Samuels, W. B. and Ladino, A., Calculations of seabird population recovery from potential oilspills in the mid-Atlantic region of the United States, *Ecol. Model.*, 21, 63, 1984.
225. Wiens, J. A. et al., Simulation modelling of marine bird population energetics, food consumption, and sensitivity to perturbation, in *Environmental Assessment of the Alaskan Continental Shelf*, Annual Report Vol. 1, U.S. Department of Commerce and U.S. Dept. of Interior, Boulder, 1979, 217.
226. Ford, R. G. et al., Modelling the sensitivity of colonially breeding marine birds to oil spills: Guillemot and kittiwake populations on the Pribilof Islands, Bering Sea, *J. Appl. Ecol.*, 19, 1, 1982.
227. Albers, P. H., Effects of oil and dispersants on birds, in *Proc. 1984 Region 9 Oil Dispersants Workshop*, U.S. Coast Guard, Santa Barbara, California, 1984, 101.
228. Cairns, D. K. and Elliot, R. D., Oil spill impact assessment for seabirds: The role of refugia and growth centres, *Biol. Conserv.*, 40, 1, 1987.
229. Matsumoto, H. and Kashimoto, T., Embryotoxicity of organic extracts from airborne particulates in ambient air in the chicken embryo, *Arch. Environ. Contam. Toxicol.*, 15, 447, 1986.
230. Brunstrom, B., Broman, D., and Naf, C., Embryotoxicity of polycyclic aromatic hydrocarbons (PAHs) in three domestic avian species, and of PAHs and coplanar polychlorinated biphenyls (PCBs) in the common eider, *Environ. Pollut.*, 67, 133, 1990.
231. Brunstrom, B., Embryolethality and induction of 7-ethoxyresorufin *O*-deethylase in chick embryos by polychlorinated biphenyls and polycyclic aromatic hydrocarbons having Ah receptor affinity, *Chem.-Biol. Interactions*, 81, 69, 1991.
232. Naf, C., Broman, D., and Brunstrom, B., Distribution and metabolism of polycyclic aromatic hydrocarbons (PAHs) injected into eggs of chicken (*Gallus domesticus*) and common eider duck (*Somateria mollissima*), *Environ. Toxicol. Chem.*, 11, 1653, 1992.
233. Mayura, K. et al., Multi-bioassay approach for assessing the potency of complex mixtures of polycyclic aromatic hydrocarbons, *Chemosphere*, 38, 1721, 1999.

234. Patton, J. F. and Dieter, M. P., Effects of petroleum hydrocarbons on hepatic function in the duck, *Comp. Biochem. Physiol.*, 65C, 33, 1980.
235. Miller, D. S., Hallett, D. J., and Peakall, D. B., Which components of crude oil are toxic to young seabirds?, *Environ. Toxicol. Chem.*, 1, 39, 1982.
236. Peakall, D. B. et al., Toxicity of Prudhoe Bay crude oil and its aromatic fractions to nestling herring gulls, *Environ. Res.*, 27, 206, 1982.
237. Trust, K. A., Fairbrother, A., and Hooper, M. J., Effects of 7,12-dimethylbenz[a]anthracene on immune function and mixed-function oxygenase activity in the European starling, *Environ. Toxicol. Chem.*, 13, 821, 1994.
238. Custer, T. W. et al., Trace elements, organochlorines, polycyclic aromatic hydrocarbons, dioxins, and furans in lesser scaup wintering on the Indiana Harbor Canal, *Environ. Pollut.*, 110, 469, 2000.
239. Hansen, D. J., The Potential Effects of Oil Spills and Other Chemical Pollutants on Marine Mammals Occurring in Alaskan Waters, Minerals Management Service Outer Continental Shelf Rep. MMS 85-0031, Anchorage, Alaska, 1985.
240. Waldichuk, M., Sea otters and oil pollution, *Mar. Pollut. Bull.*, 21, 10, 1990.
241. Baker, J. R. et al., *Otter Lutra lutra* L. mortality and marine oil pollution, *Biol. Conserv.*, 20, 311, 1981.
242. Oritsland, N. A. et al., Effect of Crude Oil on Polar Bears, Environmental Studies No. 24, Northern Affairs Program, Ottawa, 1981.
243. Lipscomb, T. P. et al., Histopathologic lesions in sea otters exposed to crude oil, *Vet. Pathol.*, 30, 1, 1993.
244. Bickham, J. W. et al., Flow cytometric determination of genotoxic effects of exposure to petroleum in mink and sea otters, *Ecotoxicology*, 7, 191, 1998.
245. Loughlin, T. R., Ballachey, B. E., and Wright, B. A., Overview of studies to determine injury caused by the *Exxon Valdez* oil spill to marine mammals, in *Proc. Exxon Valdez Oil Spill Symp.*, Symposium 18, American Fisheries Society, Bethesda, 1996, 798.
246. Duffy, L. K. et al., Evidence for recovery of body mass and haptoglobin values of river otters following the *Exxon Valdez* oil spill, *J. Wildl. Dis.*, 30, 421, 1994.
247. Jenssen, B. M., An overview of exposure to, and effects of, petroleum oil and organochlorine pollution in Grey seals (*Halichoerus grypus*), *Sci. Total Environ.*, 186, 109, 1996.
248. Williams, T. M., O'Connor, D. J., and Nielsen, S. W., The effects of oil on sea otters: Histopathology, toxicology, and clinical history, in *Emergency Care and Rehabilitation of Oiled Sea Otters: A Guide for Oil Spills Involving Fur-Bearing Marine Mammals*, Williams, T. M. and Davis, R. W., Eds., University of Alaska Press, Fairbanks, 1995, Chap. 1.
249. Monson, D. H. et al., Long-term impacts of the *Exxon Valdez* oil spill on sea otters, assessed through age-dependent mortality patterns, *Proc. Natl. Acad. Sci.*, 97, 6562, 2000.
250. Frost, K. J., Lowry, L. F., and Ver Hoef, J. M., Monitoring the trend of harbor seals in Prince William Sound, Alaska, after the *Exxon Valdez* oil spill, *Mar. Mammal Sci.*, 15, 494, 1999.
251. Robineau, D. and Fiquet, P., Cetaceans of Dawhat ad-Dafi and Dawhat al-Musallamiya (Saudi Arabia) one year after the Gulf War oil spill, *Courier Forsch.-Inst. Senckenberg*, 166, 76, 1994.
252. Simons, E.A. and Akin, M., Dead endangered species in a California oil spill, in *Proc. 1987 Oil Spill Conf.*, American Petroleum Institute, Publ. 4452, Washington, D.C., 1987, 417.
253. Albers, P. H. and Gay, M. L., Unweathered and weathered aviation kerosine: Chemical characterization and effects on hatching success of duck eggs, *Bull. Environ. Contam. Toxicol.*, 28, 430, 1982.
254. Wolfe, J. L. and Esher, R. J., Effects of crude oil on swimming behavior and survival in the rice rat, *Environ. Res.*, 26, 486, 1981.
255. Gashev, S. N., Effect of oil spills on the fauna and ecology of small mammals from the Central Ob' region, *Soviet J. Ecol.*, 23, 99, 1992.
256. Savabieasfahani, M., Lochmiller, R. L., and Janz, D. M., Elevated ovarian and thymic cell apoptosis in wild cotton rats inhabiting petrochemical-contaminated terrestrial ecosystems, *J. Toxicol. Environ. Health*, Part A, 57, 521, 1999.
257. Lochmiller, R. L. et al., Disruption of rodent assemblages in disturbed tallgrass prairie ecosystems contaminated with petroleum wastes, in *Environmental Contaminants and Terrestrial Vertebrates: Effects on Populations, Communities, and Ecosystems*, Albers, P. H., Heinz, G. H., and Ohlendorf, H. M., Eds., SETAC Press, Pensacola, FL, 2000, Chap. 13.

258. Lee, J. H. and
and lung tiss
Part A, 56, 4
259. Feuston, M.
Health, 51, 3
260. Khan, A. A. e
Contam. Tox
261. Mattie, D. R.
by oral gava
262. Bowyer, R. T
Exxon Valde
263. Bowyer, R.
marine envir
264. Ray, S. et al.
of St. Lawre
265. Martineau, I
Quebec, Car
266. Mathieu, A.
J. Toxicol. E
267. Marine Man
Mammal Co
268. Paine, R. T.
Syst., 27, 19
269. Wolfe, D. A
1994.
270. Monahan, T
in *Proc. 19*
131.
271. American
Waters, We
272. American I
273. Wiens, J. A
274. Saeed, T., A
of the crud
275. Al-Senafy,
481, 1997.