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THE *EXXON VALDEZ* OIL SPILL: A SYNTHESIS EMPHASIZING LONG-TERM EFFECTS

Final Report
for the
Synthesis Project 00600, 2001-2004

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Prepared for…

The *Exxon Valdez* Oil Spill Trustee Council
441 West 5th Avenue, Suite 501
Anchorage, Alaska 99501

Prepared by:

APPLIED marine SCIENCES
4749 Bennett Drive, Suite L
Livermore, California 94551

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THE *EXXON VALDEZ* OIL SPILL: A SYNTHESIS EMPHASIZING LONG-TERM EFFECTS[a]

Robert B. Spies[1], Stanley D. Rice[2], Mark G. Carls[2], Jeffrey W. Short[2], and Adam Moles[2]

1. Applied Marine Sciences, P.O. Box 315, Little River, CA 95456
2. National Oceanographic and Atmospheric Administration, National Marine Fisheries Service, Auke Bay Laboratory, 11305 Glacier Highway, Auke Bay, Alaska 99801-8626

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4749 Bennett Drive, Suite L
Livermore, California 94551

[a] This synthesis is based on Section V of a forthcoming book on long-term ecological change in the northern Gulf of Alaska. This report is provided to the *Exxon Valdez* Trustee Council as a final report for the synthesis, although the book will be the ultimate result of their sponsorship of the synthesis project. Because of editing and other considerations the contents of the book will likely differ from this report.

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V.A. Introduction

The tanker vessel *Exxon Valdez* ran hard aground on Bligh Reef in Prince William Sound (PWS) on March 24, 1989, spilling 42 million liters of Alaska North Slope crude oil into the northern Gulf of Alaska (Fig. 5.1). Winds and currents carried floating oil hundreds of kilometers from Bligh Reef, inundating much of western PWS, parts of the outer Kenai Coast and Lower Cook Inlet, some beaches on Kodiak Island, and portions of the Alaska Peninsula as it was carried through Shelikof Strait and west into the Gulf of Alaska (Fig. 5.2). The oil eventually washed ashore discontinuously on over 2000 kilometers of shoreline, the equivalent of a spill in New York harbor reaching Virginia.

This was the largest marine oil spill in the United States and the largest spill in a sub-Arctic ecosystem, however, the devastation that accompanied the *Exxon Valdez* oil spill (EVOS) was due as much to when and where it happened as it was to the size of the spill. Although more than 50 previous spills had been larger elsewhere in the world's oceans, the EVOS was more devastating than many larger spills because it could not have occurred at a worse time and affected so much wildlife. As the spilled oil spread, the spring plankton bloom was about to start and Pacific herring were gathering to spawn on PWS beaches. The oil inundated herring, seabird, sea otter and harbor seal habitat just prior to the breeding seasons of many vulnerable species. The oil contaminated the mouths of numerous streams where pink and chum salmon were about to spawn and the nearshore environment where fry from last year's spawning were to feed for their first several months of marine life.

The acute effects from the spill were immediate and obvious, but there was surprising oil persistence and consequential lingering effects. In the first few weeks, the acute effects on wildlife were unprecedented, with hundreds of dead sea otters and tens of thousands of bird carcasses washed ashore, deformed herring larvae developing on beaches, and intertidal communities coated with oil. Despite the massive cleanup in 1989-1991 and subsequent natural weathering, a portion of the original beach-stranded oil, estimated to be about 11.3 ha of oiled substrate in 2001, remains deep in cobble beaches (Short et al. 2004a). This lingering oil has lead to subtle chronic effects that had not been documented in previous spills.

The fate and effects of the spill for the first several years had been summarized and reported in two symposium volumes (Rice et al. 1996; Wells et al. 1995), so we focus here on the longer-term fate and effects of the oil, out to 15 years post-spill. The studies of the *Exxon Valdez* disaster provide us with insights into the effects of a major oil spill. Each spill occurs under unique circumstances, but this spill has many lessons for sub-arctic ecosystems in the future.

Two "armies" of scientist have studied the spill since 1989, those working for the United States and Alaskan governments and those working for the Exxon Corporation. Consequently there are two different interpretations of the spill effects. Profound disagreements between scientists do little to diminish the validity of our general conclusions: 1) oil persisted beyond a decade in surprising amounts and in toxic forms, 2) the residual oil was sufficiently bioavailable to induce chronic biological exposures to

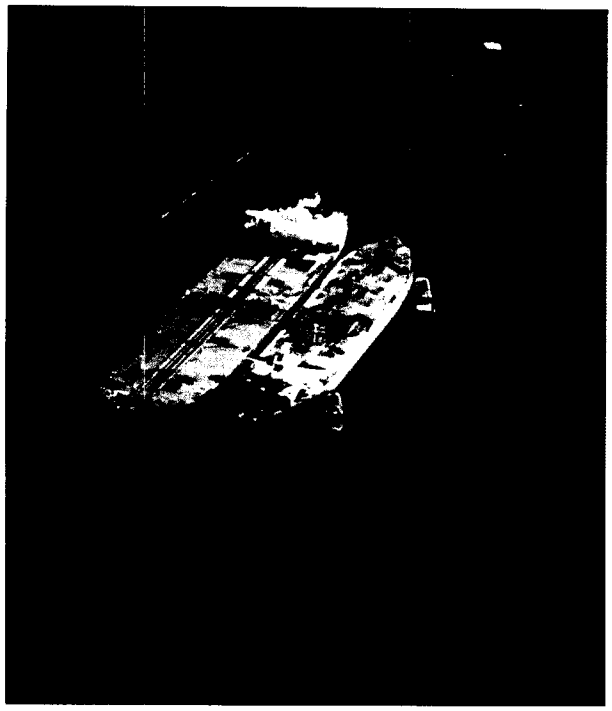


Fig. 5.1. Oil continued to leak from the *Exxon Valdez* as it was transferred to the *Exxon Baton Rouge*.

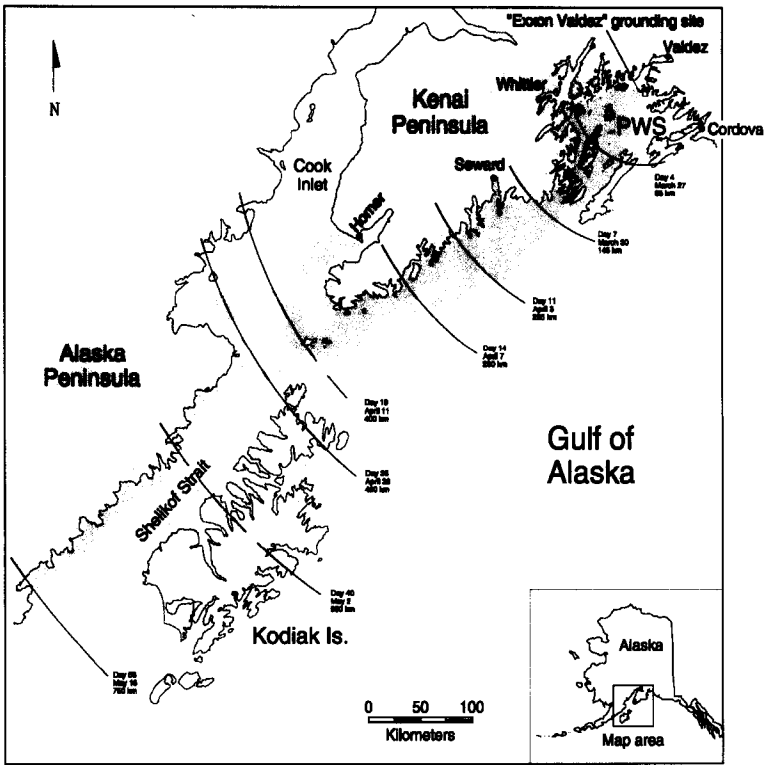


Fig. 5.2. The cumulative extent of the *Exxon Valdez* oil slick, which generally moved southwest across Prince William Sound and along the Kenai and Alaska Peninsulas.

key nearshore species, and 3) oil had both short-term and long-term effects on a wide variety of species, with prolonged effects on species associated with oiled sediments.

The *Exxon Valdez* spill is the most thoroughly studied oil spill in history. This chapter will:

1) Provide a history of events in 1989, set against the background of pre-spill ecological knowledge of Prince William Sound (PWS).
2) Describe the fate of the oil in the months and years following the spill, and how much oil remains.
3) Describe the acute, short-term, and long-term effects of the oil on key species.
4) Discuss the recovery of the ecosystem from spill effects.

V.B. Pre-spill conditions

Prince William Sound and the northern Gulf of Alaska are remote areas with a small coastal human population, and the status of the natural resources in the region, in general, were poorly documented prior to the spill. Some harvested species were tracked to help regulate fisheries, but other species were not followed and nearshore and intertidal communities were not surveyed well enough to know what their populations were in the spring of 1989.

In the mid-1970s, following large spills in England (the *Torrey Canyon*), and the United States (the Platform A blowout in the Santa Barbara Channel), the Arab Oil embargo, and with the growing desire of the United States to achieve energy independence, the Nixon Administration started a national program to evaluate the biological resources on the United States continental shelves in anticipation of increased offshore drilling and oil extraction-- the Outer Continental Shelf Environmental Assessment Program (OCSEAP). The Alaskan portion of the Program, because of its oil and gas potential, was particularly large, with an annual budget of about $20M at the height of the program. This effort resulted in a massive leap forward in scientific understanding of the Gulf of Alaska and made possible the landmark synthesis "The Gulf of Alaska" (Hood and Zimmerman 1986). There was precious little pre-spill ecological data for affected areas and species, but without the results from OCSEAP there would have been much less quantitative data on spill area resources. The boat survey data from the Fish and Wildlife Service were particularly useful for estimating the pre-spill abundances of seabirds and sea otters in PWS and murres along the shallow costal areas and islands west of PWS-all hard hit by the spill.

At about the same time that OCSEAP was underway, pioneering concepts for characterizing contamination in U.S. coastal waters in a mussel watch program were being implemented (Butler 1973; Goldberg et al. 1983; O'Connor 1994). Mussels concentrate many contaminants, including components of crude oil that can affect marine organisms, and their analysis provides an integrated picture of average water quality. Scientists from the Auke Bay laboratory predicted that a spill would be likely in PWS where the primary traffic of oil tankers originates at the oil terminal in Valdez, and initiated several years of pre-spill sampling of mussels and sediments to establish pre-spill chemical baselines (Karinen et al. 1993).

Other useful pre-spill data in Prince William Sound included:

1. Surveys of nesting adult bald eagles carried out by the Fish and Wildlife Service,
2. Pink salmon counts in spawning streams carried out by the Alaska Department of Fish and Game,
3. Herring test fishery results and aerial surveys of herring spawn carried out by the Alaska Department of Fish and Game, and
4. Harbor seal aerial surveys carried out by Alaska Department of Fish and Game.

V.C. History of the spill

The 300 m (986 ft)-long T/V *Exxon Valdez,* laden with 197 million liters (52 million gallons) of Alaska North Slope Crude oil, left the Alyeska Pipeline terminal shortly after 9 PM on March 23, 1989. A pilot guided the ship along the established route southeasterly down the center of upper Valdez Arm. She passed the Valdez Narrows and Captain Joe Hazelwood took command of the vessel. As the vessel entered Lower Valdez arm, the route ahead and to the west was littered with icebergs from the nearby Columbia Glacier. Hazelwood ordered the helmsman to take a more southerly route to avoid the ice and then relinquished the wheelhouse to a junior officer with instructions to return to the shipping lane at a particular point. The ship never did return to the designated route despite the last minute course corrections of a new helmsman, and at 12:04 AM Friday March 24, 1989, Good Friday, the *Exxon Valdez* ran onto Bligh Reef, ripping a large gash in its hull. Eight of eleven oil compartments were ruptured. The pride of the Exxon fleet was immobilized and started hemorrhaging 20 million liters (12 million gallons) of Alaska North Slope (ANS) oil into the near-pristine waters of beautiful PWS, setting the stage for a massive clean up, a large-scale scientific effort and marking a milestone in the environmental consciousness of the American public. Ironically, it was Good Friday 25 years previous that an earthquake of huge proportions rearranged thousands of kilometers of shoreline in PWS and northern Gulf of Alaska and sent a huge tsunami through Port Valdez, PWS, the northern Gulf of Alaska, and beyond.

For three quiescent days following the grounding spilled oil pooled near Bligh Reef, mainly to the southwest. During that time attempts were made to contain the oil, but the proper equipment was not available. Some dispersants were tried, but with little apparent effect without wind mixing. A test burn was even attempted. The highest priority was to offload the remaining oil from the *Exxon Valdez* to prevent an even worse spill. Although approximately 80% of the cargo was successfully removed,the 20% released was still the largest spill in U.S. waters.

Then on Monday March 27th a northerly gale (with 70 knot winds) descended on the Sound and dispersed the oil to the southwest for about 100 kilometers and any hope of containing this disaster went with it. The oil blew along the water's surface and followed the prevailing currents in a roughly counterclockwise direction in PWS. The extensive crenulated coastline of the Sound with its many islands and narrow passages to the Gulf of Alaska in the southwestern sector ensured that much of the oil would be blown onto the predominately cobble and rock beaches of these islands. Northeast-facing beaches were particularly hard hit. Oil piled up a foot or more on some beaches, pushing far up the beaches on high tides. Some of the oil retreated with ebbing tides, but some of it also

followed the dropping water level between the rocks and cobbles and became entrained into the finer material deep within the beach—to persist for many years. In some bays, floating oil continued to rise and fall with the tides for days or weeks before it was left stranded or flushed out to other parts of the sound.

The oil hit parts of the mainland on the western side of the Sound, e.g., Eshamy Bay and Main Bay, and other areas east of Whittier (see Fig. 5.2). Islands in the path of the oil within the Sound included those in the Naked Island complex: Perry Island, Lone Island, Block Island, Ingot Island, Eleanor Island, Smith Island, Knight Island and Green Island. The spill also affected parts of the northern end of Montague Island, but much of that island, greatly affected by the 1964 earthquake, was spared from the oil. The oil heavily inundated the islands defining the southwest passages to the Gulf: Latouche, Erlington, Evans and Bainbridge, as it spread towards the open Gulf of Alaska.

The floating oil was discontinuous as it exited the sound, floating in large linear rafts of oily mousse. On entering the Gulf of Alaska, the slick floated on the buoyant nearshore Alaskan Coastal Current, carrying oil to the outer parts of the southeastern Kenai Peninsula, and then much of it entering Cook Inlet. In Cook Inlet a small amount of oil was carried by strong tides north of Homer, but most of the oil was carried past the Barren Islands and south through Shelikof Strait. Some shorelines on the western side of the Strait along the Alaska Peninsula, e.g., Tonsina Bay, and touched parts of Kodiak Island as well were heavily oiled. Coating of beaches outside the sound was discontinuous and varied from heavy to light or not at all. The remaining floating oil was carried southwesterly out of Shelikof Straight and back into the Gulf of Alaska, where it presumably met the fate of oil left behind by the advancing front: evaporation, photolysis, dissolution, biodegradation, and sinking.

The cleanup operations after the spill occurred over a 3-year period, 1989-1991, and were restricted to the late spring and summer and with a decreasing effort each year. The early clean up was very widespread. In the few weeks after the spill a small but significant proportion of spilled oil was retrieved from the water's surface by skimmers, but most of the cleanup effort that eventually mounted to more than 20 million personnel hours, was expended on the beaches (Fig 5.3). On shorelines oil was picked up with shovels, or manually wiped from rocks, removed by hosing, flushed with seawater, or bioremediated with application of nutrient-laced liquid fertilizers (Mearns, 1996). Heavy equipment was also deployed to expose oil buried in beaches and berms to natural weathering. Deluging with seawater was used extensively to clean rocky beaches. About 600 commercial pressure washers sprayed hot water (usually at a temperature of 60°C) onto the beaches. A large number of moderately to heavily oiled shorelines were sprayed with hot seawater, killing much of the marine intertidal life (Lees et al. 1996). Then in 1997 (Brodersen et al. 1999), under pressure from Native Groups in PWS, a cleanup of Sleepy Bay on the northern end of LaTouche Island was undertaken.

Damage assessment by state and federal scientists was immediately undertaken to determine the spill's effects, especially to animals in the hard-hit southern sound. The task of spill assessment was enormous and scientists had to be diverted from their normal activities, but they rose to the occasion. Agencies with resource management

Fig. 5.3. Oil was removed from shorelines by shovel, manually wiping, hosing with saltwater, or bioremediated with application of nutrient-laced liquid fertilizers. In this case, oil was moved downslope by hot water, trapped within a boom, and picked up with a skimmer.

responsibilities needed to know what the effects were, how extensive and for how long lasting, but the information was also in support of legal action against Exxon Corporation; hence, the studies were "litigation sensitive" (i.e. secret) and the designs and objectives were often influenced by this legal process. There was a rush to put studies into the field in order to document spill damages soon after they occurred. Wildlife biologists with little first hand knowledge of spills had to learn about oil toxicity, behavior, and sampling to support chemical analyses. Immediate field sampling included gathering seabird, bald eagle, and sea otter carcasses; taking samples of mussels, sea water and sediments in the path of the spill; and mapping the full extent of the spill.

This was just the beginning of a massive effort ramping up to at least 40 separate studies by the State and Federal governments in 1989 alone. Agencies with responsibility responded as they saw the need, but with little coordination, peer review, or oversight in the first year. There just was not enough time. The *Exxon Valdez* Trustee Council evolved a process in subsequent years for choosing studies and managing the direction of the restoration science, an interesting story in itself, but beyond the scope of this report. As it became clear what the immediate damage was, these biological studies evolved toward understanding the long-term effects, especially to populations and how to restore damaged portions of the ecosystem. But to track recovery from damages and conduct restoration, the extent of damages had to be known. The paucity of pre-spill data made this a challenging goal. The assumptions made and the approaches taken to estimate the extent of damages and recovery from the spill contributed to the deep disagreements between the governments (State and Federal), Exxon Corporation and its scientists. (See text box).

Dueling Scientists

The controversy over the effects of the *Exxon Valdez* spill is similar to those over cigarette smoking, global warming, or other widely discussed health and environmental issues. Government scientists and policy makers come to different conclusions than corporations and their scientists. The opposing views of the governments and the Exxon Corporation in this case were aired widely in the mass media and in various technical forums and journals. A quick read of the abstracts from 2 scientific meetings held in 1993, a government-sponsored symposium in Anchorage, Alaska (Rice et al. 1996) and an Exxon-sponsored symposium in Atlanta, Georgia (Wells et al. 1995) reveals the stark contrasts in conclusions by the two armies of scientists studying spill damages and recovery. It is puzzling to environmental scientists that were not involved, let alone the public, how these widely differing accounts could arise from one reality. Adding to the puzzlement in the early years was the secrecy demanded by a government suit against Exxon that was settled in 1991.

The Exxon Corporation would benefit from a conservative estimate of damages and a rapid recovery, while the government has a mandate to protect natural resources and not minimize the spill's effects. The public might understand different interpretations by government officials and corporate leaders. But what about the scientists, aren't they supposed to be objective and truthful at all costs? The coincidence of their conclusions with their employer's view might appear to belie their role in society.

There is a less cynical interpretation that rides on assumptions about uncertainties. The assumptions are different in the government and industry camps. They arise in the following way. There is a complex chain of events that leads from spilled oil to its ultimate consequences in altered populations. There are many links in this chain and it is quite challenging to forge an unbroken certainty from start to finish. So, uncertainties arise, for example, about the size of affected populations just before the spill, the dose animals received, how toxic the oil was to certain animals in late winter, and a host of other factors. Government scientists do not demand that all links in the chain be established with certainty, only that there is a weight of evidence to conclude an effect of the spill. Exxon scientists are more conservative and demand that all links in the causal chain be established to conclude an injury occurred.

A good example of the discrepancy of conclusions is the effect of the spill on pink salmon juveniles. There were juveniles with coded wire tags in their noses released from hatcheries in western PWS in the spring of 1989. Some of the juveniles were recovered in the oiled waters around Knight Island and their growth was compared with tagged juveniles captured in unoiled areas (Willette 1996). The juveniles collected in the unoiled areas had grown significantly more than those captured in the oiled areas, and there were similar findings in a second study (Wertheimer and Celewycz 1996). Juvenile salmon captured in the latter study also had induced P4501A, a molecular marker of oil exposure (Carls et al. 1996), as did some pre-emergent fry captured in oil-affected streams (Wiedmer et al. 1996). Combined with a known relationship between rate of growth and survival to adulthood, it was possible to model the effect of reduced growth in the juvenile stage on numbers of returning adults in 1990 (Geiger et al. 1996) and led government trustees to conclude that returns of pink salmon to western PWS in 1990 had been reduced by the spill (EVOS Trustee Council 1994). Exxon-sponsored scientists disagreed, as there was no record of where the juveniles were between release from the hatchery and collection in the oiled waters, and therefore, rejected a link between the oil, slower growth in the juveniles, and adult returns (Brannon and Maki 1996).

There are other factors that lead to different conclusions, such as differing study designs; many of the government studies were substantially more robust and were more sensitive in detecting differences between oiled areas and non-oiled areas (Peterson et al. 2001). Government studies also focused commonly on the most affected areas, while Exxon scientists sampled areas less likely to be affected in greater proportions in their sampling designs, such as deep offshore areas.

The definition of recovery usually differed between the two sides. Exxon maintained that The system had recovered if healthy organisms still remained and that the population sizes merged back into the wide range of historical fluctuation. The governments maintained that a return to estimated pre-spill population sizes with a similar age composition was necessary for recovery.

V.D. Oil Fate: Transport, Weathering, and Persistence

Only early phases of transport and transformation of the petroleum hydrocarbons followed expectations. Many of the effects of the oil spill were due to the way the oil behaved in the months and years following the spill. The oil was transported, transformed, and detained in ways that defied all expectations. In order to fully appreciate how and why the petroleum hydrocarbons (the compounds that make up oil) altered the ecology of PWS, it is important to examine the types of hydrocarbons present in the sound prior to the spill, the initial transport of the oil into the environment, the effect of cleanup efforts (both natural and man-made), the transformation over time of the hydrocarbons in the oil, and the long-term persistence of some toxic compounds in the environment. Finally, we will examine how available these residual toxic hydrocarbons were to the animals of the sound.

BTEX and PAH: Petroleum's Toxic Culprits

Oil and petroleum products are made up of petroleum hydrocarbons and are the major sources of an important class of toxic chemicals known as aromatic hydrocarbons. Hydrocarbons are chemicals containing only carbon and hydrogen. The carbon atoms in aromatic hydrocarbons are joined together to form rings and are bonded to fewer hydrogen atoms than they could be (i.e. they are *unsaturated*). To be aromatic, the carbon rings must have clouds of electrons above and below them that contain a particular number of electrons (i.e. 6, 10, 14,...), in addition to the electrons between the carbon atoms that bind them together. These de-localized electrons add bonding strength, making aromatic hydrocarbons much more stable (and hence persistent in the environment) than saturated hydrocarbons, and they are also the cause of the toxicity of these molecules.

The simplest aromatic hydrocarbon is benzene, which contains six carbon atoms and six hydrogen atoms (Fig 5.4). Chemists use a kind of shorthand to depict these molecules, where two lines meeting at an angle indicate a carbon atom, a circle within a hexagonal ring of benzene stands for the six de-localized electrons, and the hydrogen atoms at the edge of the ring are not shown because there can only be one for each carbon atom.

The *alkanes* are another important class of hydrocarbons in petroleum (Fig. 5.4), and consist of carbon skeletons that are bonded to the maximum number of hydrogen atoms possible (i.e. they are *saturated*). Simple alkanes include methane, ethane, propane and two kinds of butane (see structures). When one of the hydrogen atoms of these compounds is replaced by something else, the name of the remaining hydrocarbon part is modified by replacing the –ane with –yl, as in methyl, ethyl, propyl, etc. (see structures), which are referred to generally as alkyl groups.

Any one of the hydrogen atoms on the benzene ring may be replaced by an alkyl group, which may contain any number of carbon atoms. Addition of a methyl group (one carbon) produces toluene (see structure); two methyl groups produces xylene (see structures), and there are other combinations of one or more groups that each could contain one to several carbons atoms. Note there are three different kinds of xylene,

depending on the geometric arrangement of the two methyl substituents. These six monocyclic aromatic hydrocarbons are often abbreviated as "BTEX"(benzene, toluene, ethyl benzene and toluene), and they constitute about 1.6% of fresh Alaska North Slope (ANS) crude oil, the type spilled in PWS. They are very volatile and pose a serious inhalation hazard during oil spills. Benzene in particular is a potent carcinogen.

More complicated monocyclic aromatic hydrocarbons may be formed through more alkyl substitution (Fig. 5.4). Note that as the number and complexity of these added hydrocarbons increases, so does the number of structurally distinct compounds, giving rise to rapidly increasing numbers of *isomers* (compounds with the same numbers of atoms of each element that are bonded together differently).

Polycyclic aromatic hydrocarbons (PAH) are formed when two or more aromatic rings (each containing six carbons) are joined together (Fig. 5.4). The compound biphenyl results when two benzene rings replace a hydrogen atom on each other (Fig. 5.4), while naphthalene results when two benzene rings fuse (Fig. 5.4). As with the benzene compounds, the number of PAH isomers increases rapidly with the number and complexity of saturated hydrocarbons that replace the hydrogen atoms. Hence, methylnaphthalene has two distinct isomers, dimethylnaphthalene has ten, and trimethylnaphthalene has 11. Even more isomers result when the substituting groups are different (e.g. methylethylnapthalene with 14). Environmental chemists usually combine results for these PAH isomers according to the number of carbon atoms in the substituting groups, for example the ten dimethylnaphthalene are lumped together under the label "C2-naphthalenes", where the "C2-" denotes the substituting alkyl groups include two carbon atoms (i.e. two methyl groups or one ethyl group; "C3-naphthalenes" includes the 11 trimethylnaphthalenes and the 14 methylethylnaphthalenes). The presentation of the PAH composition of ANS crude oil follows this convention in Appendix B.

Other important PAH in ANS oil include fluorene and alkyl-substituted fluorenes, the phenanthrenes, pyrenes, chrysenes, and other 4- and 5-ring PAH (Fig. 5.4).

The de-localized electrons cause PAH to be toxic in two fundamental ways. First, they are capable of reacting with biomolecules and thereby disrupting their function (whereas alkane hydrocarbons are virtually inert except when burned). For example, PAH may be transformed by cells into forms that react with DNA and RNA, interfering with cell division and with protein production. Second, certain PAH may catalyze the formation of a highly reactive form of molecular oxygen within cells when exposed to ultraviolet radiation (called photoinduced toxicity). Photoinduced toxicity may occur at part-per-billion (ppb) PAH concentrations within affected cells, and translucent biota exposed to intense sunlight are at greatest risk.

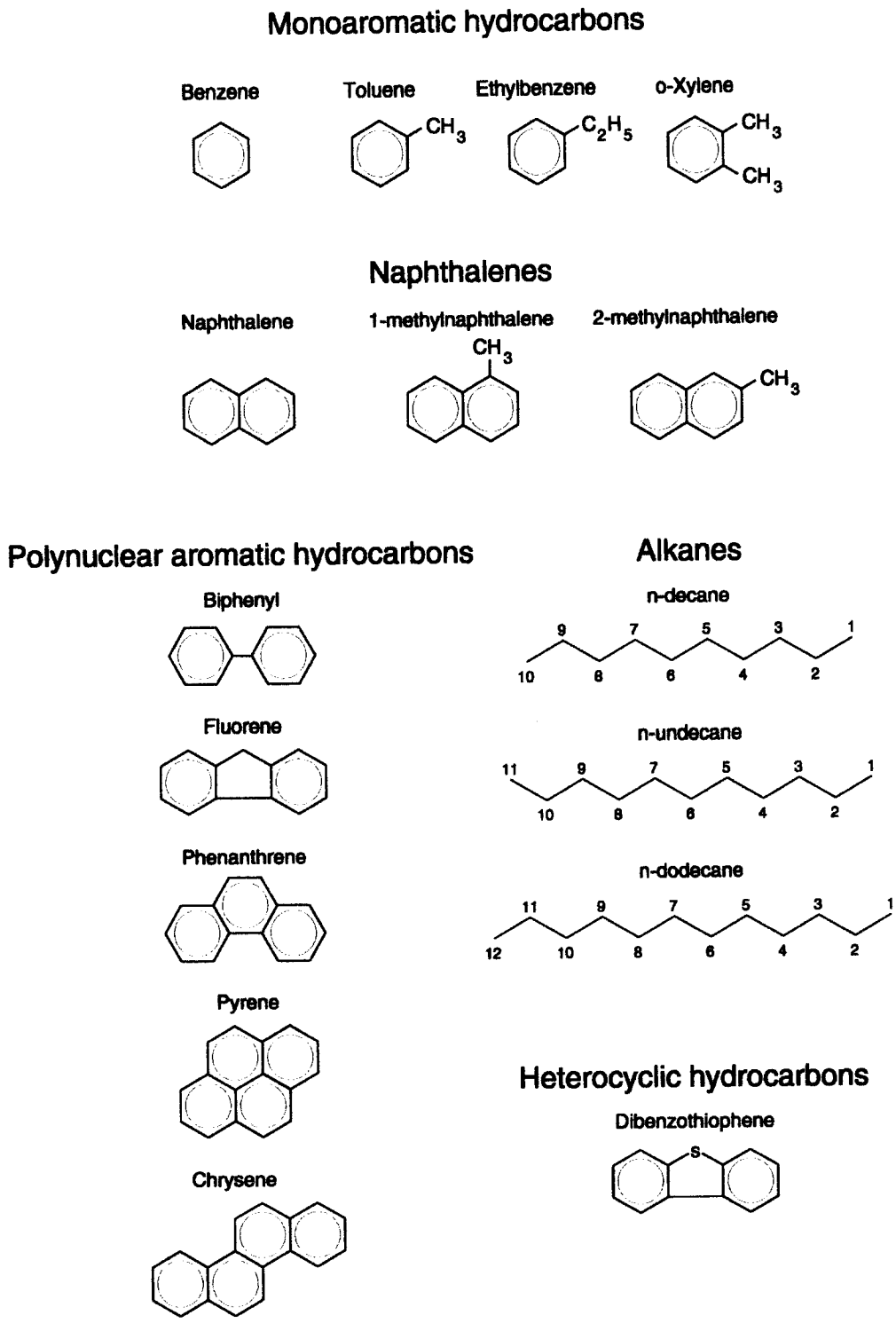


Fig. 5.4. Structure examples of aromatic, heterocyclic, and alkane hydrocarbons present in *Exxon Valdez* crude oil.

V.D.1 Contaminants in Prince William Sound Prior to the *Exxon Valdez* Oil Spill

Most of the region affected by the *Exxon Valdez* oil spill was pristine wilderness. About 10,000 people lived in PWS in 1989, almost all of them in Cordova, Valdez and Whittier. Less than two hundred people resided within the spill trajectory inside the Sound, mainly Alaska Natives in the village of Chenega, and residents of a fish hatchery at Sawmill Bay. Few people lived along the coasts of the Kenai and Alaska Peninsulas and Kodiak and adjacent islands, except for the ~8000 residents in the city of Kodiak. The primary industry in the spill region was commercial fishing, which introduced negligible contamination to shoreline sediments and biota. Small-scale mining and land-based fish processing were important industries historically within PWS, but these were few and scattered (Lethcoe and Lethcoe 1994). Contaminant effects of these activities on shorelines were attenuated by the 1964 Great Alaska Earthquake, which uplifted most shorelines within the spill trajectory from 1 m – 10 m into the supratidal (the zone immediately above the highest reach of the tides). Although contaminants from these human activities were occasionally found in inter- and shallow subtidal sediments, they were quite localized, affecting a very small fraction of the shoreline (Karinen et al. 1993).

Prior to the spill, the most likely sources of hydrocarbons on shorelines and shallow subtidal sediments within the spill region were asphalt from storage tanks in Valdez (and perhaps Whittier) that ruptured during the 1964 earthquake (Kvenvolden et al. 1995), and a natural "regional background" of hydrocarbons from eroded organic-rich shales and siltstones east of PWS. The high viscosity of the asphalt (>10,000 centipoise) prevented it from penetrating into subsurface intertidal sediments, so patches became stranded by high tides on surface rocks. Small asphalt patches may still be found firmly adhered to cobbles, boulders and bedrock above +3 m tidal elevation, but they were a small amount (<3%) relative to the *Exxon Valdez* oil remaining in PWS by 2001 (Short et al. 2004a).

Natural oil seeps were proposed as the source of natural hydrocarbons found throughout the shelf sediments of the northern Gulf of Alaska (Bence et al. 1996; Page et al. 1995), but appear now to be negligible sources, with eroded shelf rock being the major source (Short et al. 2004b). These hydrocarbons are not bioavailable because they are sequestered within coal or rock matrixes (Short et al. 2004b). The hydrocarbon "source rocks" are eroded by streams and by glaciers from outcrops of the Kulthieth and Poul Creek Formations along the southern coast of the Gulf of Alaska from Katalla to Yakutat Bay (Van Kooten et al. 2002). Finely-eroded sediments become entrained by the Alaska coastal current and transported to PWS and westward, where they settle on subtidal sediments. Concentrations of source rock PAH increase with depth, ranging from less than ~100 ng g^{-1} dry sediment in the intertidal of the spill-affected region to ~1,500 ng g^{-1} in benthic sediments of the deepest parts of the Sound (O'Clair et al. 1996; Page et al. 1995).

Deposition of PAH from forest fires on the Kenai Peninsula has also been reported (Page et al. 1999), but hydrocarbon signatures indicative of combustion sources are at trace levels except near former or present human habitation sites (Carls et al. 2004a).

V.D.2 Initial Fate of the Oil

The overall fate of the oil spilled from the *Exxon Valdez* was largely determined by the weather during the first few weeks. Oil discharge from the grounded vessel began spilling shortly after midnight on Friday, March 24, 1989 during a period of light winds and calm seas, with seawater temperatures at about 5°C. Winds were variable and below about 5 m sec^{-1} for the next 2.5 days, and the oil slick was a compact and roughly circular pool southwest of Bligh Island. During this period the oil slick spread at rates up to 2000 m^2 sec^{-1} to an area of nearly 400 km^2, with thicknesses that decreased from generally less than 1 mm after the first few hours to perhaps 0.1 mm by Sunday. The enormous increase in the surface area of the oil accelerated evaporation of the most volatile components, leading to losses of perhaps 15% by weight, including nearly all of the benzene, toluene, ethylbenzene and xylenes (BTEX), and of the saturated hydrocarbons with vapor pressures greater than that of dodecane (Payne et al. 1991). A chemical dispersant (Corexit 9527) was applied to a small portion of the slick on Friday, but it was ineffective as the sea was dead calm and the slick was too thick. A test burn of about 100 m^3 of oil was successful on Saturday, at slick thicknesses of 1–3 mm, demonstrating that evaporation rates were sufficient to permit ignition 40 hrs after the spill. A larger burn was planned for the following day, but was precluded by a storm entering the Gulf of Alaska.

Oil slicks on the water surface were distributed by wind and currents in the weeks and months following the spill (Fig. 5.5). Northeast winds up to 35 m sec^{-1} disrupted the slick and drove it toward beaches during the next three days beginning Sunday afternoon March 26th. The first landfall of the oil slick occurred Monday morning with breaking waves of 1-3 m on beaches of Naked Island and nearby islands. The oil continued to move along the east and west coasts of these islands towards Smith and Eleanor Islands, and onward toward the southwest along the coasts of Knight Island and the surrounding islands. Beaches on a complex of islands near the southwestern margin of PWS, including Latouche, Evans, and Bainbridge Islands, were affected by the oil on the last day of the storm (Wednesday, March 29th). The leading edge of the slick exited the Sound the following day as seas and winds diminished.

The storm dispersed the slick as small oil droplets in the water, and also promoted water incorporation into the oil. Oil droplet dispersion occurred at least to depths of 25 m (Short & Harris 1996), and probably considerably deeper because the water column in PWS is not stratified in late winter (Vaughan et al. 2001). The numerous small oil droplets allowed the more soluble oil components to dissolve in seawater, leaving PAH concentrations of parts per billion (ppb) weeks after the storm had passed (Short and Harris 1996). Water incorporation into the surface slick along with continued evaporative weathering lead to the formation of a "mousse" with viscosities ranging from several hundred to a few thousand centipoise (Payne et al. 1991; Bragg and Yang 1995).

The high winds thickened the slick as it approached beaches, and the breaking waves caused substantial accumulations of oil to be stranded on beaches during falling tides, blanketing the ~ 4 m vertical tidal excursion with oil (Fig 5.6). On porous beaches, pools of stranded oil percolated into subsurface sediments as the water table lowered on

Fig. 5.5. Oil transported as surface slicks drifted along coastlines. Critical areas were sometimes protected with floating booms; in this case the boom did not prevent all oil from entering the cove.

Fig. 5.6. An oil-coated beach in Snug Harbor, Prince William Sound.

the ebb tide. Even the fine-grained sediments of these beaches are very permeable (Carls et al. 2003; Bragg and Yang 1995), so the water table in the beach follows the tide closely, permitting the more viscous oil several hours to seep into the underlying sediments, to depths of 1 m in some locations (Neff et al. 1995). Subsequent rising tides failed to completely remove this subsurface oil because the increased oil viscosity inhibited displacement from finer-grained sediments. Fine-grained sediments often lay beneath coarser sediments on PWS beaches because much of the Sound affected by the spill was uplifted by the 1964 Alaska earthquake, which elevated fine-grained subtidal sediments into the intertidal zone that were subsequently buried by rock transported from the new upper intertidal to the lower intertidal by wave action. This initial percolation and retention of oil into subsurface sediments set the stage for long-term persistence.

For weeks following the storm, the oil in PWS was redistributed by wind, tide and currents, while the oil that escaped the sound became entrained in the Alaska Coastal Current and was carried southwest along the Kenai Peninsula. About 16,500 m^3 of oil ultimately beached within PWS, contaminating patches along 783 km of shoreline, compared with about 3,700 m^3 of oil that contaminated patches of beaches outside the Sound along another 1300 km of shoreline (Wolfe et al. 1994; ADNR 1991; Neff et al. 1995). The redistribution of oil within the Sound occurred as successive tides re-floated stranded oil in the intertidal and transported it to other beaches or exported it to the Gulf of Alaska during the ensuing months (Fig. 5.7). The viscosity of the oil continued to increase with weathering (mostly evaporative), and when the oil became too viscous to penetrate into beaches it formed surface pavements and tar mats that were often extensive, covering several hundred m^2 at thicknesses of up to several centimeters. These surface oil deposits were usually located in the upper intertidal zone after initial stranding at high tides, in contrast to the subsurface oil, which was most frequent at the mid-tide elevations (Short et al. 2004a).

Oil escaping into the Gulf of Alaska also became more viscous, but this oil still penetrated into subsurface sediments at some beaches (Irvine et al. 1999). Boulder-cobble beaches are more common on the outer coast of the Gulf because of the frequent large waves, and the greater porosity of these beaches permits penetration of more viscous oil. Surface oil deposits also formed on outer coast beaches, where the beach porosity did not allow oil penetration.

By the end of May 1989, the oil slick had transited Shelikof Strait and entered the southwestern margin of the Gulf of Alaska as scattered patches. Tarballs were reported near Chignik (Neff et al. 1995), with perhaps 30% of the oil ultimately dispersing into the Gulf as tarballs that slowly degraded from physical and biological processes (Wolfe et al. 1994). These processes include continued evaporation and dissolution of the more labile components, photo-oxidation, and microbial degradation. About 20% of the dispersed oil (i.e. ~3,000 m^3) ultimately became associated with enough sediment to sink to the seafloor of the Gulf (including PWS), while the remainder joined the tarball population of the northern Pacific Ocean. About half the oil discharge volume (~20,500 m^3) remained on the beaches of PWS and the Gulf of Alaska, becoming the target of unprecedented human efforts to remove it.

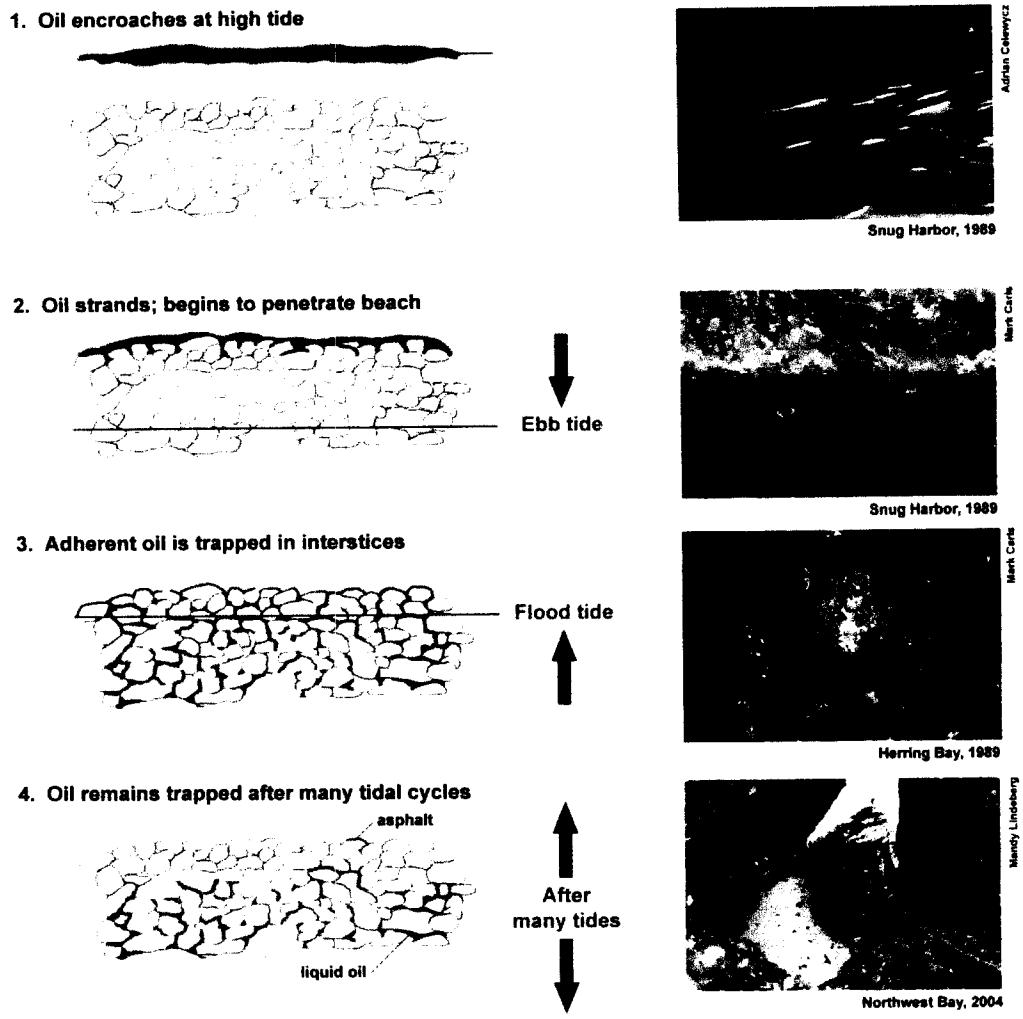


Fig. 5.7. Oil retention in intertidal beaches. Oil slicks encroached at high tide; stranded oil penetrated the highly porous sediment during the hours of ebb tide and became trapped in interstices. Subsequent tidal cycles removed oil slowly; liquid oil is still present in some beaches (2004) as illustrated in the fourth photograph.

V.D.3 Oil Clean Up Efforts

Exxon Corporation sponsored the most extensive shoreline cleanup ever attempted. Involving more than 10,000 people and $2 billion dollars, this clean-up used several methods over the first three years. During the summer of 1989, manual, hydraulic, and bioremediation methods were used on 396, 486 and 292 of 1060 beach segments, respectively. Usually the more heavily oiled beaches were treated by multiple methods (Mearns, 1996). Successively smaller clean ups were carried out during 1990 and 1991.

The most efficient cleanup method was collection of oil floating on the seasurface by skimming operations (Fig. 5.8). About 2900 - 3500 m^3 of the original spill volume was collected by oil skimming vessels within PWS, primarily during the first month (Wolfe et al. 1994). The amount of oil recovered by surface skimming was comparable with the amount recovered from oiled beaches over the next three years, which accounted for most of the cleanup expense.

Manual methods involved removing surface tar mats, oiled debris and oiled vegetation by hand, application and recovery of oil absorbance pads, and shoveling thick lenses of oil. These methods were sometimes complemented by use of oil absorbent materials attached to cables set parallel to the shoreline ("snare booms") that moved over portions of beaches driven by tidal movement. Mechanical tilling on 54 beaches exposed subsurface oil to more rapid weathering through dispersion.

The hydraulic methods were more controversial because of their greater effect on beaches. High pressure washing (usually 345 – 827 kPa), especially by hot water (approx. 60 °C), had traumatic effects on intertidal life and it was discontinued after 1989 (Fig.5.9). These methods were often coupled with high-volume flushing, wherein seawater was sprayed onto the upper intertidal, flushing the surface of the treated beach. These methods also removed fine-grained sediments, which inhibited re-colonization of some species for a decade or more (Driskell et al. 1996; Houghton et al. 1996; Lees et al. 1996). Some of the oil-laden fine-grained sediments transported oil to the shallow subtidal (Short and Babcock 1996). The higher temperatures may have sufficiently reduced the viscosity of oil promoting additional penetration into subsurface sediments on some beaches.

Bioremediation usually consisted of an application of nutrients (mainly nitrate and phosphate) in a water or lipid-soluble carrier to promote microbial decomposition of oil. Very limited addition of microbes and enzymes tailored for oil decomposition was employed on an experimental basis (Fig. 5.10). These methods often accelerated removal of surficial oil, but were less effective at removing subsurface oil. Chemical removal agents were tested only during 1989, but were not approved for widespread use. Bioremediation and manual removal methods were continued in 1990 and in 1991 on beaches where oil appeared to persist. To accelerate weathering and dispersion, a berm relocation technique was used to expose oil in storm berms in the upper intertidal zone. Hydraulic spot-washing was used on 58 beach segments in 1990 and to one segment in 1991.

Fig. 5.8. Collection of floating oil by skimming was an efficient cleanup method.

Fig. 5.9. High-pressure beach washing damaged intertidal organisms, particularly when hot water was used.

Fig. 5.10. Bioremediation, that is, the promotion of microbial decomposition of oil by application of nutrients, often accelerated removal of surface oil but was less effective at removing subsurface oil.

By the end of 1992, 5 – 8% (2040 – 4080 m^3) of the initial volume of oil spilled had been recovered (Wolfe et al. 1994). Beach cleaning also promoted dispersal of fine-grained oiled sediments to adjacent waters, which were ultimately deposited on bottom sediments over a broad area of the coastal shelf of the northern and western Gulf of Alaska.

Beach cleaning is controversial. Some methods effectively remove surface oil, but they are also destructive to the beach fauna. The long-term persistence of oil, which was not appreciated in 1989-91 to the extent it is today, adds another layer of uncertainty to judgments of how aggressive to be on beaches. Aggressive beach cleaning may be very harmful to intertidal fauna, but may be the best course of action for the protection of vertebrate predators like birds and sea otters. We do not know how to strike a balance between not treating to preserve intertidal fauna and cleaning to lessen exposure of nearshore predators.

V.D.4 Long Term Oil Persistence

Early surveys of oil persistence (1989-1993) indicated rapid dispersion

The massive beach cleanup effort required comprehensive monitoring to allocate clean-up resources and to evaluate efficacy. The initial location and extent of shorelines oiled by the spill was documented by low-altitude, low-airspeed color videotaping (Teal 1991). This provided the basis for defining the general area to be evaluated and monitored by ground-based "shoreline cleanup assessment teams" (SCAT). These teams included at minimum a geomorphologist, an ecologist and an archaeologist, who comprehensively inspected the entire shoreline within the potential affected region during beach walks or from boats when beach access was impractical (Neff et al. 1995). The SCATs received similar training and employed uniform criteria and forms for recording observations (Owens 1999). Collation of these observations provided the basis for segregating the shoreline into contiguous series of beach segments up to 2.5 km long that were bounded by readily identifiable landmarks in the field. Visual assessment of oiling intensity on each of these segments was carefully and consistently documented during the spring and summers of 1989 through 1992 (Fig. 5.11). Subsurface oil was also monitored after 1989 by excavation of thousands of pits to assess persistence.

The SCAT survey results showed oiled beaches within PWS recovered rapidly, but corresponding results for oiled beaches outside the Sound have not been reported. The cumulative length of visibly oiled beach segments inside PWS decreased from 783 km in 1989 to 10 km by 1992. The cumulative area of oiled beach was substantially less than what might be inferred from these results because the beaches were often not entirely coated by oil, especially after 1989. Very approximate estimates of oiled beach area may be calculated from the oiled shoreline lengths and percentages of intertidal area covered by oil given by Neff et al. (1995), which indicate a decline from 240 ha in 1989 to about 20 ha by 1990, and less than 1 ha by 1992. A total of 2.7 ha of oiled beach area was reported from a SCAT survey conducted in 1993 (Gibeaut and Piper 1998). Subsurface oil appeared to decline more slowly than surface oil. The surface area of beaches contaminated by moderate to very heavy subsurface oiling exceeded 5.0 ha in 1991,

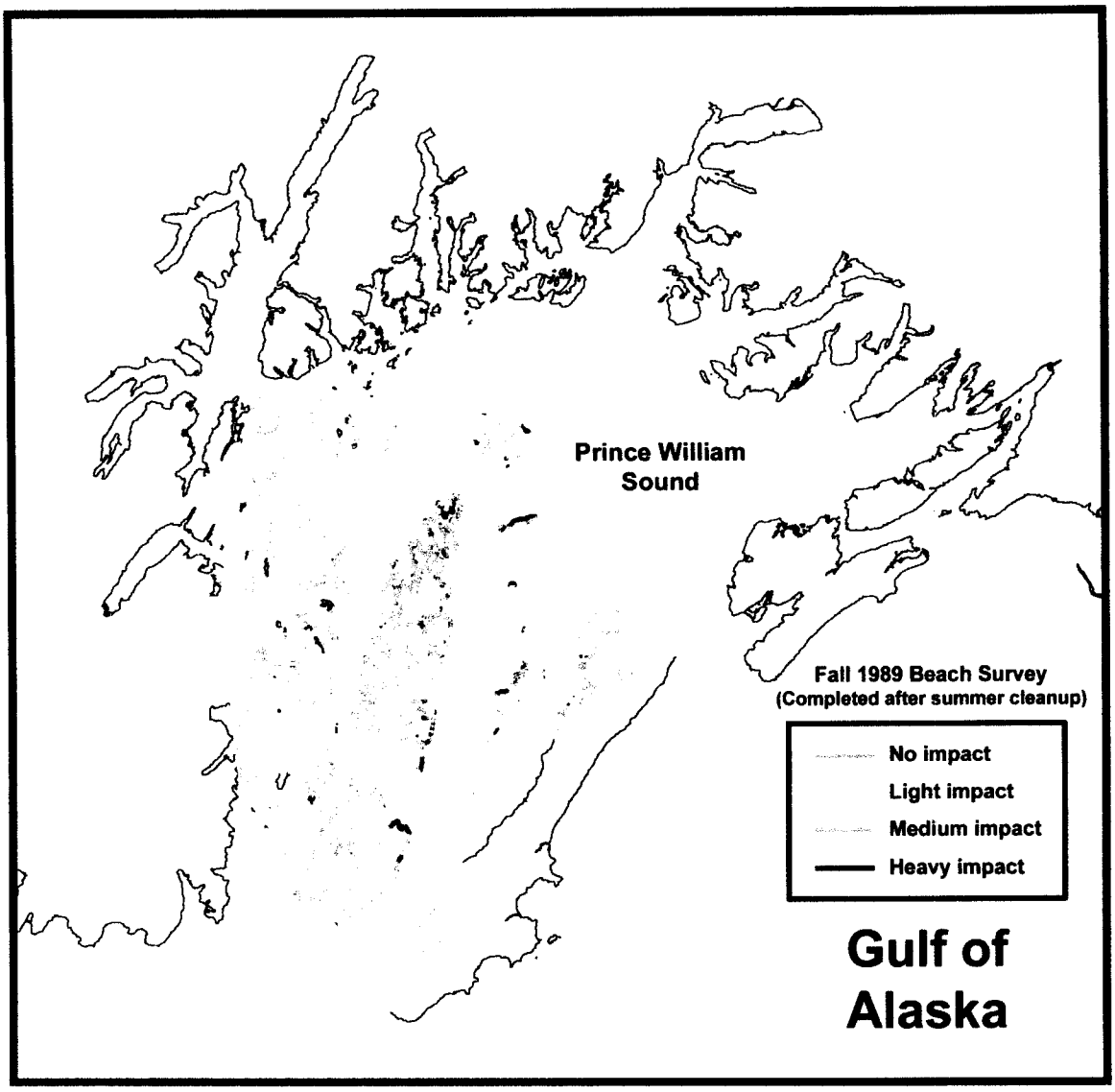


Fig. 5.11. Visual assessment of shoreline oiling, completed in fall 1989 by the Alaska Department of Environmental Conservation, after the first summer's cleanup efforts. (Gundlach et al. 1990).

and decreased by nearly 70% from 1991 to 1992 on beaches surveyed both years, but the total extent of subsurface oiling was not reported for either year (Neff et al. 1995). In 1993, the total beach area contaminated by subsurface oil was estimated as 3.4 ha, and this oil was suspected to be more firmly associated with the sediments and, hence, less easily dispersed compared with prior years (Hayes et al. 1991; Gibeaut and Piper 1998). The amount of oil remaining on PWS beaches in 1992 was estimated to be about 2% of the volume spilled, or about 817 m^3 (Wolfe et al. 1994).

Dispersion of oil from heavily affected shorelines to adjacent subtidal sediments did not result in appreciable accumulations of subtidal oil. Although fluxes of oil were substantial to the shallow subtidal the first year following the spill, these did not usually lead to accumulations because the subtidal sediments are so dynamic, leading to continued dispersion after oil deposition (Short et al. 1996). Some of the oil formed clay-oil flocs of near neutral buoyancy, promoting dispersion over a wide area of the northern Gulf of Alaska (Bragg and Yang 1995). Oiled sediments did accumulate in the shallow subtidal adjacent to heavily oiled beaches when trapped in basins. For example, at Northwest Bay, a terminal submarine sill at the bay entrance allowed oiled sediments to collect within the bay. Sediment PAH concentrations on the order of 1000 ppb that decreased with water depth occurred in 1990, but these declined rapidly during succeeding years (O'Clair et al. 1996). A resurvey of subtidal sediments during 2001 failed to detect hydrocarbons from the *Exxon Valdez* near any of five beaches that had been heavily oiled (Short et al. 2003).

The rapid dispersion of oil from the beaches of PWS indicated by the SCAT survey results implied that the remaining oil would soon be negligible after 1993 (Boehm et al. 1995). It was suggested that clay-oil floc formation would lead to rapid natural removal of subsurface oil (Bragg and Yang 1995), although the low concentrations of clay-sized sediments in the surface seawaters of PWS ($< \sim 1$ ppm) likely constrained the significance of this process (Payne et al. 1989, 2003). Comparison of the volume of oil remaining on these beaches in fall 1992 with the estimated volume that originally beached in 1989 ($\sim$ 42% of the spill volume, or $\sim$ 17,200 m^3) (Wolfe et al. 1994), and assuming first-order dispersion kinetics during the 3.5 yr interval, leads to an instantaneous dispersion rate of -0.87 yr^{-1}, or disappearance of 58% of the oil remaining over a year. Extrapolation of this dispersion rate leads to a prediction of about 60 m^3 of oil remaining after three more years of dispersion (i.e. fall 1995). Unfortunately such predictions were overly optimistic.

Surveys after 1993 changed perceptions on persistence

Studies conducted after 1993 supported the conjecture of Gibeaut and Piper (1998) that the remaining oil would be less easily dispersed by natural forces. Based on repeated surveys of fixed sampling sites from 1989 through 1997, Hayes and Michel (1998,1999) found substantial and relatively un-weathered subsurface oil at 8 of 11 stations in 1997, at sediment depths of 25 – 50 cm beneath cobble/boulder armor on coarse-grained gravel beaches in PWS. Irvine et al. (1999) found subsurface oil at five stations on the Alaska Peninsula that were sampled in 1994, at beaches that were more exposed and were more heavily armored by boulders. The PAH composition of this oil had changed little since its initial deposition. Remnant oil was also found to persist beneath mussel beds in PWS

through 1999 (Carls et al. 2001, 2004b). Oil remained so persistent at Sleepy Bay on northern LaTouche Island that another clean-up effort was made in 1997, and substantial subsurface oil deposits were found there (Brodersen et al. 1999). So, there was a surprising persistence of oil in beaches, and this realization prompted a major effort to estimate the true amount of oil remaining and the extent of beach area affected during summer 2001 inside PWS.

New methods were used for the 2001 estimate of oil in PWS (Short et al. 2004a) that were fundamentally different than those used earlier. The 2001 study employed stratified-random sampling instead of attempting a comprehensive assessment. Random sampling was used to select beaches among the heavily and moderately oiled beaches previously identified for evaluation and to locate sampling quadrats on the beaches selected. This approach has the advantage of providing a confidence interval (an estimate of the error) for the quantities estimated, at the disadvantage of not locating all of the remaining oil. The sampling was stratified according to 0.5 m tidal elevation intervals in the upper half of the intertidal. The lower intertidal zone was not sampled because the prior SCAT studies indicated the persistent oil was almost entirely in the upper intertidal (Neff et al. 1995, Gibeaut and Piper 1998).

The results from the 2001 study confirmed the oil was more persistent than predicted. The cumulative areas of beach contaminated by surface and subsurface oil were 4.13 ha (95% confidence interval: 2.07 – 7.17 ha) and 7.80 ha (95% CI: 4.06 – 12.7 ha) respectively (Short et al. 2004a). These estimates of remaining oiled areas exceeded those based on the SCAT surveys of 1992 or 1993 (Neff et al. 1995; Gibeaut and Piper 1998). The distribution of surface oil was largely disjointed from the subsurface oil distribution, with surface oil found throughout the upper intertidal, but subsurface oil most frequently found near the mid-intertidal zone (Fig. 5.12). Occasional sampling confirmed that subsurface oil occurrences extended into the more biologically productive lower intertidal, nearly to 0 m tidal elevation. The amount of subsurface oil estimated to remain in the upper intertidal in 2001 was 58 m^3 (95% CI: 27.5 – 99.4 m^3, assuming a density of 0.95 for weathered oil (Payne et al. 1991). The total amount of oil remaining was probably about twice this amount, after accounting for surface oil and for subsurface oil in the lower intertidal, which was excluded from the quantitative estimate. Hence, the amount of oil remaining on PWS shorelines in 2001 was around twice the amount predicted to remain by 1995, based on results from the SCAT surveys in the early 1990s.

The discrepancy between the 2001 survey results and expectations based on the earlier SCAT surveys is not only because the oil was more persistent than anticipated. The SCAT surveys systematically underestimated the extent and amount of oiling, with biases that increased annually during the early 1990s. The SCAT survey efforts were focused on the upper intertidal, where oil was incorrectly assumed to be most persistent (Jahns et al. 1991; Neff et al. 1995; Gibeaut and Piper 1998; Hayes and Michel 1999). Because the upper intertidal zone was drier, it was thought to be conducive to increased oil adhesion, so subsurface oil in the middle and lower intertidal zone were often overlooked (Owens 1991). Surface oil was also less visually apparent after 1989 because of surface weathering, and these biases contributed to the overly optimistic predictions of rapid dispersion in the early 1990's.

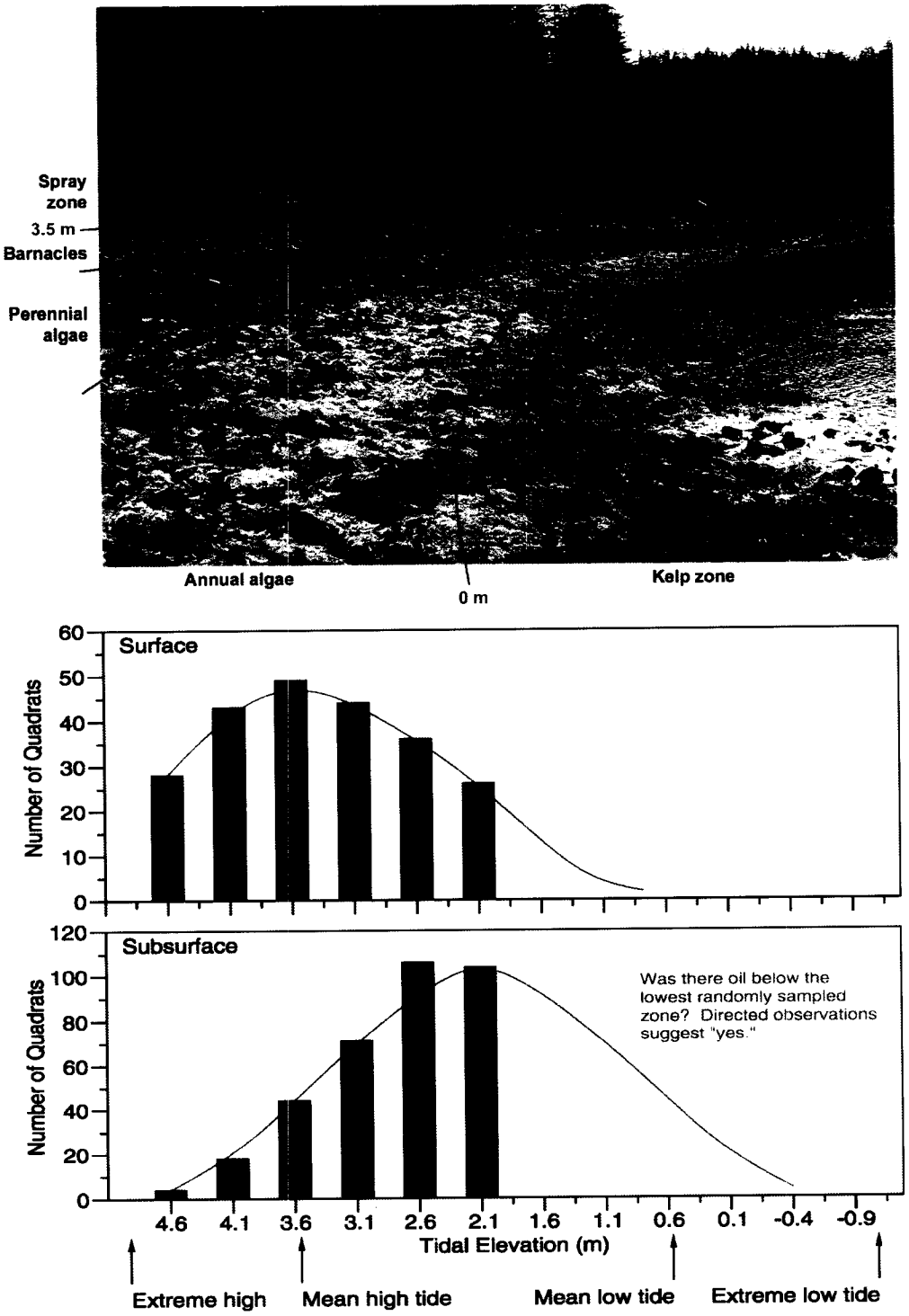


Fig. 5.12. Twelve years after the spill, the distribution of surface and buried oil in beaches was disjointed; surface oil was found throughout the upper intertidal but subsurface oil was most frequently found near the mid-intertidal zone.

The 2001 survey results imply that oil was much more abundant on PWS shorelines than anticipated during the decade following the spill, with commensurate increase in risk of exposure to fish and wildlife. The estimated volume of oil that initially affected beaches (~16,500 m^3) is based on the initial discharge volume and predictions of the NOAA On-Scene Spill Model (Galt 1991a, 1991b), which accounts for evaporation, oil that escaped to the Gulf of Alaska, and dispersion, and may therefore be regarded as relatively precise. About 80% of the oil was estimated to have dispersed from shorelines during the first year (Michel et al. 1991), and this seems plausible considering the intensive efforts to remove it during the summer of 1989, the high energy of waves associated with winter storms, and the fact that much of the oil was only loosely associated with these beaches. Assuming 3,300 m^3 of oil remained on shorelines by 1990 and first-order exponential dispersion thereafter to ~100 m^3 of oil by 2001 (accounting for surface oil and oil in the lower-intertidal) leads to an instantaneous dispersion rate of -0.32 yr^{-1}. At this rate, about 1,750 m^3 of oil would have remained by 1992, and 670 m^3 by 1995, in contrast to the ~60 m^3 anticipated by 1995 on the basis of the SCAT surveys of 1990-1992 (Fig. 5.13). Hence, oil was likely more abundant than anticipated by factors of ten or more through the mid- to late-1990s, and the subsurface proportion was often well preserved, with a complement of toxic PAH that was only slightly smaller than what was present in the oil initially (Short et al. 2004b).

V.D.5 Bioavailability of Persistent Oil

Unlike the n-alkanes, which are readily degraded by microbes, losses of PAH from oils (and hence PAH bioavailability) is determined primarily by physical factors-evaporation and dissolution. The two factors that limit these processes, and hence the bioavailability of PAH in petroleum, are the ratio of surface area to volume of the oil, and the extent of weathering. Tiny droplets or thin films of oil release PAH rapidly relative to their volume, and continue to do so until the supply of PAH is exhausted. Loss rates of individual PAH depend on molecular size, with small molecules being lost more readily. Whereas the dynamics of PAH dissolution losses are usually modeled by equilibrium thermodynamics (e.g. Shiu et al. 1988), and an even simpler approach may be used for the very sparingly soluble alkyl-substituted PAH, especially those with three or more aromatic rings or extensive alkyl substitution. The intrinsically slow dissolution rates of these compounds into media that are effectively an infinite sink means that equilibrium boundary layers may become negligibly thin, and relative PAH loss rates may then be predicted on the basis of molecular surface area. This approach was applied to the oiled sediments and mussels analyzed for the *Exxon Valdez* oil spill, and found that nearly all of the 1,500 samples evaluated showed the same pattern of PAH weathering losses. This pattern is described by $P_{i,j,t} = P_{i,j,0} \exp(-k_j w_i)$, where $P_{i,j,t}$ is the concentration of the *j*th PAH in the *i*th sample of oil at time *t*, k_j is a relative dissolution rate constant for the *j*th PAH, and w_i is a weathering parameter describing the extent of evaporative and dissolution losses.

The weathering parameter, *w*, provides a convenient means of quantitatively comparing PAH composition changes induced by physical weathering. Examples of the relation between *w* and the PAH composition of weathered oil are depicted in Fig. 5.14. Values of $w < 2$ correspond with PAH compositions dominated by naphthalenes, values of $2 < w$

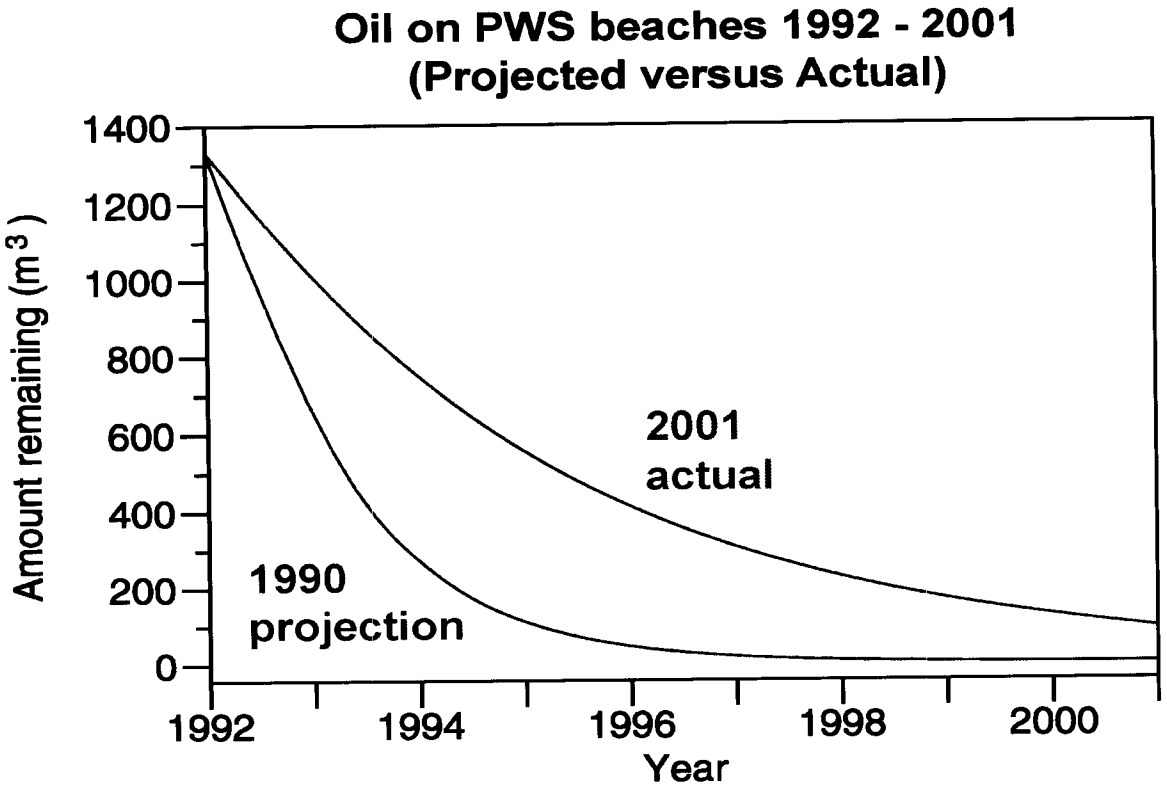


Fig. 5.13. The rate of oil loss estimated in 1990 was more rapid than the revised rate based on random survey in 2001, hence oil was likely more abundant than anticipated by factors of ten or more through the mid- to late-1990s (Short et al. 2004a).

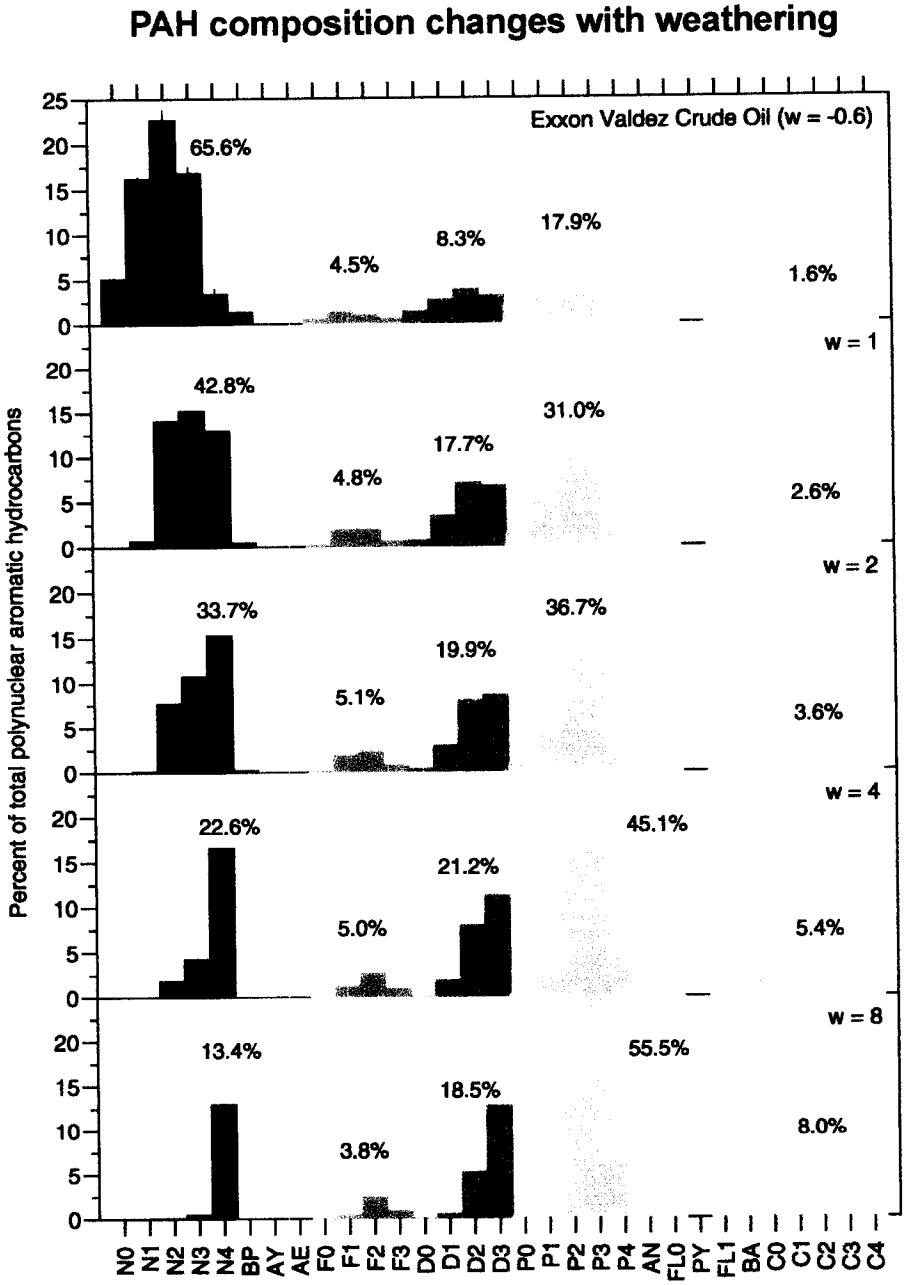


Fig. 5.14. Changes in polynuclear aromatic hydrocarbon composition as *Exxon Valdez* oil weathers, summarized by weathering coefficient *w*, as estimated by the model of Short and Heintz (1997).

< 6 by 3-ring PAH (especially phenanthrenes), and values of > 6 by 4-ring PAH (especially chrysenes). These ranges also correspond with oil weathering states that may be categorized as light, moderate and heavy, with viscosities that increase from readily mobile fluids to slightly mobile fluids to immobile asphalt-like crusts. Using this weathering scale, analysis of oiled sediments and biota during the first few years following the spill showed values that range from near zero to > 10 each year, with only a very weak trend of increased values with time (Short and Heintz 1997). This is because the oil was distributed among an array of environments, from surface layers of heavily-weathered oil to compact subsurface deposits that are little changed 12 years after deposition (Short et al. 2004b). These subsurface deposits formed persistent reservoirs of toxic PAH that were potentially available to biota that associate with the intertidal in those portions of PWS that were most heavily oiled initially, especially the intertidal infauna: pink salmon, sea otters and some sea ducks.

V.E. Effects of the spill on aquatic organisms

The spill affected organisms and communities acutely (within days and weeks), short-term (up to 2 years), and long-term (up to 15 years after the spill) and this sub-section is organized into those three periods of effect. Effects varied from severe and immediate toxicity from large doses of oil and hypothermia, to longer-term chronic effects and indirect effects. The effects of the spill on intertidal communities, fish, birds, and mammals varied depending on the severity and length of exposure. Within days and weeks, the presence of surface oil combined with BTEX compounds in the water and air resulted in acute mortalities of large numbers of animals over a wide range of habitats. The late winter and early spring are very stressful times for many animals as their stored energy has been depleted by the long winter and food is not yet very abundant prior to the start of the production season. Many organisms were therefore living on the energetic edge in the early spring of 1989, emerging from a cold winter and beginning migration and reproductive cycles. For the next few years, weathered oil was still present in habitats and in some organisms that had been initially exposed to oil. These effects, which we refer to as short term, although not immediate, were similar to those seen in many previous oil spills where response and recovery takes time. Finally, the persistence of bioavailable oil in low-energy environments such as beach sediments resulted in long-term effects, both direct and indirect, that had been rarely documented in previous oil spills.

Some species recovered faster than others, but a number of species discussed in this section continued to be affected in the months and years following the spill. Full recovery of all the aquatic species has still not happened at the time of this writing, 15 years after the spill. This is not only because the oil persisted in the environment, but also because recovery requires time after significant exposure has ceased. Most of the species in this section are still listed as recovering after a decade or more (section V.F. addresses recovery). The purpose of this examination is to address why some species in some environments were more affected than others.

First we will describe the initial mortality among seabirds, sea otters, and seals due to direct oil contact, inhalation and coating. Then we will turn our attention to animals such

as Pacific herring and pink salmon juveniles, that were exposed to oil in the short term (up to 2 years); this will also include a summary of the effects on intertidal life. Finally, we will focus on long-term effects (>2 years) on pink salmon embryos and on mussels that were the result of oil sequestered in the nearshore shallow sediments. Over longer periods of time species such as sea otters and Harlequin ducks that forage in the intertidal zone of heavily oiled habitat, experienced reductions in survival and recovery. These reductions are more likely a result of exposure to oiled substrate and or consumption of oiled prey. Indirect effects extended beyond those caused by exposure to persistent oil and included cascade effects, such as the loss of macroalgae that provide important habitat for dependent species.

An overall summary of the biological effects of the spill documented by government studies is presented in Appendix A. Much of this information is summarized in the volume from the 1993 symposium (Rice et al. 1996). As mentioned above, not all of these results are discussed in this section; rather our emphasis is on the longer-term effects of the spill.

V.E.1 Acute Effects

The initial effect of the spill was partially reflected in the death toll: 250,000 seabirds, 2800 sea otters, 300 harbor seals, and 250 bald eagles. There were about 2000 kilometers of intertidal habitat that were also inundated with oil. This estimated toll is based on carcass counts from beaches (bald eagles, Bowman et al. 1995; sea birds, Piatt and Ford 1996; and sea otters, Garrott et al. 1993), and population censuses from before and after the spill (harbor seals, Frost et al. 1994, 1999).

V.E.1.a Birds

Seabird damage from spilled oil has less to do with the amount of oil as it does with the proximity of slicks to large bird congregations and the foraging habits of the birds. Some seabirds swim extensively on the surface of the water where the potential of oiling was greatest. For example, oil near murre colonies in the Gulf of Alaska resulted in large numbers of these diving seabirds being coated with oil and dying quickly; probably more than 100,000 murres died in this way in the first month after the spill. In addition, migratory seabirds aggregate and many of them may be exposed if the aggregation is in the spill's path. Other aspects of the foraging habits of seabirds also put them at continued risk. For example, it is estimated that more than 1600 Harlequin ducks were among the quarter million seabirds killed by the *Exxon Valdez* oil spill, but their foraging habits took them throughout the intertidal zone, and exposed them to lower levels of residual oil for years (Esler et al. 2000). These and five other species of seabirds still have not recovered fifteen years after the spill (EVOSTC 2002).

Coating and acute oil toxicity initially killed birds; and, as we shall discuss later, long-term damage was also evident in shorebirds that encountered lingering oil in their habitat. Just after the spill, preening birds ingested oil coating their feathers. When oil coating was extensive and feathers lost their shape, cohesion, and insulating ability, many birds lost their ability to fly and keep warm. Without such insulation, birds quickly become

hypothermic and die. The timing of the Exxon spill was unfortunate for migratory birds as it occurred during the migratory season, thus affecting more than just resident birds, so the spill had an effect beyond the geographic boundaries of the slick.

V.E.1.b *Sea Otters*

Some of the most persistent memories of the oil spill were the dead and dying sea otters. Being the only sea mammal without blubber, the otter's dense fur and high metabolism are their only protection from the cold subarctic waters. The fur and its air bubbles also help keep them afloat. Prior to the spill, the estimated sea otter population in Alaska exceeded 100,000 with more than one third living in the path of the oil spill (Bodkin et al. 1995). Over 1000 carcasses were recovered from the spill area, their fur matted with oil. Effective insulation from the cold is lost and an otter dies when more than 20% of an otter is coated with oil(Costa and Kooyman 1982). Many more than 1000 sea otters may therefore have died and sunk, or the carcasses scavanged, their corpses never recovered. Those dead otters, which did not sink, had weakened lung membranes and damaged kidneys, both of which indicated the otters could have trouble diving. Hypothermia combined with emphysema and starvation killed an estimated total of from 750-2650 (a statistical range) sea otters during the first year after the spill (Garrott et al. 1993). In addition, 347 live sea otters were taken to rehabilitation centers in 1989, and 116 of these died at the centers from a likely combination of toxicity and shock (Rebar et al. 1995). In 1990 and again in 1991, a high proportion of adult sea otters died in PWS with mortality of recently weaned juveniles also being higher in oiled areas.

V.E.1.c Seals

Harbor seals are also vulnerable to oil, yet in PWS they did not avoid oil slicks, or oil-coated algal mats in the intertidal zone. In May 1989, 81% of 585 harbor seals observed in oiled areas of PWS were coated with oil and many were heavily contaminated (Frost et al. 1994). How detrimental the oil actually was is unclear, despite this high incidence of oiling. Only 14 dead seals, mostly pups, were recovered in PWS after the spill, although others undoubtedly died, their carcasses were never recovered. This is not a large number and many of these could have been the result of natural mortality, which is high in harbor seal pups. Harbor seals are buffered against the most deadly effect of oil contamination to warm-blooded animals, loss of insulation and hypothermia. Harbor seals rely on blubber for insulation and despite having their hair coated with oil they could stay warm. Fumes of BTEX are suspected of affecting seals, but there were no direct measurements of these volatile hydrocarbons made just above the slicks where the seals breathe.

The only conspicuous evidence that seals were affected was the lethargy of many animals. Harbor seals are normally wary and flee when people approach to within 100-200 m or when aircraft fly over them at low altitudes. In the first two to three months after the spill, however, many oiled seals could be approached on foot to within a few meters and were described as sick or unusually tame (Lowry et al. 1994). In contrast to this unusual behavior, interactions between oiled mother-pup pairs appeared normal—they seemed to remain bonded and pups nursed, even on heavily oiled mothers.

The physical condition of pups appeared normal as well, indicating that nursing and milk provisioning were successful. Since stored blubber by the mother, from before a pup is born, acts as the source of milk during the first six weeks of nursing, this strategy may have minimized the dose of spilled oil to pups born in the spring of 1989.

Examinations of dead seals often failed to determine causes of death because of the length of time between death and examination. However, 27 seals were collected and immediately examined and all were found to have several types of external and internal lesions that were consistent with oil exposure (Spraker et al. 1994). In most cases the lesions were mild and likely reversible. Lesions in the brain of some seals, however, may explain the disorientation and lethargy noted in numerous heavily oiled seals in early summer (Lowry et al 1994), and may have been responsible for the deaths of the most severely affected animals. Live oiled seals taken to rehabilitation centers did not display behavioral or clinical signs of organ dysfunction, and 15 of the 18 animals brought in survived to release (Williams et al. 1994).

V.E.1.d Sea Lions

Acute oil effects were equivocal for Steller sea lions (*Eumetopias jubatus*) that inhabited PWS. Oil did not persist as long on sea lions as harbor seals (Calkins et al. 1994). Although histological examination of adult sea lions did not reveal significant oil-related damage (Calkins et al. 1994), because of the migratory behavior of sea lions, it was not possible to do a before-and-after comparison and estimate spill mortality.

V.E.1.e Whales

Several cetacean species frequented the spill area and were even photographed in oil slick areas, but there is not enough data or carcasses to determine whether the spill affected whales. In some cases, numbers definitely declined after the spill, but carcasses were not recovered so the linkage to the oil spill is equivocal. Mysticete and odontocete whales are able to detect oil but apparently do not avoid it (Geraci 1990); killer, humpback, and gray whales and Dall's porpoises were observed in oiled waters (Harvey and Dahlheim 1994; Matkin et al. 1994; von Ziegesar et al. 1994).

The case for an oil effect on cetaceans was strongest for a single pod of killer whales resident in PWS, yet even this evidence is equivocal and circumstantial. When the AB pod was first sighted after the spill, seven fewer whales were seen than had been sighted in the previous year and six more disappeared by June 1990 (Matkin et al. 1994). These losses of about 20% per year, were much higher than any previously known natural rate and could not be explained by emigration to another pod or formation of a secondary pod (Matkin et al. 1994). Missing individuals were primarily juveniles and reproductive females. Although killer whales in AB pod had interfered with the sablefish fishery in the mid-1980's, some were killed and some of the surviving whales carried scars from bullets, the fishery was not blamed for the atypical mortalities discovered in 1989-1990 and it was suggested that the spill accounted for this unusually high loss (Dahlheim and

Matkin 1994; Matkin et al. 1994). Also, the dorsal fins of two AB males began to collapse in 1989, a possible sign of poor health, and these whales remained permanently disfigured (Matkin et al. 1994) (Fig 5.15). Mortality in other killer whale pods was not unusual.

No distressed killer whales were observed, the missing whales had last been sighted 6 months before the spill, and no carcasses were found for pathologic or toxicologic studies because these whales usually sink after dying (St. Aubin and Geraci 1994; Loughlin et al. 1996). If the spill were responsible for killer whale death, the route may have been inhalation of oil or hydrocarbon vapors (Matkin and Saulitis 1997); the pod was photographed in and near slicks in the early days of the spill. Such inhalation may inflame mucous membranes, cause lung congestion, lead to pneumonia and cause neurological damage and liver disorders (Geraci 1990). High volatile hydrocarbon concentrations (> 100 ppm) or long exposures may be required to cause damage (Geraci 1990; Matkin and Saulitis 1997) and post-spill studies suggested that concentrations did not reach this level (Matkin and Saulitis 1997). However, negative consequences of inhaling petroleum vapors from the *Exxon* spill were plausible in sea otters and harbor seals (St. Aubin and Geraci 1994).

There were even fewer observations to assess the post-spill changes in other cetacean species. Changes in humpback whale abundance, calving rates, seasonal residency time, and mortality were not significant; von Ziegesar et al. (1994) concluded that there were insufficient data to detect oil-related effects. Dall's and harbor porpoises may have been affected by the spill, but little study effort was directed toward them (Loughlin et al. 1996). One group suggested three gray whales in a moderate amount of oil appeared lethargic (Harvey and Dahlheim 1994), while Loughlin (1994) did not detect alterations in swimming speed, direction, and breathing behavior. The number of gray whale carcasses discovered in 1989 exceeded those in other years, but was not unusual compared to other regions and could be explained by coincidental timing of the survey and migration. The cause of gray whale deaths could not be determined (Loughlin 1994) and concentrations of potentially toxic chemicals in carcasses (PAHs, chlorinated hydrocarbons, and toxic metals) were low compared to concentrations in tissues of marine mammals feeding on higher trophic levels (Varanasi et al. 1994).

Cascading Effects: Killer Whales
The loss of key individuals in socially organized populations such as killer whales can induce subsequent depressed reproduction. After exceptionally high mortality of 20% between September 1988 and spring 1989 (prior to the spill) and another 20% during the following year in the AB pod of resident (fish-eating) killer whales that had been observed swimming through the spill, losses of adult females from these matriarchially-organized family groups led to suppressed reproduction (Loughlin 1994).

Fig. 5.15. Two males from AB pod of killer whales with collapsed dorsal fins.

V.E.2 Short-term Effects of the Spill

V.E.2.a Pacific herring

The *Exxon Valdez* oil spill occurred at the worst possible time for Pacific herring (*Clupea pallasi*), a keystone species of great ecological and commercial importance. In late March 1989 adult herring were entering the nearshore habitat in PWS to spawn on intertidal vegetation and rocks. Eggs were spawned in early to mid April and incubated about 24 days (Biggs and Baker 1993); thus the critical embryonic development period coincided with the highest aqueous concentrations of oil (Brown et al. 1996a; Carls et al. 2002). All available evidence indicates that the primary source of herring egg contamination by oil in PWS was the water. Low concentrations of dissolved PAH were present in water (Neff and Stubblefield 1995; Short and Harris 1996), and were accumulated from water by both caged (Short and Harris 1996) and native mussels (Brown et al. 1996a) in herring spawning areas. By contrast, oil in beach sediment was infrequently encountered and in low concentrations where detectable (Pearson et al. 1995a; Short and Babcock 1996; Short et al. 1996). Approximately 25 to 32% of the developing embryos were exposed to potentially damaging oil concentrations (Carls et al. 2002). However, the effect of lost production on the herring population could not be estimated because natural recruitment processes are poorly understood and not predictable.

Herring embryos were damaged by oil exposure (Fig. 5.16). Compared to reference embryos, those exposed hatched earlier, were longer but weighed less, and had skeletal, craniofacial, and finfold abnormalities (Hose et al. 1996; Brown et al. 1996a; Marty et al. 1997a). The severity of skeletal abnormalities, certain types of craniofacial defects (jaw abnormalities, microphthalmia, and absence of otic capsules) and the total severity index were significantly correlated to log-transformed total PAH concentrations in mussels collected nearby (Hose et al. 1996). Observed responses were confirmed with laboratory study; total PAH concentrations between 0.4 and 9.1 ppb damaged embryos (Carls et al. 1999). In PWS, April/May 1989, concentrations detrimental to herring embryos in the laboratory were well within documented aqueous TPAH concentrations, in the low parts per billion (Neff and Stubblefield 1995; Short and Harris 1996a).

Pacific herring larvae were adversely affected by oil in 1989, and this was confirmed in laboratory studies. Major oil-associated effects in larvae captured from oiled sites in spring 1989 included: small size, ascites (fluid accumulation), pericardial edema (swelling around the heart), delayed development, and genetic damage (Marty et al. 1997a). Microscopic lesions were consistent with decreased growth and increased mortality of herring larvae collected near oiled beaches (McGurk and Brown 1996), and were similar to those observed in laboratory studies of herring larvae exposed to Alaska North Slope crude oil (Kocan et al. 1996; Marty et al. 1997; Carls et al. 1999). Growth of older larvae from offshore areas also decreased throughout PWS in 1989, but a direct link to oil exposure was not possible because these larvae likely originated from a mixture of oiled and unoiled areas (Norcross et al. 1996). In contrast, the frequency of genetic defects was low and jaw size was within normal limits in PWS larvae six years after the spill (Norcross et al. 1996).

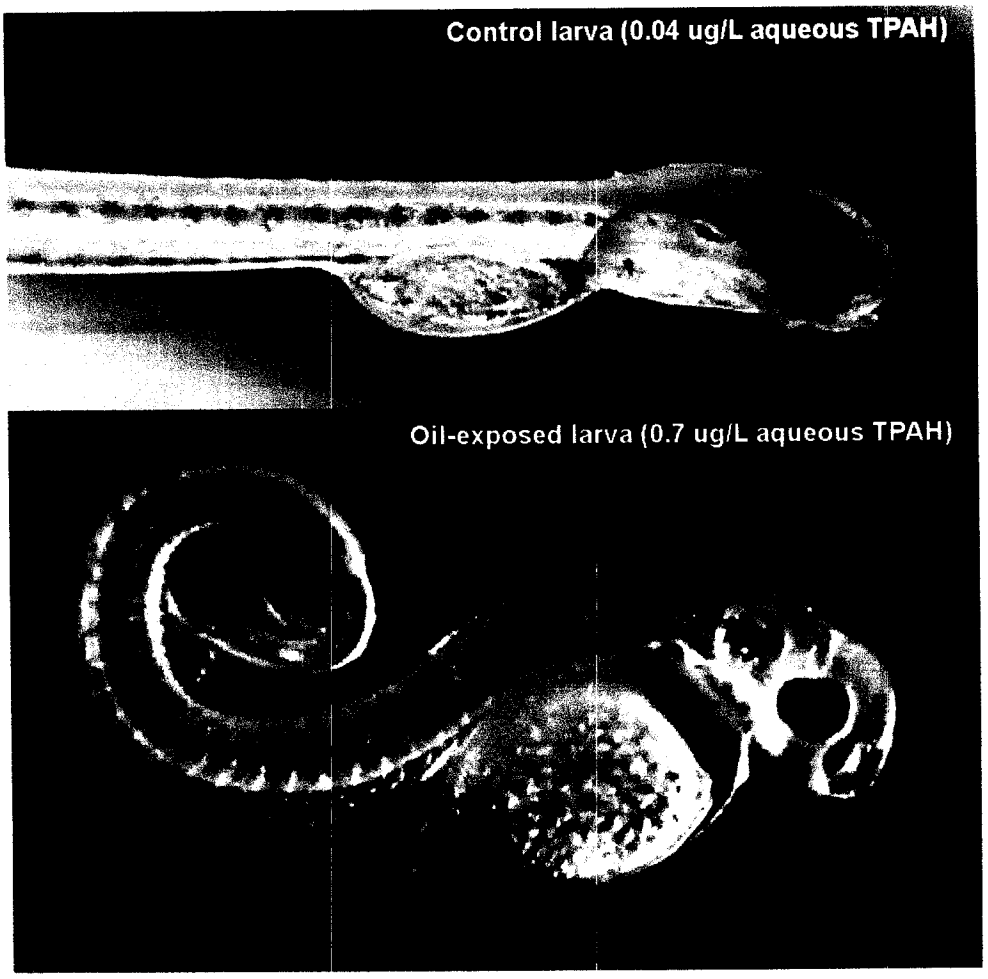


Fig. 5.16. Exposure to oil damages developing Pacific herring embryos in characteristic ways, including edema, spinal deformities, and heart defects. The abnormal embryo was exposed to oiled water for just 4 days (Carls et al. 1999).

Adult Pacific herring in the path of the slick in 1989 were exposed to hydrocarbons and exhibited significant lesions, but fish from reference sites did not (Moles et al. 1993; Marty et al. 1999). Fish from oiled sites had hepatic necrosis and elevated PAH concentrations (primarily naphthalenes) in their tissues (Marty et al. 1999). Naphthalenes were also preferentially accumulated in muscle tissue in laboratory exposures of adult herring to PAH in water (Carls et al. 2000). These fish also had evidence of oil metabolism (Thomas et al. 1997) and differential hydrocarbon uptake. Herring from oil-exposed areas had fewer nematode parasites in their body cavity than did fish from reference sites (apparently because stressed nematodes migrated into muscle tissue), and the link to sublethal oil exposure was confirmed through laboratory study (Moles et al. 1993). In later laboratory studies, exposure of wild herring to concentrations of crude oil similar to those that were encountered following the oil spill depressed immune functions and allowed expression of a viral disease, viral hemorrhagic septicemia virus (VHSV) in concert with increased liver lesions (Carls et al. 1998) (Fig. 5.17). A small fraction of the population probably died as a result of this viral outbreak. The lesions originally attributed solely to oil exposure in herring captured in PWS in 1989 may have been caused by disease, which, in turn, may have been triggered by oil exposure. In 1990 and 1991, fish sampled from oiled sites had neither oil-related lesions nor significant PAH concentrations in their tissue (Marty et al. 1999).

V.E.2.b Pink salmon juveniles

Pink salmon (*Oncorhynchus gorbuscha*) provide an important fishery in the Gulf, especially PWS, and were the most extensively studied species after the spill. At 24.5 to 49.5 million fish caught annually (hatchery raised and wild) prior to the spill (Sharr et al. 1995), the pink salmon resource is a mainstay of the fisheries economy in PWS (49.5 million fish in 2003). Pink salmon are also a major part of the PWS ecosystem with returns of wild pink salmon to the sound ranging from 1.8 to 2.1 million adult fish in summer (Bue et al. 1996), and the annual release of more than a half million hatchery-produced fry in spring. Effects were thoroughly documented at all the life stages, often at very low oil concentrations (Fig. 5.18).

At the time of the spill, fry were emerging from the spawning gravels, feeding along the shoreline, and were soon going to migrate toward the Gulf of Alaska. The primary fry migration route from PWS to the Gulf of Alaska is the same route the oil followed, the Alaska Coastal Current--through the western Sound and southwest passages-- thus exposing fry to oil (Carls et al. 1996; Wertheimer and Celewycz 1996; Willette 1996). Despite high mixing energy and large amounts of oil released, the inherent low solubility of oil in water resulted in only low concentrations (up to 6.2 ppb) (Short and Harris 1996). These low concentrations of oil in water were dominated by polycyclic aromatic hydrocarbons rather than the monocyclic aromatics, which had largely evaporated to the atmosphere. Caged mussels deployed below the surface for 2-8 weeks accumulated hydrocarbons matching the composition of the spilled *Exxon Valdez* oil (Short and Heintz 1997).

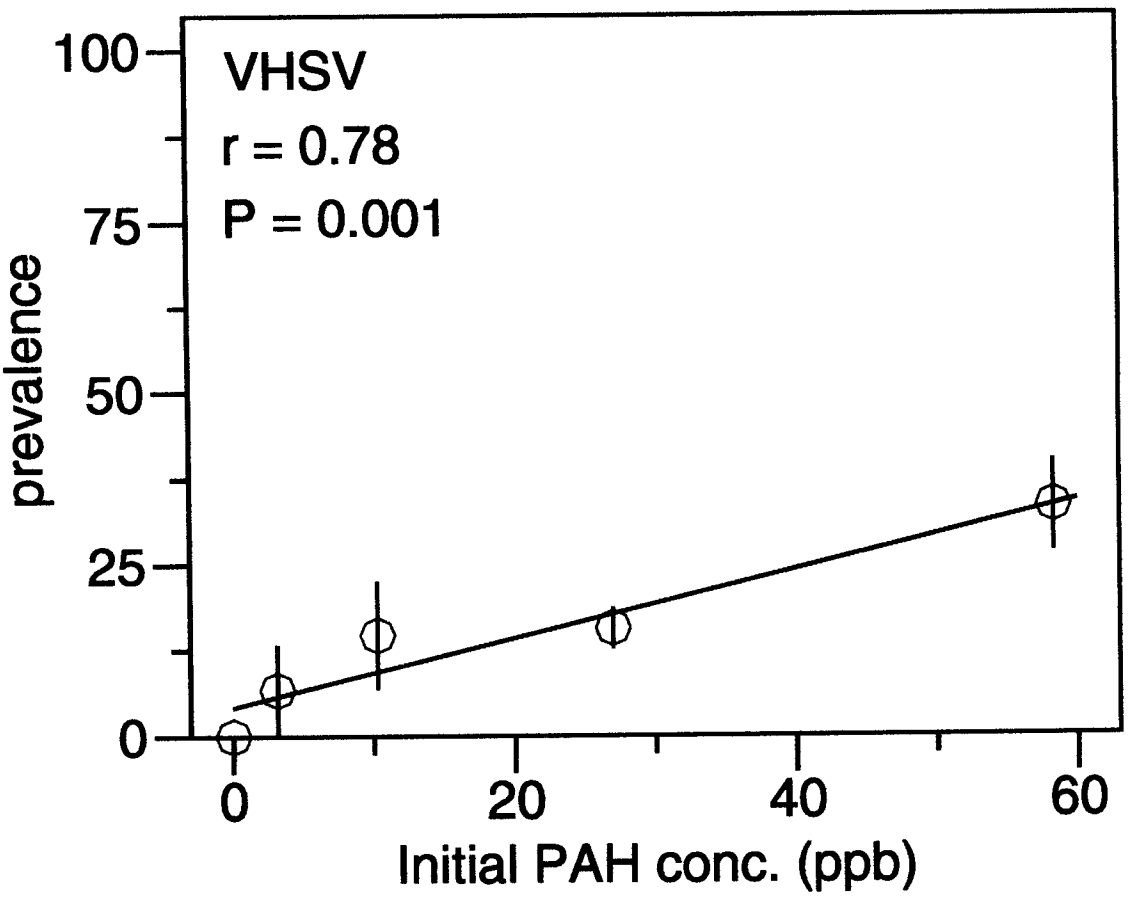


Fig. 5.17. Exposure of wild herring to concentrations of crude oil similar to those that were encountered following the oil spill resulted in expression of a viral disease, viral hemorrhagic septicemia virus (VHSV). There was a linear relationship between increasing oil concentration and greater prevalence of experimental herring with the virus expressed.

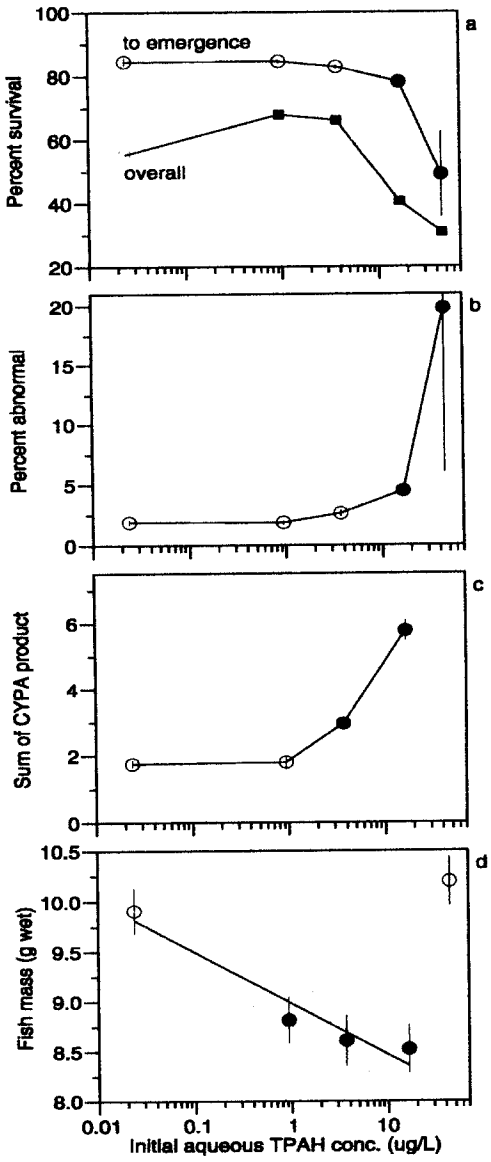


Fig. 5.18. Low-level exposure to oil in water reduced pink salmon embryo survival (a), caused abnormalities in emergent fry (b), and induced a mixed function oxygenase enzyme, cytochrome P4501A (c), also measured at emergence. Abnormalities observed at emergence included ascites, bulging eyes, malformed heads, short opercular plates, external hemorrhaging, mouth or jaw malformations, and deformed caudal fins. Growth of emergent fry, cultured for 6 months in clean water, was depressed by prior exposure to oil (d). Although growth was predictably depressed in most oil treatments, only the most vigorous fish survived the highest oil treatment and these survivors outgrew all others,an example of natural selection. Vertical bars are ± 1 standard error.

The low concentrations of oil in the water did not directly kill pink salmon fry as the hydrocarbon concentrations were far below acutely toxic concentrations and declined with evaporation and dilution. However, growth was reduced in both wild (Wertheimer et al. 1996) and hatchery-raised fry (Willette 1996) entering the sound and migrating through oiled areas (Fig. 5.19). Wild pink salmon fry collected from nearshore waters in oiled areas between April 10 and June 26, 1989 grew at half the rate of fry from reference areas (Wertheimer and Celewycz 1996). These differences were noted primarily in migration corridors rather than in the bays where the fry spent little time. Willette (1996) measured lower growth in hatchery-reared pink salmon collected in oiled areas than in reference sites in 1989. These fry were coded-wire tagged and measured at release, giving very accurate determinations of marine growth (Fig. 5.20). Fry depend on rapid growth soon after emergence to escape predation (Parker 1971; Healy 1982), thus indirect mortality likely occurred as a result of retarded growth (Geiger et al. 1996). These wild and hatchery pink salmon fry were exposed to *Exxon Valdez* oil in 1989, as evidenced by tissue PAH concentrations, induction of the biomarker enzyme cytochrome

P4501A, and hydrocarbons present in the tissues (Carls et al. 1996b; Willette 1996; Wiedmer et al. 1996) (Fig. 5.21), and oil globules observed in the stomachs and intestines of fry (Sturdevant et al. 1996). By 1990, fry grew comparably in oiled and reference portions of the Sound, with no evidence of increased P4501A enzyme induction or petroleum hydrocarbons in their tissue, so oil-reduced growth of pink salmon fry in the marine environment was restricted to 1989. Growth reduction in oil-exposed fry was probably caused by a shift in net energy from growth to hydrocarbon metabolism (Fig. 5.22). In laboratory tests, decreased growth was observed in pink salmon fry exposed to sublethal concentrations of aromatic hydrocarbons in water (Moles and Rice 1983) or in food (Carls et al. 1996a). The oil fractions having the greatest effect were the 3- and 4-ringed aromatics. Food consumption was not reduced in fry captured in oil-contaminated locations in PWS (Sturdevant et al. 1996), or in laboratory experiments with oiled food (Carls et al. 1996a), but the same concentrations of oil in the water column that reduce growth are also capable of increasing the respiration rate (Thomas and Rice 1979), suggesting that energy for growth was diverted to hydrocarbon metabolism and metabolite excretion.

Population effects of the spill were identified through modeling, with 1.9 million fewer adult pink salmon returning in 1990 (Geiger et al. 1996). These loss estimates were driven by growth reductions measured in the field studies by Wertheimer et al. (1996) and Willette (1996). There was no catastrophic effect of the spill on juvenile or adult pink salmon, probably due to very favorable marine survival conditions. Geiger et al. (1996) did not measure population effects at the stream level and natural straying rates in PWS are very high, sometimes as high as 50% and probably could have swamped any variations in fry-to-adult survival in oiled streams (Sharp et al. 1994). Oil exposure early in life has little effect on natural straying rates as embryonic exposure of pink salmon alevins subsequently released in southeast Alaska did not result in any increased straying (Wertheimer et al. 2000).

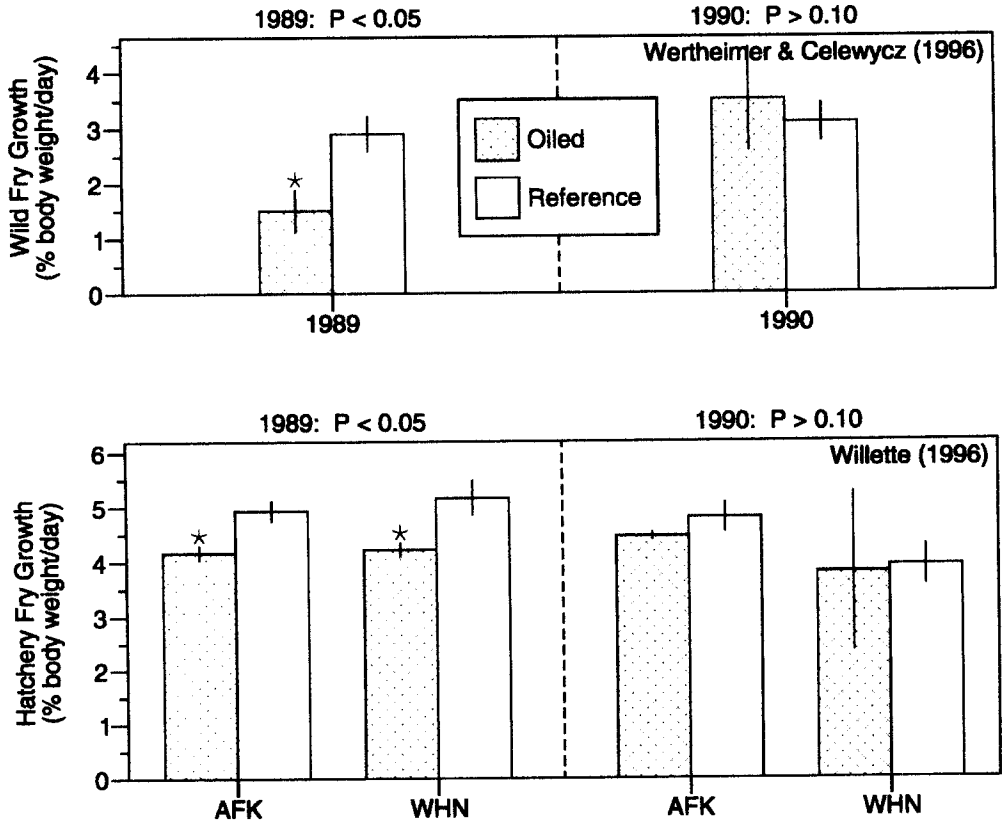


Fig. 5.19. In 1989, wild and hatchery pink salmon fry grew slower in oiled areas of Prince William Sound than in non-oiled areas; asterisks indicate significant differences between reference and oiled fry. Rates of growth in these same areas were not significantly different a year after the spill.

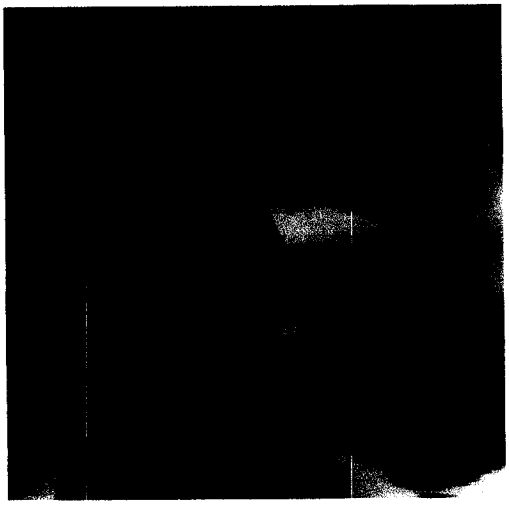


Fig. 5.20. Pink salmon juveniles with coded wire tags in their noses.

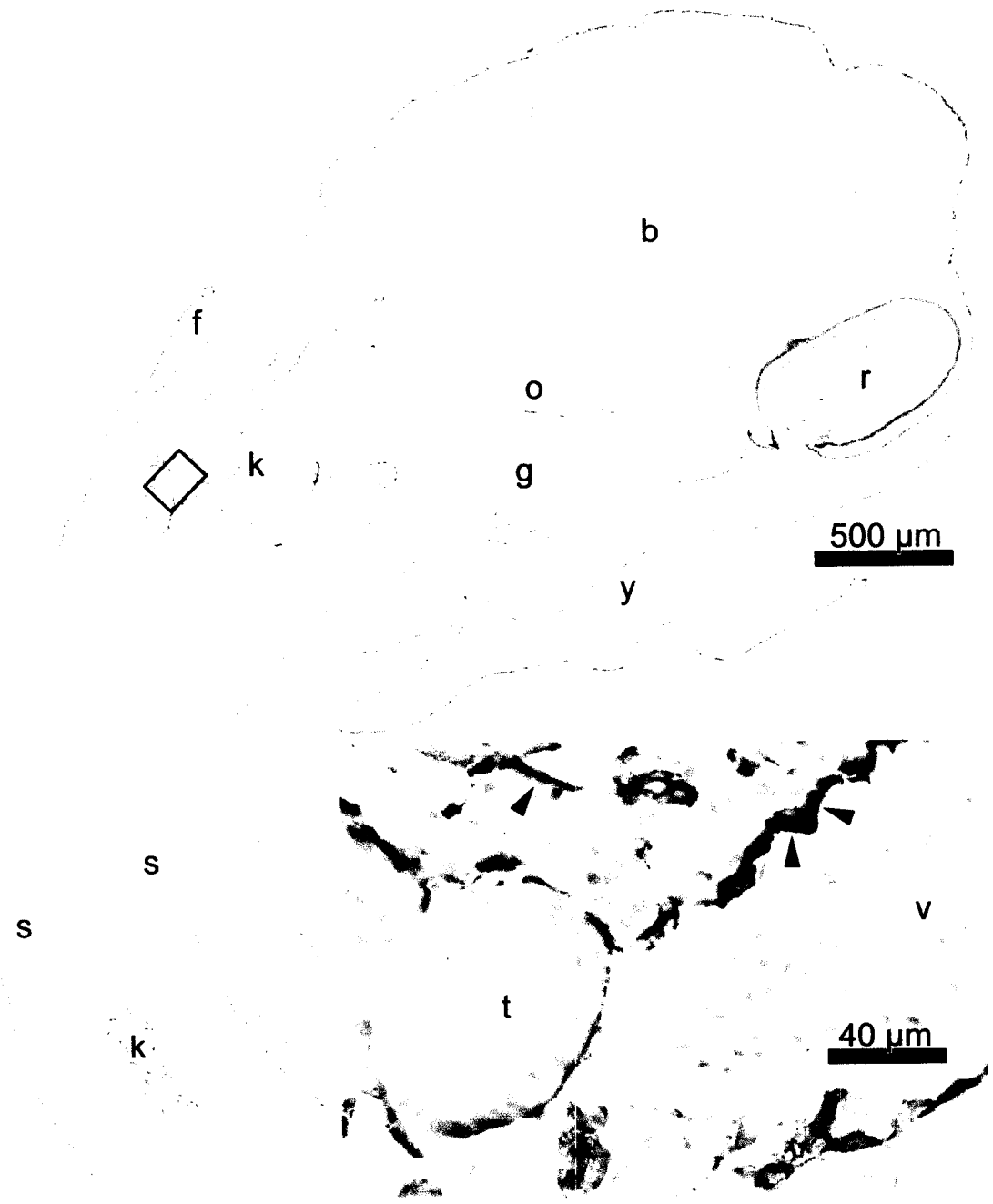


Fig. 5.21. Photomicrograph of a cross section of a 53-day-old pink salmon embryo exposed to weathered crude oil since fertilization stained for P4501A (red). The black box outlines the part of the kidney shown at high magnification in the inset. Staining is prominent in the yolk sac (y), skin (external surface), and in endothelial cells (arrowheads) of large blood vessels (v) and small sinusoids of the kidney. Staining is less intense in kidney tubules (t), and blood vessels of the gill (g) and brain (b). Also labeled are the retina (r), otolith chamber (o), pectoral fin (f), and skeletal muscle (s).

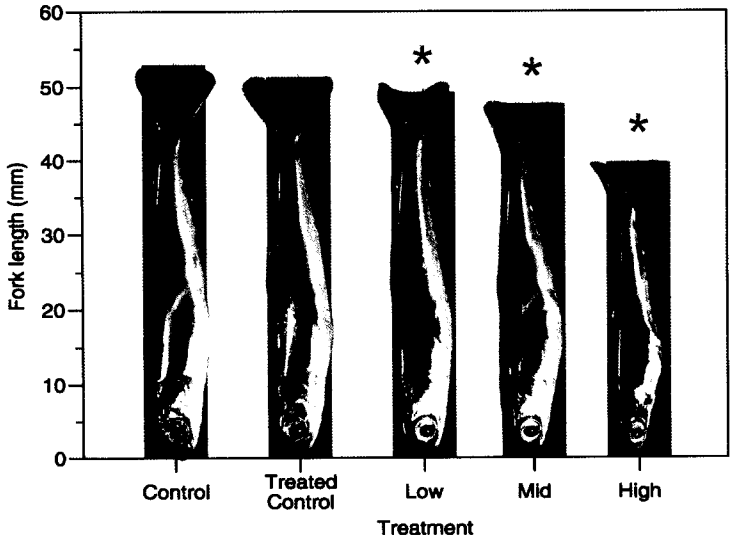


Fig. 5.22. In laboratory tests, decreased growth was observed in pink salmon fry exposed to sublethal concentrations of aromatic hydrocarbons in food (Carls et al. 1996a). Asterisks indicate significantly shorter groups.

Precipitous population declines occurred in 1992 and 1993 at the same time that plankton production was relatively low, but any linkage to long-term oil effects is speculative at best. The weight of evidence, therefore, indicates that fewer fish returned to oiled streams from the 1989-year class even though there were high returns to the region.

V.E.2.c Intertidal communities

Severe damage to the intertidal fauna lasted only about 2 years, and we address these effects in this sub-section on short-term effects of the spill. There are some important exceptions, such as clams on cleaned beaches (Driskel et al. 1996), which are discussed in chapter V.F., and recovery of algal cover in the upper intertidal (Stekoll and Deysher 2000). Of course the oil persisted in beaches much longer and brought its own set of long-term problems.

Oil slicks that came ashore were often deposited in a thick layer that smothered intertidal animals and plants (Fig. 5.23). Oil was also chemically toxic to many intertidal organisms, and its cleanup was clearly damaging (Lees et al. 1996). All different types of shorelines were affected by the oil, including rocky beaches, sand and gravel beaches and marsh. However, the most common beaches and the majority of affected shorelines were rocky with boulder and cobble.

One of the most affected members of the community was *Fucus gardneri*, the dominant intertidal brown algae (Fig. 5.24). *Fucus*, the longer-lived barnacles, *Semibalanus balanoides* and *Balanus glandula*, and mussels were greatly reduced on many rocky shorelines throughout the spill area, especially where aggressive oil cleanup techniques were used (Highsmith et al. 1996; Houghton et al. 1996; 1997; Stekoll et al., 1996). The remaining *Fucus* lost fertility (Stekoll and Deysher 2000), young gametophytes (juveniles), when they did settle and grow, dried out and died during low tides without the shading effect of the canopy, or many young plants settled and grew on barnacles and were washed off by wave action (Van Tamelen et al. 1997). Algal recruitment was shown experimentally to be reduced by tar coatings on rocks (Duncan and Hooten 1996). The greatly reduced canopy from the spill and the cleanup also set in motion a series of direct effects and indirect interactions, which apparently included settlement of opportunistic algae that are normally excluded from growing and settling, settlement of a more opportunistic barnacle, *Chathalamus dalli*, and increased oystercatcher predation on snails (Peterson 2001). Experimental removal of *Fucus* canopy in the Herring Bay region of Knight Island resulted in loss of limpets (*Tectura persona*) and the periwinkle (*Littorina sitkana*) (Highsmith et al. 1996), so both of these indirect effects were likely as well. It took *Fucus* several years to reestablish its canopy—at least until 1994 for portions of the higher intertidal zone (Stekoll and Deysher 1996; 2000). Not only were invertebrates affected, in 1990 there were significant differences in numbers of small intertidal fish sampled at oiled and unoiled sites at three habitat types in PWS: sheltered rocky, exposed rocky and coarse-textured (Barber et al. 1995), but these effects apparently only lasted until 1991.

Fig. 5.23. Oil slicks were deposited in a thick layer that smothered intertidal animals and plants. The most commonly affected shorelines were rocky with boulder and cobble.

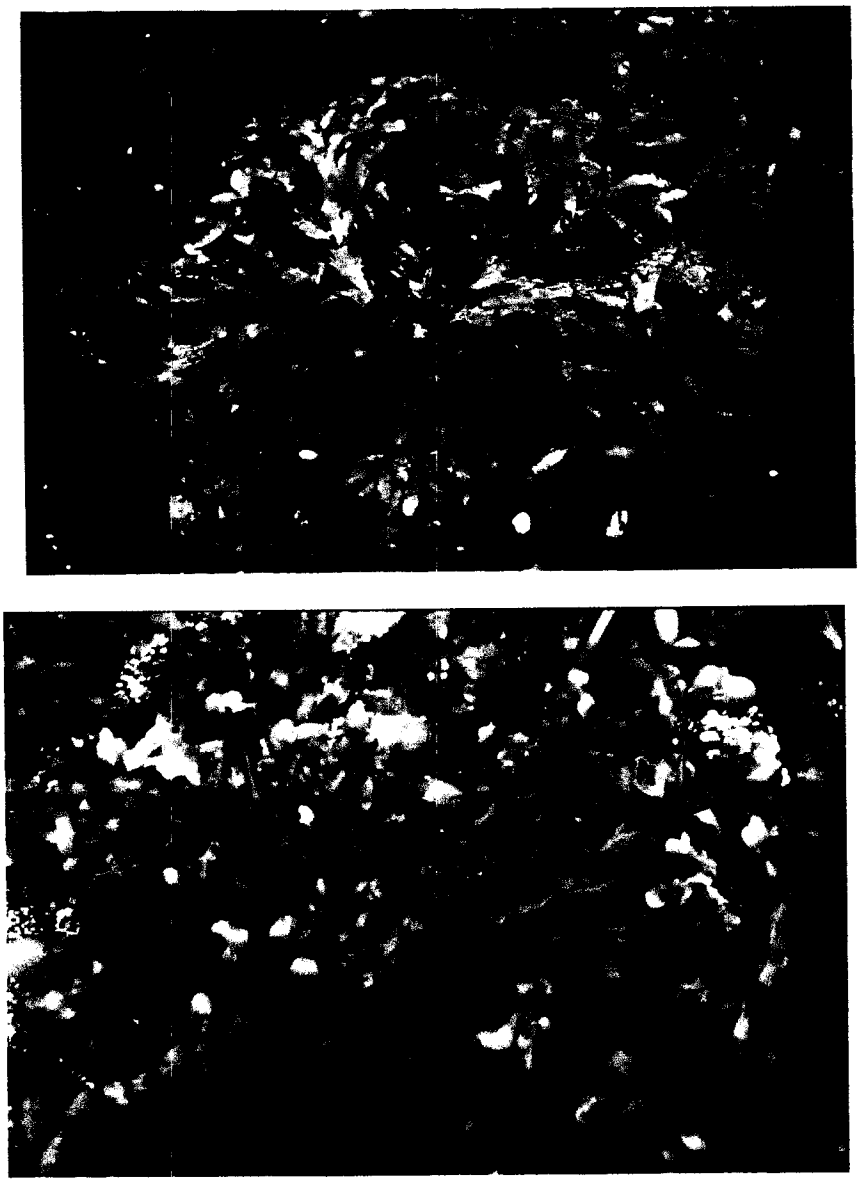


Fig. 5.24. One of the most impacted members of the intertidal community was *Fucus gardneri*, a dominant perennial brown alga: heavily oiled (top), clean (bottom).

So, in addition to learning about the recovery times of rocky intertidal communities from large-scale disturbance, the intertidal studies also provided some insight into the interrelationships between organisms in these dynamic communities.

On finer-grained beaches there was increase in oligochaetes (Stekoll et al. 1996); these annelid worms are able to feed in chronically oiled sediments and incorporate carbon from petroleum in their proteins and carbohydrates (Spies and DesMarais 1983; Bauer et al. 1990). Harpacticoid copepods were also higher on some oiled beaches (Wertheimer et al, 1996). Experiments with meiofauna (animals less than 0.4 mm in size) suggested that there could have been short-term effects on these small but important components of the fauna on soft sediment beaches (Fleeger et al. 1996).

V.E.3 Long-term Effects of the Spill

V.E.3.a Pink salmon

V.E.3.a.1 Embryos

The weight of evidence indicates the *Exxon Valdez* oil spill resulted in significant damage to pink salmon for as many as five years following the spill, as a result of reduced growth of the 1989-year class (thus reduced ocean survival) coupled with reduced survival of embryos in oiled stream deltas through 1994. About 31% of the streams used as spawning habitat by pink salmon were oiled (Geiger et al. 1996). Oil-contaminated water drained from the beaches into the streams, exposing developing embryos to PAH (Carls et al. 2003) (Fig. 5.25 and 5.26). Cytochrome P4501A enzyme induction in pre-emergent fry was noted through 1991, providing evidence that embryos were exposed to PAH from persistent intertidal oil deposits for at least three years following the spill (Wiedmer et al. 1996). Embryo mortality was consistently greater in oiled streams than in reference streams through 1993 (Bue et al. 1996; 1998; Craig et al. 1995). Pink salmon exposed in the laboratory to low part-per-billion concentrations of PAH from oil-coated gravel can result in a cascade of long-term effects on marine survival and growth (Marty et al. 1997b; Heintz et al. 1999, 2000).

V.E.3.a.2 Long-term Egg Mortality

The most dramatic example of long-term exposure to low concentrations of weathered oil occurred in the intertidal reaches of salmon streams where pink salmon eggs incubate. These long-term effects appear to be the result of the combination of lengthy exposure coupled with the sensitivity of embryonic life stages to contaminants. Approximately 75% of pink salmon in PWS deposit their eggs in the intertidal reaches of streams between mid-July and September (Helle 1964) (Fig. 5.27). Eggs deposited in the intertidal gravel hatch between late October and mid-March and the larvae, termed alevins, continue to incubate in the gravel subsisting on their yolk sac until April. When the yolk supplies are depleted in April, the fry emerge from the gravel and move immediately to the marine environment to feed. Eggs and alevins have a potential for 8-9 months of exposure to oil in the intertidal environment.

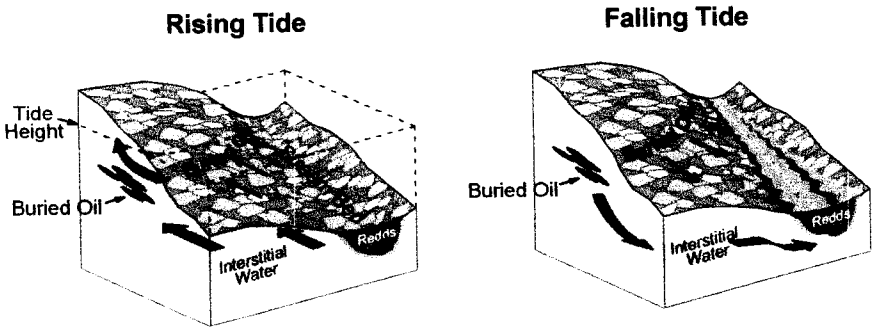


Fig. 5.25. Buried oil on stream deltas can contaminate salmon redds by leaching into streams during ebb tide. Water that passes through oiled sediment becomes contaminated with polynuclear aromatic hydrocarbons and drains downslope during falling tides, thus transferring contaminants to incubating embryos.

Fig. 5.26. Drainage of water from banks into streams and stream beds was demonstrated by release of fluorescent dye (Carls et al. 2003).

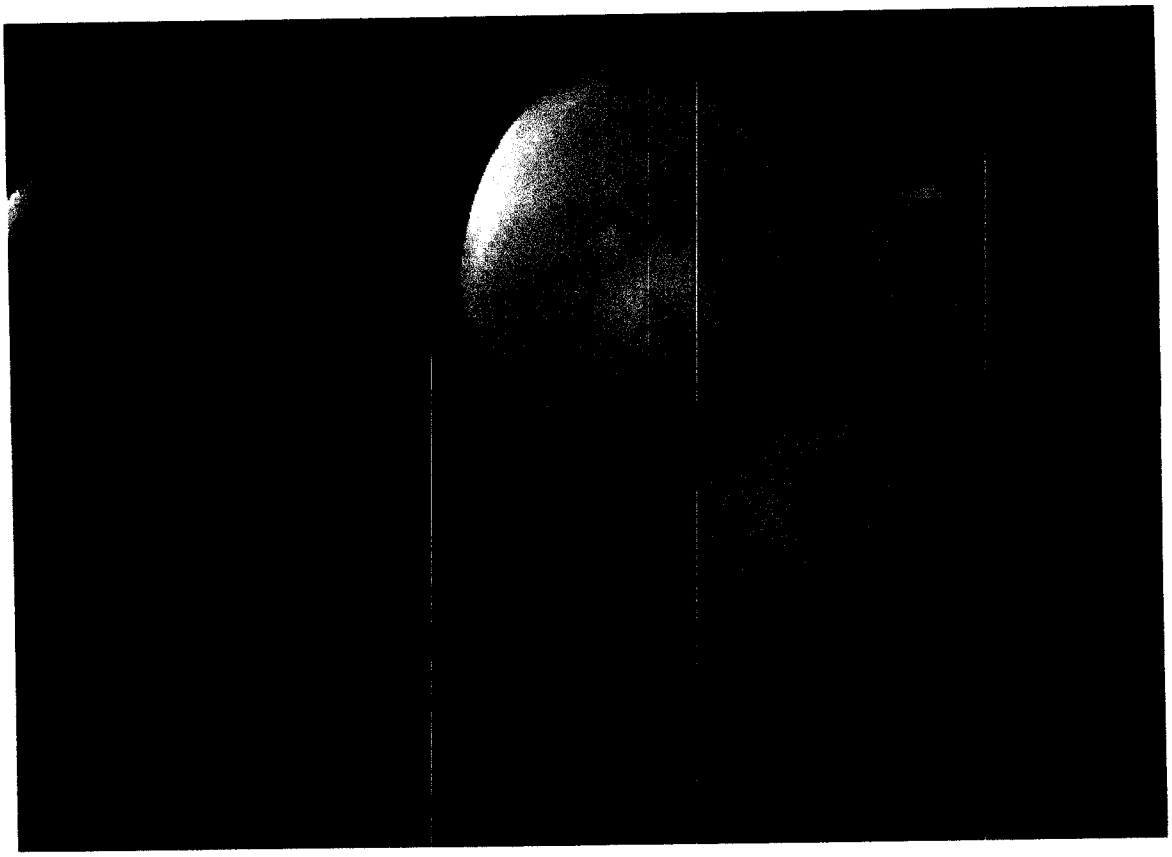


Fig. 5.27. Incubating pink salmon eggs. The top center egg is infertile and was mechanically damaged about 1 minute before it was photographed.

There was elevated egg mortality in oiled streams from 1989 through 1993, four years after the oil spill (Bue et al. 1994, 1996, 1998). Mortality rates varied each year, but relative to reference streams, the oiled streams had significantly elevated mortality rates each year (Fig 5.28). Embryos were hydraulically sampled from spawning gravels at 10 oiled and 15 reference streams from 1989 to 1997. At each stream embryos were removed from 14 locations along transects within 3 intertidal zones and one zone above mean high water. Mortality of embryos was significantly greater in oiled streams through 1993, but not significant in the years thereafter except 1997. Time of spawning and collection methods employing mechanical disturbance were proposed as possible confounding factors for concluding that there was an effect of oil on egg mortality (Brannon and Maki, 1996). However, Craig et al. (2003) continued to find oil-induced mortality when time of spawning was included as a covariate in 1991, the only year in which spawn timing in individual streams was accurately monitored. This confirmed oil as a causal factor, at least in 1991. Egg mortality was consistent with drainage of oil-contaminated water from surrounding sediment (Carls et al. 2003). Egg mortality was again greater in oiled streams in 1997 (Craig et al. 2003). The differences between oiled and reference streams in 1997 were driven primarily by elevated mortalities in three of the ten oiled streams, and these transected the beaches with the greatest oiling. Possibly these 3 streams may have shifted into new zones of oil-contaminated sediment deposits. Such shifts in intertidal stream channels are not unusual (Michel and Hayes 1994; Carls et al. 2003).

The effects of oil on salmon embryo mortalities were initially surprising, because it was obvious that there were no acute exposures directly to oil; freshwater flow in stream channels at low tide prevented the direct oiling of the spawning redds. The oil stranded along the sides of these streams was not suspected until the elevated embryo mortalities were consistently measured for several years. Murphy et al. (1999) documented the lingering oil along the banks of several streams in 1995, confirming the probable oil effects on embryos measured by Bue et al. (1996) and the elevated P4501A observed in 1989 by Weidmer et al. (1996). In 1999, a decade after the spill, lingering oil was detected in some previously oiled streams, but at low concentrations (Carls et al. 2004a). In 1999, PAH were significantly elevated in two of six previously heavily oiled streams in a pattern again consistent with stream drainage of oil-contaminated water, and chemical fingerprinting confirmed that the lingering oil matched with the spilled *Exxon Valdez* oil. Concentrations of PAH in sediment and induction of cytochrome P4501A enzymes in pink salmon embryos were correlated with PAH concentrations in streams (Carls et al. 2004b) (Fig. 5.29). This indicates PAH were bioavailable in some streams a decade after the spill. Oil was, however, not present in four streams that had been previously heavily oiled, thus only the most severely oiled beaches were still exposing embryos to PAH a decade after the spill.

Interstitial (below the beach surface) water flow within the stream beds are driven by a combination of tidal pumping and precipitation. Fluorescent tracer dyes injected into beaches during ebb tide were observed throughout most of the adjacent intertidal zone, including surface and subsurface stream water where salmon eggs incubate (Carls et al. 2003). Mean horizontal groundwater flow through intertidal gravel was rapid (4 to 7 m per hour).

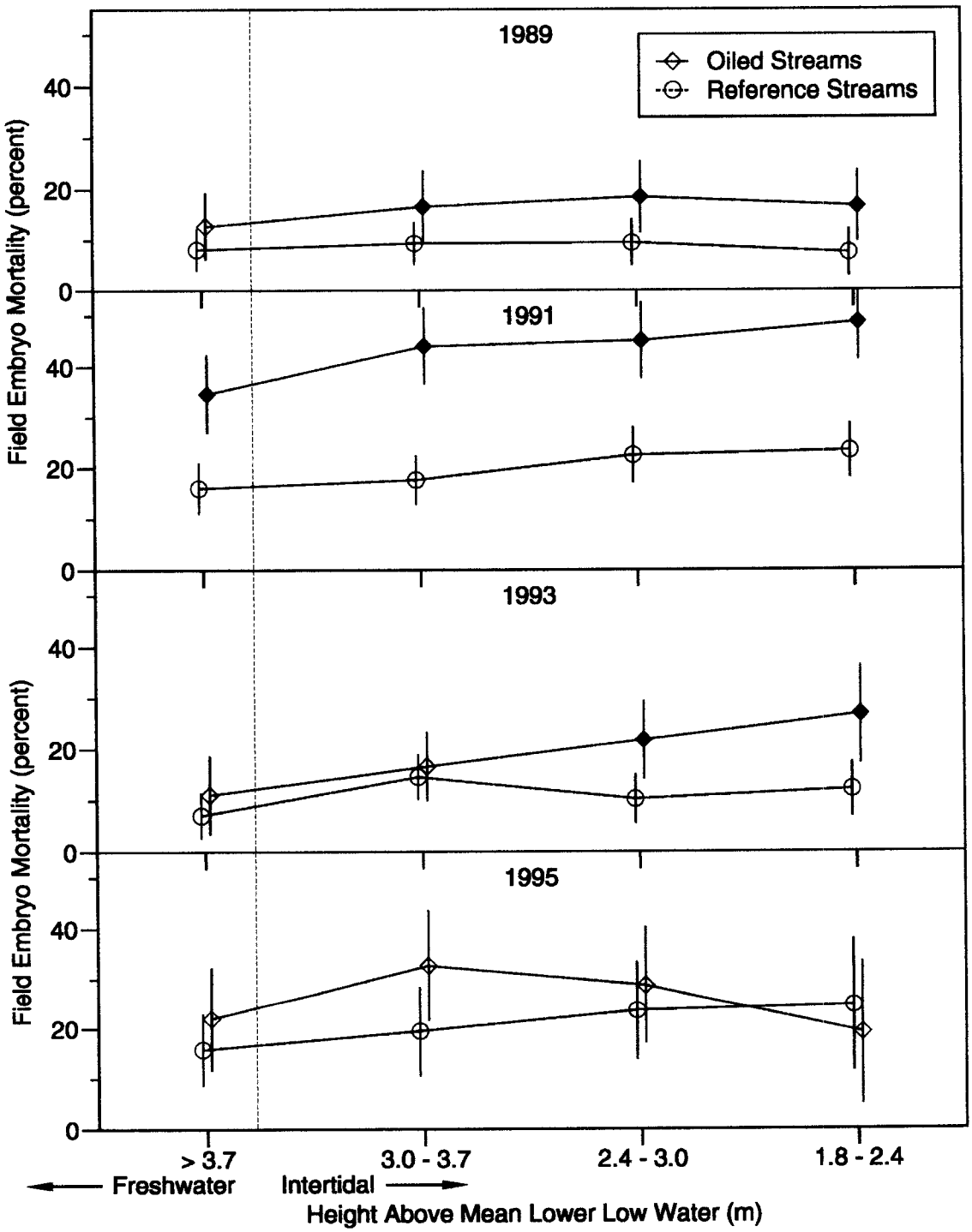


Fig. 5.28. Egg mortality in oiled streams was elevated from 1989 through 1993, four years after the oil spill (Bue et al. 1994, 1996, 1998). Mortality of embryos was significantly greater in oiled streams through 1993, but not significant in the years thereafter except 1997. Significant differences are indicated by solid symbols.

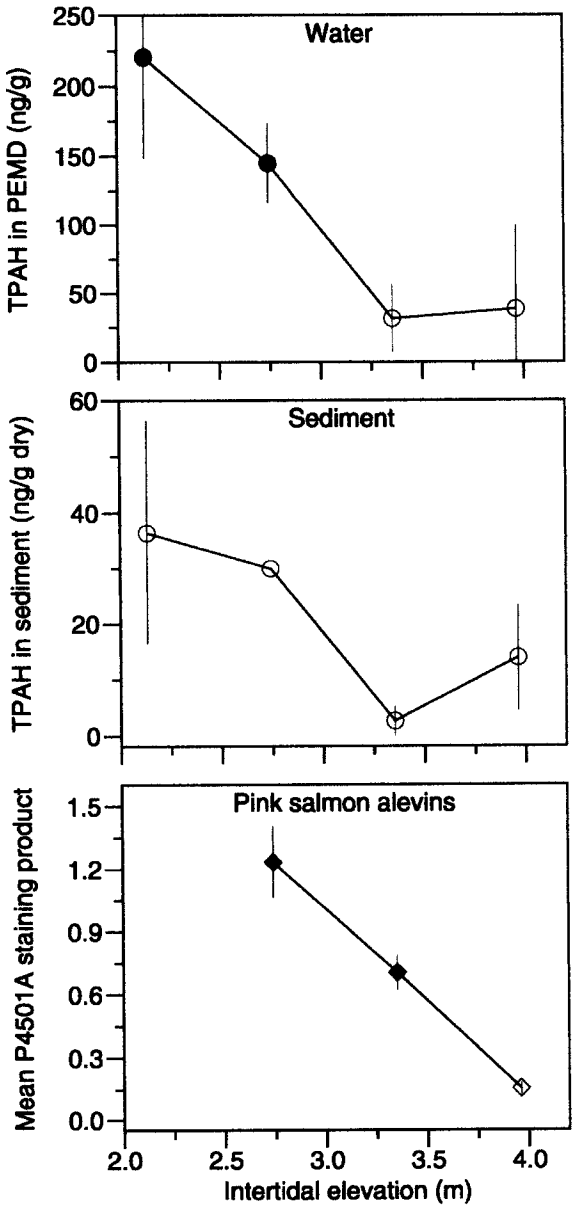


Fig. 5.29. Correspondence between PAH in water and sediment of an intertidal stream and P4501A enzyme induction in wild pink salmon alevins exposed as embryos (Carls et al. 2004). These data were collected a decade after the *Exxon Valdez* oil spill from Sleepy Creek, a stream still affected by oil: PAH concentrations in water were measured with passive samplers known as low-density polyethylene membrane devices (PEMD). The staining product is a measure of P4501A induction (intensity x occurrence). Oil was unlikely in the uppermost zone, thus it served as the reference; solid symbols highlight data significantly different from the references.

Even though oil is deposited at some distance from the stream bed, these interstitial water flows can transport PAH dissolved from subsurface oil deposits high in the intertidal zone to eggs incubating lower in the intertidal zone by gravity and a hydraulic head (Carls et al. 2003). Such an exposure mechanism was particularly relevant in the intertidal streams of PWS where the average tidal range is 3 m and most of the lingering oil was subsurface (Short et al. 2002). Pink salmon eggs and alevins were thus exposed to oil for months, which had effects later in the life cycle and apparently on the subsequent generation.

In estimating stream mortality of salmon eggs, hydraulic pumps were used to remove eggs from the stream gravel. This procedure has the potential to shock a portion of the live eggs resulting in an appearance similar to dead eggs. This may have affected some of the data. However, in a modeling study designed to retrospectively explore the accuracy of the original egg classifications, potential egg misclassification errors by Bue et al. (1996, 1998) did not explain observed survival differences between oiled and reference streams (Thedinga et al. In press). This modeling was based on experimentally determined egg misclassification rates by novice and experienced observers (Carls et al. 2004c). Estimates that potential observer bias was not sufficient to explain poorer embryo survival in oiled streams is consistent with other observations, as presented below.

The best evidence of a reproductive impairment effect on adults from oil was a controlled experiment in 1993 on pink salmon (Bue et al. 1998). Observations in 1991 of elevated embryo mortality in the freshwater sections of oiled streams above high tide, (above where the oil could have exposed incubating salmon embryos in the stream) led to questions of possible reproductive impairment of returning adults (Bue et al. 1996). Because oil contamination in this exclusively freshwater zone was unlikely, any oil-related effects would have been due to the returning adults that had been exposed to oil as embryos in 1989. This raised the possibility that returning adults had reproductive dysfunction, perhaps as a result of genetic damage as embryos.

Reproductive impairment of returning adults was tested in 1993 and 1994, when spawn originating from a series of oiled and un-oiled streams was incubated concurrently in a clean hatchery environment. In 1993, the last continuous year of an oil effect on embryo mortality rates, the embryos from adults collected in oiled streams had elevated embryo mortality rates compared to embryos from adults collected from non-oiled streams. In 1994, the experiment was repeated, but there was no statistical elevation of embryo mortality rates from the field sampling of oiled streams or from the spawn collected in oiled streams and returned to the hatchery for rearing. These results were consistent with the recovery of the oiled streams to levels that were no longer result in measurable elevated embryo mortality (Bue et al. 1998) (Fig. 5.30). Elevated oil-related mortality in the 1993 hatchery experiment paralleled elevated mortality in oiled streams observed during annual hydraulic pumping.

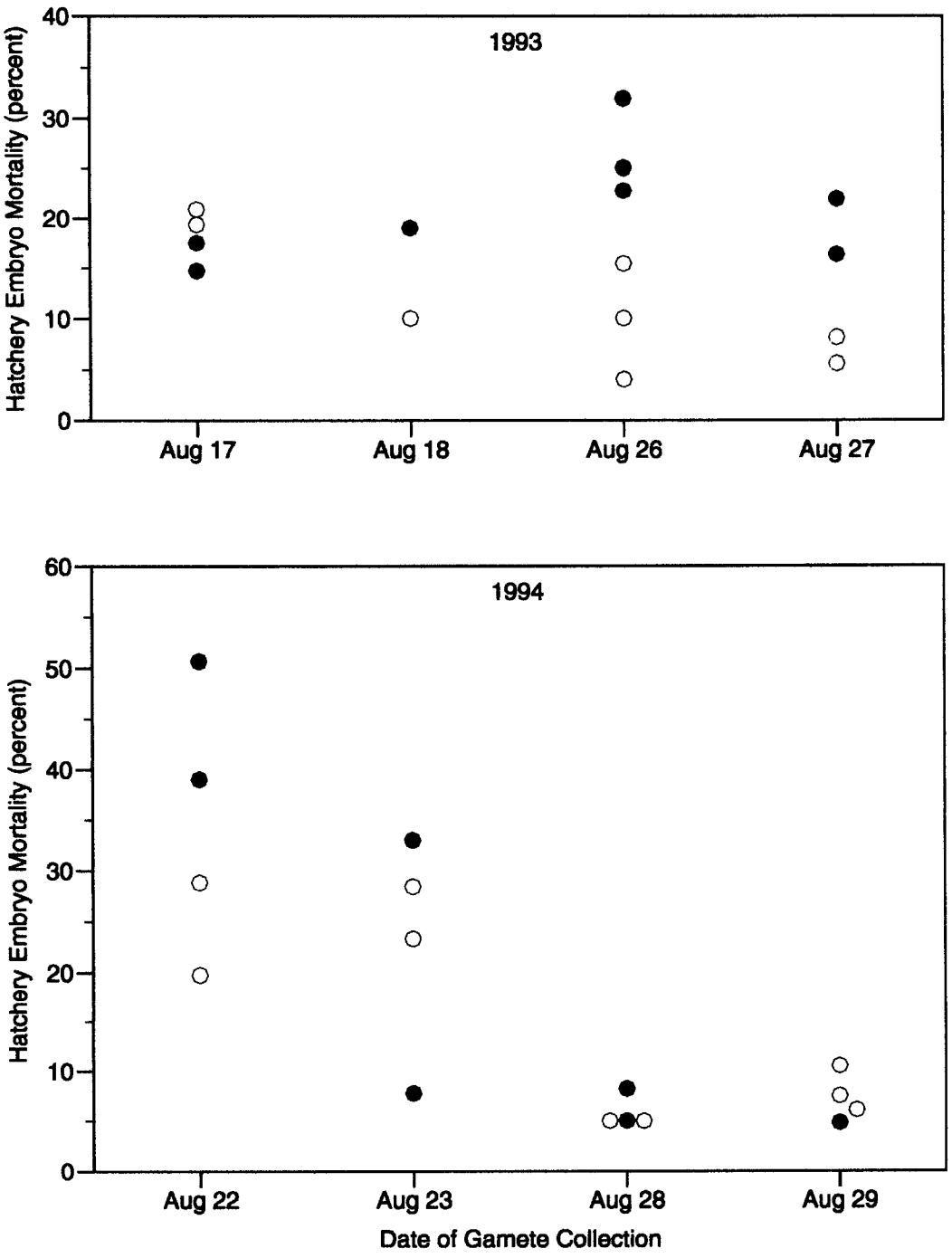


Fig. 5.30. In 1993, but not 1994, mortality of embryos produced in the hatchery from adults collected in oiled streams was higher than those in unoiled streams, which was consistent with the results of field collections of embryos from oiled and unoiled streams (Bue, 1998). Solid circles=oiled streams; open circles=unoiled streams Each data point represents approximately 27,000 eggs from 900 fish.

Thus, the embryo viability in hatchery incubators reported by Bue et al. (1998) was entirely consistent with their field observations. More importantly, the embryo viability experiment was evidence of reproductive impairment in returning adults.

V.E.3.a.3 Laboratory Studies Confirm Long-term Effects

Long-term damage from oil exposure, the effects of which appear in subsequent generations, had not previously been reported. The effects of long-term exposure to low concentrations of PAH were documented in a series of detailed laboratory studies on pink salmon embryos. These multi-generational studies were designed to explain how continued egg mortality occurred despite significant declines in oil concentrations in PWS beaches over those same years. The laboratory studies demonstrated that embryonic exposure to very low concentrations of PAH resulted in long-term effects on the morphology, reproductive capability, and marine survival of later life stages of pink salmon.

The first of these studies demonstrated that pink salmon embryos are sensitive to long-term exposure to weathered oil (Marty et al. 1997b). Larvae exposed to various levels of oil in gravel were sampled four weeks before, during, and after emergence and examined for histopathological abnormalities and induction of cytochrome P4501A. Some of the PAH on gravel dissolved into water, and uptake by eggs was mediated by water, not direct contact with oiled gravel. Significant adverse biological effects were measured at aqueous concentrations as low as 4.4 ppb. Pink salmon eggs accumulate PAH rapidly to concentrations that may exceed those in water by 9000 times (Carls et al. 2004d). Maximum total PAH concentrations in tissue likely occurred within the first two weeks of embryonic development and were controlled by the capacity of eggs to accumulate PAH and declining total aqueous PAH concentrations. Clearly the chorion, the protective membrane around these eggs, was sufficiently porous for high-molecular weight PAH to pass through and did not insulate the embryos from chemical toxicity.

Oil-related effects from exposure of embryos to weathered PAH included induction of cytochrome P4501A, development of ascities (swelling due to excess fluid retention), retarded development, and increased mortality (Marty et al. 1997b) (Fig. 5.31). The fry that emerged from oiled gravel had more yolk and hepatocellular glycogen, increased apoptosis (programmed cell death) of gonadal cells and midventral skin cells, and less food in the gastrointestinal tract than did control fish of the same age and emergence date. Histopathological problems were observed in larvae four weeks before emergence, at emergence, and 13 days after emergence. Significant gonadal apoptosis suggests a possible mechanism for the reproductive impairment observed in field-sampled fish by Bue et al. (1996). Several of these oil-related changes were indicative of premature emergence.

The long-term sensitivity of embryos was confirmed again in tests using oil-coated gravel that had been washed for nine months and sat in the open for an additional three months prior to exposure tests (Heintz et al. 1999).

Fig. 5.31. Ascities, a swelling due to excess fluid retention, was frequent in larvae exposed to weathered crude oil as embryos; (top) control, (bottom) oil-exposed larva (Marty et al. 1997).

Eggs exposed from fertilization through hatching to an initial PAH concentration of 1.0 ppb of this highly weathered oil resulted in significantly greater mortality than in controls. Sublethal effects included higher incidences of spinal deformities, opercular hypoplasia, and ascites. The biological effects were the same whether embryos were incubated in oiled gravel or in water effluent from oil gravel, demonstrating that PAH transport was via water and that contact with oiled gravels was not necessary to produce the effects. The study results suggested that the low concentrations of weathered oil produced by the stream-side deltas could cause reproductive effects observed in PWS. Comparison of these experiments to earlier works (Moles et al. 1979) demonstrate that pink salmon embryos are far more sensitive to long-term exposure to low concentrations of heavily weathered PAH than they are to short, high-concentration exposures of water-accommodated fractions of low-molecular-weight aromatic hydrocarbons.

Despite the inherent low solubility of PAH in water, toxic hydrocarbons leached from the gravel can effectively be sequestered in internal tissues of these lipid-rich embryos. Transfer of hydrocarbons from water to embryo is the result of kinetic processes rather than solubility. The kinetics of the transfer process results in more rapid accumulation of PAH by smaller embryos, such as Pacific herring because the surface area per unit volume of eggs increases inversely with the egg radius and the uptake rate is directly proportional to this surface-to-volume ratio. Comparison of the adverse effects caused by exposure to less-weathered and more-weathered oil suggests that smaller PAH (e.g., naphthalenes) contributed little to the toxicity. Consistent with this observation, comparison of three toxic mechanisms (narcosis, aryl hydrocarbon receptor agonism, and alkyl phenanthrene toxicity) in Pacific herring and pink salmon suggested alkyl phenanthrenes explained most of the toxicity (Barron et al. 2003). The concentration of large-molecular-weight PAH, which persist longer in the environment, was similar in both less- and more-weathered exposures, with similar toxic outcomes. Developing fish embryos are clearly at risk from environmentally persistent PAH.

Sublethal exposure of embryos to oil can also have delayed effects on growth and marine survival (Heintz et al. 2000). In experiments designed to test this possibility, pink salmon embryos were exposed to water contaminated with four different concentrations of PAH, again produced by percolation of water through columns of gravel coated with different amounts of weathered Alaska North Slope crude oil. Most surviving alevins appeared healthy and fish from each exposure group were transferred to saltwater net pens for feeding. A delayed effect on growth was measured in juvenile salmon that survived embryonic exposure to an aqueous total PAH concentration of 18 ppb. Over 200,000 fry from the experiment were released directly to the marine environment with coded-wire tags. Tag-bearing fish were counted upon return to their southeast Alaska natal stream two years later.

Overall marine survival rates were 0.8% (in the 19 ppb exposure group), a nearly 40% reduction compared to the 1.3% marine survival rate in control fish (Fig. 5.32). Marine survival in the 5 ppb exposure group was intermediate, 1.1%, and significantly different from the control group. In a subsequent experiment in 1996, relative marine survival in fish exposed as embryos to 14 ppb was less than half that of unexposed fish. The pink salmon experiments, both field and laboratory, demonstrate that embryonic exposure to PAH at low part-per-billion concentrations impairs development. Embryonic toxicity was not a narcotic effect associated with acute exposure, but the result of other mechanisms operating during embryonic development. Reduced growth and survival were noted long after exposure had stopped and long after PAH was eliminated from tissue, demonstrating that long-lasting or permanent damage had occurred. Roy et al. (1999) described mutagenic effects of oil on pink salmon embryos exposed to weathered *Exxon Valdez* oil, confirming that weathered oil is capable of causing genetic damage. Marty et al. (1997b) argues that abnormal histology of gonads in pink salmon alevins after lengthy low-level exposure to PAH might be sufficient to explain subsequent reproductive damage.

The cumulative effect of these injuries could decrease population productivity. Such decreases would be from the interplay of defects acquired during embryonic exposure, the increased energy needed for hydrocarbon metabolism (cytochrome P4501A is induced in pink salmon embryos at total PAH concentrations as low as 1-4 ppb), and delayed effects that decrease growth and reduce survival after exposures have ended. Not only are fewer fry produced from a set of exposed eggs (Bue et al. 1996; Heintz et al. 1999), but when coupled with a potential 40% reduction in marine survival, a modest-sized run that produces ten million eggs would produce 46,000 fewer adult fish if those eggs were exposed to 19 ppb total PAH (Rice et al. 2001). Such concentrations were still present in the interstitial waters of a number of salmon streams in 1995 (Murphy et al. 1999). Fortunately, by 1999, available evidence indicated that the majority of pink salmon habitat was recovering (Carls et al. 2004a).

V.E.3.b Mussels

The long-term exposure of mussels (*Mytilus trossulus*) to *Exxon Valdez* oil and the oiling of the intertidal habitat they occupy exposed nearshore predators to low levels of oil for more than a decade. In the aftermath of the spill, intertidal oil was retained longer than expected, thus oil was biologically available for long periods, exposing both prey and predators to oil (Fig. 5.33). Following the *Exxon Valdez* oil spill, mussel beds on sediment were recognized as vulnerable, yet valuable, habitat and were protected from cleanup activity (Babcock et al. 1996). Mussels stabilize sediment, provide physical structure for a host of intertidal organisms, and are food for a variety of invertebrate and vertebrate predators. There were no reports of catastrophic mussel loss due to oiling (Gilfillian et al. 1995a,b) and aggressive shoreline cleanup procedures such as hot-water washes and mechanical displacement of colonized substrate (Mearns 1996) were avoided on shores with extensive mussel beds (A. Weiner, personal communication). The slow natural rates of hydrocarbon loss from these set-aside habitats was not anticipated, placing the mussel community and consumer species dependent on it at risk from chronic hydrocarbon exposure (Babcock et al. 1996; Carls et al. 2001).

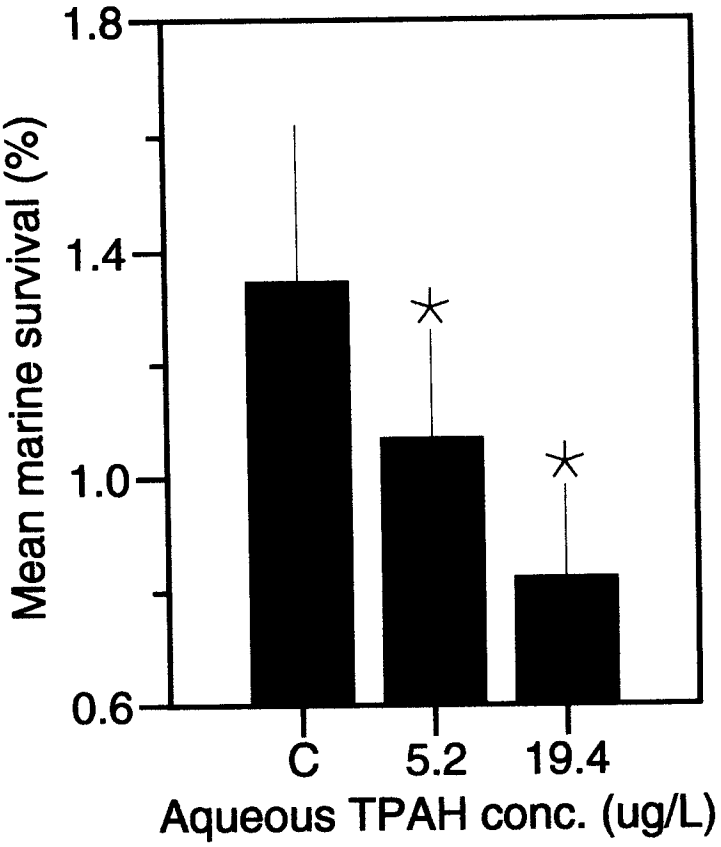


Fig. 5.32. Sublethal exposure of embryos to oil had delayed effects on marine survival. Asterisks indicate significantly reduced survival (from Heintz et al., 2000).

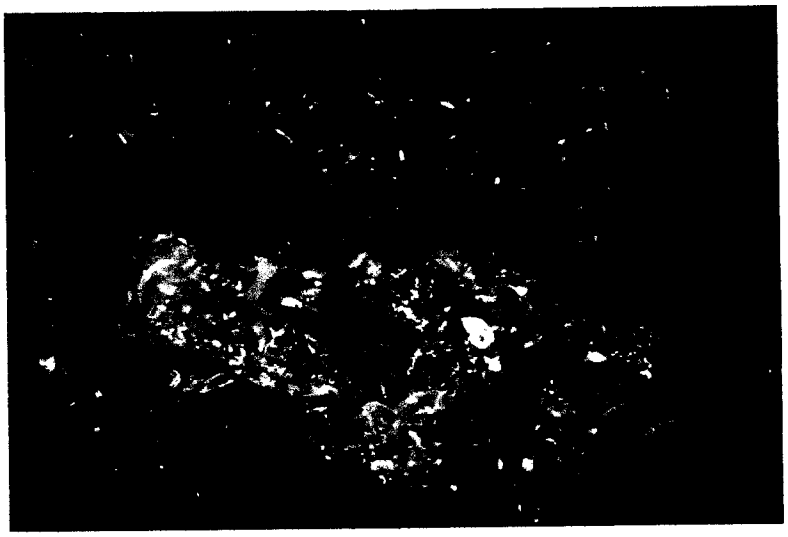


Fig. 5.33. *Exxon Valdez* oil persisted for many years in mussel beds (Carls et al. 2001).

The slow leakage of residual oil from uncleaned mussel beds along with oil from other areas of western PWS had biological consequences (Fig. 5.34). In a study of heavily oiled mussel beds, oil was estimated to persist in the sediment underlying the beds for five to 30 years (Carls et al. 2001). Physiological stress due to chronic oil exposure was observed in mussels and other bivalves (*Protothaca staminea* and *Mya arenaria*) five to 11 years after the Exxon spill (Thomas et al. 1999; Fukuyama et al. 2000; Downs et al. 2002).

Bivalve molluscs are good indicators of local petroleum hydrocarbon contamination because of their sessile nature and high capacity to bioaccumulate hydrocarbons with little effect on metabolism (Fossato and Canzonier 1976; Vandermeulen and Penrose 1978; Stegeman 1985; Livingstone et al. 1989). Mussels were often sampled to determine site-specific biological availability of EVOS oil. For example, Brown et al. (1996a,b) used PAH concentrations in mussels as surrogates for Pacific herring egg exposure and Wertheimer et al. (1994) compared hydrocarbons in mussels to those in migrating pink salmon fry to interpret their results on sub-lethal effects on fry. The National Mussel Watch Program exploits the ability of these bivalves to act as environmental samplers (O'Connor 2002).

Habitat type determined availability of oil to the intertidal community and in some habitats the oil remained for years. Persistence of oil in mussels was short (months) on shorelines not coated by oil and where mussels were only exposed to oil dissolved in water (Carls et al. 2002). Persistence of oil in mussels was long (six to ten years or more) on beaches where oil remained in soft sediment (Babcock et al. 1996; Carls et al. 2004b; Carls et al. 2004c). In a study of heavily oiled beds, total PAH concentrations in mussels from 10 of 23 beds sampled remained above background concentrations in 1995 (Carls et al. 2004b). A decade after the spill, total PAH concentrations in mussels were indistinguishable from background in 11 of 12 of these beds (Carls et al. in press). This indicator of declining biological availability of oil, hence habitat recovery, occurred despite lingering oil in sediment. The general decline of oil concentrations in sediment shows that this reservoir of oil is being depleted, allowing habitat recovery (Carls et al. 2004b) (Fig. 5.35).

The persistence of oil in intertidal sediment, mussel beds, and associated fauna may explain the decadal exposure of predators such as Harlequin ducks (*Histionicus histionicus*) and sea otters (*Enhydra lutris*) to hydrocarbons. Biochemical indicators of exposure to PAH persisted in sea otters and Harlequin ducks inhabiting oiled areas for at least 12 years (Bodkin et al. 2002; Esler et al. 2002). Both species forage in nearshore environments and exposure to oiled sediment during foraging and consumption of oil-exposed fauna are likely the principal routes of exposure for these predatory species. We do not know the relative contributions of oil in mussels on other prey species or oil in sediment to predator exposure, but recognize that consumption of oiled mussels is likely not the sole route of exposure because concentrations in mussels generally declined to background levels before exposure ceased in predators.

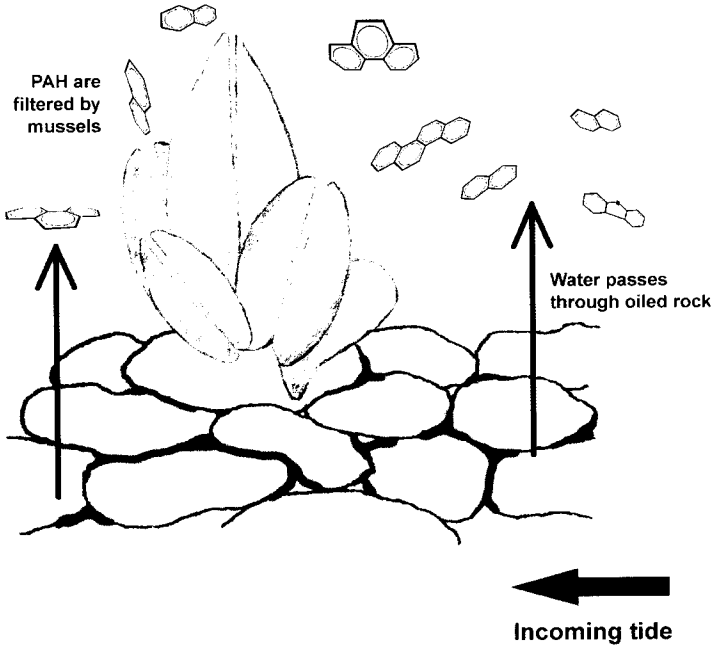


Fig. 5.34. Possible mechanism for movement of oil trapped in sediment into mussels.

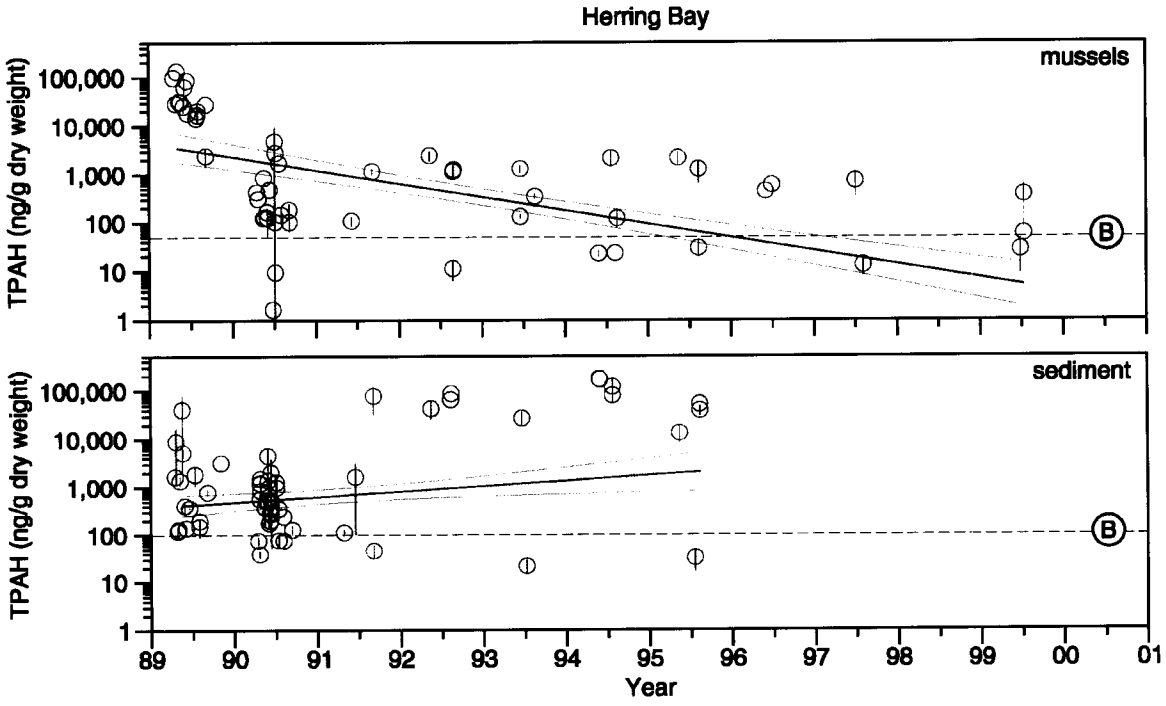


Fig. 5.35. Total PAH concentrations in mussels typically dropped in the decade after the *Exxon Valdez* spill, as was the case in Herring Bay, where oil retained by intertidal sediment served as the contaminant reservoir. Data illustrated are means ± standard error; horizontal dashed lines indicate estimated background concentrations.

The observation that remaining *Exxon Valdez* oil persisted in intertidal sediment and was toxic in the decade after the spill is consistent with other spill experience. In a review of seven well-studied oil spills, Teal and Howarth (1984) conclude that oil effects can persist for at least 6-12 years in sediment. Long-term chronic exposure to oil can reduce fitness, growth, and reproduction, and result in increased genetic abnormalities, and susceptibility to disease.

We conclude that in low-energy habitats oil may not degrade, rather be slowly released, affecting the associated species for a decade or more. Chronic, long-term exposure to oil did not cause the massive die-offs associated with oil coating, but rather apparently exerted more subtle sublethal effects on the health, recovery, and populations of resident species.

The lesson from the *Exxon Valdez* spill is that chronic non-point sources of PAH entering the waters around urban areas may exert similar effects and explain the incremental erosion of community structure and depletion of species. Concentrations of PAH up to 9 ug/L have been reported for urban watersheds (Doong and Yin, 2004), which exceed the concentrations having effects on pink salmon and Pacific herring from the *Exxon Valdez* oil spill.

V.E.3.c Otters and Harlequin Ducks

Sea otters (*Enhydra lutris*) and Harlequin ducks (*Histrionicus histrioncus*) have not fully recovered from the oil spill as of 2003, long after many species have been declared either recovered or on the road to recovery. In addition to suffering heavy losses in 1989, populations of these two species in the oiled areas of PWS have been continually exposed to oil for up to 13 years after the spill. Sea otters still have not regained pre-spill abundances in the areas around Knight Island in PWS and population effects on Harlequin ducks are likely still—not only from failure to recover the loss of over 1000 individuals in 1989, but also poor over-winter survival of adult females in the oiled western PWS (Esler et al. 2000, 2002). The poor recovery is likely a direct result of their foraging habits; these species forage on intertidal invertebrates in areas that retained oil in the sediments many years after the spill. Based on differences in physiology, enzyme biomarkers, and abundance between animals in oiled and unoiled areas, it is probable that long-term population repercussions were the result of chronic oiling of both sea otters and Harlequin ducks.

There is strong evidence that continued exposure to oil was the primary factor limiting the recovery of these species over the last 10-15 years. The continued, though declining, persistence and bioavailability of oil in nearshore environments favored by these species coupled with continued induction of liver enzymes (P4501A) used to detoxify oil, indicate that the low survival rates of both species are the result of chronic oil exposure. As of 2003, P4501A induction levels in oiled areas declined to those of reference levels for Harlequin ducks (D. Esler, personal communication) and are approaching parity for sea otters, which may signify imminent sea otter population recovery (B. Ballachey and J. Bodkin personal communications).

V.E.3.c.1 Sea Otters

Populations of sea otters in PWS were reduced radically by the *Exxon Valdez* spill and did not recover as quickly as expected or hoped (Fig. 5.36). Recovery of sea otter populations averaged about 4% per year after 1993 throughout the western (oiled) portion of the sound (Bodkin et al. 1999). Between 1993 and 2000, the number of sea otters in the spill area increased by about 600 to nearly 2700 (Bodkin et al. 2002), a similar rate of increase. This contrasted sharply with the 10% recovery rate experienced after the Russian trade in sea otter pelts stopped in the 19th century (Bodkin et al. 1999). The population at a non-oiled location in the Sound, Montague Island, doubled in the three years between 1995 and 1998. In contrast, populations at the heavily oiled Knight Island were half the size they had been prior to the spill and no signs of recovery were evident for at least a decade after the spill (Dean et al. 2000). Modeling indicated survival of otters, including those born after 1989 in oiled portions of the Sound, declined following the spill (Monson et al. 2000). While reproduction was not impaired following the spill, juvenile mortality was relatively high (Monson et al. 2000), and a disproportionately high number of prime-age sea otters died in western PWS after the spill than before (Monson et al. 2000).

Oil exposure, food supplies or demographic factors are postulated factors for the lack of recovery of sea otters at Knight Island and all these factors were evaluated for a suite of apex predators, including the sea otter, from 1996-1998 during the Nearshore Vertebrate Predator study (Peterson and Holland-Bartels 2002). Food consumption and foraging efficiency of sea otters in the vicinity of Knight Island and at the control site at Montague Island were investigated and these measures were higher at Montague Island (Dean et al. 2002). Additionally, condition of sea otters at Knight Island was equivalent to or better than the condition of otters at Montague. So, food limitation has been discounted as the primary factor constraining recovery (Bodkin et al. 2002).

What did differ between the previously oiled and unoiled sites was the amount of residual hydrocarbons present in the prey of sea otters. Ingestion of hydrocarbon-laden prey continued for several years after the offshore waters were free of hydrocarbons. Mussels and clams, which filter their food from the water, concentrate hydrocarbons in their tissues. The same slow metabolism that protects these bivalves from dying quickly following oil exposure also results in the organisms retaining and only slowly purging their systems of the hydrocarbons. The subsequently high concentrations of oil compounds found in clams persisted until at least 1996 (Fukuyama et al. 2000; Peterson 2001). The sediments in these mussel bed and eelgrass foraging areas also retained hydrocarbons for years after the spill (Jewett et al. 1999), providing another avenue of exposure for some of the sea otters which foraged in the intertidal zone at high tide. In contrast, in 1997 and 1998, the river otters that foraged for fish along the same oiled shoreline showed none of the biomarkers of exposure displayed by the sea otters.

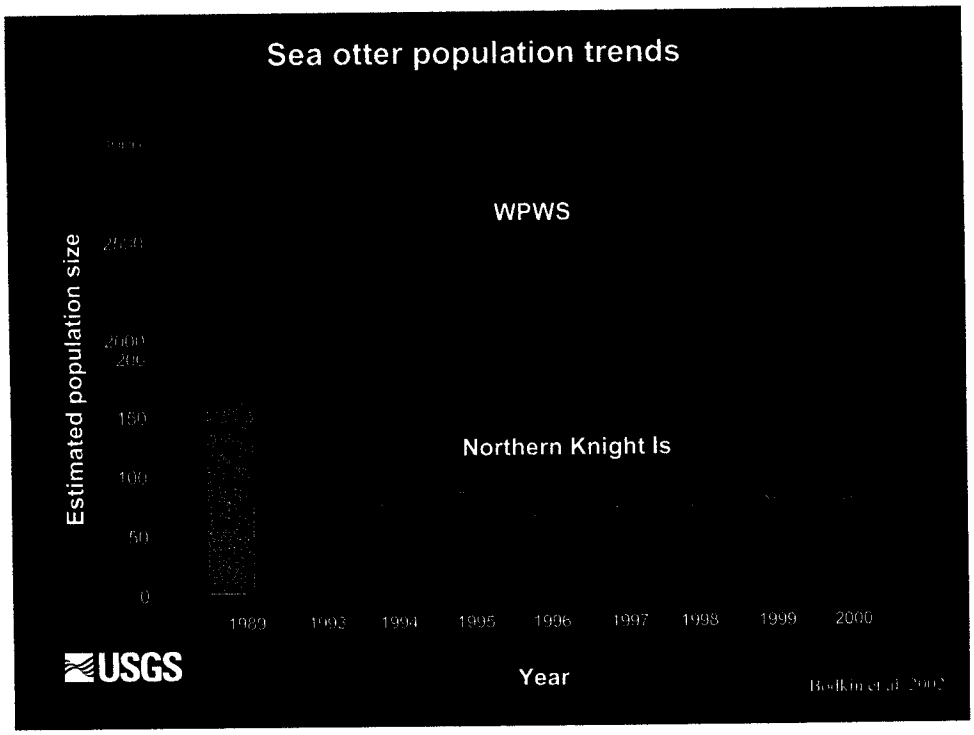


Fig. 5.36. Sea otter population sizes in Western Prince William Sound (WPWS) and the heavily oiled northern Knight Island. The estimated size pre-spill 1989 is indicated by the green bar.

Differences in foraging behavior and food sources may explain the much larger and longer lasting differences in oil exposure biomarkers in sea otters. Fish can be retrieved and eaten by river otters without foraging in oiled sediments and their fish prey may metabolize any hydrocarbons before they reach the river otter, but sea otters have to dig in sediment to capture most of their invertebrate prey and the slow metabolism of oil in the invertebrates would transmit contaminants to the sea otters.

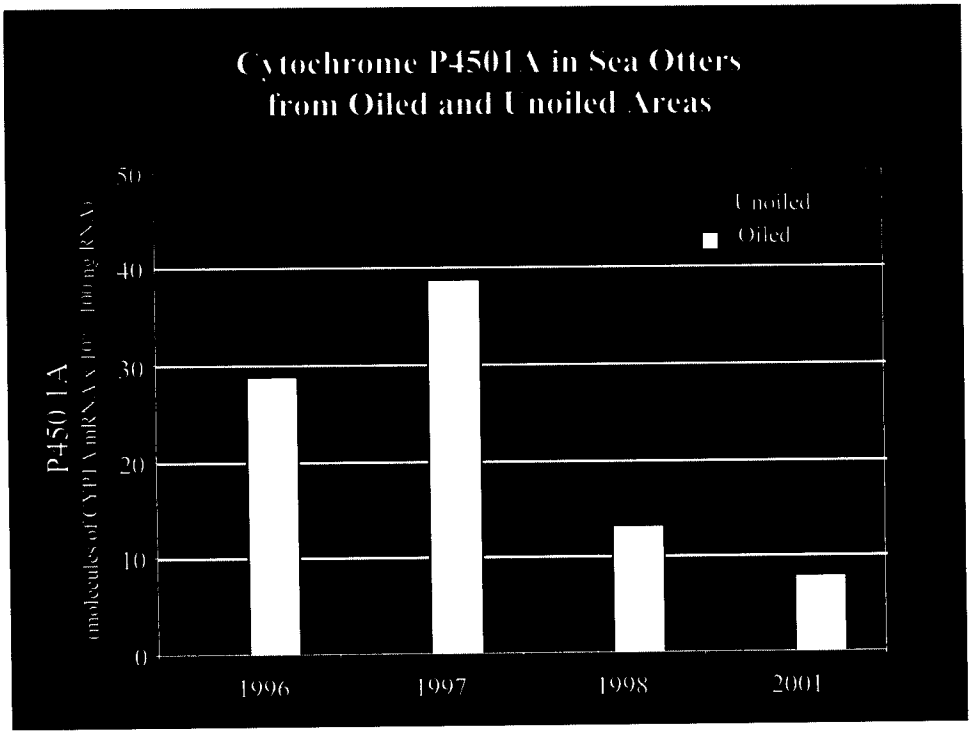
The poor survival of sea otters born after the spill due to chronic oil exposure was evaluated by the incidence and degree of induction of the hydrocarbon metabolizing enzyme CYP1A, comparing sea otters from the heavily oiled northern Knight Island with those from the Montague Island reference area. The presence of the enzyme indicates recent exposure to aromatic hydrocarbons. The results of these comparisons confirmed a continuing exposure to oil between 1996 and 1998 (Bodkin et al. 2002), and this difference continued through 2000 (B. Ballachey personal communication) (Fig 5.37). There is an apparent downward trend as values in 2001 were substantially lower in Knight Island sea otters than they were in 1997-1998 and the differences between sea otters between Knight Island and Montague Island were narrowing. Enzymes indicative of liver disease, particularly gamma-glutamyl transferase (GGT) were also elevated in serum from otters in the western Sound (Ballachey et al. 2001). During the period 1992-2001, over 30% of sea otters from oiled areas had increases in GGT whereas otters from the unoiled areas averaged 10%. Furthermore, in 2001 livers of captured live sea otters were examined by endoscopy, and 3/15 otters from Knight Island had visible liver pathologies, with at least two of them having damage sufficient to impair survival, according to two veterinarians doing the examinations.

Cascade Effects: Sea Otters

One of the potential effects of otter mortality and slow population recovery is the removal of predation pressure of a keystone species on sea urchins. If the sea otter population is too small to control urchin population size, the urchins are fully capable of overgrazing kelp beds. Dean et al. (2002) documented both a 50% reduction in sea otter populations and the subsequent increases in urchin populations following the spill. Kelp and other submerged aquatic vegetation in Alaska provide a complex structure that acts as protective habitat for juvenile fishes and invertebrates (Johnson et al. 2003). This in turn affects the seabirds and fish that prey on the residents of these kelp shelters (Estes and Duggins 1995). The well-documented and complex ecosystem dynamic among otters, urchins, and kelp could easily have been disrupted by the loss of sea otters during the spill (Peterson et al. 2003). These kinds of indirect cascades through the ecosystem are perhaps the most elusive to quantify but may have significant ecological consequences. The loss of apex predators caused directly by an oil spill can in turn cause a cascade of secondary indirect effects on the ecosystem.

V.E.3.c.2 Harlequin Ducks

About 1000 harlequin ducks died in 1989 by direct exposure to the oil slick (Piatt et al., 1990), and persistence of oil in their habitat may have contributed to poor survival for a decade or more. Harlequin ducks feed directly in the intertidal zone (Fig. 5.38) where most of the initial oil was stranded and where oil has persisted for more than



5.37. A measure of cytochrome P4501A activity (from mRNA) in livers of sea otters from unoiled Montague Island and oiled northern Knight Island from 1996 through 2001.

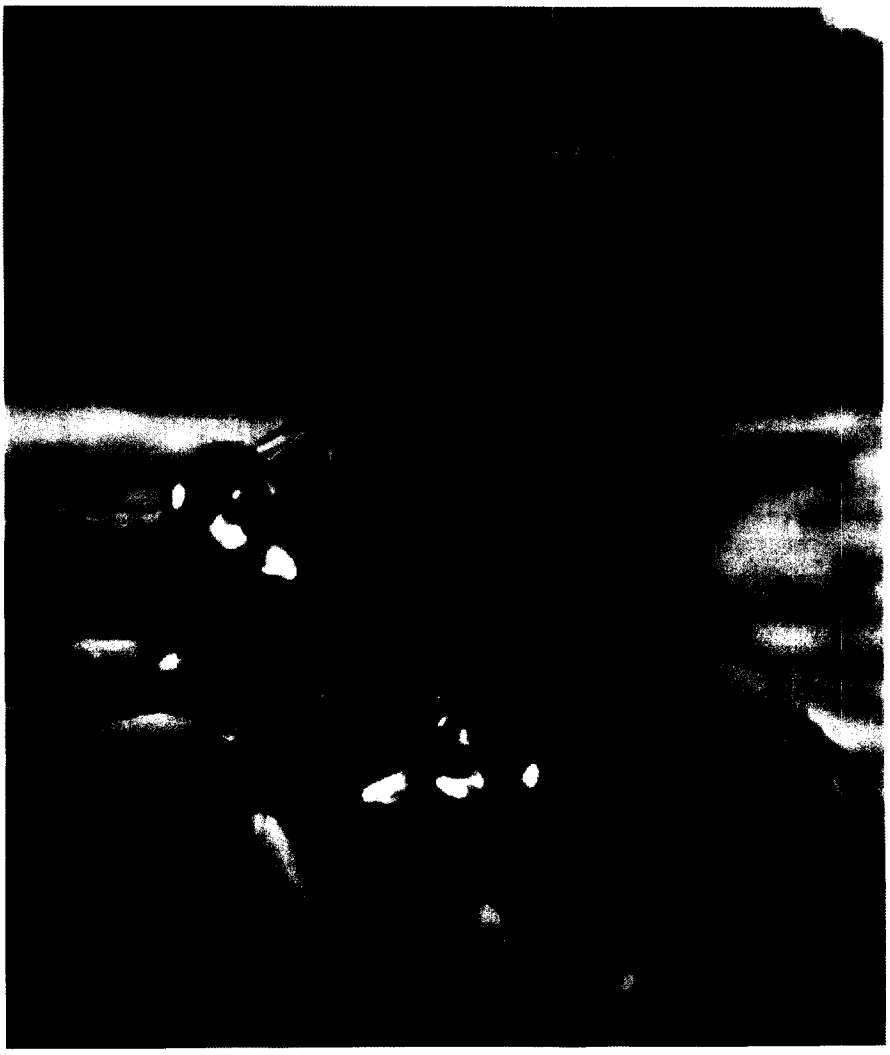


Fig. 5.38. Researcher with two Harlequin ducks captured in Prince William Sound.

a decade. Radio-tracking of overwintering adult female Harlequin ducks from oiled and unoiled sites demonstrated lower overwinter survival among the birds from heavily oiled sites (Fig. 5.39). During the winters of 1995-1996 and again in 1997-1998, an average of 22% of the birds from the heavily oiled shoreline sites of Green and Knight Islands died as opposed to only 16% of the birds from the unoiled Montague Island (Esler et al. 2002). This difference seems small but has large implications for the populations. Survival was lowest during mid-winter, December through January, in these oiled areas. The study examined the relative importance of intrinsic geographical differences and oiling history, concluding that continued exposure to oil was the most likely mechanism affecting survival. Densities of Harlequin ducks along previously oiled shoreline declined at an annual rate of 5% during this time while continuing at stable population levels along unoiled shoreline (Esler et al. 2002).

Harlequin ducks, along with many other species of shorebirds, feed on a variety of intertidal and subtidal invertebrates such as barnacles, mussels, crabs, small crustaceans, and a wide variety of snails in the winter. All these species of benthic invertebrates are intimately associated with the sediment and do not rapidly metabolize hydrocarbons. Ingestion of contaminated prey is the most probable explanation for continued exposure of these sediment-foraging ducks. Other species of seabirds also showed signs of persistent oil exposure. For example, Barrow's goldeneye, a seaduck that feeds on intertidal mussels, declined in abundance in oiled beach areas (Day et al. 1997).

Proof that ducks, including Harlequin and Barrow's goldeneye, were indeed exposed to residual oil in their habitat again is biochemical. As in other species, the oil detoxifying enzyme cytochrome P4501A was induced, indicating continued oil exposure of these species through 1998. Induction of P4501A in livers of wintering Harlequin ducks were 2.8 times higher in samples from two heavily oiled Knight Island sites than from two unoiled Montague Island sites (Trust et al. 2000). Other contaminants potentially responsible for this enzyme activity are various polychlorinated biphenyls (PCBs), but these were below detection limits at all the sites, demonstrating that differences in enzyme induction were not due to the presence of confounding contaminants.

Another clue to the specific route of contamination in sea ducks is comparison to the dietary habits and P4501A induction in another seabird, the pigeon guillemot. In pigeon guillemots, elevated levels of P4501A were restricted; levels were not elevated in their chicks. The chicks feed only on fish whereas the adults also include shallow water benthic invertebrates in their diets (Golet et al. 2002). This indicates that sea ducks foraging in shallow sediments in the winter must either be directly exposed to oil-laden sediment or to oil in their prey.

However, by 2002, overwinter survival rates and P4501A levels in Harlequin ducks in oiled and unoiled areas converged and were no longer significantly different (Esler personal communication).

V.E.4 Indirect Interactions

The direct mortality of so many animals and plants after this massive spill had indirect effects on other species. All animals depend on other species, as food, as prey or for

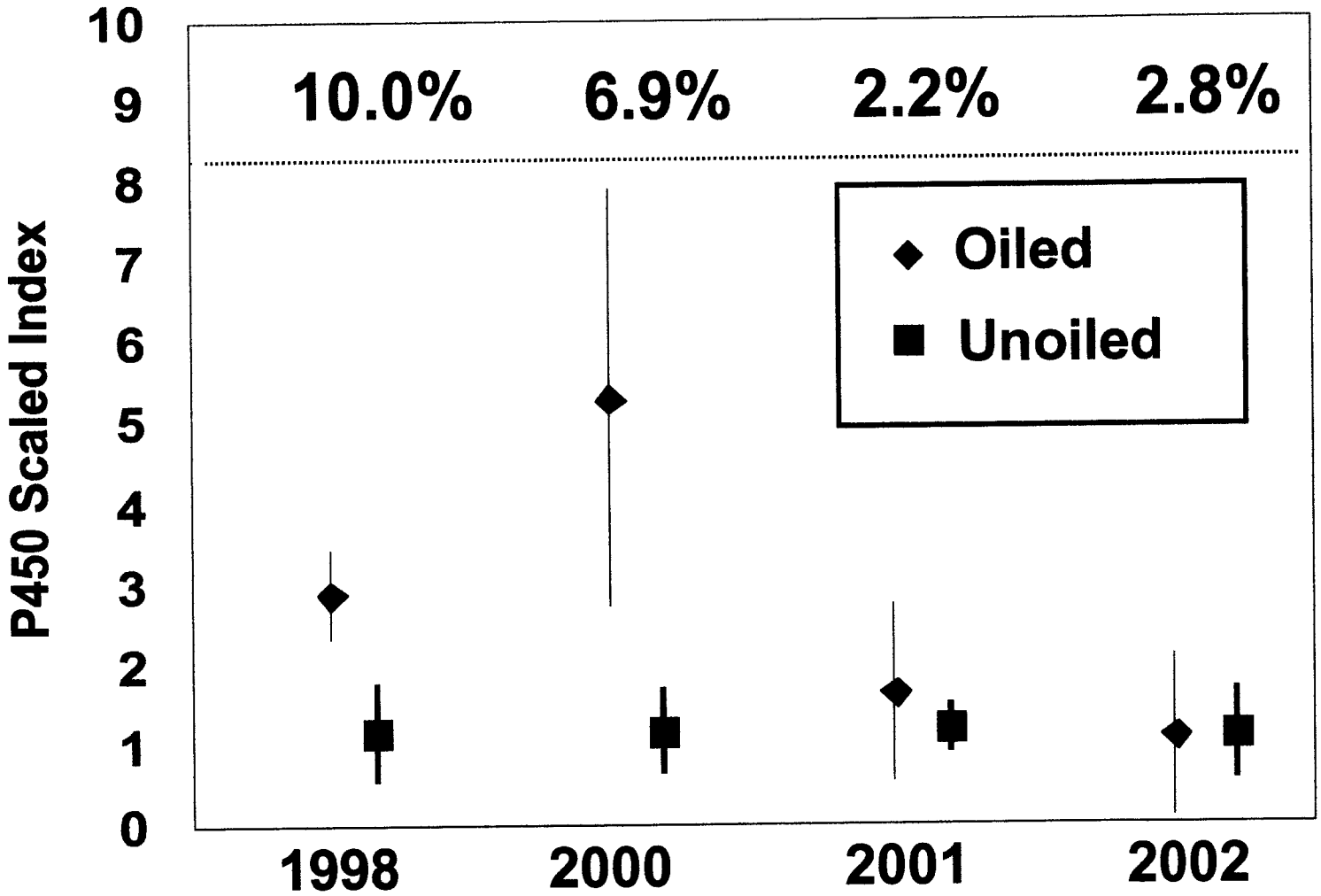


Fig. 5.39. Scaled P4501A values in livers of harlequin ducks from 1998 through 2002 compared to overwinter mortality rates (percentages at the top of the graph).

shelter. The reduction in primary or secondary consumers, reduced abundance of a top predator, or a change in competition for limited resources among predators sharing a common prey item are the most common ecological interactions. This spill that differentially affected plants and animals would induce a cascade of indirect effects as well.

The removal of keystone species such as sea otters, which so vigorously crop sub-tidal invertebrates, or the common brown intertidal algal species *Fucus*, which provides shelter for other invertebrates, has cascading effects. Indirect effects can be as important as direct trophic interactions in structuring communities. Cascading indirect effects are delayed in expression because they are mediated through changes in an intermediary. Perhaps this was nowhere more evident than in the decade-long recovery process of the rocky shorelines where changes in living habitat altered the ecosystem for the residents of these intertidal areas (Peterson 2001).

The rockweed, *Fucus* provides the primary habitat for the mollusks of PWS. Dramatic initial loss of this cover following the spill and subsequent cleanup triggered a cascade of indirect effects. Freeing the space on the rocks and the losses of important grazers such as limpets and periwinkles and predators such as whelks combined to promote initial blooms of ephemeral green algae in 1989 and 1990 and an opportunistic barnacle in 1991 (Peterson 2001). Absence of structural algal canopy leads to declines in associated invertebrates and inhibited recovery of *Fucus* itself. The recruits of rockweed plants require the cover provided by adult plants. Those *Fucus* plants that settled on the shells of barnacles became dislodged during storms because the attachment to the barnacle is not as permanent as it is to rocks.

After apparent recovery of *Fucus*, previously oiled shorelines exhibited another mass rockweed mortality in 1994, a cyclic instability probably caused by simultaneous senility of a single-aged stand (Driskell et al. 2001). The general sequence of succession on rocky intertidal shores extending over a decade after the *Exxon Valdez* oil spill closely resembles the dynamics after the *Torrey Canyon* oil spill in the UK (Hawkins and Southward 1992). Expectations of rapid recovery based on short generation times of most intertidal plants and animals proved naïve, rather, it should be realized that interspecific interactions can lead to a sequence of delayed effects for years.

The loss of more than 50% of sea otters around Knight Island in PWS apparently resulted in a greater proportion of large urchins, although the lack of pre-spill data from the oiled and unoiled sites makes the cause of this difference somewhat uncertain (Dean et al. 2000).

Fleeger et al. (2003) reviewed 150 papers on indirect effects of contaminants, including three on oil in the marine environment. In addition to altered trophic cascades and changes in competition, they identified changes in foraging behavior, such as reduced predation rates and increased susceptibility to predation. Noted were changes in habitat use as species abundance declined and the implications when multiple stressors were present. Although the authors admit that indirect effects of contaminants are more rare in marine pelagic environments than in freshwater environments due to fewer trophic

cascades, this too may be a function of the difficulty in studying the marine ecosystems. Certainly changes in habitat utilization have been noted for benthic fish and invertebrates when oil is present (Pinto et al. 1984; Moles et al. 1994; Moles and Stone 2002), potentially increasing the crowding and competition in the remaining habitat. As we have seen, indirect and cascade effects did occur following the *Exxon Valdez* oil spill.

V.F. Recovery and injury revisited

Everyone that remembers the spill wants to know if the injuries have healed and if the ecosystem is recovered. The answer to this question is not simple, and it is in fact one of the main reasons that this report was written. To understand the changes brought by the spill over the hundreds of kilometers in 1989 and lasting in some form for up to 15 years, one has to understand how this continuously dynamic ecosystem might change from other forces. Recovery of the ecosystem immediately raises the questions of the initial toll from the spill and the continuing effects. To the extent that we do not know how the system changes and in response to what forces, we have difficulty establishing the initial effects precisely and, likewise, difficulty in declaring recovery.

Yardstick for Recovery.
The *Exxon Valdez* Trustee Council faced a difficult task in reporting to the citizens of Alaska and the rest of the country on the recovery from the spill. They had to choose one of two possible standards "Recovery is the return to pre-spill population numbers" or "Recovery is the attainment of population levels that would have been there if the spill had never occurred" These are quite different concepts philosophically. The first concept ignores the fact that the ecosystem would have changed for other reasons even without the spill. But if there are defined conditions from before the spill, e.g., counts of common murres at the Barren Islands, this provides an easily defined target. The latter concept is more logical and implicitly allows for ecosystem change, but is practically impossible to determine—we have no tools to do so. In the end the EVOSTC used both definitions in not entirely consistent ways.

We can appreciate these difficulties by reference to Appendix A, which summarizes the major studies providing evidence of injury for each "major" species. Nearly all these species appeared on and "Injured Species List" established by the *Exxon Valdez* Trustee Council in 1994 and updated in 1996, 1999 and 2002. For each of the species on this list we discuss the evidence for injury and recovery and the major uncertainties.

Bald eagles--After the spill, 151 bald eagle carcasses were found, mainly on beaches of PWS. The number of actual eagles killed is not well known and could be as few as 250 or as many as 900 (Bowman et al. 1995). Since eagles dying of natural causes tend to end up in heavily forested areas, if one assumes that eagles dying from oil exposure also die in a similar fashion, then mortalities may approach the upper end of this estimate. That is, for every eagle easily found (e.g., on the beach), there are eight or nine that died in the woods. To the extent that spill mortality results in dead carcasses on beaches, then the lower end of this range is a better estimate. Population censuses are of limited value in establishing injury as previous estimates were made mainly in PWS only, they were based only on "white-headed" adults, the estimates were several years old and they likely

had a precision of about 20%, greater than the likely proportion of adult birds killed by the spill. A life table model predicted a replacement of the lost members of the population by 1994 (Bowman et al. 1995). Post-1989 population censuses could not precisely determine recovery, but a steady increase in the population supported the predictions of the life table model. Bald eagles were declared recovered in 1999 by the EVOSTC.

Black Oystercatchers--The evidence for injury to black oystercatchers were the nine oiled carcasses recovered in 1989. The number of oyster catchers killed may have been five times this amount. For a few years after the spill, oystercatchers on oiled Knight Island did not have as good reproductive success (chicks produced per nest) as they did on nearby unoiled Montague Island (Andres 1997), and for a few years after that chicks gained weight more slowly and had oil in their feces on Knight Island. However, by 1999, it was concluded that any remaining differences in reproductive success were due to other factors, such as nest predation (Andres 1999). The Black oystercatcher was moved from the recovering category to the recovered category by the EVOSTC in 2002.

Clams-These species are covered below in the intertidal category. Clams are listed, as of 2002, in the recovering category on the injured species list.

Common loons—There were 216 oiled carcasses of common loons and 179 of other species of loon retrieved from the beaches in the spill area in 1989, but no expansion factors have been postulated to estimate the total toll. There are probably only a few thousand common loons in PWS, where most of the carcasses were recovered, so it is possible that a substantial portion of the population was killed. Boat surveys in PWS showed that common loons have decreased over 50% since the mid 1970's, so the spill toll was additive to declines due to other unknown factors. There has been no recovery of the loon populations since the spill and this species was still considered not recovered as of 2002 by the EVOS Trustee Council.

Common murres--This species had the single greatest toll of any seabird or mammal, representing about 75% of the carcasses and, therefore, the same proportion of the total estimated 250,000 seabirds killed by oil in 1989. Post-spill populations of common and thick-billed murres were clearly less than the censuses of pre-spill populations in the areas of the northern Gulf of Alaska affected by the spill. However, there are millions of murres in the Gulf of Alaska and Piatt and Anderson (1996) concluded that there was no credible way to separate the losses detected from the spill to those due to climate since previous estimates. It seemed probable that the greatest losses of murres in the GOA was from the Barren Islands area in the entrance to Lower Cook Inlet, where counts had returned to pre-spill areas by about 1999. In addition, the breeding synchrony and timing that had been offset by weeks in 1989 finally retuned to pre-spill timing by 1999 (A. Kettle, unpublished). Therefore, the EVOS Trustee Council declared the murres recovered in 1999.

Cormorants--The situation with cormorants is similar to that for common loons. There were carcasses recovered (838), no postulated expansion factors for which to project total mortality, and declining populations from before the spill. Since there has not been a stabilization or increase in the abundance of this species from boat survey data, it is still

on the EVOSTC injured species list in the not recovered category.

Cutthroat trout and Dolly Varden—These anadramous fish were studied together and substantially less growth occurred in animals collected in the oil-contaminated western side of PWS in 1990 and 1991 (Hepler et al. 1996), but no differences in survival were detected. Since no pre-spill growth studies were carried out, it is possible that differences in growth rates were due to other factors, such as different water temperatures between the east and west sides of PWS. No recent studies have been published that either confirm another reason for the observed pattern of growth differences (e.g., the differences persist long after exposure has ceased, or due to a natural factor), or demonstrate that the differences in growth no longer exist. Therefore, these two species remain on the EVOSTC injured species list as recovery unknown.

Harbor seals—The injury to harbor seals was explained earlier in this section. Since the 200-300 adult seals that were probably lost because of the spill have not been replaced by natural recovery in the declining population (a 80% decline since the late 1970's), as of 2002 this species remains on the injured species list in the not recovered category.

Harlequin ducks—There were 212 carcasses recovered after the spill, representing perhaps as many as 1800 individuals. Post-spill populations censuses indicate more Harlequin ducks on the eastern side of PWS and very few broods in the oiled western portion of PWS. Pre-spill data appears unreliable with regard to the presence of duck broods in western PWS, so it is unclear if there are natural geographic differences, e.g., the longer streams in eastern PWS may be favored for breeding. The decrease in exposure to oil and recovery of adult female winter survival rates in western PWS (see discussion earlier in this section) is a strong indication that recovery is underway and perhaps this species will be removed from the injured species list soon. As of 2002 the Harlequin duck is listed as not recovered.

Pacific Herring—The effect of the oil spill on Pacific herring was discussed earlier in this section (see ***V.E.2.a***). Basically, the population in the spill area crashed in 1993-1994 from an estimated biomass of 120 metric tons and has stayed below 30 metric tons since that time. Although there is little evidence that the crash of the population, four years after the spill, is linked to oil, the problems of the population which oil conceivably could have had a part, make it difficult to declare it recovered from the spill. Therefore, the Trustee Council has continued to list this species as not recovered.

Intertidal communities—There was extensive and widespread damage to the intertidal zone over about 1500 kilometers of coastline in the spill area—from the oil and from the subsequent cleanup (Highsmith et al. 1996; Hooten and Highsmith 1996; Stekol et al. 1996; Duncan and Hooten 1996; Barber et al. 1995). Since most intertidal organisms are fixed in place and have limited ability to move there are fewer problems interpreting if they have been affected by oil. However, the lack of pre-spill data makes it difficult to know if differences between the oiled and non-oiled shorelines all arise from the spill or if there are other reasons for them. Sampling large numbers of oiled and unoiled sites partly compensates for the lack of pre-spill data on intertidal communities in determining the spill's effect, as it is less likely that other factors will systematically affect each

category of effect. In general, organisms in intertidal communities had recovered by 1991, however there were lingering effects. First, reproductive capacity of the dominant brown alga, *Fucus*, had not recovered by 1993 (DeVogelaere and Foster 1994). In addition, as of 1997 the bivalve fauna had not recovered on beaches that had been cleaned with vigorous washing and lost their fine sediments (Driskell and Fukuyama, 1996). Also, as of 1996 there were negative effects of oil on clams (*Protothaca stamminea*) in a reciprocal transplant experiment (Fukuyama et al. 2000). For these reasons, the intertidal communites are listed as recovering, but not fully recovered.

Marbled and Kittzlet's Murrelets—These two non-colonial diving seabirds forage nearshore in PWS. The marbled murrelet is found in PWS in relatively large numbers and nests mainly in old growth forest (Kuletz et al. 1995). Kittlitz's murrrelets are more rare, occur in low densities in PWS and Lower Cook Inlet (about 3-4 thousand in each area in summer; Kendall and Agler 1998) and nest mainly in rock scree and prefer to forage in glacial fjords (Day et al. 1999). Both species feed on common forage fishes most of the year and invertebrates only in early summer. There were 1100 carcasses of murrelets recovered from beaches following the spill; about 90% were marbled murrelets. The actual number killed was probably 8-10 times higher than the carcass count and may have been about 7% of the population in spill areas. Summer surveys in PWS have documented declines in marbled murrelets since the early 1970's and the decline has continued since the oil spill in both oiled and unoiled areas (Piatt and Naslund 1995). However, March surveys have shown that spring populations have neither increased nor decreased during this same time. As of 2002, the EVOSTC listed this species as recovering. There is no population trend data for the Kittlitz's murrelet, although it is likely that this species has undergone a large decline since the 1970's (Kendall and Agler 1998). This species is listed as recovery unknown by the EVOSTC.

Pigeon guillemots—This seabird occurs in small aggregations in various places in the spill area. Pigeon guillemots feed on nearshore fishes and invertebrates, and as a diving seabird, are quite vulnerable to oil. Their productivity, or reproductive success, has been linked to the availability of small forage fishes, particularly sand lance (Golet et al. 2002). The spill may have killed up to 15% of the population in PWS. Surveys have shown that this species has been declining for some time in PWS, and without the replacement of individuals lost in the spill the EVOSTC continues to list this species as not recovering.

Pink salmon--The effects of the spill on pink salmon are discussed extensively in chapters V.E.2.b and V.E.3.a. Since there was little evidence of exposure and highly unlikely that oil was affecting this species 13 years after the spill, the Exxon Valdez Trustee Council declared the pink salmon recovered from the effects of the oil spill in 2002.

River otters—There were 12 carcasses of river otters found after the spill. Since unhealthy animals may have retreated back into their dens in wooded areas near shorelines, been eaten by predators or otherwise not accounted for, the number of river otters killed could have been much greater. Some of the same difficulties in accounting for dead animals apply to estimating populations in the spill area. There is no reliable

way to directly estimate the total population size (Melquist and Hornocker 1979), and, therefore, what proportion of the population in the spill area was killed directly by the spill. Since there was little pre-spill data on river otters, the inferred injury to river otters came from a post-spill comparison of an unoiled area near Esther Island and an oiled area on Knight Island, both in PWS. Differences between these two areas included the following inferred effects of oil: about a 20% elevation of blood haptoglobin concentrations and elevated interleukin-6 concentrations (Duffy et al. 1993,1994a), a >1% lower body mass of adult males; a higher rate of latrine abandonment (Duffy et al. 1994a), and larger home territories and a less diverse diet of fish (Bowyer et al. 1994, 1995). An indirect method of estimating population densities at the two study sites using radiolabeled cesium found equal densities of river otters at the two sites (Testa et al. 1994). By May and June of 1992 the differences in haptoglobin and interleukin-6 seen in 1991 were not present (Duffy et al. 1994b).

To supplement the field observations, a series of captive experiments were carried out using river otters given sublethal doses of oil. The results revealed subtle effects on digestion and on biochemical relationships between induction of P4501A enzymes and a porphyrin related to hemoglobin metabolism (Ben-David et al. 2001, 2000; Taylor et al. 2001). Food passed through the digestive systems of high dose otters (receiving 50 ppm oil in their food) faster than the low dose (5 ppm oil in food) and control groups. Ingestion of the large amounts of oil necessary to affect this reduction in food retention time may have occurred after the spill and might explain the smaller body sizes of adult male otters observed after the spill. One of the most interesting outcomes of the oiling experiments was the effect of oil on diving physiology and food capture by river otters. Oil-exposed otters developed anemia, had greater oxygen consumption rates during exercise and made fewer dives to capture fish (Ben-David et al. 2000). Although the case is not strong, there is enough evidence from field and laboratory studies to suggest that oiling could have affected the energy available to river otters to find and capture prey as a consequence of subtle alterations of blood chemistry, and energetic fitness. Since the effects of the spill seem to have only lasted a few years, the river otters were placed in the recovered category of the injured species list in 1999.

Rockfish—A few dead rockfish were observed at the time of the spill and may have died of acute oil toxicity. No comprehensive studies of rockfish were carried out and the rockfish is still in the category of recovery unknown.

Sockeye salmon—There is no evidence that spilled oil affected this species. However, there were indirect effects of the closure of the fishery in 1989 to protect salmon fisheries elsewhere in Alaska due to public concerns about the safety of Alaskan salmon. Also, as a result of fisheries closures a larger number of spawning sockeye entered their natal streams to spawn, resulting in some ecological interactions in the nursery lakes between the rearing fry and their planktonic food base (Schmidt et al. 1996). Although the ecological reverberations may have lasted several years, it was likely that they were over or insignificant by 2001 and sockeye salmon were declared recovered by the EVOSTC in 2002.

Sub-tidal communities--The communities that were exposed to oil inhabited soft sediments extending from the lowest low tide line to a depth of 20 m. Oil in subtidal sediments showed a rapid decrease from 1990 to 1991 (about a 10 fold difference in the shallow subtidal near eelgrass beds; Dean and Jewett 2001) and about 13% of the oil remained in the subtidal sediments in 1991(Wolfe et al. 1994). Losses of oil continued through the 1990's, and as of 2001, no detectable *Exxon Valdez* oil remained in sediments near heavily oiled beaches (See chapter V.B.). Studies around Knight Island in PWS, in the heart of the spill affected area, showed little effect on kelps (Dean et al. 1996), some potential effects on flowering of eel grass (Dean et al. 1998) and that some amphipod crustaceans, of the families Phoxocephalidae and Isaeidae, were less numerous in oiled areas. These amphipods are known to be particularly susceptible to oil pollution. There were other differences in the abundances of subtidal organisms (Jewett et al. 1999), but the differences were not nearly of the magnitude that occurred in the adjoining intertidal zone. Differences seen in the subtidal zone disappeared in 1993, but then reappeared when studied again in 1995, suggesting that some of the differences seen earlier may have been due to natural phenomena such as sediment texture variation, not the oil. Organic enrichment from oil degradation has also been invoked to explain the greater abundance of some organisms at oiled sites. Lacking pre-spill data and not knowing whether remaining faunal differences indicate a lack of recovery or natural differences that existed before the spill, the subtidal communities are listed as recovery unknown on the injured species list.

V.G. Conclusion

As a result of the *Exxon Valdez* oil spill, researchers are now aware that some habitats are capable of sequestering oil for long periods of time, thus species dependent on these habitats can be exposed to oil for protracted periods (a decade or more). As persistent oil weathers, it can become more toxic per unit mass because the less toxic fraction is removed (weathering) more rapidly. Spill response strategies will need to factor in what habitats are present and their ability to retain PAH compounds to appropriately mitigate spill effects. It is also important to assess what animals are associated with the affected environment and the feeding relationships of these organisms. Even if sensitive life stages are not present during a spill, exposure is possible if their habitat, such as intertidal sediment, is able to retain hydrocarbons and subsequently release toxic compounds over long periods. A precautionary approach to oil and gas development, incidental hydrocarbon release (such as urban runoff), and spill response is advisable based on long term persistence of oil in some environments, the high sensitivity of early life stages to exposure, and the presence of trophic cascades and indirect effects.

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V.I. Appendices

Appendix A

Appendix A.
Summary of the major studies and findings on the biological impacts from the *Exxon Valdez* Oil Spill.

Species/Habitat	Study	Comparisons*	Findings*	Comments	References
Bald eagles	Carcass recovery		151 carcasses	Total spill mortality estimate 250	Bowman et al., 1993
	Pop. (adults)	PrS/PstS	pop. Increased since spill to pre-spill numbers		Bowman et al., 1993
	Reproduction	O/UO, PstS	O<UO	Chick production was lower in WPWS in 1989, but recovered in 1990; effect may have been due to disturbance	Bernatowicz et al., 1996
	Survival	O/UO, PstS	O=UO	Survival of radiotagged eagles equivalent in WPWS and unoiled areas. Life table model suggests bald eagle population would have recovered by 1995.	Bowman et al., 1995
Black oystercatchers	Carcass recovery			9 carcasses recovered	
	Repro/ feeding	O/UO, PstS	O<U (sites not replicated)	% pairs nesting decreased (89'-91'); decreased feeding rates	Andres, 1997
	Expos/repro	O/UO, PstS	O<U (sites not replicated)	Oil in chick feces; chicks gained weight more slowly	Andres, 1999
Clams	See intertidal communities below.				
Common loons	carcass recovery		216 carcasses	No species specific expansion factors have been specified to estimate total losses	
	Pop. (adults)	O/U PrS/PstS	Severe declines in other loon species (>50%)	Pre-spill data: 1972. Climate shifts appear to have affected fish-eating birds	Agler et al., 1999
Common murres	carcass recovery		Total estimated mortality: 185,000	About 22,200 of the found carcasses were murress whose deaths could be attributed to the spill	Piatt and Ford, 1996
	Pop. (adults)	O/U PrS/PstS	population declines from mid-1970s to 1991	Avail. Data cannot distinguish losses from spill and those due to climate change	Piatt and Anderson, 1996
	Reproduction	O/U, PstS var. GOA colonies	O<U	Lower breeding success in Oiled colonies; breeding delayed several weeks in affected colonies; evidence for these effects outside the spilll region as well. Avail. Data cannot distinguish losses from spill and those due to climate change. Return to pre-spill conditionsin late 1990s.	Piatt and Anderson, 1996;
Comorants (red-faced, pelagic and double crested)	carcass recovery		838 carcasses	No species specific expansion factors have been specified to estimate total losses	
Cutthroat trout	growth and survival	O/U, PstS E vs W PWS	O<U	Slower growth by 36-43% for oil exposed pop. and differences persisted 2 yrs. After spill. Survival was not different.	Hepler et al. 1996
Dolly varden	growth and survival	O/U, PstS E vs W PWS	O<U	Slower growth by 22-24% for oil exposed pop. for I yr. only. Survival was not different.	Hepler et al. 1996
Harbor seals	carcass recovery		A few carcasses recovered; difficult to kow if this was normal mortality or not		
	Pop. (adults)	O/U; PrS/PstS	O<U	Harbor seals were decreasing before the spill, but between 1988 and 1989 oiled areas lost more seals (43%) than unoiled areas (11%).	Frost et al., 1994
Harlequin ducks	carcass recovery		212		Piatt and Ford, 1996
	oil exposure	O/U, PstS E vs W PWS	O>U	Greater induction of P4501A in ducks from oiled areas.	Trust et al., 2000
	female survival	O/U, PstS E vs W PWS	O<U	Poorer winter survival in oiled areas for winters of 1995-1996 through 1997-1998	Esler et al., 2000. Also see Esler et al., 2002 for synthesis of injury and summary of population study results.

* O=oilied, UO = unoiled, PrS=pre-spill, PstS=post spill

Species/Habitat	Study	Comparisons*	Findings*	Comments	References
Intertidal communities	Pop & community	O/U, PstS All major areas in spill zone	O<U	Decreases in 1990 and 1991of most major taxa (e.g., Fucus, barnalcles, snails, mussels). Increases in oligochaetes.	Highsmith et al., 1996; Stekoll et al., 1996
	Fucus pop.	O/U, PstS herring Bay PWS	O<U	Impacts on adult population through 1990; recovered by 1992. Reproductive capapcity had not recovered by 1993. Pop.s In areas cleaned with hot water were more affected.	Van Tamelen & Stekoll, 1996
	clam (Protothaca)	O/U	O<U	Effects found on growth in clams from an oiled transferrred to an unoiled site.	Fukuyama et al., 2000
	Invertebrate recruitment	O/U experiemntal oiling herring Bay PWS	O<U	Limpets and litorine snails had lower recruitment on tarred settling plates than controls.	Duncan & Hooten, 1996
	Intertidal fish	O/U, PstS Western PWS (oiled/cleaned vs. control)	O<U	In 1990 diversity was equivalent but numbers of fish were greater in unoiled areas. Effects were not detected in 1991.	Barber et al., 1995
Subtidal communities	Pop. and community	O/U, PstS herring Bay PWS	O<U &U<O	Negative effects on some amphipod populations; enhancement of some polychsetes at oiled sties. Natural variability may have influenced findings.	Jewett et al. , 1999
Killer whales	Pop. & pod	PrS-PstS, oil-exposed pods	Very high mortality in AB transient pod	Loss of 13 individuals in 2 years out of the original 36 members of AB pod. Pod had history of harassing fishermen and had bullet scars. Injury is circumstantial. Cause(s) of death uncertain.	Matkin et al., 1994
Marbled murrelets	carcass recovery		528 identified carcasses	Total estimated mortality: 4984 (another 3143 unidentified murrelets)	Kuletz et al., 1996
	Pop.	PrS/PstS Naked Island PWS	PstS<PrS in 1989 PstS>PrS in 1990	There was a negative relationship between boat activity and abundance that affected the population in 1989.	Kuletz et al., 1996
Kittzlitz's murrelets	carcass recovery		51 identified carcasses. Some unidentified murrelet carcasses may have been this species.	255 total estimated mortality.	Kuletz et al., 1996
Pacific herring	Egg/larval impacts	O/UO, PstS	O<U	About half the depositied egss were exposed. Larval deformities and abnormalities were elevated in oiled areas. It was estimated that larval production was reduced by about 3 orders of magnitude in WPWS.	Brown et al., 1996a,b
Mussels			More than 50 oiled mussel bed identified in 1993		Karinen et al., 1993; Babcock et al., 1996
Pigeon Guillemots	carcass recovery		136 carcasses in PWS	Total spill mortality highly uncertain. May have been as high as 1-15 % of the spill area population or much lower depending on assumptions.	see Oakley and Kuletz, 1996
	Pop.	PrS/PstS Naked Island PWS	O<U Declines greater in oiled areas	Post-spill pop. around Naked Island was 43% less than in the late 1970s, but declines were area wide. Oiled areas declined greater than unoiled areas in 1989-1990.	Oakley and Kuletz, 1996
Pink salmon	larval mortality	O/UO, PstS	O<U	Surveys showed that embryonic fish (before hatching) had poorer survival in oiled streams than in unoiled stream around Knight Island, 1989-1992.	Bue et al., 1996
	larval exposure	O/UO, PstS	O>U	In 1989 and 1990 pre-emergent alevins had P450IA induction in a variety of tissues in oil-exposed, but not unoiled areas.	Wiedmer, 1996
	juvenile growth	O/UO, PstS	O<U	Juveniles that were tagged with coded wires and recovered in oiled areas had less growth than those reocvered in unoiled areas. Second study with untagged juveniles had similar results. Juveniles in oiled areas of PWS had induced P4501A.	Willette, 1996; Wertheimer and Celewitz. 1996
	juvenile exposure	O/UO, PstS	O>U	Juveniles captured in PWS had greater induction of P4501A than those captured in non-oiled areas in 1989, but 1990	Carls et al., 1996
	adult survival	O/UO, PstS	O<U	Modeling study indicated that about 1.6 milion pink salmon did not return as adults tospawn in 1990 due to the effects of oil exposure, mainly on juvenile growth	Geiger et al., 1996

* O=oilied, UO = unoiled, PrS=pre-spill, PstS=post spill

Species/Habitat	Study	Comparisons*	Findings*	Comments	References
River otters	body mass, blood haptoglobin	O/UO, PstS	O>U haptoglobin; O<U body mass	Body mass lowerin oiled area and hpatoblobins elevated	Duffy et al. 1993
	blood/enzyme chemistry	O/UO, PstS	O>U	Elevated blood haptoglobins, interleuken 6ir,asparate amino transferase, alanine aminotransferase and creatine kinase in 1991. Otters in oiled areas abandoned latrine sites at greater rates than in non-oiled areas	Duffy et al. 1994a
	fecal porphyrins	O/UO, PstS	O>U	Elevated fecal porphyrins in 1990. Otters in oiled areas abandoned latrine sites at greater rates than in non-oiled areas	Blajeski et al. 1996
	diving physiology	O/U, laboratory exposure	O>U	oil doses of 0.5 and 0.05 g/d caused anemia and increased O2 consumption during exercise	Ben-David et al. 2000
	biomarker responses	O/U, laboratory exposure	O=U	oil doses did not affect haptoglobins	Ben-David et al. 2000
	blood/enzyme chemistry	O/UO, PstS	O>U	Blood haptoglobins interleuken 6ir,no longer different by 1992.	Duffy et al. 1994b
Rockfish	adult mortality	PstS		floating carcasses of rockfish reported after spill	
Sea otters	Summary of the major studies and findings on the biological impacts from the Exxon Valdez Oil Spill.	O/UO, PrS/PstS	2,650 total estimated mortality in Prince William Sound	Estimate of total mortality had wide confidence limits	Garrott et al., 1993
	adult mortality	Intersection model estimate	500-700 total estimated mortality on Kenai Peninsula		Bodkin and Utewitz, 1994
	pathology	Examination of dead oiled otters from the wild and rehabilitation centers.	Oil caused pulmonary emphysema, followed by stress, gastric erasion and hemmhorage, heaptic and renal lipidosis, and heaptic necrosis.		Lipscomb et al., 1994
	delayed mortality	O/UO, PrS/PstS	Life table model based on carcass recoveries	A greater proportion of prime aged animals died after the spill than before.	Monson et al. 2000
	biomarker responses	O/UO, PstS	O>U	Elevated P4501A induction in oiled otters at least through 2000.	Bodkin et al. 2002
	biomarker responses	O/UO, PstS	O>U	Elevated liver transaminase enzymes (e.g., GGT)	Ballachey et al. 2001
Sockeye salmon	large excapements up rivers		Larger than usual numbers of fish went up several Alaskan rivers as a result of fishing closures; there was a threat of overgrazing by the developing juveniles in the lakes		Schmidt et al., 1996
	large excapements up rivers	Prs/Psts	Scale growth assessment revealed interactive effects among various brood years of juvenile sockeye salmon.	Spill year related effects were seen for 2-4l years in the Kenai River system and at Red and Akalura Lakes on Kodiak Island.	Ruggeroni et al., 2003

* O=oilied, UO = unoiled, PrS=pre-spill, PstS=post spill

Appendix B

Appendix B: Properties and Composition of Fresh and Weathered Alaska North Slope Crude Oil

Alaska North Slope oil is a brown-black medium crude oil with a calculated API gravity of 30.89. Following are properties and compositional data for fresh oil and for artificially weathered oil that had 30.5% of the initial mass removed by heating (Data from Wang et al. 2003):

Properties	Unweathered	30.5% Weathered
Sulfur, %	1.11	1.5
Flash Point, °C	< - 8	115
Density (@ 0 °C)	0.878	0.946
Pour Point, °C	-32	-6
Viscosity, cP (@ 0 °C)	23.2	4230
Viscosity, cP (@ 15 °C)	11.5	625

	Cumulative Weight Fraction (%)	
Boiling Point (°C)	0% weathered	30.5% weathered
40	2.5	
60	3.9	
80	6.5	
100	10.0	
120	13.4	
140	16.6	
160	19.8	
180	22.6	
200	25.2	0.5
250	32.6	7.5
300	40.7	18.7
350	49.5	31.1
400	57.7	42.8
450	66.0	54.5
500	72.8	64.2
550	79.0	72.8
600	84.1	79.9
650	88.4	85.8

Component	Concentration (weight %) 0% weathered	30.5% weathered
Saturates	75.0	64.8
Aromatics	15.0	18.5
Resins	6.1	10.3
Asphaltenes	4.0	6.4
Waxes	2.6	3.6

Component	Concentration (µg/g oil) 0% weathered	30.5% weathered
Benzene	2866	0
Toluene	5928	0
Ethylbenzene	1319	0
Xylenes†	6187	0
C_3-Benzenes‡	5620	30
Total BTEX	16300	0

Alkylated PAH	**Concentration (µg/g oil) 0% weathered**	**30.5% weathered**
Naphthalene		
C0-N	261	167
C1-N	1015	1288
C2-N	1800	2716
C3-N	1702	2575
C4-N	815	1174
Sum	5594	7919
Phenanthrene		
C0-P	209	295
C1-P	666	932
C2-P	710	988

C3-P	486	707
C4-P	296	432
Sum	2368	3354
Dibenzothiophene		
C0-D	122	174
C1-D	225	319
C2-D	318	456
C3-D	265	362
Sum	931	1312
Fluorene		
C0-F	142	197
C1-F	328	449
C2-F	447	647
C3-F	379	525
Sum	1295	1819
Chrysene		
C0-C	48	68
C1-C	74	107
C2-C	99	141
C3-C	84	115
Sum	306	430
TOTAL	**10493**	**14834**
2-m-N/1-m-N	1.49	1.41
(3+2-m/phen)/(4-/9-+1m-phen)	0.76	0.76
4-m:2/3m:1-m-DBT	1 : 0.65 : 0.34	1 : 0.65 : 0.34

Other PAHs		
Biphenyl	134.71	176.9
Acenaphthylene	12.03	18.43
Acenaphthene	13.03	20.02
Anthracene	2.88	4.55
Fluoranthene	2.88	3.81
Pyrene	8.40	11.92
Benz(a)anthracene	4.64	8.11
Benzo(b)fluoranthene	5.14	7.49
Benzo(k)fluoranthene	0.50	0.70
Benzo(e)pyrene	10.28	14.74
Benzo(a)pyrene	2.26	3.69
Perylene	3.01	4.42
Indeno(1,2,3cd)pyrene	0.13	0.25
Dibenz(a,h)anthracene	0.63	1.02
Benzo(ghi)perylene	3.13	4.91
TOTAL	**204**	**281**