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The effects of spilled oil on coastal ecosystems: lessons from the *Exxon Valdez* spill

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INTRODUCTION

Oil spilled from ships or other sources into the marine environment often occurs in close proximity to coastlines, and oil frequently accumulates in coastal habitats. As a consequence, a rich, albeit occasionally controversial, body of literature describes a broad range of effects of spilled oil across several habitats, communities, and species in coastal environments. This statement is not to imply that spilled oil has less of an effect in pelagic marine ecosystems, but rather that marine spills occurring offshore may be less likely to be detected, and associated effects are more difficult to monitor, evaluate, and quantify (Peterson *et al.*, 2012). As a result, we have a much greater awareness of coastal pollution, which speaks to our need to improve our capacities in understanding the ecology of the open oceans. Conservation of coastal ecosystems and assessment of risks associated with oil spills can be facilitated through a better understanding of processes leading to direct and indirect responses of species and systems to oil exposure.

It is also important to recognize that oil spilled from ships represents only ~9% of the nearly 700 000 barrels of petroleum that enter waters of North America annually from anthropogenic sources (NRC, 2003). The immediate effects of large spills can be defined as acute, due to the obvious and dramatic effects that are observed. In contrast, the remaining 625 000 barrels that are released each year can be thought of as chronic non-point pollution, resulting from oil entering the coastal ocean as runoff in a more consistent but much less conspicuous rate. In this chapter, we primarily address the effects of large oil spills that occur near coastlines

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and consider their potential for both acute and chronic effects on coastal communities. As described below, in some instances, the effects from chronic exposure may meet or exceed the more evident acute effects from large spills. Consequently, although quantifying chronic effects from low exposure rates can be challenging and time-consuming, the results of such efforts provide insights into the understudied effects of chronic non-point oil pollution.

Spilled oil can accumulate and persist in coastal habitats, leading to ecosystem-level effects that range from acute to chronic, direct to indirect, and lethal to sublethal. We begin by briefly exploring a range of coastal habitats where large oil spills have occurred and the nature, magnitude, and duration of documented effects that occurred under an array of conditions. This evaluation is not intended to be inclusive or exhaustive, but rather illustrative of the range of habitats, species, and interactions that have been impacted by oil spills and the resulting chronic and indirect effects detected. We next describe some of the challenges inherent in trying to understand the effects of spilled oil in environments where there is often little understanding of the status, trends, or variability of the system at the time of the spill.

We then turn our focus to work over the past 23 years on the biological effects of, and the process of recovery from, the 1989 tanker vessel (T/V) *Exxon Valdez* oil spill in Prince William Sound and the Gulf of Alaska. To that end we present an overview of the spill and the observed acute, chronic, and cascading or indirect effects, which in some cases have persisted for decades. Realized impacts were dependent on the magnitude and persistence of oil in the environment, and the phenology, behavior, trophic dynamics, and social structure of organisms. Acute and direct effects were most obvious and resulted in elevated and often high rates of mortality. Chronic and indirect spill effects were more difficult to detect. Generally, chronic exposure to persistent oil was reflected in reduced levels of individual fitness, resulting in modest changes to survival and reproduction that ultimately modify population growth rates.

We follow this discussion with case histories from four species that demonstrated different vulnerabilities to oil as reflected through habitat, life histories, and behavior, each of which demonstrated protracted periods of recovery. These include the pink salmon (*Oncorhynchus gorbuscha*), killer whale (*Orcina orca*), harlequin duck (*Histrionicus histrionicus*), and sea otter (*Enhydra lutris*). We close by reviewing the lessons learned in responding to the *Exxon Valdez* spill with the intent to improve responses related to biological resources when the next such event occurs.

REVIEW OF PRIOR SPILLS

Although our knowledge of the effects of oil spills in marine environments continues to grow, because of high variability among spills, there are few generalities to be drawn from this body of literature outside of the classifications of shoreline types used to evaluate sensitivity to spilled oil (Gundlach & Hayes, 1978; Gundlach, 2006; Adler & Inmar, 2007). Rocky exposed coasts are generally considered least susceptible, with sensitivity increasing as exposure, sediment size, and slope decrease. In general, marshes, mangroves, and estuaries are considered most sensitive and capable of sequestering oil for decades (Gundlach, 2006), although *Exxon Valdez* oil has persisted in the Gulf of Alaska in both exposed and sheltered unconsolidated sediments for more than two decades (Li & Boufadel, 2010). Multiple factors, including variation in coastal geography and geology, ocean and atmospheric conditions, type, volume and rate of oil spilled, distance from the coast, and composition of biological communities all interact to influence the nature and degree of the disturbance caused by an oil spill (Kingston, 2002).

Low latitude

In 1986, more than 50 000 barrels of crude oil were spilled into nearshore habitats comprised of coral reefs, mangroves, and seagrass beds near Galeta Marine Laboratory in Panama (Jackson *et al.*, 1989). Studies following a previous spill in the same area in 1968, the *Witwater* (Rotzler & Sterrer, 1970), providing an unprecedented baseline of data to support rigorous examination of acute and chronic effects of the 1986 spill across these habitats. The 1986 spill contaminated approximately 82 km of shoreline and affected 16 km^2 of red mangrove (*Rhizophora mangle*) and 8 km^2 of intertidal and subtidal reefs. Acute effects of the spill included large-scale mortality of scleractinian corals, with total coral cover reduced by up to 76%, and intertidal sea grasses (*Thallasia testudinum*), but with little apparent effect on subtidal seagrass. Within a few months of the spill, a 27-km band of dead mangroves was evident where oiling had occurred (Jackson *et al.*, 1989). Additionally, a diverse suite of invertebrates, including mussels, barnacles, oysters, and stomatopods (mantis shrimps) exhibited reductions in abundance. Algal assemblages also were affected through acute mortality but recovered to pre-spill abundances within 18 months. Many of the acute effects of the spill and recovery processes were consistent with what was expected from previous spills in tropical shorelines, but other results were not. Duke and colleagues (1997) estimated that sublethal damage to mangroves was 5–6 times greater than direct mortality. The widespread

mortality of subtidal corals and invertebrate assemblages associated with seagrasses was unexpected and illustrates the variability in responses to acute oiling of tropical coastlines. This event also illustrates that sublethal exposure to oil and indirect effects can have a marked effect on the structure and function of affected ecosystems. Of particular note was the shift in the size structure of stomatopods, an important prey for many consumers in the system, with smaller individuals dominating the community after the spill. In follow-up work, Guzman and others (1994) found that recovery of corals had not occurred five years after the spill and that lingering oil in the environment was a factor contributing to its reduced fecundity and growth. Long-term persistent oil in coastal mangrove and seagrass sediments was projected to delay recovery of this low-latitude tropical ecosystem by more than 20 years (Burns *et al.*, 1993).

In 2001, the T/V *Jessica* ran aground in the Galapagos Islands off Ecuador, releasing >5000 barrels of diesel and bunker oil (Edgar *et al.*, 2003). Although oiling was evident along shorelines, the effects on algae, invertebrates, fishes, and mammals generally were minor and did not persist (Born *et al.*, 2003; Edgar *et al.*, 2003; Salazar *et al.*, 2003). The moderate volume of spilled oil, a predominantly rocky shoreline, dilution of bunker oil with lighter diesel oil, offshore transport, moderate sea conditions, and warm temperatures were suggested as factors contributing to the minimal effects from this spill (Edgar *et al.*, 2003). One dramatic exception was a decline of 62% in marine iguanas from pre-spill abundance at an island affected by the spill (Wikelski *et al.*, 2002). The iguana decline was attributed to relatively low levels of residual oil in coastal waters that appear to have adversely affected digestive processes in the iguanas' hindgut. This example illustrates the complexity of mechanisms and cascading effects by which spilled oil can affect coastal marine communities.

Mid-latitude

In 2002/2003, the T/V *Prestige* spilled 460 000 barrels of bunker C, a heavy oil, off the northwest coast of Spain, eventually impacting 1000–2500 km of coastline and other marine habitats off the coasts of Portugal, Spain, and France (Juanes *et al.*, 2007; Penela-Aranez *et al.*, 2009). This event was unusual in that the leaking ship was towed 260 km offshore before it broke apart and sank in 3600 m of water, where it continued to release oil for > 8 months. Thirteen taxa, from plankton to seabirds, experienced acute population-level reductions, with most of the documented injuries among intertidal invertebrates, benthic invertebrates, and seabirds (Sanchez *et al.*, 2006; Alonso-Alvarez *et al.*, 2007; Penela-Aranez *et al.*, 2009; Munilla *et al.*,

2011). In one additional case, secondary exposure to oil through contaminated prey was responsible for both adult and egg mortality in the peregrine falcon (*Falco peregrinus*) (Zuberogoitia *et al.*, 2006). Investigations following this spill documented widespread chronic effects, primarily represented through continued exposure to polycyclic aromatic hydrocarbons (PAHs) from the *Prestige*. Chronic exposure in the animals was measured through either biomarkers of exposure or direct measures of tissue PAHs. Species that demonstrated chronic effects included benthic invertebrates, demersal fishes, and seabirds (Perez *et al.*, 2008, 2009; Martinez-Gomez *et al.*, 2009; Vinas *et al.*, 2009). Indirect effects resulting from the spill were noted in several cases. The European shag (*Phalacrocorax aristotolis*) suffered high female-biased direct mortality, which resulted in long-term reductions in reproduction at the population level (Martinez-Abrain *et al.*, 2006). In addition, chronic exposure to PAHs in yellow legged gulls (*Larus michahellis*) was correlated with reduced secondary sexual characteristics that likely resulted in reduced reproductive performance of individuals, potentially lowering population levels (Perez *et al.*, 2009). In a third example, Velando and colleagues (2005) observed a reduction in reproduction and declining chick condition in the European shag, attributed to a spill-induced reduction in its preferred prey, the sand eel (*Ammodytes* spp.). Lastly, Pineira and others (2008) found genetic-level effects on shell traits in the rough periwinkle (*Littorina saxitilis*) consistent with oil exposure. Despite the breadth of taxa that incurred acute or chronic spill effects, Penela-Aranez *et al.* (2009), in a review of the *Prestige* effects on biota, concluded that recovery was evident in less than two years. Alternatively, the work of others clearly indicates that chronic and indirect effects may have persisted and delayed recovery beyond 2004 (Velando *et al.*, 2005; Martinez-Abrain *et al.*, 2006; Martinez-Gomez *et al.*, 2009; Perez *et al.*, 2009).

High latitude

In January 1993, the grounding of the T/V *Braer* released ~600 000 barrels of light crude oil near the rocky shorelines of southern Scotland at approximately 60°N, during a period when high winds and currents and large oceanic swells rapidly dispersed and suspended spilled oil into the water column (Ritchie, 1993). Due to these conditions at the time of the spill, relatively little oil was washed ashore, and only minor or localized effects were detected on intertidal species and communities (Newey & Seed, 1995; Kingston *et al.*, 1996), with limited effects observed on seabirds and marine mammals (Goodlad, 1996; Hall *et al.*, 1996). Rather, it appeared that the oil was suspended in the water column and transported to distant sediment

basins, raising PAH levels there up to 150 times greater than background levels (Davies *et al.*, 1996). These contaminated sediments were cause for concern related to the health of finfish and shell fisheries that were closed over an area of 400 km^2 for two years after the spill. Despite the comparatively minor damage documented in this spill, recovery times for affected species were up to a decade (Newey & Seed, 1995). A variety of factors appeared to have contributed to the comparatively low impact of this spill on coastal ecosystems, including a light crude oil, a turbulent sea, and a lack of oil-susceptible beaches, such as low-energy tidelands (Ritchie, 1993). The evidence gathered from this particular spill suggests that pathways of injury to marine ecosystems do not exclusively reside along shorelines and further highlights the fact that we currently have a relatively poor understanding of the mechanisms of damage from oil spilled at sea.

For a variety of reasons it is likely that the full extent of effects of any oil spill in the sea will remain unknown. Some of these reasons include design and statistical issues related to the lack of experimental controls and replication of the treatment (spill effect), and others relate to the repeatability of sampling methods (Weins & Parker, 1995). Perhaps the most common constraint to understanding spill effects, however, is an incomplete understanding and record of the ecosystem prior to the spill, in the form of baseline data. An appropriate baseline would include not only estimates of the numbers, ages, sex, and size of all organisms present, but also the suite of interactions and physical, biological, and ecological processes that resulted in the structure, function, and variability of that community prior to the spill; a tall order indeed. To some extent, proclamations of the lack of effects of spilled oil in marine ecosystems must stem from our limited knowledge of those systems and the various mechanisms of direct and indirect effects generated by spilled oil. However, accumulating evidence from more recent spills, when resources have been made available to explore effects more closely, clearly indicate the complexity of interactions and cascading effects that result from disturbances to ecosystems caused by large oil spills in the ocean (Peterson *et al.*, 2003, 2012). In other words, we suggest that determinations on the lack of effects following spills could result more from our limited ability to detect and measure, rather than from negligible impacts (Kingston, 2002). The biological significance of oil spill effects undoubtedly will continue to be a source of debate.

Administrative, procedural, and legal requirements at different organizational (governmental and corporate) scales present further challenges to improved understanding of oil spill effects. In the case of the *Exxon Valdez* (Paine *et al.*, 1996), state and federal trustees initially focused efforts on

assessing the extent of injury caused by the spill, while Exxon Corporation focused on restoration of injured resources. The legal framework at the time of the *Exxon Valdez* spill required that Exxon Corporation pay for damages; therefore, an incentive existed to minimize damages, which pitted corporate-sponsored scientists against government-sponsored scientists, whose legal guidance focused primarily on documenting damage. Competing objectives and legal requirements occasionally produced conflicting conclusions in response to the same questions and at the least precluded scientists from working collaboratively to improve basic knowledge (Paine *et al.*, 1996). By nature, the timing and locations of oil spills are unexpected, and the window of opportunity to acquire pre-event data is limited. Responding entities generally have priorities other than ecological sampling, such as human health, spill containment, clean up, and rehabilitation. The limited capacity and priority of planning to acquire pre-event data remains a constraint to advancing our understanding of the effects of spilled oil on coastal and oceanic marine ecosystems.

THE T/V *EXXON VALDEZ* SPILL

Centered at approximately 60°N and 147°W, Prince William Sound (PWS) is a sub-arctic estuarine embayment of approximately 15 000 km^2, with more than 5000 km of shoreline. Glaciated fjords dominate the northern Sound with deep basins to > 870 m in depth dominating the central Sound. A complex shoreline results from hundreds of islands, bays and passages composed of rocky benches and unconsolidated sediments from boulder beaches to sheltered bays and estuaries of finer sediments. The marine ecosystem of PWS is influenced by cool temperatures, high precipitation, and glacial inputs. The Alaska coastal current is a dominant oceanographic feature that transports nutrients and productivity into the Sound where the presence of kelp and seagrass beds supplement microalgal production and provide additional habitat for a variety of invertebrates and fishes. The complex habitat and environment in PWS and the adjacent Gulf of Alaska results in diverse marine communities that can be broadly characterized by at least two distinctive food webs. In general, the marine phytoplankton/zooplankton community supports a diverse assemblage of forage fishes and higher trophic-level consumers, such as predatory fishes, seabirds, pinnipeds, and cetaceans. In contrast, a nearshore food web, where kelps and seagrasses supplement phytoplankton production, supports an assemblage of nearshore benthic invertebrates, such as crabs, clams, urchins, and mussels that feed consumers, such as sea ducks, shorebirds, and the sea

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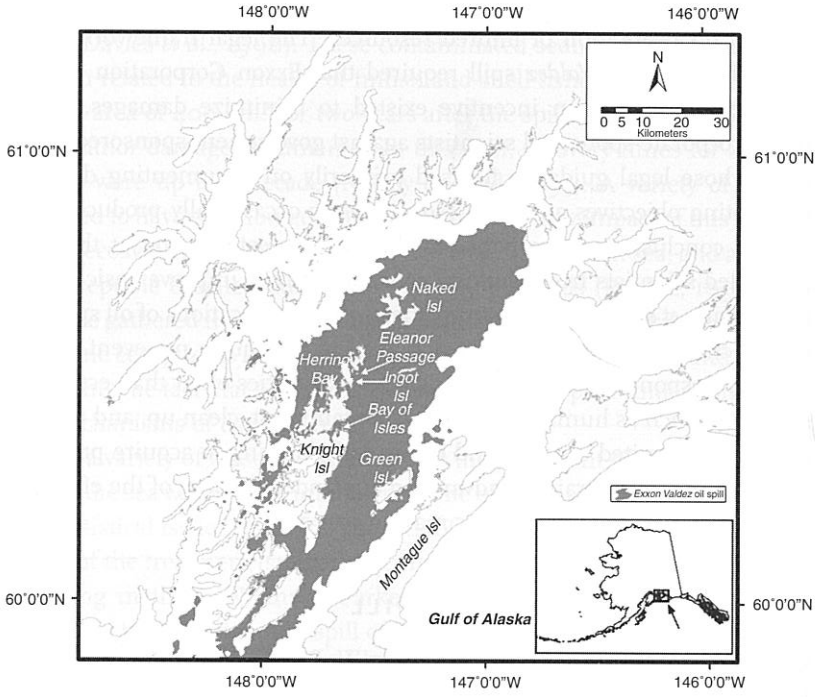

Figure 11.1 Prince William Sound and the area affected by the *Exxon Valdez* oil spill in 1989 (from Monson *et al.*, 2011).

otter. The functional distinction of these two food webs, as well as the life histories and behaviors of individual species, has important implications for the pathways and mechanisms of exposure and both direct and indirect effects of the oil spilled from the *Exxon Valdez* tanker.

On 24 March 1989 the T/V *Exxon Valdez* went aground on Bligh reef in northwest PWS, Alaska (Figure 11.1). The damaged ship eventually spilled an estimated 264 000 barrels of crude oil and ultimately contaminated >2000 km of shore in PWS and the Gulf of Alaska (Bragg *et al.*, 1994). In the weeks following the spill, ~150 000 barrels of oil washed ashore on beaches in PWS (Wolfe *et al.*, 1994), contaminating 783 km of shoreline (Short *et al.*, 2004). Although oil also contaminated shorelines on the Kenai and Alaska Peninsulas and persisted for more than a decade (Irvine *et al.*, 2006), most of the work on acute, chronic, and indirect effects of the spill on species and communities was conducted in PWS, where we will focus the remainder of this chapter.

The initial acute effects of the spilled oil were dramatic and relatively well documented, at least among birds and mammals, through carcass collections

and response activities, with acute mortality estimates of ~250 000 seabirds (Piatt & Ford, 1996), thousands of sea otters, and hundreds of harbor seals (*Phoca vitulina*) (DeGange *et al.*, 1994; Frost *et al.*, 1994a). However, less effort was directed toward quantifying acute mortality among the algae, seagrasses, invertebrates, and fishes, which suffered mortality from both direct oiling as well as clean-up efforts that included highly destructive high-pressure washing in intertidal habitats (Lees *et al.*, 1996).

Based on initial loss rates of oil in the years immediately following the spill, it was assumed that lingering oil would soon be negligible, spill effects would rapidly diminish, and recovery of affected populations would be evident within a few years (Neff *et al.*, 1995). However, long-term monitoring identified both unexpected persistence and magnitude of lingering oil in primarily unconsolidated intertidal sediments and evidence of continuing oil exposure in species of invertebrates, fishes, birds, and mammals occupying nearshore habitats (Peterson *et al.*, 2003; Short *et al.*, 2004). In the following sections, we will review published information on the persistence of lingering oil, the effects of acute and chronic oiling, and the range of mechanisms implicated in both acute and long-term exposure to spilled oil in coastal marine habitats.

LINGERING OIL

In the decade following the spill, several lines of evidence suggested that *Exxon Valdez* oil persisted in unexpected volumes in nearshore habitats and was related to the protracted period of recovery evident for some species. Evidence included both direct observations of oil in the intertidal zone (Hayes & Michel, 1999), the presence of hydrocarbons sequestered in the tissues of bivalves (Babcock *et al.*, 1996; Fukuyama *et al.*, 2000; Carls *et al.*, 2001), and biological responses in fishes, birds, and mammals (Duffy *et al.*, 1994; Trust *et al.*, 2000; Bodkin *et al.*, 2002; Esler *et al.*, 2002, 2011; Golet *et al.*, 2002; Jewett *et al.*, 2002).

In 2001, 12 years after the *Exxon Valdez* spill, Short and colleagues (2004) undertook a comprehensive field study to quantify the amount and distribution of lingering oil in PWS. Nearly 9000 pits were excavated in intertidal substrates that had been described as heavily or moderately oiled during 1989–1993. The team found that both surface residues and subsurface oil persisted on some beaches, including the majority of beaches that were classified as heavily or moderately oiled within four years after the spill. Oil deposits on beach surfaces were highly weathered and largely transformed into asphalt-like material, which was considered to have low toxicity and

low bioavailability. However, subsurface oil was liquid and much less weathered than surface residues (Michel & Hayes, 1999; Short *et al.*, 2007), leading to concerns that the subsurface oil might be both bioavailable and toxic. Short *et al.* (2004) estimated that the areal extent of subsurface oil in 2001 was 7.8 ha (95% confidence intervals: 4.1–12.7 ha), and the mass of remaining oil was 55 600 kg (95% CI: 26 100–94 400 kg; one barrel of oil = 131 kg). These values were considered to be moderate underestimates, given several factors that would lead to a low bias (Short *et al.*, 2006). For example, subsurface oil was found lower in the intertidal zone than anticipated, at elevations not sampled during the 2001 effort, leading to an underestimate by ~30% (Short *et al.*, 2006). Despite uncertainty about the exact amount of lingering oil, all estimates were well under 1% of the amount thought to have stranded initially on PWS beaches. However, the mass and volume remaining and the toxic potential of subsurface oil elicited concerns about the effects of lingering oil on wildlife populations. For example, Short and colleagues (2006) reasoned that animals that forage in intertidal habitats, like sea otters and sea ducks, would be likely to encounter subsurface oil on multiple occasions during the course of a year in the most heavily oiled areas.

Subsurface oil on PWS beaches was distributed mainly in the middle to upper intertidal zone, although a significant proportion (~30%) persisted below 1.8 m above mean lower low water (MLLW) (Short *et al.*, 2006; Figure 11.2). Occurrence of subsurface oil in lower intertidal zones raises concerns, as these elevations tend to be biologically richer than upper zones and serve as foraging sites for many vertebrates, including harlequin ducks and sea otters.

Subsurface oil is expected to decline in occurrence and extent over time through disturbance of sediments associated with storm events, foraging by intertidal animals, including sea otters, and other weathering and degradation processes. However, the rate at which attenuation occurs is unknown and presumably becomes progressively lower over time, with oil persisting longest in areas that are least susceptible to depletion processes (Short *et al.*, 2004, 2007). It is well established that some oil currently remains within sediments underlying some beaches of PWS (Li & Boufadel, 2010; Xia & Boufadel, 2011), although the amount of lingering oil, and how much it has declined since estimates were made in 2001 (Short *et al.*, 2004, 2006), are not certain.

Lingering oil from other catastrophic spills has been observed in a number of other environments (Vandermeulen *et al.*, 1982; Corredor *et al.*, 1990; Burns *et al.*, 1993; Vandermeulen & Singh, 1994; Reddy *et al.*, 2002; Bernabeu *et al.*, 2009), suggesting that persistence of lingering oil is not an

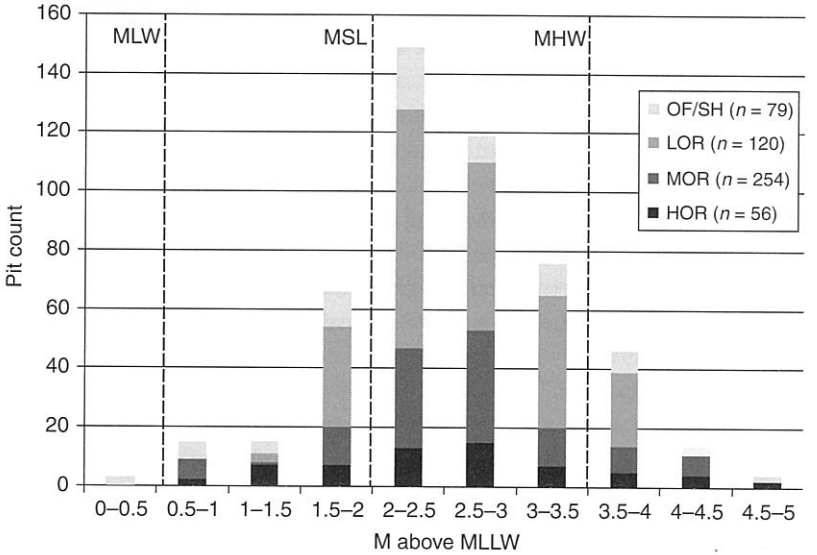

Figure 11.2 Distribution of subsurface lingering oil relative to intertidal zone elevation (*x*-axis, meters above mean lower low water) and oiling intensity (OF/SH, oil film or sheen; LOR, lightly oiled residue; MOR, moderately oiled residue; HOR, heavily oiled residue). The *y*-axis denotes the number of excavated pits meeting the indicated condition (from Short *et al.*, 2006).

issue that is specific to the *Exxon Valdez* spill. However, in the case of the *Exxon Valdez* spill, the extent of research directed at understanding persistence and distribution of lingering oil, as well as effects on wildlife, is unprecedented.

CASE STUDIES FROM THE *EXXON VALDEZ* SPILL

An extensive list of species was affected by the *Exxon Valdez* oil spill, but not all were affected equally. Some, such as the bald eagle (*Haliaeetus leucocephalus*), suffered acute oil-related mortality and reduced reproduction in 1989 but appeared to recover quickly (Bernatowicz *et al.*, 1996). However, a range of mechanisms and pathways of direct and indirect long-term effects from spilled oil on several species were revealed by the research completed in the decades following the spill. A comprehensive review of that research is beyond the scope of this chapter. Instead, we will focus on four species that perhaps reflect the breadth and complexity of mechanisms engaged when considering acute and chronic effects of a large-scale contamination event, such as an oil spill. We caution that the findings and conclusions of

some of the research that we will report may not be universally accepted and that alternative conclusions may be found in the peer-reviewed literature (Boehm *et al.*, 2007; Brannon *et al.*, 2007; Harwell *et al.*, 2012). However, our intent here is not to review the various interpretations of data regarding spill effects, but rather to point to the various ways that spilled oil can affect individuals, populations, and ecosystems. While there will always be some level of uncertainty in understanding effects of events, such as oil spills, we believe that the breadth, depth, and duration of study following the *Exxon Valdez* spill clearly demonstrate pathways and mechanisms of potential spill-related effects that serve to aid in preparing for and responding to future events.

Pink salmon

For pink salmon, biology and vulnerability to spilled oil are linked. Pink salmon adults return in the fall to their natal streams, spawn in gravel beds, and embryos/larvae develop in the gravels until the following spring. Young of the year (fry) emerge from the gravel in the spring and are immediately swept downstream to estuarine marine waters where they convert from freshwater to seawater tolerance and from using yolk for energy to predation/feeding. Fry feed along shore for the first month as they migrate to the open ocean, returning as adults 1.3 years later. Spawning streams in PWS are relatively short (most < 1 mile long), but there are > 2000 of them. The most productive spawning habitat is in the intertidal portion of a stream where up to 75% of the spawning occurs. Unfortunately, this prime spawning area places developing embryos in the habitat where most oil was stranded and subsequently persisted. In addition to the potential for oil-exposed embryos, there was concern that out-migrating fry would receive exposure as they dispersed and foraged along contaminated shorelines.

At the time of the spill, fry were beginning to emerge from the spawning gravels, feeding along the shoreline and migrating through the estuaries of PWS. An estimated 31% of the pink salmon spawning streams in the southwest portion of the Sound were oiled to some extent. Acute mortalities of the out-migrating fry were never detected. However, immediate impacts on growth of fry, and on subsequent survival, were detected in 1989. Evidence of oil exposure included tissue PAH concentrations, induction of the biomarker enzyme P4501A (Carls *et al.*, 1996; Willette, 1996), and oil globules observed in stomachs and intestines (Sturdevant *et al.*, 1996). Wild pink salmon fry collected from nearshore waters in oiled areas grew at half the rate of fry from reference areas (Wertheimer & Celewycz, 1996), and Willette (1996) found lower growth in fry released from hatcheries collected

in oiled areas than in unoiled sites. By 1990, fry grew comparably in oiled and unoiled reference portions of PWS with no evidence of increased P4501A enzyme induction or tissue hydrocarbons, demonstrating that reduced growth of pink salmon fry in the marine environment was restricted to the initial spill year when the shorelines were heavily contaminated during the migratory period.

In the fall of 1989, elevated pink salmon embryo mortalities were detected in spawning gravels of oiled beaches. This immediate impact was not unexpected as oil was still obvious on many beaches. However, further sampling continued to find elevated embryo mortalities up to four years past the spill (1993), although the differences in mortalities with reference streams declined over time (Bue *et al.*, 1994, 1996, 1998). The observation of mortality several years after the spill was unprecedented and raised questions about the exposure mechanism.

Oil exposure was initially assumed to be negligible as oil did not directly contaminate the spawning habitat (oil floats and the spawning gravels were always underwater). However, the continued elevated embryo mortalities stimulated re-examination of possible exposure mechanisms. Murphy and colleagues (1999) confirmed that lingering oil adjacent to streams was associated with elevated embryo mortalities. Further, embryo mortality was consistent with interstitial drainage of oil-contaminated water into spawning gravels from surrounding stream banks (Carls *et al.*, 2003). Concentrations of PAHs in gravels exposed to oil in this way must be very low, however, raising questions of whether a chronic PAH load would be of any significance.

In response, a series of controlled laboratory exposure tests were conducted to determine if chronic low-level exposure to PAHs could duplicate the field observation of elevated embryo mortalities. Acute toxicity studies prior to the spill required several parts per million to kill embryos (Moles & Rice, 1983), but the mechanism of exposure deduced from the field post-spill indicated that chronic exposure concentrations would be much lower, in the parts per billion (ppb). The embryonic development was long (90 days to hatch, more than 150 to emergence), and the high lipid content of the yolk suggested that long-term sequestering of low-level hydrocarbons was possible. Long-term exposure of embryos to PAH concentrations of 20–50 ppb resulted in increased deformities, slower development, histopathological damage, and lower survival (Marty *et al.*, 1997; Heintz *et al.*, 1999, 2000). Effects at these dose levels were unprecedented in the literature, but exposures were long-term, and the polycyclic composition was elevated (3–4 rings) compared to traditional acute bioassays where

1–2-ringed aromatic hydrocarbons dominated the exposure solutions. Chemical analyses for hydrocarbons in tissues and induction of cytochrome P4501A in embryonic tissues indicated the multi-ringed aromatics were easily permeating the outer egg membranes, resulting in lower growth and survival (Carls *et al.*, 2003).

The sensitivity of embryos to chronic exposure to PAH concentrations of 5–20 ppb was demonstrated through delayed impacts on growth and marine survival (Heintz *et al.*, 1999, 2000). In these experiments, pink salmon embryos were exposed to water contaminated with four different concentrations of PAH and then moved to saltwater pens for growth tests, or they were tagged and released to the environment to assess adult returns 1.3 years later. A delayed effect on growth was measured in juvenile salmon that survived embryonic exposure to a concentration of 18 ppb. Marine survival of salmon fry was tested using coded-wire tags to indicate the exposure dose during the embryonic life stage. Over a year later, the returning adults bearing the tags were decoded and counted to determine survival rates by dose (including controls treated and tagged similarly). Marine survival of pink salmon that had been exposed as embryos to 5 ppb in 1995 was reduced by 16%. Exposure to 19 ppb resulted in a 36% reduction in marine survival, indicating a dose–response relationship. The controlled laboratory exposure tests, followed by further environmental challenges (migration, growth, predation) are unprecedented and demonstrate that low-level exposures (ppb) at the embryonic life stage can affect fitness (growth) and have an impact at the population level (adult returns).

Collectively, the measure of elevated embryo mortality in the five years following the spill, identification of the exposure mechanism from contaminated beaches to spawning gravels, and measured effects on fitness following embryonic exposures provides compelling evidence of chronic impacts from lingering oil. Rice and colleagues (2001) calculated that a modestly sized run that produces 10 million eggs would produce 46 000 fewer adult fish if those eggs were exposed to 19 ppb total PAH. Such concentrations were still present in the interstitial waters of a number of salmon streams in 1995 (Murphy *et al.*, 1999).

Killer whale

Killer whales are considered a cosmopolitan species, found in all oceans. In the north Pacific, the species has been further classified into three distinct ecotypes based on genetics, diet, and behavior. These are referred to as residents, which consume fish exclusively and are organized into well-defined and stable social groups, or pods (Bigg *et al.*, 1990); transients, which

consume exclusively mammals; and offshores, which consume numerous fish species, apparently specializing in sharks (Bigg *et al.*, 1990; Ford *et al.*, 1998, 2011; Herman *et al.*, 2005). These ecotypes are morphologically, genetically, acoustically, and dietarily distinct (Matkin *et al.*, 1999; Herman *et al.*, 2005). Killer whales are long-lived, some living over 60 years with females between the ages of about 15 and 40 typically producing a single calf every four to six years (Brault & Casewell, 1993). Although not well understood, it is thought that reproductively senescent females play an important role in maintaining the social structure and dynamics of the pod. Essential to assessing the effects of the *Exxon Valdez* oil spill, resident pods exist within a social organization of matrilines that exhibit no exchange of individuals among matrilines or pods. Some exchange of individuals may occur among transient groups, although this has not occurred within the oil-impacted AT1 transient population of PWS (Matkin *et al.*, 2008).

Understanding the effects of the *Exxon Valdez* spill on killer whales would not have been possible without the five years of data collected prior to 1989. During that period, photo-identification techniques allowed delineation of killer whale pods, including the number, age, and sex of individuals within each pod in PWS and the Gulf of Alaska (Matkin *et al.*, 1994, 2008; Scheel *et al.*, 2001). Additionally, comparable studies from southeast Alaska provided critical contrasts and allowed inferences regarding oil spill effects on killer whale populations and demographics in PWS. Because of the unwavering fidelity of individuals to their matrilines in resident pods and the lack of movement of individuals out of the AT1 transient population, resighting of individuals allowed for estimation of group size, recruitment, and mortality within the resident and transient populations of the region. Baseline data available on this species served to demonstrate the invaluable nature of pre-event data and provided an unprecedented opportunity to evaluate effects of the spill on one of the ocean's top predators.

The *Exxon Valdez* provided one of the first opportunities to evaluate the potential effects of spilled oil on killer whales. Between 1984 and the spill, 11 pods of resident whales and the AT1 population of transient whales were identified and annually censused in PWS and adjacent waters in the Gulf of Alaska (Figure 11.1). During the period prior to the spill there was general stability or a slight increase in the numbers of individuals in most resident pods (Matkin *et al.*, 2008). The AB pod, which was largely resident in PWS, had declined from 28 to 26 individuals during the 5 years prior to the spill, due to the loss of older females and fisheries interactions. The AT1 transient group remained stable at 22 individuals between 1984 and

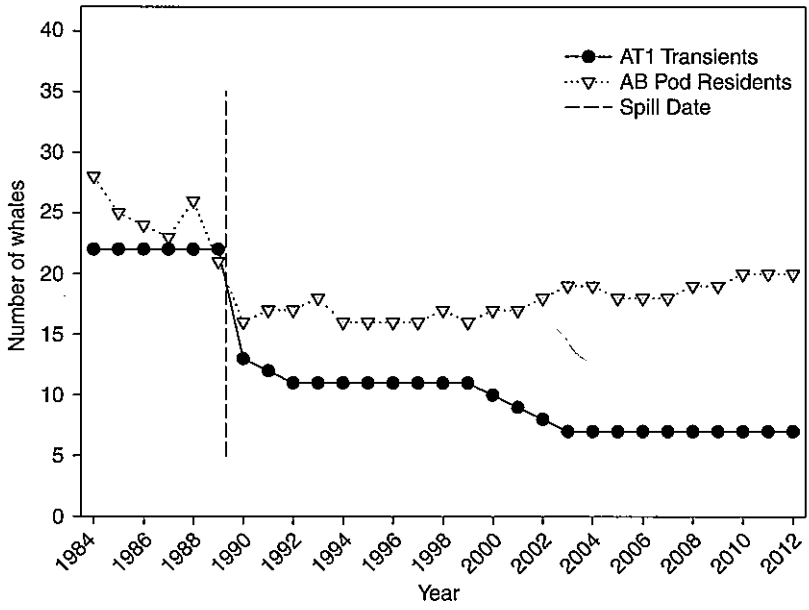

Figure 11.3 The number of killer whales (*Orcina orca*) in the AT1 transient group and AB resident pod, enumerated annually from 1984 to 2012. The vertical line in 1989 indicates the date of the T/V *Exxon Valdez* oil spill. This figure uses data that appeared in Matkin *et al.* (2008) but removes part of the AB pod (six whales) that split off in 1990 following the spill (AB25 pod). The remaining AB pod has not recovered in the 23 years following the spill, with fewer whales in 2012 than prior to the spill in fall 1988.

1989. In the days and weeks following the spill, both groups were documented swimming within the slicks of oil.

Matkin and colleagues (2008) reported a total of 13 mortalities from the AB pod following the spill. However, part of the AB pod (now known as the AB25 pod) split off permanently shortly after the spill. The matrilines, which still travel together and are now considered as the AB pod, lost 10 individuals (38%) following the spill (Figure 11.3). The individuals lost included three sexually mature females and seven juveniles. These are sex and age classes that generally demonstrate very low mortality rates (Olesiuk *et al.*, 2005). Since 1990, the AB pod has not recovered and numbered only 20 individuals in 2012. This annual growth rate of < 1% compares with a growth rate of 3.2% measured across all the unaffected pods in the Gulf of Alaska (Matkin, unpublished data, 2012). The delayed recovery of the AB pod is largely a result of increased mortality of mature and juvenile females shortly after the spill and the resultant decrease in production of calves

(Matkin *et al.*, 2008). However, social disruption resulting from acute oil exposure and mortality of key individuals cannot be ruled out as a factor contributing to delayed recovery and should be worthy of consideration when evaluating the recovery of highly social species.

At least four members of the AT1 population were observed and photographed in the oil within hours of the spill, and three of those four went missing and were eventually presumed dead when they were not resighted in 1990 and the years following (Matkin *et al.*, 2008). Nine of the 22 (41%) AT1 whales have not been observed in the 23 years since the spill and are considered fatalities (Figure 11.3). Additional mortalities reduced the AT1 population to 11 individuals by 1992, half of the pre-spill number, and by 2003 the group was reduced to 7 individuals. There has been no recruitment of calves into the AT1 population since the spill, and they are now listed as depleted under the Marine Mammal Protection Act of 1977.

The loss of 38% and 41% of the AB pod and AT1 group, respectively, following the *Exxon Valdez* spill is unprecedented in the decades of study of killer whales in the north Pacific (Ford *et al.*, 1998). Two avenues of exposure are likely to have caused or contributed to elevated mortality in killer whales, including inhalation of toxic fumes and oil and consumption of contaminated prey (Geraci, 1990; Lipscomb *et al.*, 1994). The latter may be particularly evident among transient whales that feed extensively on smaller marine mammals, such as harbor seals, which were known to have been exposed to and retained oil in fur and tissues (Frost *et al.*, 1994b; Lowry *et al.*, 1994). The lack of discovery of killer whale carcasses following exposure to spilled oil may not be surprising as individuals are known to sink after their death, but it does inject some level of uncertainty in assigning cause of death. However, the weight of evidence provided by Matkin *et al.* (2008) strongly implicates acute exposure to spilled oil as the dominant factor responsible for the declines in these two groups of killer whales.

It is likely that most acute killer whale exposure to oil occurred shortly after the spill, and that even without long-term effects, an extensive demographic lag in replacing lost individuals resulted in a recovery that has stretched over decades. However, the apparent disruption of the social structure of these highly organized groups of animals highlights the complexity of mechanisms and pathways that spilled oil or other sources of mortality can affect marine resources. Also to be considered are the trophic cascades and indirect effects on prey populations (e.g. seals, sea lions, and salmon) resulting from declines of these apex predators (Peterson *et al.*, 2003).

Harlequin duck

Harlequin ducks spend much of their annual life cycle in nearshore marine habitats and demonstrate a high degree of fidelity to relatively small areas of coast (Iverson *et al.*, 2004). As a result, they may not relocate in response to events that diminish the quality of their habitat (Iverson & Esler, 2006). They forage exclusively on marine invertebrates that occur in intertidal and shallow subtidal habitats, which put them in proximity to locations where lingering oil was known to persist (Short *et al.*, 2006). They are demographically characterized by long life spans (to 20 years) and low reproductive potential coupled with high adult survival. As a consequence, population dynamics are sensitive to even slight changes in adult survival. Overwintering in high-latitude marine environments, such as PWS, harlequin ducks exist close to their energetic threshold, which provides little margin for increasing caloric intake to meet elevated metabolic demands (Esler *et al.*, 2002), such as those resulting from exposure to PAHs.

For harlequin ducks, a number of factors indicated lack of recovery for the first decade after the *Exxon Valdez* oil spill, including reduced densities on oiled areas, numerical declines on oiled areas in concert with stable numbers on unoiled areas, and evidence of continued exposure to lingering oil (Trust *et al.*, 2000; Esler *et al.*, 2002). However, the most troubling finding was a significant depression of adult female winter survival on oiled relative to unoiled areas from 1995 to 1998 (Esler *et al.*, 2000; Figure 11.4). A similar study from 2000 to 2003 found that winter survival rates for both young and adult female harlequin ducks was similar between oiled and unoiled areas (Esler & Iverson, 2010; Figure 11.5), suggesting that the direct survival effects of lingering oil had subsided by this time.

Harlequin duck survival, as well as other demographic information including movements and fecundity, were compiled in a population model to estimate the timeline and process of population recovery (Iverson & Esler, 2006, 2010). Model results suggested that elevated female mortality, although likely highest during the first year after the spill, persisted for more than a decade and the mortality associated with the chronic phase of the spill (estimated at 772 females) was higher than the number estimated to have died during the first year after the spill (400 females). The harlequin duck population model also estimated that the most plausible timeline to full population recovery was 24 years (i.e. by the year 2013), with a range of 16–32 years (2005–2021) under best- and worst-case scenarios, respectively (Iverson & Esler, 2010; Figure 11.6).

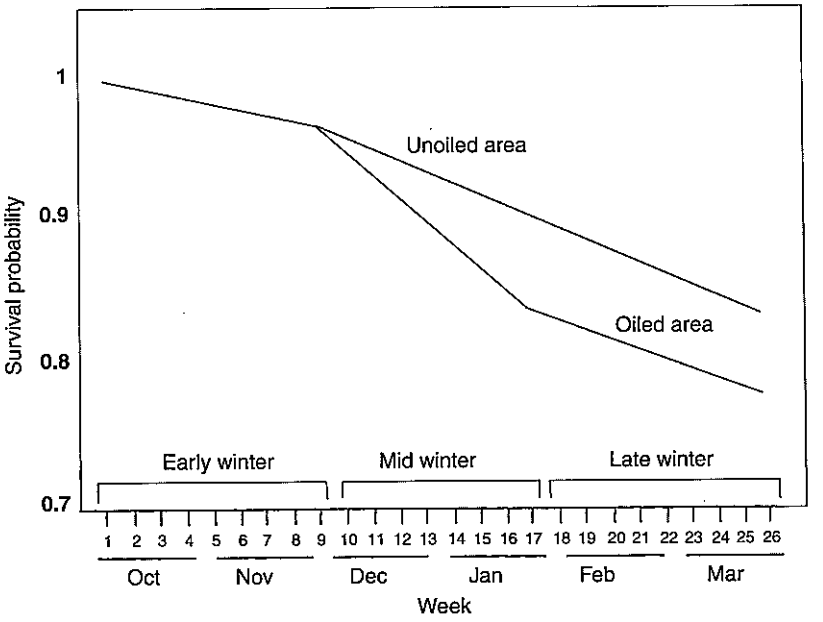

Figure 11.4 Cumulative winter survival probability of adult female harlequin ducks (*Histrionicus histrionicus*) in oiled and unoiled areas of Prince William Sound, Alaska, during 1995 to 1998 (from Esler *et al.*, 2000).

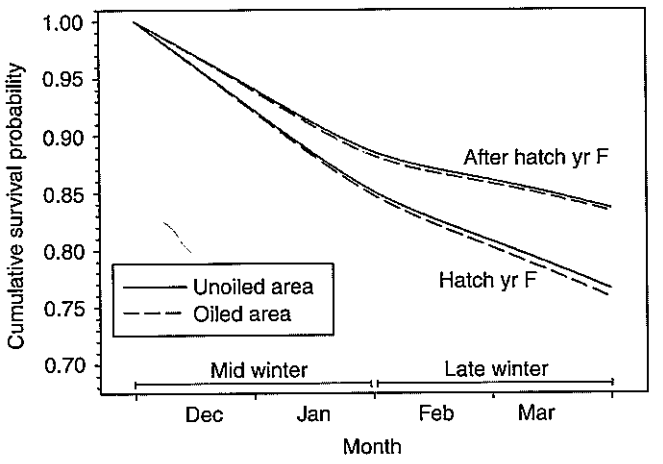

Figure 11.5 Cumulative winter survival probability of adult (after hatch year) and juvenile (hatch year) female harlequin ducks (*Histrionicus histrionicus*) in oiled and unoiled areas of Prince William Sound, Alaska, during 2000–2003 (from Esler & Iverson, 2010).

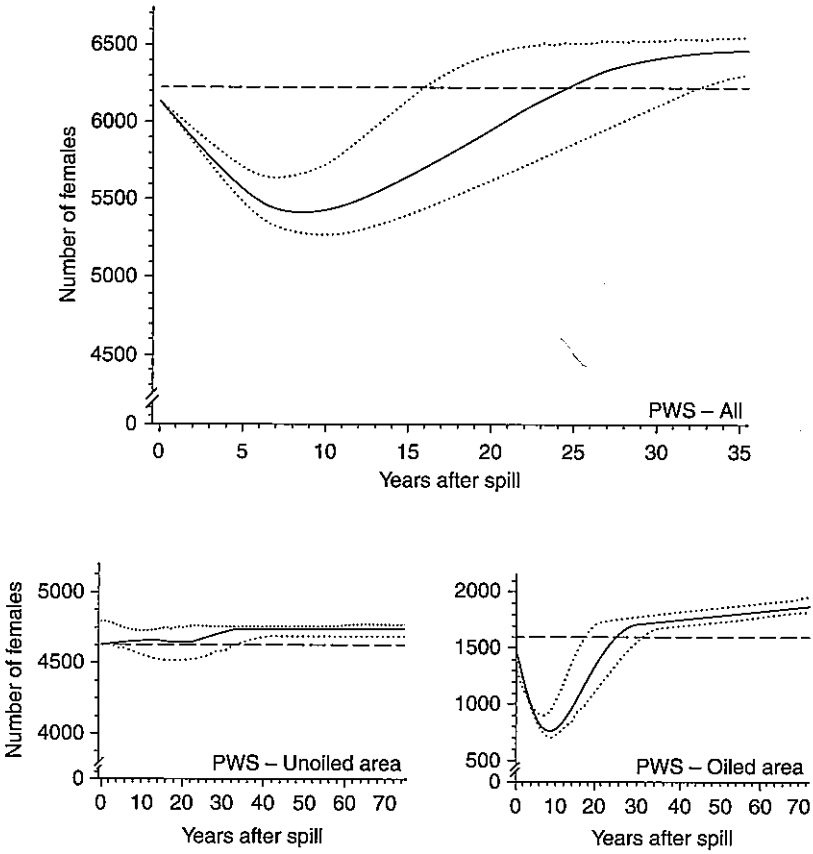

Figure 11.6 Results of a population model exercise projecting timeline to recovery of harlequin duck (*Histrionicus histrionicus*) numbers to pre-spill estimates (dashed line) following the 1989 *Exxon Valdez* oil spill. The solid line represents the most likely outcome, and dotted lines represent best- and worst-case scenarios, based on different permutations of model inputs (from Iverson & Esler, 2010).

Studies of harlequin ducks have generated the most complete data series evaluating cytochrome P4501A (CYP1A) induction since the *Exxon Valdez* oil spill. CYP1A is induced upon exposure to a limited number of compounds, including PAHs, and is widely used as a biomarker of oil exposure. In 1998, nine years after the spill, harlequin ducks from oiled areas of PWS had indicators of CYP1A that averaged nearly three times higher than those from unoiled areas (Trust *et al.*, 2000). Similar patterns were observed through 2009 (Esler *et al.*, 2010), suggesting that some harlequin ducks continued to be exposed to lingering *Exxon Valdez* oil for up to 20 years post-spill.

Unlike killer whales, for which acute oil exposure and mortality and subsequent demographic lags seems to be the most important factor constraining population recovery, harlequin duck population recovery was influenced by persistent, chronic exposure to oil that had demographic consequences for at least a decade after the spill. The importance of chronic exposure and the effects offers a novel perspective of the various mechanisms of population-level effects of oil spills, as well as insights to potential mechanisms of effects of chronic exposure to non-point source pollution.

Sea otter

Sea otters are non-migratory residents of shallow nearshore marine habitats of the north Pacific, mostly defined by the intertidal zone out to about the 40-m depth contour. Individuals display high fidelity to a relatively small annual home-range, which is usually from 10 to 100 km^2. Adult male sea otters defend small exclusive territories from other males, and female home-ranges can overlap several male territories. Because of small home-ranges with high site fidelity, the sea otter is susceptible to local disturbances or modifications to habitat at relatively small spatial scales (Bodkin & Ballachey, 2010). Adults have high survival. Females usually give birth to a single pup annually, and both sexes may live up to 20 years. These life-history attributes render populations sensitive to relatively small increases in adult mortality. Their habitat is defined by their need and ability to dive to the sea floor where they prey nearly exclusively on large benthic marine invertebrates, including bivalves, gastropods, echinoderms, and crustaceans. Most foraging takes place in shallow waters < 40 m in depth (Bodkin *et al.*, 2004). In soft sediment habitats, clams that require excavation can account for a high proportion of their diet. Lastly, reliance on dense fur and a high metabolic rate require that a sea otter consume ~25% of its own body mass daily, providing little latitude to increase energy intake in response to environmental stress, including that resulting from oil-contaminated habitat.

A number of lines of evidence indicate that sea otter populations suffered deleterious effects of the *Exxon Valdez* oil spill during both the acute and chronic stages of the spill (Bodkin *et al.*, 2002). Estimates of sea otter mortality due to acute effects of the oil spill in western PWS ranged from 750 to 2650 animals (Garrott *et al.*, 1993; Garshelis, 1997). The disparity among acute mortality estimates largely reflects the lack of accurate pre-spill estimates of sea otter population size. Using population models, Udevitz and colleagues (1996) predicted recovery of the western PWS sea otter population in 10–23 years, with maximum annual growth rates from

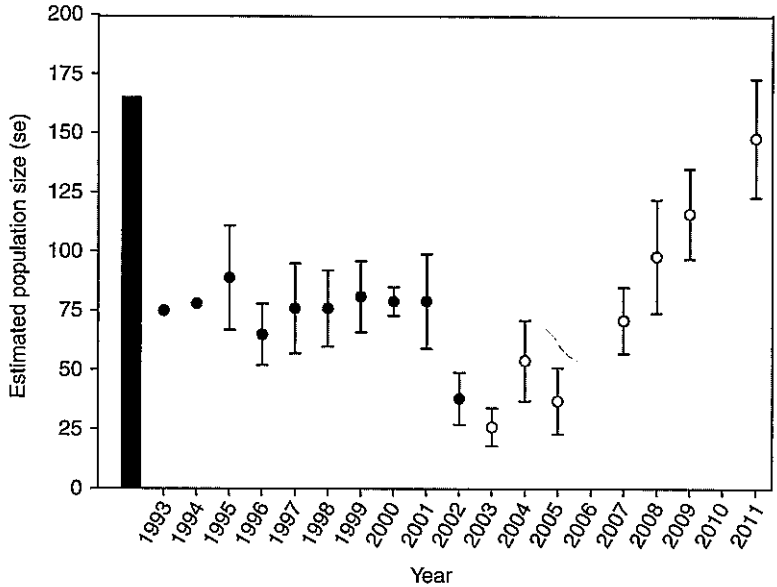

Figure 11.7 Results of aerial surveys to assess the recovery of sea otters (*Enhydra lutris*) following the 1989 *Exxon Valdez* oil spill. The survey area includes islands in the northern Knight Island Archipelago, from Herring Bay to Bay of Isles (Figure 11.1), where sea otter mortality approached 90% (Bodkin & Udevitz, 1994). The black bar reflects the estimated population size at the time of the spill based on the number of carcasses and live oiled animals removed for rehabilitation. White symbols represent the data used to estimate population growth (from Bodkin *et al.*, 2011).

10% to 14%. In the first decade post-spill, numbers of sea otters remained depressed at the heavily oiled northern Knight Island region (Figure 11.7), while recovery was evident to some extent through increases in abundance in areas less severely affected by oil (Figure 11.8). Also, population models based on age distributions of dead otters indicated that the survival of sea otters was depressed for at least a decade after the *Exxon Valdez* oil spill (Monson *et al.*, 2000).

Causes for the delayed recovery of sea otters residing in previously oiled habitats are likely related to elevated mortality rather than reduced reproduction (Bodkin *et al.*, 2002). Linkages between oil and increased mortality are documented in two studies. Ballachey and colleagues (2003) documented elevated juvenile sea otter mortality in oiled areas of PWS compared to unoiled areas. Monson and colleagues (2000, 2011) constructed mortality models based on ages at death acquired both prior to and after the spill. Elevated mortality was evident in animals that were alive at

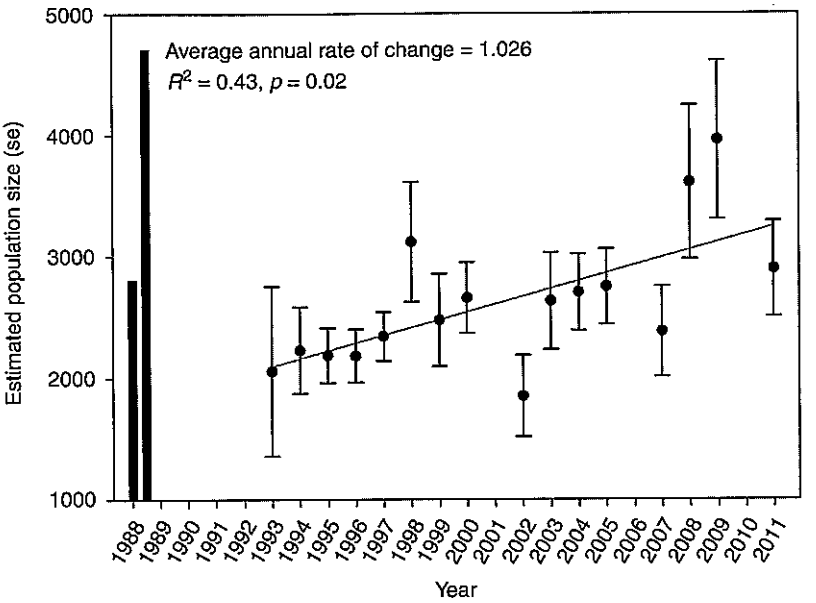

Figure 11.8 Results of aerial surveys to assess the recovery of sea otters (*Enhydra lutris*) following the 1989 *Exxon Valdez* oil spill in western Prince William Sound. The black bars reflect a range of possible pre-spill population sizes based on acute mortality estimates added to the 1993 estimate. The black bars reflect a range of possible pre-spill population sizes based on acute mortality estimates added to the 1993 estimate (from Bodkin *et al.*, 2011).

the time of the spill, as well as among those born after the spill, implicating lingering oil as a contributing factor (Monson *et al.*, 2000, 2011). Monson and colleagues (2011) estimated that the mortality associated with chronic exposure or long-term effects of acute exposure was 900 animals, suggesting that chronic exposure mortality was similar in magnitude to the known immediate, acute mortality represented by the number of carcasses recovered in 1989 following the spill.

While mechanisms of mortality related to acute oil exposure were evident through stranded live sea otters and carcass collections during the spill (Lipscomb *et al.*, 1994), pathways of exposure to relatively small amounts of lingering oil in the decades following the spill were not readily discerned. Sea otters excavate sediments when they forage for some prey, including clams, which constitute ~75% of their diet in PWS (Dean *et al.*, 2002). Sea otter foraging excavations routinely disturb sediments at depths below which lingering oil occurs (Short *et al.*, 2004). Therefore, if sea otters forage at sites with patches of lingering oil, they are likely to be exposed either through

consumption of prey that have assimilated hydrocarbons or by disturbing oiled sediments and releasing lingering oil (Fukuyama *et al.*, 2000), which could then adhere to their fur and subsequently be ingested upon grooming. Bodkin and colleagues (2012) evaluated the degree of spatial and temporal overlap of foraging otters and lingering oil to determine whether these were plausible pathways of exposure.

Based on sea otters with abdominally-implanted time and depth recorders, Bodkin and colleagues (2012) found that, of more than a million foraging dives, most (82%) were subtidal and not a risk for encountering lingering oil. However, all individuals ($N = 19$) foraged in intertidal zones at least some of the time, averaging between 8 and 91 intertidal foraging dives per day. Within the intertidal zone, foraging dives occurred most frequently at lower elevations, where lingering oil was less common (Figure 11.2), but an average of 3–38 dives per day occurred at elevations >1.8 m above MLLW, where most lingering oil persisted. Based on these foraging data and the distribution of lingering oil during 2001 and 2003 (Short *et al.*, 2004, 2006), Bodkin and colleagues (2012) estimated that sea otters would encounter subsurface lingering oil an average of 10 times each year, ranging from 2 to 24 times, depending on individual foraging routines (Figure 11.9). Perhaps more

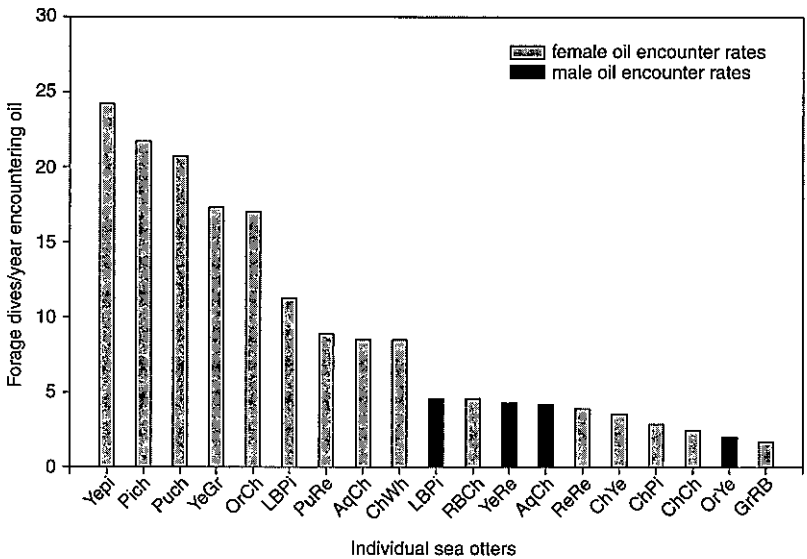

Figure 11.9 Estimated number of foraging dives per year in which individual sea otters (*Enhydra lutris*; $N = 19$) would encounter lingering, subsurface oil in intertidal zones of Prince William Sound, Alaska (from Bodkin *et al.*, 2012).

importantly, there was a strong seasonal component to intertidal foraging, with a pronounced peak from late spring to early summer. This is the period when most adult females will have small pups, making them most likely to encounter oil when they are least prepared physiologically to deal with the added metabolic costs associated with oil contamination.

Bodkin and colleagues (2012) also observed sea otter foraging excavations on soft sediment beaches within the heavily oiled northern Knight Island area, including beaches that were known to contain patches of lingering oil. Further, some sediment samples taken from or near sea otter foraging pits were determined to have elevated levels of PAHs. Collectively, the results of Bodkin and colleagues (2012) confirm that sea otter exposure to sequestered oil via their foraging activities was a likely pathway. These findings indicate that, as a function of their extensive foraging activity, the population of sea otters in the northern Knight Island area may have been an important agent in disruption and depletion of patches of lingering oil through their estimated excavation of more than a million intertidal pits annually (Bodkin *et al.*, 2012).

As another indicator of occurrence and effects of oil exposure, a technique for measuring differential gene expression in response to hydrocarbon exposure has been developed based on laboratory studies with mink (*Mustela vison*) (Bowen *et al.*, 2007). Gene transcript profiles indicated that sea otters from PWS in 2008 had differential transcription relative to those from captive and wild reference areas (Figure 11.10). Particularly for those otters captured near northern Knight Island, there were a number of genes with elevated transcription, including those related to tumor formation, cell death, inflammation, and heat shock (Miles *et al.*, 2012). These patterns were consistent with variation in gene expression of mink exposed to oil in lab studies (Bowen *et al.*, 2007), suggestive of exposure of sea otters to lingering *Exxon Valdez* oil and compromised health and physiological functioning (Miles *et al.*, 2012). Some of the factors indicated by differential gene expression (e.g. tumor formation) could be the result of historical, not necessarily contemporary, exposure in these long-lived animals.

In summary, sea otter populations residing in the path of spilled oil suffered high rates of mortality through acute exposure, resulting in large-scale declines in abundance that unexpectedly persisted for more than two decades. As a result of behavior that structures sea otter populations in relatively small geographical areas, those animals residing in habitats where lingering oil was sequestered in shallow sediments had potential access to that oil. Relying on a diet that is dominated by several species of clam that burrow in shallow sediments provided a direct pathway to

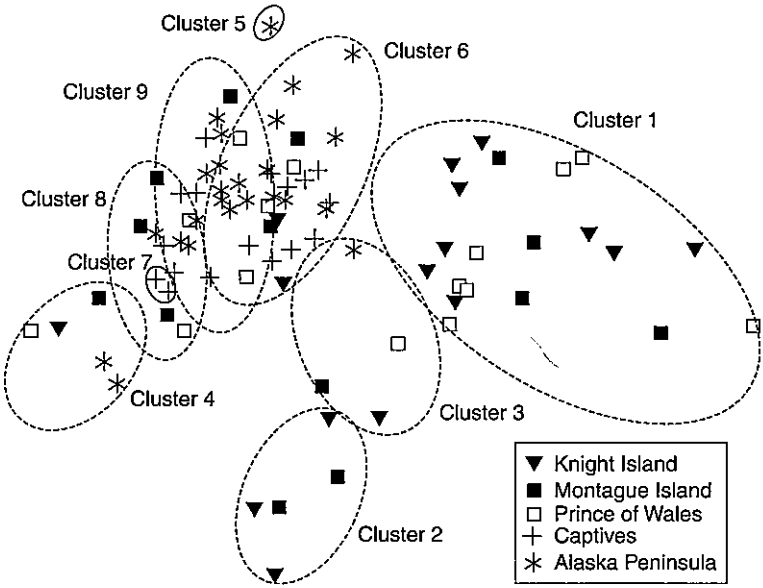

Figure 11.10 Multivariate, non-parametric, multidimensional scaling of gene transcription profiles of sea otters (*Enhydra lutris*) sampled at three locations in Prince William Sound, Alaska, at the Alaska Peninsula, and in captivity at aquaria. Statistics indicated significant separation among all clusters. Clusters 1 through 3 were dominated by sea otters from Prince William Sound, particularly heavily oiled Knight Island (from Miles *et al.*, 2012).

lingering oil. Chronic exposure to oil and possibly latent acute exposure effects apparently led to elevated mortality, contributing to a recovery period of more than two decades. Processes of population injury and recovery were very similar between sea otters and harlequin ducks, suggesting that ecological attributes may be stronger predictors of vulnerability to oil spill effects than taxonomic relationships.

LESSONS LEARNED

A diverse array of factors contribute to the magnitude, duration, and recovery from effects of marine oil spills, including the type of oil; magnitude, timing, rate, and duration of the spill; environmental conditions; and the physical, biological, and ecological conditions over which the spill occurs. Because of unique complexities and uncertainties associated with individual spills, predicting the full range of effects from future spills will remain problematic. However, the experience and knowledge gained from several

decades of research into the effects of spills across a wide range of conditions provides lessons that may be used to better prepare for and document the effects of future spills. In the following three paragraphs we present our thoughts on generalities and lessons to be drawn from the literature on spills. In the final three paragraphs, we provide concluding remarks on our experiences specifically with the *Exxon Valdez*.

Initial short-term acute responses to oil spills and contamination of the shorelines are predictable and were most often reflected in direct mortality of birds and mammals. However, for most marine species (e.g. kelps and seagrasses, invertebrates, reptiles, and fishes), even acute effects remain poorly understood and require additional attention in preparation and response to future spills. Sublethal and chronic effects can lead to reduced fitness that may be translated into reduced survival rates, ultimately affecting recovery of injured populations. Long-term consequences of spills are significant, less predictable, and will remain difficult to document, and should be considered as potentially equal or greater in importance to acute effects.

If there is a single consistent message from those scientists that have studied the effects of past oil spills, it is that the lack of baseline data is the greatest single impediment to understanding the effects of spilled oil on marine ecosystems. Our ability to accurately and defensibly determine the effects of future oil spills on ecosystems, communities, and individuals, and subsequent recovery processes, will be determined largely on the quantity and quality of data available to describe the affected systems prior to the spill. Because ecosystems and populations are dynamic at various spatial and temporal scales, baseline data from one or more unaffected, or reference populations, immediately prior to the perturbation will be essential. Attempts to describe spill effects in the absence of baseline data for affected and unaffected populations will lead to uncertainty in conclusions and will be subjected to the valid criticism of an inadequate experimental design to assign cause to effect. Additionally, lack of accurate and defensible baseline data on populations hampers definition of meaningful restoration endpoints.

The work done to date associated with past spills highlights the diverse and often unpredicted pathways and mechanisms by which spilled oil affects marine ecosystems. It is not simply acute contamination of individual organisms that constitutes spill effects, but rather the complex interactions between the presence and persistence of oil in the environment and the ways in which it can affect organisms and communities. The examples from the *Exxon Valdez* we review above illustrate this complexity and highlight the need to consider and explore potential interactions

between oil and individuals in the context of basic biology, behavior, and ecology.

Long-term oil persistence can lead to long-term consequences. The lack of recovery of sea otters and harlequin ducks in specific areas of PWS was alarming and after several years of study, was eventually linked to the presence of toxic subsurface oil. The apparent pathway of exposure was through diet and foraging behavior and mediated through the fitness of individuals causing relatively modest reductions in survival rates, rather than acute mortalities like those that were easily detected in the first months of the spill. Nevertheless, a localized population effect was detected, and full recovery was hampered for nearly two decades. In both sea otters and harlequin ducks, chronic mortality was nearly equal to or exceeded acute mortality. Response to future spills requires consideration of the potential for long-term consequences from persistent oil in the environment and from persistent effects from acute oil mortality, such as the disruption to the social structure and future reproduction in killer whale pods.

Recovery is dependent on many factors, including chemical recovery of the habitat and species' reproductive biology. Full recovery of pink salmon, sea otters, and harlequin ducks could not happen until the chronic contamination of lingering oil abated. Pink salmon, with high fecundity (2500 eggs per female) and a short life history (2 years), rebounded quickly when habitat oil exposure was removed. In contrast, long-lived/low reproductive potential species, such as the killer whale, may take decades to fully recover, if at all. Although the species affected by future spills will differ, biology, behavior, and ecology of affected species and communities should inform planning and response efforts in terms of the potential for both acute and long-term spill effects.

Lastly, we return to the finding that spills from transporting oil in marine waters represent only ~9% of the total amount of anthropogenic sources of oil entering the environment, and ultimately the oceans, where it is deposited in nearshore habitats. Evidence from laboratory experiments and acute marine oil spills clearly points to the adverse effects of elevated PAHs on both the fitness and health of individual organisms; yet accepting and understanding the potential for chronic oil exposure effects from the 91% of oil that enters the environment via human activities other than marine spills remains a challenge. The results of studies directed at monitoring the process of population recovery from chronic exposure effects of large oil spills provides a new perspective that demonstrates the potential for adverse effects from chronic oil pollution in coastal communities.

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