REVIEW

Long-Term Ecosystem Response to the Exxon Valdez Oil Spill

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The ecosystem response to the 1989 spill of oil from the Exxon Valdez into Prince William Sound, Alaska, shows that current practices for assessing ecological risks of oil in the oceans and, by extension, other toxic sources should be changed. Previously, it was assumed that impacts to populations derive almost exclusively from acute mortality. However, in the Alaskan coastal ecosystem, unexpected persistence of toxic subsurface oil and chronic exposures, even at sublethal levels, have continued to affect wildlife. Delayed population reductions and cascades of indirect effects postponed recovery. Development of ecosystem-based toxicology is required to understand and ultimately predict chronic, delayed, and indirect long-term risks and impacts.

Before the Exxon Valdez oil spill, information available for constructing risk assessment models to predict ecological impacts of petroleum hydrocarbons was limited to selective, largely short-term monitoring after previous oil spills and to tests of acute toxicity in laboratory-tolerant taxa (*1*). After the tanker Exxon Valdez grounded on Bligh Reef in northern Prince William Sound on 24 March 1989, the magnitude of the spill, extent of shoreline contamination, and evident high mortality of wildlife prompted an evaluation of ecological impacts of unprecedented scope and duration extending now for more than 14 years (*2*–*5*). The release of 42 million liters of Alaskan North Slope crude oil contaminated to some degree at least 1990 km of pristine shoreline. Prince William Sound was most severely affected, but the oil spread more than 750 km to the southwest along the Kenai Peninsula, Kodiak archipelago, and the Alaska Peninsula (Fig. 1). Years of study provide a new understanding of long-term biological impacts and recovery processes in a coastal ecosystem populated by abundant marine mammals, seabirds, and large fishes (*2*–*5*).

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Delays in recovery and emergence of long-term impacts are understood by bringing an ecosystem perspective to ecotoxicology (*6*). The ecosystem framework extends ecotoxicology to include interactions among multiple abiotic and biological components rather than treating each species separately and restricting assessment to acute short-term impacts (*7*). Disagreements exist between Exxon- and government-funded scientists (*8*), and unknowns persist, especially in understanding how multiple processes combine to drive observed dynamics. Nevertheless, these uncertainties do little to diminish the general conclusions: oil persisted beyond a decade in surprising amounts and in toxic forms, was sufficiently bioavailable to induce chronic biological exposures, and had long-term impacts at the population level. Three major pathways of induction of long-term impacts emerge: (i) chronic persistence of oil, biological exposures, and population impacts to species closely associated with shallow sediments; (ii) delayed population impacts of sublethal doses compromising health, growth, and reproduction; and (iii) indirect effects of trophic and interaction cascades, all of which transmit impacts well beyond the acute-phase mortality.

Acute-Phase Mortality

After the release of crude oil from the Exxon Valdez into Prince William Sound (PWS), acute mortality followed a pattern largely predictable from other oil spills. Because marine mammals and seabirds require routine contact with the sea surface, these taxa experience high risk from floating oil (*2*, *6*). Oiling of fur or feathers causes loss of insulating capacity and can lead to death from hypothermia, smothering, drowning, and ingestion of toxic hydrocarbons. Accordingly, mass mortalities of 1000 to 2800 sea otters (*9*) and unprecedented numbers of seabird deaths estimated at 250,000 (*10*) were documented during the days after the spill. An estimated 302 harbor seals, a short-haired marine mammal, were killed not by oiled pelage but likely from inhalation of toxic fumes leading to brain lesions, stress, and disorientation (*2*). Mass mortality also occurred among macroalgae and benthic invertebrates on oiled shores from a combination of chemical toxicity, smothering, and physical displacement from the habitat by pressurized wash-water applied after the spill (*5*, *7*).

Persistence of Oil: Ecosystem Sequestration

Only early phases of transport and transformation of the petroleum hydrocarbons followed expectations (*11*). About 40 to 45% of the oil mass grounded in 1989 on 787 km of PWS beaches; another 7 to 11% was transported to contaminate 1203 km of Gulf of Alaska shoreline (*11*, *12*). About 2% remained on intertidal PWS beaches after 3.5 years (*11*); this reflected an exponential decay rate of -0.87 year^{-1}, which in turn produced a loss of 58% over a year. Unexpectedly (*3*), rates of dispersion and degradation diminished through time, as most oil remaining after October 1992 was sequestered in environments where degradation was suppressed by physical barriers to disturbance, oxygenation, and photolysis (*12*). A 2001 survey of intertidal PWS shorelines revealed 55,600 kg of often little weathered, Exxon Valdez oil in intertidal subsurface sediments and a perhaps equal mass of high-intertidal degraded surface oil and lower-intertidal, minimally weathered subsurface oil (*13*). This represents a decay rate from 1992–2001 of only -0.22 to -0.30 year^{-1} (20 to 26% loss over a year) from the 806,000 kg estimated to be present on PWS beaches in 1992.

Sedimentary refuges inhibited degradation and sequestered persistently toxic oil in the intertidal zone of coarse-grained gravel shores where geomorphologic armoring by boulders and cobbles inhibited disturbance by waves (*12*). Some of this oil was similarly trapped under mussel beds providing an

enduring route of entry into many food chains (*14*). The subsurface cobbles and gravels of stream banks (*15*) harbored biologically available oil, exposing and killing pink salmon embryos through at least 1993 (*16*). Thus, heavily oiled coarse sediments formed and protected subsurface reservoirs, sequestering oil from loss and weathering in intertidal habitats containing fish eggs and invertebrate predators (sea otters, seaducks, and shorebirds).

Long-Term Population Impacts

Chronic exposures of sediment-affiliated species. Chronic exposures for years after the spill to oil persisting in sedimentary refuges were evident from biomarkers in fish (*17*), sea otters (*18*), and seaducks (*19*) intimately associated with sediments for egg laying or foraging. These chronic exposures enhanced mortality for years. In 1989, prediction of oil risk to fishes was based largely on testing acute toxicity in short-term (~4-day) laboratory exposures to the water-soluble fraction dominated by 1- and 2-ringed aromatic hydrocarbons (*8*). After the spill, fish embryos and larvae were chronically exposed to partially weathered oil in dispersed forms that accelerate dissolution of 3-, 4-, and 5-ringed hydrocarbons largely missing from the traditional laboratory toxicity assays (*15*). Laboratory experiments showed that these multiringed polycyclic aromatic hydrocarbons (PAHs) from partially weathered oil at concentrations as low as 1 ppb are toxic to pink salmon eggs exposed for the months of development and to herring eggs exposed for 16 days (*20*, *21*). This process explains the elevated mortality of incubating pink salmon eggs in oiled rearing streams for at least 4 years after the oil spill (*16*).

After 1989, sea otter recovery of about 4% per annum (averaged throughout western PWS) has fallen far short of the 10% expected from earlier population recovery after termination of trade in sea otter pelts (*22*). At heavily oiled northern Knight Island, sea otters have remained at half the estimated prespill numbers with no recovery initiated by 2000, whereas an unoiled Montague Island population doubled just in the period from 1995 to 1998 (*23*). Spring carcass collections in 1976–85 and again in 1989–98 produced age-at-death data, which allowed population modeling to demonstrate that sea otter survival in the oiled portion of PWS was generally lower in the years after the spill and declined rather than increased after 1989 (*24*). This response surprisingly included higher mortality of animals born after the spill, implicating a substantial contribution from chronic exposure. Persistent exposure to oil in 1996–98 is confirmed by higher levels of the detoxification enzyme CYP1A in individuals from northern Knight than from Montague Island (*18*). Abundance of sea otter prey (clams, mussels, crabs) did not differ between Knight and Montague during this period, so prey availability fails to explain suppression of population recovery (*23*). Suspension-feeding clams and mussels concentrate and only slowly metabolize hydrocarbons, which leads to chronically elevated tissue contamination that persisted in one prominent prey of sea otters, the clam *Protothaca staminea*, until at least 1996 (*7*). Sediments in protected areas, including oiled mussel beds and shallow eelgrass habitats (*25*), also retained contamination, with recovery to background in oiled mussel beds estimated from repeated sampling to require up to 30 years (*14*). Thus, foraging sea otters suffered chronic exposure to residual petroleum hydrocarbons from both sediment contact and ingestion of bivalve prey. In contrast, piscivorous river otters showed little evidence of chronic oil exposure even along heavily oiled shorelines, implying that foraging in sediments entails greater risk (*18*).

Among marine birds, harlequin ducks exhibited the most unanticipated chronic impact. Radio tracking of adult females revealed higher mortality rates while overwintering in 1995–96 through 1997–98 on heavily oiled Knight and Green Island shores (22%) than on unoiled Montague Island (16%), a difference with significant implications for population trajectories (*26*). Harlequin ducks, which prey on intertidal benthic invertebrates, showed induction of the CYP1A detoxification enzyme in 1998, which in the absence of corresponding patterns in other potential inducers like polychlorinated biphenyls (PCBs) indicates ongoing exposure to oil 9 years after the spill (*20*). Body mass of harlequins in late winter was negatively related to CYP1A levels in 1998, which suggests that a mechanism involving energetics led to the observed elevation in over-winter mortality rates (*27*). Reflecting the sensitivity of harlequin duck population dynamics to adult female survival, fall PWS densities of harlequins on oiled shores declined at an annual rate of about 5% in 1995-97, as compared with stable numbers on unoiled shores (*26*).

Other marine birds that forage in shallow sediments showed evidence of persistent exposure to residual oil after the spill. Barrow's goldeneye, a seaduck that overwinters in coastal Alaska and forages in intertidal mussel beds, declined in abundance in oiled relative to unoiled bays immediately after the spill with no evidence of recovery through 1991 (*28*). Along oiled Knight Island shorelines, Barrow's goldeneye showed chronic exposure to oil into winter 1996–97, as evidenced by induction of CYP1A (*19*). The association between foraging on littoral benthic invertebrates and chronic exposure to residual toxins from the oil is illustrated by differences among age classes in pigeon guillemots. This seabird, which restricts its foraging to the near-shore environment, suffered acute mortality during the spill (*10*). In 1999, 10 years after the oil spill, the chicks of pigeon guillemots, which are fed only fish, showed no evidence of ongoing exposure to toxics, whereas the adults, which include shallow-water benthic invertebrates in their diets, had elevated CYP1A in their livers (*29*).

Sublethal exposures leading to death from compromised health, growth, or reproduction. Several studies documented cascades of

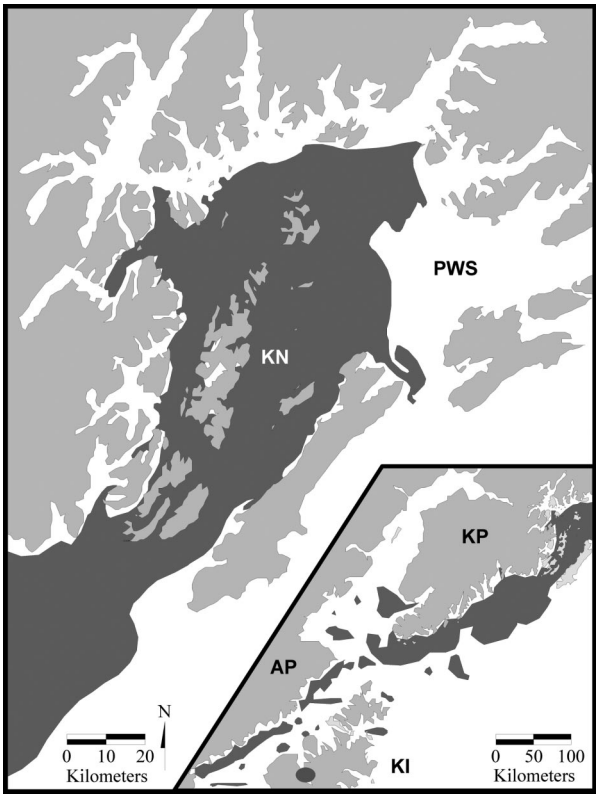

Fig. 1. Map of the spread of oil and the shorelines (indicated in black) contaminated to some degree after the grounding of the Exxon Valdez at Bligh Reef in northern Prince William Sound. Oil was transported to the southwest, striking Knight (KN) and other PWS islands, the Kenai Peninsula (KP), the Kodiak Island archipelago (KI), and the Alaska Peninsula (AP).

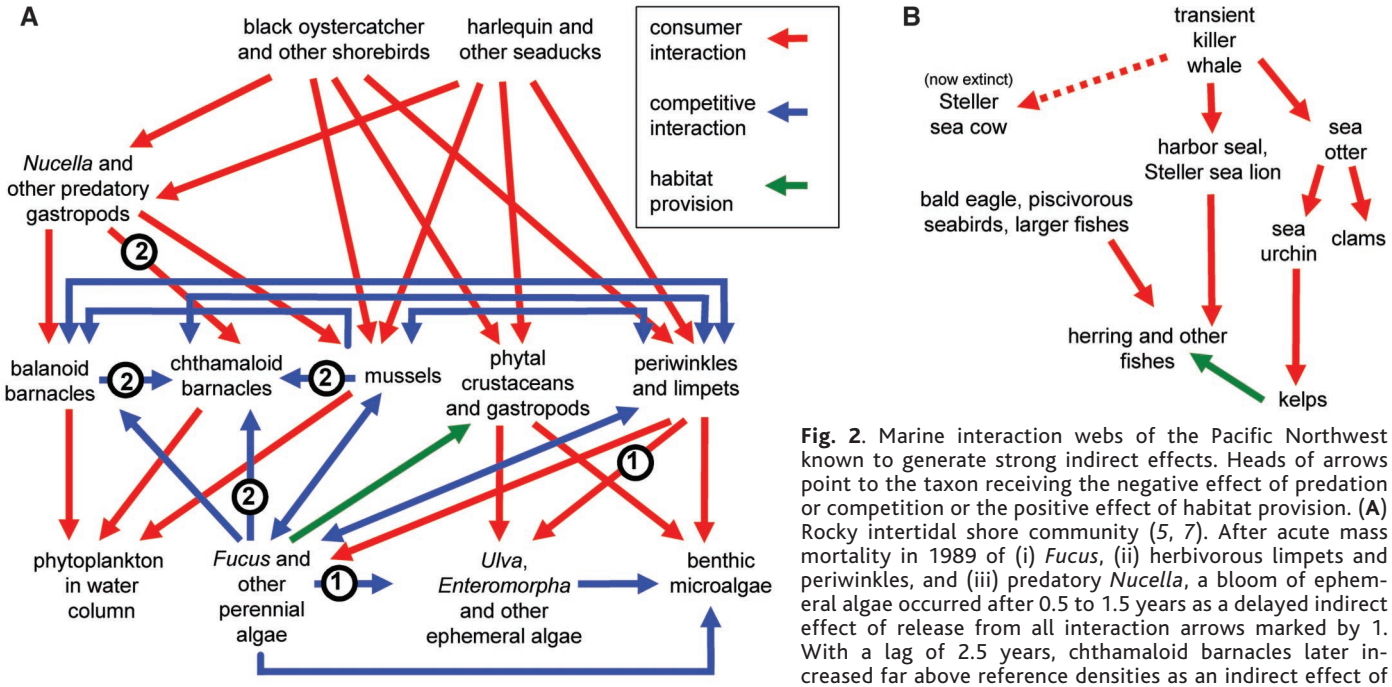

Fig. 2. Marine interaction webs of the Pacific Northwest known to generate strong indirect effects. Heads of arrows point to the taxon receiving the negative effect of predation or competition or the positive effect of habitat provision. (**A**) Rocky intertidal shore community (*5*, *7*). After acute mass mortality in 1989 of (i) *Fucus*, (ii) herbivorous limpets and periwinkles, and (iii) predatory *Nucella*, a bloom of ephemeral algae occurred after 0.5 to 1.5 years as a delayed indirect effect of release from all interaction arrows marked by 1. With a lag of 2.5 years, chthamaloid barnacles later increased far above reference densities as an indirect effect of release from all interaction arrows marked by 2 (*4*, *7*). (**B**) Subtidal kelp forest community (*36*, *41*). Despite acute loss of over 50% of the sea otters at heavily oiled northern Knight Island, there exists only limited evidence of initiation of this potentially strong trophic cascade. Some patches of larger sea urchins have appeared but no explosion of their abundance and no evident overgrazing of kelp have been seen even in the absence of sea otter recovery to date (*22*, *23*).

events indirectly affecting individual survival or reproduction after sublethal exposures. Oil exposure resulted in lower growth rates of salmon fry in 1989 (*8*), which in pink salmon reduce survivorship indirectly through size-dependent predation during the marine phase of their life history (*30*). After chronic exposures as embryos in the laboratory to <20 ppb total PAHs, which stunted their growth, the subsequently marked and released pink salmon fry survived the next 1.5 years at sea at only half the rate of control fish (*21*). In addition, controlled laboratory studies showed reproductive impairment from sublethal exposure through reducing embryo survivorship in eggs of returning adult pink salmon that had previously been exposed in 1993 to weathered oil as embryos and fry (*31*). These definitive experimental demonstrations of compromised survival and reproduction from sublethal dosing conform with a growing understanding of how exposure to xenobiotics at sensitive early stages in vertebrate development can lead to enhanced mortality and reproductive impairment later in life through endocrine disruption and developmental abnormalities (*32*). Abnormal development occurred in herring and salmon after exposure to the Exxon Valdez oil (*14*, *20*).

Support for the inference that sublethal effects of chronic exposure to toxics through ingestion of oil led to population-level impacts on shorebirds comes from studies of the black oystercatcher. In summer 1989, pairs of black oystercatchers with foraging territories on heavily oiled shores showed reduced incidence of breeding and smaller eggs than those that bred elsewhere (*33*). Chick mortality was enhanced in proportion to degree of shoreline oiling in both 1989 and 1990. Subsequent study (*34*) revealed that black oystercatchers indeed consumed oiled mussels and that parents gathering prey on oiled shores in 1991 and 1992 fed chicks more to achieve less growth than on unoiled shores, which implies energetic or developmental costs and reproductive impairment from ingestion of toxics 3 years after the spill. Fledging late or at small size has negative implications for chick survivorship.

Cascades of indirect effects. Indirect effects can be as important as direct trophic interactions in structuring communities (*35*). Cascading indirect effects are delayed in operation because they are mediated through changes in an intermediary. Perhaps the two generally most influential types of indirect interactions are (i) trophic cascades in which predators reduce abundance of their prey, which in turn releases the prey's food species from control (*36*); and (ii) provision of biogenic habitat by organisms that serve as or create important physical structure in the environment (*37*). Current risk assessment models used for projecting biological injury to marine communities ignore indirect effects, treating species populations as independent of one another (*7*, *8*), even in rocky-shore systems, where basic community ecology would indicate otherwise (*38*).

Indirect interactions (Fig. 2A) lengthened the recovery process on rocky shorelines for a decade or more (*7*). Dramatic initial loss of cover by the most important biogenic habitat provider, the rockweed *Fucus gardneri*, triggered a cascade of indirect impacts. Freeing of space on the rocks and the losses of important grazing (limpets and periwinkles) and predatory (whelks) gastropods combined to promote initial blooms of ephemeral green algae in 1989 and 1990 and an opportunistic barnacle, *Chthamalus dalli*, in 1991. Absence of structural algal canopy led to declines in associated invertebrates and inhibited recovery of *Fucus* itself, whose recruits avoid desiccation under the protective cover of the adult plants. Those *Fucus* plants that subsequently settled on tests of *Chthamalus dalli* became dislodged during storms because of the structural instability of the attachment of this opportunistic barnacle. After apparent recovery of *Fucus*, previously oiled shores exhibited another mass rockweed mortality in 1994, a cyclic instability probably caused by simultaneous senility of a single-aged stand (*5*, *39*). The importance of indirect interactions in rocky shore communities is well established (*38*), and 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 (*40*). Expectations of rapid recovery based on short

generation times of most intertidal plants and animals are naïve and must be replaced by a generalized concept of how interspecific interactions will lead to a sequence of delayed indirect effects over a decade or longer (*7*).

Indirect interactions are not restricted to trophic cascades or to intertidal benthos. Interaction cascades defined broadly include loss of key individuals in socially organized populations, which then suffer subsequently enhanced mortality or depressed reproduction. After exceptionally high mortality of 20% between September 1988 and spring 1989 and another 20% during the following year in the AB pod of resident (fish-eating) killer whales that had been observed to swim through the spill, losses of adult females from these matriarchially organized family groups led to suppressed reproduction (*2*). In another pod (AT1) of transient (mammal-eating) killer whales, the 40% loss during the spill is leading to likely disintegration (*2*). Furthermore, the most compelling example in all of marine ecology of a trophic cascade radically modifying a marine community comes from the Gulf of Alaska kelp ecosystem (*36*). Unless eliminated by killer whales that have lost their traditional, larger marine mammal prey (*41*), sea otters control sea urchin populations, preventing them from overgrazing kelp and other macroalgae, and thereby retaining structural habitat for fishes and invertebrates (Fig. 2B). Given the spill loss of about 50% of the sea otters from PWS, there is potential for this cascade to influence recovery dynamics, but evidence of its operation to date is limited to reduction in otter foraging and increase in urchin sizes (*18*). Nevertheless, should sea otters be eliminated from an area by an oil spill, the repeatability of the otter-urchin-kelp cascade is sufficiently strong that risk assessment models can confidently include its implications. In contrast, limited understanding of the importance of behaviorally mediated indirect effects in driving community dynamics (*42*) still prevents their inclusion in risk modeling.

Table 1. Changing paradigms in oil ecotoxicology, moving from acute toxicity based on single species toward an ecosystem-based synthesis of short-term direct plus longer-term chronic, delayed, and indirect impacts.

Old paradigm	Emerging appreciation
Physical shoreline habitat	
Oil that grounds on shorelines other than marshes dominated by fine sediments will be rapidly dispersed and degraded microbially and photolytically.	Oil degrades at varying rates depending on environment, with subsurface sediments physically protected from disturbance, oxygenation, and photolysis retaining contamination by only partially weathered oil for years.
Oil toxicity to fish	
Oil effects occur solely through short-term (~4 day) exposure to water-soluble fraction (1- to 2-ringed aromatics dominate) through acute narcosis mortality at parts per million concentrations.	Long-term exposure of fish embryos to weathered oil (3- to 5-ringed PAHs) at ppb concentrations has population consequences through indirect effects on growth, deformities, and behavior with long-term consequences on mortality and reproduction.
Oil toxicity to seabirds and marine mammals	
Oil effects occur solely through short-term acute exposure of feathers or fur and resulting death from hypothermia, smothering, drowning, or ingestion of toxics during preening.	Oil effects also are substantial (independent of means of insulation) over the long term through interactions between natural environmental stressors and compromised health of exposed animals, through chronic toxic exposure from ingesting contaminated prey or during foraging around persistent sedimentary pools of oil, and through disruption of vital social functions (care giving or reproduction) in socially organized species.
Oil impacts on coastal communities	
Acute mortality through short-term toxic exposure to oil deposited on shore and the shallow seafloor or through smothering accounts for the only important losses of shoreline plants and invertebrates.	Clean-up attempts can be more damaging than the oil itself, with impacts recurring as long as clean-up (including both chemical and physical methods) continues. Because of the pervasiveness of strong biological interactions in rocky intertidal and kelp forest communities, cascades of delayed, indirect impacts (especially of trophic cascades and biogenic habitat loss) expand the scope of injury well beyond the initial direct losses and thereby also delay recoveries.

Implications of Changing Paradigms of Oil Ecotoxicity

It is well known that acute tests of toxicity in the laboratory are insufficient for ecotoxicological risk assessment (*43*). It has also been clear that tests of chronic exposures are needed to fully understand impacts of petroleum and other toxins in the marine environment (*6*). Support grows for inclusion of a range of physiological, biochemical, and histopathological evaluations of toxicity, facilitated by rapid development of molecular tools. Furthermore, ecologists have long acknowledged the potential importance of interaction cascades of indirect effects. Now synthesis of 14 years of Exxon Valdez oil spill studies documents the contributions of delayed, chronic, and indirect effects of petroleum contamination in the marine environment (Table 1). Expanding the scope of the fundamental basis of ecotoxicology beyond reliance on short-term acute toxicity to include delayed, chronic, and indirect effects operating over longer periods is analogous to developing ecosystem-based management of forest (*44*) and fisheries (*45*) resources to embrace the nexus of ecosystem interactions. Our synthesis implies necessary modifications of environmental standards for water quality, stormwater control, chronic low-level oil releases, and many other human activities. Vague concerns about the role of poor water quality in the steady declines of estuarine-dependent fisheries may now find renewed focus on a specific class of contaminants, the multi-ringed PAHs, in physically protected sedimentary spawning and nursery habitats. In light of delayed impacts of the Exxon Valdez (Table 1) and the San Cristobal oil spill in the Galapagos Islands during 2001 (*46*), the growing role played by risk assessment modeling in a priori environmental decision making and a posteriori estimation of natural resource injury needs reconsideration. Much incentive exists for advancing the predictive capacity of ecology to allow more confident modeling of chronic, indirect, and delayed effects of stressors through ecosystem-based frameworks.

References and Notes

1. Preparation of this review involved synthesis of an extensive and still-growing literature. The associated Supporting Online Material (SOM) identifies additional sources of relevant information.
2. T. R. Loughlin, Ed., *Marine Mammals and the "Exxon Valdez"* (Academic Press, San Diego, 1994).
3. P. G. Wells, J. N. Butler, J. S. Hughes, Eds., *"Exxon Valdez" Oil Spill: Fate and Effects in Alaskan Waters* [ASTM (American Society for Testing and Materials), Philadelphia, 1995].

4. S. D. Rice, R. B. Spies, D. A. Wolfe, B. A. Wright, Eds., *Proceedings of the "Exxon Valdez" Oil Spill Symposium* (American Fisheries Society, Bethesda, MD, 1996).
5. R. T. Paine *et al.*, *Annu. Rev. Ecol. Syst.* **27**, 197 (1996).
6. National Research Council, *Oil in the Sea III: Inputs, Fates, and Effects* (National Academy Press, Washington, DC, 2002).
7. C. H. Peterson, *Adv. Mar. Biol.* **39**, 1 (2001).
8. S. D. Rice *et al.*, *Rev. Fish. Sci.* **9**, 165 (2001).
9. R. A. Garrott, L. L. Eberhardt, D. M. Burn, *Mar. Mammal Sci.* **9**, 343 (1993).
10. J. F. Piatt, R. G. Ford, *Am. Fish. Soc. Symp.* **18**, 712 (1996).
11. D. A. Wolfe *et al.*, *Environ. Sci. Tech.* **28**, 561A (1994).
12. M. O. Hayes, J. Michel, *Mar. Poll. Bull.* **38**, 92 (1999).
13. J. W. Short *et al.*, *Environ. Sci. Technol.*, **38**, 19 (2004).
14. M. G. Carls *et al.*, *Mar. Environ. Res.* **51**, 167 (2001).
15. M. L. Murphy *et al.*, *Trans. Am. Fish. Soc.* **128**, 909 (1999).
16. B. G. Bue, S. Sharr, J. E. Seeb, *Trans. Am. Fish. Soc.* **127**, 35 (1998).
17. S. C. Jewett, T. A. Dean, B. R. Woodin, M. K. Hochberg, J. J. Stegeman, *Mar. Environ. Res.* **54**, 21 (2002).
18. J. L. Bodkin *et al.*, *Mar. Ecol. Prog. Ser.* **241**, 237 (2002).
19. K. A. Trust, D. Esler, B. R. Woodin, J. J. Stegeman, *Mar. Poll. Bull.* **40**, 397 (2000).
20. G. D. Marty *et al.*, *Can. J. Zool.* **75**, 989 (1997).
21. R. A. Heintz *et al.*, *Mar. Ecol. Prog. Ser.* **208**, 205 (2001).
22. J. L. Bodkin, B. E. Ballachey, M. A. Cronin, K. T. Scribner, *Conserv. Biol.* **13**, 1378 (1999).
23. T. A. Dean, J. L. Bodkin, S. C. Jewett, D. H. Monson, D. Jung, *Mar. Ecol. Prog. Ser.* **199**, 281 (2000).
24. D. H. Monson, D. F. Doak, B. E. Ballachey, A. M. Johnson, J. L. Bodkin, *Proc. Natl. Acad. Sci. U.S.A.* **97**, 6562 (2000).
25. S. C. Jewett, T. A. Dean, R. O. Smith, A. Blanchard, *Mar. Ecol. Prog. Ser.* **185**, 59 (1999).
26. D. Esler, J. A. Schmutz, R. L. Jarvis, D. M. Mulcahy, *J. Wildl. Manag.* **64**, 839 (2000).
27. D. Esler *et al.*, *Mar. Ecol. Prog. Ser.* **241**, 271 (2002).
28. R. H. Day *et al.*, *Ecol. Appl.* **7**, 593 (1997).
29. G. H. Golet *et al.*, *Mar. Ecol. Prog. Ser.* **241**, 287 (2002).
30. M. Willette, R. T. Cooney, K. Heyer, *Can. J. Fish. Aquat. Sci.* **56**, 364 (2000).
31. R. A. Heintz, J. W. Short, S. D. Rice, *Environ. Toxicol. Chem.* **18**, 494 (1999).
32. M. R. Arkoosh, T. K. Collier, *Human Ecol. Risk Assess.* **8**, 265 (2002).
33. B. E. Sharp, M. Cody, R. Turner, *Am. Fish. Soc. Symp.* **18**, 748 (1996).
34. B. A. Andres, *J. Wildl. Manag.* **61**, 1322 (1997).
35. T. H. Schoener, in *Mutualism and Community Organization: Behavioural, Theoretical, and Food-Web Approaches*, H. Kawanabe, J. E. Cohen, K. Iwasaki, Eds., (Oxford Univ. Press, New York, 1993), pp. 365–411.
36. J. E. Estes, D. O. Duggins, *Ecol. Monogr.* **65**, 75 (1995).
37. C. G. Jones, J. H. Lawton, M. Shachak, *Oikos* **69**, 373 (1994).
38. B. A. Menge, *Ecol. Monogr.* **65**, 21 (1995).
39. D. C. Driskell, J. L. Ruesink, D. C. Lees, J. P. Houghton, W. B. Lindstrom, *Ecol. Appl.* **11**, 815 (2001).
40. S. J. Hawkins, A. J. Southward, in *Restoring the Nation's Marine Environment*, G. W. Thayer, Ed. (Maryland Sea Grant Publication, College Park, 1992), pp. 584–631.
41. J. E. Estes, M. T. Tinker, T. M. Williams, D. F. Doak, *Science* **282**, 473 (1998).
42. L. M. Dill, M. R. Heithaus, C. J. Walters, *Ecology* **84**, 1151 (2003).
43. K. D. Kimball, S. A. Levin, *Bioscience* **35**, 135 (1985).
44. N. L. Christensen *et al.*, *Ecol. Appl.* **6**, 664 (1996).
45. L. W. Botsford, J. C. Castilla, C. H. Peterson, *Science* **277**, 509 (1997).
46. M. Wikelski, V. Wong, B. Chevalier, N. Rattenborg, H. L. Snell, *Nature* **417**, 607 (2002).

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Supporting Online Material

www.sciencemag.org/cgi/content/full/302/5653/2082/DC1
SOM Text
References and Notes