Exxon Valdez Oil Spill (EVOS) Legacy: Shifting Paradigms in Oil Ecotoxicology[1]

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Abstract. *Oil is much more toxic to coastal fish, birds, and mammals than previously predicted by short-term laboratory bioassay studies used during the 1970s and 1980s to develop a "paradigm" or model understanding of oil toxicity. Hundreds of comprehensive field assessments and lab studies conducted by government and academic researchers after the Exxon Valdez oil spill (EVOS) show that oil is persistent in important shoreline environments and causes long-term, population-level injury to coastal sealife. These 1990s studies frame a new oil toxicity paradigm, showing that risk evaluation or "ecotoxicity" models developed in the 1970s severely understate environmental damage from chronic oil pollution. Public policies based on the 1970s oil toxicity paradigm are not adequately protective of sealife. Policies guiding every phase of oil use from production to consumption and waste disposal need to be reevaluated in light of the 1990s oil toxicity paradigm.*

1970s Oil Toxicity Paradigm: History & Limitations

With the passage of the federal Clean Water Act in 1972, scientists developed standards to protect fish and wildlife in marine and fresh water environments from harmful levels of oil, among other chemicals. Scientists used short-term (usually 96-hour) laboratory "bioassays" as a way of exposing organisms to oil dissolved in the water column or the "water soluble fraction" (WSF) and then measuring the effects of this exposure (usually as mortality) to determine what levels of oil were harmful (*1*).

The oil toxicity paradigm[3] that emerged as a result of these bioassays (*2*) held that the primary compounds of concern in crude oil, which is composed of hundreds of different hydrocarbons, were the 1- and 2-ring aromatic hydrocarbons, which dissolve rapidly in water or air. Other larger aromatic hydrocarbons (3-5 rings) were more toxic, but they did not dissolve or mix into the water rapidly, and were not a factor in the short-term bioassays. The 1- and 2-ring aromatic hydrocarbons were quick in toxic action, but also short in duration – easily diminished by dilution, volatilization, and dispersal. Hence, the 1970s oil toxicity paradigm was based on *acute* toxicity, with toxic concentrations to fish and invertebrates in the low parts per million. There was some concern for long-term toxicity and safety factors were

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[3]*Note: in this paper, a "paradigm" is a theoretical framework created by scientists to explain a functional relationship in natural science. Paradigms are fluid, not static, models and they shift to accommodate new understanding as science advances. For example, a scientific paradigm once held that the world was flat, but we no longer believe this.*

suggested by guess work: for example, 1 / 100th of the acute toxicity *should* be safe.

What was Wrong with the 1970s Acute Toxicity Paradigm for Oil?

There are two basic problems with the acute toxicity model that evolved out of the 1970s – persistence and toxic mechanism. First, persistence of toxic compounds was not considered to be a problem. 1- and 2-ring aromatics do not persist in the natural environment, but their larger 3- , 4- , 5- ring cousins can. The larger compounds are not volatile, not soluble, but much more difficult to degrade. Second, the mechanism for toxicity is very different between the small and large aromatics. The 1- and 2-ring aromatics are toxic to membrane function and cause a "narcosis" type of toxicity. This acts quickly and leads to a variety of system failures. Fish, for example, loose equilibrium when exposed to 1-ring aromatics, there are function failures in gills and other organs, and the fish typically die within minutes of exposure.

In contrast, the mechanism of toxicity for larger compounds operates within the cell, where proteins and DNA can be directly affected. Embryos will suffer injury where cellular DNA is damaged and then replicated during embryonic growth, creating more cells with damaged DNA. Rather than causing an acute narcosis death, this damage affects "fitness" and results in a juvenile that is less capable of normal growth, avoiding predators, or capturing prey. In contrast to a direct narcosis death, this mechanism is more indirect (getting eaten, for example), but the result is still a loss in numbers of recruiting individuals. Populations slowly decline.

The limitations of the 1970s oil toxicity paradigm, based on acute toxicity, are such that it cannot be used to predict oil toxicity in an environment where oil may persist for some time (*1*). Acute bioassays were designed originally to measure potency of insecticides, not assess environmental safety to wildlife, where there are complex and long-term interactions among growth, body condition, maturation, diseases, reproduction, and predation.

Until now after results of EVOS studies have been compiled, resource and environmental managers only had available the 1970s oil toxicity paradigm to use to establish water quality standards and develop environmental risk models. A body of public policy (environmental laws) emerged, based on the 1970s paradigm, supposedly to protect aquatic and marine life from oil pollution.

1990s Oil Toxicity Paradigm & Supporting Studies

The 1970s oil toxicity paradigm failed to predict the long-term impacts of the EVOS, stemming from persistent oiling and subsequent bioavailability of oil in critical nearshore habitats (*1, 3*). As part of the Restoration Program undertaken by the federal and Alaska state governments, scientists designed comprehensive field and lab studies to explore and explain the population-level impacts that occurred, notably, in Prince William Sound, where nearly half of the oil from the *Exxon Valdez* had stranded on beaches (*4*). These studies and the resulting 400+ peer-reviewed papers frame the new 1990s oil toxicity paradigm. The persistence of substantial amounts of oil for more than a decade in biologically important, protected shoreline habitats, such as deltas of anadromous fish

streams, mussel beds, and boulder-cobble shores (*1, 3*), was unanticipated and has induced the long-term exposures that underlie the new 1990s paradigm.

The 1990s oil toxicity paradigm holds that the compounds of concern are not the 1- and 2-ring aromatic hydrocarbons but 3-, 4- , 5-ring PAHs, or polycyclic aromatic hydrocarbons that were ignored in the 1970s paradigm. PAHs are persistent and bioavailable: PAHs are toxic during chronic exposure to early developmental life stages of herring and pink salmon at 0.4 to 1 part per *billion*, respectively, or levels 1,000 times lower than predicted by the 1970s paradigm (*5, 6*). A range of maladies was found in a variety of fish, birds, and mammals from field exposure to PAHs at levels of low parts per billion (ppb) (Table 1). Both direct and indirect effects were reported. In brief, these findings are as follows.

FISH. After the EVOS, weathered oil characterized by 3- , 4- , 5-ring PAHs was trapped in protected beach environments such as subsurface groundwater of anadromous fish streams for at least 4-8 years (*7*). PAHs were bioavailable to embryos and larvae of pink salmon as the PAHs were absorbed across the yolk membrane of eggs: prolonged exposures for months during incubation to levels as low as 1 ppb were found to be toxic (*6*). In addition to enhanced embryo mortality through chronic exposure to PAHs in weathered oil in groundwater (*8*), "sublethal" (not directly toxic) oil exposure led to population-level impacts. Evidence of higher rates of abnormal development and larval deformity in pink salmon and herring following oil exposure imply enhanced mortality (*5, 6*). Exposure of salmon fry to *Exxon Valdez* oil resulted in lower growth rates in 1989 and increased subsequent mortality through predation (*9, 10*). Finally, controlled laboratory studies of embryo development demonstrated reproductive impairment in the form of lower embryo survival of eggs from returning adult pink salmon that had been exposed to PAHs in weathered oil in streams during incubation as eggs and fry (*11*).

The 1990s paradigm of oil ecotoxicity to fishes incorporates both enhanced embryo mortality and delayed reproductive impacts of chronic exposure of embryos to persistent PAHs in weathered oil at low ppb concentrations, and it includes population-level consequences of sublethal impacts on growth of juvenile stages.

MARINE MAMMALS. Prior to the EVOS the widely accepted risk assessment model predicting population-level impacts to marine mammals and seabirds held that this wildlife had to be physically oiled and the resulting loss of insulation to fur or feathers led to hypothermia, drowning, and death. While the EVOS confirmed this model during the early weeks of the spill in that thousands of sea otters (*12*) and hundreds of thousands of seabirds (*13*) died from physical contact with oil, researchers also found that other processes caused previously unanticipated long-term population-level effects.

Smooth-skinned mammals–documented for harbor seals (*14*) and killer whales (*15*)–declined in abundance in 1989 in oiled areas of Prince William Sound. Brain lesions, evident in necropsies of seals implicate inhalation of toxic fumes, the 1- and 2-ring aromatics, and were considered to have caused mortality through observed behavioral disorientation, lethargy, and stress response (*16*). Killer whales in Prince

William Sound experienced unprecedented losses in the years following the spill. Early losses arose from direct toxic exposures, whereas long-term, delayed

Table 1. Evidence of Effects of Chronic Oil Pollution. Examples of species, life stage, connection to the intertidal zone, and lowest level of PAHs causing effect (in parts per billion). "Elevated P450 enzyme" indicates PAHs are bioavailable; further effects as noted. From Peterson (2001) and Rice et al. (2001).

Species	Life Stage	PAH s (ppb)	Connection to intertidal (Effect)
Pink salmon	Embryo	1 µg / g	Early development (death, genetic damage to 1st, 2nd generation)
Pink salmon	Juvenile	1 µg / g	Nursery (decreased growth & reduced marine survival)
Dolly Varden char	Juvenile, adult	low ppb	Forage (decreased growth for 1 yr)
Cut-throat trout	Juvenile, adult	low ppb	Forage (decreased growth for 2 yr)
Pacific herring	Egg, embryo	1 µg / g	Early development (death)
Black oystercatchers	Adult	low ppb	Nest (delayed recovery due to problems with rearing chicks)
Harlequin ducks	Adult	low ppb	Forage on mussels (depressed over winter survival of females, 9 yr)
Barrow's goldeneye	Adult	low ppb	Forage on mussels (depressed recovery, elevated P450 enzyme, 9 yr)
Cormorants, murres, black-legged kittiwake, pigeon guillemot (PG), loons, mergansers	Adult	low ppb	Forage on high lipid fish (delayed recovery for 9 yr (loons 5 yr); PG lower productivity of young, elevated P540 enzyme 9 yr)
Masked greenling	Adult	0.40 µg / g	Resident (elevated P450 enzyme up to 7 years post spill)
Sea otters	Juvenile	low ppb	Forage on mussels (high mortality for up to 3 yrs)
Sea otters	Adult	low ppb	Forage (high mortality of prime breeding age adults for 5 yr)
River otters	Adult	low ppb	Forage (expanded feeding territories, poor condition, elevated P450 enzyme

effects on survival, reproduction, and recruitment success were the indirect consequences of loss of parents and experienced older members, disrupting the social structure of the pods (*17*).

In addition to the thousands of early sea otters deaths caused by acute toxicity, long-term studies revealed processes inhibiting recovery of otters in heavily oiled areas. Intensive documentation of sea otter population dynamics for over a decade after the EVOS revealed a reduced population growth rate and increased death rate of prime-age and juvenile sea otters in oiled areas of Prince William Sound (*18*). Sea otters feed heavily on clams that they dig out of eelgrass beds and on mussels and crabs. Clams and mussels sequester (absorb and store in their bodies) oil hydrocarbons: sediment in eelgrass beds and under mussel beds remained contaminated with PAHs from *Exxon Valdez* oil, which remained bioavailable to sea otters through their shellfish diet (*19*).

The 1990s paradigm of oil ecotoxicity to marine mammals recognizes risk from inhalation of toxic fumes, behavioral interdependencies among social animals, and long-term exposure to oil through diet and residual weathered oil in sediments.

SEABIRDS. Guilds of seabirds that feed in nearshore habitats suffered greater initial declines, delayed declines, and delayed recovery compared to those that feed offshore (*20, 21*). In particular, species of seaduck that feed heavily on mussels such as Barrow's goldeneyes and harlequins showed no evidence of recovery through the 1998 survey (*22*) and continued exposure to PAHs, as evidenced by high levels of enzymes that metabolize or break down oil (*23*). For years after the EVOS, harlequins experienced high over-wintering mortality rates and continued population decline in oiled areas of Prince William Sound (*24*). Black oystercatchers, a shorebird that feeds heavily on mussels, also had reduced incidence of breeding, smaller eggs, and reduced growth of offspring in oiled areas in 1989 (*25*). Results of studies on seabirds imply that energetic costs of metabolizing oil ingested through diet are substantial and create sublethal effects on growth, body condition, and reproduction (*26*) with population-level impacts (*27*).

The 1990s paradigm of oil ecotoxicity to seabirds recognizes risk from long-term exposure to oil through diet and subsequent sublethal effects on reproduction, growth, and survival with population-level impacts.

INDIRECT EFFECTS. The current risk assessment models used for predicting population-level effects of oil pollution lack all indirect effects and treat species populations as independent of one another. Studies after the EVOS demonstrated two main types of indirect effects in communities of sealife associated with rocky shores: loss of critical habitat through loss of species that provide structural habitat and "trophic-level" (food web) interactions among species (*3*).

The macroalga *Fucus* provides critical habitat, a virtual seaweed forest, for a variety of marine invertebrates that serve as prey for seabirds and shorebirds, sea and land mammals, and young pelagic and benthic fish (*3*). Dramatic loss of *Fucus* in the intertidal zone by oiling and the pressurized hot water (*28*) wash inhibited recovery of both the *Fucus* itself, which depends upon recruits being protected from desiccation by the seaweed canopy

(*29*), and also the community of invertebrates that shelters under the seaweed (*30*). The subsequent sequence of community development and species succession extended over a decade as opportunistic species of fauna and flora were gradually replaced by single-aged stands of *Fucus*, which died in cycles, starting the whole process again (*31*).

In the Gulf of Alaska, large reductions in sea otter populations, not spill-related, have been shown to predictably reduce predation on sea urchins, which then can experience a population explosion and overgraze their kelp and macroalgal foods. The consequent loss of the kelp forests has dramatic negative impacts on the fish and invertebrate community that resides within the forest and subsequently on the seabirds and marine mammals that prey on these resources (*32*). The potential for such a trophic cascade existed in Prince William Sound after the EVOS, but it was not fully realized as only the initial phase of increased sizes of sea urchins was documented in oiled areas with depleted sea otter populations (*19*).

Another indirect trophic impact, however, was realized in Prince William Sound when populations of important species of forage fish crashed after the EVOS (*33*). Herring in particular are critically important to seabirds and marine mammals because of their high lipid (fat) content and surface schooling habits, making them nutritious and easy to capture (*34*). Several fish-eating seabirds, including murres, cormorants, mergansers, pigeon guillemots, and black legged kittiwakes (*21*), and marine mammals, such as harbor seals (*14*), have exhibited persistent reductions in abundance in oiled areas since the EVOS.

The 1990s paradigm of oil ecotoxicity recognizes risk of delayed recovery of apex consumers (seabirds and marine mammals) due to indirect, bottom-up trophic interactions of oil inducing prey limitation. It also recognizes that interspecific interactions will lead to a sequence of delayed indirect effects on rocky intertidal communities.

Public Policy Implications

In light of the recent research on chronic oil pollution, the current regulatory framework is grossly inadequate to protect marine life from chronic, non-point source discharges, especially along urbanized coastlines. The current regulatory framework is based on outdated risk assessment models (acute toxicity models based on narcosis) that fail to recognize (a) chronic direct population-level effects from persistent PAHs; (b) sublethal, indirect, and trophic-level effects of weathered oil; and (c) the importance of habitat quality in maintaining population structure (*1, 3*).

Streams and estuaries serve as critical habitat, a nursery, for vulnerable early developmental life stages of many species of fish and other sealife: these habitats also receive bulk chronic hydrocarbon discharges. Scientists estimate that the amount of highway runoff in the US to be about one quart of oil per person per year. This means that for every 50 million people the equivalent of an EVOS (or 11 million gallons as reported by Exxon) is dumped every year, year after year, into productive coastline habitats as urban run-off (*1*). Clearly, if sustainable coastal fish populations and other wildlife are to co-exist with industrialized societies, our focus needs to shift to the prevention, control, and restoration of these habitats from

contamination–whether it is from acute spills or chronic non-point source pollution.

One place to start is with our federal water quality standards for PAHs, which are currently 300 ppb. Scientists now recognize a toxicity threshold of 1 ppb aqueous PAHs for habitats where fish eggs and larvae rear (*35*). Revisions to federal storm-water discharge regulations should be based on the 1990s oil toxicity paradigm (*1*), where chronic toxicity mechanisms are the concern not short-term narcosis.

Resource managers and oil spill response managers currently use outdated ecotoxicity models from the 1970s to assess only the short-term acute toxicity risks and damage from oil pollution and, in so doing, severely understate environmental impacts of chronic oil pollution (*1*, *3*). The regulatory framework governing oil discharge from offshore drilling platforms, oil tankers, and oil facilities regulated by federal discharge permits needs to be re-examined in light of the 1990s oil toxicity paradigm. Policies governing natural resource damage assessment following oil spills also fail to reflect this new appreciation of impacts of long-term toxicity. For example, the Oil Pollution Act of 1990 has effectively eliminated long-term biological damage assessment and long-term monitoring in oil spills after the *Exxon Valdez*.

A precautionary approach to oil and gas development and use seems advisable in the face of mounting evidence that oil is far more persistent and deadly in protected nearshore habitats than previously recognized. Unless restrictive regulations of anthropogenic PAH sources are adopted to minimize the ubiquitous chronic oil pollution, public resources–land, water, fish, and sealife–will subsidize at great cost the environmental burden of our oil dependency.

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