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Letter to the Editor: COMMENT ON "HYDROCARBON COMPOSITION AND TOXICITY OF SEDIMENTS FOLLOWING THE *EXXON VALDEZ* OIL SPILL IN PRINCE WILLIAM SOUND, ALASKA, USA"

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Letter to the Editor

COMMENT ON ''HYDROCARBON COMPOSITION AND TOXICITY OF SEDIMENTS FOLLOWING THE *EXXON VALDEZ* OIL SPILL IN PRINCE WILLIAM SOUND, ALASKA, USA''

To the Editor:

In their recent paper, Page et al. [1] concluded that spilled oil lost toxicity due to weathering and that it dispersed from beaches so rapidly that the potential for toxicity is now, 13 years after the spill, negligible. Their conclusions, however, are based on insensitive toxicity tests, a statistical design with low power, and invalid assumptions regarding the extent and location of remaining oil.

Although Page et al. [1] make a convincing case that the narcotic toxicity of *Exxon Valdez* oil declined rapidly after the spill, the claim that *all* oil toxicity has similarly declined requires a higher standard of evidence. They estimate sediment toxicity from mortality in 10-d amphipod bioassays; however, measures of lethal responses in adult animals are usually less sensitive than sublethal measures, particularly in early life stages [2–5]. To fully assess the toxic potential of contaminated sediment, evaluation of multiple taxa is frequently recommended [e.g., 6–8]. The absence of acute sediment toxicity does not demonstrate the absence of benthic degradation [9].

Only large alterations of community structure are detectable with the study design used by Page et al. [1]. An observed 20% decrease in species richness at total polycyclic aromatic hydrocarbon (PAH) concentrations in sediment equivalent to about 0.2% oil (see Table 9 and Fig. 6 [1]) is not detected as significant at $\alpha = 0.05$. A statistical power analysis indicates this sampling design has less than 50% probability of detecting as significant reductions in species richness of 50% (using Student's *t* test with unequal variances and the data in Fig. 6). This insensitivity is the consequence of low sampling effort and resulting low power. The worst-case intertidal sampling program of Page et al. [1] represents three tide stages, two habitats, and two oiling classifications sampled during each of four years, with a cumulative area sampled less than 3 m^2. This sample area was selected from a worst-case impact area spanning many hectares. The effect of oil on community structure is thoroughly obscured by inadequate estimates of the variance contributions from each statistical stratum (most strata were ignored in the statistical analyses). Sublethal responses leading to reduced reproductive output or survival, for example, are almost certain to remain undetected by this sampling design.

The assumption that total PAH concentrations in associated sediment are reliable measures of community exposure is not necessarily valid for the intertidal, where interstitial water flows (driven by tidal pumping or precipitation) can transport PAH dissolved from subsurface oil deposits to distant infauna along hydraulic gradients [10]. Such an exposure mechanism is especially relevant in intertidal Prince William Sound, where the average tidal range is about 3 m, annual precipitation is measured in meters, and most remaining oil is subsurface [11]. Page et al. [1] collected their samples from the uppermost 10 cm of upper intertidal sediments; therefore, correlation of total PAH with actual exposure of infaunal communities to PAH dissolved from the spilled oil may be substantially weaker than assumed. Infaunal communities with low total PAH concentrations in sediment may actually be exposed to intermittent but harmful PAH dissolved in interstitial water flows. The resulting exposure misclassification further reduces statistical power.

Finally, recent evidence does not support their assertion that the remaining oil is so rare that there is ''. . . a very small likelihood of sampling such deposits using an unbiased random sampling design'' [1]. Such a design revealed approximately 11 ha of contaminated shoreline with about 65,000 L of remaining oil buried in the middle and lower intertidal of Page et al.'s study area [11,12]. Along the 74 km of beaches in Prince William Sound that remained heavily or moderately oiled after 1989, probability-based sampling determined that approximately 7% of the beach area between +1.8 to 4.8 m tide height contained subsurface oil in 2002 [12]. This cryptic oil is usually considerably less weathered than surface oil deposits; hence, it contains more biologically available PAH than surface oil deposits. These PAH can dissolve into tidally driven interstitial water flows. Because Page et al. [1] focused most of their sampling effort on highly weathered surface oil in the upper intertidal, which no longer contained PAH likely to cause narcosis, the potential for toxic effects of the remaining oil is underestimated.

The issues we raise are not merely academic. Several studies have implicated remaining *Exxon Valdez* oil as the proximate source of PAH currently inhibiting recovery of sea duck and sea otter populations that forage in the intertidal of the most heavily oiled portion of the spill zone in Prince William Sound [13–16]. Biochemical indicators of PAH exposure in these species are closely associated with the geographical distribution of the remaining oil. Exposure is likely caused by the ability of the oil to disturb middle- and lower-intertidal sediment where the bulk of less-weathered subsurface oil remains. Together with recent estimates of the amount of remaining oil [11], evidence of exposure and impaired population recovery is compelling evidence that *Exxon Valdez* oil continues to exert deleterious effects on wildlife in parts of this region. That persistent oil causes long-term damage should be no surprise; persistent physiological and community disruption lasting at least a decade has been reported for other spills [e.g., 17–20].

The study by Page et al. [1] uses an approach to the environmental assessment of the *Exxon Valdez* oil spill noted by Peterson et al. [21], wherein study design choices that consistently lower statistical power are used, predictably leading to the conclusion of absence of effects. In the present case, Page et al. [1] consider only the least toxic mode of PAH

action (narcosis), compare this with community structure impacts assessed using sampling methods that are only likely to detect declines in species richness exceeding 50%, and depend on a questionable measure of exposure (total PAH in surface sediments).

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The authors' reply:

In their comments on our recent publication of the toxicology results of a triad-based study of the effects of the *Exxon Valdez* oil spill [2], Rice et al. [1] assert that narcotic toxicity of oil-contaminated sediments is not a sensitive predictor of overall sediment toxicity, and they question the validity of the amphipod bioassay used. This assertion is not consistent with reported sediment toxicity studies. The mode of action for acute and chronic toxicity to aquatic organisms of polycyclic aromatic hydrocarbons (PAH) and many other nonpolar organic chemicals can be attributed to narcosis [3], which may manifest itself as lethality or as a wide variety of sublethal or chronic responses, depending on the concentration of toxicant at sites of action. The sediment toxicity test with the amphipod, *Rhepoxinius abronius*, is widely accepted, including by the National Oceanic and Atmospheric Administration and the U.S. Environmental Protection Agency [4,5], as a representative and sensitive toxicity test. For this reason it has been used in the development of models of bioavailability and toxicity of nonpolar organic chemicals, including (PAH), in sediments (see references in Page et al. [2]). *Rhepoxynius abronius* has been shown to be among the most sensitive of sediment toxicity test organisms to toxicants including PAH [4,5], with a wide range in response, relatively high discriminatory power, and intermediate precision [5].

Long et al. [6] made extensive use of sediment toxicity tests with amphipods, particularly *Rhepoxynius*, in developing sediment quality guideline values for marine and estuarine sediments. The sediment toxicity threshold concentration for