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Reply to Jenkins et al.: Evidence for contaminating oil exposure is closely linked in space and time to biological effects

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LETTER

Reply to Jenkins et al.: Evidence for contaminating oil exposure is closely linked in space and time to biological effects

Our original article (1) linked exposure of resident killifish to contaminating oil from the *Deepwater Horizon* (DWH) oil spill with significant biological responses, including genome expression, protein expression, and tissue morphology. Given decades of laboratory studies on the effects of crude oil in many species, including fish, and after extensive field studies following the *Exxon Valdez* spill, some of the responses we captured are recognized as diagnostic of exposure to, and effects from, the toxic components of weathered crude oil (e.g., ref. 2). Jenkins et al. (3) raise two main objections to our study. They assert that the analytical chemistry was not consistent with detected biological responses, and that chemicals other than DWH-derived crude oil polyaromatic hydrocarbons (PAHs) could have caused the observed biological responses. PAHs are quickly metabolized by vertebrate animals (4); therefore, it is not surprising that chemical analyses of tissues showed little differences among sites and time points. The hydrophobicity of PAHs also explains why concentrations in water were low, tending to accumulate in sediments. It is in the heavily contaminated sediments of Grande Terre, LA (GT), relative to other sites, that the most notable differences were observed during the study, consistent with GT being the only of our six field sites that was contaminated by DWH oil. Ultimately, sediments were the main reservoir of contaminating oil following the *Exxon Valdez* spill (2). We drew attention to this point in our original publication (1). It is important to emphasize that we integrated multiple types of data to rigorously document the location and timing of spilled oil contamination, including analytical chemistry of water, tissues, and sediments, along with satellite imagery and photographic evidence. These data clearly indicate that, of our six field sites, only the GT site was contaminated with spilled oil, and the oil arrived after our first sampling time point, but before our second sampling time point (Fig. 1 in ref. 1). Our observed biological responses coincided with the timing and the location of oil contamination. Chemicals other than PAHs, such as dioxins and polychlorinated biphenyls, may induce similar biological responses, but to our knowledge, there exists no evidence of a polychlorinated biphenyl or dioxin spill event that occurred at the GT field site only, within 2 mo after our preevent baseline data measurement, and coincident with the arrival of contaminating oil.

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The authors declare no conflict of interest.

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