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Semipermeable membrane devices link site-specific contaminants to effects: PART II – A comparison of lingering Exxon Valdez oil with other potential sources of CYP1A inducers in Prince William Sound, Alaska

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ABSTRACT

We deployed semipermeable membrane devices (SPMDs) on beaches for 28 days at 53 sites in Prince William Sound (PWS), Alaska, to evaluate the induction potential from suspected sources of cytochrome P450 1A (CYP1A)-inducing contaminants. Sites were selected to assess known point sources, or were chosen randomly to evaluate the region-wide sources. After deployment, SPMD extracts were analyzed chemically for persistent organic pollutants (POPs) and polycyclic aromatic hydrocarbons (PAH). These results were compared with hepatic CYP1A enzyme activity of juvenile rainbow trout injected with the same extracts prior to clean-up for the chemical analyses. Increased CYP1A activity was strongly associated with PAH concentrations in extracts, especially chrysene homologues but was not associated with POPs. The only apparent sources of chrysene homologues were lingering oil from Exxon Valdez, asphalt and bunker fuels released from storage tanks during the 1964 Alaska earthquake, creosote leaching from numerous pilings at one site, and PAH-contaminated sediments at Cordova Harbor. Our results indicate that PWS is remarkably free of pollution from PAH when nearby sources are absent as well as from pesticides and PCBs generally.

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1. Introduction

Oil from the T/V Exxon Valdez (EVO) has been surprisingly persistent on some beaches in western Prince William Sound (PWS), after deposition following the spill in 1989. Estimated loss rates of <4% yr^{-1} for the buried oil imply persistence for decades (Short et al., 2007a), presenting a potentially long-term reservoir of bioaffective contaminants to wildlife. However, distinguishing any long-term biological effects of this oil from those of other contaminant sources is problematic.

Several species that interact with the intertidal zone in the spill-impacted part of PWS have shown prolonged expression of cytochrome P450 1A (CYP1A), a substrate-inducible enzyme that is a sensitive biomarker of exposure to planar contaminants such as polycyclic aromatic hydrocarbons (PAH), certain polychlorinated biphenyls (PCBs) and other persistant organic pollutants (POPs). Expression of CYP1A in PWS nearshore vertebrates such as sea otters (*Enhydra lutris*) harlequin ducks (*Histrionicus histrionicus*) and crescent gunnels (*Pholis laeta*) has been highest in animals taken from locations where EVO has been most persistent, and has appeared to decrease as the oil dispersed (Bodkin et al., 2002; Jewett et al., 2002; Trust et al., 2000). These patterns suggest that exposure to remaining EVO, a known CYP1A inducer (Woodin et al., 1997), may be the source of the CYP1A induction observed in these animals.

Whether the EVO remaining on PWS beaches is sufficiently bioavailable to induce CYP1A in wildlife is a matter of some controversy. Once hardened into highly viscous tar mats and asphalt pavements, the resistance to mass transfer of PAH is so great that loss rates of PAH may become extremely low. However, sea otters and ducks can disturb sediments while foraging (Kvitek and Oliver, 1992; Calkins, 1978), and any subsurface oil encountered could contaminate their pelage or plumage. Oil ingested during subsequent preening (Bodkin et al., 2002; Trust et al., 2000) provides an exposure pathway for eliciting CYP1A induction and potentially toxic effects (Schwartz et al., 2004).

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Evidence of CYP1A induction alone is not sufficient to identify the source of the inducing agent when numerous potential sources may be present (Short and Springman, 2006). In PWS, several sources of potential CYP1A inducers besides EVO must be considered. Putative sources include atmospheric transport of POPs (Stern et al., 1997), PAH associated with erosion of terrestrial hydrocarbon source rock outcrops transported hydraulically to the northern Gulf of Alaska and then by the Alaska Coastal Current to PWS where they are deposited on the seafloor (Page et al., 1996; Short et al., 2007b), POPs associated with adult salmon returning to PWS to spawn (Krümmel et al., 2003; Ewald et al., 1998), small spills of diesel and bilge oils from marine vessels, residues of asphalt and bunker fuels derived from the Monterey Formation in California and released from storage tanks in PWS that ruptured during the 1964 Alaska earthquake (Kvenvolden et al., 1995), and operations associated with continuing industrial activity in PWS such as salmon hatcheries. Resolving contributions from these prospective sources to observed CYP1A responses in biota presents a considerable challenge.

One promising approach for evaluating the CYP1A induction potential of a contaminant source involves injecting fish with contaminants accumulated by semipermeable membrane devices (SPMDs) deployed at locations strongly affected by the candidate source (Springman et al., 2008). This approach does not require knowledge of the chemical identity of the suspected contaminant, although analysis may provide this. Rather, it examines the biomarker response from the bioavailable complex mixture without selective filtration if the accumulated contaminants are injected without cleanup. At a minimum, prospective contaminant sources that are not capable of eliciting CYP1A responses in biota can be identified at high confidence when the injected contaminants fail to elicit a response provided the amount of contaminant injected is comparable with those likely to be accumulated through passive equilibrium partitioning by the same organisms exposed in the field.

Our objectives in this study are to distinguish which contaminant sources in PWS are capable of eliciting CYP1A activity in dosed fish and to identify which compounds of the sampled complex mixture are most responsible. To accomplish this, we deployed SPMDs at a variety of locations that included all the prospective contaminant sources mentioned above. In Part I of this study, we measured the CYP1A induction potential of contaminants extracted from deployed SPMDs and injected without clean-up into juvenile rainbow trout (*Oncorhynchus mykiss*, Springman et al., 2008). Here we present chemical analysis results of extracts from SPMDs deployed to evaluate the compounds most likely responsible for induction, and of additional SPMDs deployed to better characterize contaminant sources and distributions in PWS.

2. Methods

2.1. Study sites

We deployed SPMDs at 53 sites within PWS during 2004, including known and suspected contaminant source sites and sites selected at random to characterize the pollution burden of the Sound (Fig. 1). Known contaminant source sites include boat

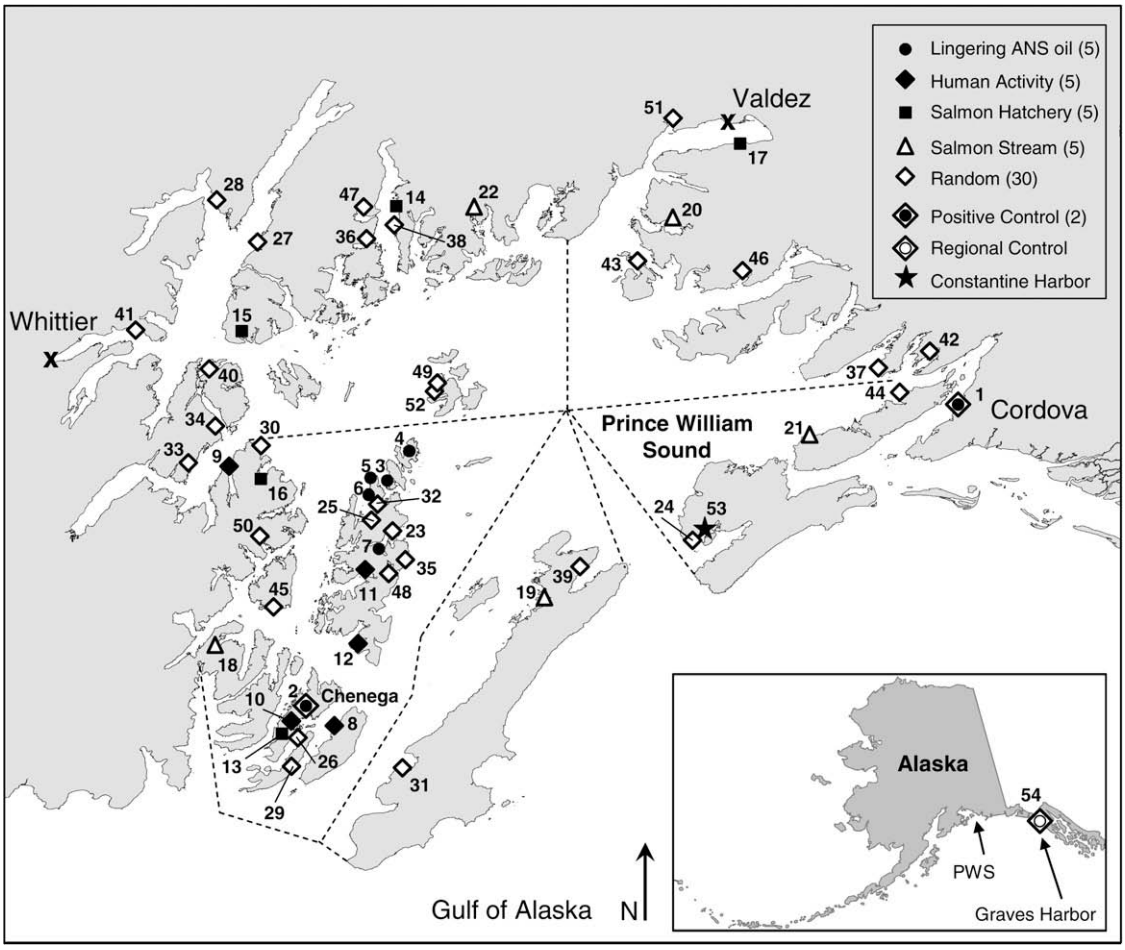

Fig. 1. Map of study region. Site numbers are also listed in Table 2. Dashed lines indicate Alaska Department of Fish and Game districts.