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Improved flatfish health following remediation of a PAH-contaminated site in Eagle Harbor, Washington

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ABSTRACT

Eagle Harbor in Puget Sound, WA became a Superfund site in 1987 due to polycyclic aromatic hydrocarbons (PAHs) released chronically from a nearby creosoting facility. Early studies here (1983–1986) demonstrated up to an ~80% prevalence of toxicopathic liver lesions, including neoplasms, in resident English sole (*Parophrys vetulus*). These lesions in English sole are consistently associated with PAH exposure in multiple field studies, and one laboratory study. Later studies (1986–1988) incorporated biomarkers of PAH exposure and effect, including hepatic CYP1A expression and xenobiotic–DNA adducts, and biliary fluorescent aromatic compounds (FACs). Before site remediation, lesion prevalences and other biomarker values in this species from Eagle Harbor were among the highest compared to other sites in Puget Sound and the US Pacific Coast. To sequester PAH-contaminated sediments, in 1993–1994, a primary cap of clean sediment was placed over the most-contaminated 54 acres, with a 15-acre secondary cap added from 2000–2002. Lesion prevalences and biomarker values before primary capping were reduced compared to 1983–1986, consistent with facility closure in 1988 and shore-based source controls begun in 1990. Liver lesion risk, hepatic CYP1A activities, and levels of biliary FACs from fish collected immediately after and at regular intervals up to 2 years after primary capping were variable relative to pre-capping. Over the entire monitoring period since primary capping (128 months), but particularly after 3 years, there was a significantly decreasing trend in biliary FACs, hepatic DNA adducts and lesion risk in English sole. In particular, lesion risk has been consistently low (<0.20) compared to primary cap initiation (set at 1.0), from ~4 years after primary capping through April 2004. These results show that the sediment capping process has been effective in reducing PAH exposure and associated deleterious biological effects in a resident flatfish, and that longer term monitoring of pollutant responses in biological resources, such as resident fish, is needed in order to demonstrate the efficacy of this type of remediation.

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1. Introduction

Extensive evidence indicates that exposure to polycyclic aromatic hydrocarbons (PAHs) and their derivatives produces a variety of adverse biological effects in marine fish. While much of this information is derived from laboratory studies, there is an increasing body of evidence from field studies supporting these relationships between PAH exposure and biological injury. Several of the most definitive field studies in this regard have been done in Puget Sound, Washington (Myers et al., 1987, 1998, 2003; Rhodes et al., 1987; Johnson et al., 1988; Casillas et al., 1991; Landahl and Johnson, 1993) where one of the most heavily PAH-contaminated sites has been in Eagle Harbor, on the east side of Bainbridge Island in Puget Sound, WA (Fig. 1). Studies done by Northwest Fisheries Science Center (NWFSC)/National Oceanic and Atmospheric Administration (NOAA) scientists since 1983 in Eagle Harbor showed that resident epibenthic flatfish such as English sole (*Parophrys vetulus*) there were at increased risk of developing serious toxicopathic liver diseases, including cancer, and of exhibiting impaired reproductive function and altered physiological responses (Malins et al., 1985; Myers et al., 1987; Rhodes et al., 1987; Johnson et al., 1988; Casillas et al., 1991; Stein et al., 1992). In fact, toxicopathic hepatic lesion prevalences (Malins et al., 1985; Myers et al., 1987) and other values for biomarkers of PAH exposure in Eagle Harbor (Stein et al., 1992; Myers et al., 1998) were among the highest recorded in Puget Sound, with up to ~80% of English sole affected with these liver lesions.

Due largely to these findings, as well as the high levels of PAHs in the sediments (up to 120,000 ppb $\sum$PAHs), English sole stomach contents (up to 84,000 ppb $\sum$PAHs) (Malins et al., 1985)

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Fig. 1. Map showing location of Eagle Harbor, WA in Puget Sound.

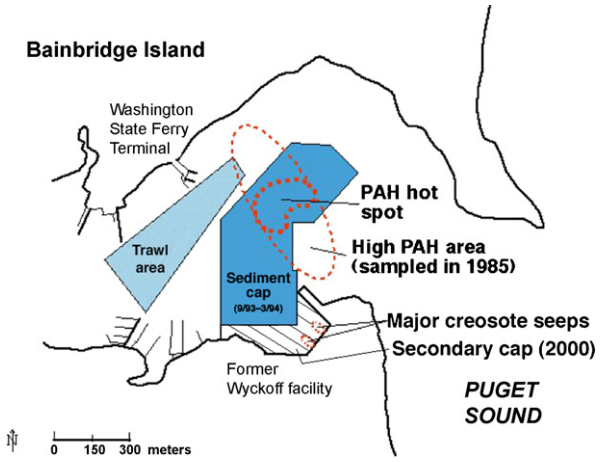

Fig. 2. Map of area of Eagle Harbor in which fish sampling (bottom trawling) operations were conducted, in relation to the past area of high sediment PAHs. Also shown are the areas sequentially capped: (1) in September 1993 to March 1994 with sediments composed of dredge material from the Snohomish River (polygonal shaded area including the PAH "hot spot"), covering 54 acres, and (2) in November 2000–February 2002 as a secondary cap of clean sand, joining the southern edge of the original cap with the northern shoreline of the former Wyckoff facility, adding another 15 acres of area capped with clean sediments.

and edible marine biota, Eagle Harbor was designated a Superfund site in 1987. These high levels of PAH exposure were a result of long-term and continuing releases of creosote from the onshore facility which had pressure-treated wood pilings with coal tar creosote almost continuously since 1903, referred to as the Wyckoff site/facility after the name of the most recent company operating the facility. The United States Environmental Protection Agency (USEPA) completed a Remedial Investigation/Feasibility Study in 1989, and in December 1991 proposed a phased cleanup of the harbor which included capping of PAH-contaminated sediments (http://yosemite.epa.gov/R10/CLEANUP.NSF/sites/Wyckoff). In June 1993, under Superfund Removal authority, the USEPA recommended placement of relatively uncontaminated (<570 ppb $\sum$PAHs) Snohomish River dredge material over the most contaminated area of Eagle Harbor by a combined process of release of dredge material from split-hull barges in the deeper areas of Eagle Harbor and wash-off of similar material from regular barges in the shallower regions of the area to be capped. In late September 1993, the USEPA and US Army Corps of Engineers began placement of this sediment cap as a means of (1) controlling transport of contaminants; (2) isolating contaminants from marine biota; and (3) providing clean habitat for benthic organisms, thus reducing overall risk of exposure to PAHs contained in the sediments. This primary capping process was completed in March 1994, resulting in placement of an approximately three-foot thick cap of 275,000 cubic yards of clean sandy material and associated wood debris over an original area of approximately 54 acres of the most highly contaminated subtidal sediments within Eagle Harbor (Fig. 2), including a PAH "hot spot" documented in 1983 (Malins et al., 1985). Other remediation actions have included: (1) construction of an onshore groundwater extraction and treatment system in 1990 and subsequent intermittent operation of this system up through the present; (2) installation in November 2000–March 2001 of a 2000 feet long sheet pile wall driven 10–100 feet deep along the entire shoreline of the former Wyckoff site to curtail continuing subtidal and intertidal seeps of free creosote into Eagle Harbor sediments. These seeps had resulted in multiple areas within the capped area displaying locally elevated surface high molecular weight PAH concentrations exceeding both the Washington State Sediment Quality Standards and Minimum Cleanup Levels, up to ~72,000 ppb (USEPA/USACOE, 1999); and (3) from November 2000–February 2002, extension of the original sediment cap from its southern edge to the north-facing shoreline of the former Wyckoff creosoting facility to further sequester the continuing subtidal and intertidal seeps of free creosote. This placement of ~170,000 cubic yards of clean sand created another 15 acres of clean sediment cap over PAH-contaminated regions of Eagle Harbor (Fig. 2).

The primary objective of this study was to assess the efficacy of these remediation measures in Eagle Harbor in terms of reducing PAH exposure and improving the health of resident fish. Several biochemical measures of PAH exposure and early toxicological response were monitored in the English sole, a resident flatfish species that has been routinely used as a sentinel species in Puget Sound. In addition, we performed histopathological examinations of the livers of the same English sole assayed for PAH exposure and early response, and determined the occurrence of a spectrum of toxicopathic hepatic lesions involved in the stepwise process of hepatocarcinogenesis. These lesions have been strongly and consistently associated with PAH exposure in wild English sole (Myers et al., 1987, 2003). Moreover, certain neoplasia-related liver lesions have been induced in sole by injections of a PAH-rich fraction extracted from Eagle Harbor sediment (Schiewe et al., 1991). These four measures, or biomarkers, of PAH exposure and effect, are described below.

Because of the high rate of metabolism and depuration of PAHs by many species of fish, direct measurement of parent compounds in tissues is not generally a useful indicator of exposure of fish to PAHs (Varanasi et al., 1989a). Accordingly, methods have been developed to determine such exposure, based on our increased understanding of the mechanisms and pathways of PAH metabolism in fish.

The first biomarker used in this study is the measurement of fluorescent aromatic compounds (FACs) in bile by high performance liquid chromatography (HPLC) with fluorescence detection. Many polar and fluorescent metabolites of PAHs are primarily excreted via the hepatobiliary system in fish. This method provides a semi-quantitative means for rapidly assessing recent PAH exposure in fish and has the added advantage of being able to be 'fine-tuned'