Estimating risk at a Superfund site using passive sampling devices as biological surrogates in human health risk models

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ARTICLE INFO

ABSTRACT

Passive sampling devices (PSDs) sequester the freely dissolved fraction of lipophilic contaminants, mimicking passive chemical uptake and accumulation by biomembranes and lipid tissues. Public Health Assessments that inform the public about health risks from exposure to contaminants through consumption of resident fish are generally based on tissue data, which can be difficult to obtain and requires destructive sampling. The purpose of this study is to apply PSD data in a Public Health Assessment to demonstrate that PSDs can be used as a biological surrogate to evaluate potential human health risks and elucidate spatio-temporal variations in risk. PSDs were used to measure polycyclic aromatic hydrocarbons (PAHs) in the Willamette River; upriver, downriver and within the Portland Harbor Superfund megasite for 3 years during wet and dry seasons. Based on an existing Public Health Assessment for this area, concentrations of PAHs in PSDs were substituted for fish tissue concentrations. PSD measured PAH concentrations captured the magnitude, range and variability of PAH concentrations reported for fish/shellfish from Portland Harbor. Using PSD results in place of fish data revealed an unacceptable risk level for cancer in all seasons but no unacceptable risk for non-cancer endpoints. Estimated cancer risk varied by several orders of magnitude based on season and location. Sites near coal tar contamination demonstrated the highest risk, particularly during the dry season and remediation activities. Incorporating PSD data into Public Health Assessments provides specific spatial and temporal contaminant exposure information that can assist public health professionals in evaluating human health risks.

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1. Introduction

Urban rivers that are used by local residents for recreational purposes such as boating, and sport or subsistence fishing are often heavily polluted. Public Health Assessments inform the public about the relative risks of these activities in a specific area by providing information about potential exposures and the likelihood that those exposures could lead to adverse health effects. A Public Health Assessment develops an estimated human exposure dose based on environmental and contaminant data for a specific site and existing regulatory standards (ATSDR, 2005) (for more information about Public Health Assessments please see Section 1 in Supplementary information). Currently, exposure due to consumption of resident organisms is based on tissue contaminant data

from fish or shellfish harvested in the area. However, obtaining organisms for analysis can be difficult, usually requires destruction of the organism and often provides limited specific spatial or temporal information (Huckins et al., 2006). Studies have highlighted spatial and temporal variations in contamination and exposure (Ko and Baker, 2004; Brown and Peake, 2006) and others have called for their consideration in risk assessments (Linkov et al., 2002). Recently, developing methodology for more accurately assessing exposure has become a priority for risk assessment (Birnbaum, 2010). Passive sampling devices (PSDs) can be strategically deployed to address spatial and temporal issues in bioavailable contaminant concentrations, an issue that has been shown to significantly affect risk (Huckins et al., 2006).

PSDs, such as semipermeable membrane devices (SPMDs), simulate biological membranes and lipid tissue and thus sequester only the freely-dissolved or bioaccessible fraction of lipophilic organic contaminants. Huckins et al. (2006) reviewed over 30 studies with side-by-side comparisons of SPMDs with organisms and found good correlations with finfish and bivalves, though few studies have investigated PAHs specifically (Peven et al., 1996; Baussant et al., 2001; Verweij et al., 2004; Boehm et al., 2005; Ke et al., 2007). Correlations between PAHs in SPMDs and organisms

have been found in terrestrial and aquatic systems, although investigators observed differences in the composition of the PAHs sequestered by organisms and PSDs (Baussant et al., 2001; Ke et al., 2007; Tao et al., 2008). Baussant et al. (2001) found that lower molecular weight PAHs predominated in caged finfish while Ke et al. (2007) measured higher concentrations of PAHs in SPMDs compared to tissue from caged carp. While these studies demonstrate that PSD concentrations can be correlated to organism tissue concentrations, they do not link the PSD concentrations to human health risks.

Recent lab and field trials have resulted in simpler and cheaper variants of SPMDs (Adams et al., 2007; Sower and Anderson, 2008; Allan et al., 2009). These PSDs are constructed from low density polyethylene lay-flat tubing without triolein and designated lipid-free tubing samplers, or LFTs. PSDs, such as the LFT used in this study, offer numerous advantages over using organisms for environmental assessment including simplicity, low cost, fast and minimal extraction and clean-up procedure, no metabolic activity and no organisms are destroyed. Though numerous physical, physiological and ambient factors affect concentrations in organisms, all accumulate contaminants like PSDs: from water across biological membranes (Huckins et al., 2006). Also, unlike organisms, PSDs spiked with performance reference compounds provide chemical specific calibrations of time-integrated, bioavailable concentrations that can be standardized across studies (Huckins et al., 2006; Adams et al., 2007). Using PSDs to determine the time integrated water concentration of contaminants is well established, however, this is the first demonstration of the direct application of PSD data for assessing potential human health risks from consumption.

PSDs are particularly useful in areas where point sources are significant contributors to contamination and where seasonal fluctuations in contaminant concentrations are suspected. To this end, the Portland Harbor Superfund megasite on the Willamette River in Portland, Oregon (river miles or RM 3.5–9.2) is an ideal model system for examining the application of PSD data to Public Health Assessments to elucidate potential exposures and risks in an urban river. Portland Harbor is an industrialized area containing several PAH point sources including coal tar and a remediated former creosoting plant, which is its own Superfund site within the larger harbor site. Additional sources of PAHs in the lower Willamette include ship, train and vehicle emissions, combined sewer overflows, urban runoff, atmospheric deposition and petroleum product leaks and spills. Additionally, significant seasonal flow and precipitation fluctuations occur on the river and seasonal variations in contamination concentrations have been observed (Sower and Anderson, 2008).

The Willamette River is used extensively for both sport and subsistence fishing. Eating contaminated fish from the harbor is considered the most significant health risk from chemical contamination at the site (ATSDR, 2006). Although fish advisories have been issued for some areas, based on exposure to other industrial contaminants, the most recent Public Health Assessment could not evaluate risk from exposure to PAHs due to insufficient fish data. Of 39 species of resident fish in this area, eight constitute the most likely to be caught and consumed by local sport and subsistence fishers, including walleye, black crappie, white crappie, smallmouth bass, pikeminnow, yellow bullhead, carp and largescale sucker. Clams and crayfish are also commonly harvested for consumption. Details about resident fish as well as fish consumption data for different population groups is available in the Portland Harbor Public Health Assessment (ATSDR, 2006).

The purpose of this study is to apply PSD data in a Public Health Assessment to demonstrate that PSDs can be used as a biological surrogate to elucidate spatial and temporal variations in potential human health risks. To achieve this, the PSD mass concentrations

of PAHs were substituted for fish tissue contaminant concentrations. The spatial and temporal distribution of PSD measured PAH concentrations were applied to cancer and non-cancer human health risk assessment models.

2. Methods

2.1. Study area

The study area was the lower 18.5 miles of the Willamette River, up to its confluence with the Columbia River. Samplers were placed at 13 sites on west (W) and east (E) sides of the river channel from 2004 to 2006 (Fig. 1). The sites were located upriver (RMs 18.5E, 17E, 15.5E, 13W, and 12E), downriver (RM 1E) and within the Portland Harbor Superfund megasite (RMs 3.5E, 3.5W, 5W, 6.5W, 7W, 7E and 8E). Residential and commercial uses dominate the upriver area whereas the Superfund megasite area is heavily industrialized and contains PAH point sources including creosote and coal tar contaminated sites at RMs 7E and 6.3W respectively. In addition, urban runoff and combined sewer overflows affect the area. Undeveloped or agricultural areas predominate downriver from the harbor.

The study period overlapped with remediation activities that were carried out at RM 6.3 from August to October, 2005. During this time submerged tar from a manufactured gas plant (MGP) site was removed by dredging and a cap was placed over the contaminated sediment. The temporal effects of this remediation activity are analyzed separately from the seasonal data and serve to highlight the importance of having specific spatial and temporal data for effective risk assessment in areas affected by sporadic peaks in contaminant inputs.

2.2. Chemicals and solvents

PAH standards (purities ⩾99%) were obtained from ChemService, Inc. (West Chester, PA, USA) and Pesticide or Optima® grade cleanup and extraction solvents from Fisher Scientific (Fairlawn, NJ, USA) were used. The 16 target analytes, which correspond to the USEPA 16 priority PAHs, included naphthalene, acenaphthene, acenaphthylene, fluorene, anthracene, phenanthrene, fluoranthene, pyrene, chrysene, benz(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, dibenz(a,h)anthracene benzo(ghi)perylene, and indeno(1,2,3-c,d)pyrene.

2.3. Sample collection, extraction and analysis

LFT passive samplers were constructed and fortified with performance reference compounds (PRCs) using methods described in Sower and Anderson (2008) Briefly, additive-free, 2.7 cm wide, low-density polyethylene membrane (Barefoot) from Brentwood Plastic, Inc. (St. Louis, MO, USA) was cleaned with hexanes, cut into 100 cm strips, fortified with dibenz(ah)anthracene as a PRC and heat sealed at both ends.

From 2004 to 2006 samplers were deployed in multiple 21-d events during July or August ("dry season") and October or November ("wet season"). This period represents the transition from the lowest precipitation and river flows of the year to relatively high precipitation and flows. In 2006 two sampling events were added from May through June, the transition from high to low flow. Stainless steel cages were loaded with five LFTs and suspended 3 m above the river bottom at each site with an anchor–cage–float system described elsewhere (Sethajintanin and Anderson, 2006).

A YSI® sonde was used during sampler deployment and retrieval to collect water chemistry data including temperature, pH, specific conductivity, and oxidative-reductive potential (ORP). LFT
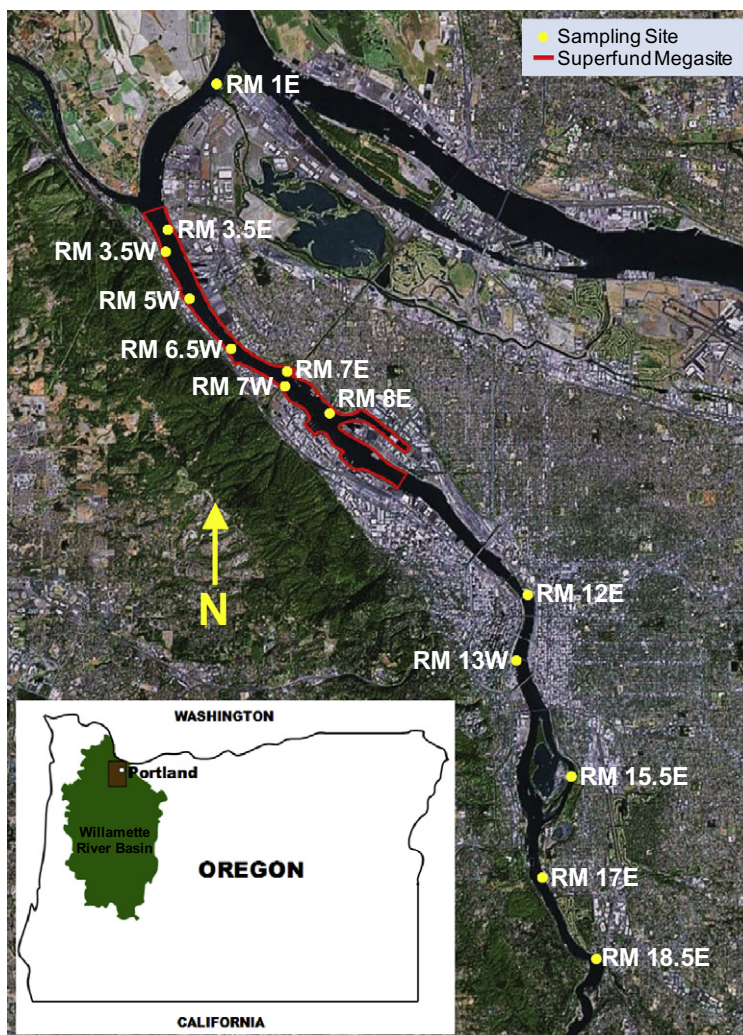
Fig. 1. Sampling sites on the lower Willamette River 2004–2006. Each site is designated by a yellow circle. Not all sites were used every deployment. The red line indicates the approximate boundaries of the Portland Harbor Superfund megasite.

field cleanup and laboratory extraction were carried out as described in Sower and Anderson (2008). Field quality control consisted of duplicate samplers at RMs 7W and 8E, field blanks, trip blanks and field cleanup blanks. Laboratory quality control included reagent blanks, high and low concentration fortifications, and unexposed fortified LFTs.

After extraction, samples were analyzed by HPLC with diode-array (DAD) and fluorescence (FLD) detectors. DAD signals were 230 and 254 nm and FLD excitation and emissions were 230 and 332, 405, 460, respectively. Flow was 2.0 mL min^{-1} beginning with 40/60% acetonitrile and water and steadily ramping to 100% acetonitrile over a 28 min run per column maker recommendations.

2.4. Exposure, cancer, non-cancer and ecological risk modeling

Water concentrations were calculated using equations provided in Huckins et al. (2006). PSD concentrations for risk models are based on the mass of contaminant collected vs. the mass of the sampler. This mass:mass concentration treats the PSD as a direct biological surrogate and represents the amount of contaminant an organism would take up through passive partitioning. PAHs do not biomagnify in fin fish and chemical uptake from water and/or pore water has been described as the most likely dominant route of uptake for fish and shellfish (Connell, 1990;

Huckins et al., 2006). LFT concentrations best reflect exposure of organisms residing in the water column; benthic fauna and infauna may be exposed to different sediment and/or pore water PAH concentrations.

Exposures and human health endpoints were calculated by substituting the PSD mass concentrations for the fish tissue contaminant concentrations in models previously used for the Portland Harbor Public Health Assessment (ATSDR, 2006).

The PSD mass concentrations of PAHs that are recognized as carcinogenic by the USEPA (benzo(a)anthracene, chrysene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, indeno(1,2,3,-c,d)pyrene, and dibenzo(a,h)anthracene) were used for cancer risk modeling. The PSD mass concentrations of PAHs that are not recognized as carcinogens were used in the non-cancer endpoint risk model. The other equation variables and default values in both the cancer and non-cancer risk models are the same as those used in the Portland Harbor Public Health Assessment (ATS-DR, 2006) (Table S1).

Exposure ($\mu g\ kg^{-1}\ d^{-1}$) was calculated using Eq. (1) where C is the mass concentration (PSD substituted for fish), CF is a conversion factor, EF and ED are exposure frequency and duration, respectively, BW is body weight and AT is the averaging time (Table S1).

$$\text{Exposure} = \frac{C \times CF \times IR \times EF \times ED}{BW \times AT} \qquad (1)$$

The ingestion rates (IR) are the 90th (17.5 g d^{-1}) and 99th percentiles (142.4 g d^{-1}) for fish consumption that were used in the Portland Harbor Public Health Assessment (ATSDR, 2006) that evaluated local sport and subsistence angling populations. These rates may not apply to other situations. In order to assess potential public health implications of exposure, estimates of exposure can be compared to estimates of a dose that is likely to be without appreciable risk of deleterious effects, such as minimal risk level (MRLs) or reference doses (RfD) (ATSDR, 2006).

Excess cancer risk was determined by normalizing the slope factors for carcinogenic PAHs to benzo(a)pyrene and then multiplying by the sum contaminant exposure (Eq. (2)). Unacceptable cancer risk, as a matter of policy, was set at an excess of one in one million (1×10^{-6}).

$$\text{Excess cancer risk} = \text{Exposure} \times \text{slope factor} \qquad (2)$$

For the non-cancer endpoint each contaminant's exposure was divided by a chronic RfD or MRL to determine a hazard quotient (HQ) for the chemical (Eq. (3)). The sum of the HQs for the individual chemicals yields the hazard index (HI) and, as a matter of policy, a HI exceeding one represents an unacceptable risk.

$$\text{Hazard quotient} = \frac{\text{Exposure}}{\text{Rfd or MRL}} \qquad (3)$$

Analysis of exposure data was carried out using S-plus® (8.0, Insightful Corp.); Wilcoxon rank sum tests were used for seasonal comparisons and Kruskal–Wallis was used for analysis of spatial differences in exposure, followed by multiple comparisons using the Tukey 95% simultaneous confidence intervals method. Spatial and temporal differences in cancer risk were analyzed using Mann-Whitney rank sum tests in SigmaStat®. SigmaPlot® was used for graphing.

3. Results

A total of 110 samples, from 3 years and 10 different sampling events are included in this study: six dry (summer) and four wet season (fall and spring) events, defined by river flow. The wet season is defined as flow greater than 300 m^3 s^{-1}; median flows were higher during the wet season (494 m^3 s^{-1}) than during the dry season (246 m^3 s^{-1}, $p < 0.001$). Results for water chemistry parameters support the seasonal delineation; the dry season had higher temperature (22 vs. 16 °C), higher specific conductivity (0.1 vs. 0.08 mS cm^{-1}), lower ORP (139 vs. 196 mV; all $n = 17$, $p < 0.05$), but no difference in pH (7.4, $p = 0.9$).

Relative standard deviation (RSD) for the PAH concentrations at duplicate sites averaged 19%. Target compounds in blanks were either non-detect or below levels of quantitation. Results are recovery corrected. Recoveries from method spikes range from an average 43% for NAP, the lowest molecular weight and most volatile PAH, to 108% for IPY, with an overall average of 77%.

A detailed analysis of spatial and temporal variations of water concentrations of PAHs in the lower Willamette River can be found in Sower and Anderson (2008). Briefly, the sum concentration of 16 PAH analytes ($\sum_{16}$PAH) in the Superfund area (11.4 ng L^{-1}) is significantly higher than upriver sites (3.1 ng L^{-1}, $p < 0.001$), but not downriver sites. The upriver area does not exhibit significant variation among sites, but Superfund sites do. RMs 7W, 6.5W and 5W are consistently the most contaminated sites. None of the average concentrations for any site exceed the EPA human health Water Quality Criteria for consumption of water (3.8 ng L^{-1}) or 'water + organism' (18 ng L^{-1}) (US EPA, 1999) for the total carcinogenic PAHs, though some sites exceeded the threshold seasonally or during specific sampling events.

3.1. Comparison of PSD and fish tissue concentration data

While it is widely understood that humans do not consume passive samplers, comparisons of PAH concentrations in PSDs and fish tissue from the Portland Harbor Superfund site demonstrate that using PSD concentrations in a Public Health Assessment would provide a reasonable and conservative estimate of exposure that would be protective of human health without significantly overestimating risk. Table 1 presents fish tissue data from the Lower Willamette Group (Integral et al., 2009), some of which was used in the Portland Harbor Public Health Assessment (ATSDR, 2006) as well as PSD data from this study. The fish and shellfish were collected from Portland Harbor during a period that overlapped with the PSD study; however these two studies are unrelated to one another. Furthermore, it is important to highlight that PAHs were not included in the Portland Harbor Public Health Assessment because of insufficient data (ATSDR, 2006); therefore, the data presented in Table 1 is based on a limited sample set. The side-by-side comparison demonstrates that PSDs from this study captured the magnitude, range and variability of PAH concentrations that have been reported in a variety of fish and shellfish tissues from the harbor and provide an estimate of exposure that is realistic and protective.

3.2. Spatial and temporal variations in PAH exposure

As detailed in Section 2, exposure to PAHs from consumption of fish is dependent on a number of factors; some of which have standard values in risk assessment models, and others that are determined for specific human populations, such as consumption rates of organisms. In this study, the mass:mass concentrations of PAHs in LFT passive samplers are substituted for fish tissue concentrations in the exposure formula. Exposure is therefore a factor of consumption rate on the measured PSD concentrations.

To avoid confounding the interpretation of spatial differences in exposure to PAHs, data that were acquired during the tar removal dredging in the Superfund have been removed from these analyses. The effects of remediation activities on exposure and risk are discussed later in the results.

Significant differences in PSD concentrations of the $\sum_{16}$PAH were observed within and outside of the Superfund megasite ($p < 0.001$). A median $\sum_{16}$PAH concentration of 603 µg kg^{-1} in the Superfund was significantly greater than 431 µg kg^{-1} at upriver sites ($p < 0.001$) but not greater than the downriver area. Similarly, significant differences in carcinogenic PAHs were observed ($p < 0.001$), where median exposure was greater within the Superfund (12.1 µg kg^{-1}) than upriver (5.7 µg kg^{-1}) but not downriver.

A more detailed analysis of PSD concentrations shows significant differences between sites, both within and outside of the Superfund megasite for $\sum_{16}$PAH and carcinogenic PAHs ($p = 0.002$ and $p < 0.001$ respectively). Exposure to $\sum_{16}$PAH is greater at RM 7W and 3.5W (1110 and 1150 µg kg^{-1} medians respectively) than three upriver sites, which had median concentrations between 353 and 466 µg kg^{-1}. A similarly high median concentration of $\sum_{16}$PAH was observed in PSD extracts from RM 6.5W (1270 µg kg^{-1}); however this site was not differentiated from other sites in the analysis, likely due to a smaller sample size. Furthermore, the PSD concentration at RM 7W was significantly greater than at RM 8E (448 µg kg^{-1} median), though both sites are located within a mile of each other in the Superfund megasite (Fig. 2).

Median PSD concentrations of carcinogenic PAHs were greater at RM 3.5W (22.3 µg kg^{-1}) than RMs 18.5E, 17E and 12E (4.2, 5.5 and 6.4 µg kg^{-1} respectively). Additionally, it was greater than at RM 8E (10.1 µg kg^{-1}), which is also located in the Superfund megasite. Interestingly, RM 7W did not differentiate itself from other sites with regards to carcinogenic PAHs, although it had

Table 1
Concentrations of PAHs in PSDs and fish and shellfish tissue from the Portland Harbor Superfund site.

| Chemical	Concentration (μg kg^{-1}) – average (maximum)						
	PSD[a]		Fish and shellfish from Portland Harbor Superfund[b]				
	Superfund	Upstream	Smallmouth bass	Carp	Sculpin	Crayfish	Clam
Naphthalene	1.0 (6.5)	0.7 (3.8)	10 (86)	20 (56)	19 (250)	0.82 (2.9)	25 (78)
Acenaphthene	5.5 (54)	0.02 (1.1)	13.7 (95)[c]	34.1 (75)[c]	NA	NA	NA
Fluorene	13 (84)	5.4 (70)	9.31 (69)[c]	22.3 (53)[c]	NA	NA	NA
Phenanthrene	44 (219)	4.9 (24)	20 (85)	10 (16)	6.8 (33)	52 (97)	35 (300)
Fluoranthene	170 (850)	24 (57)	2.77 (36)[c]	NA	NA	10.2 (130)[c]	NA
Benz(a)anthracene	51 (504)	10 (44.6)	NA	NA	NA	2.01 (80)[c]	NA
Chrysene	36 (172)	10 (28)	20 (85)	NA	NA	2.16 (87)[c]	NA
Pyrene	170 (733)	35 (92)	2.9 (39)[c]	NA	NA	4.02 (83)	NA
Benzo(a)pyrene	14 (70)	4.1 (21)	0.64 (1.3)	NA	NA	1.1 (7.5)	34 (490)
$\sum_{16}$PAH	819 (3094)	397 (1147)	71.5 (308)	85.5 (222)	52.3 (550)	71.2 (477)	478 (4980)
$\sum$carcinogenic PAH	23 (123)	7.6 (25.2)	2.5 (6.8)	2.1 (2.8)	3.18 (9.8)	22 (170)	220 (2700)

NA indicates that data was not available for this publication.

Notes: PSD average and maximum concentrations are based on measurements made during the study period; data obtained in the superfund during tar removal remediation were not included. Concentrations in organisms correspond to reported whole body measurements in fish and shellfish obtained from the Portland Harbor Superfund site in an unrelated study. Not all analytes used to compute reported totals were available to be shown in this table.

[a] PSD measured concentrations of PAH analytes (this study).
[b] Data from the Lower Willamette Group Portland Harbor RI/FS (Integral et al., 2009) except where noted (c).
[c] Data from Portland Harbor Public Health Assessment (ATSDR, 2006).
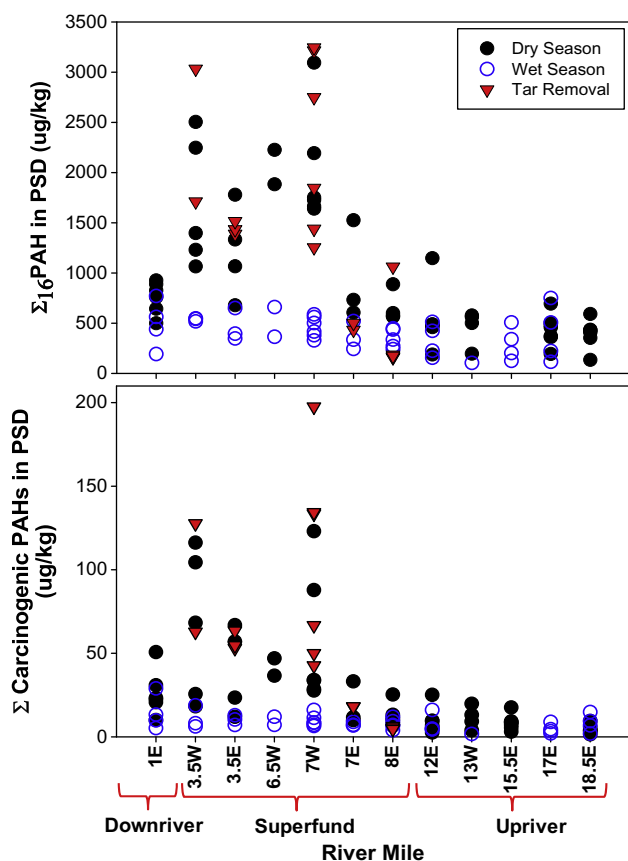
Fig. 2. $\sum_{16}$PAH and carcinogenic PAHs in PSDs. Mass-to-mass concentration of sum PAHs and sum carcinogenic PAHs in passive sampling devices (PSDs) at sites downriver, upriver and within the Portland Harbor Superfund megasite. Each point represents one observation during the dry season (closed circles), wet season (open circles) or tar removal remediation (triangles). These values were used in place of fish tissue concentrations to calculate exposure for risk assessment models.

significantly higher levels of total PAHs than sites located both within and outside of the Superfund area (Fig. 2).

No differences in exposure to total or carcinogenic PAHs were observed between the wet and dry seasons at the upriver or down-river sites. In contrast, significant differences were observed between the wet and dry season for both carcinogenic and total PAHs within the Superfund megasite ($p < 0.001$). PSD concentrations of PAHs in the Superfund megasite were greater during the dry than the wet season (1470 and 442 μg kg^{-1} respectively). Similarly, median concentrations of carcinogenic PAHs were 33.5 μg kg^{-1} in the dry season compared to 8.5 μg kg^{-1} in the wet season.

### 3.3. Spatial and temporal variations in cancer and non-cancer risk

All areas exceed the established threshold of one excess cancer risk in 1 000 000 (1×10^{-6}). Estimated risk of cancer in excess of the background rate for the Superfund megasite and downriver (1.3×10^{-5} and 1.7×10^{-5}, respectively, for average consumption) were significantly higher than upriver sites (4.5×10^{-6}, $p < 0.001$) (Fig. 3). Within the Superfund megasite, estimated excess risk was up to five times greater at RM 7E than 7W; sites located on opposite banks and separated by only a few hundred meters (Table 2). These estimated numbers of cancer cases in excess of the background are based on the assumption, from the risk assessment model applied here, that all individuals are equally exposed.

Non-cancer risk from PAHs was also higher at Superfund and downriver sites than urban areas ($p < 0.001$) with RMs 7W, 6.5W, and 3.5W exhibiting the highest hazard quotients, though all were below one by more than two orders of magnitude (Table 2).

The increased PAH concentrations in the Superfund area during the dry season result in significantly elevated risk (Fig. 3). Both non-cancer HQs and excess cancers increased during the dry period ($p = 0.004$ and $p < 0.001$ respectively), however the non-cancer HQs remained below unacceptable risk levels (Table 2). The cancer model predicts four times greater cancer risk from fish consumption in the Superfund area during the dry season compared to wet season. Notably, the excess cancer risk at RM 7W from average consumption of PSD measured mass concentrations increases by sevenfold during the dry season from 7.8×10^{-6} to 6.0×10^{-5} ($n = 18$, $p = 0.005$).

3.4. Effects of remediation activities on risk

Dredging of a coal tar contaminated site at RM 6.3W removed more than 11 500 m^3 of submerged tar contamination from August
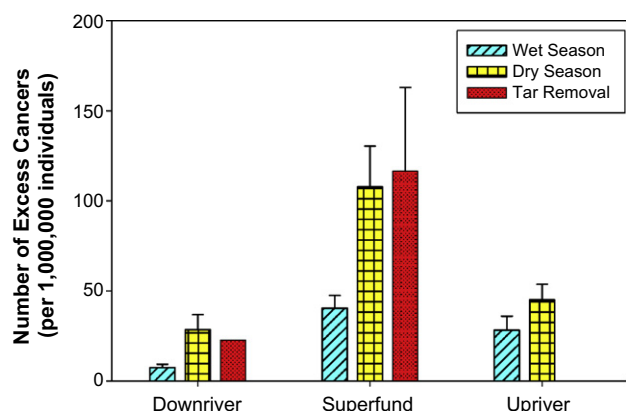
Fig. 3. Estimated number of cancers, in excess of the background rate, per 1 000 000 individuals exposed to carcinogenic PAHs. Calculations are based on average fish consumption rates; where PSD concentrations have been substituted for fish tissue concentrations. Data from all sites located in each area of the river (upriver, downriver and within the Superfund) were averaged for the wet and dry seasons and observations associated with tar removal remediation activities are presented separately. Error bars represent 95% confidence intervals, based on variability in the PSD measured concentrations for each site-season, and only one observation was made at the downriver site during tar removal. See Table 2 for statistical analyses of these data.

to October 2005. LFT samplers downriver at RM 3.5W and upriver at RM 7W during this period accumulated significantly elevated $\sum_{16}$PAH and carcinogenic PAH concentrations (Fig. 2). September 2005 samples, taken during the middle of the dredging activity, from RM 7W down to RM 1 are the highest concentrations of $\sum_{16}$PAH and carcinogenic PAHs recorded during this study (Fig. 2). The median $\sum_{16}$PAH and carcinogenic PAH pre- and post-tar removal are significantly lower than the tar removal median. The highest observed carcinogenic PAH concentrations in water measured during this study occurred at RMs 7W and 5W in September 2005 (71 ng L^{-1} and 20 ng L^{-1}, respectively). At RM 3.5W, chrysene and benz(a)anthracene exceed the US EPA Water Quality Criteria limit of 3.8 ng L^{-1} for the consumption of water and organism, while at RM 7W benzo(b)fluoranthene and benzo(a)pyrene exceeded this limit and chrysene and benz(a)anthracene exceeded the 18 ng L^{-1} limit for the consumption of organisms.

4. Discussion

PSDs are well established for determining the water concentrations of freely dissolved and thus bioavailable, organic contaminants (Huckins et al., 2006; Adams et al., 2007; Anderson et al., 2008). Their use for risk assessment is less well established, however, they respond to the need for biologically relevant exposure data (Birnbaum, 2010) and they can be standardized across studies. Furthermore, initial comparisons of PAH concentrations in PSDs and fish tissue demonstrate that PSDs capture the magnitude and variability of PAH exposure, and thus are an adequate surrogate for this parameter in some risk models. Obtaining PSD data from sites within and outside of the Superfund area provided a more representative range of concentrations for highly mobile fish species that are likely to move through large areas of the river and might avoid contaminated areas. Conversely, less motile or sessile organisms, such as crayfish and clams from the Superfund area had concentrations of PAHs in their tissues more closely aligned with PSD data from the Superfund area. Fin fish, unlike PSDs, ingest and metabolize PAHs, however passive partitioning has been shown to be the principal route of uptake (Connell, 1990) and the results of this study concord with other publications that demonstrate the comparability of PSDs (SPMDs) with finfish and bivalves (Huckins et al., 2006). As mentioned by other researchers (Baussant et al., 2001; Ke et al., 2007) a comparison of this study to fish tissue data from the area (Table 1) demonstrated higher concentrations of low molecular weight PAHs in fin fish than PSDs. This observation merits further study; however, due to these compounds classification as non-carcinogenic and their relatively high MRLs, the lower concentrations observed in PSDs do not have a significant effect on the outcomes of a Public Health Assessment based on the PSD data.

Using PSDs as direct biological surrogates by measuring unmetabolized parent compounds through mass:mass concentrations reveals a more complete exposure potential. In Portland Harbor, the large number of PSD samples over several seasons and years, provided a much more complete understanding of risk for the area, with specific spatial and temporal resolution that proved to be significant. Notably, risk from exposure to PAHs from consumption of fish had not been evaluated in the Public Health Assessment for Portland Harbor due to insufficient fish data. Using PSDs in place of organisms eliminates problems associated with capturing

Table 2
Cancer and non-cancer risk associated with consumption of fish estimated by PSD[a],[b].

River location and season		N	Non-cancer hazard quotient consumption			Excess cancer risk consumption		
			Average	High	p-Value	Average	High	p-Value
Upriver		37						
	Wet	13	2.0×10^{-4}	1.3×10^{-3}		3.8×10^{-6}	3.1×10^{-5}	
	Dry	24	4.0×10^{-4}	3.3×10^{-3}	0.07	5.6×10^{-6}	4.6×10^{-5}	0.31
Superfund megasite		64						
	Wet	28	9.3×10^{-4}	7.6×10^{-3}		6.7×10^{-6}	5.4×10^{-5}	
	Dry	36	3.3×10^{-3}	2.7×10^{-2}	0.004	2.6×10^{-5}	2.1×10^{-4}	<0.001
Superfund no tar events		45						
	Wet	22	3.6×10^{-4}	3.0×10^{-3}		6.5×10^{-6}	5.3×10^{-5}	
	Dry	23	2.9×10^{-3}	2.4×10^{-2}	<0.001	2.2×10^{-5}	1.8×10^{-4}	<0.001
RMs 7W and 5W		28						
	No tar	19	1.1×10^{-3}	8.5×10^{-3}		1.5×10^{-5}	1.2×10^{-4}	
	Tar	9	4.5×10^{-3}	3.7×10^{-2}	0.02	9.1×10^{-5}	7.4×10^{-4}	<0.001
Downriver		10						
	Wet	4	3.4×10^{-4}	1.5×10^{-2}		9.1×10^{-6}	7.4×10^{-5}	
	Dry	6	1.9×10^{-3}	2.8×10^{-3}	0.11	1.8×10^{-5}	1.5×10^{-4}	0.18
			Threshold = 1			Threshold = 1.0×10^{-6}		

All test are at $\alpha = 0.05$.
[a] Mann–Whitney rank sum tests within location between seasons.
[b] p-Values are for comparisons between seasons.

samples, destructive sampling and analyzing compounds in an analytically complex biological matrix.

Temporal disparities in exposure and estimated risk were observed in the Superfund area. Several studies have observed higher PAH concentrations with increasing precipitation, flows, and urban runoff (Ko and Baker, 2004; Gasperi et al., 2005; Brown and Peake, 2006) and Stout et al. (2004) note that storm water is the greatest contributor to sediment PAHs over time. However, our data demonstrate an opposite tendency, where the dry season is associated with higher water concentrations, higher exposure, and consequently higher risk, in the Superfund area. Dilution does not explain the concentration and risk disparities between wet and dry seasons in the Superfund area either. Unlike the Superfund sites, upriver and downriver areas do not demonstrate seasonal variations. If the observed differences in the Superfund were due to dilution, this should be a uniform effect in the river. One potential explanation for the seasonal differences observed only within the Superfund site, and especially at 7W, 6.5W and 3.5W, is that contaminant diffusion from sediments into overlying water is responsible for high concentrations. The contamination may be from riverbank sediments and higher wet season flows could inhibit groundwater movement into the river due to hydraulic pressure and bank storage (Winter et al., 1998; Sower and Anderson, 2008). Another possible explanation is that higher summer temperatures cause greater contaminant diffusion from the sediment to the water column. Further investigation is required to elucidate sources of seasonal disparities in PAH contamination in the Superfund area.

A sediment cap over creosote contaminated sediments at RM 7E, installed prior to this study, was found to be effective in preventing PAH contamination into the overlying water column (Sower and Anderson, 2008) but did not diminish RM 7W high concentrations. The cause of the significant difference observed between sites located in close proximity to one another, such as RMs 7E, 7W and 8E, merits further study. It also highlights the importance of considering spatial differences in risk on a small scale, which can be achieved by taking PSD data into account in risk assessments.

While remediation of contaminated sites is desirable, few studies have assessed the potential impacts of dredging on exposure and risk during and after remediation (Committee on Sediment Dredging at Superfund Megasites, 2007). This study provided an opportunity to evaluate the effects of dredging on PAH bioavailability and potential human health risks from exposure. Prior to capping, dredging at RM 6.3W removed significant quantities (>11 500 m³) of coal tar; however the area remains a higher risk with higher freely-dissolved PAH concentrations than surrounding areas, particularly in the dry season.

This study demonstrates an association between variable flows, sediment disturbance and freely-dissolved and, thus, bioavailable contamination in the water column. Although the dredging produced a spike in exposure to PAHs, and a corresponding increase in risk values, the duration of the effect was limited to the time that it took to complete the operation. The short duration of the disturbance would only be expected to have an immediate and more substantial effect on aquatic organisms. Though fish kills were observed within the containment area, none were observed outside the barriers (Parametrix, 2006).

The site downriver from the Superfund megasite, RM 1E, is not significantly different in concentration from the Superfund sites. While the Portland Harbor Public Health Assessment only sampled within the Portland Harbor Superfund sites, our data demonstrate that the downriver site has similar concentrations and could pose similar health risks. Seasonal and spatial information like this could be useful to public health officials when constructing a health assessment or determining where to post warning signs.

5. Conclusions

PSDs provide spatially and temporally resolved contaminant exposure information that, as demonstrated here, can be incorporated into risk assessment models. This study revealed significant spatial and temporal differences in risk that would not have been elucidated in a traditional risk assessment, such as the Portland Harbor Public Health Assessment. Although it is clear that humans do not consume PSDs, their application as a biological surrogate in risk assessment models has the potential to provide specific spatial and temporal contaminant exposure information that can assist public health professionals in accurately evaluating human health risks. Furthermore, using PSDs for risk assessment has the advantages of larger sample size, non-destructive sampling and comparability across studies. PSDs provide biologically relevant exposure data for risk assessment that could be used when organism data is not available or to complement, and further refine, other measures of exposure.

Acknowledgements

Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.chemosphere.2011.06.051.

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