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Combined Effects of Contaminant Desorption and Toxicity on Risk from PAH Contaminated Sediments

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A strong inverse correlation was observed between the polycyclic aromatic hydrocarbon (PAH) mass fraction desorbed, a surrogate measure of bioavailability, and relative carcinogenicity, as quantified by potency equivalency factors (PEFs), for two study sediments from the New York/New Jersey Harbor estuary. Because compounds with the highest toxicity, such as dibenz(a,h)anthracene and benzo(a)pyrene (BAP), also tended to be the least rapidly and least extensively desorbed, the U.S. Environmental Protection Agency (EPA) default guidance may dramatically overestimate risk from exposure to PAH-contaminated soils or sediments. A "relative risk index" (RRI) was developed to account for the combined effects of compound-specific bioavailability and toxic potency in estimating excess cancer risk. Using this approach, estimated excess cancer risk may be diminished by as much as a factor of 159 times versus default EPA guidance. Also, the hierarchy of estimated risk between study sediments and among treatment fractions of study sediments differed using the two approaches, implying that the default approach may inaccurately determine site clean-up priorities. The percentage contribution of each potentially carcinogenic priority PAH to total excess cancer risk was computed under various scenarios. In each case, the contribution of BAP to total excess cancer risk was remarkably invariable, for example, ranging from 48% to 52% in one sediment, and 44% to 54% in the other, over four different exposure durations. These results suggest that BAP may be an excellent indexing compound for gauging relative exposure risk across sediments. Other important contributors to total excess cancer risk were benz(a)anthracene and dibenz(a,h)anthracene. Together, these three compounds comprised nearly 90% of total excess cancer risk from all PAHs in every scenario. This integrated RRI approach may enable regulators to more accurately gauge relative risks and make more informed sediment management decisions.

KEY WORDS: Bioavailability; cancer endpoints; exposure assessment; polycyclic aromatic hydrocarbons (PAHs); potency equivalency factors (PEFs)

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1. INTRODUCTION

Multiple studies have found the rate and extent of contaminant removal decreases with contaminant-sediment (or soil) contact time (Alexander, 2000; MacLeod & Semple, 2000; Reid *et al.*, 2000; Shor & Kosson, 2000). The phenomenon is especially pronounced for hydrophobic organic contaminants (HOCs) such as polycyclic aromatic hydrocarbons (PAHs). Because of significant mass transport limitations with aging, total contaminant concentration may

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Table I. Selected Properties of 4-, 5-, and 6-Ring Priority PAHs Measured in Newtown Creek (NC) or Piles Creek (PC) Sediments

	PAH Conc. # ($mg\ kg^{-1}$)			Mol. Wt.[a] ($g\ mol^{-1}$)	Aqueous Solubility[a] ($mg\ L^{-1}$)	log K_{ow}[a]	Cancer Class[b]	PEF EPA[c]	PEF Nisbet and LaGoy[d]
	NC	PC	Rings						
Fluoranthene	3.8	6.4	4	202	0.26	5.2	D		0.001
Pyrene	3.7	6.3	4	202	0.135	5.2	D		0.001
Benz(a)anthracene	2.0	3.2	4	228	0.014	5.9	B2	0.1	0.1
Chrysene	3.8	3.3	4	228	0.002	5.9	B2	0.001	0.01
Benzo(b)fluoranthene	1.9	4.1	5	252	0.0015	6.5	B2	0.1	0.1
Benzo(k)fluoranthene	0.8	1.7	5	252	0.00081	6.8	B2	0.01	0.1
Benzo(a)pyrene	1.1	3.1	5	252	0.0038	6.5	B2	1 (index)	1 (index)
Dibenz(a,h)anthracene	0.6	1.6	5	278	0.0005	7.2	B2	1	5
Benzo(g,h,i)perylene	1.6	2.9	6	276	0.00026	6.9	D		0.01
Indeno(1,2,3-CD)pyrene	0.8	1.9	6	276	0.00019	7.7	B2	0.1	0.1
Total PAHs	21	35							
Total cPAHs	11	19							

[a]Data from Mackay *et al.* (1992).
[b]Cancer class from U.S. EPA guidance (2002).
[c]Potency equivalency factor (PEF) values after EPA guidance (1993).
[d]Potency equivalency factor (PEF) values after Nisbet and LaGoy (1992).

be a poor predictor of the bioavailable[5] concentration (Alexander, 2000; U.S. EPA, 2000). Thus, the real danger posed by exposure to such aged contaminants in the environment is uncertain. For example, one recent study computed "environmentally protective" PAH concentration thresholds in soil 3 to 10 times higher than California default assumptions when considering the unavailable fraction (Stroo *et al.*, 2000). Current standard approaches that do not explicitly consider bioavailability (e.g., U.S. EPA, 1993) may magnify or distort actual risks. As a result, scarce resources may be diverted away from instances where the risks to human health are the greatest.

PAHs are commonly detected in U.S. surface waters (Kolpin *et al.*, 2002), and in the sediments underlying U.S. ports, harbors, and other industrialized waterways (NRC, 1997; U.S. EPA, 1998). The U.S. Environmental Protection Agency (EPA) has designated 17 PAHs as priority pollutants, and 7 of these as level B2 "probable human carcinogens" (U.S. EPA, 2001) (Table I). Various PAHs may also cause other adverse health effects, including liver, kidney, and hemoglobin toxicity (U.S. EPA, 2001). Sources of PAHs in the environment include naturally occurring deposits, incomplete combustion of fossil fuels, oil refining, and petrochemical spills. A combination of low aqueous solubility, low vapor pressure, and high hydrophobicity causes PAHs to accumulate in sediments. The rate and extent of PAH mass transport from sediment depends on many factors, including structure-activity properties of individual PAHs, sediment properties (e.g., sediment particle size and structure, and organic matter concentration and properties), temperature, aqueous chemistry (e.g., dissolved organic carbon concentration and properties), and time (Shor *et al.*, 2003b). A recent study has shown bioavailability of sediment-sequestered PAHs to microorganisms is limited by slow mass transport to the aqueous phase (Shor *et al.*, 2003a). Another study showed the quantity of PAHs that are in the "rapidly desorbing fraction" might control the extent of PAH biodegradation in sediments (Cornelissen *et al.*, 1998).

In addition to controlling the bioavailability of PAHs in sediments to microorganisms, mass transport affects bioavailability of PAHs in other systems as well. In one study, PAHs passed more quickly through monkey skin and into receptor solution when they were applied in an acetone/artificial sweat solution versus applied in oil; moreover, more hydrophobic PAHs, including benzo(a)pyrene (BAP), only passed through the skin in measurable quantities when applied in solution of acetone/sweat (Sartorelli *et al.*, 1999). Another study investigated bioavailability of PAHs to worms (Mayer *et al.*, 1996): in 4 hours at 22°C in a well-mixed system, only about 5% of PAHs were extracted from each of two field-aged

[5] Where bioavailability is defined after Alexander as "the accessibility of a chemical for assimilation and possible toxicity" (Alexander, 2000).

PAH-contaminated sediments in a solution of digestive fluids collected from marine deposit feeders. The authors observed the highest percentage solubilization for the 3- and 4-ring PAHs, while more carcinogenic 5- and 6-ring compounds including BAP were only about 3% solubilized. These studies suggest that more carcinogenic PAHs may also be less bioavailable. Integration of compound- and sediment-specific mass transport properties with relative toxicity has important implications for both human health and ecological risk assessment, as suggested by other reports (Brown *et al.*, 1999; Peters *et al.*, 1999). Although the decreased bioavailability of more hydrophobic PAHs has been incorporated into equilibrium models such as equilibrium partitioning theory (EPT) (DiTorro *et al.*, 1991), exposure is typically not an equilibrium process. Other factors such as sequestration status (Kraaij *et al.*, 2002) and desorption kinetics should also be considered to more accurately describe exposure and predict risk.

This study introduces a theoretical framework for more accurately accounting for bioavailability in human health risk calculations; however, the broader implications may also be applicable to ecological risk assessment. The approach is based on computing the "relative risk index" (RRI), which incorporates compound-specific bioavailability and toxicity for a given biota-sediment contact time. The RRI approach is general and can be used to compute relative risks from exposure to any mixture of organic or inorganic contaminants within any solid matrix, provided relative toxic potency data are available for a given risk endpoint. In this study, excess cancer risk from human exposure to PAH-contaminated sediments is used as a test case. The experimental data presented here demonstrate the substantial variability in PAH availability and predicted risk as a function of sediment source and fraction, PAH compound, and contact time. Most notably, the RRI approach to incorporating bioavailability into risk prediction results in a significant diminution in total predicted excess cancer risk as compared with the default approach due to the combined effects of low bioavailability for PAHs with the highest toxicity.

2. CONCEPTUAL MODEL

Risk from exposure to hazardous substances like PAHs is typically computed by incorporating a measure of the total quantity of contaminant to which an individual has been exposed, or dose, with a measure of toxicity per dose. The results may be expressed in the form of a hazard quotient for noncancer human health endpoints, or for carcinogenic compounds, in terms of excess cancer risk (the number of additional cancer deaths that might result in a lifetime of exposure to a population of 1 million people). Although the U.S. EPA has designated 7 PAHs as "probable human carcinogens," a cancer-slope factor (the dose-response relation needed to compute excess cancer risk) is available only for benzo(a)pyrene (BAP) (U.S. EPA, 2001). As reviewed recently (Reeves *et al.*, 2001), there have been several approaches to estimate the carcinogenicity of other PAHs. One approach is to apply the cancer slope factor for BAP to the total concentration of all potentially carcinogenic PAHs in a given matrix (the "total cPAH" approach). This approach may overestimate the actual cancer risk, because most PAHs are less toxic than BAP. Other researchers have examined the available data and have proposed "potency equivalency factors" (PEFs) to weight the relative carcinogenicity of other PAHs relative to BAP. PEF values reported by Nisbet and LaGoy (1992) and provisional guidance by the U.S. EPA (1993) (Table I) continue to be widely used and cited (Hussain *et al.*, 1998; Peters *et al.*, 1999) and are consistent with other PAH potency equivalency studies (Collins *et al.*, 1998).

Using the PEF approach, the cancer slope factor for BAP, CsF_{BAP}, is applied directly to the BAP-equivalent dose, BAP_{eq} dose:

$$\text{Risk} = CsF_{BAP} \cdot BAP_{eq}\ \text{dose} \tag{1}$$

where BAP_{eq} dose is defined as the summation of the doses of each PAH, i, weighted by its corresponding potency equivalency factor, (PEF_i):

$$BAP_{eq}\ \text{dose} = \sum_i \text{dose}_i \cdot PEF_i. \tag{2}$$

In turn, the dose from exposure to PAH-contaminated sediment is computed by multiplying the sediment concentration by a set of exposure parameters appropriate for a given exposure scenario. For example, typical equations for computing dose from exposure to sediment via incidental ingestion and dermal contact pathways (after Adams *et al.*, 1994) are:

(I) for incidental ingestion of contaminated sediment [mg PAH kg^{-1} day^{-1}]:

$$\text{dose}_i = \frac{C_i \cdot IR \cdot EF \cdot ED \cdot cf}{BW \cdot AT}, \text{ or} \tag{3}$$

(II) for dermal contact with contaminated sediment [mg PAH kg^{-1} day^{-1}]:

$$\text{dose}_i = \frac{C_i \cdot AB \cdot SA \cdot AF \cdot EF \cdot ED \cdot cf}{BW \cdot AT} \quad (4)$$

where

AB = Dermal adsorption fraction [dimensionless]
AF = Dermal adherence rate for sediment [mg cm^{-2}]
AT = Exposure averaging time [days]
BW = body weight [kg]
C_i = Concentration of compound, i [mg kg^{-1}]
cf = Conversion factor [10^{-6} kg mg^{-1}]
ED = Exposure duration [years]
EF = Exposure frequency [days $year^{-1}$]
IR = Ingestion rate [mg exposure-day^{-1}]
SA = Dermal surface area exposed [cm^2]

For the incidental ingestion exposure pathway, EPA guidance is to use the total PAH concentration in sediment in computing a dose (U.S. EPA, 1997). This approach does not take into account the fraction of the total soil or sediment PAH concentration that is permanently bound, or the PAH fraction that does not desorb during the biota-sediment contact time interval. For the dermal exposure pathway, EPA guidance is to use the total concentration of a given hydrophobic compound, scaled by a dermal absorption fraction, AB, where $AB = 0.13$ for PAHs (U.S. EPA, 1999), regardless of sediment characteristics, molecular properties of the compound of concern, or biota-sediment contact time. These default approaches may be inadequate. A better approach may be to explicitly incorporate a measure of bioavailability into the estimation of exposure dose. In such a formulation, the total concentration of a given PAH in sediment, C_i, in Equation (3) and the product of C_i and dermal absorbed fraction, AB, in Equation (4) would be replaced by $C(t)_i'$, which is the PAH concentration that is bioavailable in a given time interval, t. The time interval is defined as the characteristic duration of contact between the matrix and the receptor.

The bioavailable fraction must be established for each different sediment, for each contaminant, and is defined only for a given biota-sediment contact time. The time dependence on the bioavailable fraction is critical. As with release of other contaminants from other solid matrices, desorption of PAHs from soils and sediments is a slow diffusive process driven by the magnitude of the concentration gradient between internal regions where contaminants are sequestered and the solid surface where a lower concentration is maintained (Shor *et al.*, 2003b). The longer sediment is in contact with the skin, lungs, or gut of humans or other biota, the longer the surface concentration remains depleted, and a greater percentage of total contaminants will have an opportunity to desorb. Also, for a given exposure event, the rate of contaminant release is greatest initially, and lessens at longer biota-sediment contact times. Subsequent exposure to other parcels of sediment must be considered as independent exposure events, and the "bioavailability interval clock" reset. For this reason, it makes a substantial difference to bioavailability, and therefore risk, if a person is exposed to contaminated sediment for 10 minutes on each of 1,000 separate occasions, or 1,000 minutes on each of 10 separate occasions. Thus, the correct approach is to incorporate the biota-sediment contact time into the bioavailable contaminant concentration. Other temporal parameters gauging the frequency of exposure in a person's lifetime are modeled as usual.

With this modification, Equations (3) and (4) become

(I) for incidental ingestion of contaminated sediment [mg PAH kg^{-1} day^{-1}]:

$$\text{dose}_i = \frac{C(t)_i' \cdot IR \cdot EF \cdot ED \cdot cf}{BW \cdot AT}, \text{ and} \quad (5)$$

(II) for dermal contact with contaminated sediment [mg PAH kg^{-1} day^{-1}]:

$$\text{dose}_i = \frac{C(t)_i' \cdot SA \cdot AF \cdot EF \cdot ED \cdot cf}{BW \cdot AT}. \quad (6)$$

In each case, all exposure parameters besides $C(t)_i'$ are independent of bioavailability. In order to explore how bioavailability affects risk, independent of other exposure parameters, all other terms are grouped together into a single parameter called "other parameters," OP. Thus, the dose of a given PAH, i, resulting for a given hypothetical exposure scenario j, can be written as

$$\text{dose}_{i,j} = C(t)_i' \cdot OP_j. \quad (7)$$

Because each of the terms making up OP is independent of bioavailability, sediment, or PAH characteristics, they can be removed as a group from the summation to compute a BAP-equivalent dose. Therefore, combining Equations (1), (2), and (7) yields a compact form to describe bioavailability-dependent risk:

$$\text{Risk} = \text{CsF}_{\text{BAP}} \cdot OP_j \cdot \sum_i (C(t)_i' \cdot \text{PEF}_i), \quad (8)$$

where an appropriate CsF_{BAP} must be defined for the exposure pathway, j. In order to facilitate comparison of relative risk from exposure to different PAH-contaminated sediments (for a given exposure scenario), the term "relative risk index," RRI [mg PAH kg^{-1}], is defined as

$$\mathrm{RRI} = \sum_i (C(t)'_i \cdot \mathrm{PEF}_i). \quad (9)$$

RRI is the component of total risk that involves the combined effects of compound-specific bioavailability and toxicity for a given biota-sediment contact time. Finally, the fractional contribution of each compound to total relative risk index, RRI_i, is defined as

$$\mathrm{RRI}_i = \frac{C(t)'_i \cdot \mathrm{PEF}_i}{\sum_i (C(t)'_i \cdot \mathrm{PEF}_i)} \times 100\%. \quad (10)$$

3. METHODS

3.1. Study Sediments

The effects of bioavailability on relative excess cancer risk were tested using study sediments from two sites, Piles Creek (40°36.53′N, 74°13.60′W) and Newtown Creek (40°44.28′N, 73°56.75′W), in the New York/New Jersey Harbor estuary. Piles Creek runs through an intertidal marsh region in an industrialized area and is characterized by coarsely graded sediment, a high concentration of vascular plant debris (primarily *Spartina alterniflora* and *Phragmites australis)*, and 3.7% organic carbon. Newtown Creek is an industrial waterway in New York City characterized by finely graded sediment dominated by silts and clays, low vascular plant debris, and relatively higher organic carbon content (6%). Sediment samples were collected from both sites, homogenized, and stored at 4°C until use as described previously (Rockne *et al.*, 2002). Split samples of sediments were further fractionated into high- and low-density portions (less than or greater than 1.7 g cm^{-3}) by equilibrium settling in a solution of CsCl in water, as described previously (Mayer *et al.*, 1993). Extensive chemical and physical characterization data for both whole and fractionated sediments are reported elsewhere (Rockne *et al.*, 2002).

PAH content for sediments and sediment fractions were quantified in triplicate by a hot acetonitrile sonication extraction procedure as described (Rockne *et al.*, 2002). Briefly, wet sediments were dewatered by centrifugation (8000*g*, 20 min), and PAHs were extracted into hot acetonitrile in sealed Teflon Oak Ridge centrifuge tubes (85°C, 120 min) in an ultrasonic bath, and analyzed by reverse-phase HPLC separation with fluorescence and photodiode array peak quantification and identification. Mean PAH recovery efficiency was 76% from a NIST standard using the method described.

3.2. Desorption Studies

PAH desorption as a function of time was measured for both sediments by a modification of the method of Cornelissen *et al.* (1997) as described previously (Rockne *et al.*, 2002). Briefly, a known mass of wet sediment, Tenax®-TA beads (60–80 mesh, Alltech Associates Inc., Deerfield, IL), 0.45 μm filtered seawater, and $HgCl_2$ (to prevent microbial growth) were brought together and shaken (150 rpm) at 23 ± 0.5°C in the dark. At specified time intervals (approximately doubling between time periods of 30 minutes and one year) the sediment-seawater slurry was separated from the used beads and fresh Tenax beads were added. Used beads were then extracted with hexane and analyzed as described (Rockne *et al.*, 2002). Total PAH recoveries from the procedure (comparing PAH masses in split samples of untreated sediments with the cumulative masses desorbed plus the residual PAH masses remaining in the sediments at the end of the desorption experiments) were 99 ± 22% and 95 ± 24% for whole Piles Creek and Newtown Creek sediments, respectively (Rockne *et al.*, 2002).

Tenax beads in the slurry acted as an aqueous-phase "PAH sink," rapidly absorbing PAHs as they desorbed from the sediment and partitioned into the aqueous phase. Excess Tenax beads were added to ensure rapid and complete absorption of all PAHs in solution, and before beads began to be saturated with PAHs they were replaced with fresh beads. In this way, the aqueous PAH concentration was kept to near zero, and therefore the PAH concentration at the sediment surface (in local equilibrium with the aqueous phase) was also small. These experimental conditions were designed to engender maximum desorption without using high temperatures, harsh solvents, or altering the physical structure of the sediment. The results of the Tenax desorption experiments may therefore be considered a conservative estimate of bioavailability because it is unlikely that biological exposure pathways could provide equal PAH absorption capacity in the aqueous phase or as favorable mixing conditions to maintain the maximum-concentration gradient-driving diffusion. However, the experimental conditions could not replicate the conditions of the

lungs, skin, or gastrointestinal tract, where biological and chemical processes may enhance PAH uptake by mechanisms not considered in this study.

4. RESULTS AND DISCUSSION

4.1. PAH Distribution and Desorption Trends

The total priority PAH concentration in the study sediments for 4-, 5-, and 6-ring PAHs was 35 mg kg^{-1} for Newtown Creek sediment and 21 mg kg^{-1} for Piles Creek sediment (Table I). PAHs smaller than four rings were not included in this report because they do not contribute substantially to carcinogenicity (Nisbet & LaGoy, 1992; U.S. EPA, 1993). The ratios of both total PAH concentration and total carcinogenic PAH concentration between Newtown Creek and Piles Creek sediments were nearly 2:1. For the two most carcinogenic compounds, BAP and DBA, concentration ratios between the sediments were 2.8:1 and 2.6:1, respectively. Therefore, in terms of total PAH concentration, total cPAHs, and concentration distribution of PAHs, it would seem that exposure to Newtown Creek sediment is likely to present a greater risk than equivalent exposure to Piles Creek sediment (Table II).

However, information on PAH concentration distribution alone is inadequate to predict risk because it ignores the bioavailability of the contaminants. Mass transport is a kinetic process; therefore, it is essential to consider time when estimating bioavailability. In a short period of time only a small proportion of the PAHs are likely to be desorbed and, therefore, to be bioavailable. However, for much longer biota-sediment contact times, a much larger fraction can be desorbed. The cumulative extent of desorption for all 10 4-, 5-, and 6-ring Priority PAHs, including all Class B2 Priority PAHs, are shown for both study sediments at desorption intervals of 30 minutes, 1 day, 1 month, and 1 year, along with the undesorbed residual (Fig. 1) (desorption data were not available for benzo(g,h,i)perylene or indeno(1,2,3-CD)pyrene for Piles Creek sediment). In general, a larger fraction of PAHs desorb from Piles Creek sediment than from Newtown Creek sediment at all points in time.[6] The trend is the most pronounced at shorter time periods, where about 20% of fluoranthene and pyrene are desorbed in just 30 minutes from Piles Creek sediment, but only 6% are desorbed from Newtown Creek sediment. The ratio of mass fraction desorbed at 30 minutes between Piles Creek and Newtown Creek sediments averages 6:1 for all PAHs, and the difference is largest for the less hydrophobic compounds.[7]

The PAH distribution suggests that exposure to Newtown Creek sediment is a greater toxicity threat than equivalent exposure to Piles Creek sediment. However, the lower bioavailability of PAHs in Newtown Creek sediment may mitigate this risk, especially for short biota-sediment contact times appropriate for many realistic exposure scenarios. Even for chronic sediment exposure (occurring repeatedly over many months or years) each exposure episode

Table II. Comparison of Relative Risk from Hypothetical Exposure to PAH-Contaminated Newtown Creek (NC) and Piles Creek (PC) Sediments for Four Biota-Sediment Contact Times

	PEF EPA[a]		PEF Nisbet and LaGoy[b]	
	NC	PC	NC	PC
Sum priority PAHs (mg kg^{-1})	38	22	38	22
Total cPAHs (mg kg^{-1})	19	11	19	11
BAP_{eq}Conc (mg kg^{-1})	5.6	2.2	6.1	2.8
RRI (mg kg^{-1})				
0.5 hour	0.036	0.071	0.053	0.096
1 day	0.45	0.57	0.58	0.77
1 month	1.7	1.1	2.4	1.5
1 year	2.3	1.3	3.4	1.8
Risk ratio				
Total cPAHs/RRI				
0.5 hour	532	157	359	115
1 day	43	19	33	14
1 month	11	10	8	8
1 year	8.2	8.6	5.6	6.0
EPA default (100% availability)/RRI				
0.5 hour	159	31	116	29
1 day	13	3.8	11	3.6
1 month	3.2	2.0	2.6	1.9
1 year	2.5	1.7	1.8	1.5
EPA default (13% availability)/RRI				
0.5 hour	21	4.1	15	3.8
1 day	1.7	0.50	1.4	0.47
1 month	0.42	0.26	0.33	0.25
1 year	0.32	0.22	0.23	0.20

[a]Potency equivalency factor (PEF) values after EPA guidance (1993).
[b]Potency equivalency factor (PEF) values after Nisbet and LaGoy (1992).

[6] The key factors and controlling mechanisms resulting in differential rate and extent of PAH desorption from these study sediments are described in detail elsewhere (Rockne *et al.*, 2002; Shor *et al.*, 2003b).

[7] Hydrophobicity is quantified by the log octanol-water equilibrium distribution coefficient, log K_{ow} (values are reported in Table I). Log K_{ow} is lower for less hydrophobic PAHs, and higher for more hydrophobic PAHs.

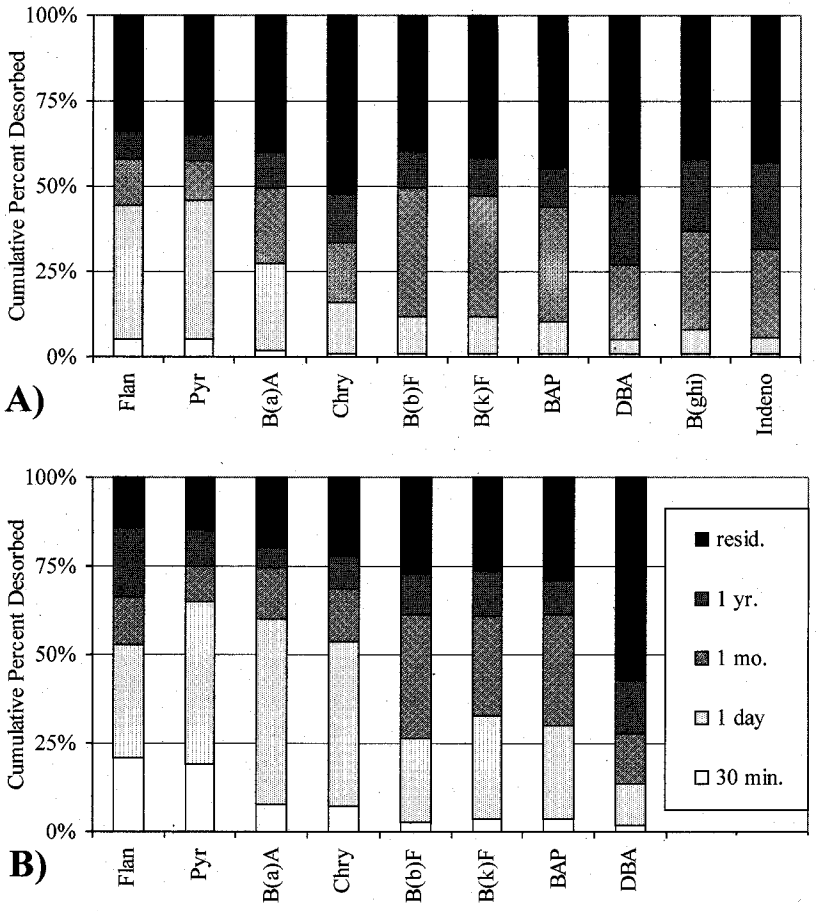

Fig. 1. Cumulative mass desorbed as a percentage of total desorbed plus residual for (A) whole Newtown Creek sediment and (B) whole Piles Creek sediment by 30 minutes (white), 1 day (dots), 1 month (weave), 1 year (tile), and undesorbed residual (black). Abbreviations are Flan—fluoranthene; Pyr—pyrene; B(a)A—benz(a)anthracene; Chry—chrysene; B(b)F—benzo(b)fluroanthene; B(k)F—benzo(k)Fluroanthene; BAP—benzo(a)pyrene; DBA—dibenz(a,h)anthracene; B(ghi)—benzo(g,h,i)perylene; Indeno—indeno(1,2,3-CD)pyrene.

is an independent event in terms of desorption time. Therefore, short biota-sediment contact times occurring repeatedly are the appropriate model for even chronic dermal contact and ingestion exposure scenarios.

4.2. Bioavailability is Inversely Correlated with Toxicity

For both study sediments, smaller, less hydrophobic PAHs are more rapidly and extensively desorbed, while the larger, more hydrophobic PAHs tend to desorb more slowly. Larger PAHs also tend to have a greater nondesorbable residual. The size of this nondesorbable residual fraction of total sediment PAHs is important because this fraction is almost certainly not bioavailable, and therefore probably does not contribute to exposure risk. The compound with the largest residual fraction in both sediments is DBA, and second largest in Piles Creek sediment is BAP. Larger, more hydrophobic compounds—especially BAP and DBA—are also the most carcinogenic. Conversely, less carcinogenic compounds like fluoranthene and pyrene tend to be more rapidly and extensively desorbed. Therefore, the connection between comparatively high toxicity and comparatively low bioavailability was examined further.

PEF values of Nisbet and LaGoy (1992) were regressed on the percentage of mass desorbed for all PAHs individually for each desorption interval for each study sediment (ordinary least squares linear regression with intercept; significance of slope term measured by 1-tailed Student's t-test). For Newtown Creek sediment, the log of PEF was significantly inversely correlated with the percentage desorbed at 30 minutes ($p = 0.012$), 1 day ($p = 0.013$), and 1 year ($p = 0.05$). For Piles Creek sediment, log of PEF was significantly correlated with the percentage desorbed at 1 month ($p = 0.0005$) and at 1 year ($p = 0.0004$). Insignificant correlations ($p > 0.05$) were observed in the remaining cases. The fact that the most toxic compounds also tend to be the least desorbable for our two study sediments has clear and important implications for risk assessment. In the next section, the

combined effects of compound-specific toxicity and bioavailability will be examined in terms of total predicted excess cancer risk.

4.3. Current Guidance May Overpredict and Distort Actual Exposure Risk

The sum of priority PAHs, total carcinogenic PAHs (cPAH), and BAP-equivalent total sediment concentration all indicate that exposure to Newtown Creek sediment would present greater risk than would equivalent exposure to Piles Creek sediment (Table II). However, when desorption kinetics are taken into account, relative risk from exposure to Piles Creek sediment may be twice that of Newtown Creek sediment for exposure scenarios where the typical biota-sediment contact time is 30 minutes. For typical biota-sediment contact times of 1 day, exposure to Piles Creek sediment is still likely to pose greater risk than exposure to Newtown Creek sediment, although the margin is not as large. At long desorption times, the difference in exposure risk between Newtown Creek and Piles Creek sediment becomes less, and are similar to the ratios between total PAH concentrations and total carcinogenic PAH concentrations (Table II).

The magnitude of risk from exposure to these study sediments computed by the RRI approach is very different from risks estimated by other approaches (Table II). For 1-month exposures, the EPA default approach (total BAP-equivalent concentration, as per oral exposure scenario, using EPA provisional PEF values) results in relative risks 2 to 3 times higher than those predicted by the RRI approach. For 1-day exposure to Piles Creek sediment, risks computed by the default approach are 4 times higher than the RRI approach; for 1-day exposure to Newtown Creek sediment, the EPA default approach would estimate risk 13 times higher. For very short-term exposure risk (30 minutes) the default approach might overestimate risks by as much as 31 times in Piles Creek sediment and 159 times in Newtown Creek sediment. Large discrepancies also exist between the RRI approach and the conventional approach using PEF values from Nisbet and LaGoy (1992) (Table II).

4.4. BAP and Two Other PAHs Dominate Total Risk

The percentage contribution to total risk by compound was calculated for biota-sediment contact time of 1 day according to Equation (10) using PEF values after both Nisbet and LaGoy (1992) and U.S. EPA (1993). The results (Fig. 2) show that BAP constitutes nearly 50% or more of total risk for both sediments using both sets of PEF values. Using EPA PEF values, the contribution of BAP to total RRI increases to nearly 70% for Newtown Creek sediment and to 65% for Piles Creek sediment. When the PEF values of Nisbet and LaGoy are used (where $PEF_{DBA} = 5$) DBA constitutes about a quarter of the total risk for both sediments; however, using EPA PEF values (where $PEF_{DBA} = 1$), DBA constitutes only 7% of total risk. Compounds with PEF values of 0.1 (benz(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, and indeno(1,2,3-CD)pyrene) contribute from 2% to 20% to total risk depending on the set of PEF values used. The relatively large desorbable mass of benz(a)anthracene makes it the largest contributor to risk of compounds with a PEF of 0.1. Assuming EPA PEF values, benz(a)anthracene contributes 20% to total risk in Piles Creek sediment: 3 times higher than DBA, which has a PEF that is 10 times higher. Together, BAP, DBA, and benz(a)anthracene contribute 87% of total PAH risk in Newtown Creek sediment, and 88% for Piles Creek sediment (Fig. 2).

The 4-ring PAH benz(a)anthracene desorbs rapidly from Piles Creek sediment in the first day, and then continues to desorb more slowly for up to 1 year (Fig. 3). Conversely, almost none of the DBA desorbs from Newtown Creek sediment on the first day and slowly approaches 50%, which is desorbed in 1 year. Because different PAHs desorb by different mechanisms and at dramatically different rates, the relative contribution of various PAHs to total risk will change as a function of time. In general, rapidly desorbing compounds contribute more to risk at short time periods than at long time periods (Tables III and IV). For example, using EPA PEF values, rapidly desorbing benz(a)anthracene contributes 12% to total risk at 1 day, but only 6% at 1 year, while the contribution of the slowly desorbing 5-ring PAH DBA grows from 6% at 1 day to 11% at 1 year.

BAP, which has an intermediate desorption rate compared with the other two dominant risk contributors, is a consistent contributor to total risk. Using Nisbet and LaGoy's PEF values, BAP contributed between 44% and 54% of total RRI in Newtown Creek sediment, and between 48% and 52% of total RRI in Piles Creek sediment for all biota-sediment contact times (between 30 minutes and 1 year) (Table III). Similarly, using EPA PEF values, where the toxic potency of DBA is significantly discounted, BAP contributed between 65% and 73% of total RRI

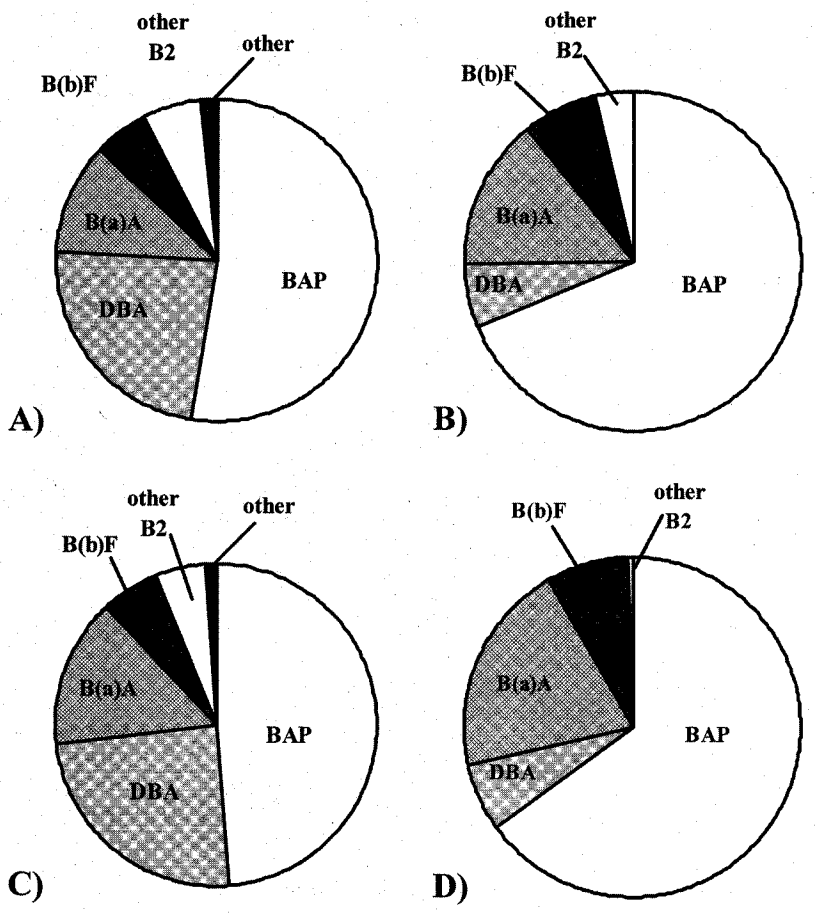

Fig. 2. Percentage contribution of various PAHs to total relative risk index (RRI) for biota-sediment contact time of 1 day computed by Equation (10) for: (A) Newtown Creek sediment using PEF values after Nisbet and LaGoy (1992); (B) Newtown Creek sediment using PEF values after EPA (1993); (C) Pile Creek sediment using PEF values after Nisbet and LaGoy; and (D) Piles Creek sediment using PEF values after EPA. Abbreviations are: B(a)a -benz(a)anthracene; B(b)k-benzo(k)fluoranthene; BAP-benzo(a)pyrene; DBA-dibenz(a,h)anthracene; "other B2" are the sum of chrysene, benzo(k)fluoranthene, and indeno(1,2,3-CD)pyrene; "other" are all other class-D priority PAHs included in the study.

in Newtown Creek sediments, and between 65% and 70% of RRI in Piles Creek sediments (Table IV). These results suggest that BAP may be useful as a surrogate compound to evaluate *relative* risk from exposure to mixtures of PAHs in different sediments.

4.5. Application of RRI Approach to Compare Risk Among Sediment Fractions

Organic matter-rich low-density sediment fractions can be greatly enriched in PAH content; therefore, removal of the low-density fractions may be a viable contaminated sediment management strategy (Ghosh *et al.*, 2000; Rockne *et al.*, 2002). For example, in Piles Creek sediment, the low-density fraction contains 85% of all PAHs but constitutes only 4% of total sediment mass. However, the PAHs in the low-density fractions desorb more slowly, and to a lesser extent, compared with PAHs in whole or high-density fractions (Ghosh *et al.*, 2000; Rockne *et al.*, 2002). Furthermore, the low-density fraction has been shown to limit the rate of release of PAHs from whole sediments (Rockne *et al.*, 2002). Therefore, it is unclear if removal of the low-density portion would be beneficial or detrimental to achieving overall reduction in risk from exposure to contaminated sediments.

One possible means of readily establishing relative risk among sediments and sediment fractions may be to simply evaluate and compare the cumulative desorption of BAP. This analysis was done for various biota-sediment contact times from whole and fractionated Newtown Creek and Piles Creek sediments. The results suggest that removal of the PAH-enriched low-density fractions would not diminish exposure risk (Table V). The RRI for BAP at 1 day is 0.31 for whole sediment, and 0.32 for high-density sediment. (Given that the density fractionation process is a binary separation, the high-density fraction is simply whole sediment with the low-density portion removed.) In Piles Creek sediment, the RRI for the high-density fraction is about 3 times *higher* than whole sediment (0.94 vs 0.37 for high-density and whole Piles Creek sediment. Therefore, while removal of the low-density portion may be a valid

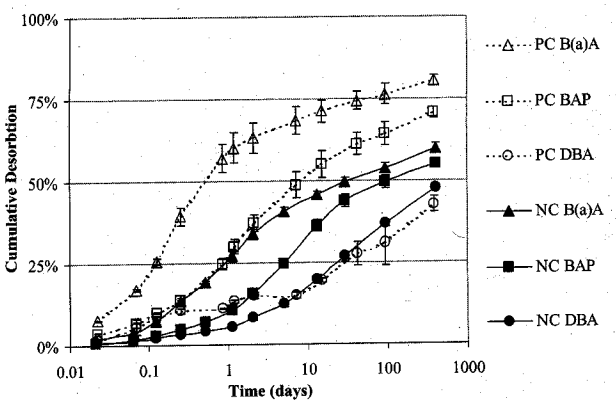

Fig. 3. Comparison of cumulative mass desorbed as a percentage of total extractable concentration for Piles Creek sediment (open markers) or Newtown Creek sediment (solid markers) for the compounds most influencing total relative risk index. Abbreviations are: PC—Piles Creek; NC—Newtown Creek; B(a)A—benz(a)anthracene (triangles); BAP—benzo(a)pyrene (squares); and DBA—dibenz(a,h)anthracene (circles). Average and range of duplicates. Note logarithmic time scale.

Table III. Percentage Contribution by Compound to Excess Cancer Risk, as Computed by Equation (10), Using PEF Values After Nisbet and LaGoy (1992), for Four Biota-Sediment Contact Times

	30 Minutes	1 Day	1 Month	1 Year
Newtown Creek				
Fluoranthene	0.76	0.59	0.19	0.15
Pyrene	0.73	0.56	0.17	0.14
Benz(a)anthracene	8.1	11	4.9	4.2
Chrysene	0.37	0.57	0.29	0.29
Benzo(b)fluroanthene	4.3	5.6	5.7	4.9
Benzo(k)fluroanthene	1.9	2.7	2.6	2.3
Benzo(a)pyrene	44	53	54	47
Dibenz(a,h)anthracene	35	23	29	36
Benzo(g,h,i)perylene	0.45	0.39	0.44	0.49
Indeno(1,2,3-CD) pyrene	3.8	2.4	3.3	4.2
Total	100.00	100.00	100.00	100.00
Piles Creek				
Fluoranthene	0.69	0.21	0.14	0.15
Pyrene	1.0	0.43	0.26	0.24
Benz(a)anthracene	16	15	9.8	8.4
Chrysene	2.2	1.9	1.3	1.2
Benzo(b)fluroanthene	5.2	5.7	6.9	6.7
Benzo(k)fluroanthene	2.8	3.2	3.2	3.1
Benzo(a)pyrene	49	49	52	48
Dibenz(a,h)anthracene	24	25	27	32
Total	100.00	100.00	100.00	100.00

strategy to meet EPA sediment contaminant concentration limits, it may not be a prudent strategy in terms of risk management. These results further emphasize the need to move away from soil and sediment concentration limits in environmental regulations to an integrative approach including mass transport, bioavailability, and prediction of risk.

Table IV. Percentage Contribution by Compound to Excess Cancer Risk, as Computed by Equation (10), Using PEF Values After U.S. EPA (1993), for Four Biota-Sediment Contact Times

	30 Minutes	1 Day	1 Month	1 Year
Newtown Creek				
Benz(a)anthracene	11.9	15	6.7	6.1
Chrysene	0.05	0.07	0.04	0.04
Benzo(b)fluroanthene	6.3	7.3	7.8	7.2
Benzo(k)fluroanthene	0.3	0.4	0.4	0.3
Benzo(a)pyrene	65	69	73	70
Dibenz(a,h)anthracene	10	6	8	11
Indeno(1,2,3-CD) pyrene	5.6	3.1	4.5	6.1
Total	100.0	100.0	100.0	100.0
Piles Creek				
Benz(a)anthracene	21	20	13	12.1
Chrysene	0.3	0.3	0.2	0.2
Benzo(b)fluroanthene	7.0	7.6	9.3	9.6
Benzo(k)fluroanthene	0.4	0.4	0.4	0.4
Benzo(a)pyrene	65	65	70	68
Dibenz(a,h)anthracene	6.3	6.6	7.1	9.3
Total	100.0	100.0	100.0	100.0

Table V. Relative Risk Among Sediment Density (ρ) Fractions: Comparison of RRI for BAP

	Whole	High-ρ	Low-ρ
Newtown Creek			
30 minutes	0.02	0.03	0.06
1 day	0.31	0.32	0.38
1 month	1.27	0.79	1.47
Piles Creek			
30 minutes	0.05	0.14	0.98
1 day	0.37	0.94	3.34
1 month	0.76	1.41	nd

nd: not determined.

5. CONCLUSIONS

The EPA has estimated that 10% of the sediments underlying U.S. surface waters are "sufficiently contaminated with toxic pollutants to pose potential risks to humans" (U.S. EPA, 1998). Since all contaminated sediment cannot possibly be treated, more accurate ways of predicting relative risk are urgently needed. In this study, consideration for the combined effects of compound-specific bioavailability and toxicity resulted in risk estimates up to 2 orders of magnitude lower than those calculated using EPA default parameters. Results from the current EPA approach also differed from this approach in gauging the relative

contribution of various compounds to total risk, and the relative magnitude of risk among contaminated study sediments and various sediment fractions. Studies that attempt to integrate mass transport and toxicity into exposure assessment may enhance the accuracy of risk prediction. Additional studies probing the interrelationships between mass transport, bioavailability, and toxicity in other systems are needed.

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