

# Parent and Halogenated Polycyclic Aromatic Hydrocarbons in Seafood from South China and Implications for Human Exposure

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**ABSTRACT:** This work extends previous studies on the occurrence of persistent halogenated compounds in consumer seafood from South China. Residual levels of 16 U.S. EPA priority polycyclic aromatic hydrocarbons (PAHs) and 9 halogenated PAHs (HPAHs) were determined in three kinds of seafood products collected from 11 coastal cities in South China from June to October 2005. The results indicated that PAH components were low but detectable in a large number of seafood samples under investigation. The benzo(*a*)pyrene (BaP)-like TEQ concentrations of HPAHs were higher than those of PAHs for all three kinds of seafood. The relative contributions of each seafood group to the total estimated daily intake of PAHs and HPAHs were also analyzed. Shellfish contributed the most to the total exposure for all subgroups, followed by shrimp. Overall, the excess cancer risks (ECRs) induced by HPAHs were much greater than the risks posed by PAHs. Both ECRs for PAHs and HPAHs were far below  $10^{-4}$ , showing no significant cancer risk via seafood consumption for people in South China. Sensitivity analysis results show the oral cancer slope factor of BaP is the most influential variable that contributed most to the total variance of risk for all subgroups.

**KEYWORDS:** polycyclic aromatic hydrocarbons, halogenated polycyclic aromatic hydrocarbons, seafood, health risk assessment, toxic equivalency quotient

## INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs) result from both natural processes, such as volcanic eruptions and diagenesis,<sup>1</sup> and human activities, such as incomplete combustion or pyrolysis of carbonaceous materials.<sup>2,3</sup> They were recognized as a carcinogenic class of compounds in the late 1920s. As derivatives of PAHs, halogenated PAHs (HPAHs), including chlorinated PAHs and brominated PAHs, are a group of compounds composed of PAHs and one or more halogen atoms attached to the aromatic skeleton, which show more toxicity than their corresponding parent PAHs.<sup>4–6</sup>

Due to the ubiquity of PAHs and HPAHs in the environment, people can be exposed to them via various pathways, and a large number of studies on human exposure to PAHs have already been published.<sup>7–12</sup> In contrast, human exposure to HPAHs present in the environment has not been studied extensively to date. Because certain HPAHs have a greater toxicity than their corresponding parent PAHs,<sup>4–6</sup> HPAHs in the environment and the implications for human exposure deserve our concern. For PAHs, food has been documented as a major route of exposure for nonsmokers and nonoccupationally exposed populations.<sup>7–11</sup> As for HPAHs, the contribution of dietary ingestion is not yet clear due to a lack of information on the subject. A previous study discussed PAHs and HPAHs in rice and the consequential human health implications in China,<sup>13</sup> but these contaminants in seafood have not been addressed adequately so far, to our knowledge. Because large quantities of seafood products are produced in and exported from China's Pearl River Delta, the dietary intake of organic pollutants via seafood consumption should be a concern not only in South China but also throughout the globe.

The present study aimed to survey a large number of seafood products from the coastal region of southern China for parent and halogenated PAHs contamination. The data acquired enabled us to perform risk assessments related to the consumption of seafood products and to identify potential health risks, extending our previous studies on the occurrence of persistent halogenated compounds in consumer seafood from South China.<sup>14–16</sup> To assess the human intake of parent and halogenated PAHs via seafood consumption under worst-case scenarios, we selected samples with high levels of persistent halogenated compounds as cited in previous studies,<sup>14–16</sup> which we discuss in the following sections.

## MATERIALS AND METHODS

**Chemicals and Materials.** Internal standards (2-fluorobiphenyl and *p*-terphenyl-*d*<sub>14</sub>) and surrogate standards (acenaphthene-*d*<sub>10</sub>, phenanthrene-*d*<sub>10</sub>, chrysene-*d*<sub>12</sub>, and perylene-*d*<sub>12</sub>) were purchased from Dr. Ehrenstorfer GmbH (Augsburg, Germany). A standard solution of 16 U.S. EPA priority PAHs was purchased from Chem Service (West Chester, PA, USA). The 16 PAHs included in the present study were naphthalene (Nap), acenaphthylene (Acy), acenaphthene (Ace), fluorine (Flu), phenanthrene (Phe), anthracene (Ant), fluoanthene (Flu), pyrene (Pyr), benzo[*a*]anthracene (BaA), chrysene (Chr), benzo[*b*]fluoranthene (BbF), benzo[*k*]fluoranthene (BkF), benzo[*a*]pyrene (BaP), indeno[1,2,3-*cd*]pyrene (IcdP), dibenzo[*a,h*]anthracene (DahA), and benzo[*g,h,i*]perylene (BghiP). The sum of these 16 PAHs is designated  $\Sigma_{16}$ PAH. 9-Chlorophenan-

Received: November 13, 2012

Revised: January 31, 2013

Accepted: February 4, 2013

Published: February 4, 2013

Table 1. Detection Rates (DR, %) and Concentrations (ng/g ww) of PAHs and HPAHs in Seafood Products from South China

	crab ( <i>n</i> = 29)			shrimp ( <i>n</i> = 43)			shellfish ( <i>n</i> = 66)		
	DR	mean	range	DR	mean	range	DR	mean	range
Nap	0	<RL <sup>a</sup>	<RL	0	<RL	<RL	0	<RL	<RL
Acy	76	0.20	<RL–0.43	72	0.20	<RL–0.54	82	0.27	<RL–0.87
Ace	55	0.19	<RL–0.97	47	0.14	<RL–0.47	76	0.22	<RL–0.59
Fle	90	0.55	<RL–1.44	86	0.58	<RL–1.52	97	0.86	<RL–1.99
Phe	72	1.92	<RL–4.32	95	1.97	<RL–4.37	92	3.35	<RL–9.02
Ant	59	0.31	<RL–2.14	44	0.24	<RL–3.67	76	0.25	<RL–2.02
Flu	66	0.52	<RL–1.04	86	0.52	<RL–1.33	98	1.67	<RL–7.45
Pyr	79	0.30	<RL–0.79	70	0.34	<RL–0.89	94	1.03	<RL–3.74
BaA	38	<RL	<RL	0	<RL	<RL	48	0.17	<RL–0.96
Chr	3	<RL	<RL–0.19	2	<RL	<RL–0.14	58	0.35	<RL–1.86
BbF	31	<RL	<RL–0.13	2	<RL	<RL–0.22	47	0.20	<RL–1.02
BkF	21	<RL	<RL–0.14	2	<RL	<RL–0.21	24	<RL	<RL–1.16
BaP	7	<RL	<RL–0.40	0	<RL	<RL	26	<RL	<RL–0.84
IcdP	14	<RL	<RL	0	<RL	<RL	0	<RL	<RL
DahA	0	<RL	<RL	0	<RL	<RL	0	<RL	<RL
BghiP	3	<RL	<RL	0	<RL	<RL	3	<RL	<RL
Σ <sub>16</sub> PAH		4.13	<RL–12.5		4.09	<RL–13.6		8.63	<RL–32.0
2-BrFle	100	1.10	0.96–1.70	100	1.15	0.97–2.26	100	1.26	0.96–2.43
9-BrPhe	76	0.54	<RL–1.94	81	0.57	<RL–1.60	94	1.13	<RL–8.6
9-BrAnt	100	1.25	0.38–9.01	100	0.96	0.38–2.19	100	1.72	0.37–8.02
9,10-Br <sub>2</sub> Ant	100	1.01	0.89–2.71	100	1.06	0.89–3.31	100	1.19	0.89–3.92
1-BrPyr	100	1.97	0.36–22.1	100	2.41	0.34–42.9	100	2.50	0.37–18.2
7-BrBaA	100	1.35	0.81–2.44	100	1.58	0.81–6.73	100	1.40	0.80–4.80
9-ClPhe	100	1.50	0.74–15.4	100	0.96	1.75–2.19	100	1.16	0.75–2.35
2-ClAnt	100	1.21	0.90–4.59	100	1.12	0.91–1.99	100	1.44	0.90–9.50
9,10-Cl <sub>2</sub> Ant	83	1.67	<RL–3.62	93	1.79	<RL–3.33	91	1.77	<RL–6.34
Σ <sub>9</sub> HPAH		1.35	0.81–2.44		1.58	0.81–6.73		1.39	0.79–4.76

<sup>a</sup><RL, lower than the reporting limit (0.125 ng/g wet weight).

threne (9-ClPhe), 2-chloroanthracene (2-ClAnt), and 9,10-dichloroanthracene (9,10-Cl<sub>2</sub>Ant) were purchased from Aldrich (St. Louis, MO, USA). 1-Bromopyrene (1-BrPyr), 2-bromofluorene (2-BrFle), 9-bromophenanthrene (9-BrPhe), 9-bromoanthracene (9-BrAnt), and 9,10-dibromoanthracene (9,10-Br<sub>2</sub>Ant) were obtained from Acros Organics (Geel, Belgium). 7-Bromobenz(a)anthracene (7-BrBaA) was purchased from Tokyo Kasei Kogyo Co., Ltd. (Tokyo, Japan). The sum of these nine HPAHs is designated Σ<sub>9</sub>HPAH. All solvents were redistilled using a glass system. SX-3 Bio-Beads used in a gel permeation chromatograph were purchased from Bio-Rad Laboratories (Hercules, CA, USA). Neutral silica gel (80–100 mesh) was Soxhlet extracted with methanol and methylene chloride for 48 h and was activated at 180 °C for 12 h and deactivated with distilled water (3%, w/w) prior to use. Sodium sulfate was baked at 450 °C and stored in sealed containers. All glassware was hand-washed with detergent and tap water, rinsed with deionized water, and baked at 450 °C for at least 4 h before use.

**Sample Collection and Pretreatment.** Seafood samples were collected on the basis of a market basket-based survey. A total of 138 samples, representing 6 species of shrimps, 2 species of crabs, and 10 species of shellfish commonly consumed by local residents, were randomly collected from local fishery markets in 11 coastal cities of Guangdong Province, South China, from June to October 2005. Upon collection, samples were stored in polyethylene bags, kept on ice, and brought back to the laboratory immediately. They were stored at –20 °C until analyzed. More information on the sampling process was detailed in the previous study.<sup>14</sup>

Frozen samples were thawed and rinsed individually with purified water to remove possible impurities. Approximately 20 g (wet weight, ww) of muscle or soft tissue was taken and homogenized. Upon freeze-drying for 48 h, all samples were ground into powders and stored at –20 °C until chemical analysis was performed. After being spiked with the surrogate standards, each sample was Soxhlet extracted

with an acetone and hexane mixture (1:1 in volume) for 48 h. The extract was first subjected to a gel permeation chromatograph based on a 50 cm × 2.5 cm i.d. glass column packed with 40 g of Bio-Beads SX-3 for lipid removal and was then subjected to chromatographic separation on a glass column (45 cm × 1.0 cm i.d.) packed with alumina–silica gel (6:12, v/v) and 2 cm anhydrous sodium sulfate on the top. The fraction containing parent and halogenated PAHs was eluted with 70 mL of a mixture of hexane and methylene chloride (7:3, v/v) and was then concentrated to 0.5 mL. Internal standards were added to each extract prior to the instrumental analysis.

**Instrumental Analysis and Quality Control.** Concentrations of PAHs and HPAHs were determined by GC-MS (Agilent 7890A GC equipped with 5975C MSD; Agilent Technologies, Foster City, CA, USA) with splitless injection. Gas chromatographic separation was accomplished using a 30 m DB-5MS fused silica capillary column (0.25 mm i.d. and 0.25 μm film thickness (J&W Scientific, Folsom, CA)). The column oven temperature was initially programmed from 60 to 200 °C at a rate of 10 °C/min, to 214 °C at a rate of 2 °C/min, to 254 °C at a rate of 5 °C/min (held for 2 min), and finally to 290 °C at a rate of 18 °C/min (held for 17 min). The mass selective detector was operated in the selected ion monitoring. Mass spectra were acquired in the electron impact mode with an impact voltage of 70 eV. Finally, data acquisition and processing were performed with Agilent ChemStation system.

Quantification of PAHs and HPAHs was carried out with an internal calibration procedure. The lowest concentration of the calibration standards was chosen as the reporting limit for the target analytes (0.125 ng/g wet weight (ww) for a 20 g sample). Samples of procedural blanks and spiked blanks were processed with each batch of samples. Recoveries of the surrogate standards, naphthalene-*d*<sub>8</sub>, acenaphthene-*d*<sub>10</sub>, phenanthrene-*d*<sub>10</sub>, chrysene-*d*<sub>12</sub>, and perylene-*d*<sub>12</sub>, from all procedural blanks and spiked blanks were 60 ± 11, 81 ± 12, 83 ± 10, 75 ± 5, and 73 ± 8%, respectively. The quality control

standards for PAHs and HPAHs were analyzed for each 10 samples to monitor the instrumental stability. After deducting a blank value, the concentrations of PAHs and HPAHs in the current study were presented on a wet weight basis. The reported concentrations were not corrected with the recoveries of the surrogate standards.

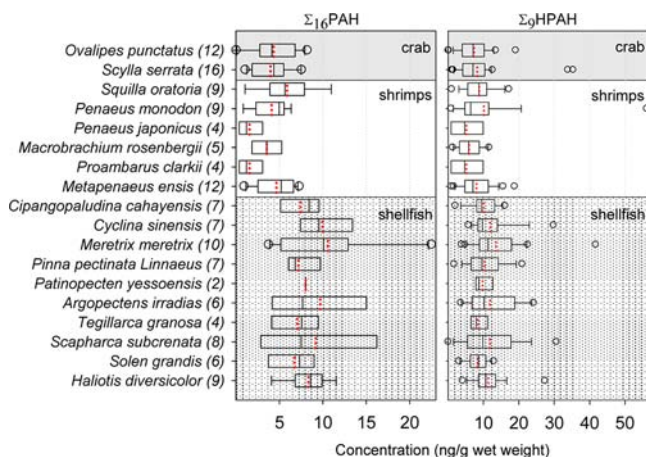
**Exposure Assessment and Data Analysis.** Dietary exposure was estimated on the basis of the results from our dietary survey conducted in April 2006, and the concentration values obtained from the present study. For samples with an analyte concentration below the reporting limit, half the reporting limit was used for the estimation. The estimated daily intake (EDI) was calculated as  $EDI (ng/kg\ bw/day) = \text{seafood consumption (g/day)} \times \text{target compound concentration (ng/g/body weight (kg))}$ . Additionally, the average body weights of different age groups were derived from a previous study.<sup>14</sup>

Because the toxic equivalency quotient (TEQ) can be regarded as a better index for the potent toxicity than the concentration, we also calculated the TEQ of PAHs and HPAHs; the calculation of TEQ was based on a previously reported protocol.<sup>13</sup> Briefly, the TEQ of PAH can be obtained by the concentration of an individual PAH, multiplying the toxic equivalency factors of the corresponding PAH relative to BaP.<sup>17</sup> For the TEQ of HPAHs, the calculation is the same, but the toxic equivalency factors are replaced with the toxic potency values of HPAHs relative to BaP.<sup>5</sup>

In addition, the excess cancer risk (ECR) induced by dietary exposure to PAHs and HPAHs via seafood consumption was also assessed using the procedure detailed in a previous study.<sup>13</sup> An average lifespan of 70 years is assumed. The oral cancer slope factor of BaP was assumed as  $7.3 (mg/kg/day)^{-1}$  by the integrated risk information system of the U.S. EPA.<sup>18</sup>

## RESULTS AND DISCUSSION

**PAHs and HPAHs in Seafood.** The concentrations of PAHs and HPAHs in consumer seafood from South China are summarized in Table 1 and Figure 1. According to the seafood



**Figure 1.** Comparison of  $\Sigma_{16}$ PAH and  $\Sigma_9$ HPAH concentrations in crab, shrimp, and shellfish. (Dotted lines in boxes represent the arithmetic mean concentrations. Numbers of samples are given in parentheses.)

categories, the highest level (mean) of  $\Sigma_{16}$ PAH was detected in shellfish (8.63 ng/g ww), followed by crab (4.13 ng/g ww) and shrimp (4.09 ng/g ww). For  $\Sigma_9$ HPAH, the decrease sequence was shrimp (1.58 ng/g ww) > shellfish (1.39 ng/g ww) > crab (1.35 ng/g ww). Actually, the levels of  $\Sigma_{16}$ PAH and  $\Sigma_9$ HPAH in the three kinds of seafood were comparable. With the exception of Nap (after the deduction of a high blank value, Nap was deemed as zero in the present study), the other low molecular weight PAHs have higher detection rates than those of high molecular weights PAHs (Table 1). For example, the detection

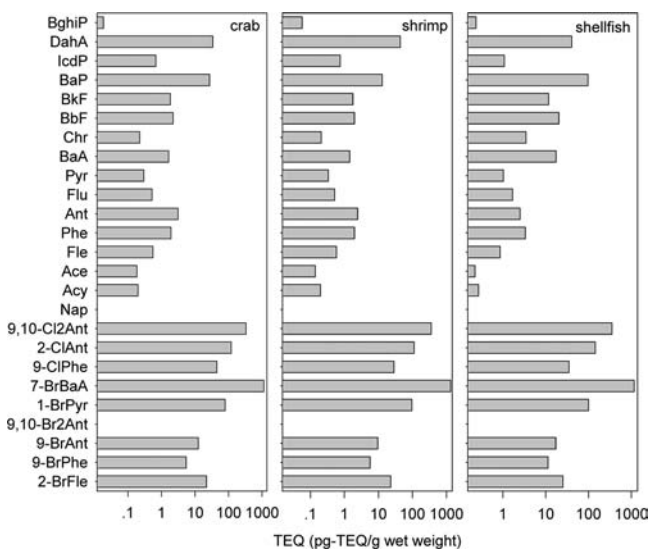
rates of Acy, Ace, Fle, Phe, Ant, Flu, and Pyr in the three varieties of seafood were all near or above 50%. This can be attributed to high molecular weight PAHs making the bioconcentration more difficult. For HPAHs, with the exception 9-BrFle and 9,10-Cl<sub>2</sub>Ant, detection rates of the other HPAHs all reached 100%. This indicates that HPAHs were more easily bioconcentrated by the crab, shrimp, and shellfish than PAHs. Overall, the parent and halogenated PAHs in shellfish were slightly higher than those in the crab and shrimp. We postulated that the difference in the levels of PAHs and HPAHs between species is likely due to the different ecological characteristics of different species, such as feeding habits and habitats. For example, in comparison to crab and shrimp, shellfish lives close the surface sediment, which acts as an ultimate sink for organic pollutants brought into the aquatic environment.<sup>19</sup> Again, the filter-feeding behavior of shellfish might also affect the bioaccumulation of PAHs and HPAHs. In addition, because the seafood samples were collected randomly from local markets, the different biological properties, such as age, gender, weight, and lipid content of the individual samples, as well as various environmental factors, including the proximity to local PAHs and HPAHs sources, may all have potentially influenced the levels of these compounds in individual samples.

Polycyclic aromatic hydrocarbons result from both natural processes and human activities. The halogenation of PAHs to form the corresponding HPAHs is a well-known reaction.<sup>20</sup> Currently, automobile emissions and waste incineration are considered the two major emission sources of HPAHs in the environment.<sup>5,21,22</sup> These pollutants can enter aquatic environments in a number of ways. Sediments act as an ultimate sink for organic pollutants brought into the aquatic environment from direct discharges, surface runoff, and atmospheric dry and wet deposition. The crab, shrimp, and shellfish are scavengers in benthic environments; therefore, contaminated aquatic environments, such as surface sediments, may be major sources of target compounds in this seafood. However, because the seafood samples were collected randomly from local markets, source identification of target compounds is difficult to carry out.

The contents of  $\Sigma_{16}$ PAH in three kinds seafood from South China were comparable to or slightly higher than those in Catalonia, Spain (2.864 ng/g fresh weight in fish and shellfish).<sup>23</sup> The concentrations of individual PAH congeners in shrimp and crab from areas in Mississippi affected by the Deepwater Horizon oil spill were higher than those in shrimp and crab from South China;<sup>24</sup> the severe pollution of seafood in Mississippi can be attributed to the oil spill. In England,<sup>25</sup> the concentration of 18 PAHs in commercial shrimp (36–8930 ng/g ww) was also higher than concentrations in shrimp from South China (<RL–13.6 ng/g ww). Crabs caught from Lake Timsah (Egypt) contained significantly higher concentrations of 13 PAHs (1319–3767 ng/g; dry weight or wet weight was not specified in this study).<sup>26</sup> These values were 3 orders of magnitude higher than those in crabs collected from South China. Again, shellfish along the coast of the Gulf of Naples with the average concentration of 217 ng/g ww<sup>27</sup> was also beyond our measured result (8.63 ng/g ww in shellfish). The size of the shellfish is also an important factor affecting the concentrations of PAHs; however, these data were not available. Therefore, the size of the shellfish was not taken into account for this comparison. To our knowledge, no study

on HPAHs in seafood has been published to date; therefore, no comparison was conducted in the present study.

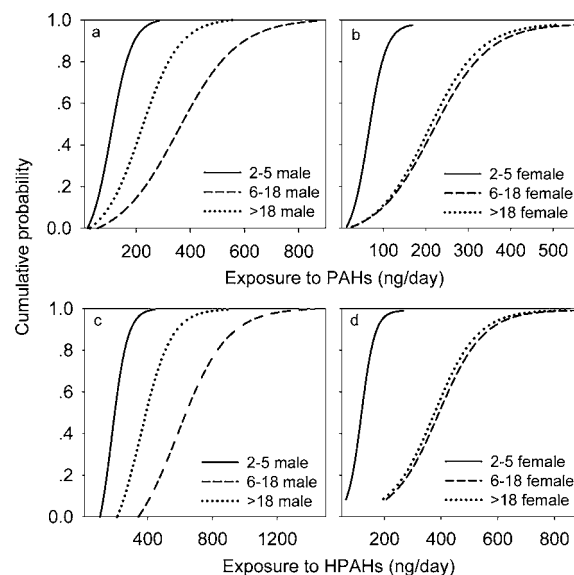
The TEQ could be a better index than concentration for determining potent toxicity.<sup>13</sup> The BaP-like TEQs for individual PAHs and HPAHs were calculated and are shown in Figure 2. The calculated mean TEQs of  $\Sigma_{16}$ PAHs were 74.5,



**Figure 2.** Toxic equivalency quotient relative to BaP of PAHs and HPAHs in seafood from South China.

69.2, and 201 pg TEQ/g ww for crab, shrimp, and shellfish, respectively. DahA and BaP were the major contributors to the total TEQ of  $\Sigma_{16}$ PAHs for all three kinds of seafood, although the levels of all three are below the reporting limit. The TEQs of HPAHs (2-BrFle, 9-BrPhe, 9-BrAnt, 7-BrBaA, 9-CIPhe, 2-ClAnt, 1-BrPyr, and 9,10-Cl<sub>2</sub>Ant,  $\Sigma_8$ HPAH) were calculated on the basis of the toxic potency value of HPAH relative to BaP except 9,10-Br<sub>2</sub>Ant, for which the toxic potency value relative to BaP was not available (Figure 2). The total TEQ concentrations of  $\Sigma_8$ HPAH were 1751, 1961, and 1858 pg TEQ/g ww for crab, shrimp, and shellfish, respectively. Among the individual HPAHs, the TEQ of 7-BrBaA accounted for >60% of the TEQ concentrations of  $\Sigma_8$ HPAHs in the three kinds of seafood. Obviously, the concentration profiles of PAHs and HPAHs will be different from their TEQ profiles for all of the seafood samples. Overall, HPAHs have higher TEQ concentrations than PAHs, due to higher concentrations and higher toxicity levels of HPAHs compared to the corresponding parent PAH (Figure 2).

**Daily Intake of PAHs and HPAHs.** The cumulative probability distributions of the EDI of PAHs and HPAHs via the seafood consumption of each subgroup in South China are displayed in Figure 3. For 2–5-, 6–18-, and >18-year-old groups, the average EDI values of  $\Sigma_{16}$ PAH were 21, 69, and 42 ng/day for males and 13, 42, and 39 ng/day for females, respectively. For 2–5-, 6–18-, and >18-year-old groups, the average EDI values of  $\Sigma_8$ HPAH were 48, 150, and 92 ng/day for males and 29, 91, and 85 ng/day for females, respectively. Apparently, the 6–18-year-old group has the highest EDI, whereas the 2–5-year-old group shows the lowest exposure for both males and females (Figure 3). It is notable that the exposure estimations were affected by the grouping situation. More subgroups will improve the accuracy of the exposure estimations.

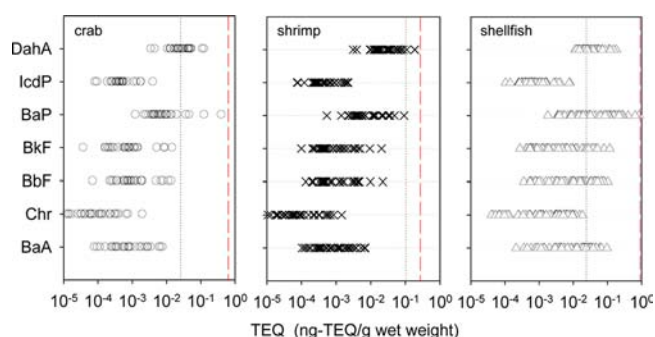


**Figure 3.** Probability distributions of daily exposure to PAHs and HPAHs via seafood consumption for population groups in South China.

The relative contributions of each seafood group toward the total EDI of PAHs and HPAHs were also analyzed. Shellfish contributed the most to the total exposure for all subgroups, followed by shrimp. Crab, shrimp, and shellfish all contributed 0.5–18, 2–37, and 52–98% for PAHs exposure and 3–14, 7–36, and 58–90% HPAHs exposure for males 6–18 years of age. The significantly high contribution from shellfish results from its relatively high consumption rate when compared to the other seafood items, whereas the opposite is true for crab and shrimp.

Seafood is only a small portion of the overall dietary pattern, according to a previous dietary survey in South China.<sup>14</sup> Therefore, the contribution of exposure to PAHs and HPAHs as a result of seafood consumption in comparison to total food exposure proved to be minor. For example, our results were 2–3 orders of magnitude lower than exposure to PAHs via the total diet in the United Kingdom, The Netherlands, Italy, New Zealand, and Spain; in those countries, food exposure to PAHs were all >3000 ng/day.<sup>28–32</sup> More recently, PAHs and HPAHs in rice and human exposure via rice consumption in China have been studied,<sup>13</sup> suggesting that the EDI of PAHs and HPAHs via rice ingestion were also far beyond the EDI related to exposure via seafood ingestion.

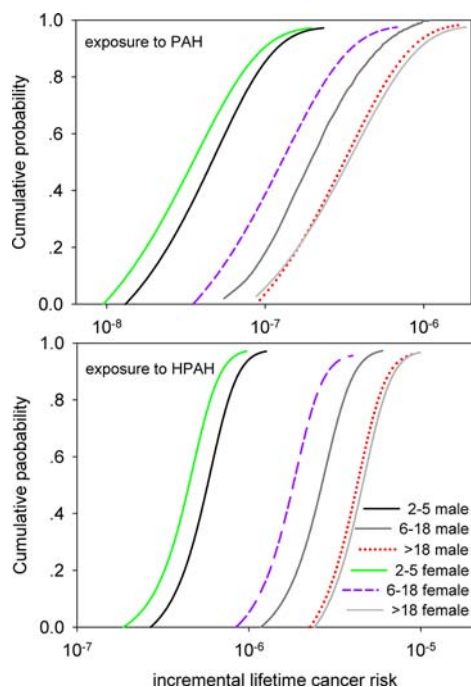
However, compared with the levels of concern (LOCs) for different subgroups estimated with the method detailed in a previous study,<sup>30</sup> the TEQ concentrations of the seven individual carcinogenic PAHs in the three kinds of seafood overlapped with the lowest end of the LOCs (Figure 4). For example, the TEQ concentrations of BaP and DahA in crab were beyond the lowest LOC estimated for carcinogenic PAHs (0.06 ppb TEQ<sub>BaP</sub> for >18-year-old females who consume one seafood meal per day). Similar situations were also observed in shrimp and shellfish. It is notable that the TEQ concentrations of almost all the carcinogenic PAHs present in shellfish, except Chr and IcdP, were beyond the LOC for 6–18-year-old males (0.02 ppb TEQ<sub>BaP</sub>, one seafood meal per day). Assuming that the LOCs of HPAHs are the same as those of PAHs, we found that HPAHs in seafood from South China are all beyond the lowest LOCs estimated for carcinogenic PAHs. Therefore,



**Figure 4.** TEQ concentrations of seven carcinogenic PAH compounds in crab, shrimp, and shellfish. The dotted line and long dash line in each box show the minimum and maximum LOCs for subgroups who consume one seafood meal per day, respectively. LOC values for the other subgroups fall somewhere between them.

whereas human health risks induced by PAHs and HPAHs in seafood should not be ignored, the contribution of exposure via seafood consumption to the total food exposure showed to be minor.

**Health Risk Assessment.** The cumulative probability distributions of the calculated ECRs for subgroups in South China are presented in Figure 5. The median values of ECRs



**Figure 5.** Cumulative probability of incremental lifetime cancer risk induced by PAHs and HPAHs via seafood consumption for population groups in South China.

associated with exposure to  $\Sigma_{16}$ PAH via seafood consumption were estimated to be  $4.2 \times 10^{-8}$ ,  $3.3 \times 10^{-8}$ ,  $2.0 \times 10^{-7}$ ,  $1.4 \times 10^{-7}$ ,  $3.1 \times 10^{-7}$ , and  $3.5 \times 10^{-7}$  for 2–5-year-old males, 2–5-year-old females, 6–18-year-old males, 6–18-year-old females, >18-year-old males, and >18-year-old females, respectively; the above subgroups of ECRs induced by  $\Sigma_9$ HPAH were estimated to be  $5.9 \times 10^{-7}$ ,  $4.6 \times 10^{-7}$ ,  $2.7 \times 10^{-6}$ ,  $1.8 \times 10^{-6}$ ,  $4.1 \times 10^{-6}$ , and  $4.6 \times 10^{-6}$ , respectively. The median values of ECRs induced by  $\Sigma_{16}$ PAH for all subgroups fell in the range of  $10^{-8}$ – $10^{-7}$ , which is lower than the acceptable risk level ( $10^{-6}$ ).<sup>18</sup>

$\Sigma_{16}$ PAH ECR values at the 97th, 99th, 92th, and 90th percentiles for 6–18-year-old males, 6–18-year-old females, >18-year-old females, and >18-year-old males were  $>10^{-6}$ , indicating high potential carcinogenic risk. For 2–5-year-old children, ECRs induced by  $\Sigma_{16}$ PAH were far below the acceptable risk level ( $10^{-6}$ ). For  $\Sigma_9$ HPAH, the median values of ECRs for 2–5-year-old males and females were below the acceptable risk level ( $10^{-6}$ ); however, the other four population groups were higher than the acceptable risk level ( $10^{-6}$ ), but lower than the priority risk level ( $10^{-4}$ ).<sup>31</sup> The ECR values (for  $\Sigma_9$ HPAH) at the 92th and 99th percentiles for 2–5-year-old males and females, respectively, and those at the zeroth percentile for the other four subgroups were all  $>10^{-6}$ , indicating a high potential carcinogenic risk (Figure 5). Overall, the ECRs induced by HPAHs were much greater than those induced by PAHs. This can be attributed to higher levels and higher toxicities of HPAHs than PAHs. However, for all population groups, both ECRs for PAHs and HPAHs were far below  $10^{-4}$ , showing no significant cancer risk related to seafood consumption for people in South China.

Studies regarding dietary health risk assessments for PAHs, especially for HPAHs, via seafood consumption are rather limited. Here we compare our results with other studies that conducted dietary health risk assessments for PAHs. It is not strange that the highest ECR value in the present study ( $4.6 \times 10^{-6}$  for >18-year-old females induced by HPAHs via seafood consumption) is comparable with the lowest ECR values in dietary health risk assessments for PAHs in other studies.<sup>8,13,32</sup> This can be attributed to the fact that the portion of seafood in the dietary pattern is small.

To evaluate the impact of the variability and uncertainty of parameters on the estimation of ECR, a quantitative sensitivity analysis was conducted with Soft Crystal Ball 11. Our results indicate the oral cancer slope factor of BaP had the greatest contribution (>55%) to the total variance of the risk for all of the subgroups, followed by TEQ concentrations in seafood items, especially shellfish. The contribution to the total variance from body weight and intake rates were only <15 and <3%, respectively. This preliminary study found the most influential factor to be the oral cancer slope factor of BaP, upon which further studies should be conducted to improve the accuracy of risk assessment.

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### Funding

This work was supported by the National Natural Science Foundation of China (No. 41071303 and 40973087).

### Notes

The authors declare no competing financial interest.

## ACKNOWLEDGMENTS

We thank the four anonymous reviewers for their valuable comments on the manuscript.

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