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Concentrations and congener profiles of polybrominated diphenyl ethers (PBDEs) in blood plasma from Hong Kong: Implications for sources and exposure route

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HIGHLIGHTS

- Concentrations of $\sum$PBDEs ranged from 0.56 to 92 ng g^{-1}, lipid weight.
- Fish consumption was more important than dust for Hong Kong people exposure to PBDE.
- There is a spatial distribution and terrestrial source of BDE-28 for Hong Kong people.
- There was no significant correlation among PBDE congeners and MeO-BDEs, OH-BDEs or BRPs.

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ABSTRACT

There was limited information about bioaccumulation of polybrominated diphenyl ethers (PBDEs) in humans of the general population of Hong Kong. Therefore, the present study was conducted to determine concentrations and congener profiles of PBDEs in blood plasma from Hong Kong, evaluate their sources and correlations with other organobrominated compounds, and investigate exposure routes from fish and dust. Concentrations of $\sum$PBDE$_{22}$ ranged from 0.56 to 92 ng g^{-1}, lipid weight (lw), with a median of 5.4 ng g^{-1}. BDE-47 was the dominant congener, accounting for 26% of $\sum$PBDE$_{22}$. Concentrations of PBDE congeners in market fish were significantly ($r^2 = 0.89$, $p < 0.001$) correlated with plasma. Positive but no significant correlations were observed, between concentrations of PBDE congeners in indoor dust from workplaces ($r^2 = 0.46$, $p = 0.081$) and homes ($r^2 = 0.49$, $p = 0.10$), with concentrations of PBDE in human blood plasma. The results indicated that dietary exposure, particularly consumption of fish, is a major pathway through which people in Hong Kong are exposed to PBDEs. Furthermore, our data revealed a spatial distribution and terrestrial source of BDE-28 for local people. Results of the present study, which was the first systematic study to investigate concentrations of PBDEs in blood of Hong Kong people, provides useful information to which future measurements can be compared.

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1. Introduction

Polybrominated diphenyl ethers (PBDEs) are a class of brominated flame retardants (BFRs) widely used in a variety of consumer products such as polyurethane foams, textiles, and electric appliances [1]. They are persistent and degrade slowly in the environment and are therefore ubiquitous in both biotic and abiotic environments all over the world [2]. PBDEs have been reported to be toxic to the animals, and have the potential to cause adverse effects on humans. The time trend study of Unites States residents revealed that there have been increasing concentrations of PBDE in blood serum of humans from the mid-1980s [3]. Due to the extensive use and relatively great potential for bioconcentration and biomagnification, PBDEs now have been detected in blood of humans from all over the world including the United States [4], Canada [5], the United Kingdom [6], and Japan [7].

Humans are exposed to PBDEs through their diet, inhalation and incidental ingestion of dust. Previous studies have revealed that concentrations of PBDEs in blood serum were positively correlated with consumption of fish, which contributed approximately 40–50% of the total dietary intake of PBDE [8]. Our recent data revealed that concentrations of PBDEs and their MeO–/OH–structural analogs were relative great in Hong Kong market fish [9], thus it is reasonable to assume that PBDEs could be accumulated into Hong Kong residents via consumption of fish. Furthermore, our previous study indicated that indoor dust collected from workplaces or homes in Hong Kong were greater than those in developed counties such as Australia [10]. The risk assessment indicated that the non-dietary ingestion of dust ($101–404\,ng\,d^{-1}$) was the dominant pathway for children PBDE daily intake [10]. All this evidence suggested that Hong Kong residents might be exposed toxicologically relevant concentrations of PBDEs.

There was very limited information about bioaccumulation of PBDE into the general population of humans of Hong Kong. The only study of PBDEs in blood of persons from Hong Kong revealed that concentrations of PBDE were significantly greater in smokers than nonsmokers [11]. All samples in this study were collected from one blood donor center. Most of the donors may live or work near the center, and the study group was not a random sample of the population. Recent results indicated that $\sum$PBDE ($3.4\,ng\,g^{-1}$ fat) in human breast milk collected from Hong Kong were comparable to the international median levels of the 15 other countries participating in the 2002–2003 WHO exposure study [12]. However, milk can only be obtained from the female population and only during lactation. Compared to other human samples, blood is more easily collected and can cover a wide range of ages of both genders. Therefore, blood is considered to be a good matrix in which to assess concentrations of pollutants in the general population. Our recent study reported that hydroxylated (OH–) and methoxylated (MeO–) PBDEs and bromophenols (BRPs) can be detected in blood plasma of humans in Hong Kong [13].

In the current study, 117 blood plasma samples were collected by the Red Cross in Hong Kong, the world's most densely populated coastal city. Concentrations of PBDE in blood plasma of the general population were assessed. Specifically, the objectives of the present study were to: (1) indentify concentrations of PBDEs in blood plasma samples representative of Hong Kong general population; (2) evaluate congener profiles and their correlations with each other and other PBDEs structure analogs; (3) examine the factors affecting body accumulation of PBDEs; and (4) investigate the contribution of fish consumption and indoor dust inhalation for PBDE human body bioaccumulation.

2. Materials and methods

2.1. Sample collection and preparation

Studies were performed in accordance with the guidelines and approval of Human Investigation Ethics Committee of Department of Biology, Hong Kong Baptist University. A total of 117 participants (blood donors were Southern Han Chinese in origin, female 54, male 63) were recruited during February 2011. All participants were determined to be eligible as blood donors based on their health history statuses and a screening by nurses at the Hong Kong Red Cross before recruitment into the study. Blood plasma samples in the present study were from people younger than 65 or older than 16 years old were excluded. Samples were classified into five age groups 16–20 ($n=9$), 21–30 ($n=36$), 31–40 ($n=22$), 41–50 ($n=28$), 51–65 ($n=22$) years of age. Samples of blood were collected in heparinized tubes, maintained at 4 °C, and centrifuged at $1000\times g$ for 15 min to allow collection of the plasma fraction. All samples of plasma were kept at −20 °C until extraction.

2.2. Extraction and instrumental analysis

Approximately 3 g of plasma for each sample were transferred to a clean centrifuge tube and spiked with a known amount of PCB-60 (5 ng) and PCB-137 (5 ng). The mixture was mixed and equilibrated overnight. Hydrochloric acid (6 M, 1 ml) and 2-propanol (5 ml) were added and mixed. The sample was subsequently extracted three times with a hexane/MTBE mixture (1:1; 5 ml). The organic extracts were combined and concentrated. The lipid content was determined by gravimetric method the content of each sample was used to express concentrations of PBDE on a lipid normalized basis [14]. After measuring the lipid contents, the organic extracts were removed lipid by concentrated sulfuric acid and concentrated to 1 ml before clean up on a florisil column as described previously [15]. After concentrated and resolved in 50 μl *n*-hexane, duterated internal standards (^{13}C-labeled BDE-3, ^{13}C-labeled BDE-28, ^{13}C-labeled BDE-153, and ^{13}C-labeled BDE-197) were added prior to instrumental analyses. The detailed procedures used to identify and quantify PBDEs are described in the Supporting Information according to our previously published study [9]. The 22 targeted PBDE congeners are BDE-3, 7, 15, 17, 28, 47, 49, 66, 71, 77, 85, 99, 100, 119, 126, 138, 153, 154, 156, 183, 184, and 191 (IUPAC congener numbers). Details of quantification parameters of target compounds are described in Table S1.

2.3. Quality assurance/quality control

For every sequence of 15 samples, a solvent blank, a procedural blank, and a known amount PBDE standard solution were processed to ensure that the samples and the analysis process. Several quality control criteria were used to ensure correct identification and quantification of the target compounds: first, retention times were compared with those of authentic reference compounds; second, ratios of the two characteristic ions were determined to be within 15% of the theoretical values; third, the signal-to-noise (S/N) ratio was determined to be greater than 3 for the selected ions; forth, the amount of analytes in the sample had to be at least two times that in the blank sample. If any of these three criteria failed, the congener was excluded from the data set. The limit of detection (LOD), defined as concentrations of analytes that gave rise to a peak with a signal-to-noise ratio (S/N) of 3, was determined for each analyte by use of a batch standard (Table 1). Recoveries of PCB-60 and PCB-137, spiked into blood plasma, ranged from 85 to 109% (mean 93.4%) and from 89 to 107% (mean 95.1%), respectively. Recoveries of the solvent spiked samples for PBDEs ranged from 89.2 to 112%.

2.4. Data analysis

The results of PBDEs were not adjusted since recoveries were consistent and sufficiently great. If the concentration of an individual congener was less than the LOD, its concentration was assumed to be LOD/2 for statistical analysis, while it was set at zero for sum, mean and median calculations. Concentrations of PBDE were presented as ng g^{-1}, lipid weight (lw). Data analysis was performed using SPSS 17.0 for Windows. The data were log-transformed prior to conducting statistical tests. Normality was confirmed by use of the Kolmogorov–Smirnov test. Student t test was used to compare the concentrations of pollutants between female and male. The effects of age on pollutants body burden were determined by one-way ANOVA. The correlation between PBDE individual congeners in human plasma of general population with their concentrations in twenty market fish species (n = 279) and indoor dust (n = 55) were investigated respectively. PBDE congeners in market fish and indoor dust were detected in our previous studies [9,10] and listed at Table S2. Correlation analyses between concentrations of PBDE, MeO-BDEs, OH-BDEs and BRP congeners and their corresponding total concentrations in human plasma were conducted using pair-wise correlation method. Concentrations of other PBDE structural analogs were detected in our recent study [13] and listed at Table S3. The accuracy of the data was determined to be two significant figures.

3. Results and discussion

3.1. Concentrations of PBDE in blood plasma

Concentrations of $\sum$PBDE$_{22}$ ranged from 0.56 to 92.2 ng g^{-1}, with a median value of 5.36 ng g^{-1}, lw (Table 1). Previous studies indicated that concentrations of $\sum$PBDE in humans in South China have been increasing in recent years [16]. However, the difference detected PBDE congeners made it hard to compare our data ($\sum$PBDE$_{22}$, 0.56–92.2 ng g^{-1}, median 5.36 ng g^{-1}, lw) with those of others about the PBDE concentrations in human samples collected from the PRD ($\sum$PBDE$_7$, 1.5–17 ng g^{-1}, median of 4.4 ng g^{-1}, lw [17]; and $\sum$PBDE$_{16}$, median 600 ng g^{-1}, lw, [16]). When compared with general populations from around the world, the median concentration of $\sum$PBDE$_{22}$ 5.4 ng g^{-1}, lw in human blood plasma of people in Hong Kong was greater than that in blood of people in Sweden ($\sum$PBDE$_{10}$ 2.07 ng g^{-1}, lw) [18], Germany ($\sum$PBDE$_{16}$ 4.53 ng g^{-1}, lw) [19], Belgium ($\sum$PBDE$_{11}$ 3.64 ng g^{-1}, lw) [20], Singapore ($\sum$PBDE$_7$ 2.60 ng g^{-1}, lw) [21], Japan ($\sum$PBDE$_7$ 3.52 ng g^{-1}, lw) [19], and Northern China ($\sum$PBDE$_{21}$ 2.90 ng g^{-1}, lw) [22], comparable with that in people from the New Zealand ($\sum$PBDE$_6$ 6.29 ng g^{-1}, lw) [23], but less than those in people from the United States ($\sum$PBDE$_6$ 61.0 ng g^{-1}, lw) [3] and Canada ($\sum$PBDE$_4$ 13.4 ng g^{-1}, lw) [5].

Table 1
The concentrations (ng g^{-1}, lw) of PBDE congeners and $\sum$PBDE$_{22}$ in human blood plasma collected from Hong Kong.

	Mean ± SD	Median	Min	Max	LOD	FOD
BDE-47	1.84 ± 2.32	1.42	0.18	17.58	0.10	100
BDE-28	1.79 ± 6.23	0.54	ND	46.08	0.05	98.2
BDE-99	1.20 ± 1.35	0.97	0.06	9.72	0.05	100
BDE-100	0.70 ± 4.02	0.10	ND	30.78	0.05	77.6
BDE-153	0.42 ± 1.11	0.17	ND	7.19	0.05	91.4
BDE-184	0.42 ± 1.86	0.14	ND	14.32	0.05	82.8
BDE-183	0.23 ± 0.60	0.11	ND	4.63	0.05	87.9
BDE-154	0.21 ± 0.21	0.15	ND	1.21	0.05	87.9
BDE-17	0.19 ± 0.06	0.09	ND	1.97	0.05	70.7
BDE-71	0.09 ± 0.11	0.05	ND	0.63	0.05	55.2
BDE-85	0.08 ± 0.10	0.04	ND	0.37	0.05	50.0
BDE-49	0.08 ± 0.08	0.05	ND	0.42	0.05	56.8
BDE-191	0.07 ± 0.16	0.04	ND	1.18	0.05	46.6
BDE-15	0.07 ± 0.10	0.04	ND	0.58	0.03	70.7
BDE-66	0.06 ± 0.09	0.04	ND	0.66	0.03	77.6
BDE-156	0.05 ± 0.08	0.03	ND	0.57	0.05	34.5
BDE-126	0.04 ± 0.05	0.03	ND	0.23	0.03	48.2
BDE-7	0.04 ± 0.05	0.03	ND	0.35	0.03	60.3
BDE-138	0.04 ± 0.03	0.03	ND	0.10	0.03	55.2
BDE-3	0.04 ± 0.04	0.03	ND	0.25	0.03	56.9
BDE-119	0.03 ± 0.03	0.02	ND	0.15	0.02	50.0
$\sum$PBDE$_{22}$	9.48 ± 14.8	5.36	0.56	92.2	–	–

ND, not detected; LOD, limit of detection; FOD, frequency of detection.

3.2. Congener profiles

In order of average dominant contribution, the eight predominant PBDE congeners are BDE-47, BDE-28, BDE-99, BDE-100, BDE-153, BDE-184, BDE-183 and BDE-154 (Table 1). The average proportions of $\sum$PBDE$_{22}$ contributed by individual congeners were: BDE-47 (26%) > BDE-99 (18%) > BDE-28 (16%). These three congeners made up 5.1 to 90% (median 66%) of the $\sum$PBDE$_{22}$. BDE-47 was the predominant congener detected in human plasma, which was similar to the trend observed for BDE-47 in other human tissues collected from people in this region, such as hair and milk [10,17], which suggested the wide spread use of technical penta-BDEs in this area. The lack of heavier PBDEs (octa- and nona-PBDEs) detected in plasma of Hong Kong residents is similar to previous studies that nona and deca-BDE congeners were not detected or were not abundant in the general population from other countries such as Singapore [21].

In order to compare the relative proportions of individual congeners observed in this study with those from other countries and DE-71, an important commercial penta-BDE mixture and the presumptive source of PBDEs in human blood, the sum of the seven predominant PBDE congeners, BDE-28, -47, -99, -100, -153, -154, and -183 were normalized to 100%. The predominant congeners in blood plasma of people in Hong Kong were BDE-47 (41%) and BDE-99 (28%), followed by BDE-28 (16%), BDE-153 (4.9%), BDE-154 (4.3%), BDE-183 (3.2%) and BDE-100 (2.9%) (Fig. 1). The predominant congener in blood plasma of humans in Hong Kong is BDE-47, which is also the predominant congener in blood plasma of people in North America [24] and some European countries such as Sweden [18]. The results may be explained by the enhanced environmental availability of BDE-47 due to its relatively greater vapor pressure [25]. Furthermore, much of BDE-47 can also come from the diet and from natural marine sources, particularly seafood [19]. Results of a previous study revealed that BDE-47 was the predominant PBDE congener in 20 fish species collected from Hong Kong markets, accounting for 29% of $\sum$PBDE$_{22}$ [9]. The relative proportion of $\sum$PBDE$_{22}$ contributed by BDE-28 (16%) observed in blood plasma in people from Hong Kong was greater than that observed in other regions, followed by the South China. This result suggests that the contribution of less brominated PBDE congeners is greater in this region than it is in other countries. The reason for this observation will be further discussed below. The percentage of BDE-99 in DE-71 is greater than the relative proportion of this congener in blood plasma collected from people in Hong Kong and other compared regions and countries. Neglecting the potential for slightly different uptake efficiencies of these congeners, the results of the study presented here suggest that BDE-99 might be the least persistent PBDE congener in DE-71 that can accumulate into human blood [26]. The selective environmental elimination of BDE-99 has also been observed in some species such as common carp [19]. Relatively small contributions of hexa-BDEs, which contain primarily BDE-153 and -154, and octa-BDEs, which are comprised primarily of BDE-183 were observed in plasma of people in Hong Kong compared with people from other countries or regions [19]. The

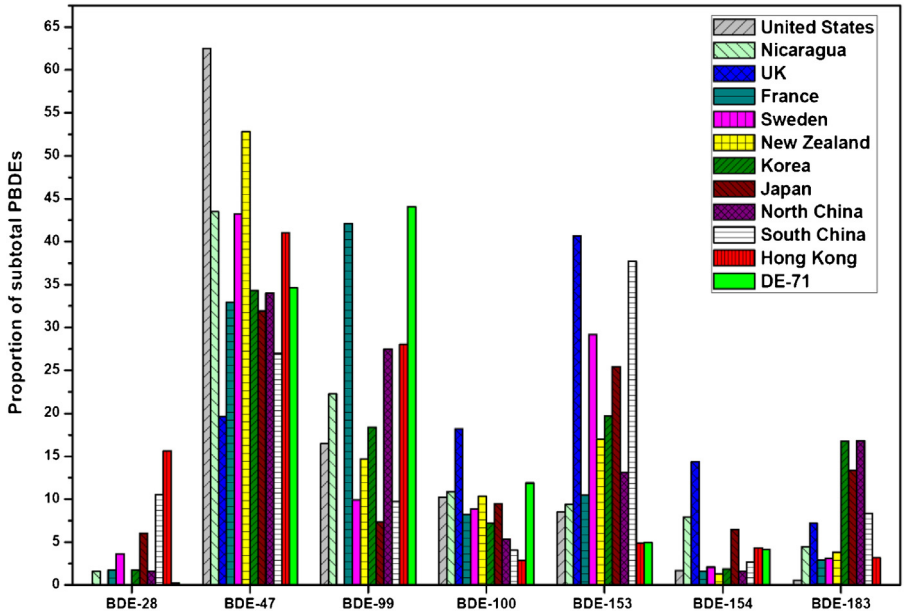

Fig. 1. The proportions of seven most dominant PBDE congeners in plasma collected from different counties or regions in the world. The data normalized to the sum of BDE-28, -47, -100, -153, -154, and -183 were from published data: the United States [24]; Nicaragua [42]; UK [6]; France [43]; Sweden [18]; New Zealand [23]; Korea [44]; Japan [45]; North China [22]; South China [17]; Hong Kong (the present study); and DE-71 [46].

differences in profiles of relative concentrations of individual PBDE congeners might be related to dietary habits or temporal trends in the exposure to various PBDE technical mixtures.

3.3. Gender, age and other contributing factors

There was no significant (t-test: $p = 0.88$) difference between $\sum PBDE_{22}$ concentrations in females (1.53–92.2 ng g^{-1}, median 5.46 ng g^{-1}, lw) and males (0.56–57.1 ng g^{-1}, median 5.26 ng g^{-1}, lw). It was different with previous studies that concentrations of $\sum PBDEs$ in adipose samples of males were greater than those in females [27], but consistent with previous studies that there is no significant difference for the PBDE distribution in males or females blood from Dalian, China [28] and Australia [29]. It suggested that gender was not the determining factor for PBDE bioaccumulation in human plasma.

Previous studies indicated that concentrations of persistent lipophilic compounds such as PBDEs tend to increase with age and body fat content [11]. No significant difference (one-way ANOVA: $p = 0.71$) was observed among concentrations of $\sum PBDE_{22}$ in blood plasma of the five age groups (16–20, 21–30, 31–40, 41–50, and 51–65) studied in Hong Kong. Furthermore, no significant ($p > 0.05$) relationship was observed between $\sum PBDE_{22}$ concentration and lipid content of blood plasma. These results

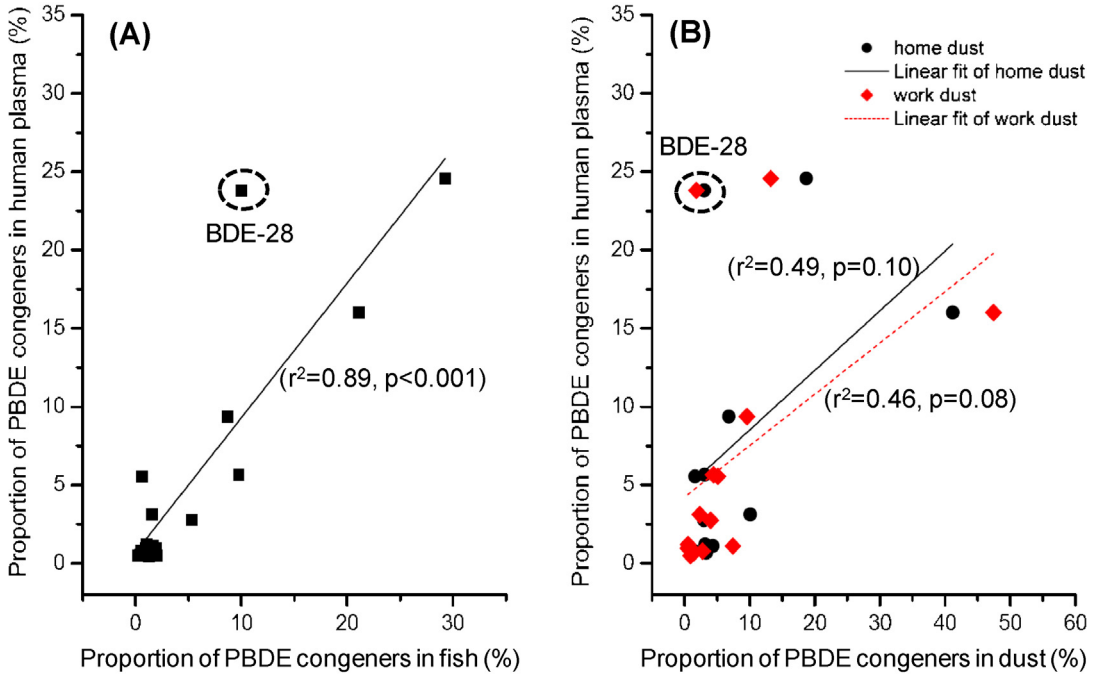

Fig. 2. Correlations between fish (A) or dust (B) concentrations of PBDE congeners and their corresponding concentrations in blood plasma of Hong Kong residents.

Table 2
Pairwise correlations between the concentrations of predominant PBDE, MeO-BDE, OH-BDE and BRP congeners and their corresponding total concentrations in human plasma.

	BDE-47	BDE-28	BDE-99	BDE-100	BDE-153	BDE-184	BDE-183	BDE-154	$\sum$PBDE$_{22}$	3-MeO-BDE-47	$\sum$MeO-BDEs	6-OH BDE-47	$\sum$OH-BDEs	2,4,5-TBP	$\sum$BPRs
BDE-47	1														
BDE-28	.08	1													
BDE-99	**.90****	.06	1												
BDE-100	**.92****	.05	**.85****	1											
BDE-153	**.52****	**.82****	**.49****	**.52****	1										
BDE-184	**.92****	.05	**.86****	**.99****	**.52****	1									
BDE-183	**.93****	.04	**.89****	**.98****	**.52****	**.98****	1								
BDE-154	**.79****	.10	**.77****	**.65****	**.47****	**.65****	**.73****	1							
$\sum$PBDE$_{22}$	**.75****	**.50****	**.75****	**.75****	**.81****	**.76****	**.77****	**.65****	1						
3-MeO-BDE-47	.02	.21	.05	.01	.19	.03	.11	.10	.11	1					
$\sum$MeO-BDEs	.04	.21	.10	.03	.20	.05	.13	.12	.12	**.99****	1				
6-OH-BDE-47	.00	−.08	.24	−.09	−.04	−.07	−.05	.08	−.04	−.01	.03	1			
$\sum$OH-BDEs	.00	−.13	.24	−.07	−.14	−.06	−.04	.11	−.12	.14	.18	**.92****	1		
2,4,5-TBP	.33	−.22	.07	.08	−.21	**.43***	.33	**.45***	.24	.21	.21	.34	.05	1	
$\sum$BPRs	−.10	.05	−.19	−.09	−.04	−.07	−.08	−.08	−.09	.16	.16	−.14	.01	**.92****	1

Bold means a statistical significance.
** Correlation is significant at the 0.01 level (2-tailed).

are consistent with the results of previous studies that concentrations of PBDE in tissues of humans were not correlated with age or lipid contents in China [16,22]. This might due to that PBDEs are relative new chemicals in the Hong Kong environment (they started to enter the China environment since 1980), therefore the age and fat may not yet be the most important factor in explaining their distribution in general population in this area.

3.4. Accumulation of PBDEs via fish and indoor dust

Fish are the dominant dietary source of PBDEs in some countries such as Finland and Belgium [8,30], even though the consumption rates of fish and PBDE concentrations in fish in those countries were less than that of Hong Kong. The results of a recent study suggested that dietary intake of PBDEs from fish and other seafood was greater than from other food items such as meat and poultry in Hong Kong [31]. Furthermore, indoor dust inhalation has been hypothesized to be a major route for human exposure to PBDEs [10]. Therefore, the correlations between body bioaccumulation of PBDEs and fish consumption and dust inhalation have been investigated in the present study.

The significant ($r^2 = 0.89$, $p < 0.001$), positive correlation was observed between proportions of individual PBDE congeners in market fish muscle (data from our previous study [9] are listed at Table S2) and blood plasma of humans in Hong Kong (Fig. 2A), suggesting that fish consumption is an important exposure pathway for exposure of residents of Hong Kong to PBDE. Although the data of the $\sum$PBDE concentrations in market fish samples were not one to one correspondences with human blood samples, their individual PBDE congener proportions in both human plasma of general population and general market fish species were one to one correspondences, and therefore the regression analyses could illustrate the role of fish consumption in PBDE body accumulation for general Hong Kong people. These results were consistent with those of earlier studies in Northern Europe [32] and Japan [33], which found associations between concentrations of PBDE in human tissues and rate of consumption of fish.

The results of previous studies have also suggested that PBDEs in indoor air and dust might be another potential source of exposure that results in accumulation of PBDE by humans [34]. This is especially true for children, where ingestion of indoor dust can lead to as much as a 100-fold greater exposure to PBDE for toddlers than for adults [35]. In the present study, positive but not statistically significant correlations were observed between the proportions of individual PBDE congeners in indoor dust [10] collected from workplaces ($r^2 = 0.46$, $p = 0.081$) and homes ($r^2 = 0.49$, $p = 0.10$) with concentrations in blood plasma of people in Hong Kong (PBDE concentrations in dust samples from our previous study [10] are listed at Table S2). Similar to fish consumption analyses, the individual PBDE congener concentrations in both human plasma and indoor dust were one to one correspondences. This was different from the result that dust is a primary PBDE exposure pathway for American [36] while similar to another study conducted on 34 German homes found no significant correlation between dust and serum concentrations of PBDEs [37]. It might be due to the fact that concentrations in dust in European countries and Hong Kong are orders of magnitude less than those in the U.S.A. [38]

Both food consumption and indoor dust are pathways for Hong Kong general population exposure to PBDEs. Dietary intake of PBDEs via fish consumption (265–831 ng d^{-1}) was significantly greater than accumulation via inhalation (1.41–277 ng d^{-1}) by people in Hong Kong [10].

3.5. Correlation of organobrominated compounds

Proportions of BDE-28 in blood plasma of Hong Kong and South China were greater than those in blood of people in other regions, which suggests a local source for this congener. Alternatively, the proportion of BDE-28 in blood plasma (15.2 ± 14.1%) was greater than that in fish muscle (9.94 ± 6.24%) and indoor dust (2.37%) (Fig. 2). This result is suggestive that there are other sources such as metabolism for the BDE-28 accumulation in humans. This hypothesis is supported by the results of a previous study, performed in Norway, which revealed that correlation coefficients of between concentrations of BDE-28 in seafood and human plasma were weak and sources other than diet were likely contributing to exposure [39].

Pairwise correlations were analyzed between concentrations of predominant PBDE, MeO-BDE, OH-BDE and BRP congeners and their corresponding total concentrations in human plasma (Table 2) (MeO-BDE, OH-BDE and BRP concentrations in blood samples from our previous study [13] are listed at Table S3). Concentrations of all of the predominant individual PBDE were significantly ($p < 0.01$) correlated with each other except for BDE-28, which was only correlated with BDE-153 (Table 2). These results suggested that BDE-28 might be biotransformated or metabolized after assimilation from individual congeners (such as BDE-153). It was also supported by the fact that the concentrations of BDE-28 in plasma of people in the North China were significantly greater than would be predicted from congener profiles based on various mixtures [22]. Further studies are needed to investigate whether there are specific or higher activity enzyme systems involved in BDE-28 generation for people living in this region.

Furthermore, there was no significant correlation among PBDE congeners and MeO-BDEs, OH-BDEs or BRPs in blood plasma in the present study. This result is consistent with those of previous studies about the tissues of marine animals collected from different global locations [40]. This might due to MeO-/OH-BDEs being inter-converted and synthetic PBDEs used in flame retardants, are not precursors of MeO-BDEs or OH-BDEs in blood plasma of humans in Hong Kong [41]. Although there were significant positive correlations between PBDE congeners (BDE-184 and BDE-154) and 2, 4, 5-TBP, these metabolic relationships have never been reported and need further study.

4. Conclusions

Concentrations of $\sum PBDE_{22}$ ranged from 0.56 to 92 ng g^{-1}, lipid weight (lw), with a median of 5.4 ng g^{-1}. BDE-47 was the predominant congener detected in human plasma. The results suggested that fish consumption is the dominant intake pathway for exposure of resident of Hong Kong to PBDEs, suggesting that appropriate safety measure should be applied to protect people from potential subtle, multi-generational effects of exposure to PBDE in food, particularly fish products. The spatial distribution and terrestrial sources of BDE-28 requires further study to confirm whether it is biotransformated in vivo by people living in this region. The blood collected from donors in Hong Kong Red Cross in the present study could serve as a benchmark matrix for studying the status and trends in body burdens of PBDEs in the general populations of South China.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jhazmat.2013.07.033.

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