Exposure of Hong Kong residents to PBDEs and their structural analogues through market fish consumption

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ABSTRACT

High concentrations of polybrominated diphenyl ethers (PBDEs) and their structural analogues (such as methoxylated (MeO) and hydroxylated (OH) PBDEs) had been observed in environmental samples and human tissues. This study evaluated the occurrence, potential source and human exposure of these organobrominated compounds via market fish consumption in Hong Kong. The contamination of 22 PBDEs, 7 MeO-BDEs, 15 OH-BDEs and 3 bromophenols (BRPs) were analyzed in 20 fish species (279 samples). The estimated daily intakes of PBDEs, MeO-BDEs, OH-BDEs and BRPs via fish consumption ranged from 4.4 to 14, 0.50 to 4.3, 0.02 to 0.43 and 0 to 0.21 ng/kg day for Hong Kong residents, respectively, based on 50th and 95th centile concentrations. BDE-47 and 99 were found to be the major PBDE congeners while 2′-MeO-BDE-68, 6-MeO-BDE-47 and 3-MeO-BDE-47 were the dominant MeO-BDEs. Concentrations of OH-BDEs and BRPs were 10–100-fold less than those of PBDEs, with small frequencies of detection (max 36.7%). Dietary intake of PBDEs via fish consumption by Hong Kong residents was greater than many developed countries, such as the USA, UK, Japan and Spain. To our knowledge, this is the first report to estimate the dietary intake of MeO/OH-BDEs and BRPs via fish consumption. Our results indicated that the toxicity potential of these compounds should not be neglected.

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1. Introduction

Polybrominated diphenyl ether (PBDE), a subcategory of brominated flame retardants (BFRs), has been produced and used in large quantities worldwide since the 1970s [1]. Uncontrolled discharges of materials containing BFRs can lead to unintentional environmental exposure to these chemicals. Over the last 30 years, concentrations of PBDEs in human blood, breast milk and other tissues have been increasing with doubling times of approximately 4–6 years [2]. Recently, it has been found that the structural analogues of PBDEs, such as hydroxylated (OH-), methoxylated (MeO-) BDEs and bromophenols (BRPs) are ubiquitous in the environment and human tissues [3–6]. The OH- and MeO-BDEs exhibited even greater potency for some adverse effects such as disruption and imbalance between androgens and estrogens in organisms than PBDEs. Concerns over these organobrominated compounds are of

particular interests since they are structurally similar to the thyroid hormone thyroxin (T4) and could alter thyroid hormone homeostasis [7], estradiol synthesis and cause neurotoxicity [8].

Relatively high concentrations of PBDEs were observed in blood and breast milk collected from people living in South China (ranged from 1.5 to 17 ng g^{-1}) [9], serum (the 95th percentile for $\sum$PBDEs was 291 ng g^{-1}, l.w., n = 2062) [10] and blood ($\sum$PBDEs ranged from 4.6 to 365 ng g^{-1}, l.w., n = 100) [11] collected from United States. These studies noted marked increase in PBDEs in human tissues over several decades. It was revealed that concentrations of PBDEs in blood serum were positively correlated with fish consumption [12], which contributed approximately 40–50% of the total dietary PBDE intake [13–15]. In fact, fish have been found to be the dominant dietary source of PBDEs in some counties such as Finland and Belgium [14,16], even though consumption rates of fish in those countries were less than that of Hong Kong. Freshwater and marine fish, are the major dietary source of protein for many Hong Kong residents. A recent study suggested that dietary intake of PBDEs from fish and other seafood was greater than other food items such as meat and poultry in Hong Kong [17].

In addition to PBDEs, MeO/OH-BDEs and BRPs have been detected in human serum collected from Nicaragua ($\sum$OH-BDEs

ranged from 11 to 120 pmol g^{-1}, l.w. in a waste disposal site, while from 3.1 to 5.6 pmol g^{-1}, l.w. in referents) [6] and United States (the total concentration of HO-PBDEs and bromophenols ranged from 2.0 to 900 ng g^{-1}, l.w., mean of 79 ng g^{-1}, l.w.) [18]. These compounds may originate from biotransformation or directly from dietary intake [6]. It is reasonable to assume some PBDE metabolites detected in humans, especially MeO-BDEs, may derive from dietary intake, because MeO-BDEs have been detected at relatively high concentrations in fish [19]. However, there is no published data concerning the dietary intake of MeO/OH-BDE and BRP in some common food items such as fish. In addition, there is no information about MeO/OH-BDEs and BRPs in human tissues in relation to dietary food intakes.

The objectives of the present study were: (1) to indentify and quantify PBDEs and their structural analogues in edible fish at Hong Kong markets; (2) to assess potential sources and bioaccumulation behavior of these compounds; and (3) to evaluate the dietary intake of these organobrominated compounds via fish consumption and the associated potential health risks of Hong Kong residents.

2. Materials and methods

2.1. Sample collection and treatment

Twenty species of commonly consumed fish were purchased from local fish markets from May to November 2009. Their scientific and common names, origins (production area), and feeding strategies are listed in Table S1. At least six individual samples were collected for all species except Mandarin fish, which had only 3 individuals because of their large body mass (1.4–1.6 kg). Altogether, there were 279 fish samples for the analysis. All samples were stored in ice boxes and were directly transported back to the laboratory. The edible muscle of fish was dissected, freeze-dried, and homogenized by grounding into powder prior to analyses.

2.2. Chemicals analyses

The detail extraction, clean up and quantification procedures are described in the Supporting Information. Target chemical standards included 22 PBDE congeners (IUPAC congener numbers 3, 7, 15, 17, 28, 47, 49, 66, 71, 77, 85, 99, 100, 119, 126, 138, 153, 154, 156, 183, 184 and 191), 9 MeO-BDE congeners (7 target compounds: 2′-MeO-BDE-28, 4′-MeO-BDE-49, 2′-MeO-BDE-68, 6-MeO-BDE-137, 6-MeO-BDE-47, 3-MeO-BDE-47, 5-MeO-BDE-47, surrogate: ^{13}C-6-MeO-BDE-47 and internal standard: ^{13}C-6-MeO-BDE-100), 17 OH-BDE congeners (15 target compounds: 3-OH-BDE-47, 5-OH-BDE-47, 5′-OH-BDE-99, 6′-OH-BDE-99, 2′-OH-BDE-7, 3′-OH-BDE-7, 2′-OH-BDE-28, 4′-OH-BDE-17, 6-OH-BDE-47, 2′-OH-BDE-66, 4-OH-BDE-42, 6-OH-BDE-90, 6-OH-BDE-85, 3-OH-BDE-100 4-OH-BDE-90; surrogate: ^{13}C-6-OH-BDE-17 and internal standard: ^{13}C-6-OH-BDE-100) and 3 BRP congeners (2, 4-dibromophenol, 2,4,5-tribromophenol and 2,4,6-tribromophenol). Chemical standards used in the present study were purchased from Cambridge Isotope Laboratories (Andover, MA, USA), Accustandard (New Haven, CT, USA) and Wellington Laboratories (Guelph, ON, Canada), except the last 11 OH-BDE target congeners which were synthesized in the Department of Biology and Chemistry of City University of Hong Kong with purities >98%. All solvents used were GC grade obtained from Tedia Company, Inc. (Fairfield, Ohio, USA). All other reagents were of analytical grade or HPLC grade.

2.3. QA/QC

To test the recovery rates, the standard reference material (SRM) 2977 (mussel tissue) obtained from the National Institute of Standards and Technology (NIST, USA) was used for PBDE analysis, and the surrogates of ^{13}C-6-MeO-BDE-47 and ^{13}C-6-OH-BDE-17 were used for MeO-BDEs and OH-BDEs (BRPs), respectively. Rates of recovery were 86.3–105.4% for PBDEs in SRM 2977, 76.3–121.2% and 64.5–132.1% for MeO-BDEs and OH-BDEs (BRPs) in spiked samples. The variation coefficient of PBDE concentrations between duplicate samples was less than 15%. For each set of 15 samples, a procedural blank (solvent), a sample duplicate and a SRM 2977 were processed. The method detection limit (MDL) was calculated as a mean of the background signal plus three times the standard deviation in the blank samples. MeO-BDEs, OH-BDEs and BRPs was undetectable in procedural blanks. The MDL ranged from 0.01 to 0.05 ng g^{-1} for PBDEs (BDE28, BDE47, BDE85, BDE-99 and BDE153 were detected in blank samples) and 1–5 pg g^{-1} for MeO-BDEs, OH-BDEs and BRPs in various samples (wet weight, ww). For those measurements less than the MDL, a proxy value of half the MDL was assigned. Due to the small rates of recovery of MeO-BDEs, OH-BDEs and BRPs, they were corrected for recovery by use of surrogates in matrix spike samples. However, the results of PBDEs were not adjusted because the recovery rates were acceptable.

2.4. Data analyses

Concentrations were presented as ng g^{-1}, ww, for PBDEs and MeO-BDEs and pg g^{-1}, ww for OH-BDEs and BRPs to keep the data at two significant figures. Data analyses were performed using SPSS 17.0 for Windows. Normality was confirmed by Kolmogorov–Smirnov test. Concentrations of PBDEs, MeO/OH-BDEs and BRPs were analyzed using two independent t-test, Wilcoxon rank sum test, one-way ANOVA and Kruskal–Wallis test.

2.5. Estimated daily intake and risk evaluation

The daily intake (DI) from fish of all target compounds and the hazard Quotient (HQ) for BDEs 47, 99 and 153 were calculated using Eqs. (1) and (2) [20], respectively.

$$DI = \frac{C \times CR \times EF \times ED}{BW \times AT};$$ (1)

$$HQ = \sum PBDE \, Congener \, Hazard \, Quotients = \sum \frac{DI}{RfD}$$ (2)

C is the concentration of detected compounds, CR is the average fish consumption rate for the general Chinese population (70 g day^{-1}) [21], exposure frequency (EF) is assumed to be 365 days/year; exposure duration (ED) 30 years, and body weight (BW) 60 kg. For DI estimation, averaging time (AT) was assumed to be 10,950 days (EF × ED). Estimates of DI were reported as ng/kg day for PBDEs and MeO-BDEs, pg/kg day for OH-BDEs and BRPs, based on the measured 50th and 95th centile concentrations (ww). The HQs for BDEs 47, 99 and 153 and $\sum$PBDEs were calculated by using reference dose (RfD) values reported in the draft U.S. EPA IRIS Toxicological Evaluations and published data [22–24].

3. Results and discussion

3.1. Concentrations of PBDEs and structural analogues

Concentrations of $\sum$PBDEs, $\sum$MeO-BDEs, $\sum$OH-BDEs and $\sum$BRPs in Hong Kong market fish are shown in Fig. 1. Concentrations of $\sum$PBDEs ranged from 1.9 to 16 ng g^{-1}, with the median value of 4.1 ng g^{-1}. There was no significant (t-test: $p = 0.594$) difference between $\sum$PBDEs concentrations in freshwater fish (mean
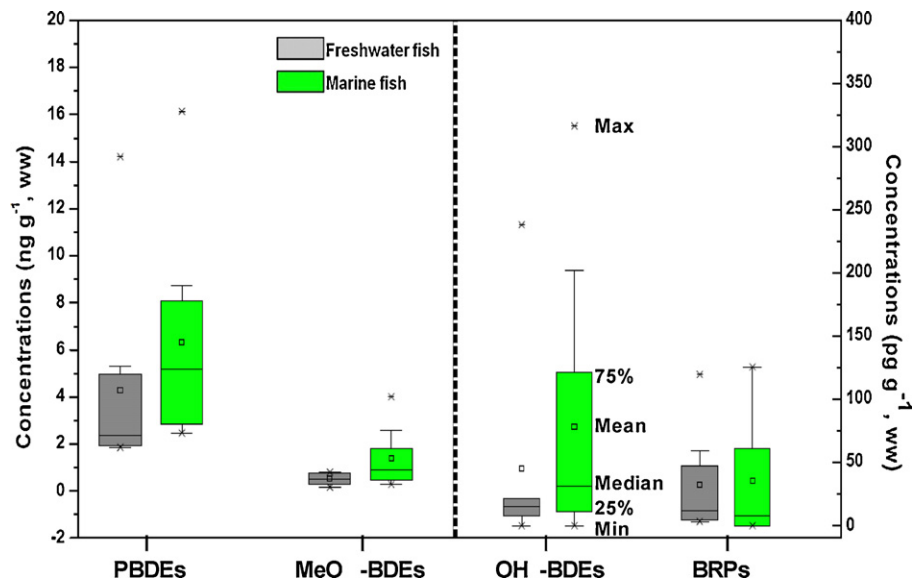
Fig. 1. Concentrations of $\sum$PBDEs, $\sum$MeO-BDEs, $\sum$OH-BDEs, and $\sum$BRPs in 10 freshwater and 10 marine fish species collected from a Hong Kong market.

of 4.3 ng g^{-1}) and marine fish (mean of 6.3 ng g^{-1}). Concentrations of $\sum$PBDEs in the present study were generally greater than those reported from the Netherlands ($\sum$PBDE$_5$, 0.01–4.8 ng g^{-1}, ww) [25], United States ($\sum$PBDE$_{13}$, 0.011–3.7 ng g^{-1}, ww) [26], Japan ($\sum$PBDE$_6$, 0.02–1.65 ng g^{-1}, ww) [27], Canada ($\sum$PBDE$_{18}$, <0.01–5.5 ng g^{-1}, ww) [28], Spain ($\sum$PBDE$_6$, 0.77 to 2.02 ng g^{-1}, ww) [29] and southern China ($\sum$PBDE$_{11}$, <0.0012–3.85 ng g^{-1}, ww) [30]. In general, the concentrations $\sum$PBDE$_{22}$ in Hong Kong market fish were comparable to or greater than those from other countries. This result is consistent with those of previous studies, which indicated that concentrations of PBDEs in fish from China were relatively high [31,32].

Unlike PBDEs, the concentrations of $\sum$MeO-BDEs in freshwater fish (9.0×10^{-2} to 1.7 ng g^{-1}, mean of 5.1×10^{-1} ng g^{-1}) were significantly (*t*-test: $p < 0.05$) less than those of marine fish (2.5×10^{-1} to 6.5 ng g^{-1}, mean of 1.4 ng g^{-1}). Concentrations of $\sum$MeO-BDE$_7$ in Hong Kong market fish were greater than that in tuna collected from North Pacific Ocean ($\sum$MeO-BDE$_{12}$, 0.27–0.76 ng g^{-1}, ww) [3] and adipose of Chinese sturgeon (0.14 ± 0.06 ng g^{-1}, ww) collected from Yangtz River [33]; but were comparable to anchovy (*Coilia* sp.) from the Yangtze River Delta, China ($\sum$MeO-BDE$_2$, N.D. to 8.2 ng g^{-1}, mean of 1.0 ng g^{-1}, ww) [19]. However, the concentrations were definitely less than that reported for some marine mammals, which could reach micrograms per gram (0.99 μg g^{-1}), based on lipid weights [34].

The concentrations of $\sum$OH-BDEs in freshwater fish (N.D.—238 pg g^{-1}, mean of 45.1 pg g^{-1}) were also significantly (Wilcoxon rank sum test: $p < 0.05$) less than those in marine fish (N.D.—316 pg g^{-1}, mean of 78.1 pg g^{-1}). However, the difference was not observed for $\sum$BRPs (Wilcoxon rank sum test: $p > 0.05$, mean of 32.0 and 35.4 for freshwater and marine species respectively). The OH-BDEs and BRPs concentrations were significantly less than those detected in albatross liver (0.27 ± 0.30 ng g^{-1}) [3], but comparable with those previously reported in fish plasma (ranged from 2.7 pg g^{-1} in white bass to 49 pg g^{-1} in longnose gar) [35]. The concentration differences might be due to different species, and/or the analytical methods.

3.2. Congener profiles

The concentrations of PBDEs, MeO/OH-BDEs, BRPs and their predominant congeners in the 20 fish species are listed in

Table 1. Of the 22 detected PBDEs, the proportions to $\sum$PBDEs of the most common congeners decreased as the following trend: BDE-47 (28.9%) > BDE-99 (20.9%) > BDE-28 (9.94%) > BDE-153 (9.63%) > BDE-100 (8.58%) > BDE-154 (5.24%). These six congeners made up 68.2 to 89.8% (mean 81.4%) of the total PBDEs. The lack of heavier PBDEs (octa- and nona-PBDEs) detected in both freshwater and marine fish might be related to the source profile or the fact that they were less soluble and more reliable in water [19]. The compositional profiles of PBDE congeners obtained in the present study were similar to previous studies obtained in fish collected from the Pearl River Delta (PRD) [15,32] and farmed or wild salmon collected from around the world [36].

Among the 7 detected MeO-BDE congeners, the most abundant MeO-BDE congeners were 2'-MeO-BDE-68 (mean 0.31 ng g^{-1}), followed by 6-MeO-BDE-47 (mean 0.29 ng g^{-1}) and 3-MeO-BDE-47 (mean 0.21 ng g^{-1}), together contributing 46.5–93.8% (mean 83.7%) of $\sum$MeO-BDEs in fish muscle. Previous studies suggested that 3-MeO-BDE-47 could not be detected or the concentration was much less than 2'-MeO-BDE-68 and 6-MeO-BDE-47 [37,38]. This is the first study showing that 3-MeO-BDE-47 had comparable concentrations with 2'-MeO-BDE-68 and 6-MeO-BDE-47 in environmental samples.

Concentrations of OH-BDE and BRP congeners were 10–100-fold less than PBDEs. Of the 15 OH-BDEs analyzed, 2'-OH-BDE-28 had the greatest detection frequency (36.7%), followed by 6'-OH-BDE-99 (11.7%) and 3-OH-BDE-47 (8.3%). 6-OH-BDE-90, 5'-OH-BDE-99, 6-OH-BDE-85, 3-OH-BDE-100 and 4-OH-BDE-90 could not be detected in all fish samples. The detection frequencies of other congeners ranged from 1.7 to 6.7%. For BRPs, the detection frequency was 26.7, 20.0 and 18.3% for 2,4,5-TBP, 2,4-DBP and 2,4,6-TBP, respectively. 6-OH-BDE-47 was the predominant congener in tissues of Chinese Sturgeon [33], bottlenose dolphin [39] and fish [35]. However in the present study, the detection frequency for this congener was only 1.67% in Hong Kong market fish.

3.3. Potential source apportionments

Freshwater carnivorous fish, such as the mandarin fish, had significantly (one way-ANOVA: $p < 0.01$) greater concentrations of PBDEs and MeO-BDEs than fish with other feeding strategies, such as omnivores, bottom feeders, herbivores and filter feeders. This could be explained by greater fugacity of persistent organic

Table 1
Concentrations of the predominant PBDEs, MeO- and OH-BDEs and BRPs in fish muscle from Hong Kong.[a]

Number	Name	PBDEs (ng g⁻¹)			MeO-BDEs (ng g⁻¹)				OH-BDEs (pg g⁻¹)				BRPs (pg g⁻¹)			
		BDE-47	BDE-99	$\sum$PBDEs	2-MeO-BDE-68	6-MeO-BDE47	3-MeO-BDE-47	$\sum$MeO-BDEs	2′-OH-BDE-28	6′-OH-BDE-99	3-OH-BDE-47	$\sum$OH-BDEs	2,4-DBP	2,4,5-TBP	2,4,6-TBP	$\sum$BRPs
Freshwater fish																
1	Tilapia	0.44	0.43	2.1 ± 0.43	0.04	0.03	0.11	0.25 ± 0.06	20	N.D.	N.D.	20 ± 3.3	N.D.	3.9	N.D.	3.9 ± 0.5
2	Spotted snakehead	1.5	0.93	4.4 ± 0.38	0.06	0.28	0.13	0.52 ± 0.05	15	N.D.	N.D.	15 ± 7.3	120	N.D.	N.D.	120 ± 36
3	Snakehead	1.1	0.89	5.0 ± 0.99	0.15	0.14	0.14	0.52 ± 0.11	N.D.	N.D.	N.D.	8.0 ± 3.9	56	3.2	N.D.	60 ± 47
4	Rice field eel	0.53	0.78	2.4 ± 0.19	0.16	0.48	0.10	0.79 ± 0.35	12	4	N.D.	12 ± 10	47	N.D.	N.D.	47 ± 12
5	Mud carp	0.33	0.35	1.9 ± 0.42	0.35	0.11	0.14	0.74 ± 0.45	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	4.7	4.7 ± 3.5
6	Mandarin fish	6.8	3.1	14 ± 4.7	0.45	0.08	0.18	0.80 ± 0.13	N.D.	N.D.	N.D.	116 ± 26	46	N.D.	N.D.	46 ± 19
7	Grey mullet	0.64	0.42	1.9 ± 0.47	0.05	0.04	0.02	0.16 ± 0.03	12	226	N.D.	238 ± 44	N.D.	3.2	N.D.	3.2 ± 2.4
8	Grass carp	0.29	0.36	1.9 ± 0.07	0.04	0.05	0.05	0.30 ± 0.18	21	N.D.	N.D.	21 ± 0.9	N.D.	6.4	5.4	12 ± 6.5
9	Catfish	2.5	0.64	5.3 ± 1.6	0.05	0.05	0.18	0.32 ± 0.19	7.4	N.D.	N.D.	21 ± 4.8	N.D.	8.0	N.D.	8.0 ± 7.4
10	Bighead carp	2.0	0.18	3.7 ± 1.3	0.13	0.30	0.13	0.64 ± 0.26	N.D.	N.D.	N.D.	N.D.	N.D.	3.2	13	16 ± 4.7
Marine fish																
11	Yellow seafin	0.82	0.64	2.5 ± 1.4	0.10	0.15	0.13	0.45 ± 0.16	N.D.	316	N.D.	316 ± 33	N.D.	7.9	N.D.	7.9 ± 3.1
12	Yellow croaker	0.54	1.7	5.2 ± 1.0	0.32	1.4	0.67	2.6 ± 0.44	N.D.	N.D.	20	20 ± 17	N.D.	N.D.	N.D.	N.D.
13	Tongue sole	1.9	1.6	8.7 ± 1.8	0.31	0.07	0.51	1.1 ± 0.25	23	N.D.	14	37 ± 11	52	3.2	N.D.	55 ± 28
14	Snubnose pompano	3.3	1.8	7.7 ± 2.5	0.15	1.3	0.17	1.8 ± 0.13	N.D.	110	N.D.	121 ± 23	126	N.D.	N.D.	126 ± 73
15	Orange-spotted grouper	0.72	0.46	2.9 ± 0.8	0.32	0.38	0.07	0.82 ± 0.01	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
16	Golden threadfin bream	0.78	1.1	3.7 ± 0.4	0.29	0.11	0.04	0.48 ± 0.12	9.2	N.D.	N.D.	42 ± 39	N.D.	N.D.	3.7	3.7 ± 2.5
17	Goldspotted rabbitfish	1.6	1.7	5.6 ± 0.4	0.70	0.28	0.18	1.3 ± 0.80	N.D.	N.D.	N.D.	202 ± 36	67	N.D.	N.D.	67 ± 35
18	Bleeker's grouper	0.72	0.87	2.7 ± 0.6	0.18	0.04	0.03	0.28 ± 0.03	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
19	Bigeye	1.5	1.1	8.1 ± 0.7	0.36	0.15	0.24	0.92 ± 0.45	11	N.D.	N.D.	11 ± 9.1	33	N.D.	N.D.	33 ± 14
20	Bartail flathead	2.6	3.1	16 ± 4.7	2.1	0.23	1.0	4.0 ± 2.7	24	N.D.	7.4	31 ± 13	57	3.8	N.D.	61 ± 27

[a] ng g⁻¹ for MeO-BDE while pg g⁻¹ for OH-BDEs and BRPs

Table 2
Bivariate correlations between the lipid contents, concentrations of MeO-BDE congeners, $\sum$MeO-BDEs and $\sum$PBDEs in fish muscle.

	LC (%)	$\sum$MeO-BDEs	2-MeO-BDE28	2-MeO-BDE68	6-MeO-BDE47	3-MeO-BDE47	5-MeO-BDE47	4-MeO-BDE49	6-MeO-BDE137	$\sum$PBDEs
LC (%)	1									
$\sum$MeO-BDEs	.147	1								
2-MeO-BDE28	−.146	**.646****	1							
2-MeO-BDE68	−.205	**.851****	**.629****	1						
6-MeO-BDE47	**.675****	**.475****	.014	.016	1					
3-MeO-BDE47	.029	**.837****	**.583****	**.697****	.199	1				
5-MeO-BDE47	−.098	**.808****	**.738****	**.766****	.127	**.699****	1			
4-MeO-BDE49	−.032	**.750****	**.637****	**.632****	.227	**.642****	**.887****	1		
6-MeO-BDE137	.021	**.609****	**.492****	**.576****	.174	**.403****	**.706****	**.703****	1	
$\sum$PBDEs	−.007	**.412****	**.436****	**.388****	.066	**.417****	**.327***	**.337****	.243	1

Bold means that the correlation is significant.
* Correlation is significant at $p < 0.05$ level (2-tailed).
** Correlation is significant at $p < 0.01$ level (2-tailed).

compounds in carnivorous fish, at higher trophic levels. However of the marine fish, the bottom feeding Bartail flathead and Tongue sole, had statistically significantly (Kruskal–Wallis test: $p < 0.05$) greater concentrations of PBDEs than fish with other feeding habits. This might due to the fact that wild marine bottom feeding fish can accumulate PBDEs from bacteria, fungi, algae, plants and sediments [40,41]. In our study, marine fish contained significantly (t-test: $p < 0.05$) higher concentration of MeO-BDEs than freshwater fish, while concentrations of PBDEs were comparable between marine and freshwater fish. It suggested that at least some of the MeO-BDEs in the fish muscle are derived from natural sources instead of biotransformation of PBDEs [19]. This hypothesis is supported by the following observations: (1) The two dominant MeO-BDE congeners (2′-MeO-BDE-68 and 6-MeOBDE-47) have been shown to be natural products produced by marine organisms instead of by metabolism [34]; (2) higher concentration of PBDEs would be observed in marine fish if MeO-BDEs are metabolized from PBDE due to the fact that concentrations of MeO-BDEs were higher in marine fish than freshwater fish; and (3) higher concentrations of MeO-BDEs in marine fish suggested that the source of MeO-BDEs may be from the sea. No significant correlation was observed between lipid contents and concentrations of either PBDEs or MeO-BDEs. This result suggests that lipid may not be the sole factor influencing concentrations of PBDEs and their OH- and MeO-analogues in the fish muscle [33]. Other factors such as species, age, gender, feeding strategies and the surrounding environment may also affect PBDEs accumulation in the fish muscle. It is difficult to evaluate the age, body size and gender-related exposure among fish species in the present study.

Bivariate correlations among lipid contents and concentrations of MeO-BDE congeners, $\sum$MeO-BDEs and $\sum$PBDEs were calculated (Table 2). Concentrations of nearly all MeO-BDE congeners were significantly and positively correlated with each other, $\sum$MeO-BDEs and $\sum$PBDEs, but not with 6-MeO-BDE47. Concentrations of 6-MeO-BDE-47 in the muscle of fish were positive correlated with only lipid contents and concentration of $\sum$PBDEs. In addition, 6-MeO-BDE-47 in freshwater fish (mean $0.16\,\mathrm{ng\,g^{-1}}$) was significantly ($t$-test: $p < 0.05$) less than that in marine fish (mean $0.42\,\mathrm{ng\,g^{-1}}$). This is consistent with previous studies that indicated marine fish had greater concentrations of trace organic

contaminants such as OCPs [42] and PBDEs [32] which were accumulated from environments. Furthermore, some studies have suggested that MeO/OH-BDEs with methoxy/hydroxyl group in an *ortho* position were natural products [37,43]. Therefore, it is likely that 6-MeO-BDE-47 detected in the marine fish is a natural product accumulated from the marine environment [34,44], while 3-MeO-BDE-47 and 5-MeO-BDE-47 are the biotransformation products of PBDEs [3]. Stable carbon isotope measurements have been used to confirm that 6-MeO-BDE-47 and 2′-MeO-BDE-68 are natural products [34]. Although there is possibility that 6-MeO-BDE-47 was biotransformed from 6-OH-BDE-47 in fish muscle [45], the low detection frequency (1.67%) of 6-OH-BDE-47 in the present study limited the further investigation about the interconversion of these two compounds.

Due to the limited detection frequency (max 36.7%), it is difficult to estimate the factors which affected OH-BDEs and BRPs concentrations in market fish. The greatest concentrations of $\sum$OH-BDEs ($316\,\mathrm{pg\,g^{-1}}$) and $\sum$BRPs ($109\,\mathrm{pg\,g^{-1}}$) were found in Yellow seafin and Snubnose pompano, respectively. There was no correlation between $\sum$BRPs and $\sum$OH-BDEs or between those classes and $\sum$PBDEs or $\sum$MeO-BDEs in the 20 fish. Our results were contradictory with a former study that found significant correlations between $\sum$MeO-BDEs and $\sum$OH-BDEs in albatross and polar bears [3]. This is possibly due to the fact that fish muscle had different sources of OH-BDEs or might have been a technical artifact of the small frequency of detection of BRPs and OH-BDEs in fish muscle. Based on the results of *in vivo* exposures, it has been suggested that OH-BDEs with a hydroxyl moiety at the *meta* or *para* position are the transformation products of PBDEs. OH-BDEs of natural origin should have a hydroxyl group at the *ortho* position [46]. The predominant congeners 2′-OH-BDE-28 and 6′-OH-BDE-99 in Hong Kong market fish are thus most likely to be found due to accumulation from the natural sources [3,33].

3.4. Intake estimation and risk evaluation

Estimated dietary intakes of organobrominated compounds associated with consumption of Hong Kong market fish were calculated (Table 3). The intake of PBDEs via consumption of Hong

Table 3
The estimated daily intake of PBDEs, MeO-, OH-BDEs and BRPs based on 50th and 95th concentrations with consumption of Hong Kong market fish.

	PBDEs (ng/kg day)		MeO-BDEs (ng/kg day)		OH-BDEs (pg/kg day)		BRPs (pg/kg day)	
	50th	95th	50th	95th	50th	95th	50th	95th
Freshwater fish	2.8	12	0.49	1.5	22	283	8.8	203
Marine fish	6.1	16	0.99	4.3	26	420	0	209
Total fish	4.4	14	0.68	3.1	23	352	1.6	208

Kong market fish was estimated to range from 4.4 to 14 ng/kg day on the basis of the 50th and 95th centile concentrations, respectively. Generally, DI values for PBDEs from fish consumption for Hong Kong residents were greater than those in the United States (3.0×10^{-2} to 2.5 ng/kg day) [20], Japan (3.0×10^{-2} to 2.78 ng/kg day, 60 kg bw) [27], UK (1.3 ng/kg day, 70 kg bw) [47] and Spain (3.0×10^{-1} ng/kg day, 70 kg bw) [29]. Considering that dietary intake of PBDEs in the present study (265–831 ng/day) was greater than accumulation via inhalation (2.7–9.2 ng/day) in the PRD [15], it is reasonable to assume that PBDEs intake via fish consumption is a major pathway for the exposure of Hong Kong residents to PBDEs. The estimated daily consumption rate of fish (70 g/day) used in the present study was the average value for people in China [21]. The estimated PBDEs intake via fish consumption would be greater if the greater consumption rate 164.4 g fish/day that has been reported for people in Hong Kong [48].

To assess the potential health risk caused by PBDEs, the U.S. EPA proposed RfD values for BDEs 47, 99, and 153 as part of the IRIS Toxicological Review for each PBDE congener [22–24]. The lowest observed adverse effect value of 1 mg $\sum$PBDEs/kg day [49] was used to calculate hazard quotients. Hazard quotients were calculated as the quotient of the 50th or 95th centile of the DI divided by the RfD (Table S3). When the 95th centile HQ is greater than 1.0, a refined risk assessment would subsequently be conducted to further ascertain the real risk. When both HQ values based on the 50th and 95th centile are greater than 1.0, an initiation of appropriate management strategies should be considered. HQs for all congeners and $\sum$PBDEs are less than 1.0, this may suggest a small non-cancer risk associated with the consumption of these Hong Kong market fish. It should be noted that the HQ values were based on the estimation of PBDE intake for adults with an average consumption rate of 70 g/day for the general Chinese population, the sensitive populations, such as fishermen who might eat even more fish, children and the elderly, were not considered.

The daily intake of MeO-BDEs via fish consumption ranged from 5.0×10^{-1} to 4.3 ng/kg day for Hong Kong residents. Daily intakes of MeO-BDE were less than the DI of PBDEs for Hong Kong people, but were comparable to or even greater than the DI of PBDEs for other countries such as the United States [20], Japan [27] and UK [47]. The DI of OH-BDEs and BRPs ranged from 22 to 4.3×10^2 pg/kg day and 0 to 2.1×10^2 pg/kg day, respectively. Most previous studies dealing with MeO/OH-BDEs and BRPs focused on their levels/distribution in liver or blood of high tropic level animals such as bear or ringed seals [3,4]. There is no published data about the dietary intakes of these compounds for comparison of the results obtained in this study. Furthermore, there is only limited toxicological information and no RfD for these organobrominated compounds. However, recent studies have found that some of the OH-/MeO-BDEs, especially OH-BDEs, exhibited greater neurotoxic, thyroid system disrupting and estrogenic potency than analogous PBDE congeners [8,50]. Therefore, the detected MeO/OH-BDEs and BRPs in the present study showed that these compounds should not be neglected in risk assessment, although their concentrations might be less than those of their parent compounds.

There is no previous intake estimation of MeO/OH-BDE and BRPs via ingestion intake or other pathways. However, recent studies reported the detection of several OH-BDE congeners in human blood [6,18], and the concentrations of OH-BDEs (4.1–120 pmol g^{-1}, l.w.) were greater in children consuming large amount of fish (0–8 meals/month) than those without fish consumption (3.1–4.1 pmol g^{-1}, l.w.) [6]. Although OH-BDEs in human blood could be of natural origin [6], further studies are essential to reveal the significance of the dietary source of OH-BDE, especially from fish consumption. Most previous studies have focused on concentrations in animals and there is limited published data on concentrations of OH and MeO-BDE in air, water and food samples [34,51]. Therefore, the role of dietary intake of these compounds in human exposure should be further investigated, especially their distribution in other environmental samples such as other food items and their metabolism in human body.

Overall, exposure to PBDEs via consumption of market fish by Hong Kong residents obtained in the present study was greater than those in other countries, but the associated non-cancer risks were small. However, the investigation in the present study only focused on the selected PBDEs. It is well known that many POPs such as OCPs and PAHs and other chemicals are reported to be contained in fish [42,52]. Considering that PBDEs could be accumulated in the organism and there were possibilities of synergistic effects when other pollutants are taken up simultaneously, more attention should be paid to the PBDE contamination in fish collected from these areas. The detection of MeO/OH-BDEs and BRPs in the edible fish indicated that these compounds exist in food items, and their toxicity potential should not be neglected.

Acknowledgements

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.jhazmat.2011.05.036.

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