BROMINATED FLAME RETARDANTS AND PERSISTENT ORGANIC POLLUTANTS IN INDONESIA: ASSESSMENT OF HUMAN EXPOSURE

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Introduction

The emerging contaminants, brominated flame retardants (BFRs) have become compounds of interest due to similarities in chemical structure, properties and toxic potencies to some persistent organochlorines (OCs), such as polychlorinated biphenyls (PCBs) and dioxins which are well-known endocrine disrupters. Among BFRs, polybrominated diphenyl ethers (PBDEs) and hexabromocyclododecanes (HBCDs), have been widely detected in the environment and animal tissues/organs and their concentrations seem to be increasing[1], and thus these compounds are of concern for their possible effects on human health. Although the primary route of human exposure is still unclear, humans are most probably exposed to BFRs via food intake, as is the case for OCs. However, due to usage of BFRs in household consumer products and their subsequent presence at high levels in house dust, direct exposure via inhalation and ingestion of particulate-bound BFRs in indoor air/dust in work and/or home environments may also significantly contribute to human exposure[1]. The present study aims to assess the exposure of Indonesian people to BFRs in comparison to PCBs by 1) determining concentrations in exposure media such as soil, house dust and food, 2) examining body burdens based on measurements of the contaminants in breast milk, and 3) estimating the average intake values for adults and children.

Materials and methods

Various environmental/exposure media such as surface soil (*n*=4), house dust (*n*=11), shellfish (*n*=5), freshwater fish (*n*=5), marine fish (*n*=20), beef (*n*=3), poultry (*n*=2), egg (*n*=1) and dairy products (*n*=3) as well as human breast milk (*n*=40) were used in this study. Human breast milk samples were collected from three locations representing urban (Jakarta), suburban (Bogor) and rural areas (Purwakarta and Lampung) in Indonesia during 2003-2007, whereas other samples were mainly collected from Jakarta and Bogor. Furthermore, available data in the literature[2,3] were also used in this study.

Analysis of BFRs and PCBs was performed following methods described elsewhere[3], with slight modification depending on sample matrices. Fourteen PBDE congeners (BDE-3, -15, -28, -47, -99, -100, -153, -154, -183, -196, -197, -206, -207, -209), HBCDs (α-, β- and γ-HBCD) and 62 PCB isomers were measured in this study. Using the labeled internal standards as surrogates and QA/QC procedures described previously[3], quantification of PBDEs and PCBs was carried out by a gas chromatograph coupled with a mass spectrometry detector (GC-MS) in the negative chemical ionization mode, and by a liquid chromatograph with a tandem mass spectrometry detector (LC-MS-MS) using electrospray ionization for assessing the isomeric composition of HBCDs.

The exposure pathways evaluated included inhalation, soil/dust dermal contact, soil/dust and food ingestion. For estimation of daily intake of contaminants, we used the exposure factors for non-diet scenarios following the US-EPA approach[4], food consumption data from WHO[5] and breast milk lactation rate for infants from Sudaryanto et al[3].

Results and discussion

Human body burden and exposure media concentrations

BFRs and PCBs were detected in breast milk and various environmental exposure media (Table 1), suggesting wide environmental contamination and human exposure to these compounds in Indonesia. PCBs, which have longer history and larger amount of usage than BFRs[3], were found at higher concentrations than PBDEs and HBCDs in all

the samples, except for house dust. Higher concentrations of BFRs than PCBs in house dust indicate that home environment can be one of the important human exposure sources to PBDEs and HBCDs. Among BFRs, levels of PBDEs were higher than those of HBCDs, concomitant with the difference in their usage. For instance, mean value of ΣPBDEs (2.0 ng/g lipid wt.) in human breast milk was 5 times higher than ΣHBCDs (0.43 ng/g lipid wt.). ΣPBDE concentrations in human milk in the present study are comparable to those of general population of Asia and Europe, but one or two orders of magnitude lower than in North America[1,3,4], a region which consumed much of technical penta-BDE. With respect to HBCDs, few studies have reported the body burden in humans. The levels found in the human breast milk of the present study were similar to the countries such as Japan (1.8 ng/g lipid wt.)[6], Sweden (0.42 ng/g lipid wt.)[7], and Mexico (1.2 ng/g lipid wt.)[7].

Table 1. Concentration of PBDEs, HBCDs and PCBs (ng/g lipid wt.) in human breast milk and various environmental exposure media in Indonesia*

	Breast milk	[1]Air	[2]Soil	[2]House Dust	Shellfish	Marine Fish	Freshwater Fish	Beef	Chicken	Dairy Products	[3]Egg
n	40	2	4	11	5	20	5	3	2	3	1
Lipid (%)	2.1 (0.56-4.9)	-	-	-	1.5 (1.2-1.9)	3.3 (0.61-8.5)	7.6 (5.3-11)	3.6 (1.8-5.9)	5.1 (3.3-6.9)	24 (1.0-44)	7.9
BDE-3	nd	-	0.0072 (nd-0.023)	nd	nd	nd	nd	nd	nd	nd	nd
BDE-15	0.023 (nd-0.11)	nd	0.0053 (nd-0.010)	0.054 (0.020-0.090)	0.038 (nd-0.10)	0.24 (nd-3.2)	0.17 (0.10-0.31)	0.011 (nd-0.034)	nd	0.031 (0.0025-0.082)	0.0033
BDE-28	0.054 (nd-0.34)	2.6 (nd-5.2)	0.0046 (0.0019-0.0075)	0.18 (nd-0.55)	0.14 (0.080-0.19)	0.44 (nd-4.5)	0.60 (0.27-0.99)	0.015 (nd-0.045)	nd	nd	nd
BDE-47	0.37 (0.090-2.1)	11.45 (10-13)	0.10 (0.063-0.13)	1.2 (0.18-3.8)	1.8 (0.78-2.9)	11 (0.12-130)	11 (6.4-19)	0.40 (0.060-0.72)	0.38 (0.19-0.57)	0.082 (nd-0.18)	0.033
BDE-99	0.16 (nd-0.84)	0.85 (0.20-1.5)	0.12 (0.079-0.15)	1.3 (0.29-4.1)	1.3 (0.49-2.2)	5.3 (0.023-72)	2.5 (0.050-6.6)	0.34 (nd-0.57)	0.52 (0.27-0.77)	0.023 (nd-0.045)	0.33
BDE-100	0.14 (nd-0.65)	nd	0.030 (0.018-0.043)	0.19 (nd-0.52)	0.42 (0.14-0.66)	1.9 (0.018-19)	1.6 (0.99-2.0)	0.12 (0.087-0.17)	0.098 (0.050-0.15)	0.016 (nd-0.036)	0.062
BDE-153	0.30 (nd-2.7)	nd	0.039 (0.016-0.058)	0.85 (0.29-1.7)	0.018 (nd-0.070)	1.0 (0.018-19)	0.88 (0.31-1.5)	nd	nd	nd	nd
BDE-154	0.026 (nd-0.20)	nd	0.023 (0.0085-0.038)	0.21 (nd-0.21)	0.010 (nd-0.020)	1.2 (nd-11)	2.2 (0.55-3.8)	nd	nd	nd	nd
BDE-183	0.15 (nd-1.4)	nd	0.11 (0.043-0.16)	3.5 (1.3-8.2)	0.028 (nd-0.11)	0.14 (nd-2.0)	0.27 (nd-0.59)	nd	nd	0.010 (nd-0.028)	nd
BDE-196	nd	-	0.061 (0.027-0.082)	1.7 (0.72-3.4)	nd	0.47 (nd-7.4)	nd	0.62 (0.30-0.94)	0.12 (nd-0.24)	0.87 (nd-2.6)	0.086
BDE-197	0.27 (nd-3.2)	-	0.060 (0.0083-0.12)	1.9 (0.86-4.5)	nd	1.4 (nd-23)	0.088 (nd-0.19)	0.071 (nd-0.17)	0.12 (0.042-0.20)	0.076 (.010-0.076)	0.038
BDE-206	0.042 (nd-0.83)	-	0.19 (0.082-0.28)	7.9 (2.3-16)	nd	0.12 (nd-1.4)	0.075 (nd-0.17)	0.12 (nd-0.20)	0.17 (0.051-0.29)	0.17 (nd-0.46)	nd
BDE-207	0.18 (nd-1.5)	-	0.21 (0.083-0.29)	5.9 (2.5-8.8)	0.10 (nd-0.25)	0.081 (nd-1.3)	0.11 (nd-0.30)	0.37 (0.040-0.88)	0.43 (0.22-0.64)	0.30 (0.018-0.77)	0.074
BDE-209	0.23 (nd-3.6)	nd	3.4 (0.88-7.0)	170 (36-390)	0.73 (nd-2.9)	0.19 (nd-2.9)	0.70 (nd-1.7)	1.9 (0.29-4.1)	1.7 (1.0-2.3)	1.8 (0.051-5.0)	0.21
[9]ΣPBDEs	1.2 (0.40-8.2)	15 (14-16)	0.44 (0.24-0.56)	7.7 (3.4-16)	3.8 (1.7-6.1)	22 (0.23-260)	19 (15-25)	0.89 (0.61-1.1)	1.0 (0.51-1.5)	0.16 (0.0025-0.34)	0.43
ΣPBDEs	2.0 (0.49-13)	16 (14-16)	4.4 (1.3-8.2)	190 (46-430)	4.6 (2.0-6.1)	24 (0.47-270)	20 (15-25)	3.9 (1.7-7.3)	3.5 (2.1-4.9)	3.4 (0.10-9.3)	0.84
α-HBCD	0.43 (0.20-1.0)	-	2.4 (0.011-5.9)	17 (3.3-52)	1.9 (0.62-3.3)	4.1 (0.040-39)	4.4 (1.5-7.9)	0.060 (nd-0.095)	0.091 (0.065-0.12)	0.074 (0.019-0.18)	0.11
β-HBCD	nd	-	0.25 (nd-0.69)	3.0 (0.47-12)	nd	0.014 (nd-0.15)	0.27 (nd-0.95)	nd	nd	nd	nd
γ-HBCD	nd	-	1.4 (nd-4.1)	3.5 (0.71-11)	0.12 (nd-0.46)	0.56 (nd-2.0)	1.7 (nd-6.7)	nd	nd	nd	nd
ΣHBCDs	0.43 (0.20-1.0)	-	4.1 (0.013-11)	23 (5.7-75)	2.0 (0.62-3.8)	4.7 (0.068-41)	6.4 (1.6-16)	0.060 (nd-0.095)	0.091 (0.065-0.12)	0.074 (0.019-0.18)	0.11
ΣPCBs	29 (8.4-62)	40 (28-51)	-	11 (1.5-21)	160 (96-210)	330 (9.7-1100)	110 (21-150)	170 (2.2-520)	2.1 (1.8-2.3)	51 (1.5-110)	6.6

*Note: [1]pg/m3 (Wurl et al., 2006), [2]ng/g sample, [3]pooled samples, [9]ΣPBDEs= total PBDEs from mono-hepta congeners, -= not analyzed, nd=not detected

Concentrations of ΣPBDEs and ΣHBCDs in house dust ranged from 46-430 ng/g dust (mean: 190 ng/g dust) and 5.7-75 ng/g dust (mean: 23 ng/g dust), respectively; which were one or two orders of magnitude higher than outdoor surface soils (4.4 ng/g soil and 4.1 ng/g soil, respectively), implying that indoor environment was more contaminated by BFRs than outdoor. Outdoor air was also contaminated by PBDEs and PCBs as represented by their detection in coastal areas of Java Island[2], with concentrations at two locations being 14-16 pg/m^3 for PBDEs and 28-51 pg/m^3 for PCBs. Among the food items, concentrations of ΣPBDEs, ΣHBCDs and ΣPCBs were higher in fish, whereas shellfish, meat, eggs and dairy products were one or two orders of magnitude lower (Table 1).

PBDE congener profiles and implications of exposure sources and pathways

Except for BDE-3 and -196, all the remaining congeners were identified in human milk of the present study (Table 1), indicating that general population of Indonesia might have been exposed to commercial penta-, octa- and deca-BDE sources. The availability of all these PBDEs formulations was confirmed by the profiles seen in soils and house dust, the two matrices considered as representing original sources and containing all markers of technical mixtures[8] (Table 1). The dominance of BDE-209 in soil and house dust (>80% of ΣPBDEs) indicates that the presence of PBDEs in Indonesian environment is mainly caused by deca-BDE commercial mixtures, consistent with the high consumption of deca-BDE in Asia[1].

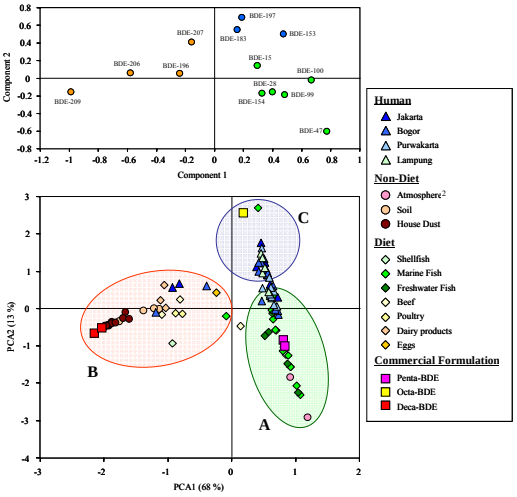

Fig. 1. Principal component analysis of PBDE congener profiles in human, environment, food products and commercial mixtures

To delineate the potential exposure pathways, congener profiles in humans were further examined using principal component analysis (PCA) against compositions of PBDEs in diet, non-diet and available commercial formulations[8] (Fig. 1). After the factor-loading plot, the PCA showed three clusters (A, B and C) of variables that had larger loading for each factor. Most congener profiles in human breast milk (cluster A) were close to fish and penta-BDE formulation (Fig. 1), which is characterized by tetra- to penta-BDE congeners (Table 1, Fig. 1), suggesting fish as an important source of PBDEs and high bioavailability of lower brominated congeners. Food is generally the major source of lower brominated BDEs, from tetra- to penta-BDEs[9]. Furthermore, some donors also showed high percentage of higher brominated congeners (Table 1), ranging from hexa- to deca-BDE. In cluster C, congener profiles were dominated by BDE-206, -207 and BDE-209 which is similar to dust/soil, meat and dairy products. The similarity between PBDE congener profiles in some human milk and dust samples suggest that non-dietary exposure pathways may also be an important route of exposure. It has also been hypothesized that dust/soil ingestion are possible routes of PBDEs exposure in humans[1]. Profiles of human samples in cluster C were characterized by higher brominated congeners (BDE-197, -183, -153) and did not resemble the profiles in environmental media, indicating exposure to deca-BDE that has been environmentally/biologically degraded (such as debromination) resulting in the observed profile. It has been shown in animal studies that BDE-209 can be debrominated into BDE-183 and, subsequently, BDE-153[10]. The large number of samples containing BDE-183 and located close to the octa-BDE mixtures (cluster C) may also suggest additional exposure from octa-BDE formulation.

Intake estimation

Figure 2 shows the estimated intakes of HBCDs, PBDEs and PCBs by adults and infants, considering various exposure pathways. The total intake of BFRs, PBDEs (52 ng/day) and HBCDs (9.8 ng/day) by adults were lower than PCBs (560 ng/day). This intake pattern agreed with the body burdens estimated from breast milk concentrations in which the levels of BFRs were much lower (>10 times) than PCBs (Table 1). In the case of PCBs, dietary intake was almost the only source to human (~100 %), particularly the high intake was from fish and shellfish (~95%) and low from meat (~3%), dairy products (1.3%) and others (<1%). The contribution of non-dietary intake, such as inhalation, soil/dust dermal contact and ingestion, to human exposure to PCBs was negligible (<0.20 %). In contrast, in case of BFRs, non-dietary sources also significantly contributed to human exposure (accounting for about 12% and >17% to the total daily intake of PBDEs and HBCDs, respectivel), particularly significant proportion of BFR intake was from soil/dust ingestion (Fig. 2). Food sources still contributed a large proportion to the total daily intake of BFRs (>80%), especially fish and shellfish (~75% for PBDEs and ~85% for HBCDs). Among the congeners and isomers, BDE-47, -209, -99, -100, -153, -154 and α-HBCD contributed the most to the total PBDEs and HBCDs intake, respectively, with fish and dust being the most important sources. Fish was a major contributor of lower brominated congeners, whereas higher brominated congeners, particularly BDE-209, came from dust (Fig. 2).

Generally, the intake estimation of total PBDEs was based on few target congeners from tetra- to hexa-BDE[11, 12, 13, 14], but to some extent up to octa-[9] and deca-BDE[4]. In this study, the average daily dietary intake of PBDEs by Indonesians was 38 ng/day and 42 ng/day for the sum from mono- to hepta-BDE and to deca-BDE, respectively (Fig. 2), which was similar to that in Sweden (41 ng/day)[11] and Finland (44 ng/day)[12], but lower than in Japan (73 ng/day)[13], Spain (82 ng/day)[9], UK (91 ng/day)[14] and USA (90 ng/day)[4]. The total intake of PBDEs (52 ng/day) was also lower than that for UK (110 ng/day)[14] and USA (540 ng/day)[4]. However, the contribution of dust ingestion to the total intake of PBDEs (16%) was close to that in UK (19%)[14]. In USA, the intake via dust ingestion was much larger and accounted for ~66 % of the total intake[4].

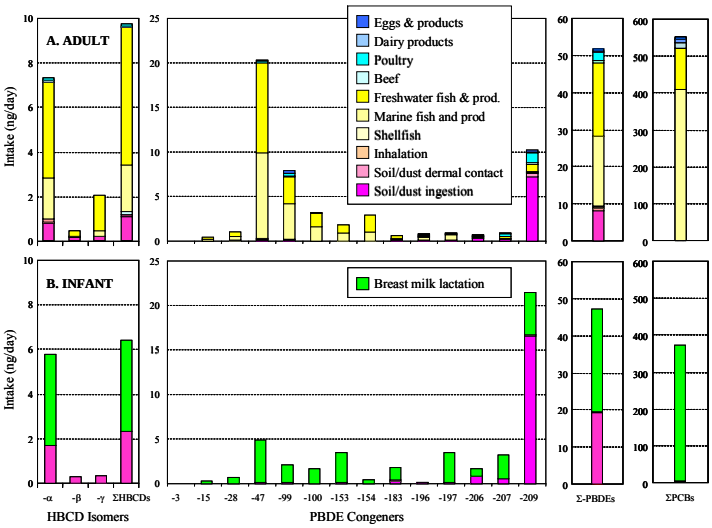

Fig. 2. Daily intake of BFRs and PCBs by adults and infants

The exposure pathways of adults to BFRs and PCBs were also compared with that of infants who are assumed to have intake only during a short period (<2 years) from breast milk and dust ingestion/dermal contact (Fig. 2). Although the total daily intake of BFRs by infants was comparable to adults, the contribution from dust ingestion to the total intake was much greater than that for adults accounting for 41 % for PBDEs and 36 % for HBCDs. In contrast, infant exposure to PCBs was mainly from breast feeding (~100%). Total PBDE intake by infants was mainly contributed by BDE-209, which was different in case of adults (Fig. 2).

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