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Polybrominated diphenyl ethers and synthetic musks in umbilical cord Serum, maternal serum, and breast milk from Seoul, South Korea

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ABSTRACT

Fetal and maternal exposure levels of two emerging pollutants, polybrominated diephenyl ethers (PBDEs) and synthetic musks, were measured in Korean general population to assess prenatal and postnatal exposures in infants. For this purpose, paired samples of breast milk, maternal and cord blood were collected from 20 Korean women in 2007. In comparison to data from other countries and previous data from Korea, relatively higher and gradually increasing concentrations for PBDEs were found in Korean breast milk (<LOQ to 590 ng g^{-1} lipid wt; median = 90 ng g^{-1}). Differences in PBDEs and musk concentrations were found among age groups and parity levels. PBDEs concentrations in breast milk were lower in the younger mothers and/or the mothers with multiple parities, while these trends were not found for musks. Compared with PBDEs, concentrations of musks were significantly lower in breast milk than in serum and a little correlation in concentrations among the three human biological matrices were observed. The differences in the profiles of musks relative to PBDEs were due to different clearance rates between these two compounds. The average hazard quotients (HQs) for daily intake of PBDEs by infants via lactation were 0.62, 0.42, and 0.19 for BDE-47, BDE-99, and BDE-153, respectively.

1. Introduction

Emerging contaminants such as PBDEs have received attention due to their persistent, bioaccumulative, and toxic potentials (i.e., PBT nature) (Hardy et al., 2003; Law et al., 2006). Contrary to legacy persistent organic pollutants (POPs) such as organochlorines, the levels of these emerging pollutants continue to increase in the environment (Norén and Meironyté, 2000; Hites, 2004; Wang et al., 2007). Penta- and octa-BDEs have been shown to affect the thyroid hormone homeostasis (Zhou et al., 2002). Furthermore, it has been shown that exposure to BDE-47 and -99 during the critical neonatal period caused neurotoxic effects in adult mice (Eriksson et al. 2001). Polycyclic musks are another class of environmental pollutants to the great extent general population can be exposed through the use of consumer products. In recent years, concerns have been raised on estrogenic activity and carcinogenicity of polycyclic musks (Bitsch et al., 2002; Brunn et al., 2004). Thus, exposure to these emerging pollutants during the sensitive period in development could pose a potential risk for human health.

PBDEs, comprising of penta-, octa-, and deca-BDE products, are widely used as flame retardants, especially in household products such as electronics/electrics equipments, textiles, paints, and furnishings (www.bsef.com). PBDEs have been used as a popular brominated flame retardant (BFR) until a ban and/or restriction on the use of penta- and octa-BDE mixtures has been proposed in many countries. Despite increase in regulation and decline in consumption worldwide (DEPA, 1999; Renner, 2000), the BFR market in South Korea, sharing ~56% of flame retardant market, has increased annually by 13.5% on the average. Domestic brominated flame retardants (BFR) consumption in South Korea in 2002 was 49,050 tons, of which 25% (i.e., 12 408 tons) was estimated to be PBDEs with deca-BDE accounting for 12 324 tons (Kim et al., 2007).

Synthetic musks are used as ingredients in fragrances for consumer products such as perfumes, cosmetics, soaps, shampoo, detergents, fabric conditioners, cleaning products, and air fresheners as an alternative for the natural musk (HERA, 2004). The production of polycyclic musks such as 7-acetyl-1,1,3,4,4,6-hexamethyl-1,2,3,4-tetrahydronaphthalene (AHTN) and 1,3,4,6,7,8-hexahydro-4,6,6,7,8,8-hexamethylcyclopenta-γ-2-benzopyran (HHCB) has

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increased since 1950, with a corresponding decline in production of nitro-musks such as musk ketone (4′-tert-butyl-2′,6′-dimethyl-3′,5′-dinitroacetophenone, MK), musk-xylene (1-tert-butyl-3, 5-dimethyl-2,4,6-trinitrobenzene, MX) and musk-moskene (1,1,3,3,5-pentamethyl-4,6-dinitro-2H-indene, MM) due to their potential toxic effects (Rimkus, 1999; Peck and Hornbuckle, 2004). In 1996, the worldwide production of polycyclic musks, 90–95% of which were HHCB and AHTN, was 5600 tons (Rimkus, 1999). A substantial amount from a few mg g^{-1} to a few percent of musk compounds were reported to be impregnated in consumer products (HERA, 2004; Reiner and Kannan, 2006; Roosens et al., 2007). There is no domestic report on musk consumption and/or emission in South Korea.

A recent meta-analysis of POP contamination in Korea suggested that emerging pollutants could pose more serious issues than the legacy POPs because of rapid economic growth in recent decades promoted the consumption of these emerging pollutants without any regulations (Kim et al., 2007). However, there is little or no report on the human exposure to PBDEs and/or musks in Korea.

Maternal serum, umbilical cord serum, and breast milk have been used as matrices to biomonitor the extent of human exposure, in particular maternal body burdens as well as pre- and post-natal exposure of neonates, through placenta and lactation (Mazdai et al., 2003; Bi et al., 2006; Gómara et al., 2007). The objectives of this study were to report, for the first time, the levels and accumulation profiles of PBDEs and musks in sera (maternal and umbilical cord) and breast milk from Korean general population and to assess prenatal and postnatal exposure levels to these two pollutants by infants. Maternal sera, umbilical cord sera and milk samples were collected from the same individuals for this analysis. Very few studies have examined such paired sera and milk samples to assess fetal and maternal exposures.

2. Materials and methods

2.1. Study design and sample collection

Twenty volunteers of age >25 years with a median age of 32 years from general population in Seoul, Korea, donated serum, cord serum and breast milk. Mothers' height, weight, age, and number of previous pregnancy, and weight and gender of the baby were recorded at the time of sampling (Table 1 in Supplementary

Table 1
PBDE and synthetic musk concentrations range, (mean/geometric mean); *n* in maternal serum, umbilical serum, and breast milk samples from Seoul, Korea.

Sample ID	Maternal serum	Umbilical cord serum	Breast milk
Sample number	(*n* = 20)	(*n* = 20)	(*n* = 17)
Lipid (%)	0.15–1.1 (0.58/0.51); 20	0.05–0.27 (0.16/0.14); 20	0.63–5.2 (3.08/2.64); 17
PBDEs (ng/g lipid)			
BDE-17	NA	NA	<0.60–4.5 (0.71/0.47); 5
BDE-28	<1.2[a]–8.1 (4.1[b]/0.74[b]); 2[c]	<4.9–28(4.1/3.0); 2	0.90–13(2.1/1.0); 8
BDE-49	NA	NA	0.60–2.4(0.52/0.39); 3
BDE-47	<4.1–60(6.1/3.0); 4	<16–230(36/14); 4	3.6–180(31/21); 17
BDE-66	<3.8(nc[d]/nc); 0	<15(nc/nc); 0	0.6–9.3(1.2/0.51); 4
BDE-100	<3.1–9.8 (2.3/1.9); 2	<12–91 (11/7.6); 2	<1.2–52 (11/4.3); 13
BDE-99	<3.8–20(3.3/2.5); 3	<15–96(19/11); 4	<3.0–280(54/17); 14
BDE-85	<3.4 (nc/nc); 0	<13(nc/nc); 0	0.60–19(3.5/0.81); 5
BDE-154	<3.1–6.0 (2.0/1.8); 2	<12(nc/nc); 0	0.60–27(3.7/0.91); 7
BDE-153	<3.4–49(8.5/4.0); 8	<13–59 (16/11); 7	0.60–39(10/3.1); 12
BDE-138	<2.8(nc/nc); 0	<11 (nc/nc); 0	O.60–4.4 (0.54/0.35); 1
BDE-183	<3.3 (nc/nc); 0	<13(nc/nc); 0	<6.0 (nc/nc); 0
BDE-197	<3.2–120 (11/3.3); 5	<13–96(17/10); 6	<6.0 (nc/nc); 0
BDE-203	<15–26(8.5/8.1); 1	<60–140(35/32); 1	O.90 (nc/nc); 0
BDE-196	<5.2 (nc/nc); 0	<21 (nc/nc); 0	<3.0 (nc/nc); 0
BDE-207	<14–110 (12/8.3); 1	<57–78(33/31); 2	<2.1 (nc/nc); 0
BDE-209	<30–45(18/16); 2	<120(nc/nc); 0	<12–84(13/8.9); 5
ZPBDEs(+209)[e,g]	0.00–270 (37/7.8); 11	0.00–480(89/13); 11	7.6–570 (120/72); 17
EPBDEs(+209)[f,g]	<100[j]–310 (82/71); 11	OgO[1]–610(270/250); 11	<41[j]–590(140/90); 17
£PBDEs(-209)[e,h]	0.00–270 (32/4.5); 11	0.00–480(89/13); 11	7.6–570(110/70); 17
SPBDEs(-209)[f,h]	<70[i]–300(65/52); 11	<280[i]–550(210/190); 11	<27[j]–580(130/78); 17
penta-BDE[f,k]	<7.9–79(9.5/5.5); 4	<31–330 (56/26); 4	5.1–450 (85/42); 17
octa-BDE[f,l]	<34–180 (35/26); 10	<130–190(91/84); 9	<17–74(22/16); 12
synthetic musks (ig/g lipid)			
HHCB	O.17–1.4 (0.50/0.38); 18	O.67–2.7 (0.91/0.71); 14	0.055–0.515 (0.216/0.180); 17
AHTN	<0.17–1.4 (0.30/0.17); 7	<0.67–2.7(0.56/0.43); 3	O.015–0.091 (0.036/0.024); 11
HHCB-lactone	<0.17–0.80 (0.25/0.20); 13	<0.67–2.2 (0.64/0.54); 12	O.015–0.100 (0.016/0.010); 2
Musk-xylene	O.17–0.51 (0.11/0.09); 1	<0.67 (nc/nc); 0	0.015–0.22(0.031/0.018); 9
Musk-moskene	<0.17–0.36 (0.14/0.12); 7	0.67–1.0(0.41/0.38); 3	O.015–0.035 (0.010/0.009); 3
Musk-ketone	<0.17(nc/nc); 0	<0.67 (nc/nc); 0	0.015–0.15(0.057/0.032); 11
total musk[e]	0.00–2.1 (1.1/1.1); 19	0.56–4.9(1.7/1.2); 20	0.055–0.87 (0.35/0.20) "
total musk[f]	<1.01–2.4 (1.4/1.3); 19	2.2–6.2 (3.2/3.0); 20	0.092–0.88 (0.37/0.30); 17

[a] Values prefixed with "<" indicate respective LOQ.
[b] (Mean/geometric mean) were calculated assuming "<LOQ = half LOQ".
[c] The number of samples where concentration was detected over LOQ.
[d] Not calculated because of <LOQ in all samples.
[e] Total PBDEs concentration calculated assuming "<LOQ = 0" for individual compounds.
[f] Total PBDEs concentration calculated assuming "<LOQ = half LOQ" for individual compounds.
[g] Total PBDEs concentration including BDE-209.
[h] Total PBDEs concentration excluding BDE-209.
[i] Median value of total PBDEs on behalf of geometric mean because of including "zero" value.
[j] LOQ of total concentration was calculated from the sum of LOQs of individual compounds in the group. NA = not analyzed.
[k] Penta-BDE mixture = S(BDE-47, and BDE-99).
[l] Octa-BDE mixture = S(BDE-153, -154, -183, -196, -197, -203).

material). Maternal serum was obtained one day before delivery and fetal serum was obtained from the umbilical cord vein immediately after delivery. Breast milk was collected at 3–10 d after the delivery. In this study, 57 individual samples including 20 maternal serum and 20 umbilical cord serum and 17 breast milk samples, were analyzed for PBDEs and synthetic musks. Of the 20 paired samples, three breast milk samples were lost (due to breakage) in storage and 17 paired maternal and cord serum and mother's breast milk samples were analyzed. Samples were kept in pre-cleaned glass bottle at −70 °C until chemical analysis.

2.2. Chemical analysis and QA/QC

Seventeen BDEs including tri- to deca-BDE (BDE congeners, 17, 28, 47, 49, 66, 100, 99, 85, 154, 153, 138, 183, 197, 203, 196, 207 and 209) and six synthetic musks including polycyclic musks (HHCB, AHTN, and HHCB-lactone) and nitro-musks (musk-xylene, musk-moskene, and musk-ketone) were analyzed following the methods described elsewhere (Johnson-Restrepo et al., 2007; Reiner et al., 2007). The detailed procedures are described in Supplementary material. In brief, after passed through gel permeation chromatography (GPC), extracts of milk and serum were further purified by a silica gel packed cartridge for musks and by multi-layered silica gel packed column for PBDEs. Tri- to hexa-BDE and octa- to deca-BDE congeners were determined using GC-MSD with 15-m Restek Rxi-5 ms column and GC-ECD with 5-m DB-5 ms column, respectively. Musks were determined by GC-MSD with 30-m Restek Rxi-5 ms capillary column.

Recoveries of surrogate standards were acceptable within 70–110% for of ^{13}C-labeled PBDE congeners and 75–135% for d_3-AHTN. The concentrations lower than three times the standard deviation of blank measurements are reported as below the limit of quantitation (LOQ). The LOQs in serum and milk were 7.3–175 pg g^{-1} (wet weight) and 20–200 pg g^{-1}, respectively, for PBDE congeners, and 1 ng g^{-1} and 0.5 ng g^{-1}, respectively, for musks.

3. Results and discussion

Concentrations of individual compounds and detection frequencies are presented in Table 1. In this study, two average concentrations are provided based on the treatment of LOQ values; first as <LOQ = zero (i.e., where the concentrations below LOQ were treated as zero) and the second as <LOQ = half LOQ value (where the concentrations below LOQ were assigned half the LOQ value). The concentrations are presented on a lipid weight basis unless otherwise mentioned. The median lipid contents were 0.59% (range 0.15–1.09%) in maternal serum, 0.15% (0.05–0.27%) in cord blood, and 3.33% (0.63–5.15%) in breast milk.

3.1. PBDEs in sera and breast milk

High concentrations for both individual congeners and total PBDEs were detected in Korean breast milk samples (Table 1). As for BDE-47 which was detected above LOQ in all of 17 breast milk samples, 15 samples contained levels over 10 ng g^{-1}. The total concentration of tri- to deca-BDE (Σtri-decaPBDEs) in breast milk ranged from 26.4 to 586 ng g^{-1} with a median of 90 ng g^{-1} (when <LOQ was treated as half the LOQ value). This value is comparable to (range = 7.60–570; median = 72.2) when <LOQ was treated as zero, indicating that the median values are not significantly affected by the treatment of LOQ values for breast milk samples.

Of the sera samples analyzed, four maternal sera and four cord sera contained BDE-47 concentrations above the LOQs (4.1 and 16 ng g^{-1}, respectively) (Table 1). Similarly, the number of samples with BDE-100, −99 and −153 detected was 2, 3, and 8, respectively, in maternal serum, and 2, 4, and 7, respectively, in umbilical cord serum. BDE-47 (100%), 100 (76%), 99 (82%) and 153 (71%) were abundant in breast milk but were rarely found in serum (Fig. 1). The low detection frequency of PBDE congeners in sera can be explained by not only high LOQs but also relatively low lipid content in sera. LOQ values in the present study are one order of magnitude greater compared with other studies (Hites, 2004; Frederiksen et al., 2009; references in Fig. 4). Thus, the detection of PBDE congeners in low lipid-containing serum requires much greater sensitivity or higher sample volume.

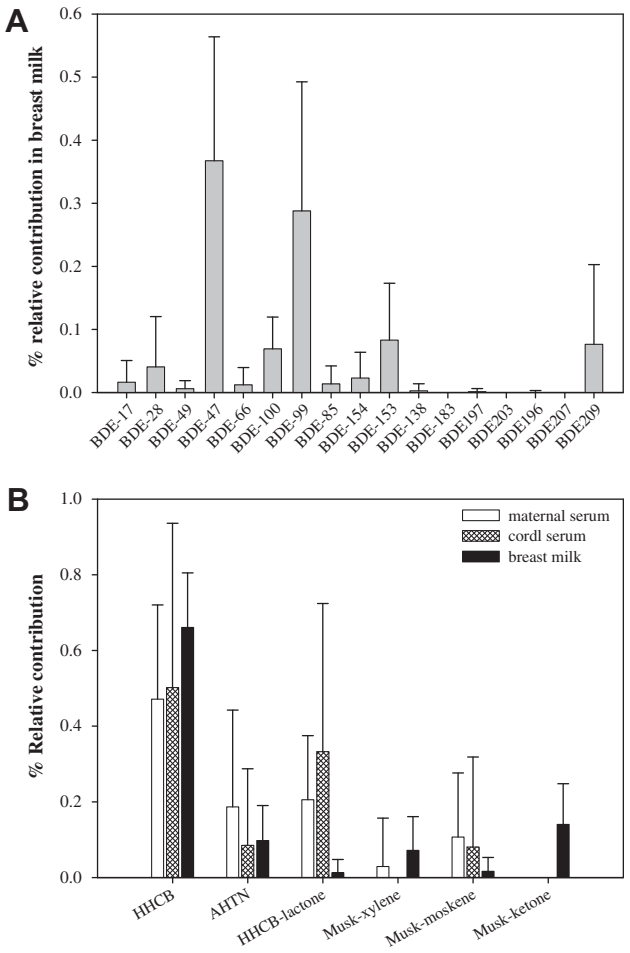

Fig. 1. Relative contribution of individual PBDEs (A) and musks (B) to corresponding total concentrations. The contribution was calculated by substituting <LOQ with 0.

Median concentrations of Σtri-decaPBDEs (<LOQ = 0) were 7.8 ng g^{-1} in maternal serum and 13 ng g^{-1} in umbilical cord serum. When the <LOQ was treated as half the value of LOQ, the median Σtri-decaPBDEs was 56 ng g^{-1} in maternal serum and 200 ng g^{-1} in umbilical cord serum, which is similar to that in breast milk. The lipid content normalized levels were frequently reported to be comparable or within a factor of two between maternal and breast milk (Mazdai et al., 2003; Meironyté et al., 2003; Bi et al., 2006). Therefore, it is more appropriate for values <LOQ be treated as half LOQ rather than zero.

3.2. Synthetic musks in sera and milk

Polycyclic musks comprised over 60% of the concentration of total synthetic musks in sera and milk (Table 1). Concentration of total polycyclic musks was <508–2140 ng g^{-1} (median = 915) in maternal serum,<2000–5220 ng g^{-1} (1660) in umbilical cord

serum, and 69.9–686 ng g^{-1} (199) in breast milk. HHCB was the predominant compound with a contribution of 30% in sera and 60% in breast milk to total musk concentrations. The second dominant musk was AHTN and HHCB-lactone. Although the concentration of AHTN was two-fold higher than that of HHCB in 20% of serum where both compounds were detected simultaneously, most sera (30 of total 40 sera) did not contain detectable levels for AHTN. In breast milk, HHCB had seven-fold greater level, on average, than AHTN. There is no information about use or consumption of musks in Korea. According to historical use volume inventory of musks in Europe, HHCB usage is about three times greater than AHTN (HERA, 2004). Furthermore, lipophilicity of HHCB (i.e., octanol–water partition coefficient (K_{ow})) is 1.6-fold higher than AHTN (HERA, 2004). Thus, a discrepancy of internal exposure between the two compounds is attributable to the differences in use volume and physico-chemical properties.

The most frequently detected musk was HHCB (90% in maternal serum, 70% in umbilical cord serum, and 100% in breast milk) followed by HHCB-lactone (>60%) in maternal and cord serum while AHTN (65%) was frequently detected in breast milk (Fig. 1). Three nitro-musks were rarely detected in serum samples, whereas in over half of the breast milk samples musk-xylene and musk-ketone were detected above the LOQ. The levels of HHCB were not correlated among maternal serum, umbilical cord serum, and breast milk (Spearman rank sum test; $p > 0.3$ for all cases). However, a strong correlation was observed between HHCB and its oxidation product, HHCB–lactone, in maternal serum (Spearman's rho = 0.82, $p < 0.01$) and umbilical cord serum (Spearman's rho = 0.86, $p < 0.014$) only when pairs with detectable concentrations were tested. The average lipid-normalized concentrations of HHCB and AHTN were 2–10 times higher in maternal serum than in breast milk (Wilcoxon test; $p = 0.019$, $n = 14$ for HHCB and $p = 0.068$, $n = 4$ for AHTN).

Lack of correlation of HHCB concentration among the three different matrices could be explained by the routes of exposure. Dermal exposure is thought to be a major pathway of human exposure to musks (Reiner and Kannan, 2006). It is also possible that compound-specific metabolism and excretion occurs in humans. Musk-xylene is excreted with an half-life of 63–107 d through urine as an amine metabolite via hydrolysis and reduction (Kokot-Helbling et al., 1995; Riedel and Dekant, 1999). Too fast excretion of musks to distribute between blood and other tissues such as breast milk could bring about a relatively low concentration in breast milk than in serum.

3.3. Comparison between PBDEs and musk

Direct comparison of PBDE concentrations among maternal serum, cord serum, and breast milk was not possible due to the low detection frequency in serum samples. Nevertheless average lipid-normalized concentrations of BDE congeners were slightly higher in breast milk than sera samples (Table 1). As for HHCB, AHTN, and HHCB-lactone, significantly greater concentrations were found in maternal serum or cord serum than in paired breast milk, and further in maternal serum than in paired cord serum (Wilcoxon test; $p < 0.05$ for each case). This implies that relative importance of exposure pathway to neonates is different between the two pollutant groups studied; that is, prenatal exposure through placenta could be a major pathway for musk exposure in infants while postnatal exposure through lactation can be a major pathway for PBDEs exposure in infants. Apparent differences were further observed between PBDEs and musk distribution in breast milk. An opposite trend with maternal age was observed for PBDEs and musk concentrations (Fig. 2). Breast milk from younger mothers contained greater concentrations of PBDEs. Further, an apparently increasing trend with age was observed for HHCB. Second, the number of parity seemed to have a different effect on PBDEs and musk concentration

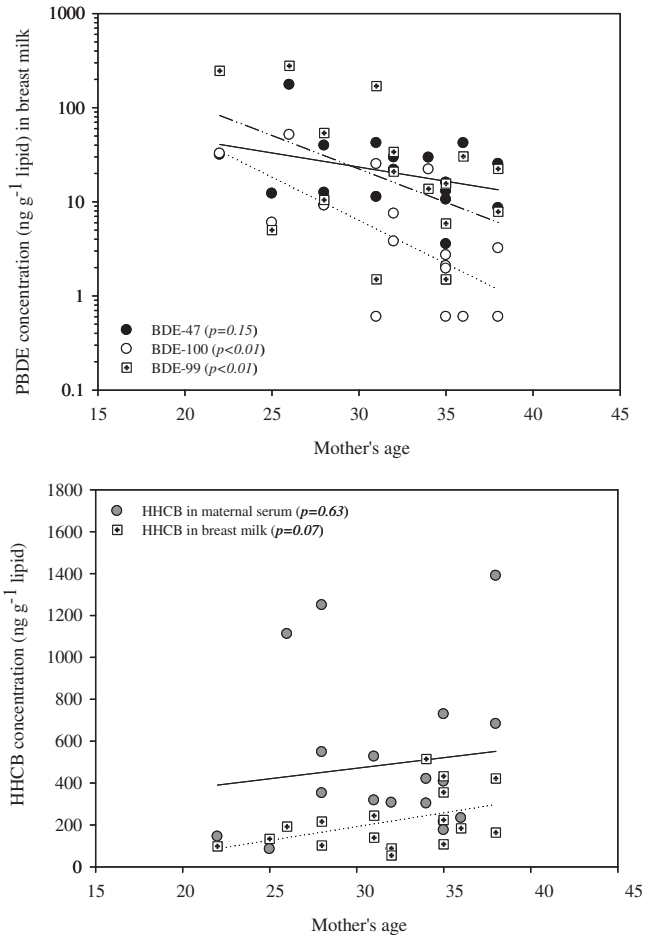

Fig. 2. Relationship between concentrations of PBDEs/HHCB in mother's milk/maternal serum and age.

(Fig. 3). Mothers who had previously nursed two babies contained the significant lower PBDEs in milk than did those who nursed one or non (Mann–Whitney U test; $p < 0.01$ for BDE-47 and 100, and $p = 0.06$ for BDE-99). However, no such relationship with parity was observed for HHCB ($p = 0.35$). Furthermore, there was no correlation between PBDEs and musks, although strong correlations were found among some musk compounds as well as among PBDE congeners in breast milk (Spearman rank correlation test; Table 2 in Supplementary material).

These findings suggest differences in exposure pathways and elimination rates of PBDEs and musks in the body. Earlier studies reported shorter half-lives in human body for musk-xylene than PBDEs; 63–107 d for musk-xylene (Kokot-Helbling et al., 1995) versus 19–78 months for PBDE congeners (Geyer et al., 2004), indicating that human body residues reflect relatively recent exposure for musks. The difference in elimination rate also means that age-related accumulation can be different between the two contaminant groups; for example, musks with 2–3 months half-life can be more efficiently and quickly eliminated by urinary excretion rather than lactation compared with PBDEs with several months to years of half-lives in the body. Consequently, age-related differences and parity effect are not likely observed for compounds that are metabolized and eliminated quickly from the body. Dermal absorption is known to be main exposure pathway of musk (Hawkins et al., 2002). Several personal care products such as perfumes and lotions, containing HHCB and AHTN at up to a few percent of product, are directly applied on skin (Reiner and Kannan, 2006; Roosens et al., 2007). Therefore, musk exposure could depend on

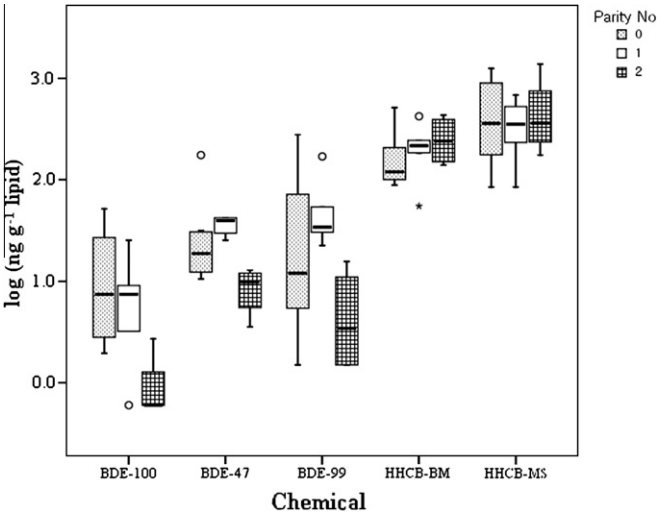

Fig. 3. HHCB concentration in maternal serum (MS) and breast milk (BM) and PBDE concentration in breast milk with the number of previous pregnancies (n = 9 for '0', n = 5 for '1', n = 4 for '2'). Horizontal lines within the box indicate median value.

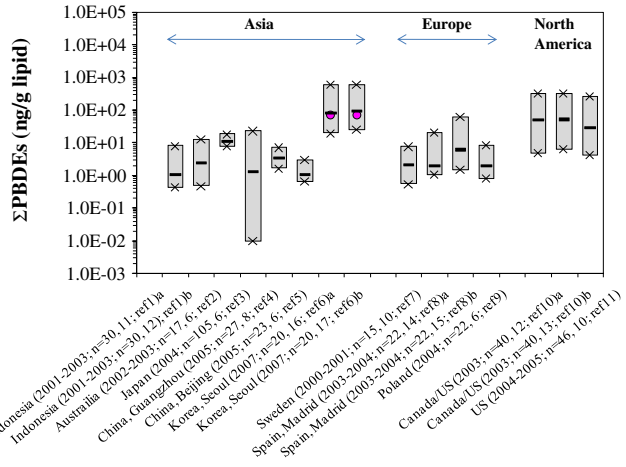

Fig. 4. Median (horizontal line within the bar) and range (upper and lower limit of the bar) of ΣPBDE concentrations measured in breast milk samples worldwide. The lines and filled circles in two Korea's bars indicate the median values with <LOQ as half LOQ value and with <LOQ as 0 for individual congeners, respectively. The values with 'b'- include BDE-209 additionally to the values with 'a'-mark (ref1 – Sudaryanto et al., 2008; ref2 – Tomes et al., 2007; ref3 – Eslami et al., 2006; ref4 – Bi et al., 2006; ref5 – Li et al., 2008; ref6 – this study; ref7 – Meironyté et al., 2003; ref8 – Gómara et al., 2007; ref9 – Jaraczewska et al., 2006; ref10 – She et al., 2007; ref11 – Wu et al., 2007).

the amount and/or a kind of personal care products, which is individual-specific. Compared with musk, the ingestion of diet or dust could be major contributor to PBDE exposure (Frederiksen et al., 2009; Johnson-Restrepo and Kannan, 2009). Whereas legacy chlorinated POPs showing a positive correlation of concentration with maternal age (Sudaryanto et al., 2008), most reports for PBDEs present little or no the age-dependent correlation (Thomsen et al., 2002; Sjödin et al., 2003; Thomas et al., 2006; Eslami et al., 2006). However, these findings should be carefully discussed, considering that the significant age-dependent trend disappeared for both PBDEs and musks when compared within a group with the same parity number for both PBDEs and musks ($p > 0.1$), and that significantly low PBDE concentration in mothers having two previous parity can be confounded by age effect because they were over the age of 35 for three of four mothers. This might be partly a small sample size studied.

3.4. Comparison with other studies

Both BDE-47 and -99 were predominant and constituted 37% and 29% of ΣPBDEs determined in breast milk, respectively (Fig. 1). The observed contribution of BDE-47 is comparable to or slightly lower than those in most previous studies (Hites, 2004; Frederiksen et al., 2009). It is worth to note that BDE-209 was detected in 2 of 10 maternal sera and 5 of 17 breast milk and accounted for over 50% and 22% of total PBDEs, in those samples, respectively. Other octa- and nona-BDE were detected in maternal serum and cord serum (see Table 1). BDE-197 was detected at 20 ng g^{-1} in five maternal and six cord sera. Metabolic biodegradation of BDE-209 to lower brominated BDEs is a possible explanation for the presence of other higher brominated congeners (Kierkegaard et al., 1999). Detection of octa- and nona-BDE without BDE-183 could be an evidence of BDE-209 metabolism in human body. BDE-209 has been analyzed in relatively few other studies; contribution of BDE-209 to total PBDE concentrations varied from 1% in North American breast milk (She et al., 2007) to >50% in Spanish breast milk (Gómara et al., 2007).

Concentrations of PBDEs measured in the general population of this study are higher than those reported in several Asian and European countries (Fig. 4). Noticeably, the concentration of PBDEs in Korean breast milk was comparable to or slightly higher than that of North America which has been known to be one order of magnitude greater than those of other Continents (Hites, 2004; Wang et al., 2007). One previous study (Lee et al., 2007) reported PBDEs concentration (excluding BDE-209) in Korean blood; 92 Korean sera collected in 2001 from 30 incinerator workers, 51 residents near incinerators, and 11 reference individuals from other small city. The observed concentration in that study was 5.83–47.2 ng g^{-1} lipid wt. (median = 17) and there was no significant difference among the three population groups. So, the concentration in reference group or all the groups of that study can be compared with our population. The median based lipid-normalized PBDE concentration (excluding BDE-209 and < LOQ = 0) was fourfold higher in our population.

Very few studies have reported musks in human milk. HHCB and AHTN concentration of <5–917 ng g^{-1} lipid (median = ~130) and <5–144 ng g^{-1} lipid (~50) was measured in human milk from Massachusetts, USA (Reiner et al., 2007). Similar concentrations were also found in Danish human milk, with a median concentration of 147 ng g^{-1} for HHCB, 17.5 ng g^{-1} for AHTN, 14.9 ng g^{-1} for musk-ketone, and 9.44 ng g^{-1} for musk-xylene (Duedahl-Olesen et al., 2005). The musk concentrations in Korean breast milk were similar to those reported for other countries.

3.5. High PBDEs exposure in Koreans

The high PBDE concentration in this study was fairly well predicted from levels of control group in 2001 by an exponentially increasing curve suggested by the previous studies (Hites, 2004). In Korea, personal computers were available in 60% of the houses in 2001 and increased to 84% of the houses in 2007. According to a national surveillance on human activity, Korean house-wives spend ~17 h a day in indoor environments and use computer for over 10 h per week. Increasing use of electronic/electric equipments and consequent contamination of indoor environment may be a plausible reason for high a PBDE exposures in Korean mothers. However, there is no report for PBDEs in indoor environment in Korea. Another source of exposure is consumption of PBDE-contaminated fish. Among biological samples, fishes are known to contain high levels of PBDEs (Hites, 2004; Frederiksen et al., 2009). Korea is one of the major consumers of fishes/shellf-

ishes in the diet (86 g d^{-1} per person). Thus, fishery product intake combined with indoor contamination could contribute to considerable exposure to PBDEs exposures for the Koreans.

We estimated daily intakes of infants via breast milk ingestion using daily breast milk consumption rate of the first 5 months (123 ± 44 g kg bw^{-1} d^{-1}) (Lee et al., 1997). Average estimated daily intakes were 124 ng kg^{-1} d^{-1} for BDE-47, 210 ng kg^{-1} d^{-1} for BDE-99, 42 ng kg^{-1} d^{-1} for BDE-100, 39 ng kg^{-1} d^{-1} for BDE-153, 724 ng kg^{-1} d^{-1} for HHCB, and 122 ng kg^{-1} d^{-1} for AHTN. We also calculated hazardous quotients (HQs) for PBDEs, by dividing the estimated daily intake (EDI) by the corresponding RfD values for no-observed-adverse, non-carcinogenic effects (USEPA, 2006): 0.2 μg kg^{-1} d^{-1} for BDE-47 and BDE-153 and 0.1 μg kg^{-1} d^{-1} for BDE-99. A HQ value less than unity indicates that the chemical is less likely to pose a significant health risk to the exposure group (infants). Average HQs of PBDEs in the infant population were 0.62, 0.42, and 0.19 for BDE-47, 99, and 153, respectively. The calculated HQ for PBDE congeners were similar with values reported for Massachusetts infants (Johnson-Restrepo et al., 2007). Toxic effects of synthetic musks are not clearly understood and further studies are needed on this issue.

4. Conclusions

Our findings indicate that women in Seoul are exposed to relatively high levels of PBDEs and therefore, infant exposure could be high. Apparent differences in age- and parity-related increase in concentrations, and distribution of concentrations among the three human biological matrices (maternal blood, cord blood and breast milk) were found between PBDEs and musks. PBDE concentrations were higher in younger mothers and in mothers nursing their babies for the first time. Compared with PBDEs, musk concentrations are significantly lower in breast milk than in serum. These differences in the profiles and patterns of musk and PBDEs in human tissues could be due to different metabolism and excretion rates in the body. As a result, a relative importance of prenatal exposure via placenta and postnatal exposure through lactation does not likely the same for PBDEs and musks. Due to the small sample size, these comparisons are made with caution and further investigation into exposure and body burden is needed before further generalizations can be made. The results of this preliminary study suggest that further, long-term and large-scale studies will be required to assess exposure levels in a broader population, to identify the exposure sources in Korea, and to examine possible adverse effects associated with high levels of exposure during fetal development and nursing.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.chemosphere.2010.04.009.

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