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Special Distribution of Polybrominated Diphenyl Ethers in Brain Tissues of Free-range Domestic Hens and Ducks from a Village near an Electronic Waste Recycling Site in South China

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Abstract The rural village, Taizhou of Zhejiang Province, had been exposed to e-waste recycling for years, the polybrominated diphenyl ether (PBDE) levels in hens and ducks were high. The concentration of $\sum$PBDEs in the brain was the lowest among tissues of individual hens and ducks after correction for the respective lipid content. Also, the concentration ratio of BDE-153 versus BDE-154 (153/154) of brain was the highest among tissues of individual hens and ducks. Our results indicate that the hindrance of blood–brain barrier to compounds, such as high molecular weight and non-planar conformation (steric hindrance), contributed to the low concentration of PBDEs in the brain tissue of hens and ducks, especially in cases exposed to high levels of PBDE.

Keywords Polybrominated diphenyl ether · Brain tissue · Hen · Duck

Polybrominated diphenyl ethers (PBDEs) are a group of additive brominated flame retardants (BFRs) that have been used widely in commercial and household products for decades. As these flame retardants are not permanently bound to the polymer matrix, they leach out during natural operational life, as well as during processing, recycling or combustion of the polymeric material. Because of the heavy demand by global markets in the last decade, PBDEs are nearly ubiquitously distributed in the environment (Fu et al. 2009). As a result of their lipophilicity, persistence, bioaccumulation and potential toxicity (including thyroidogenic, estrogenic, and hepatic effects, as well as neurodevelopmental disorders, because of prolonged exposure to PBDEs), two of the three commercial PBDE mixtures ("penta-BDE" and "octa-BDE") have been banned by the European Union and several states in the U.S.

With continuously increasing contamination in environment matrixes (de Wit et al. 2010), there are increasing concerns about the bioavailability and degradation of PBDEs in biota. In addition, some "special" properties of PBDEs, compared to other POPs, have been found. It was reported that lower (tetra- and penta-) PBDEs accumulated and were biomagnified in biota, while the higher congeners (deca-) were prevalent in aquatic environments and sediments (Martin et al. 2004). Dietary exposure studies with rats (Huwe and Smith 2007) and fish (Stapleton et al. 2006) revealed that the bioavailability of Deca-BDE produced a range of results that might be attributed to matrix effects, the dose and other effects. There are many studies focused on the tissue localization of PBDEs in animals (Naert et al. 2007; Gebbink et al. 2008; Luo et al. 2009). Little attention has been paid to the bioaccumulation of PBDEs in brain tissue, for example, in bears (Gebbink et al. 2008), rats (Huwe et al. 2008) and birds of prey (Voorspoels et al. 2006a; Naert et al. 2007). In this work, we investigated the contamination and tissue distribution of PBDEs in free-range domestic hens and ducks raised in a rural village at

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an e-waste recycling region in South China. The primary aim of the present study was to compare the PBDE concentrations and congener patterns in the brain with that in other tissues of these animals.

Methods and Materials

PBDE analytical standard method 1614 native PAR stock solution EO-5278 (unlabeled BDE-28, 47, 99, 100, 153, 154, 183, 209), method 1614 labeled surrogate stock solution EO-5277 ($^{13}C_{12}$ labeled BDE-28, 47, 99, 100, 153, 154, 183, 209), and method 1614 labeled injection internal stock solution EO-5275 ($^{13}C_{12}$ labeled CB-52, 138), were purchased from Cambridge Isotope Laboratories, Inc. (Andover, MA). Hexane and dichloromethane used for the extraction and cleanup procedures were pesticide grade (J.T. Baker, USA), and other solvents and reagents were of analytical grade.

In November 2006, four free-range hens (*Gallus domesticus*), named as HA (2.5 kg, 3 years), HB (2.1 kg, 3 years), HC (0.65 kg, 1.5 years) and HD (1.2 kg, 2 years), were purchased from different farmers who lived in a small rural village in Taizhou of Zhejiang Province. In addition, three ducks (*Anas platyrhynchos domesticus*), named as DA (2.7 kg, 1 year), DB (2.8 kg, 1 year) and DC (1.75 kg, 1 year), which were free-range in the paddy field outside this village, were also collected. The live hens and ducks were brought to the laboratory and blood samples (about 20 mL) were taken following euthanasia. Then, the liver, brain, pectoral muscle, intestines and mesenteric fat tissues were excised, and designated as -B, -L, -Br, -M, -I and -Mf, respectively. For unknown reasons, there was no mesenteric fat in hen C and the ducks. After being homogenized in a tissue homogenizer, 0.5–8 g of various tissues and blood were freeze-dried, and kept frozen at −20°C until analysis.

Eight PBDE congeners (BDE-28, 47, 99, 100, 153, 154, 183 and 209) were detected in samples using $^{13}C_{12}$ isotope dilution methods that are described below. Dried tissue or blood samples were ground with anhydrous sodium sulfate into free-flowing powder. The samples were extracted with 200 mL of hexane/dichloromethane (1:1, v/v) in Soxhlet extraction mode for 24 h. An aliquot of the extract was used for gravimetric lipid determination. All samples were spiked with eight $^{13}C_{12}$-labeled PBDE recovery standards (EO-5277) prior to extraction and cleanup. Then, the concentrated extracts were cleaned on a column (15 mm, i.d.) packed, from the bottom to top, with 1 g activated silica gel, 3 g basic silica gel (EPA Method 1614, Sect. 7.5.1.3), 1 g activated silica gel, 4 g acid silica gel (44% concentrated sulfuric acid, w/w), 4 g acid silica gel (22% concentrated sulfuric acid, w/w), 1 g activated silica gel, and 1 cm anhydrous sodium sulfate. After elution with 100 mL of hexane, the concentrated eluate was loaded on another column (8 mm i.d.) containing 2 g silver nitrate ($AgNO_3$) silica (10%, w/w), 2 g activated silica gel and 1 cm anhydrous sodium sulfate. PBDEs were separated from PCBs by two elutions: 40 mL hexane for PCBs and 40 mL hexane/dichloromethane (1:1, v/v) for PBDEs. The PBDE fractions were collected and reduced to 30 μL under a gentle N_2 stream. Then, $^{13}C_{12}$-labeled internal injection standards (EO-5275) were added prior to the GC injection. Throughout the extraction, cleanup and analysis procedure, the analytes were protected from light by wrapping the containers with aluminum foil, or by using amber glassware.

The samples were analyzed on an Agilent 6890 series gas chromatograph coupled with an Agilent 5973 mass spectrometer. For the analysis of BDE-28, 47, 99, 100, 153, 154 and 183, the mass spectrometer was operated with the electron impact ionization (EI) source in the selected ion monitoring (SIM) mode, and a DB-5MS (30 m × 0.25 mm i.d., 0.25 μm film thickness) capillary column was used. The monitored ions, (m/z) 405.8/407.8, 483.7/485.7, 403.8/405.8, 403.8/405.8, 481.7/483.7, 481.7/483.7 and 561.6/563.6, were selected for unlabeled BDE-28, 47, 99, 100, 153, 154 and 183, respectively. The monitored ions 417.8/419.8, 495.7/497.7, 415.8/417.8, 415.8/417.8, 493.7/495.7, 493.7/495.7 and 573.6/575.6, were selected for $^{13}C_{12}$-labeled BDE-28, 47, 99, 100, 153, 154 and 183, respectively. The temperature of the MS ion source (electron energy 70 eV) and transfer line were kept at 230 and 300°C, respectively. The GC oven temperature program was carried out as follows: initial temperature of 100°C held for 1 min, increased to 150°C at 10°C/min, held for 5 min, and then increased to 280°C at 5°C/min, and to 290°C at 10°C/min, held for 15 min. Helium was used as the carrier gas at a flow rate of 1 mL/min. For the determination of BDE-209, the mass spectrometer was operated with negative chemical ionization (NCI) in the SIM mode, and a DB-5 MS (15 m × 0.25 mm i.d., 0.25 μm film thickness) capillary column was used. The monitored ions, (m/z) 484.7/486.7 and 494.7/496.7, were selected for unlabeled and $^{13}C_{12}$ labeled BDE-209, respectively. Methane was used as a chemical ionization moderating gas and helium was used as the carrier gas at a flow rate of 1.5 mL/min. The ion source and interface temperatures were set to 150 and 300°C, respectively. The temperature program was from 80°C (1 min) to 200°C at 10°C/min, and to 300°C at 20°C/min (held for 15 min). In both experiments, the splitless injection mode was used and 1 μL was injected (injector temperature 265°C) with a solvent delay set at 6 min.

Quality control was performed by regular analyses of procedural blanks, and regular injection of solvent blanks and standards. The residue concentrations in samples

Table 1 Concentrations of 8 target PBDE congeners (ng/g lipid weight), ∑PBDEs (sum of the BDE-28, 47, 99, 100, 153, 154, 183, 209) and the concentration ratio of BDE-99 vs. BDE-100 (99/100) and BDE-153 vs. BDE-154 (153/154) in various tissues of hens(H) and ducks(D)

	BDE28	BDE47	BDE100	BDE99	BDE154	BDE153	BDE183	BDE209	∑PBDEs	99/100	153/154
HA-I	0.33	30.43	7.89	20.50	3.09	9.25	2.29	33.03	106.82	2.60	2.99
HA-M	0.47	40.08	9.31	26.30	3.74	10.32	2.53	50.02	142.75	2.83	2.76
HA-Br	0.13	3.57	1.31	4.00	0.54	1.89	0.32	2.98	14.76	3.05	3.51
HA-L	0.19	30.98	7.03	18.00	2.16	7.26	1.31	15.53	82.47	2.56	3.36
HA-Mf	0.59	56.85	13.47	40.68	5.26	14.48	3.12	15.06	149.51	3.02	2.76
HA-B	ND[a]	7.69	3.00	11.18	1.23	3.37	0.79	21.05	48.31	3.73	2.73
HB-I	0.75	46.83	7.34	37.22	6.78	80.81	190.11	2712.11	3081.94	5.07	11.92
HB-M	0.39	20.71	3.53	14.24	2.86	33.34	75.70	1393.56	1544.31	4.03	11.66
HB-Br	0.07	4.57	0.71	2.93	0.98	16.61	11.69	34.55	72.12	4.11	17.04
HB-L	0.13	22.32	4.70	10.57	3.84	58.87	55.50	247.32	403.25	2.25	15.32
HB-Mf	0.60	63.22	13.01	40.96	7.80	100.20	343.85	644.36	1214.00	3.15	12.85
HB-B	ND[a]	7.97	2.97	4.17	2.12	10.25	38.85	288.94	355.26	1.40	4.85
HC-I	0.08	19.67	13.96	31.85	15.81	85.30	39.13	557.95	763.76	2.28	5.40
HC-M	0.49	19.18	15.61	23.22	10.08	37.15	14.43	430.30	550.47	1.49	3.68
HC-Br	0.13	2.49	1.49	2.86	1.04	7.88	1.34	3.62	20.85	1.91	7.59
HC-L	0.46	26.57	25.22	14.16	12.33	29.71	8.22	115.16	231.81	0.56	2.41
HC-B	ND[a]	3.71	5.10	3.06	3.54	9.20	3.62	29.36	57.60	0.60	2.60
HD-I	0.42	57.72	14.96	37.09	6.10	20.16	16.90	279.29	432.64	2.48	3.30
HD-M	0.34	36.69	10.83	22.83	4.49	13.33	9.30	251.76	349.56	2.11	2.97
HD-Br	0.02	3.87	1.29	2.84	0.42	2.41	0.75	3.04	14.63	2.21	5.75
HD-L	0.44	46.80	14.23	16.33	5.39	16.04	5.28	128.45	232.96	1.15	2.98
HD-Mf	0.45	83.86	20.02	52.90	7.84	25.66	18.01	148.54	357.28	2.64	3.27
HD-B	ND[a]	7.22	4.01	3.97	2.15	2.91	2.51	13.15	35.91	0.99	1.36
DA-I	41.55	1927.89	676.09	3376.51	201.80	1089.95	1799.74	12039.52	21153.05	4.99	5.40
DA-M	8.31	679.94	177.87	956.96	125.93	670.76	928.97	16226.27	19775.00	5.38	5.33
DA-Br	1.57	156.71	41.48	208.78	27.84	237.20	320.27	671.63	1665.49	5.03	8.52
DA-L	3.82	1018.66	225.88	1122.40	211.33	1221.01	1310.78	114210.91	119324.79	4.97	5.78
DA-B	0.86	136.48	27.09	129.11	16.50	68.10	32.54	19234.36	19645.04	4.77	4.13
DB-I	24.25	1476.41	399.43	2105.11	261.17	1306.91	2240.20	9737.19	17550.67	5.27	5.00
DB-M	6.78	743.81	189.89	835.24	111.84	620.33	1395.84	18094.67	21998.39	4.40	5.55
DB-Br	0.92	155.04	49.61	210.29	26.75	244.63	237.86	73.45	998.55	4.24	9.15
DB-L	3.15	876.53	249.56	907.26	151.09	880.79	2053.74	77776.43	82898.55	3.64	5.83
DB-B	ND[a]	334.66	87.97	361.11	58.89	387.29	1005.29	32731.69	34966.91	4.10	6.58
DC-I	0.40	56.11	65.24	226.02	65.61	378.37	628.24	898.12	2318.10	3.46	5.77
DC-M	0.57	105.20	108.90	398.40	84.89	521.34	687.68	1039.75	2946.72	3.66	6.14
DC-Br	0.03	22.62	24.89	84.49	16.89	123.09	71.25	16.50	359.77	3.39	7.29
DC-L	0.14	105.99	110.88	368.10	91.45	504.93	616.47	827.58	2625.55	3.32	5.52
DC-B	ND[a]	28.15	31.55	115.39	32.76	202.30	298.93	926.04	1635.11	3.66	6.18

The blood, liver, brain, muscle, intestines and mesenteric fat tissues were expressed as -B, -L, -Br, -M, -I and -Mf, respectively

[a] *ND* not detected

below quantificational limits were regarded to be equal to zero in the calculation of the sum, means, and so on. The limit of quantifications (LOQ) defined as the mean value of corn oil matrix procedural blanks (spiked with eight $^{13}C_{12}$-labeled PBDE recovery standards) plus a safety margin of 3× the standard deviation (SD) of the procedural blanks were 0.005–0.03 ng/g lipid weight for BDE-28, 47, 99, 100, 153, 154 and 183 in the EI-MS mode, and 0.04 ng/g lipid weight for deca-BDE in NCI-MS mode. The recovery rates of $^{13}C_{12}$-labeled BDE-209 spiked into tissue samples ranged from 60 to 90%, and for the other $^{13}C_{12}$-labeled PBDEs, it was 75–110%.

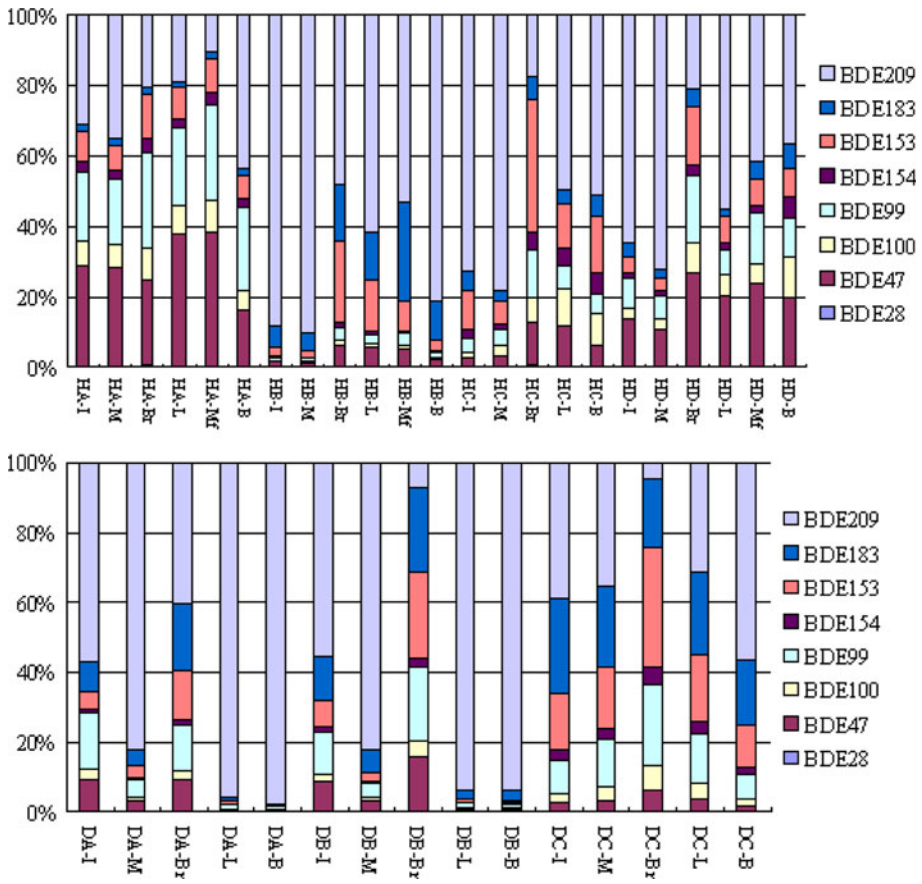

Fig. 1 Percentage of PBDE congeners in tissues of hens (*top*) and ducks (*bottom*), the blood, liver, brain, muscle, intestines and mesenteric fat tissues were expressed as -B, -L, -Br, -M, -I and -Mf, respectively

Results and Discussion

Except for BDE-28, which was not detected in blood samples from the hens, and duck B and C (Table 1), 8 target PBDE congeners were found consistently in all tissues of the hens and ducks. In general, the PBDE concentrations in ducks were higher than in hens; this may be explained by the paddy field, which the free-range ducks frequented near the e-waste dump. No significant correlations were found between PBDE concentrations and the weight or age of the hens or ducks. Compared with other studies, as the village had been exposed to e-waste recycling for years, the PBDE levels found in this study were similar to that of fowl from another e-waste recycling site in Guangdong Province of China (Luo et al. 2009), but much higher than the market-based samples (Voorspoels et al. 2007).

As shown in Table 1, the concentration of $\sum$PBDEs (sum of the BDE-28, 47, 99, 100, 153, 154, 183 and 209) in brain was the lowest among tissues of individual hens and ducks. Additional data also showed lower concentrations of PBDEs in brain than in other tissues after correction for the respective lipid content (Voorspoels et al. 2006a). This was similar to the PCDD/Fs and coplanar PCBs found in brain tissues of wild herring gulls from Bohai Bay, North China (Wan et al. 2006). It is possible that these toxicants are hindered from crossing the blood–brain barrier because of the polar lipid composition of the brain (Ishaq et al. 2000; Maervoet et al. 2005).

Being small, neutral and lipophilic molecules, PBDEs may enter the brain by passive diffusion or by transporter-mediated uptake. Voorspoels et al. (2007) considered that PBDEs theoretically had ideal physicochemical properties to diffuse freely across biological membranes, such as the blood–brain barrier, by the theoretical passive drug absorption models (Camenisch et al. 1996). Since brain tissue consists mainly of relatively polar lipids (e.g., polar phospholipids) (Tilbury et al. 1997), compared with triacylglycerol-rich tissues, these polar phospholipids constitute unfavorable sites for the accumulation of PCBs (Sandermann 2003). The same hypothesis could be applied to the bioaccumulation of PBDEs in brain tissues. We presume that the ability of the blood–brain barrier to hinder the transport of certain compounds is also involved in the low PBDE concentrations in the brain, especially in the case of high levels of PBDE pollution.

In the present study, as the ducks and hens were sampled from an e-waste recycling region, the BDE-209 was consistently found in brains, and was the major component part of the $\sum$PBDEs in other tissues (Fig. 1). Except for Hen A

Table 2 PBDEs congener correlation of brain (Br), mesenteric fat (Mf), muscle (M), liver (L) and intestine (I) versus blood (B) of hens and ducks, respectively

PBDE congener	Hens					Ducks			
	Br vs. B (n = 4)	Mf vs. B (n = 3)	M vs. B (n = 4)	L vs. B (n = 4)	I vs. B (n = 4)	Br vs. B (n = 3)	I vs. B (n = 3)	M vs. B (n = 3)	L vs. B (n = 3)
BDE28	NA[a]	NA[a]	NA[a]	NA[a]	NA[a]	NA[a]	NA[a]	NA[a]	NA[a]
BDE47	0.907	−0.821	0.528	0.166	0.685	0.763	0.602	0.825	0.670
BDE100	0.695	0.999	0.886	0.994	0.848	0.708	−0.011	0.561	0.580
BDE99	0.993	−0.537	0.526	0.639	−0.900	0.551	0.160	0.357	0.289
BDE154	0.667	0.999	0.876	0.973	0.984	0.043	0.421	−0.269	−0.383
BDE153	0.903	0.985	0.965	0.880	0.975	0.146	0.312	−0.244	−0.392
BDE183	1.000	1.000	0.997	0.999	0.993	−0.075	0.499	0.819	0.716
BDE209	0.999	0.974	0.970	0.844	0.988	0.164	0.806	0.944	0.727

[a] *NA* not available

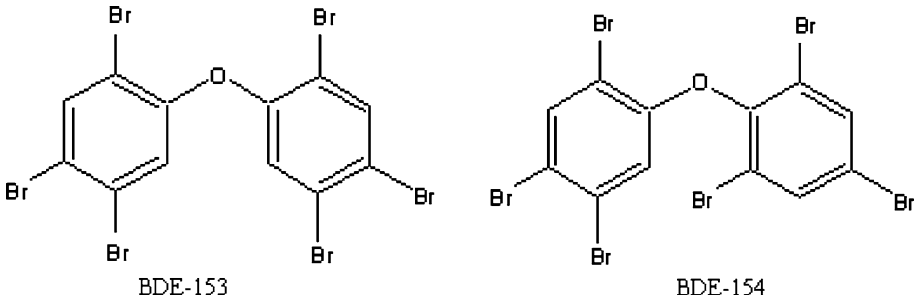

Fig. 2 Structure of BDE-153 and BDE-154

which had the lowest PBDE contamination among these samples, the percentage of BDE-209 in ∑PBDEs in the brain was the lowest among tissues of individual hens and ducks, suggesting the greater hindrance of the blood–brain barrier to the crossing of higher molecular weight molecules.

Although BDE-153 prevailed over BDE-154 in birds (Voorspoels et al. 2006a) and other terrestrial species (Voorspoels et al. 2006b), in this work, the concentration ratio of BDE-153 versus BDE-154 (153/154) in brain was the highest among tissues of individual hens and ducks (Table 1). However, the concentration ratio of BDE-99 versus BDE-100 (99/100) in brain was not the highest. Several studies have shown the metabolism of higher brominated PBDEs via debromination in biota (Huwe and Smith 2007). Since the brain is not the major metabolic organ, the debromination of higher brominated PBDEs in brain may not be faster than other metabolic organs (for example, liver). Furthermore, as shown in Table 2, the PBDE congener correlation of various tissues versus blood showed that the highest correlation difference between BDE-153 and BDE-154, both in hens and ducks, appeared in the brain versus blood. This indicates the difference in the penetration of BDE-153 and BDE-154 across the blood–brain barrier. Thus, the highest concentration ratio of BDE-153 versus BDE-154 being in the brain might be caused by the easier penetration of BDE-153 crossing the blood–brain barrier than BDE-154.

Figure 2 illustrates the structures of BDE-153 and BDE154. As there are two ortho-substituted bromine atoms in BDE-154, similar to the structure of PCBs (McKinney et al. 1983), BDE154 is less prone to form the coplanar conformation (minimum bulk) than BDE-153, thus creating a steric hindrance. Therefore, BDE-154 has a lower penetrability than BDE-153 when crossing the blood–brain barrier. The lower molecular weight of BDE-99 and BDE-100 compared to that of BDE-153 and BDE-154, may weaken the effect of the molecular conformation when crossing the blood–brain barrier. As a result, the relative selectivity of BDE-99 over BDE-100 in the brains of hens and ducks was not as obvious as the relative selectivity of BDE-153 over BDE-154.

In brief, as the village had been exposed to e-waste recycling for years, the PBDE levels in the hens and ducks used in this study were very high. Our results indicate that the hindrance of the blood–brain barrier to compounds, especially to molecules with high molecular weight and non-planar conformation (steric hindrance), contributed to

the low concentration of PBDEs in the brain tissue of hens and ducks collected in a rural village at an e-waste recycling region in South China.

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References

Camenisch G, Folkers G, van de Waterbeemd H (1996) Review of theoretical passive drug absorption models: historical background, recent developments, and limitations. Pharm Acta Helv 71:309–327

de Wit CA, Herzke D, Vorkamp K (2010) Brominated flame retardants in the Arctic environment—trends and new candidates. Sci Tot Environ 408:2885–2918

Fu S, Yang ZZ, Zhang L, Li K, Xu XB (2009) Composition, distribution, and characterization of polybrominated diphenyl ethers in sandstorm depositions in Beijing, China. Bull Environ Contam Toxicol 83:193–198

Gebbink WA, Sonne C, Dietz R, Kirkegaard M, Riget FF, Born EW, Muir GDC, Letcher RJ (2008) Tissue-specific congener composition of organohalogen and metabolite contaminants in East Greenland polar bears (*Ursus maritimus*). Environ Pollut 152(3): 621–629

Huwe JK, Smith DJ (2007) Accumulation, whole-body depletion, and debromination of decabromodiphenyl ether in male sprague-dawley rats following dietary exposure. Environ Sci Technol 41:2371–2377

Huwe JK, Hakk H, Birnbaum LS (2008) Tissue distribution of polybrominated diphenyl ethers in male rats and implications for biomonitoring. Environ Sci Technol 42:7018–7024

Ishaq R, Karlson K, Näf C (2000) Tissue distribution of polychlorinated naphthalenes (PCNs) and non-ortho chlorinated biphenyls (non-ortho CBs) in harbour porpoises (*Phocoena phocoena*) from Swedish waters. Chemosphere 41:1913–1925

Luo XJ, Liu J, Luo Y, Zhang XL, Wu JP, Lin Z, Chen SJ, Mai BX, Yang ZY (2009) Polybrominated diphenyl ethers (PBDEs) in free-range domestic fowl from an e-waste recycling site in South China: levels, profile and human dietary exposure. Environ Int 35:253–258

Maervoet J, Beck V, Roelens SA, Covaci A, Voorspoels S, Geuns JMC, Darras VM, Schepens P (2005) Uptake and tissuespecific distribution of selected polychlorinated biphenyls in developing chicken embryos. Environ Toxicol Chem 24:597–602

Martin M, Lam PKS, Richardson BJ (2004) An Asian quandary: where have all of the PBDEs gone? Mar Pollut Bull 49:375–382

McKinney JD, Gottschalk KE, Pedersen L (1983) A theoretical investigation of the conformation of polychlorinated biphenyls (PCBs). J Molec Struct 104:445–450

Naert C, Van Peteghem C, Kupper J, Jenni L, Naegeli H (2007) Distribution of polychlorinated biphenyls and polybrominated diphenyl ethers in birds of prey from Switzerland. Chemosphere 68:977–987

Sandermann JH (2003) Differential lipid affinity of xenobiotics and natural compounds. FEBS Letters 554:165–168

Stapleton HM, Brazil B, Holbrook RD, Mitchelmore CL, Benedict R, Konstantinov A, Potter D (2006) In vivo and in vitro debromination of decabromodiphenyl ether (BDE 209) by juvenile rainbow trout and common carp. Environ Sci Technol 40:4653–4658

Tilbury KL, Stein JE, Meador JP, Krone CA, Chan SL (1997) Chemical contaminatants in harbour porpoise (*Phocoena phocoena*) from the north Atlantic coast: tissue concentrations and intra- and inter-organ distribution. Chemosphere 34:2159–2181

Voorspoels S, Covaci A, Lepom P, Escutenaire S, Schepens P (2006a) Remarkable findings concerning PBDEs in the terrestrial top-predator red fox (*Vulpes vulpes*). Environ Sci Technol 40:2937–2943

Voorspoels S, Covaci A, Lepom P, Jaspers VLB, Schepens P (2006b) Levels and distribution of polybrominated diphenyl ethers in various tissues of birds of prey. Environ Pollut 144(1):218–227

Voorspoels S, Covaci A, Neels H, Schepens P (2007) Dietary PBDE intake: a market-basket study in Belgium. Environ Int 33:93–97

Wan Y, JY HU, An W, Zhang ZB, An LH, Hattori T, Itoh M, Masunaga S (2006) Congener-specific tissue distribution and hepatic sequestration of PCDD/Fs in wild herring gulls from Bohai Bay, North China: comparison to coplanar PCBs. Environ Sci Technol 40:1462–1468