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Brominated flame retardants and phenolic endocrine disrupters in Finnish human adipose tissue

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Abstract

Brominated flame retardants and phenolic compounds, of which several have been shown to exhibit endocrine disrupting effects, were screened in extracts of Finnish human adipose tissue samples. The samples were collected during autopsy from 39 subjects, of which 23 were males and 16 females. The samples were homogenised and extracted, and then cleaned-up by preparative gel permeation chromatography. The phenolic compounds were determined in silylated extracts. A total of 21 individual compounds were analysed in the extracts by gas chromatography–mass spectrometry (HRGC–LRMS) in the selected ion monitoring mode. The most commonly occurring compounds were 4-octylphenol diethoxylate, 4,4′-dihydroxybiphenyl, and 2,2′,4,4′-tetrabromodiphenyl ether (BDE-47), but also some other alkylphenols, pentabromophenol, and 2,2′,4,4′,5-penta- and 2,2′,4,4′,5,5′-hexabromodiphenyl ether could be detected in 1–6 samples. The concentrations were ranging from trace amounts to 71 ng/g of lipid weight. The mean concentration of BDE-47 was 1.20 ng/g lipids, however, in 15 of the samples the concentration was below the detection limit. Compared to other European studies the average concentration of BDE-47 obtained in this study is at the lower end of the reported concentrations.
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1. Introduction

Halogenated, mostly brominated flame retardants (BFRs) have been used in large quantities in many products such as TV sets, computers, and household textiles to reduce fire risk. The compounds are very widespread in the environment (de Wit, 2002). Most concern has been expressed over polybrominated biphenyls (PBBs) and polybrominated diphenyl ethers (PBDEs). PBBs have similar properties to PCBs, and PBDEs have been shown to induce neurotoxic effects in mice (Eriksson et al., 1998) and to disrupt the thyroid hormone system in rats and mice (Hallgren et al., 2001). PBDEs have been detected in human breast milk (Darnerud et al., 1998; Meironyté et al., 1999; Strandman et al., 2000), in human blood samples (Sjödin et al., 1999; Thomsen et al., 2002), and in human adipose tissue (Stanley et al., 1991; Hardell et al., 1998; Meneses et al., 1999; Strandman et al., 1999; Meironyté Guvenius et al., 2001; Covaci et al., 2002; She et al., 2002; van Bavel et al., in press). Also 2,4,6-tribromophenol (BP3) has been detected in human serum (Thomsen et al., 2002). PBDEs belonged to the most widely used flame retardants in the 1990s (EHC, 1997). 2,4-Dibromophenol (BP2), BP3, and pentabromophenol (BP5) are high production volume ($\geqslant 5000$ t/year) flame retardants, whereas pentabromotoluene (PBT) is a moderate

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production volume flame retardant (1000–5000 t/year) (EHC, 1997). Hexabromobenzene (HBBz), tribromoaniline (TBA), and 2,2′,4,4′,5,5′-hexabromobiphenyl (HxBB) are no longer in use as flame retardants (EHC, 1997).

Phenolic compounds, such as the alkylphenols 4-octylphenol (OP) and bisphenol A (BPA), have raised much concern as estrogen-mimicking compounds. These compounds and other 4-OH-alkylphenols such as 2-*tert*-butyl-4-hydroxyanisole (BHA), and 4-*sec*-butyl- (SBP), 4-*tert*-butyl- (TBP), and 4-*tert*-pentylphenol (TPP), as well as hydroxybiphenyls have shown estrogenic effects in vitro assays (Jobling and Sumpter, 1993; Soto et al., 1995). SBP, TPP, TBP, and 4,4′-dihydroxybiphenyl (OH_2BP) are industrial chemicals. TBP and TPP are e.g., used in the manufacture of resins and bactericidal detergents. BPA is used in the synthesis of polycarbonate plastics and 2- and 4-hydroxybiphenyl (2-OHBP, 4-OHBP) are used in the rubber industry. 2-OHBP is also used as a disinfectant and as a fungicide and germicide, and 4-OHBP in the manufacture of resins. BHA is a widely used food antioxidant. OP and 4-octylphenol di- and triethoxylate (OP2EO and OP3EO) are degradation products of 4-octylphenol polyethoxylates, which are nonionic industrial surfactants. Some alkylphenols, among them BPA, 2-OHBP, and 4-OHBP have been detected in surface waters, sediments, and sewage sludge (Bolz et al., 2001).

The main objective of this work was to screen Finnish human adipose tissue on the occurrence of a large number of brominated flame retardants and endocrine disrupting phenolics. To our knowledge, other brominated flame retardants than PBDEs have not been analysed before in human adipose tissue. This study also adds to previously available data on PBDE levels in human adipose tissue samples from Finland. Moreover, we considered it necessary to add to the very limited knowledge on the possible occurrence of alkylphenols in human adipose tissue.

2. Experimental

2.1. Chemicals

The solvents and reagents were the same as described in a previous work (Smeds and Saukko, 2001). The individual reference compounds were obtained from various commercial sources: 4,4′-dibromooctafluorobiphenyl (BFB), 4-hydroxynonafluorobiphenyl (HNFB), OP2EO, OH_2BP, BPA, TBP, SBP, and OP from Aldrich Chemical Co.; 2-OHBP, HBBz, TBA, TPP, BP2, BP3, and BP5 from Acros Organics; BHA and 4-OHBP from ICN Biomedicals, Inc.; PBT and HxBB from Accu-Standard; 2,2′,4,4′-tetrabromodiphenyl ether (BDE-47), 2,2′,4,4′,5-pentabromodiphenyl ether (BDE-99), and 2,2′,4,4′,5,5′-hexabromodiphenyl ether (BDE-153) as a mix solution (“BDE-MXA”) from Wellington Laboratories. The Igepal CA-210 product from Aldrich Chemical Co. consisting of OP2EO contained OP3EO as an impurity according to GC–MS analysis and interpretation of the mass spectra. According to GC–MS the product consisted of 87% OP2EO and 11% OP3EO. OP3EO was also quantified in the adipose tissue extracts.

2.2. Samples

The adipose tissue samples were obtained from routine medico-legal autopsies and some of them were the same as in a previous work (Smeds and Saukko, 2001). A total of 39 samples were collected from 23 males and 16 females aged 14–95 years. The samples were collected from the abdominal, perirenal, pericardial, or mammary region. The average age of the male subjects was 59 and of female subjects 65 years. The samples used in the analysis of PBDEs were from the 23 male and 14 of the female subjects (average age 65 years), in the analysis of bromophenols from 17 of the male and 12 of the female subjects, and of alkylphenols from seven of the male subjects (aged 43–82 years, average age 64 years) and six of the female subjects (aged 51–90 years, average age 77 years). Eighteen lipid extracts (prepared as described below) consisted of composite samples obtained by combining an aliquot of lipid extract from two or three different fat depots of the same person. The rest of the samples were exclusively from one region (the male samples from the perirenal and abdominal region, the female samples from the mammary region). The samples were stored in glass vials at −25 °C until extraction and clean-up.

2.3. Sample pretreatment

The thawn approximately 10-g samples were extracted and cleaned-up by GPC as described previously (Smeds and Saukko, 2001). Briefly, the samples were homogenised in *n*-hexane:acetone 85:15 (v/v), and approximately 0.6 g of the lipid extract dissolved in dichloromethane (DCM):cyclohexane 1:1 (v/v) was gravimetrically eluted through a glass column packed with Bio-Beads S-X1 using DCM:cyclohexane 1:1 (v/v) as eluent. The 200–450 ml fraction was collected, the solvent was evaporated to a few ml in a rotary evaporator at 35–40 °C and evaporated to dryness with nitrogen gas. The extract was weighed and dissolved in 1 ml *n*-hexane. The sample was then divided into two 0.5-ml aliquots. To one aliquot, HNFB was added as quantitative standard in an amount corresponding to 0.5 μg/g lipid extract. The samples were silylated by addition of bis-(trimethylsilyl)-trifluoroacetamide (BSTFA, 0.25 ml) and storing of the solution at 70 °C in 30 min. The solvent was evaporated to 50–100 μl. The samples were

stored at −25 °C until GC–MS analysis of phenolic compounds. Three of the phenolics were flame retardants (BP2, BP3, and BP5).

The other aliquot was further cleaned-up by Florisil chromatography. The solution was eluted through a Florisil packed glass column with 7.5 ml of *n*-hexane and then 9 ml of hexane:DCM 20:80 (v/v). BFB was added to the solution as a quantitative standard in an amount corresponding to 0.5 μg/g of lipids. The solvent was evaporated to 50–100 μl and stored at −25 °C until GC–MS analysis of flame retardants.

2.4. GC–MS–SIM analysis

The GC–MS–SIM analyses were performed with a VG7070E mass analyser interfaced to a Dani 3800 GC as described before (Smeds and Saukko, 2001). The column used was HP-1 with a length of 30 m, an i.d. of 0.25 mm, and a film thickness of 0.25 μm. In the analysis of phenolics the GC oven temperature was raised from 80 to 300 °C at a rate of 8 °C/min, and in the analysis of flame retardants from 130 to 300 °C at a rate of 8 °C/min. The criteria for SIM identification were the same as in our previous work (Smeds and Saukko, 2001). Ions monitored are listed in Tables 1 and 2.

2.5. Quality control

At regular intervals, solvent blanks were subjected to the entire analytical procedure to determine background interference. Clean fat for fortification purposes was obtained by collecting the 100–200 ml GPC fraction and concentrating to dryness. The fat was fortified with known amounts of the phenolics. The fortification level was 11 μg/g fat for OP3EO, 200–250 μg/g for 4-OHBP, BPA, and BP5, and 60–100 μg/g fat for the other compounds. The fortified sample was gel filtrated as the other samples. The recoveries were determined by GC–FID and the compounds were quantified against BFB, which was added just prior to the GC analysis.

3. Results and discussion

3.1. GPC recoveries

The GPC recoveries of the phenolics ranged from 64% to 95%. The average recoveries were: TBP 67.6% (RSD 11.8%, $n = 5$), SBP 78.0% (RSD 9.4%, $n = 5$), TPP 75.8% (RSD 7.4%, $n = 5$), 2-OHBP 83.6% (RSD 4.7%, $n = 5$), OP 95.2% (RSD 7.3%, $n = 5$), BP2 76.0% (RSD 6.1%, $n = 4$), BHA 64.0% (RSD 31.9%, $n = 3$), OP2EO 67.0% (RSD 5.4%, $n = 3$), OH_2BP 87.0% (RSD 13.0%, $n = 3$), OP3EO 82.3% (RSD 18.3%, $n = 3$), BP5 88.3% (RSD 2.4%, $n = 3$), BPA 88% (RSD 19.3%, $n = 2$), and 4-OHBP 73% (RSD 0%, $n = 2$). All compounds except BHA showed acceptable extraction repeatabilities (RSD<20%). The concentrations of phenolics in adipose tissue extracts presented in Table 3 have been

Table 1
Ions monitored for quantitative and qualitative determinations of BFRs using GC–MS/SIM

Compound	Molecular formula	m/z	Fragment ion	Theor. peak area ratio	Relative retention time
4,4′-Dibromooctafluorobiphenyl (BFB)	$C_{12}F_8Br^{81}Br$	455.82	$M^+ + 2$	1.00	1.00
Tribromoaniline (TBA)	$C_6H_4NBr_3$	326.79	M^+	0.34	1.01
	$C_6H_4NBr_2{}^{81}Br$	328.79	$M^+ + 2$	1.00	
	$C_6H_4NBr^{81}Br_2$	330.78	$M^+ + 4$	0.98	
Pentabromotoluene (PBT)	$C_7H_3Br_3{}^{81}Br_2$	485.61	$M^+ + 4$	1.00	2.25
	$C_7H_3Br_2{}^{81}Br_3$	487.61	$M^+ + 6$	0.98	
Hexabromobenzene (HBBz)	$C_6{}^{79}Br_4{}^{81}Br_2$	549.51	$M^+ + 4$	0.77	2.65
	$C_6{}^{79}Br_3{}^{81}Br_3$	551.50	$M^+ + 6$	1.00	
2,2′,4,4′-Tetrabromodiphenyl ether (BDE-47)	$C_{12}H_6OBr_3{}^{81}Br$	483.71	$M^+ + 2$	0.68	2.80
	$C_{12}H_6OBr_2{}^{81}Br_2$	485.71	$M^+ + 4$	1.00	
2,2′,4,4′,5-Pentabromodiphenyl ether (BDE-99)	$C_{12}H_5OBr_3{}^{81}Br_2$	563.62	$M^+ + 4$	1.00	3.24
	$C_{12}H_5OBr_2{}^{81}Br_3$	565.62	$M^+ + 6$	0.98	
2,2′,4,4′,5,5′-Hexabromobiphenyl (HxBB)	$C_{12}H_4OBr_4{}^{81}Br_2$	625.53	$M^+ + 4$	0.77	3.50
	$C_{12}H_4OBr_3{}^{81}Br_3$	627.53	$M^+ + 6$	1.00	
2,2′,4,4′,5,5′-Hexabromodiphenyl ether (BDE-153)	$C_{12}H_4OBr_4{}^{81}Br_2$	641.53	$M^+ + 4$	0.77	3.67
	$C_{12}H_4OBr_3{}^{81}Br_3$	643.53	$M^+ + 6$	1.00	

Table 2
Ions monitored for quantitative and qualitative determinations of phenolics

Compound	Molecular formula	m/z	Fragment ion	Peak area ratio	Relative retention time
4-*tert*-Butylphenol (TBP)	$C_{12}H_{19}OSi$	207.12	$[M\text{-}CH_3]^+$	1.00	0.74
	$C_{12}H_{19}O^{29}Si$	208.12	$[M\text{-}CH_3]^+$	0.19[a]	
	$C_{13}H_{22}OSi$	222.14	M^+	0.15[a]	
4-*sec*-Butylphenol (SBP)	$C_{11}H_{17}OSi$	193.10	$[M\text{-}C_2H_5]^+$	1.00	0.76
	$C_{12}H_{19}OSi$	207.12	$[M\text{-}CH_3]^+$	0.074[a]	
	$C_{13}H_{22}OSi$	222.14	M^+	0.14[a]	
4-*tert*-Pentylphenol (TPP)	$C_{12}H_{19}OSi$	207.12	$[M\text{-}C_2H_5]^+$	1.00	0.77
	$C_{13}H_{21}OSi$	221.14	$[M\text{-}CH_3]^+$	0.075[a]	
	$C_{14}H_{24}OSi$	236.16	M^+	0.071[a]	
2-*tert*-Butyl-4-hydroxyanisole (BHA)	$C_{13}H_{21}O_2Si$	237.13	$[M\text{-}CH_3]^+$	1.00	0.99
	$C_{13}H_{21}O_2{}^{29}Si$	238.13	$[M\text{-}CH_3\ ]^+$	0.18[a]	
	$C_{14}H_{24}O_2Si$	252.15	M^+	0.83[a]	
4-Hydroxynonafluorobiphenyl (HNFB)	$C_{15}H_9OF_9Si$	404.03	M^+		1.00
2,4-Dibromophenol (BP2)	$C_8H_9Br_2OSi$	306.88	$[M\text{-}CH_3]^+$	0.51[b]	1.03
	$C_8H_9Br^{81}BrOSi$	308.88		1.00	
	$C_8H_9{}^{81}Br_2OSi$	310.87		0.49[b]	
2-Hydroxybiphenyl (2-OHBP)	$C_{14}H_{15}OSi$	227.09	$[M\text{-}CH_3]^+$	1.00	1.14
	$C_{14}H_{15}O^{29}Si$	228.09	$[M\text{-}CH_3]^+$	0.21[a]	
	$C_{15}H_{18}OSi$	242.11	M^+	0.63[a]	
2,4,6-Tribromophenol (BP3)	$C_8H_8Br_3OSi$	384.79	$[M\text{-}CH_3]^+$	0.34[b]	1.41
	$C_8H_8Br_2{}^{81}BrOSi$	386.79		1.00	
	$C_8H_8Br^{81}Br_2OSi$	388.78		0.98[b]	
4-Hydroxybiphenyl (4-OHBP)	$C_{14}H_{15}OSi$	227.09	$[M\text{-}CH_3]^+$	0.92[a]	1.41
	$C_{14}H_{15}O^{29}Si$	228.09	$[M\text{-}CH_3]^+$	0.18[a]	
	$C_{15}H_{18}OSi$	242.11	M^+	1.00	
4-*n*-Octylphenol (OP)	$C_{10}H_{15}OSi$	179.09	$[M\text{-}C_7H_{15}]^+$	1.00	1.51
	$C_{16}H_{27}OSi$	263.18	$[M\text{-}CH_3]^+$	0.016[a]	
	$C_{17}H_{30}OSi$	278.21	M^+	0.11[a]	
4-Octylphenol diethoxylate (OP2EO)	$C_{14}H_{23}O_2Si$	251.15	$[M\text{-}C_7H_{15}O]^+$	1.00	1.74
	$C_{18}H_{31}O_2Si$	307.21	$[M\text{-}C_3H_7O]^+$	0.037[a]	
	$C_{19}H_{34}O_2Si$	322.23	$[M\text{-}C_2H_4O]^+$	0.039[a]	
4,4′-Dihydroxybiphenyl (OH_2BP)	$C_{17}H_{23}O_2Si_2$	315.12	$[M\text{-}CH_3]^+$	0.14[a]	1.99
	$C_{18}H_{26}O_2Si_2$	330.15	M^+	1.00	
	$C_{18}H_{26}O_2Si^{29}Si$	331.15	M^+	0.30[a]	
Bisphenol-A (BPA)	$C_{20}H_{29}O_2Si_2$	357.17	$[M\text{-}CH_3]^+$	1.00	2.09
	$C_{20}H_{29}O_2Si^{29}Si$	358.17	$[M\text{-}CH_3]^+$	0.33[a]	
	$C_{21}H_{32}O_2Si_2$	372.19	M^+	0.19[a]	
4-Octylphenol triethoxylate (OP3EO)	$C_{16}H_{27}O_3Si$	295.17	$[M\text{-}C_7H_{15}O]^+$	1.00	2.11
	$C_{16}H_{27}O_3{}^{29}Si$	296.17	$[M\text{-}C_7H_{15}O]^+$	0.19[a]	
	$C_{21}H_{38}O_3Si$	366.26	$[M\text{-}C_2H_4O]^+$	0.040[a]	
Pentabromophenol (BP5)	$C_8H_6Br_3{}^{81}Br_2OSi$	544.61	$[M\text{-}CH_3]^+$	1.00	2.33
	$C_8H_6Br_2{}^{81}Br_3OSi$	546.60		0.98[b]	
	$C_8H_6Br^{81}Br_4OSi$	548.60		0.48[b]	

[a] Experimental value, mean of five analyses of the standard solution.
[b] Theoretical value, based on the ratios for the isotope clusters.

Table 3
Concentrations and occurrence frequencies of detected compounds in adipose tissue extracts

Compound	Conc., ng/g lipids			No. of positive samples/ total no. of samples
	Individual/range	Mean ± SD	Median	
TBP	2.16, 53.8	–	–	2/13
TPP	1.60, 14.1	–	–	2/13
OP	1.59, 4.60	–	–	2/13
BPA	14.9, 47.2	–	–	2/13
BHA	13.8	–	–	1/13
2-OHBP	0.89	–	–	1/13
4-OHBP	0.7–8.6	1.22 ± 2.36	1.49	6/13
OH_2BP	0.4–29.5	5.30 ± 8.25	5.03	9/13
OP2EO	6.0–70.7	26.3 ± 21.3	16.1	13/13
OP3EO	3.56, 6.57	–	–	2/13
BP5	5.1, 11.7	–	–	2/29
BDE-47	0.20–15.5	1.20 ± 2.91	0.55	22/37
BDE-99	0.23–5.13	0.26	0.74	5/37
BDE-153	0.29, 0.30, 2.87	0.09	0.30	3/37

corrected for loss during sample preparation represented by these average recovery percentages.

3.2. *Occurrence and concentrations of analysed compounds in extracts of adipose tissue*

Table 3 shows concentrations and occurrence frequencies of detected compounds in the lipid extracts.

3.2.1. *Alkylphenols*

The background amount of a compound determined in the blanks was subtracted from the amount determined in the samples. Most of the phenolics were present in the blanks (in amounts ranging from 0.6 to 5.9 ng) with the exception of the halogenated phenolics, OH_2BP, OP3EO, and 4-OHBP. SBP could not be detected in any sample above the background contamination level, and 2-OHBP and BHA in only one sample. The compounds were quantified against the most abundant ion (Table 2) and two ions were used for confirmation of the identity by comparing the relative abundancies obtained in the sample with those obtained in the standard solution. One problem with the analysis of the phenolics was that the ions used for confirmation were usually much less abundant, making an unambigous identification impossible at low concentrations. The detection limit was estimated to be approximately 0.5 ng/g for most of the phenolics, however, somewhat lower for OH_2BP (0.3 ng/g) and higher for BP5 (2 ng/g). BPA seemed to be present in many samples but could not be quantified because of overlapping peaks at m/z 357 and 358.

To our knowledge, alkylphenols have not been detected previously in human adipose tissue at levels above the background contamination. Müller et al. (1998) were able to detect 4-octylphenol, but the concentration was at the analytical background level, i.e., ranging from 0.58 to 4.07 ng/g lipids. Schaefer et al. (2000) were not able to detect BPA and BHA and the concentration of 4-octylphenol was unknown because of disturbing high background values.

The findings that alkylphenols are not present or are present at low levels in adipose tissue are supported by studies indicating that some alkylphenols have a low bioaccumulation potential (Certa et al., 1996; Upmeier et al., 1999, 2000). These studies indicate that bioaccumulation occurs only after excessive doses which lead to saturated detoxification pathways. On the other hand, one study has indicated that 4-nonylphenol is distributed into the lipid phase of the human body within 2 h after oral application (Müller et al., 1998).

3.2.2. *Flame retardants*

Of the ten analysed BFRs only the PBDEs and BP5 could be detected. In our study only the PBDE congeners BDE-47, -99, and -153 were determined. These three PBDEs have been reported to account for 84–94% of the total amount of PBDEs in adipose tissue (Meironyté Guvenius et al., 2001).

The concentration of BDE-47 ranged from 0.20 to 15.5 ng/g lipids with an average of 1.20 ng/g in all samples (Table 3). The compound was considerably more frequently occurring among the male subjects (74% positive samples) than the female subjects (50% positive samples). Moreover, the average concentration was considerably higher among the male (1.59 ng/g) than the female subjects (0.56 ng/g) although the average age of the males was lower than that of the females. Very similar results were obtained in a study of 13 Spanish subjects (Meneses et al., 1999). The average BDE-47

concentration of all subjects was 1.36 ng/g; 1.58 ng/g of the males and 0.59 ng/g of the females, and the average age of the males was lower than that of the females. Contradictory results have been obtained in different studies on the sex and age correlation of PBDEs. Also in a Swedish study by Hardell et al. (1998), the average BDE-47 level was higher among the male than the female subjects, but in a Belgian study by Covaci et al. (2002) and in a study of Hungarian subjects (van Bavel et al., in press), the levels were higher among the female subjects. In a study of female subjects, She et al. (2002) found an inverse relationship between PBDE levels and age, but Hardell et al. (1998) and Covaci et al. (2002) found no correlation between PBDE levels and age.

BDE-47 could be detected in 59% of the samples, BDE-99 in five and BDE-153 in only three samples. Meneses et al. (1999) were able to detect BDE-47, -99, and -153 in all the analysed samples and the same was the case in a previous Finnish study (Strandman et al., 1999) ($n = 10$), in a Swedish study (Meironyté Guvenius et al., 2001) ($n = 5$), and in a North American study (She et al., 2002) ($n = 23$). However, in all these studies the overall concentration level was higher than in the present study with smaller variations between individuals. It is possible that PBDEs could have been detected in some more samples in the present study if the detection limits would have been lower (they were approximately 0.2 ng/g lipids) and the method would have been more optimised for recovery and detection of PBDEs. In a very recent study of Swedish ($n = 53$) and Hungarian ($n = 27$) human adipose tissue samples (van Bavel et al., in press), the average levels of BDE-47 were low (0.87 and 0.75 ng/g lipids, respectively). BDE-47 could not be detected in all, and BDE-99 and -153 in any of the Hungarian samples.

BDE-47 was the major congener in all samples in which all the three congeners could be detected (i.e., samples in which the concentration of BDE-47 exceeded 0.9 ng/g). BDE-47 has been the major congener in most human samples studied, including milk and blood (Meironyté et al., 1999; Sjödin et al., 1999; Strandman et al., 1999; Strandman et al., 2000; Meironyté Guvenius et al., 2001; She et al., 2002; Thomsen et al., 2002). This indicates a predominant exposure via food as BDE-47 is usually the major congener in fish, wildlife, and environmental samples (de Wit, 2002). However, in blood from electronics dismantlers, BDE-153 and hepta-BDE dominated (Sjödin et al., 1999). In two studies of human adipose tissue from the general population, the average BDE-153 level was higher than the BDE-47 level (Meneses et al., 1999; Covaci et al., 2002). This might be due to a more extensive exposure via inhalation of indoor air contaminated with higher-brominated PBDEs from flame-retarded electronic equipment, textiles, and furniture in the home environment or in the workplace than via food in these population groups.

The average PBDE levels measured in the present study are comparable with levels reported in several European studies: in Spain the average BDE-47 and -99 concentrations were 1.36 and 0.42 ng/g lipids, respectively (Meneses et al., 1999), in Belgium 1.45 and 0.29 ng/g lipids, respectively (Covaci et al., 2002), and in Sweden and Hungary the average BDE-47 concentrations were 0.87 and 0.75 ng/g lipids, respectively (van Bavel et al., in press). In a North American study (California), the PBDE levels were much higher with average BDE-47, -99, and -153 concentrations of 33, 11, and 16 ng/g lipids, respectively. The high levels may be due to a more extensive use of flame retardants in California than in European countries. In the previous investigation of Finnish human adipose tissue (Strandman et al., 1999), the PBDE levels were considerably higher than in our study. The average BDE-47 concentration was 7.3 ng/g lipids and also the BDE-99 and BDE-153 levels were higher (2.2 and 2.3 ng/g, respectively). The differences in concentration levels between the present and the previous Finnish study may be due to pure chance, taking into consideration the small number of samples analysed in the previous study ($n = 10$). Levels in Finnish human milk were similar to the levels measured in the present study; the average BDE-47 and -99 concentrations were 1.31 and 0.39 ng/g lipids, respectively (Strandman et al., 2000). Also in Sweden very different PBDE levels have been measured in different studies. Hardell et al. (1998) obtained an average BDE-47 concentration of 5.1 ng/g lipids in a control group ($n = 27$), whereas van Bavel et al. (in press) obtained an average concentration of 0.87 ng/g lipids ($n = 53$). The reasons why PBDE levels are similar in some European studies and higher in other studies are unknown. More studies should be done and exposure routes should be investigated more thorougly. Perhaps groups of people having similar exposure sources should be studied and compared instead of comparing randomly selected subjects from different countries.

The analyses in the present study were partly carried out on the same samples as in our previous study in which PCBs and pesticides were analysed (Smeds and Saukko, 2001). An obvious correlation between the concentration levels of these compounds and PBDEs cannot be observed. Covaci et al. (2002) found a correlation between PBDE and PCB levels in Belgian samples and suggested that the origin of these compounds might be the same, i.e., a common dietary source. Although this would be the case, the PBDEs have many other sources for the general population like electronic devices, plastic materials, and textiles which may contaminate the indoor air. The role of these sources is not very well known and may in some cases be larger than that of the dietary sources.

4. Conclusions

Some endocrine disrupting alkylphenols could be detected in adipose tissue at low concentration levels. Most alkylphenols could be detected only in a few samples, but 4-octylphenol diethoxylate and 4,4′-dihydroxybiphenyl were more commonly occurring. PBDE levels in the adipose tissue samples were low; similar to the levels reported in some recent European studies. Low levels of these compounds indicate that they cannot be considered as a threat to human health, but more studies should be done as it is possible that the levels may be higher in other population groups.

Previously, we have analysed PCBs and halogenated pesticides in Finnish human adipose tissue samples (Smeds and Saukko, 2001). The present study was our second work on screening Finnish human adipose tissue on the occurrence of a large number of endocrine disrupting chemicals. Only a small number of the analysed chemicals could be detected in the studied samples. As a summary, the most dominating compounds were 4,4′-DDE, PCBs, and pentachlorobenzene.

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