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Occurrence of Polychlorinated Biphenyls and Polybrominated Diphenyl Ethers in Belgian Human Adipose Tissue Samples

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Abstract. Levels of polychlorinated biphenyls (PCBs) and polybrominated diphenyl ethers (PBDEs) were measured in 53 human adipose tissue samples. The samples consisted of adipose tissue from 31 men and 22 women having a mean age of 53 years. No information about diet or occupational exposure was collected. Cleanup was performed using a glass column containing acidified silica, deactivated alumina, and anhydrous sodium sulphate. Subsequently, samples were analyzed by high-resolution gas chromatography/tandem mass spectrometry. PBDE concentrations (sum of BDEs 28, 47, 99, 100, 154, 153, and 183) ranged between 1.23 and 57.2 ng g^{-1} lipid weight and were comparable with levels in samples from other European countries. The sum of seven International Council for the Exploration of the Sea (ICES) indicator PCB congeners (PCBs 28, 52, 101, 118, 138, 153, and 180) ranged from 126 to 2090 ng g^{-1} lipid weight. No age dependency was found for PBDEs (Pearson correlation −0.023, $p = 0.873$), whereas PCBs showed higher correlation coefficients with age (Pearson correlation 0.613, $p < 0.0005$). There was no relationship between PBDE and PCB levels (Pearson correlation −0.010, $p = 0.943$).

Polychlorinated biphenyls (PCBs) and polybrominated diphenyl ethers (PBDEs) are pollutants that have been identified worldwide in environmental and biologic matrices, *i.e.*, air (Sjödin *et al.* 2001a; Montone *et al.* 2003), water (Binelli *et al.* 2004; Law *et al.* 2003), sediment (Allchin *et al.* 1999; de Boer *et al.* 2003; Sapozhnikova *et al.* 2004), fish (Focardi *et al.* 1996; Hites 2004, Johnson and Olson 2001; Akutsu *et al.* 2001), wildlife (Law *et al.* 2003), and human adipose tissue (Guvenius *et al.* 2001; Meneses *et al.* 1999; She *et al.* 2002; Pauwels *et al.* 2000; Costabeber and Emmanuelli 2003), blood (Sjödin *et al.* 1999; Sjödin *et al.* 2001b; Orloff *et al.* 2003; Thomsen *et al.* 2001a, 2001b), and breast milk (Erdoğrul *et al.* 2004; Kazda *et al.* 2004; Schecter *et al.* 2003; Lind *et al.* 2003; Norén and Meironyté 2000; Meironyté *et al.* 1999).

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PCBs have been widely used as electric insulators in transformers and capacitors, as heat-transfer fluids, and as additives in pesticides, adhesives, plastics, and paints (Safe 1994). A substantial part of the environmental burden of these compounds has resulted from disposal practices or leakage from industrial facilities and chemical waste disposal sites.

PBDEs are a group of additive flame retardants that are mixed with or dissolved into potentially flammable materials. The quantity and type of flame retardants employed depends on the application and fire protection requirements (Sjödin *et al.* 2001a). PBDEs are commercially produced as three mixtures, *i.e.*, penta-BDE, octa-BDE, and deca-BDE, indicating their degree of bromination (Sjödin *et al.* 2003). These three mixtures each contain fewer congeners than the former commercial PCB mixtures, and their general compositions are listed in Table 1. As a consequence, the number of PBDE congeners found in environmental samples is much lower than the number of PCB congeners.

PCBs and PBDEs are chemically stable and lipophilic and have been shown to be ubiquitous environmental pollutants. They have a tendency to accumulate in lipid-rich tissues and magnify up the food chain, increasing in concentration at each successively higher trophic level. Octa-, nona-, and deca-BDEs are detected in biota and humans, but they show no biomagnification potential (Burreau *et al.* 2004). The major human exposure route appears to be through intake of various kinds of fish, meat, and dairy products (Sjödin *et al.* 2003), although consensus on this point has not yet been reached. Occupational exposure may on certain occasions contribute significantly to the body burden of PBDEs (Sjödin *et al.* 1999; Thomsen *et al.* 2001b).

Despite the fact that PBDE concentrations in humans are significantly lower than those of PCBs, they are a growing reason for concern because PBDE levels have increased exponentially since the late 1970s (Alaee and Wenning 2002). A study by Meironyté (2002) showed decreasing levels of BDE 47 in breast milk from 1998 to 2000, but no large differences were noticed in levels of higher brominated PBDEs. In contrast, PCB levels have decreased in past decades (Schecter *et al.* 2004).

Table 1. General composition of three commercially produced PBDE mixtures given in percent of BDE congeners present[a]

Technical product	Tri-BDE (%)	Tetra-BDE (%)	Penta-BDE (%)	Hexa-BDE (%)	Hepta-BDE (%)	Octa-BDE (%)	Nona-BDE (%)	Deca-BDE (%)
Penta-BDE	0–1	24–38	50–62	4–8				
Octa-BDE				10–12	43–44	31–35	9–11	0–1
Deca-BDE							0.3–3	97–98

[a] Data from World Health Organization / International Programme on Chemical Safety (1994).
BDE = Brominated diphenyl ethers.

Toxic effects of PBDEs and PCBs include immunotoxicity, reproductive effects, teratogenicity, endocrine disruption, and carcinogenicity (Meerts *et al.* 2000; Legler and Brouwer 2003; Gill *et al.* 2004). However, because of the wide variation in physicochemical properties of individual congeners, generalized statements about their toxicology are difficult to make. Generally, the technical penta-BDE products seem to cause effects at comparably the lowest dose, whereas much higher doses are needed for effects of the deca-BDEs (Darnerud 2003).

The European Union has recently closed a 10-year scientific risk assessment of deca-BDE and concluded that it poses an acceptably low risk to human health and that no further risk decrease measures beyond those already being applied are necessary. According to Council Directive 2003/11/EC (2003), the use of penta- and octa-BDE, and the placement on the market of articles containing one or both of these substances, has been banned within the European Union since August 15, 2004. The Belgian legislation has set maximum limits for the total sum of seven International Council for the Exploration of the Sea (ICES) indicator PCB congeners (PCBs 28, 52, 101, 118, 138, 153, and 180) in different food matrices. The main objective of this work was to determine the levels and profiles of PCBs and PBDEs in adipose tissues of people living in Belgium.

Materials and Methods

Samples

Fifty-three human adipose tissue samples from the abdominal fat region were obtained by autopsy between 2001 and 2003 in two different Belgian laboratories from deceased individuals who died from natural or accidental causes. The mean age of the dead subjects was 53 years (range from 19 to 84) (Table 2). There were 31 men and 22 women. For each subject, the province of residence in Belgium was known, but no information was available about the type of residence area (rural or industrial). No information about diet or occupational exposure was provided. Animal fat tissue samples, taken within the framework of the Belgian national monitoring program for PCBs and containing PCB levels lower than the decision limit (CCα), were used to prepare a pool of blank extracted animal fat for quality-control purposes. From this pool, a sample was analyzed, and a new pool was prepared if PBDE levels were higher than CCα. Samples were stored at −20°C till analysis.

Materials and Reagents

Individual PBDE standards—International Union of Pure and Applied Chemistry (IUPAC) numbers 28 (2,4,4′-tribromodiphenyl ether), 47 (2,2′,4,4′-tetrabromodiphenyl ether), 99 (2,2′,4,4′,5-pentabromodiphenyl ether), 100 (2,2′,4,4′,6-pentabromodiphenyl ether), 153 (2,2′,4,4′,5,5′-hexabromodiphenyl ether), 154 (2,2′,4,4′,5,6′-hexabromodiphenyl ether), and 183 (2,2′,3,4,4′,5′,6-heptabromodiphenyl ether)—were purchased from Wellington Laboratories (Ontario, Canada). Individual PCB standards—IUPAC numbers 28 (2,4,4′-trichlorobiphenyl), 52 (2,2′,5,5′-tetrachlorobiphenyl), 101 (2,2′,4,5,5′-pentachlorobiphenyl), 118 (2,3′,4,4′,—5-pentachlorobiphenyl), 138 (2,2′,3,4,4′,5′-hexachlorobiphenyl), 153 (2,2′,4,4′,5′-hexachlorobiphenyl), and 180 (2,2′,3,4,4′,5,5′-heptachlorobiphenyl), together with PCB-Mix 3 (10 ng μl^{-1} of each congener)—were purchased from Dr. Ehrenstorfer (Augsburg, Germany). Internal standards—Mirex, PCB 143 (2,2′,3,4,5,6′-hexachlorobiphenyl), polybrominated biphenyl (PBB) 155 (2,2′,4,4′,6,6′-hexabromobiphenyl), and injection standard PBB 103 (2,2′,4,5′,6-pentabromobiphenyl)—were also purchased from Dr. Ehrenstorfer. A standard stock solution containing all seven PBDE congeners was prepared at a concentration of 1 ng μl^{-1} in nonane. Individual standard stock solutions of PBBs 155 and 103 in nonane, and PCB 143 and Mirex in isooctane, were prepared at 10 ng μl^{-1}.

All reagents and solvents were of analytic reagent grade. Iso-octane, n-hexane Suprasolv®, and anhydrous sodium sulphate were obtained from Merck (Darmstadt, Germany). Nonane was purchased from Sigma Aldrich nv/sa (Bornem, Belgium). Acidified silica was prepared by adding 35.5 ml concentrated sulphuric acid p.a. (Merck) to 100 g silica gel (0.063 to 0.200 mm; Merck) and mixing thoroughly. Water was obtained by a Milli-Q gradient system (Millipore, Brussels, Belgium). Preparation of deactivated alumina was done by adding 5 ml water to 45 g alumina B activity I (ICN Biomedicals, Eschwege, Germany). Silane-treated glass wool was obtained from Alltech Associates (Deerfield, IL).

Cleanup of Adipose Tissue

The analysis method was evaluated and fully validated and is described in detail elsewhere (Naert *et al.* 2004). Ten grams adipose tissue was put on a folded Ederol filter paper (VWR, Leuven, Belgium) in a glass funnel together with anhydrous sodium sulphate (10 g). The fat was melted in a microwave oven (600 W for 2 minutes) and received in a glass recipient. Internal standards PBB 155 (40 µl 0.1 ng μl^{-1}), Mirex (20 µl 1 ng μl^{-1}), and PCB 143 (20 µl 10 ng μl^{-1}) were added to the melted fat (2 g). A glass column filled with acidified silica, deactivated alumina, and anhydrous sodium sulphate was used for cleanup. PBDEs and PCBs were eluted with 40 ml n-hexane. The eluate was evaporated in a Rotavapor (Büchi, Switzerland) at 40°C to ca. 4 ml. This solution was transferred to a graduated glass vial (Egilabo, Kontich, Belgium). Injection standard PBB 103 (40 µl 0.1 ng μl^{-1}) and keeper solvent iso-octane were added. This mixture was concentrated under nitrogen at 40°C to 100 µl and divided between two gas chromatography–mass spectrometry (GC-MS) vials.

GC-MS

Analysis of the adipose tissue samples was performed on a Finnigan GCQ gas chromatograph coupled to a Finnigan GCQ mass

Table 2. Concentrations (ng g^{-1} lipid weight) of PBDEs and PCBs in Belgian adipose tissue samples (n = 53)

Sample no.	Age (y)	Sex	PCB sum	PBDE sum
1	19	M	171	7.70
2	21	M	126	2.83
3	22	F	144	5.93
4	23	M	173	2.97
5	27	F	261	3.74
6	27	M	346	6.07
7	28	M	267	8.41
8	30	F	380	4.94
9	30	M	316	3.52
10	31	M	344	7.37
11	32	M	516	10.0
12	34	M	360	7.65
13	34	F	605	12.6
14	37	F	274	5.32
15	37	M	216	2.50
16	38	F	785	9.02
17	40	F	793	7.75
18	42	M	389	5.35
19	44	F	432	2.96
20	44	F	516	19.9
21	44	F	194	1.76
22	46	F	251	1.23
23	47	F	2001	7.27
24	47	F	1033	4.63
25	47	M	602	57.2
26	49	M	1092	29.0
27	50	F	1137	5.16
28	50	M	1118	6.10
29	55	F	1049	3.52
30	60	M	151	5.30
31	61	F	1063	2.55
32	64	M	1077	5.22
33	65	M	816	4.51
34	65	M	529	4.15
35	66	M	997	7.02
36	69	M	472	21.0
37	70	M	1570	6.50
38	70	M	515	5.93
39	71	M	1375	6.19
40	72	F	1092	1.62
41	74	M	912	3.12
42	74	M	1165	9.40
43	74	F	1891	4.90
44	74	M	2090	7.28
45	75	F	1163	2.20
46	75	M	1001	3.59
47	75	F	1100	14.0
48	76	F	535	1.99
49	77	M	542	23.3
50	81	M	812	4.15
51	83	M	1561	3.91
52	83	M	868	3.26
53	84	F	869	6.05

PBDE = Polybrominated diphenyl ether.
PCB = Polychlorinated biphenyl.

spectrometer (Austin, Tx) operating in electron impact – tandem mass spectrometry (MS/MS) mode. A 25 m × 0.22 mm × 0.25 μm HT8 capillary column (SGE, Achrom, Zulte, Belgium) was used for the separation of the PCB and PBDE congeners. A 2-μl aliquot of the final sample extract was injected in the splitless mode (CTC 200 series injector, Zwingen, Switzerland). The oven was programmed from 70°C for 1 minute to 170°C at a rate of 30°C min^{-1}, then to 300°C (15 minutes) at a rate of 8°C min^{-1}.

Quality Control

A six-point calibration curve in matrix was made with every sample sequence by fortifying blank animal fat samples with a mixture of seven PBDE congeners at levels of 0.2, 0.5, 1, 2, 5, and 10 ng g^{-1} per congener and seven PCB congeners at levels of 5, 10, 20, 100, 300, and 500 ng g^{-1} per congener. Because PBDEs were expected to occur in a lower concentration range than PCBs, PBDE calibration curves were determined in a lower linear range.

Given that levels of PCBs 138, 153, and 180 were significantly higher than levels of PCBs 28, 52, 101, and 118, concentrations of the former were calculated with internal standard PCB 143. Concentrations of the latter were calculated with internal standard Mirex.

To be acceptable (Beltest I014-Rev4-17/7/2000-17 2000), the correlation coefficient (R^2) of the calibration curves had to be at least 0.995 for each congener. In every sequence, a blank animal fat sample was subjected to the entire analytic procedure to determine possible contamination.

Retention times, ion chromatograms, and intensity ratios were used as identification criteria. According to Commission Decision 2002/657/EC (2002), relative retention times (RRTs) of the analyte may not differ more than ± 0.5 % of the RRT of the calibration standard. The relative intensities of the detected ions, expressed as a percentage of the intensity of the most abundant ion, must correspond to those of the samples fortified at comparable concentrations within the tolerances mentioned by Commission Decision 2002/657/EC (2002). The signal-to-noise ratio for each diagnostic ion must be >3:1.

Results

A summary of the PBDE and PCB levels found in Belgian human adipose tissue is listed in Tables 2 and 3. PCB- and PBDE-chromatograms of a human adipose tissue sample extract are shown in Figures 1 and 2. The total PBDE concentration (sum of BDEs 28, 47, 99, 100, 153, 154, and 183) in Belgian human adipose tissue ranged between 1.23 and 57.2 ng g^{-1} lipid weight (median 5.32 ng g^{-1} lipid weight). BDEs 47, 153, and 183 were the predominant PBDE congeners and accounted for 83% (range 6 % to 100%) of the total PBDE content. BDE 153 was present in all of the adipose tissue samples. Levels of BDEs 47 and 183 were higher than that of BDE 153 in nine and five samples, respectively. BDEs 28 and 154 were only detected in some of the tissue samples.

The sum of 7 ICES PCB congeners (PCBs 28, 52, 101, 118, 138, 153, and 180) ranged from 126 to 2090 ng g^{-1} lipid weight (median 605 ng g^{-1} lipid weight). PCB congeners 52 and 101 were only detected in a minority of the adipose tissue samples. The sum of PCBs 138, 153, and 180 constituted 76% to 100% of the total sum of PCBs. PCB 153 was the dominant PCB congener in 43 samples, whereas PCB 180 was the most abundant in the remaining 10 samples.

No age dependency was found for PBDEs (Pearson correlation −0.023, $p = 0.873$), whereas PCBs showed higher correlation coefficients with age (Pearson correlation 0.613, $p < 0.0005$) (Fig. 3). PBDEs showed no correlation with the

Table 3. Distribution of PBDE and PCB congeners in Belgian adipose tissue samples (n = 53)

	Mean	Median	Range
Compound		ng g^{-1} lipid weight	
PCB 28	7.2	4.0	< CCα–52
PCB 52	2.4	2.3	< CCα–3.6
PCB 101	4.1	2.6	< CCα–11
PCB 118	27	23	1.9–84
PCB 138	181	149	19–543
PCB 153	310	274	55–848
PCB 180	232	205	11–931
BDE 28	0.40	0.20	< CCα–2.03
BDE 47	2.12	0.88	< CCα–14.3
BDE 99	1.56	0.47	< CCα–7.98
BDE 100	0.80	0.72	< CCα–1.91
BDE 153	3.68	2.40	0.70–25.1
BDE 154	0.93	0.93	< CCα–1.28
BDE 183	1.62	0.78	< CCα–15.4

BDE = Brominated diphenyl ether.
PCB = Polychlorinated biphenyl.

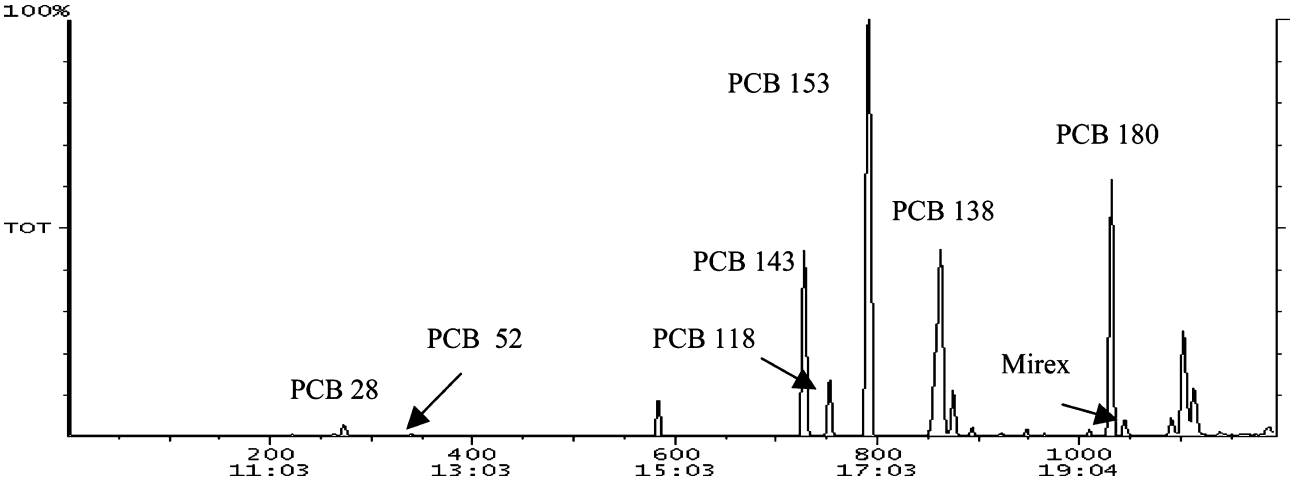

Fig. 1. PCB chromatogram of a human adipose tissue sample extract (sample no. 20)

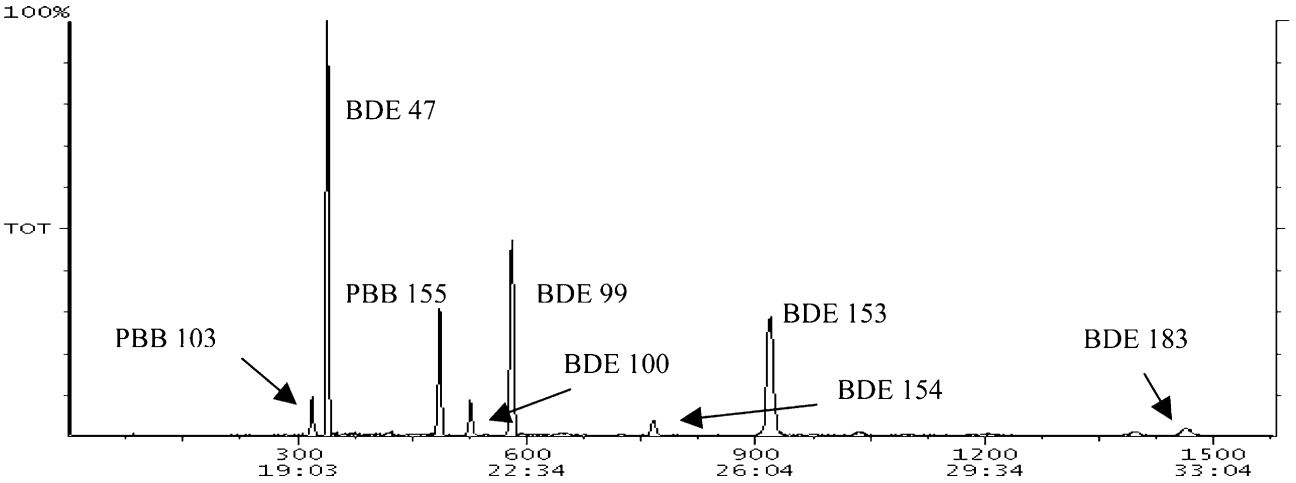

Fig. 2. PBDE chromatogram of a human adipose tissue sample extract (sample no. 20)

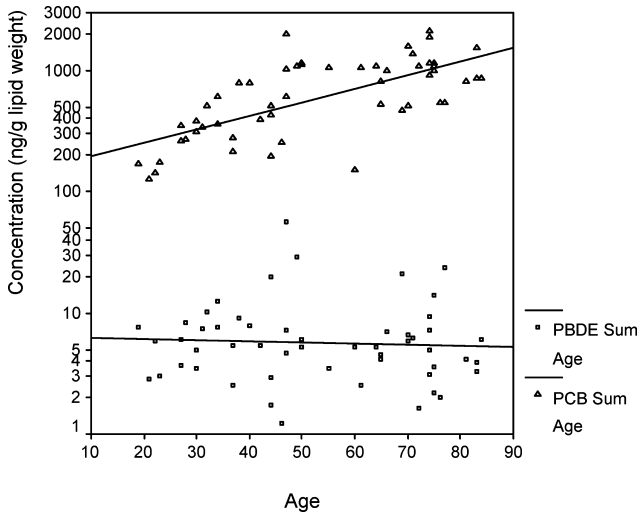

Fig. 3. Scatterplot of PBDE and PCB concentrations (ng g^{-1} lipid weight) in Belgian human adipose tissue samples (n = 53) versus age

sum of PCBs (Pearson correlation −0.010, $p = 0.943$). High Pearson correlation coefficients were found between PCBs 138 (Pearson correlation 0.909, $p < 0.0005$), 153 (Pearson correlation 0.960, $p < 0.0005$), and 180 (Pearson correlation 0.913, $p < 0.0005$) and the sum of PCBs, whereas correlations between BDEs 47 (Pearson correlation 0.685, $p < 0.0005$), 153 (Pearson correlation = 0.815, $p < 0.0005$), and 183 (Pearson correlation 0.809; $p < 0.0005$) and the sum of PBDEs were lower. There was no sex-related difference for PBDEs ($t = -0.526$, $p = 0.601$) and PCBs ($t = 1.297$, $p = 0.201$). Analysis of variance showed no significant province-related difference in total PBDE ($F = 2.937$, $p = 0.102$) and PCB ($F = 2.427$, $p = 0.144$) concentrations.

Discussion

Method

The extraction, cleanup, and GC-MS/MS procedures have been fully evaluated and validated elsewhere (Naert *et al.* 2004). Validation of the method was done according to Commission Decision 2002/657/EC (2002). Specificity, decision limit (CCα), detection capability (CCβ), recovery, and precision were determined. CCα is defined as the limit at and above which it can be concluded with an error probability of α that a sample is noncompliant (α = 1%). CCβ is the smallest content of the substance that may be detected, identified, and/or quantified in a sample with an error probability of β (β = 5%). Decision limits for PBDEs and PCBs ranged from 0.06 to 0.15 ng g^{-1} lipid weight and from 0.35 to 1.22 ng g^{-1} lipid weight, respectively. Detection capabilities were all between 0.23 and 0.55 ng g^{-1} lipid weight for PBDEs and between 0.98 and 2.29 ng g^{-1} lipid weight for PCBs (Table 4). Measurement uncertainty was determined according to the Co-operation on International Traceability in Analytical Chemistry (EURACHEM/CITAC) guide (Ellison *et al.* 2000). Expanded measurement uncertainty varied between 23.5% and 42.8% for PCBs and between 16.5% and 28.2% for PBDEs (Table 4).

Levels of PBDEs in Human Adipose Tissue Samples

Concentrations of PBDEs in 53 adipose tissue samples from the Belgian population were comparable with previously reported data in Europe (Guvenius *et al.* 2001; Covaci *et al.* 2002; Meneses *et al.* 1999), but they were considerably lower than PBDE concentrations in human adipose tissue samples from California (She *et al.* 2002; Petreas *et al.* 2003). According to Petreas *et al.* (2003) this might be because of California regulations mandating that all polyurethane foam and textiles used in furnishings pass a flammability test. Another explanation might be that the relative use of commercial penta-BDE compared with deca-BDE is highest in the United States (Bromine Science and Environmental Forum 2003).

BDE 47 was the predominant congener in human adipose tissue samples from Sweden (Guvenius *et al.* 2001) and the United States (She *et al.* 2002, Petreas *et al.* 2003). In contrast, BDE 153 was the major PBDE congener in the examined Belgian adipose tissue samples. High BDE 153 levels were also present in adipose samples from Spain (Meneses *et al.* 1999), Belgium (Covaci *et al.* 2002), and Japan (Choi *et al.* 2003) and in human milk samples from the Faroe Islands (Fängström *et al.* 2004).

According to Covaci *et al.* (2002), this might be related to the difference in diets containing food items in which the higher brominated (hexa- to deca-BDE) congeners are preferentially bioaccumulated. According to Bocio *et al.* (2003), tetra-BDEs and penta-BDEs constitute 69% of the total dietary intake of PBDEs. In contrast, hexa-BDEs represent only 16% of the total dietary intake. As a consequence, other exposure routes, *e.g.*, dermal uptake and inhalation, could also be at the origin of the elevated BDE 153 levels. The occurrence of these higher BDE 153 levels compared with BDE 47 may also reflect elevated exposure to octa-BDE, which contains more of the higher brominated PBDE congeners (Gill *et al.* 2004).

In accordance with other previously reported data (Sjödin *et al.* 1999; Sjödin *et al* 2003; Covaci *et al.* 2002; Kazda *et al.* 2004; Schecter *et al.* 2003), no significant relationship with age could be observed. This lack of correlation might indicate that PBDE levels are increasing in the West European environment.

Levels of PCBs in Human Adipose Tissue Samples

PCB profiles and levels were comparable with profiles found in Sweden (Guvenius *et al.* 2001), Italy (Mariottini *et al.* 2000), Wales (Duarte-Davidson *et al.* 1994), Spain (Costabeber and Emmanuelli 2003), and Belgium (Covaci *et al*. 2002). In contrast, concentrations (mean 756 ng g^{-1} lipid weight) were higher than levels found by Pauwels *et al.* (2000) (mean 334 ng g^{-1} lipid weight) in Belgian female adipose tissue samples. In that study, the mean age of the subjects (32 years) was considerably lower than in our study (53 years). This supports findings in studies by Costabeber and Emmanuelli (2003), Covaci *et al.* (2002), Duarte-Davidson *et al.* (1994), and our present study that PCB body burden increases with age.

It is known that the PCB pattern shifts from lower to higher chlorinated congeners when organisms move to higher trophic levels. The higher concentrations of PCBs 153 and 180 (persistent congeners) and the absence of lower chlorinated

Table 4. Results of the different validation parameters

	CCα	CCβ	
Compound	ng g^{-1} lipid weight		Expanded measurement uncertainty (%)[a]
PCB 28	0.56	1.75	32.4
PCB 52	0.55	1.70	42.8
PCB 101	0.38	1.25	23.5
PCB 118	0.35	0.98	27.4
PCB 138	0.35	1.28	27.7
PCB 153	0.95	2.21	32.9
PCB 180	1.22	2.29	28.4
BDE 28	0.06	0.23	23.1
BDE 47	0.06	0.31	28.2
BDE 99	0.09	0.30	17.6
BDE 100	0.09	0.40	16.5
BDE 153	0.10	0.55	19.8
BDE 154	0.08	0.46	18.8
BDE 183	0.15	0.53	24.7

[a] Data were obtained at 2 ng g^{-1} level for PBDEs and the 10 ng g^{-1} level for PCBs
BDE = Brominated diphenyl ether.
CCα = Decision limit.
CCβ = Detection capability.

congeners indicate that the principal source of contamination with PCBs was from the diet and not from direct exposure (Chu *et al.* 2003).

However, the PCB profile found in the Belgian human adipose tissue samples was different from that found in Turkey (Çok and Şatiroğlu 2004). The concentrations of PCBs 28, 52, and 101 in the Turkish study were higher than those found in studies from industrialized countries. According to Çok and Şatiroglu (2004), this could be attributable to the fact that exposure to PCBs still continues in Turkey.

Although the province of residence was known, it was not known for how long and in what kind of area (rural or industrialized) the subjects resided. Therefore, no assumption could be made about the correlation between place of residence and PCB or PBDE levels. The lack of correlation between PCBs and PBDEs indicate other exposure pathways of PBDEs compared with PCBs and suggests there could be other important sources of PBDE exposure than food.

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References

Akutsu K, Obana H, Okihashi M, Kitagawa M, Nakazawa H, Matsuki Y, et al. (2001) GC/MS analysis of polybrominated diphenyl ethers in fish collected from the Inland Sea of Seto, Japan. Chemosphere 44:1325–1333

Alaee M, Wenning RJ (2002) The significance of brominated flame retardants in the environment: Current understanding, issues and challenges. Chemosphere 45:579–582

Allchin CR, Law RJ, Morris S (1999) Polybrominated diphenylethers in sediments and biota downstream of potential sources in the UK. Environ Pollut 105:197–207

Beltest I014-Rev4-17/7/2000-17 (2000) Determination of polychlorinated biphenyls in animal feed, animal fat, egg and egg products, milk products and other foodstuffs. Beltest, Belgium

Binelli A, Ricciardi F, Provini A (2004) Present status of POP contamination in Lake Maggiore (Italy). Chemosphere 57:27–34

Bocio A, Llobet JM, Domingo JL, Corbella J, Teixido A, Casas C (2003) Polybrominated diphenyl ethers (PBDEs) in foodstuffs: Human exposure through the diet. J Agric Food Chem 51:3191–3195

Bromine Science and Environmental Forum (2003) Major brominated flame retardants volume estimated: Total market demand by region in 1999 and 2001. Bromine Science and Environmental Forum, Brussels, Belgium

Burreau S, Zebühr Y, Broman D, Ishaq R (2004) Biomagnification of polychlorinated biphenyls (PCBs) and polybrominated diphenyl ethers (PBDEs) studied in pike (*Esox lucius*), perch (*Perca fluviatilis*) and roach (*Rutilus rutilus*) from the Baltic Sea. Chemosphere 55:1043–1052

Choi JW, Fujimaki S, Kitamura K, Hashimoto S, Ito H, Suzuki N, et al. (2003) Polybrominated dibenzo-p-dioxines, dibenzofurans, and diphenyl ethers in Japanese human adipose tissue. Environ Sci Technol 37:817–821

Chu S, Covaci A, Schepens P (2003) Levels and chiral signatures of persistent organochlorine pollutants in human tissues from Belgium. Environ Res 93:167–176

Çok I, Şatiroğlu MH (2004) Polychlorinated biphenyl levels in adipose tissue of primiparous women in Turkey. Environ Int 30:7–10

Commission Decision 2002/657/EC (2002) Official Journal of the European Community, No. L 221/8

Costabeber I, Emmanuelli T (2003) Influence of alimentary habits, age and occupation on polychlorinated biphenyl levels in adipose tissue. Food Chem Toxicol 41:73–80

Covaci A, de Boer J, Ryan JJ, Voorspoels S, Schepens P (2002) Distribution of organobrominated and organochlorinated Contaminants in Belgian Human Adipose Tissue. Environ Res Sec A 88:210–218

Darnerud PO (2003) Toxic effects of brominated flame retardants in man and wildlife. Environ Int 29:841–853

de Boer J, Wester PG, van der Horst A, Leonards PEG (2003) Polybrominated diphenyl ethers in influents, suspended particulate matter, sediments, sewage treatment plant and effluents and biota from the Netherlands. Environ Pollut 122:63–74

Duarte-Davidson R, Wilson S, Jones K (1994) PCBs and other organochlorines in human tissue samples from the Welsh population: I-adipose. Environ Pollut 84:69–77

European Community Council Directive No 2003/11/EC (2003) Official Journal of the European Community, No. L 42/45

Ellison SLR, Rosslein M, Williams A (2000) Quantifying uncertainty in analytical measurement, 2nd ed. EURACHEM/CITAC

Erdoğrul O, Covaci A, Kurtul N, Schepens P (2004) Levels of organohalogentated persistent pollutants in human milk from Kahramanmaras region, Turkey. Environ Int 30:659–666

Fängström B, Strid A, Athanassiadis I, Grandjean P, Weihe P, Bergman Å (2004) A retrospective study of PBDEs in human milk from the Faroe Islands. The Third International Workshop on Brominated Flame Retardants. BFR 2004: 33–36

Focardi S, Fossi C, Leonzio C, Corsolini S, Parra O (1996) Persistent organochlorine residues in fish and water birds from the Biobio River, Chile. Environ Monit Assess 43:73–92

Gill U, Chu I, Ryan JJ, Feeley M (2004) Polybrominated diphenyl ethers: Human tissue levels and toxicology. Rev Environ Contam Toxicol 182:55–96

Guvenius DM, Berman Å, Norén K (2001) Polybrominated diphenyl ethers in Swedish human liver and adipose tissue. Arch Environ Contam Toxicol 40:564–570

Hites R (2004) Polybrominated diphenyl ethers in the environment and in people: A meta-analysis of concentrations. Environ Sci Technol 38:945–956

Johnson A, Olson N (2001) Analysis and occurrence of polybrominated diphenyl ethers in Washington State freshwater fish. Arch Environ Contam Toxicol 41:339–344

Kazda R, Hajšlová , Poustka J, Čajka T (2004) Determination of polybrominated diphenyl ethers in human milk samples in the Czech Republic. Comparative study of negative chemical ionisation mass spectrometry and time-of-flight high-resolution mass spectrometry. Anal Chim Acta 520:237–243

Law RJ, Alaee M, Allchin CR, Boon JP, Lebeuf M, Lepom P, et al. (2003) Levels and trends of polybrominated diphenylethers and other brominated flame retardants in wildlife. Environ Int 29:757–770

Legler J, Brouwer A (2003) Are brominated flame retardants endocrine disruptors? Environ Int 29:879–885

Lind Y, Darnerud PO, Atuma S, Aune M, Becker W, Bjerselius R, et al. (2003) Polybrominated diphenyl ethers in breast milk from Uppsala County, Sweden. Environ Res 93:186–194

Mariottini M, Aurigi S, Focardi S (2000) Congener profile and toxicity assessment of polychlorinated biphenyls in human adipose tissue of Italians and Chileans. Microchem J 67:63–71

Meerts IATM, van Zanden JJ, Luijks EAC, van Leeuwen-Bol I, Marsh G, Jacobsson E, et al. (2000) Potent competitive interactions of some brominated flame retardants and related compounds with human transthyretin in vitro. Toxicol Sci 56:95–104

Meironyté D, Norén K, Bergman Å (1999) Analysis of polybrominated diphenyl ethers in Swedish human milk. A time trend related study, 1972–1997. J Toxicol. Environ Health A 58:329–341

Meironyté D (2002) Organohalogen contaminants in humans with emphasis on polybrominated diphenyl ethers. Doctoral thesis, Karolinska Instituted, Stockholm, Sweden

Meneses M, Wingfors H, Schuhmacher M, Domingo JL, Lindström G, von Bavel B (1999) Polybrominated diphenyl ethers detected in human adipose tissue from Spain. Chemosphere 39:2271–2278

Montone RC, Taniguchi S, Weber RR (2003) PCBs in the atmosphere of King George Island, Antarctica. Sci Total Environ 308:167–173

Naert C, De Saeger S, Van Peteghem (2004) Development of a gas chromatography-ion trap mass spectrometry based method for the quantification of polybrominated diphenylethers and polychlorinated biphenyls in adipose tissue. Rapid Commun Mass Spectrom 18:1–6

Norén K, Meironyté D (2000) Certain organochlorine and organobromine contaminants in Swedish human milk in perspective of past 20-30 years. Chemosphere 40:1111–1123

Orloff KG, Dearwent S, Metcalf S, Kathman S, Turner W (2003) Human exposure to polychlorinated biphenyls in a residential community. Arch Environ Contam Toxicol 44:125–131

Pauwels A, Covaci A, Weyler J, Delbeke L, Dhont M, De Sutter P, et al. (2000) Comparison of persistent organic pollutant residues in serum and adipose tissue in a female population in Belgium, 1996-1998. Arch Environ Contam Toxicol 39:265–270

Petreas M, She JW, Brown FR, Winkler J, Windham G, Rogers E, Zhao GM, et al. (2003) High body burdens of 2,2′, 4, 4′-tetrabromodiphenyl ether (BDE-47) in California women. Environ Health Perspect 111:1175–1179

Safe S (1994) Polychlorinated biphenyls (PCBs): Environmental impact, biochemical and toxic responses, and implications for risk assessment. Crit Rev Toxicol 24:87–149

Sapozhnikova Y, Bawardi O, Schlenk D (2004) Pesticides and PCBs in sediments and fish from the Salton Sea, California, USA. Chemosphere 55:797–809

Schecter A, Vuk MP, Päpke O, Ryan JJ, Birnbaum L, Rosen R (2003) Polybrominated diphenyl ethers (PBDEs) in US mothers' milk. Environ Health Perspect 111:1723–1729

Schecter A, Päpke O, Staskal D, Tung KC, Ryan JJ, Rosen R, et al. (2004) PBDE Contamination of U.S. food and human milk and PBDE, PCDD/F, PCB levels in U.S. Human Blood (1973 and 2003). The Third International Workshop on Brominated Flame Retardants. BFR 2004:27–32

She JW, Petreas M, Winkler J, Visita P, McKinney M, Kopec D (2002) PBDEs in the San Francisco Bay area: Measurements in harbor seal blubber and human breast adipose tissue. Chemosphere 46:697–707

Sjödin A, Hagmar L, Klasson-Wehler E, Kronholm-Diab K, Jakobsson E, Bergman Å (1999) Flame retardant exposure: Polybrominated diphenyl ethers in blood from Swedish workers. Environ Health Perspect 107:643–648

Sjödin A, Carlsson H, Thuresson K, Sjölin S, Bergman Å, Ostman C (2001a) Flame retardants in indoor air at an electronics recycling plant and at other work environments. Environ Sci Technol 35:448–454

Sjödin A, Patterson DG, Berman Å (2001b) Brominated flame retardants in serum from US blood donors. Environ Sci Technol 35:3830–3833

Sjödin A, Patterson DG, Berman Å (2003) A review on human exposure to brominated flame retardants—Particularly polybrominated diphenyl ethers. Environ Int 29:829–839

Thomsen C, Lundanes E, Becher G (2001a) A simplified method for the determination of tetrabromobisphenol A and polybrominated diphenyl ethers in human plasma and serum. J Sep Sci 24:282–290

Thomsen C, Lundanes E, Becher G (2001b) Brominated flame retardants in plasma from three different occupational groups in Norway. J Environ Monit 3:366–370

World Health Organization/International Programme on Chemical Safety (1994) Environmental health criteria 162: Brominated diphenyl ethers. World Health Organization, Geneva, Switzerland