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ScienceDirect
Chemosphere xxx (2007) xxx–xxx
CHEMOSPHERE
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Distribution of polychlorinated biphenyls and polybrominated diphenyl ethers in birds of prey from Switzerland

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Received 31 August 2005; received in revised form 23 December 2006; accepted 5 January 2007

Abstract

Polychlorinated biphenyls (PCBs) and the structurally related polybrominated diphenyl ethers (PBDEs) have been associated with chronic neurotoxicity involving reduced motor activity and impaired attentiveness. Such neurobehavioral effects indicate that the central nervous system may represent an important target organ for the action of these persistent contaminants in wildlife. As a consequence, the brain of different terrestrial and aquatic birds collected in Switzerland was analysed for PCBs and PBDEs. In parallel, the same contaminants were examined in the accompanying adipose tissue. After clean-up by means of glass columns containing acidified silica, deactivated alumina and anhydrous sodium sulphate, the samples were analysed by high resolution gas chromatography/tandem mass spectrometry (HRGC-MS/MS).

Median PCB concentrations in the brain (sum of PCB 28, PCB 52, PCB 101, PCB 118, PCB 138, PCB 153 and PCB 180) ranged between 13 ng g^{-1} wet weight (ww) in blackbirds (*Turdus merula*) and 428 ng g^{-1} ww in sparrow hawks (*Accipiter nisus*). Median PBDE concentrations in the brain (sum of BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154 and BDE 183) ranged from below the decision limit in buzzards (*Buteo buteo*) and blackbirds, to 14 ng g^{-1} ww in sparrow hawks.

After correction for the respective lipid content, higher PCB or PBDE concentrations in brain compared to adipose tissue, were found in three sparrow hawks, four buzzards and in all investigated blackbirds. These results suggest that a deficit in the neuroprotective function of the blood–brain barrier may cause unexpected levels of PCBs and PBDEs in the central nervous system.

Keywords: PCB; PBDE; Birds of prey; Adipose tissue; Brain tissue

1. Introduction

PCBs (polychlorinated biphenyls) and PBDEs (polybrominated diphenyl ethers) are distinct members of the group of persistent organic pollutants (POPs). In the past, PCBs have been used as electric insulators in transformers and capacitors, as heat transfer fluids as well as additives in pesticides, adhesives, plastics and paints (Safe, 1994). PCBs are likely to enter the environment directly from point sources due to inappropriate disposal practices, leakage from industrial facilities or chemical waste disposal sites and, to a lesser extent, from global recycling (Meijer et al., 2003). PBDEs are widely used as flame retardants in textiles, plastics, cars and building materials. The commercial products consist of three distinct technical mixtures, i.e. Penta-BDE, Octa-BDE and Deca-BDE, reflecting the varying degree of bromination of the diphenyl ethers. As these flame retardants are not permanently

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0045-6535/$ - see front matter © 2007 Elsevier Ltd. All rights reserved.
doi:10.1016/j.chemosphere.2007.01.009

bound to the polymer matrix, they leach out during natural operational life as well as during processing, recycling or combustion of the polymeric material (D'Silva et al., 2004). Due to their lipophilicity and resistance to chemical degradation, PCBs and PBDEs are nearly ubiquitously distributed in the environment, food, biota and humans. In addition, PCBs and also some PBDEs are characterized by their tendency to accumulate in lipid-rich tissues and magnify along the food chain, increasing in concentration at each successively higher trophic level (Muir et al., 1992).

Predatory birds respond to relatively low levels of POPs, but their sensitivity varies considerably with respect to compound and species (Herzke et al., 2005). Organochlorine contaminants may impair reproduction, affect the ability to compete for food and habitat, and contribute to reduced parental attentiveness (Ulfstrand et al., 1971; Fry, 1995; Savinova et al., 1995; Vorkamp et al., 2004). For PBDEs, several studies indicate that the most critical effects result from their disturbance of the thyroid hormone homeostasis and from their chronic neurotoxicity, generating behavioural alterations such as deficits in spontaneous motor activity (Porterfield, 2000; Branchi et al., 2002; McDonald, 2002; Darnerud, 2003; Gill et al., 2004; Kodavanti and Ward, 2005).

Since birds of prey are highly positioned on the food chain, they are very suitable to study the bioaccumulation in specific target organs (Fox and Lock, 1978). PCB levels in unhatched eggs of Goshawks from Northern Germany have decreased since the 1970s and are relatively constant during the last 8–10 years (Scharenberg and Looft, 2004). This trend is also visible in gull eggs from the Great Lakes (Stow, 1995). Braune et al. (2001) observed a significant decline of PCB levels in seabird eggs from the Canadian Arctic from 1975 until 1998. In contrast, PBDE levels (sum of BDE 47, BDE 99 and BDE 100) in herring gull eggs from the Great Lakes region have risen exponentially (Norstrom et al., 2002). Similarly, PBDE levels in the eggs of several marine and freshwater birds from British Columbia are increasing with doubling times of 5–6 years (Elliott et al., 2005). This general increase of PBDE levels is also detectable in fish, marine mammals and humans (Hites, 2004). However, few data exist on the level of PCBs and PBDEs in the central nervous system, which constitutes a sensitive target for the toxicological action of both types of contaminants. As a consequence, we determined the PCB and PBDE concentrations in the brain of birds of prey (*Accipiter nisus* and *Buteo buteo*) that were found dead or dying from accidental causes, and compared the values with those from adipose tissue of the same animals. Also the PCB and PBDE levels in these predatory birds were compared with those from an additional terrestrial and an aquatic species. It was our objective to investigate if there is a relationship between brain and adipose tissue concentrations and how efficiently the central nervous system is protected from the deposition of persistent organic pollutants.

2. Experimental

2.1. Sample collection

Sixty eight brain and 50 adipose tissue samples of buzzards (*Buteo buteo*), sparrow hawks (*Accipiter nisus*), cormorants (*Phalacrocorax carbo sinensis*) and blackbirds (*Turdus merula*) from different places in Switzerland were obtained from 2003 until 2005 by the Swiss Ornithological Institute. Sample details are given in Table 1. All birds included in this study were found dead or dying from accidental causes. No bird was killed for the purpose of this study. Due to food deprivation, adipose tissue could not be collected from every specimen. Samples were stored at −20 °C until analysis.

A pool of extracted fat, from blank samples taken within the framework of the Belgian monitoring programme for PCBs, was used as blank for validation and quality control purposes. A sample of this pool was analysed for PBDEs and a new pool was prepared if PBDE levels were above the decision limit (CCα: the limit at and above which can be concluded with an error of probability of α that a sample is non-compliant). Similarly, a pool of porcine brain tissue that contained PCB and PBDE levels lower than the respective decision limits (CCα) was prepared and used as blank. Decision limits (CCα) and detection capabilities (CCβ: the smallest content of the substance that may be detected, identified and/or quantified in a sample with an error probability of β) for PCBs and PBDEs are presented in Table 2.

2.2. Standard solutions

PBDE standards: 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 from Wellington Laboratories (Ontario, Canada). 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′5,5′-hexachlorobiphenyl) and 180 (2,2′,3,4,4′,5,5′-heptachlorobiphenyl) together with PCB-Mix 3 (10 ng μL^{-1} of PCB 28, PCB 52, PCB 101, PCB 118, PCB 138, PCB 153 and PCB 180) were 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 (Augsburg, Germany). A stock solution containing all seven PBDE congeners was prepared at a concentration of 1 ng μL^{-1} in nonane. Individual stock solutions of PBB 155 and PBB 103 in nonane

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Table 1
Details of different birds of prey samples investigated in this study

Sample number	Species	Year of birth	Sex	Found in	Date	Cause of death	Weight (g)
B1	Buzzard	–	–	Sursee	23.05.2004	Thudded	725
B3	Buzzard	–	–	Ennetbürgen	24.01.2004	Starvation	500
B4	Buzzard	–	–	Ebikon	16.01.2004	Starvation	520
B5	Buzzard	–	–	Zell	11.05.2004	Wing trauma, infection	860
B6	Buzzard	–	–	Schötz	26.04.2004	Nerval wing trauma, infection	855
B7	Buzzard	–	–	Zürich	07.07.2004	Nerval wing trauma	620
B8	Buzzard	–	–	Rain	18.06.2004	Hit by car	845
B9	Buzzard	–	–	Reussbühl	19.12.2004	Hit by car	715
B10	Buzzard	–	–	Willisau	17.08.2004	wing fracture	830
B11	Buzzard	–	–	Emmen	15.07.2004	Starvation	620
B12	Buzzard	–	–	Wallisellen	2.03.2004	Wing fracture	790
B13	Buzzard	–	–	Sempach	1.03.2004	Wing fracture	–
B14	Buzzard	–	–	Menziken	4.03.2004	Suspected poisoning	–
B15	Buzzard	–	–	Menziken	4.03.2004	Suspected poisoning	–
B16	Buzzard	–	–	Luzern	10.11.2003	Leg fracture	–
B17	Buzzard	–	–	Schüpheim	3.02.2004	Hit by car, poisoning	–
B18	Buzzard	–	–	Hildisrieden	5.01.2004	Hit by car	–
B19	Buzzard	–	–	Luzern	6.01.2004	Fracture	–
B20	Buzzard	–	–	Schüpheim	3.04.2004	Wing trauma	–
B21	Buzzard	–	–	Wauwilermoos	28.02.2004	Leg trauma	–
B22	Buzzard	–	–	Grosswangen	10.03.2004	Open leg fracture	–
B23	Buzzard	–	–	Hellbrühl	7.03.2004	Wing fracture	–
B24	Buzzard	–	–	Hildisrieden	4.11.2004	Hit by car	–
B25	Buzzard	–	–	Oberkirch	16.02.2005	Femur/joint fracture	700
B26	Buzzard	–	–	Nottwil	8.05.2005	Ratite (back trauma)	690
B27	Buzzard	–	–	Neuenkirch	9.03.2005	Headtilt (hit by car)	730
B28	Buzzard	2004	–	Sempach	21.02.2005	Starvation	535
B29	Buzzard	2004	–	Dietwil	7.05.2005	Starvation	490
B30	Buzzard	2004	–	Neuenkirch	1.03.2005	Leg trauma	675
B31	Buzzard	2005	–	Abtwil	4.03.2005	Ratite (trauma)	895
B32	Buzzard	2005	–	Littau	19.02.2005	Starvation	600
B33	Buzzard	–	–	Rottenschwil	14.03.2005	Lead poisoning	550
B34	Buzzard	–	–	Sarnen	24.05.2005	Joint fracture (wing)	850
B35	Buzzard	2004	–	Kaltbach	26.03.2005	Back and wing trauma	460
B36	Buzzard	2005	–	Luzern	30.05.2005	Open humerus fracture	550
B37	Buzzard	2005	–	Altwis	18.03.2005	Paralysis	685
B38	Buzzard	2005	–	Wald ZH	25.02.2005	Unknown	650
B39	Buzzard	2005	–	Werthenstein	21.02.2005	Craniocerebral injury	575
S1	Sparrow hawk	–	Female	Neuenkirch	18.12.2003	Wing fracture	245
S2	Sparrow hawk	–	–	Neuenkirch	25.03.2004	Starvation	285
S3	Sparrow hawk	–	Female	Weggis	29.10.2003	Wing luxation	232
S4	Sparrow hawk	–	Female	Büron	7.01.2004	Thudded	275
S5	Sparrow hawk	2004	–	Sacheln	19.01.2005	Thudded	250
S6	Sparrow hawk	2002	Male	Eschenbach	18.05.2004	Nerval wing trauma	135
S7	Sparrow hawk	–	Female	Kleinwangen	22.04.2004	Nerval wing trauma	230
S8	Sparrow hawk	–	–	Basel	21.11.2004	Thudded	180
S9	Sparrow hawk	–	Male	Oberkirch	2.11.2004	Hit by car	125
S10	Sparrow hawk	–	Male	Eschenbach	17.02.2005	Thudded	245
S11	Sparrow hawk	–	Male	Rothenburg	30.04.2005	Hit by car	285
C1	Cormorant	2004	–	Lake of Sempach	26.01.2005	Drowned	2000
C2	Cormorant	2003	–	Lake of Sempach	26.01.2005	Drowned	2870
C3	Cormorant	–	–	Lake of Sempach	26.01.2005	Drowned	2650
C4	Cormorant	–	–	Lake of Sempach	26.01.2005	Drowned	3400
C5	Cormorant	–	–	Lake of Sempach	26.01.2005	Drowned	3125
C6	Cormorant	–	–	Lake of Sempach	08.2005	Drowned	2190
C7	Cormorant	–	–	Lake of Sempach	08.2005	Drowned	2595
BB1	Blackbird	–	–	Schlossried	10.11.2003	Thudded	–
BB2	Blackbird	–	–	Luzern	10.01.2004	Victim of cat	–
BB3	Blackbird	–	–	Horw	15.12.2003	Git by car	–
BB4	Blackbird	–	–	Sursee	30.01.2004	Victim of cat	–
BB5	Blackbird	Jung	–	Luzern	–	Unknown	75

(*continued on next page*)

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Table 1 (*continued*)

Sample number	Species	Year of birth	Sex	Found in	Date	Cause of death	Weight (g)
BB6	Blackbird	Adult	–	Zürich	01.07.2005	Unknown	40
BB7	Blackbird	Jung	–	Luzern	–	Unknown	45
BB8	Blackbird	Jung	–	Luzern	–	Unknown	60
BB9	Blackbird	Jung	–	Hochdorf	13.08.2005	Victim of cat	30
BB10	Blackbird	Jung	–	Meggen	13.08.2005	Euthanasia	22.5
BB11	Blackbird	Jung	–	Luzern	–	Unknown	45
BB12	Blackbird	Jung	–	Luzern	–	Unknown	45

Age, sex and weight are not provided for all animals.

Table 2
Decision limits (CCα) and detection capabilities (CCβ) for PCBs and PBDEs in adipose and brain tissue (ng g^{-1} wet weight)

Compound	Brain tissue		Adipose tissue	
	CCα	CCβ	CCα	CCβ
PCB 28	0.81	1.10	1.06	2.24
PCB 52	0.84	1.01	1.54	3.09
PCB 101	0.46	0.54	0.92	1.30
PCB 118	0.43	1.62	0.98	1.23
PCB 138	0.47	0.84	1.06	2.15
PCB 153	0.46	0.57	1.23	2.56
PCB 180	0.93	1.24	0.57	1.35
BDE 28	0.33	0.50	0.25	0.89
BDE 47	0.33	0.49	0.33	0.65
BDE 99	0.47	0.66	0.23	0.62
BDE 100	0.42	0.76	0.22	0.55
BDE 153	0.67	1.14	0.34	0.49
BDE 154	0.65	0.92	0.26	0.58
BDE 183	0.76	0.93	0.45	0.85

as well as PCB 143 and Mirex in isooctane were prepared at 10 ng μL^{-1}.

2.3. Materials and reagents

All reagents and solvents were of analytical grade. Isooctane, *n*-hexane Suprasolv® and anhydrous sodium sulphate were obtained from Merck (Darmstadt, Germany). Nonane was purchased from Sigma Aldrich (Bornem, Belgium). Acidified silica was prepared by adding 35.5 mL concentrated sulphuric acid p.a. (Merck) to 100 g silica gel (0.063–0.200 mm, Merck) and mixing thoroughly. Deactivated alumina was prepared 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, USA).

2.4. Extraction and clean-up

To prevent contamination, we used only glassware that was washed extensively and rinsed twice with hexane (Acros, Geel, Belgium). Plastic materials were not used in order to avoid contamination. Four grams of homogenised brain tissue or two grams of homogenised adipose tissue were dried with anhydrous sodium sulphate (20 g). This mixture was transferred into a hexane-rinsed centrifugation tube. Internal standards PBB 155 (80 μL 0.1 ng μL^{-1}) and PCB 143 (40 μL, 1 ng μL^{-1}) were added in the case of brain tissue and the internal standards PBB 155 (40 μL, 0.1 ng μL^{-1}), PCB 143 (40 μL 5 ng μL^{-1}) and Mirex (40 μL 0.5 ng μL^{-1}) were added in the case of adipose tissue. PBDEs and PCBs were extracted in two steps, by thoroughly shaking the mixture in the centrifugation tube with 25 mL *n*-hexane. After centrifugation for 10 min at 3000 rpm, the hexane extract was evaporated in a rotary evaporator at 40 °C to about 5 mL and cleaned-up as described by Naert et al. (2004); using a glass column that was subsequently filled with *n*-hexane (25 mL), acidified silica (12 g), deactivated alumina (3 g) and anhydrous sodium sulphate (3 g). PBDEs and PCBs were eluted from the column with 40 mL *n*-hexane. The eluate was evaporated in a rotary evaporator at 40 °C to ca. 4 mL. This solution was transferred to a graduated glass vial (Egilabo, Kontich, Belgium). Keeper solvent isooctane (100 μL), the injection standard PBB 103 (brain: 80 μL, 0.1 ng μL^{-1}; adipose: 40 μL, 0.1 ng μL^{-1}) and, in the case of brain tissue samples, also Mirex (40 μL, 1 ng μL^{-1}) were added. This mixture was concentrated under nitrogen at 40 °C to 100 μL and divided over 2 GC–MS vials.

2.5. Gas chromatography–mass spectrometry

Analysis of the samples was done using a GCQ gas chromatograph coupled to a GCQ mass spectrometer (Finnigan, Austin, Texas, USA). The autosampler was a CTC 200 series injector (Zwingen, Switzerland). Separation of the PBDE and PCB congeners was performed using a HT8 capillary column (25 m × 0.22 mm × 0.25 μm, SGE, Achrom, Zulte, Belgium). Carrier and collision gas was Alphagaz 2 helium (Air Liquide, Liege, Belgium). Instrument set points and data acquisition were under control of the GCQ software.

A 2 μL aliquot of the final sample extract was injected with a splitless period after injection of 1 min. The injection temperature was set to 300 °C and transferline temperature was 275 °C. The oven was programmed from 70 °C for 1 min to 170 °C at a rate of 30 °C min^{-1}, then to 300 °C (15 min) at a rate of 8 °C min^{-1}. The method for optimisation of the excitation voltage and of the amount of energy needed to hold the precursor ion in the trap during excita-

tion (q-value) has been described by De Saeger et al. (2005). Additional optimisation of these parameters for PBDE analysis was carried out by a 3-level factorial design (Statgraphics Plus for Windows 3.0, Statistical Graphics Corporation, Englewood Cliffs, NY, USA). Peak area ratios of the most abundant ion in relation to PBB 103 were measured to generate response surface graphs for each component and to locate the optimal excitation voltage and q-value.

2.6. Validation

Validation of the method was carried out according to Commission Decision 2002/657/EC. A quantitative confirmatory method implies that specificity, decision limit (CCα), detection capability (CCβ), recovery and precision need to be determined. Since validation of the method regarding adipose tissue has already been described by Naert et al. (2004) and only a different extraction step was applied, a limited validation procedure to determine decision limit (CCα) and detection capability (CCβ) for adipose tissue was carried out.

With exception of assay specificity, all other validation parameters regarding the analysis of brain tissue were determined on the most abundant ion. To determine decision limits and detection capabilities, five calibration curves with six data points ranging from 0 to 10 ng g^{-1} per PBDE congener and 0–40 ng g^{-1} per PCB congener were obtained. After identification, peak ratios (peak area/internal standard) were plotted against the added concentration. The concentration at the y-intercept plus 2.33 times the standard deviation of the within-laboratory reproducibility of the intercept is defined as the decision limit or CCα ($\alpha = 1\%$). CCβ or detection capability was determined by using the same calibration curves. CCβ equals the concentration at the decision limit plus 1.64 times the standard deviation of the within-laboratory reproducibility of the mean measured at the decision limit ($\beta = 5\%$). To challenge the specificity of the method, at least 10 different blank samples were analysed and checked for interfering compounds in the region of interest where the target analyte is expected to elute. Recovery was determined by fortifying blank samples before extraction and clean-up at three different levels (1, 2 and 4 ng g^{-1} per congener for PBDEs and 5, 20 and 40 ng g^{-1} per congener for PCBs). The results obtained with these samples were compared with those from blanks fortified after clean-up. Within-laboratory coefficients of variation (CV%) were calculated with blank samples fortified as outlined before. Measurement uncertainty was determined according to the EURACHEM/CITAC guide (Ellison et al., 2000). The global uncertainty was calculated as a combination of the uncertainty associated with precision and the uncertainty associated with bias. Although this is the combined uncertainty, the results are expressed as an expanded uncertainty, which corresponds to twice this value.

2.7. Quality control

A six point calibration curve in matrix was made by fortifying blank animal fat and brain samples with a mixture of 7 PBDE congeners at a level of 0.5, 1, 2, 5, 10, 20 ng g^{-1} per congener. Brain was fortified with 7 PCB congeners at 1, 5, 10, 20, 40, 80 ng g^{-1} per congener, whereas adipose tissue was fortified with PCBs at a concentration of 1, 5, 20, 50, 100, 300 ng g^{-1} per congener. Since PBDEs occur in a lower concentration range than PCBs, PBDE calibration curves were determined in a lower linear range. Given that levels of PCB 138, PCB 153 and PCB 180 in adipose tissue were significantly higher than levels of PCB 28, PCB 52, PCB 101 and PCB 118, concentrations of PCB 138, PCB 153 and PCB 180 were calculated using, as internal standard, PCB 143. Concentrations of PCB 28, PCB 52, PCB 101 and PCB 118 were calculated using Mirex as the internal standard. The correlation coefficient for each congener was >0.995 (Beltest I014-Rev4-17/7/2000-17, 2000). A blank adipose or brain tissue sample was subjected to full analysis to rule out possible contaminations. Retention times, ion chromatograms and intensity ratios were used as the identification criteria (Commission Decision, 2002).

3. Results and discussion

3.1. Methodology and validation

Different extraction solvents, e.g. hexane, hexane/acetone (1/1), hexane/acetone/dichloromethane (1/1/1), were tested but only the use of hexane yielded satisfactory recoveries of >80%. The following clean-up of brain and adipose tissue extracts as well as the optimisation of GC-MS/MS parameters has been described elsewhere (Naert et al., 2004). An additional optimisation, using experimental design, was performed to select the most efficient GC–MS/MS settings for PBDE analysis. A response surface plot obtained for BDE 47 is presented in Fig. 1. An overview of optimal excitation voltage and q-value for each PBDE congener is presented in Table 3.

A different extraction step had been added to the previously published clean-up procedure for the analysis of adi-

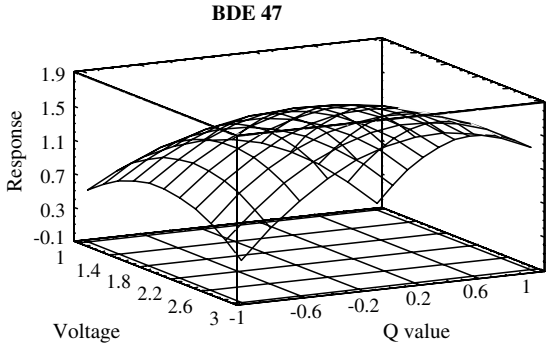

Fig. 1. Response surface plot of BDE 47.

Table 3
Optimal excitation voltage and q-value for each PBDE congener

PBDE congener	q-value	Excitation voltage (V)
BDE 28	0.300	1.5
BDE 47	0.300	2
BDE 99	0.300	2
BDE 100	0.300	2
BDE 153	0.300	2.5
BDE 154	0.300	2.5
BDE 183	0.300	3

pose tissue (Naert et al., 2004). Thus, decision limits (CCα) and detection capabilities (CCβ) for PCBs and PBDEs in adipose tissue were reassessed. In parallel, a full validation procedure according to Commission Decision 2002/657/EC was carried out for the analysis of brain tissue. Decision limits and detection capabilities for PCBs and PBDEs in adipose and brain tissue are presented in Table 2. Specificity of the method was inferred from the fact that no interfering peaks were detected when analysing 10 blank brain samples. Under repeatability conditions, within-laboratory coefficients of variation (CV%) were between 2.1% and 14% for PCB congeners and between 3.2% and 12% for PBDE congeners. Under within-laboratory reproducibility conditions, these coefficients ranged between 3.6% and 15% for PCBs and between 4.1% and 16% for PBDEs. Such low CV% values are in accordance with Commission Decision 2002/657/EC stating that for mass fractions $<100\ \mu g\ kg^{-1}$ simple application of the Horwitz equation would not be acceptable. Recovery of individual PCB congeners varied from 92% to 107% and recovery of individual PBDE congeners ranged from 85% to 109%. The expanded measurement uncertainty varied from 36% to 65% for PCBs and from 9.1% to 16% for PBDEs.

3.2. Influence of the nutrition status on the PCB and PBDE levels in brain and adipose tissue

Because in some cases the amount of sample was insufficient to determine the lipid content, a wet weight basis was chosen for calculating PCB and PBDE concentrations. Whenever possible, the percentages of the hexane extractable lipids in the tissue probes were determined. These measurements yielded a lipid content in the brain of $7.6 \pm 2.3\%$. The proportion of lipid in adipose tissue was $78 \pm 5\%$. In starved animals, these lipid contents in brain and adipose tissue remain unchanged, but the amount of adipose tissue diminishes such that the lipophilic pollutants that were stored in the adipose compartment of the body are remobilised to other sites including the brain (Voorspoels et al., 2004; Wienburg and Shore, 2004). Indeed, many buzzards displayed only a limited amount of adipose tissue and the brain of these buzzards contained higher PCB and PBDE levels than the brain of well-fed counterparts (PCB: $t = 3.559$, Sig. $= 0.003$; PBDE: $t = 2.162$, Sig. $= 0.037$). In addition, partial correlation coefficients were calculated to explore a possible relationship between body weight, corrected for each species, and contaminant levels. In three bird species, e.g. buzzards, sparrow hawks and cormorants, negative correlations could be established between the overall body weight and the concentration of PCB and PBDE in both brain and adipose tissue.

3.3. PCB and PBDE levels in brain and adipose tissue

Because the PCB and PBDE concentrations did not follow normal distributions, median values were used for further comparisons (Table 4). The median PCB concentrations (sum of PCB 28, PCB 52, PCB 101, PCB 118, PCB 138, PCB 153 and PCB 180) in the brain ranged from 13 ng g^{-1} wet weight (ww) in cormorants to 428 ng g^{-1} ww in sparrow hawks. In adipose tissue, the median PCB concentrations ranged from below the decision limit in blackbirds to 25951 ng g^{-1} ww in sparrow hawks. The median PBDE concentrations (sum of BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154 and BDE 183) in brain ranged from below the decision limit in blackbirds to 14 ng g^{-1} ww in sparrow hawks. Similarly, the median PBDE concentrations in adipose tissue ranged from below the decision limit in blackbirds to 709 ng g^{-1} ww in sparrow hawks. A summary of the PCB and PBDE levels found in brain and adipose tissue of all animals from the four investigated species is presented in Table 5. Overall, significant correlations could be established between PCB and PBDE levels in brain tissue (Spearman's rho $= 0.753$, Sig. < 0.0005) and between PCB and PBDE levels in adipose tissue (Spearman's rho $= 0.944$, Sig. < 0.0005).

Among the four bird species tested, sparrow hawks showed the highest PCB and PBDE levels. The lowest PCB and PBDE contamination was found in blackbirds. In general, PCB and PBDE concentrations in the brain were lower than in the adipose tissue. This overall trend was retained when lipid-normalized concentrations between brain and adipose tissue were compared. The heterogeneous partitioning between brain and adipose tissue could be ascribed to the blood–brain barrier, which is thought to protect against the accumulation of persistent organic pollutants. In fact, a key role of the blood–brain barrier in preventing the deposition of contaminants in the brain is indicated by the relative uniform concentration of PCBs throughout the various organs of fish, which are known to display a much more permeable blood–brain barrier (Bachour et al., 1998). Alternatively, the lower PCB or PBDE levels in the brain may be related to the different proportion of neutral lipids such as triglycerides. While in fat triglycerides constitute >90% of the total lipid content, in the brain the main lipid constituents are cholesterol and phospholipids (Covaci et al., 2004). Intriguingly, however, at least one sparrow hawk (S6) and three blackbirds (BB1, BB2 and BB4) displayed higher absolute PCB and PBDE concentrations in the brain tissue than in adipose tissue. One buzzard (B27) and an additional blackbird (BB3) displayed higher PBDE levels in the brain than in the adipose tissue. Another four blackbirds (BB7, BB8,

Table 4
Median PCB and PBDE levels (ng g^{-1} wet weight) in brain and adipose tissue samples from three different kinds of birds of prey species and blackbirds

Compound	Buzzard		Sparrow hawk		Cormorant		Blackbird	
	Brain tissue	Adipose tissue	Brain tissue	Adipose tissue	Brain tissue	Adipose tissue	Brain tissue	Adipose tissue
PCB 28	<CCα (<CCα – 6.9)	2.8 (<CCα – 22)	3.3 (<CCα – 14)	2.9 (<CCα – 36)	0.98 (<CCα – 6.1)	59 (23–158)	<CCα (<CCα – 20)	<CCα (<CCα – 0.88)
PCB 52	<CCα	<CCα (<CCα – 41)	1.9 (<CCα – 7.7)	7.4 (<CCα – 56)	<CCα	7.6 (3.7–14)	<CCα (<CCα – 4.9)	<CCα
PCB 101	<CCα (<CCα – 11)	9.0 (<CCα – 61)	6.2 (3.0–85)	280 (11–1315)	0.62 (<CCα – 0.96)	42 (17–91)	<CCα (<CCα – 5.2)	<CCα
PCB 118	2.0 (<CCα – 56)	36 (<CCα – 174)	27 (7.1–285)	1289 (61–7417)	15 (4.4–23)	872 (481–2027)	<CCα (<CCα – 14)	<CCα (<CCα – 3.0)
PCB 138	9.1 (<CCα – 246)	293 (39–947)	142 (50–1266)	6700 (253–24019)	30 (11–83)	2276 (1405–5675)	3.4 (<CCα – 19)	<CCα (<CCα – 39)
PCB 153	12 (2.3–447)	444 (79–2816)	164 (89–3624)	8572 (806–25543)	35 (13–70)	3758 (1647–7046)	6.7 (0.73–112)	<CCα (<CCα – 22)
PCB 180	10 (<CCα – 240)	332 (26–1389)	98 (45–2169)	6700 (620–21452)	11 (3.6–37)	1559 (523–3468)	3.3 (1.1–45)	<CCα (<CCα – 13)
BDE 28	<CCα	<CCα (<CCα – 0.42)	<CCα (<CCα – 1.3)	<CCα (<CCα – 2.0)	<CCα	<CCα (<CCα – 0.49)	<CCα	<CCα
BDE 47	<CCα (<CCα- 6.9)	6.2 (<CCα – 24)	3.9 (1.1–40)	199 (15–553)	0.54 (<CCα – 1.3)	41 (21–71)	<CCα (<CCα – 24)	<CCα (<CCα – 0.82)
BDE 99	<CCα (<CCα – 7.8)	4.3 (<CCα – 41)	4.4 (1.1–80)	2 60 (20–690)	<CCα	20 (11–24)	<CCα	<CCα
BDE 100	<CCα (<CCα – 3.0)	1.9 (<CCα – 4.8)	2.0 (<CCα – 29)	114 (9.8–212)	<CCα	20 (3.1–31)	<CCα (<CCα – 3.6)	<CCα
BDE 153	<CCα (<CCα- 10)	5.0 (0.45–76)	0.80 (<CCα – 27)	78 (7.4–153)	<CCα	8.9 (1.9–12)	<CCα	<CCα
BDE 154	<CCα (<CCα – 2.1)	1.2 (<CCα – 13)	1.3 (<CCα – 6.2)	27 (4.3–73)	<CCα	9.0 (1.7–26)	<CCα	<CCα
BDE 183	<CCα (<CCα – 6.1)	0.50 (<CCα – 40)	1.0 (<CCα – 2.0)	30 (4.1–90)	<CCα	<CCα	<CCα	<CCα

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Table 5
Concentrations (ng g^{-1} wet weight) of PCBs and PBDEs in birds of prey and blackbird from Switzerland

Sample number	Species	Brain tissue		Adipose tissue	
		Sum PCB	Sum PBDE	Sum PCB	Sum PBDE
B1	Buzzard	17	<CCα	173	8.6
B3	Buzzard	171	1.8	–	–
B4	Buzzard	622	13	–	–
B5	Buzzard	5.7	<CCα	179	3.0
B6	Buzzard	9.5	<CCα	770	21
B7	Buzzard	6.6	0.68	566	8.7
B8	Buzzard	72	0.84	1031	25
B9	Buzzard	7.6	0.68	392	24
B10	Buzzard	35	17	2045	185
B11	Buzzard	950	13	–	–
B12	Buzzard	8.7	0.70	435	16
B13	Buzzard	14	<CCα	943	16
B14	Buzzard	11	<CCα	1367	19
B15	Buzzard	12	<CCα	1339	26
B16	Buzzard	6.9	11	3471	19
B17	Buzzard	12	<CCα	1563	30
B18	Buzzard	2.3	<CCα	470	5.2
B19	Buzzard	261	5.6	5351	65
B20	Buzzard	12	<CCα	1932	17
B21	Buzzard	57	<CCα	–	–
B22	Buzzard	181	3.9	–	–
B23	Buzzard	127	0.74	–	–
B24	Buzzard	121	0.87	–	–
B25	Buzzard	52	0.96	2318	36
B26	Buzzard	15	<CCα	1477	50
B27	Buzzard	289	6.0	749	5.3
B28	Buzzard	384	6.8	–	–
B29	Buzzard	249	5.6	–	–
B30	Buzzard	27	<CCα	1026	13
B31	Buzzard	5.6	<CCα	1180	8.7
B32	Buzzard	222	5.6	–	–
B33	Buzzard	761	31	–	–
B34	Buzzard	22	2.1	2177	76
B35	Buzzard	188	1.7	302	9.9
B36	Buzzard	192	8.1	–	–
B37	Buzzard	17	<CCα	1476	28
B38	Buzzard	192	2.0	–	–
B39	Buzzard	31	<CCα	2177	70
S1	Sparrow hawk	207	4.6	18788	526
S2	Sparrow hawk	7439	168	12362	352
S3	Sparrow hawk	344	4.6	2599	64
S4	Sparrow hawk	776	12	50093	997
S5	Sparrow hawk	231	13	26025	1052
S6	Sparrow hawk	4452	154	1789	109
S7	Sparrow hawk	428	8.9	25951	709
S8	Sparrow hawk	2327	62	–	–
S9	Sparrow hawk	2105	57	–	–
S10	Sparrow hawk	333	14	79836	1637
S11	Sparrow hawk	390	15	50994	1660
C1	Cormorant	71	1.3	5527	141
C2	Cormorant	91	0.63	10147	99
C3	Cormorant	145	0.60	17921	133
C4	Cormorant	32	<CCα	4186	40
C5	Cormorant	38	0.54	4262	108
K6	Cormorant	119	<CCα	8386	101
K7	Cormorant	210	<CCα	15199	69
BB1	Blackbird	41	27	0.88	<CCα
BB2	Blackbird	198	15	<CCα	<CCα
BB3	Blackbird	8.8	1.0	68	0.82

(*continued on next page*)

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Table 5 (*continued*)

Sample number	Species	Brain tissue		Adipose tissue	
		Sum PCB	Sum PBDE	Sum PCB	Sum PBDE
BB4	Blackbird	62	1.4	<CCα	<CCα
BB5	Blackbird	34	<CCα	44	<CCα
BB6	Blackbird	60	<CCα	–	–
BB7	Blackbird	9.2	<CCα	<CCα	<CCα
BB8	Blackbird	11	<CCα	<CCα	<CCα
BB9	Blackbird	4.5	<CCα	–	–
BB10	Blackbird	13	<CCα	<CCα	<CCα
BB11	Blackbird	2.7	<CCα	<CCα	<CCα
BB12	Blackbird	13.3	<CCα	–	–

BB10 and BB11) contained higher absolute concentrations of PCB in the brain than in adipose tissue. Moreover, after correction for the varying total lipid content, higher PCB levels in brain fat compared to adipose fat were detected in three buzzards (B1, B27 and B35), three sparrow hawks (S2, S3 and S6) and in all investigated black birds. In some cases, the same animals also contained higher PBDE concentrations in brain fat compared to adipose fat. These observations indicate that there are important interspecies and individual differences in the susceptibility for accumulation of persistent organic pollutants in the central nervous system.

3.4. Differences in PCB and PBDE levels between species

ANOVA and Bonferroni-adjusted comparisons were performed to determine differences in mean PCB and PBDE levels between species. Significant differences in PCB and PBDE levels could be established between sparrow hawks, on the one hand and buzzards, cormorants

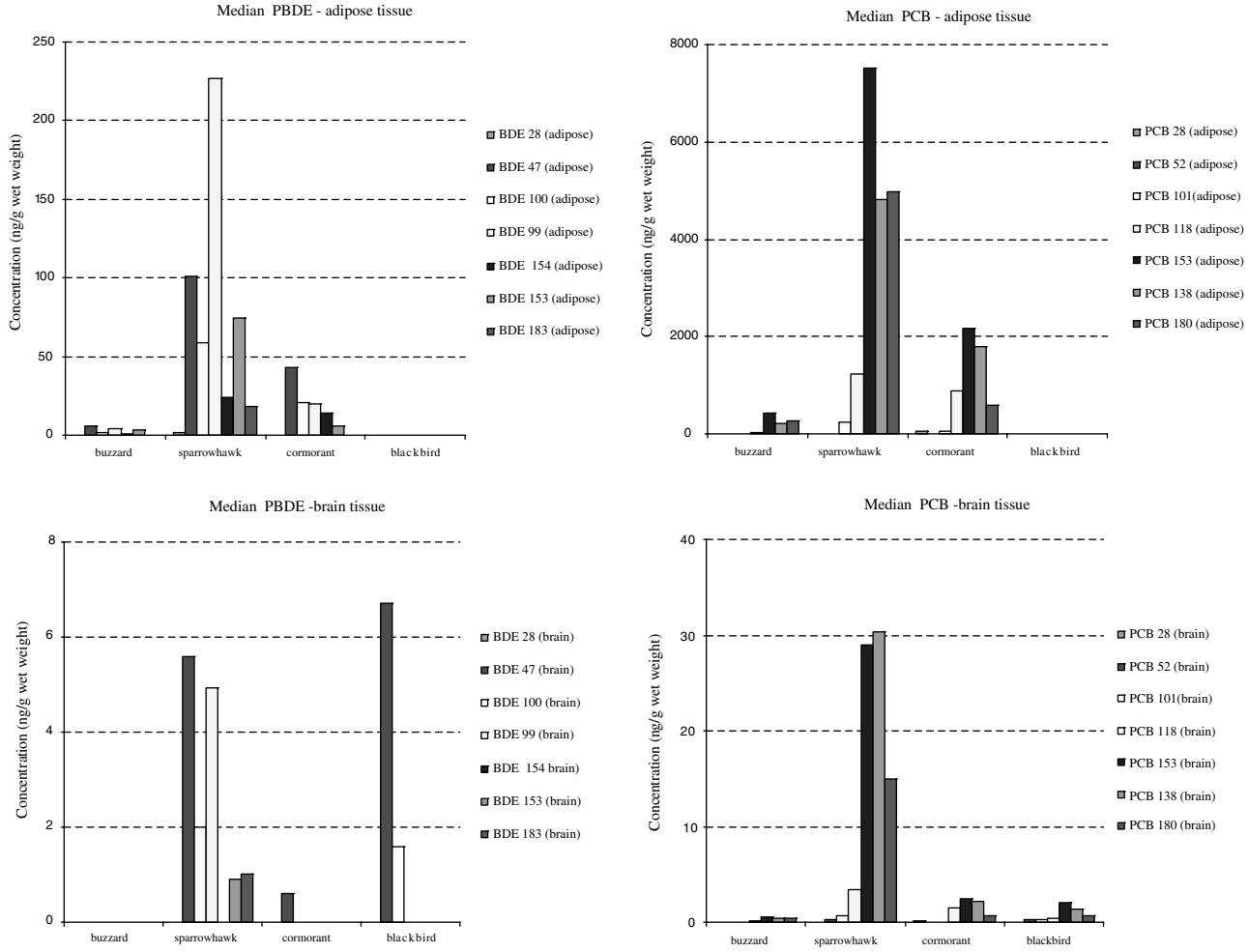

Fig. 2. Median PCB and PBDE patterns in adipose and brain tissue of birds of prey and blackbird from Switzerland.

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and blackbirds on the other hand. Significant interspecies disparities in tissue levels have also been reported by Hoshi et al. (1998), Senthilkumar et al. (2002), Tanabe et al. (1998) and Wienburg and Shore (2004). Despite the fact that both buzzards and sparrow hawk are feeding in a terrestrial environment, the differences in PCB and PBDE levels and congener patterns between buzzard and sparrow hawk are much more pronounced than those between buzzard and fish eating cormorants.

PCB 138, PCB 153 and PCB 180 were the predominant PCB congeners in adipose and brain tissue of all species accounting for 91% (ranging from 56% to 100%) and 93% (ranging from 82% to 98%) of the total PCB content, respectively (Fig. 2). This finding was comparable to congener patterns found in eggs from Norwegian predators (Herzke et al., 2005), in tissues from British birds (Boumphrey et al., 1993) and in fulmars from the Faroe Islands (Fängström et al., 2005). It is known that the PCB pattern shifts from lower to higher chlorinated congeners when these pollutants are transferred to organisms of higher trophic levels (Chu et al., 2003). BDE 47, BDE 99, BDE 100 and BDE 153 were the major congeners in adipose and brain tissue of all species. This observation is reminiscent of the PBDE profiles found in birds of prey from Australia (Symons et al., 2004), in eggs of little owls from Belgium (Jaspers et al., 2005), in eggs of Norwegian predatory birds (Herzke et al., 2005) and in birds of prey of Flanders (Voorspoels et al., 2004). Nevertheless, specific differences in the contribution of the different PBDE congeners between species could be observed in the present study (Fig. 2). In fact, BDE 99 was the most abundant congener in samples from sparrow hawks, in contrast to cormorants where BDE 47 was the most abundant congener. It has been suggested by Law et al. (2003) that birds feeding in terrestrial environments may be more exposed to the higher brominated BDE congeners than aquatic species. The heptabrominated diphenyl ether congener 183 was detected only in terrestrial birds of prey (Fig. 2).

Acknowledgement

The authors would like to thank Consella Ochieng for her technical support and enthusiasm.

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