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Human exposure to polybrominated diphenyl ethers (PBDE), as evidenced by data from a duplicate diet study, indoor air, house dust, and biomonitoring in Germany

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ABSTRACT

Polybrominated diphenyl ethers (PBDE) are used as flame retardants in a wide variety of products. As part of the Integrated Exposure Assessment Survey (INES), this study aimed to characterize the exposure of an adult German population using duplicate diet samples, which were collected daily over seven consecutive days, and indoor air and house dust measurements. Our study population consisted of 27 female and 23 male healthy subjects, aged 14–60 years, all of whom resided in 34 homes in southern Bavaria. In these 34 residences the air was sampled using glass fiber filters and polyurethane foams and the dust was collected from used vacuum cleaner bags.

The median (95th percentile) daily dietary intake of six Tetra- to HeptaBDE congeners was 1.2 ng/kg b.w. (3.3 ng/kg b.w.) or 67.8 ng/day (208 ng/day) (calculated from the 7-day median values of each study subject). Concentrations in indoor air and dust (cumulative Tri- to DecaBDE congener readings) ranged from 8.2 to 477 pg/m³ (median: 37.8 pg/m³) and 36.6 to 1580 ng/g (median: 386 ng/g), respectively. For some congeners, we identified a significant correlation between air and dust levels.

The median (95th percentile) blood concentration of total Tetra- to HexaBDE congener readings was 5.6 (13.2) ng/g lipid. No significant sex differences were observed, but higher blood concentrations were found in younger participants. Using a simplified toxicokinetic model to predict the body burden from exposure doses led to results that were of the same order of magnitude as the measured blood concentrations.

Based on these measurements and given our exposure assumptions, we estimated for the total tetra- to heptabrominated congener count an average (high) comprehensive total daily intake of 1.2 ng/kg b.w. (2.5 ng/kg b.w.). Overall, our results suggest that dietary exposure is the dominant intake pathway at least in our study population, responsible for 97% (average intake) and 95% (high intake) of the total intake of an adult population.

1. Introduction

Polybrominated diphenyl ethers (PBDE) are a class of substances with 209 individual congeners depending on the location and number of bromine atoms. These compounds do not occur naturally in the environment and have been produced for several decades primarily in the form of three technical mixtures (penta-, octa- and decabromo diphenyl ethers). PBDE are used as additive flame retardants in the production of various plastics and textiles. They can be found in electronic devices such as computers and TV sets as well as in upholstery and carpets (NICNAS, 2001).

Since August 2004, the use and import of products containing more than 0.1% penta- or octabromo diphenyl ethers has been banned in the European Union (EU, 2003). In the EU, the Restriction of Hazardous Substances (RoHS) Directive came into force in 2003, requiring that new electrical and electronic equipment on the market from this date forward would not contain PBDE, including DecaBDE, in quantities exceeding maximum prescribed concentrations (RoHS, 2003). In 2005, DecaBDE was exempted from this restriction by a Commission Decision 2005/717/EC; however, in April 2008, the European Court of Justice annulled the exemption decision. Before this court decision, certain member states like Sweden had banned the use of DecaBDE. In Germany, the industrial manufacturers had voluntarily agreed to phase out PBDE in 1986. In 1993, Germany placed official limits on PBDE use in plastic production under its Dioxin Ordinance to protect workers' health because of the tendency of PBDE to form

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brominated dioxins and furans during thermal processes including extrusion of plastics.

In the US, California became the first state to ban PBDE in 2003. This legislation banned penta- and octabromo diphenyl ethers beginning in 2006. Since then, at least eight additional states have followed with similar bans.

A number of studies have suggested that PBDE are highly persistent, bioaccumulative and globally distributed in the environment. They have been found in human tissues (NICNAS, 2001; Law et al., 2008). Our toxicological knowledge of PBDE is limited and is focused mainly on mixtures — we have much less information about individual congeners (NICNAS, 2001; HC, 2006). Some animal studies have suggested that the critical effects are exhibited on the liver, in development, neurodevelopment and in the endocrine system (NICNAS, 2001; HC, 2006; Kuriyama et al., 2007). For example, rats that are pre- and perinatally exposed to relatively low doses of pentabrominated BDE 99 presented disturbed hormone levels. The corresponding tissue concentrations were not much higher than the levels in the adipose tissue of non-occupationally exposed humans (Kuriyama et al., 2007).

Globally, differences in production, use and policy regulations have led to a broad distribution of PBDE throughout the environment but also to marked geographical differences (Frederiksen et al., 2009). The recent exposure of the human population is complex. Scientists long believed that dietary intake is the most important source of human exposure, but there are a growing number of studies showing that indoor environments should be considered as well (Lorber, 2008; Law et al., 2008; Frederiksen et al., 2009).

The goal of our study was to improve the knowledge of the various routes of human exposure to PBDE in Germany and to estimate the contributions to total exposure as accurately as possible. The aim was to measure PBDE congeners in duplicate diet samples, in blood samples and in indoor air and dust samples.

Duplicate diet samples and blood samples were analyzed for the 17 tri- to heptabrominated congeners BDE 17, -28, -47,-49, -66, -71, -77, -85, -99,-100, -119, -126, -138, -153, -154, -183. BDE 209 was not measured in the blood and duplicate diet samples.

Indoor air and dust was collected from the participants' residences and analyzed quantitatively for the 16 tri- to decabrominated congeners BDE 17, -28, -47, -66, -71, -77, -85, -99, -100, -119, -138, -153, -154, -183, -190, and -209. The first 13 are main congeners of technical pentabromo diphenyl ether while BDE 183 and 190 are major congeners of technical octabromo diphenyl ether and BDE 209 is the main congener of technical decabromo diphenyl.

The duplicate diet, dust, and air samples were converted into intakes, and these intakes were used to predict the net body burden using a simplified toxicokinetic model. This study is part of the Integrated Exposure Assessment Survey (INES) that aims to create a framework for an integrated exposure assessment approach. The research includes data from different organic pollutants across various environmental media, in food, and in the context of net body burdens (Fromme et al., 2007a).

2. Materials and methods

2.1. Study population, location

The study population was comprised of 27 female and 23 male healthy adults who had never been occupationally exposed to the target analytes. The female participants were between 14 and 60 years old (median: 35 years) and the male subjects were between 15 and 56 years of age (median: 33 years). Body weights ranged from 42 to 90 kg (median: 66 kg) for women and from 50 to 107 kg (median: 76 kg) for men. The study was carried out in the city of Munich and in nearby suburban and rural areas of southern Germany.

The study protocol was approved by the ethics committee of the Bavarian Chamber of Physicians, and all participants provided their written informed consent.

2.2. Food duplicate sampling

Daily duplicate food portions ($n = 350$) prepared as for consumption were collected between April and October of 2005. The participants collected daily duplicate diet samples over 7 consecutive days, including one weekend. The study subjects obtained information sheets about study objectives and sampling procedure and were trained to collect the samples just before the 7 days-period. All study subjects consumed a normal mixed diet. Solid and liquid foods were collected as separate composite samples in precleaned 2 L glass jars. They were stored in a cooler with ice packs during the sampling day and transported daily to the laboratory where non edible parts were removed and the total amount of food and drink was weighed. All samples were homogenized on the same day in the laboratory and stored at −20 °C until analysis. A detailed description of the study conditions was given previously (Fromme et al., 2007b,c).

2.3. Food analysis

Food samples were allowed to thaw at room temperature and thoroughly mixed. About 30 g of homogenized food samples were dried with 150 g anhydrous sodium sulfate in a glass column, which was filled with 1 cm sea sand layer. After 2 h, the lipid portion was extracted from the solid phase using a n-pentane/acetone mixture (50/50) (v/v), and determined gravimetrically after the solvent had been removed in a rotation evaporator. Thereafter, the lipid was redissolved in 10 ml of n-hexane. To the solution, 1000 pg of native Mirex was added as internal standard. The solution then was treated with sulfuric acid at 60 °C for 1 h. The organic phase was separated and dried in a nitrogen stream. The dried sample was redissolved in 300 µl n-hexane and fractionated in two parts using a Pasteur pipette, which served as a small column (filled with 2.2 g of activated aluminium oxide). The first fraction was eluted with n-hexane and the second fraction, with a mixture of dichloromethane and n-hexane (50/50) (v/v). The second fraction, which contained PBDE, was dried in a nitrogen stream, dissolved in n-hexane, and additionally purified with 1 g of silica gel in another small column using n-hexane as the mobile phase. Finally, the purified sample was completely dried and dissolved in 50 µl of isooctane.

Sample analysis was performed using GC/MS (HP 5890 Series II GC, Finnigan Mat, model 8200 MS). A 30 m long DB 5 column with ID of 0.25 mm and a film thickness of 0.25 µm was used. The injection was splitless with an injector temperature of 300 °C. The applied temperature program was as follows: start with 90 °C, held for 1 min, increased up to 250 °C (temperature rate of 20 °C/min), held for 5 min, increased again up to 300 °C (2 °C/min), and held for 6 min. Helium 5.0 served as carrier gas with a flow rate of 1.23 ml/min. The transfer line temperature was 250 °C.

The ion source temperature of the MS was 210 °C. The electron impact ionization was performed with an electron energy of 70 eV. Perfluorokerosene was used as a reference compound. The PBDE were detected in the SIM mode.

As to the quality assurance, the detection limit of the analyzed PBDE using GC/MS was determined by diluting PBDE standard solution with n-hexane until their corresponding signal to noise ratios in the mass fragmentograms were 3. The limit of detection for each sample was calculated in a concentration unit (pg/g lipid) by dividing the detection limit of each congener by lipid weight of each sample. In order to rule out any mistakes in the determination of PBDE, which could derive from contaminated laboratory glass equipments or solvents, and to ensure a good quality control, a blank sample was inserted in each prepared sample series. Then the PBDE values of the blank samples were subtracted from

the total values of the analyzed sample of the series. Each sample was analyzed twice and the tri- to heptabrominated congeners were quantified. By the analytical method used, it was not possible to obtain reproducible results for the analysis of BDE 209 in diet samples, since this congener is too heavy to be vaporized and separated by applying GC techniques. Therefore, we have included BDE 209 in the analysis.

2.4. Sampling and analysis of blood samples

Blood was collected once from each participant during the time course of the duplicate study by venipuncture. After centrifugation the serum was immediately frozen until analysis.

About 30 g of homogenized serum were dried with 150 g anhydrous sodium sulfate in a glass column, which was filled with 1 cm sea sand layer. Afterwards, 100 µl of a standard solution containing 2,2′,4,4′-Tetrabromo[$^{13}C_{12}$]-diphenylether (BDE 47), 2,2′,4,4′,5-Pentabromo [$^{13}C_{12}$]diphenylether (BDE 99) and 2,2′,4,4′,5,5′-Hexabromo[$^{13}C_{12}$] diphenylether (BDE 153) with a concentration of 30 pg/µl was inserted to the dried sample in the column. Afterwards, the samples underwent the same analytical procedure as described for food samples and the same congeners were quantified. For the blood samples, the same quality assurance procedure was applied as described for food samples.

2.5. Sampling and analysis of air and dust samples

In the 34 residences in which the 50 participants lived, air was sampled for 24 h in the living room using a medium volume sampler with a flow rate of 4.0 m³/h. The position of sampler and cartridge was in accordance to the international guideline EN ISO 16000-12., (2008). The sampling cartridge of stainless steel was filled with a glass fibre filter ($d = 50$ mm) and two cylindric polyurethane foams (PUF) ($d = 55$ mm, $h = 50$ mm). PUFs were pre-cleaned according to VDI 3498-2 (VDI 2002) and wrapped in aluminium foil. Before sampling the first PUF was spiked with 10 ng of $^{13}C_{12}$-BDE 139 as sampling standard. The residents were asked to air the living room as usual during sampling. Duration and type of airing was documented as well as the residence of persons in the living room during sampling.

The whole sample preparation was performed in amber glass ware. PUFs and filter were extracted together in a soxhlet apparatus with toluene for 20–24 h after addition of 10 ng $^{13}C_{12}$-BDE 28, -47, -99, -100, -153, -154, -183 and 50 ng $^{13}C_{12}$-BDE 209 as internal standards. The extracts were first purified on a mixed silica column (3 g silica 60/33% 1 M sodium hydroxide and 12 g silica/44% conc. sulfuric acid) eluted with 170 ml of n-heptane. A fractionation on 5 g alumina B Super I followed. After pre-elution with 50 ml of a 98/2 (v/v) mixture of n-hexane and dichloromethane the PBDE were eluted with 50 ml of a 50/50 (v/v) mixture of n-hexane/dichloromethane. The PBDE fraction was dried in a nitrogen stream and spiked with 10 ng $^{13}C_{12}$-BDE 138 as recovery standard before GC/MS analysis.

One vacuum cleaner bag was collected from each household on the day of indoor air sampling. The samples were sieved <2 mm according to the German guideline VDI 4300-8 (VDI 2001) and thoroughly homogenized overnight. Aliquots of 1 g were extracted, purified and analyzed in the same way as described for air samples.

Analysis was performed using GC/MS (Trace GC, DSQ MS, Thermo-Fisher Scientific) with a 15 m Rtx-5SilMS capillary column (ID = 0.25 mm, film thickness = 0.1 µm). 2 µl of each sample were injected into a PTV-injector in the solvent-split mode with pressure pulse (6 bar).

The injector parameter were: solvent-split flow 20 ml/min, injection time 0.3 min, temperature program: 80 °C (0.3 min)–14.5 °C/s–300 °C (0.8 min), splitless time 0.8 min.

The GC temperature program was as follows: start with 100 °C, held for 1.35 min, increased up to 180 °C (temperature rate of 30 °C/min), increased up to 260 °C (20 °C/min), increased up to 300 °C (10 °C/min), and held for 8 min. Helium 5.0 served as carrier gas with a flow rate of 1.2 ml/min. The transfer line temperature was 300 °C.

The MS was run with electron impact ionization with a temperature of the ion source of 250 °C. PBDE were detected in the SIM mode. For each degree of bromination of Tri- to HeptaBDE the two most intensive masses of the molecule ion cluster were detected for native and $^{13}C_{12}$-labeled congeners. For BDE 209 and $^{13}C_{12}$-BDE 209 the two most intensive masses of the $[M-2Br]^+$-fragment were detected. The 16 congeners BDE 17, -28, -47, -66, -71, -77, -85, -99, -100, -119, -138, -153, -154, -183, -190 and -209 were quantified.

Quantification was performed using the internal $^{13}C_{12}$-labeled standards and the average relative response factors which were checked at the beginning of each sequence and after each sixth sample by analysis of a standard solution (Br_3DE–Br_7DE: native 0.25 ng/µl, $^{13}C_{12}$ 0.5 ng/µl; BDE 209: native 1.25 ng/µl, $^{13}C_{12}$ 5.0 ng/µl). A maximum deviation of 20% was accepted. Congener-specific average relative response factors were determined with a 3-point-calibration with solutions containing constant levels of the eight $^{13}C_{12}$-labeled PBDE congeners used for quantification and of the recovery standard $^{13}C_{12}$-BDE 138. The concentrations of the 16 native congeners and the sampling standard $^{13}C_{12}$-BDE 139 were in a range of 0.05–0.5 ng/µl (0.25–2.5 ng/µl for BDE 209).

A blank sample was processed and analyzed in parallel with each sample series. In spite of a number of rigorous measures to minimize laboratory blank samples some of the results of the air samples had to be corrected with the corresponding blank values. The amount in the blank sample was subtracted from the value in the sample if the blank value was not higher than 50%. If the blank value was >50% of the corresponding value in the sample, the result was <limit of quantification (LOQ) and the blank value was set = LOQ. In all other cases without quantifiable blank values, the LOQ was a signal to noise ratio of 10 and the limit of detection (LOD) was a signal to noise ratio of 3.

For air samples the LOD were 0.04–0.18 pg/m³ for Br_3- to Br_7DE-congeners and 0.12–13.4 pg/m³ for BDE 209. For dust samples LOD for Br_3- to Br_7DE-congeners were 0.007–0.13 µg/kg.

To determine the precision of the whole analytical method, two independent analyses were performed for one dust sample. The deviation was 1–16% (mean 7%) for the single PBDE congeners.

2.6. Method to predict body burden from intake data

To convert the congener-specific intake doses to body burdens we used the simplified one-compartment toxicokinetic model described by Lorber (2008). Assuming that the PBDE contaminants obey first-order kinetics and accumulate in body lipids, the equation for congener-specific/route-specific changes in blood lipid concentration c_{BDE} (ng/g l.w.) over time is

$$\frac{\Delta C_{BDE}}{\Delta t} = \frac{E_{BDE}(t)*ABS_{BDE}}{BFM(t)} - k_{BDE}*C_{BDE}(t)$$

where E_{BDE} is the congener-specific/route-specific (CS/RS) daily dose in ng/day, ABS_{BDE} is the CS/RS absorption fraction, BFM the body fat mass in g, and k_{BDE} is the congener-specific first-order dissipation rate in the body, measured in day^{-1}. The dissipation rate k may be expressed as $k = 0.693/t_{1/2}$, where $t_{1/2}$ is the congener-specific half-life. Assuming that the absorption and k remains constant over time, BL is constant over time and that the body is challenged with a constant dose over time, the steady-state lipid concentration is calculated as

$$C_{BDE} = \frac{E_{BDE}*ABS_{BDE}}{BFM*\frac{0.693}{t_{1/2}}}$$

To predict the body burden concentrations from the above equation, we had to make certain assumptions. The body fat mass (BFM) percentage depends on age and gender (Jackson et al., 2002). For adolescents we used the data from a cross-sectional observational German study of 2554

healthy participants that reported BFM reference values at six-monthly intervals for both sexes (Schaefer et al., 1998). Because no German reference values have been published to date for adults, we used the BFM from a multicenter, retrospective study of 1866 healthy persons performed in Italy (Coin et al., 2008).

No human data are currently available for the fraction of PBDE absorbed after oral exposure. We used the data from rats reported by McDonald (2005) with an absorption fraction of 0.94 for BDE 47, 0.78 for BDE 99, 0.93 for BDE 100 and 0.9 for BDE 153. Because data for BDE 183 and BDE 209 are not available, the absorption fraction was set at 0.9 for both of these congeners. Consistent with Lorber (2008), we used the same absorption fractions for the inhalation pathway. The bioavailability from dust intake was calculated using the data published by Huwe et al. (2008). In their study, male rats were orally exposed to PBDEs in dust at two doses within 21 days. For both dosage groups, the average retention percentages were 69% (BDE 47), 44% (BDE 99), 78% (BDE 100), 73% (BDE 153), 48% (BDE 183) and 4% (BDE 209).

The half-lives of hepta- to decabrominated PBDEs in humans have been published by Thuresson et al. (2006). The underlying calculations were based on exposure assessments of rubber workers and electronic dismantlers who donated blood during a period when they reported no work-related exposure to PBDEs. A control group without any known occupational exposure was also assessed. BDE 183 and BDE 209 were shown to exhibit half-lives of 94 days and 15 days, respectively. In the case of the lower brominated PBDE species, Geyer et al. (2004) calculated half-lives in adult humans by extrapolating from the measured half-lives in rats. We used their half-lives of 1086 days for BDE 47, 1955 days for BDE 99, 1050 days for BDE 100, 4235 days for BDE 153 and 2100 days for BDE 154 in all of our calculations.

2.7. Statistical analyses

We analyzed our data using SPSS 13.0 and SAS 9.1 (SAS Institute Inc., Cary, NC, USA). If not stated otherwise, values below the LOD were assigned half of the LOD. The median of the daily dietary PBDE intake over seven consecutive days was calculated for each participant and used for subsequent analysis. All correlations were evaluated using the Spearman rank correlation coefficient. Age was categorized into three categories (14–29, 30–45, 46–60 years of age). Sex- and age-related differences were assessed using the Wilcoxon rank sum and Kruskal–Wallis test, respectively. Random effects models were used to estimate the intraclass correlation coefficient (ICC) between the food samples of seven consecutive days. The ICC was defined as ratio of between-subject variance to total variance and was adjusted for sex and age.

3. Results

3.1. Dietary intake from duplicate diet samples

Female and male participants consumed a daily average of 2692 g of food (solid and liquid) (range: 1837–4488 g) and 3405 g of food (range: 1945–5663 g), respectively. The observed differences in food consumption between the female and male study subjects were statistically significant ($p = 0.006$).

Overall, we were able to detect BDE 47, BDE 99, BDE 100, BDE 153, BDE 154 and BDE 183 in 54%, 99%, 47%, 13%, 9% and 76% of the 350 duplicate samples, respectively. The concentrations of the main quantified congeners ranged from 34 to 5062 pg/g lipid for BDE 99, 27–6873 pg/g lipid for BDE 183, 47–5193 pg/g lipid for BDE 47 and 17–2214 pg/g lipid for BDE 100. The other PBDE congeners were found to exceed the lower limit of detection in only a few samples and therefore not used for further calculations.

The variability of the individual daily dietary PBDE intake over seven sampling days is shown in Fig. 1. The intraclass correlation coefficient (ICC) ranged between 0.33 and 0.46. Thus, within-subject variability may be of greater magnitude than between-subject variability.

For each study subject, we calculated the median dietary PBDE intake for a seven-day period. Descriptive dietary intake statistics using these 50 medians of each study subject are listed in Table 1. The highest median (95th percentile) intake rates were observed for BDE 99, BDE 183 and BDE 47, with levels of 0.20 (1.03) ng/kg b.w., 0.42 (1.35) ng/kg b.w. and 0.15 (0.70) ng/kg b.w., respectively.

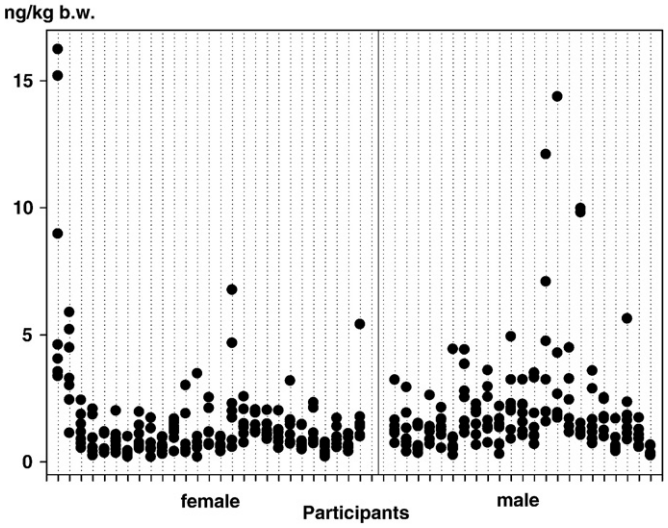

Fig. 1. Variation of single daily dietary PBDE intake over seven consecutive days in 50 participants (Sum of BDE 47, 99, 100, 153, 154 and 183). Each dot represents 1 day of intake, hence there are 7 dots per person.

Overall, we observed a higher dietary intake in males than females for the congeners BDE 100 ($p = 0.023$) and BDE 183 ($p = 0.003$) and for the sum of the six quantified tetra- to heptabrominated congeners. We failed to identify any significant age-related differences in daily PBDE intake.

3.2. PBDE levels in human blood

PBDE concentrations were analyzed in the blood samples of 47 study participants (25 females and 22 males, Table 2). Each subject provided only one blood sample. BDE 153, BDE 47, BDE 99 and BDE 100 were quantified in 94%, 87%, 77% and 49% of the samples, respectively. Across the whole group, the median (95th percentile) blood concentrations of the aforementioned congeners were 2.4 (6.6) ng/g l.w., 1.8 (5.3) ng/g l.w., 0.8 (1.9) ng/g l.w. and 0.6 (2.4) ng/g l.w. Congener BDE 183 was found to exceed the LOD only in two samples, at levels of 2.31 ng/g l.w. and 0.54 ng/g l.w. The results of the other PBDE congeners were below the limit of detection.

There were no significant sex differences. We identified higher concentrations in those aged 14–29 years, reaching significance for all target congeners except BDE 153. We failed to identify any significant correlations in terms of the individual blood concentrations and the corresponding median daily dietary intake, as calculated from duplicates.

3.3. PBDE concentrations in indoor air and dust samples

The concentrations of the nine frequently detected PBDE congeners in indoor air (sum of gaseous and particulate phase contaminants) are listed in Table 3. BDE 47, BDE 209 and BDE 28 exhibited the highest median contributions to total air concentrations with 25%, 25% and 11% of the total amounts of the nine frequently quantifiable congeners, respectively. The sum of all quantified congeners in our air samples spanned a wide range, from 8.24 to 477 pg/m³. The highest maximum values occurred for BDE 209 (438 pg/m³) and BDE 47 (169 pg/m³). Certain of the other analyzed congeners, including BDE 71, BDE 77, BDE 85, BDE 119, BDE 138 or BDE 190, were found to exceed the LOD only in a few samples.

With regard to the dust concentrations, BDE 209 was the predominant congener and it accounted for 90% (median) of the totals (Table 3). The levels of this congener ranged from 30 up to 1460 ng/g, probably reflecting the intensive use of technical decabrominated diphenyl ether in consumer products. BDE 99 and BDE 47 followed with proportions of 4% and 3%, respectively, of the total PBDE concentrations in dust.

Using log transformed values, we identified a significant correlation between air and dust readings for BDE 28, BDE 47, BDE 99 and BDE 100 (r: 0.597, $p = 0.001$; r: 0.478, $p = 0.006$; r: 0.511, $p = 0.003$; r: 0.353, $p = 0.048$) and also for the sum of the tri- to hexabrominated congeners (up to BDE 154) with r: 0.388 and $p = 0.028$. We failed to identify a significant association between PBDE air and dust concentrations and blood levels.

3.4. Predicted body burden consistent with intake dose as compared to measured blood concentrations

Overall, the body burden was predicted to equal 1.05 ng/g l.w. for BDE 47, 2.10 ng/g l.w. for BDE 99, 0.37 ng/g l.w. for BDE 100 and 1.36 ng/g l.w. for BDE 153 (see Table 2). The percentages of the total dose attributed to BDE 47, BDE 99, BDE 100 and BDE 153 were about 22%, 24%, 8% and 29%, respectively, for those four congeners. Diet-related exposure was the dominating intake dose for the four congeners, accounting for 95% of the predicted total body burden.

Table 1
Daily dietary intake of PBDE of 50 study subjects in ng/kg b.w.

	Female ($n=27$)				Male ($n=23$)			
	Mean	Median	95th percentile	Range	Mean	Median	95th percentile	Range
BDE 47	0.22	0.15	1.24	0.06–1.37	0.18	0.15	0.41	0.07–0.43
BDE 99	0.33	0.18	1.97	0.06–2.17	0.27	0.25	0.48	0.06–0.49
BDE 100	0.08	0.06	0.39	0.02–0.43	0.08	0.09	0.16	0.02–0.16
BDE 153	0.06	0.05	0.16	0.02–0.18	0.06	0.05	0.17	0.02–0.17
BDE 154	0.06	0.05	0.16	0.02–0.18	0.05	0.05	0.16	0.02–0.17
BDE 183	0.37	0.27	1.14	0.42–1.22	0.79	0.81	1.95	0.08–2.11
$\sum BDE_5$[a]	0.82	0.49	4.12	0.27–4.39	0.71	0.66	1.32	0.26–1.37
$\sum BDE_6$[b]	1.20	0.88	4.27	0.38–4.58	1.47	1.42	2.98	0.43–3.00

Note: for each study subject the median of seven sampling days was used for further descriptive analysis in this table.
[a] Sum of BDE 47, 99, 100, 153, and 154.
[b] Sum of BDE 47, 99, 100, 153, 154, and 183.

Taking the uncertainties of the used toxicokinetic model and the assignments of the parameters into account, we cautiously conclude from Table 2 that the predictions are close to the measured blood concentrations for BDE 47 and BDE 100. In contrast, our prediction for BDE 99 was higher than the measured concentrations (2.10 vs. 0.75 ng/g l.w.). Conversely, our PDE 153 prediction was somewhat lower than the observed blood concentrations. Because PBE 183 was quantifiable in only 2 of 47 blood samples, comparing its predicted and observed body burden is not meaningful. If all values below the LOD are set to equal half of the LOD, the measured BDE 183 concentration becomes 0.71 ng/g l.w., substantially higher than the predicted value of 0.19 ng/g l.w.

3.5. Overall intake using exposure data

Taking into account all the potential routes of human exposure to PBDE, we estimated the "average" and "high" daily intakes for a non-occupationally exposed adult German population (summarized in Table 4).

We used the results from animal experiments published by McDonald (2005) and Huwe et al. (2008) to generate a known set of congener-specific absorption efficiencies. We estimated exposure via inhalation using the average of the mean daily inhalation values for females and males (13.3 m^3/day) (US-EPA, (1997). We assumed that people generally spend 90% of their time indoors. For the remaining 10% of the time, outdoor exposure was estimated using data from published winter and spring PBDE measurements in Vienna in 2005/2006 (Uhl and Gans, 2007). These data include PBDE contaminants in both gaseous and particulate phases. Indoor air exposure and non-dietary intake through the ingestion of house dust were estimated using the median and 90th percentile. Dust intake was assessed by combining measurement results with an assumed average adult intake rate of 50 mg dust per day as derived by the US-EPA (US-EPA, (1997). Dietary intake was similarly estimated based on the median and 90th percentile values. We calculated descriptive statistics using the median values of seven consecutive sampling days for each study subject. We failed to take representative drinking water measurements, and accordingly we ignored this pathway altogether. Other researchers have suggested that exposure via this pathway may be negligible (Lorber, 2008).

Consistent with our assumptions, we estimated for the sum of tetra- to heptabrominated congeners an average (and high) comprehensive total daily intake of 1.2 ng/kg b.w. (and 2.5 ng/kg b.w.). Our results suggest that dietary exposure is the dominant intake pathway at least in our study population, responsible for 97% (average intake) and 95% (high intake) of the total intake for an adult population. Using an average or high intake scenario only for the more volatile lower brominated BDE 47, the contribution of diet to total intake was reduced to 94% and 86%, indicating that the inhalation pathway is somewhat more important for these congeners.

Table 2
Measured PBDE congener concentrations in blood of 47 subjects compared to predicted body burdens (in ng/g lipid weight).

	BDE 47	BDE 99	BDE 100	BDE 153
Measured concentrations				
Range	0.23–6.44	0.19–2.19	0.27–2.71	0.86–8.19
Mean	1.88	0.86	0.81	2.84
95th percentile	5.25	1.89	2.41	6.57
Median	1.81	0.75	0.58	2.37
Predicted concentrations				
95th percentile	5.22	5.44[a]	1.67	3.89
Median	1.05	2.10	0.37	1.36

[a] Predicted after exclusion of two outliers with a dietary intake higher than a factor 6 and 8 compared to the mean congener-specific intake.

4. Discussion

4.1. Blood concentration

Following research in the 1970s, we now have a comprehensive understanding of the PBDE body burden in humans. Several studies have shown that the contamination level in the blood of non-occupationally exposed populations was highest for individuals in the United States as compared to Europeans. Japanese citizens appear to have similar exposure rates to Europeans (Hites, 2004; Kawashiro et al., 2008). Our study suggests that the mean PBDE levels in Germany are currently in the same range as those reported from various European countries over the past decade (Guvenius et al., 2003; Thomas et al., 2006; Karlsson et al., 2007; Thomsen et al., 2007; Gómara et al., 2007; Knutsen et al., 2008; Antignac et al., 2009; Covaci and Voorspoels, 2005). We conclude from Fig. 2 that the distribution pattern of the congeners in this study with a dominance of BDE 153 and BDE 47 is in accordance with previously published data from several European countries. In contrast, very high PBDE concentrations, especially of BDE 47, were observed in the blood of US residents (Sjödin et al., 2008b). Our results of higher PBDE concentration in younger participants are in line with further results from the US NHANES study (Sjödin et al., 2008b). The higher concentrations found in the youngest age group were explained by their lifestyle and activity differences.

4.2. Indoor air and dust

Our results for indoor air PBDE are comparable with data from a passive air sampling study conducted in 2003 to 2004 in 31 UK homes. Scientists reported a median concentration of 24 pg/m^3 for all tri- to hexabrominated congeners (Harrad et al., 2006). In a Swedish study that included four homes, only BDE 28, BDE 47 and BDE 66 were found to be above the detection limits at concentrations of 8–28 pg/m^3, <117–171 pg/m^3 and <2– 6 pg/m^3, respectively, while BDE 99 and BDE 100 were not above the relatively high detection limits of 39 and 158 pg/m^3 (Karlsson et al., 2007). In contrast to the European situation, higher levels of 100 pg/m^3 (median) and 288 pg/m^3 (geometric mean) for the sum of the Tri- to HexaBDE were observed in 74 homes in Ottawa and in 24 residences in Boston, respectively (Wilford et al., 2004; Allen et al., 2007).

To date, BDE 209 has only been analyzed in air in two studies from the US and Sweden. In Boston, air was collected from 20 urban residences in 2006 where the median concentrations were nearly a factor of 10 higher than in our study (94.8 and 94.2 pg/m^3 in the bedrooms and main living areas, respectively) (Allen et al., 2007). The Swedish study measured BDE 209 in air samples from four homes, but found this congener to be above the detection limit in only one sample, at concentrations of 257 pg/m^3 (limit of detection: 175 pg/m^3) (Karlsson et al., 2007).

Table 5 suggests that Tri- to HeptaBDE concentrations in Europe and Asia are substantially lower than in North America. Although the percentage contribution of BDE 209 on total concentrations was only 20 to 63% in Canada and the US, it reached 98% in the UK and 71–90% in the Swedish and German studies. Within Europe, significantly higher

Table 3
Concentration of PBDE congeners in indoor air and dust of residences (*n*: 34).

	Air (pg/m³)				Dust (ng/g)			
	Mean[a]	Median	95th percentile	Range	Mean[a]	Median	95th percentile	Range
BDE 28	5.58	4.21	14.8	1.06–17.2	3.39	0.25	1.69	0.07–1.87
BDE 47	19.1	9.39	98.2	3.17–169	23.7	9.08	173	1.71–255
BDE 66	0.59	0.31	2.9	<0.02–4.87	0.55	0.19	4.0	0.03–5.14
BDE 99	9.66	2.65	78.7	<0.52–189	35.2	12.5	228	1.83–390
BDE 100	1.93	0.54	14.7	<0.23–33.3	6.47	2.49	45.5	0.28–81.1
BDE 153	1.24	0.27	11.7	<0.08–22.8	5.02	2.69	28.2	0.30–41.1
BDE 154	0.62	0.20	5.5	<0.06–10.9	3.56	1.62	27.5	0.17–42.1
BDE 183	1.40	0.44	10.3	<0.14–21.5	9.24	4.26	48.8	0.29–60.4
BDE 209	33.3	9.50	229	0.87–438	354	312	839	29.7–1460
$\sum BDE_7$[b]	38.4	19.9	211	5.22–439	74.9	30.0	448	5.88–814
$\sum BDE_9$[c]	73.1	37.8	466	8.24–477	438	386	1195	36.5–1580

[a] Concentrations <LOD were set = 0 for calculation of mean values, concentrations <LOQ were set = 0.5 LOQ.
[b] Sum of BDE 28, 47, 66, 99, 100, 153, and 154.
[c] Sum of BDE 28, 47, 66, 99, 100, 153, 154, 183, and 209.

Table 4
Estimated adult daily intake of PBDE for the general population.

	Concentration		Intake pg/day		Intake pg/kg b.w.[a]	
	Median	90th percentile	Average scenario	High scenario	Average scenario	High scenario
BDE 47						
Indoor air	9.39 pg/m³	48.32 pg/m³	113	580	1.9	9.7
Outdoor air	9.20 pg/m³	15.6 pg/m³	12	20	0.2	0.3
Dust	9.08 ng/g	56.25 ng/g	450	2813	7.5	46.9
Diet			9630	20,390	161	340
BDE 99						
Indoor air	2.65 pg/m³	9.17 pg/m³	32	110	0.5	1.8
Outdoor air	2.50 pg/m³	4.50 pg/m³	3	6	0.1	0.1
Dust	12.49 ng/g	98.32 ng/g	625	4916	10.4	81.9
Diet			15280	30,030	255	501
BDE 100						
Indoor air	0.54 pg/m³	2.34 pg/m³	6	28	0.1	0.5
Outdoor air	0.87 pg/m³	1.50 pg/m³	1	2	0.02	0.03
Dust	2.49 ng/g	16.25 ng/g	124	813	2.1	13.6
Diet			3880	10,110	65	169
BDE 153						
Indoor air	0.27 pg/m³	1.87 pg/m³	3	22	0.05	0.4
Outdoor air	0.30 pg/m³	9,69 pg/m³	0.4	12	0.01	0.2
Dust	2.69 ng/g	11.25 ng/g	135	563	2.3	9.4
Diet			3070	8410	51	140
BDE 154						
Indoor air	0.20 pg/m³	0.69 pg/m³	2	8	0.03	0.1
Outdoor air	0.22 pg/m³	0.30 pg/m³	0.3	0.4	0.01	0.01
Dust	1.62 ng/g	6.14 ng/g	81	307	1.4	5.1
Diet			3030	8410	51	140
BDE 183						
Indoor air	0.44 pg/m³	2.62 pg/m³	5	31	0.1	0.5
Outdoor air	0.90 pg/m³	37.8 pg/m³	1	49	0.02	0.8
Dust	4.26 ng/g	22.09 ng/g	213	1105	3.6	18.4
Diet			30,320	81,990	505	1367
BDE 209						
Indoor air	9.45 pg/m³	60.1 pg/m³	113	721	1.9	12.0
Outdoor air	12.0 pg/m³	29.8 pg/m³	16	35	0.3	0.6
Dust	312 ng/g	624 ng/g	15,600	31,200	260	520
Σ $PBDE_6$[b]						
Indoor air	12.8 pg/m³	48.8 pg/m³	154	586	2.6	9.8
Outdoor air	11.7 pg/m³[c]	26.7 pg/m³[d]	15	35	0.3	0.6
Dust	39.1 ng/g	154 ng/g	1955	7700	32.6	128.3
Diet			69,609	176,913	1194	2496

Average intake scenario based on median concentrations and high intake scenario based on 90th percentiles as not otherwise stated.
[a] For an adult with 60 kg b.w.
[b] Sum of BDE 47, 99, 100, 153, 154, and 183.
[c] Mean of all measured congeners except BDE 209 in a summer and winter period.
[d] Mean of the maximum values of single congeners.

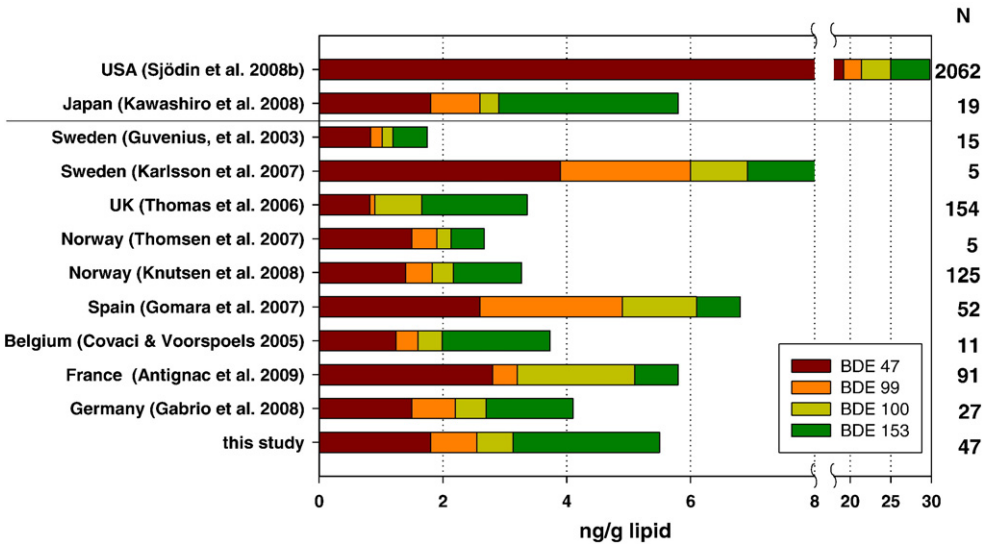

Fig. 2. Share of PBDE congeners in human blood (median values of each congener).

concentrations of BDE 209 were observed in the dust samples collected from various regions in the UK compared to values reported from two other countries. There is some evidence that the UK accounted for a disproportionately large share of the EU market for PBDE because of the UK's more stringent fire safety regulations (Harrad et al., 2008a). The international differences are consistent with the different use-patterns in Europe and North America, with a ~50-fold higher usage of technical PentaBDE in the latter (Harrad et al., 2008a). Use of BDE 209 in North America is three times that in Europe. However, the exposure profile in North America exhibited some heterogeneity. This may be partly explained by different regional regulations. For example, Zota et al. (2008) found elevated PBDE dust exposure in California, which may be a consequence of the state's more aggressive furniture flammability standards. The median dust concentrations of BDE 47, BDE 99 and BDE 100 in 49 California homes were 4–10 times higher than previously reported in North America and the maximum concentrations were the highest ever reported in indoor dust.

In Germany, we found levels of PBDE in vacuum cleaner dust to be on the same order of magnitude as those reported in 2001 to 2003 (Knoth et al., 2003) and in 10 samples from different sampling sites (Sjödin et al., 2008a). Similar concentrations were also observed in a 2006 study from the South West of Germany (Gabrio et al., 2008). The median BDE 209 concentrations in these four studies ranged from 63 ng/g to 312 ng/g. From the results of Knoth et al. (2003) and Sjödin et al. (2008a), we note contributions of BDE 209 to total PBDE of 81% and 71%, respectively. By contrast, our study suggested a contribution of 90%. Since the two other

Table 5
Dust levels (in ng/g) in this study compared to other studies (median values).

Location	*N*	28	47	99	100	153	154	183	209	References
Europe										
Germany	15–40	0.6	17.1	23.9	4.2	6.0	2.7	6.1	265	Knoth et al. (2003)
3 cities; Sweden	5	2.2	51	79	24	4.9	3.9	4.8	470	Karlsson et al. (2007)
Scotland; UK	9	0.29	16.3	35.8	5.7	6.5	4.0	8.9	3796	Pless-Mulloli et al. (2006)
Birmingham; UK	28/16 [a]	0.35	13	23	4.2	5.2	3.3	13	2800	Harrad et al. (2008a)
Birmingham; UK	30	<dl	10	20	3.4	5.0	2.8	4.2	8100	Harrad et al. (2008b)
Newcastle, UK	10	–	22	28	4	5	3	5	10000	Sjödin et al. (2008a)
5 cities, Germany	10	–	<14	10	<6	<6	–	<6	63	Sjödin et al. (2008a)
Munich; Germany	34	0.25	9.1	12.5	2.5	2.7	1.6	4.3	312	This study
North America										
Ottawa; Canada	64	15	1100	1800	490	470	380	44	1100	Wilford et al. (2005)
Toronto; Canada	28/16 [a]	4.1	140	330	65	43	39	9.0	560	Harrad et al. (2008a)
Atlanta; US	10	–	430	880	150	140	80	70	2000	Sjödin et al. (2008a)
Boston; US	11	–	670	1010	174	107	93	–	~<500	Wu et al. (2007)
Washington DC; US	16	14.8[b]	644	676	119	64.4	72.8	17.6	1350	Stapleton et al. (2005)
Amarillo and Austin; US	20/17[c]	14	410	820	160	110	89	16	1300	Harrad et al. (2008a)
Boston; US	20	6.4[b]	338	536	76.9	47.0	35.0	15.1	1811	Allen et al. (2008)
California; US	49	–	2700	3800	684	–	–	–	–	Zota et al. (2008)
Albany, US	12	<0.1	40.1	95	16.1	25.9	9.6	<21	903	Johnson-Restrepo and Kannan (2009)
Asia/Australia										
Wellington; New Zealand	20	0.65	24	51	5.8	5.4	5.1	–	–	Harrad et al. (2008a)
Singapore	31	0.6	20	24	4.2	6.9	3.5	8.5	1000	Tan et al. (2007)
Kuwait	17	0.12	2.7	0.7	0.7	0.7	0.9	1.3	82.9	Gevao et al. (2006)
Australia	10	–	60	100	18	13	9	14	730	Sjödin et al. (2008a)
Brisbane, Australia	10	–	91	184	38	23	–	102	377	Toms et al. (2009)

[a] 28 samples analysed for Tri-HexaBDEs, 16 samples analysed for Tri-DecaBDEs.
[b] Sum of congener 28 and 33.
[c] 20 samples analysed for Tri-HexaBDEs, 17 samples analysed for Tri-DecaBDEs.

Table 6
Calculated daily PBDE dietary intake from various studies in ng/kg b.w. (values < LOD = 0.5 LOD, as not otherwise stated).

Dietary intake	Study location	Sampling years	Sum of congeners	References
Market basket surveys				
0.73 [a]	Finland	1997/99	BDE 47, 99, 100, 153, 154	Kiviranta et al. (2004)
0.73 [a]	Canada	1998	BDE 28, 47, 99, 100, 153, 154	Ryan and Patry (2001)
0.69 [a]	Sweden	1999	BDE 47, 99, 100, 153, 154	Darnerud et al. (2006)
1.40	Spain	2000	BDE 47, 99, 100, 153, 154, 183	Bocio et al. (2003)
3.55 [a]	The Netherlands	2001/02	BDE 28, 47, 99, 100, 153, 154	de Winter-Sorkina et al., 2003
1.06	Norway	2002–2006	BDE 47, 99, 100, 153, 154	Knutsen et al. (2008)
0.79	The Netherlands	2003/04	BDE 47, 99, 100, 153, 154	Bakker et al. (2008)
1.2[b,c]	UK	2003/04	BDE 47, 99, 100, 153, 154	FSA (2006)
1.3 (m)[d] 0.9 (f)[d]	USA (Texas)	2003/04	BDE 17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 209	Schecter et al. (2006)
2.1 (m)[b] 1.8 (f)[c]	Australia	2004	22 congeners	FSANZ (2006)
0.58[a]	Belgium	2005	BDE 28, 47, 99, 100, 153, 154, 183	Voorspoels et al. (2007)
1.1	Spain	2006	BDE 28, 47, 99, 100, 153, 154, 183	Domingo et al. (2008)
Duplicate diet studies				
1.51[a,e] 1.95[a,b]	UK	1999/00	BDE 47, 99, 100, 153, 154	Harrad et al. (2004)
1.1[e]	Japan	2002–2005	Mono- to DecaBDE	Nomura et al. (2007)
0.17[a]	Belgium	2007	BDE 28, 47, 99, 100, 153, 154, 183	Roosens et al. (2009)
0.66 (m) 0.49 (f)	Germany	2005	BDE 47, 99, 100, 153, 154	This study
1.42 (m) 0.88 (f)			BDE 47, 99, 100, 153, 154, 183	

(m): male; (f): female.
[a] For an adult of 60 kg b.w.
[b] Average consumption behaviour.
[c] <LOD = 0.
[d] Adults 20–39 years.
[e] <LOD = LOD.

studies were conducted before penta- and octabrominated diphenyl ethers were banned in the EU, we hypothesize that more BDE 209 and/or other polybrominated substances may have recently been used in Germany in place of the less brominated (i.e., restricted) PBDE.

Consistent with other studies, we observed a correlation between indoor air values and dust concentrations for certain congeners. In Ottawa, indoor air was collected from 74 randomly selected homes and dust was sampled from the family vacuum cleaners in 68 of the same homes (Wilford et al., 2004, 2005). Correlations were found between pentamix congener levels in dust and in air from the same homes, but not for congeners of the more highly brominated mixtures. Allen et al. (2007) measured PBDE concentrations in the main living rooms and bedrooms of 20 Boston homes and identified significant correlations for PentaBDE, but not for DecaBDE, in the bedrooms. No significant associations were identified for either Penta- or DecaBDE in the main living area. They concluded that researcher-collected dust cannot generally be used as a predictor of airborne concentrations and they argued that additional factors have to be considered. Moreover, the same working group described other critical factors with regard to house dust levels. They identified significant differences within the homes (elevated living room concentrations), and between vacuum bag dust and researcher-collected dust (lower in the vacuum bags). They failed to identify significant time-related differences over an 8-month sampling period. In addition, Harrad et al. (2008b) measured the within-room spatial variability and the temporal variability every month over a 9–10 month period in UK residences and offices. They reported substantial within-room variability and temporal variability. The reasons for these differences are not yet clear.

The various influencing factors with regard to sampling method and location, as well as the substantial differences in terms of house characteristics and source variability, may be an explanation of the lack of correlation between air and dust concentration with body burden in our study. By contrast, Karlsson et al. (2007) did find a positive relationship between dust and plasma levels for net total PBDE (BDE 28 to BDE 154) in a small sample of five, although this result was strongly dependent on one of the five observations. However, no association was observed when DecaBDE was included in their calculations. Three other studies in the US and Australia have simultaneously measured PBDE in dust and breast milk. Although Wu et al. (2007) found a significant association for 12 mothers living in the greater Boston area, Sharp and Lunder (2004) observed no such relation among 10 participants in different US geographies. Toms et al. (2009) found only a significant correlation between the air and milk concentrations for BDE 99 but none between dust and milk in a study of 10 subjects in Brisbane, Australia.

4.3. Dietary intake

Table 6 lists the dietary intake based on food shopping basket surveys or duplicate diet studies performed in Europe and North America. Except in two cases, all data were calculated using the "median-bound" scenario by assuming that values below the limit of detection were equal to one-half of the LOD. Compared to our study, similar median PBDE intakes were observed in studies from Finland, Sweden, The Netherlands, Belgium and Spain, all using a market basket approach. This comparison clearly shows that the dietary intake in Europe is in the same range as that for Canada (Ryan and Patry, 2001). An expanded US survey (Schecter et al., 2006; Lorber, 2008) reported that the total contamination level in European food samples was comparable to that in the US. In most European countries fish products are the main contributors to total PBDE intake, but in the US meat and meat products tend to account for most of the exposure to these chemicals (Schecter et al., 2008).

In Japan, Ashizuka et al. (2007) determined the PBDE levels of 13 food groups from 6 regions in 2004 to 2005 with a mean dietary intake of 111 ng/day (1.56 ng/kg b.w., assuming a body weight of 70 kg). In addition, the daily PBDE intake solely from fish purchased in 3 Japanese regions was estimated to total 0.58 ng/kg b.w. across all PBDEs (Ashizuka et al., 2008).

To date, three other duplicate diet studies have been performed — in the UK in 1999/2000 (Harrad et al., 2004), in Japan in 2002 to 2005 (Nomura et al., 2007), and in Belgium in 2007 (Roosens et al., 2009). In the UK study, 10 omnivorous diet samples were investigated, each representing the dietary intake of seven consecutive days. In contrast to our study, Harrad et al. (2004) identified a slightly higher intake of PBDE ranging from 1.0 to 3.9 ng/kg b.w. In Japan, 12 samples were collected every year from 2002 to 2004, and 9 samples were collected in 2005. Each sample represented all meals, snacks and drinks as prepared for consumption over a period of three consecutive days. In the latter investigation, the daily PBDE intake ranged from 0.18 ng/kg b.w. to 8.0 ng/kg b.w. (median: 1.1 ng/kg b.w.). DecaBDE was responsible for 50% of exposure in most food samples. The median daily intakes in 2002, 2003, 2004, and 2005 were recorded as 0.78, 2.0, 1.0 and 0.9 ng/kg b.w., respectively. In Belgium, a relatively low dietary intake of 10.3 ng per day (median) of Tri- to HeptaBDE were observed in a study of 19 students.

4.4. Correlation of dietary exposure and plasma levels

We did not observe a significant correlation between the dietary intake and plasma levels despite the fact that food was the major exposure route. Up to now, the relationship of dietary PBDE exposure and body burden is not fully understood. In one study serum levels were strongly correlated with dietary exposure for men eating fish from a

contaminated lake (Thomsen et al., 2008). However, this relationship was less pronounced in another Norwegian study on persons eating only food with background levels of contamination, showing a weak correlation for men (only for BDE 47 and BDE 154) but none for females (Knutsen et al., 2008). For Latvia and Sweden, Sjödin et al. (2000) found that people reporting a high intake of fatty Baltic fish consumption had significant higher plasma levels than the group with no or low fish consumption. In contrary, no significant relationship between breast milk concentrations of PBDE and dietary intake recorded by questionnaires was observed in Sweden and Italy (Lind et al., 2003; Ingelido et al., 2007).

In a New Jersey study, the geometric mean was consistently higher for anglers who reported eating their fish catch, but the differences to persons who eat no local fish did not reach statistical significance (Morland et al., 2005). Moreover, Anderson et al. (2008) reported a positive association with shellfish and catfish consumption and a weak association with years of sport fish consumption.

In line with our results, a recent study from Belgium could not found a correlation between serum levels and dietary intake resulting from a duplicate diet study (Roosens et al., 2009). One explanation of the absence of a relationship between dietary exposure and body burden in both studies may be due to the low levels of contamination in food in these countries. A positive association was observed especially in such studies when consumption of higher contaminated fish was observed or in subsets of the population like anglers, who had also a high amount of fish intake in relation to total food intake. Further on, the duplicate diet approach can cover only a small period of time, and dietary habits as well as the contamination level could be different in the past.

4.5. Predicted body burden from intake data

Lorber (2008), who predicted the body burden of eight congeners using exposure data from the US, concluded that the predictions were reasonably close to the measurements for six of the eight congeners. Consistent with the results of this study, we observed that the predicted values were higher than measured in the case of BDE 99. In the case of three other congeners, we identified slightly lower predicted values. For BDE 47, a factor of 4 difference has been reported when comparing predicted to measured values in the US (Lorber, 2008), while in our study the difference was only a factor of 1.7. For BDE 153, the differences were factors of 2.7 (US) and 1.7 (this study).

We failed to measure BDE 209 in our food samples. Accordingly, we can only predict body burden for this contaminant from air and dust exposure. Using these data, the total predicted body burden estimated from these pathways was 0.0009 ng/g l.w. To date, BDE 209 has only been measured in certain European food surveys. In a food shopping basket survey that was representative of the Belgian population, BDE 209 did not to exceed LOQ in any food samples (Voorspoels et al., 2007). Gómara et al. (2006) reported that BDE 209 was dominant in oil and eggs, but they failed to calculate the dietary intake of this congener. The UK Food Standards Agency estimated a daily BDE 209 intake of 4.5 ng/kg b.w. across all age groups based on average consumption across the whole diet (FSA, (2006). Moreover, Knutsen et al. (2008) investigated the dietary exposure of 184 participants, not representative of the general population, and reported the range of PBDE intake among Norwegians with average and high fish consumption statistics. Since we failed to conduct a food survey for our German sample, we used dietary BDE 209 intakes of 4.5 ng/kg b.w. (UK) and 1.38 ng/kg b.w. (Norway) for our calculations. This led to body burdens of 0.36 ng/g l.w. and 0.11 ng/g l.w., respectively.

4.6. Overall estimates using intake data

Our study suggests that dietary intake is the dominant exposure pathway in our adult German study population, accounting for up to 97% of the total intake (sum of Tetra- to HeptaBDE contaminant levels) using an average intake scenario. Our results are consistent with findings from Harrad et al. (2004), who calculated that the median dietary exposure accounts for 93% of the net daily exposure in the U.K.

Our estimated average total daily intake from diet, dust ingestion and inhalation of 1.2 ng/kg b.w. for adults in Germany was lower than figures reported for the U.K. (Harrad et al., 2006, Harrad et al., 2004), Canada (Jones-Otazo et al., 2005), and the US (Lorber, 2008, Johnson-Restrepo and Kannan, 2009). This may be due to the lower contamination levels of indoor air and dust in Germany, especially when compared with data from the US and Canada (Harrad et al., 2008a).

4.7. Health effects

In 2005, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) concluded that the available toxicity data is inadequate to derive a tolerable daily intake (TDI) for PBDE. They also concluded that PBDE congeners do not exhibit a common mechanism of action (JECFA, 2006). The limited toxicity data suggests that in the case of the more toxic PBDE congeners (e.g., BDE 47, PBDE 99), adverse effects are unlikely to occur at doses of less than approximately 0.1 μg/kg b.w. The No Observable Effect Level (NOEL), determined from animal studies to examine developmental neurotoxicity or thyroid hormone changes, ranged from 0.14 to 1.0 mg/kg b.w. (McDonald, 2005). Various research groups have reported that neuro-developmental disturbances following fetal and/or postnatal exposure are probably most sensitive to PBDE-induced toxicity (summarized in McDonald, 2005; Costa and Giordano, 2007). Kuriyama et al. (2005) orally exposed rat dames on gestational day 6 to single doses of 0.06 and 0.3 mg/kg b.w. BDE 99 and identified neurobehavioral changes in offspring that had been exposed to the higher dose. However, behavioral changes were also present at puberty in the lowest dose group tested. Since this data represents the Lowest Observable Effect Level (LOEL) reported for a congener to date, we used this to estimate the potential health risks related to PBDE exposure in Germany.

To guarantee a conservative approach, we assumed that all other measured congeners are as toxic as BDE 99. We added together relevant estimates for all the congeners for which data from our study were available and we compared total exposure doses with the aforementioned LOEL data. Our average and high total daily PBDE intakes (including BDE 47 through BDE 183) were 1.2 ng/kg b.w. and 2.5 ng/kg b.w., respectively. In comparison to the LOEL, the margins of exposure (MOS) were calculated as 50,000 and 23,000, respectively.

However, McDonald (2005) has described an approach that takes into account the significant interspecies differences in terms of toxicokinetics. For example, the half-life of BDE 99 in humans is assumed by Geyer et al. (2004) to be 55-fold higher than in rodents. Therefore, McDonald (2005) used this factor, together with an uncertainty factor of 10 for human inter-individual variability and a factor of 3 for interspecies toxicodynamic differences. The resulting “tolerable” intakes using the aforementioned approach are about 30-fold and 14-fold higher than the average and high total daily intakes derived from our data.

5. Conclusion

Our study of PBDE exposure in adults via different environmental media is one of the most comprehensive data set in Europe. The results thus contribute to deeper understanding of PBDE exposure in a general adult population. Our calculated dietary intake data suggests that although the exposure of adults in Germany remains relatively low and diet is the most significant pathway. Using an average and high intake scenario, food intakes are responsible for approximately 97% and 95% of the total daily intake, respectively.

In conclusion, the current dietary intake of an adult German population is well below the levels that are associated with PBDE toxicity. We suggest that this exposure is unlikely to pose a health risk. We note

that exposure doses are considerably higher in other parts of the world (e.g., North America) and the higher intakes of certain other subsets of the population, like toddlers (due to dust intake) and breast-fed infants, may prove problematic.

To date, various studies have analyzed the exposure in residences and in ambient air, but only limited data has been available for other micro-environments. The total exposure may therefore be somewhat underestimated. In offices and in cars, air concentrations may be elevated by 3 times and 1.7 times, respectively (Harrad et al., 2006). The same research team also measured vacuum cleaner dust harvested from one square meter of carpet in residences, offices and upholstered car seats (Harrad et al., 2008b). The average concentration of Tri- to HexaBDE was 3.2 higher in offices and 30-fold higher in cars compared to residences. BDE 209 was the dominant congener in all micro-environments, and especially in cars and residences. These results are consistent with Lagalante et al. (2009), who collected dust from 60 automobiles in the US. Similarly, Mandalakis et al. (2008) reported high levels of PBDE in the interior of vehicles. Older cars exhibited lower concentrations, but higher indoor temperatures led to elevated contaminant counts. Furthermore, PBDE exposure is common in aircraft cabins, with higher serum concentrations exhibited by subjects sampled after intercontinental flights (Christiansson et al., 2008).

As part of our future research efforts, we plan to gather more data from different microenvironments to better understand the current exposure of the general population.

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