Retrospective Time-Trend Study of Polybrominated Diphenyl Ether and Polybrominated and Polychlorinated Biphenyl Levels in Human Serum from the United States

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Six polybrominated diphenyl ethers (PBDEs), one hexabromobiphenyl [polybrominated biphenyl (PBB)], and one hexachlorobiphenyl [polychlorinated biphenyl (PCB)] were measured in 40 human serum pools collected in the southeastern United States during 1985 through 2002 and in Seattle, Washington, for 1999 through 2002. The concentrations of most of the PBDEs, which are commercially used as flame retardants in common household and commercial applications, had significant positive correlations with time of sample collection, showing that the concentrations of these compounds are increasing in serum collected in the United States. In contrast, PCB and PBB levels were negatively correlated with sample collection year, indicating that the levels of these compounds have been decreasing since their phaseout in the 1970s. *Key words:* **PBB, PBDE, PCB, polybrominated biphenyl, polybrominated diphenyl ethers, polychlorinated biphenyl, retrospective time trend, serum.** *Environ Health Perspect* **112:654–658 (2004). doi:10.1289/ehp.6826 available via** *http://dx.doi.org/* **[Online 14 January 2004]**

Flame retardants (FRs) are incorporated into potentially flammable materials such as plastics, rubber, and textiles to slow down or inhibit the initial phase of a developing fire. Thus, FRs are important in modern society, limiting the consequences of fires and saving lives. However, some FRs persist in the environment (Hale et al. 2003; Law et al. 2003; Norstrom et al. 2002) and bioaccumulate or biomagnify. One class of these chemicals is the polybrominated diphenyl ethers (PBDEs), manufactured in different degrees of bromination, for example, pentaBDE, octaBDE, and decaBDE. The lower brominated pentaBDE product is primarily used in polyurethane foam for upholstery applications, whereas the mixtures with higher degrees of bromination are used for applications such as housings for electrical appliances.

PBDEs are a group of environmental contaminants with a global distribution and are present in wildlife species in both aquatic (Hale et al. 2003; Kierkegaard et al. 1999) and terrestrial (Norstrom et al. 2002; Sellström 1999) environments. However, concentrations of PBDEs in humans and wildlife in North America are much higher than concentrations reported in Europe (Asplund et al. 1999; Sjödin et al. 2003a).

Retrospective time-trend studies conducted using environmental samples originating from the Swedish environment showed a peak in environmental levels in the mid-1980s, after which environmental concentrations decreased or remained unchanged (Kierkegaard et al. 1999; Sellström 1999). This trend has been shown for herring gull eggs (Sellström 1999) originating from Stora Karlsö, a small island in the Baltic Sea, and in pike (Kierkegaard et al. 1999) collected in Lake Bolmen in Sweden. These data indicate that a potential lag exists between the time a positive effect is observed in the environment and the time a similar effect is noticeable in a human population. Concentrations of the lower brominated PBDEs in the Great Lakes region in North America reportedly doubled every 2.6–3.1 years from 1981 through 2000 (Norstrom et al. 2002). Considering the current high use of the lower brominated PBDE product (pentaBDE) in the United States, this observation is not surprising.

The aim of the present inventory was to determine the levels of PBDEs (n = 6), 2,2′,4,4′,5,5′-hexabromobiphenyl (BB-153), and 2,2′,4,4′,5,5′-hexachlorobiphenyl (CB-153) in archived serum samples collected over 1985–2002 in order to assess the concentration change over time in serum for the target analytes. The occurrence of brominated flame retardants (BFRs) was to be compared with that of the stable and persistent polychlorinated biphenyl (PCB) congener CB-153.

Methods and Materials

Serum samples. Archived serum pools used as quality assurance/quality control (QA/QC) pools in the measurement of organohalogen compounds and/or serum lipids were used in the present study. Serum pools (n = 36) from 1985–1997 and 2002 were collected in the southeastern United States by the Interstate Blood Bank (Memphis, TN). Serum samples from 1999–2002 were collected in Seattle, Washington, by Solomon Park Research Laboratories (Kirkland, WA). Serum pools (n = 16) were prepared according to the principles and methods that formed the basis for the National Committee for Clinical Laboratory Standards guidelines (NCCLS 1999). Additionally, for this study the serum lipid concentration of the serum pools was determined using commercially available test kits from Roche Diagnostics Corporation (Indianapolis, IN) for the quantitative determination of total triglycerides (product no. 011002803-0600) and total cholesterol (product no. 011573303-0600). Final determinations were made on a Hitachi 912 Chemistry Analyzer (Hitachi, Tokyo, Japan).

The number of donors in the serum pools from the southeastern United States ranged from 40 to 200, and serum from six to eight blood donors was used to make the pools originating from Seattle. Because of the lower number of blood donors in the pools originating from Seattle, the average for each year is included in Table 1 and used for all statistical handling of the data.

All serum samples were collected by convenient sampling and hence may not be considered representative of the general U.S. population. The project was exempt for human subjects review because the serum specimens were pooled and no personal identifying information was collected.

A mixture of calf serum (0.5 mL; Gibco BRL, Grand Island, NY) and water (3.5 mL) was used as method blanks.

Certified reference standards. We used two internal standard spiking solutions from Cambridge Isotope Laboratories (Andover, MA) for the BFRs and PCBs. The spiking standard for BFRs contained the following $^{13}C_{12}$-labeled congeners at a concentration

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of 7.5 pg/μL in methanol: 2,2,4′-tribromodiphenyl ether (BDE-28); 2,2′,4,4′-tetraBDE (BDE-47); 2,2′,4,4′,5-pentaBDE (BDE-99); 2,2′,4,4′,6-pentaBDE (BDE-100); 2,2′,4,4′,5,5′-hexaBDE (BDE-153); 2,2′,4,4′,5,6′-hexaBDE (BDE-154); 2,2′,3,4,4′,5′,6-heptaBDE (BDE-183); decaBDE (BDE-209); and BB-153. The spiking standard for PCBs contained $^{13}C_{12}$-CB-153 at a concentration of 7.5 pg/μL in methanol. The recovery spiking standard was also obtained from Cambridge Isotope Laboratories and contained the labeled compounds 1,2,3,4-tetrachlorodibenzo-*p*-dioxin ($^{13}C_6$-1,2,3,4-TCDD, 2.5 pg/μL); 3,3′,4,4′-tetraBDE ($^{13}C_{12}$-BDE-77, 7.5 pg/μL); 2,2′,3,4,4′,6-hexaBDE ($^{13}C_{12}$-BDE-139, 7.5 pg/μL); and 2,2′,3,3′,4,5,5′,6,6′-nonachlorinated biphenyl ($^{13}C_{12}$-CB-208, 10 pg/μL). The solvent for the recovery standard was *n*-hexane containing 10% and 2% by volume of nonane and dodecane, respectively.

We used a 10-point calibration curve spanning the concentration range 0.2–2,000 pg/μL containing the $^{13}C_{12}$-labeled BFRs and $^{13}C_{12}$-CB-153 at a concentration of 75 pg/μL for the gas chromatography/isotope dilution high-resolution mass spectrometry (GC-IDHRMS) analyses. The calibration curve contained the following native analytes: 2,2′,4-triBDE (BDE-17); BDE-28; BDE-47; 2,3′,4,4′-tetraBDE (BDE-66); 2,2′,3,4,4′-pentaBDE (BDE-85); BDE-99; BDE-100; BDE-153; BDE-154; BDE-183: 2,2′,3,4,4′,5,5′,6-octaBDE (BDE-203); BDE-209; BB-153; and CB-153.

Chemicals. Reagents and solvents used in the present study were of the highest grade available and/or intended for pesticide residue analysis and were used only after verification by GC-IDHRMS analysis monitoring for the BFRs listed above. Dichloromethane (DCM), *n*-hexane, and methanol were of pesticide grade and purchased from TEDIA (Fairfield, OH), HPLC-grade water also was purchased from TEDIA. Hydrochloric acid (37% in water 99.999%) was purchased from Aldrich (Milwaukee, WI). Formic acid (minimum 88%, ACS grade) was purchased from EM Science, an affiliate of Merck (Darmstadt, Germany). *n*-Nonane (< 99%) and silica gel (63–200 μm) were purchased from Sigma (St. Louis, MO).

Solid-phase extraction (SPE) cartridges packed with OASIS HLB sorbent (540 mg) were obtained from Waters Corporation (Milford, MA). Custom-made SPE cartridges containing activated silica (0.1 g; top layer) and silica:sulfuric acid 2:1 by weight (1 g; bottom layer) were obtained from Applied Separations (Allentown, PA).

Cleaning of glassware and other consumables. Before use, all glassware was cleaned in a dishwasher Steam Scrubber (Labconco, Kansas City, MO) and heated overnight in a Thelco laboratory oven (Precision, Winchester, VA) at 250°C. Pasteur pipettes were not washed in the dishwasher but were heated directly in the same oven overnight. Teflon-lined screw caps and Teflon-lined silicone septa were sonicated in methanol and air dried before use.

Instrumentation. Addition of internal standards, denaturation of the sample using formic acid, and dilution of the sample with water before extraction were automated with a 215 Liquid Handler (Gilson, Middleton, WI) fitted with a 402-syringe pump (Gilson). The syringe pump was equipped with a 250-μL syringe as well as a 10-mL syringe (Gilson). The syringe pump was connected via 10.5-mL coiled-transfer tubing to a septum-piercing probe. A methanol bottle was connected to the syringe pump to purge the transfer tubing and rinse the septum-piercing probe (Gilson) used for drawing and dispensing standard solutions and reagents. Samples were mixed by rotation using an 818 AutoMix (Gilson). This instrumentation was controlled and operated with UniPoint (version 5.5) software (Gilson).

SPE and cleanup were automated using the Rapid Trace SPE workstation (Zymark, Hopkinton, MA). This modular SPE system was controlled and operated using Rapid Trace workstation software (version 1.20; Zymark).

A Rapid Vap (Labconco, Kansas City, MO) was used for sample evaporation employing vortex action, vacuum, and an elevated temperature to aid the evaporation process.

IDHRMS analysis was performed on a MAT95XP instrument (ThermoFinnigan MAT; Bremen, Germany). The chromatographic separations were carried out using a 6890N gas chromatograph (Agilent, Atlanta, GA) fitted with a DB5HT capillary column (15 m, 0.25 mm inner diameter, 0.10 μm film thickness; Agilent). Splitless injection was carried out with an injector temperature of 280°C; the oven was programmed from 140°C (1 min) to 320°C (1.5 min) with a ramp rate of 10°C/min. The source temperature was 280°C in the electron impact mode using a filament bias of 40 eV.

Sample preparation. The analytical method used for sample extraction and cleanup has been recently published (Sjödin et al. 2003b, in press). Briefly, the serum samples were processed in batches of 30 including method blanks [*n* = 3; containing calf serum (0.5 mL) and water (3.5 mL)] and QA/QC samples (*n* = 3; human serum). Serum samples (4 g) were weighed into 16 × 100 mm test tubes fitted with Teflon-lined silicone septa. The samples were transferred to the Liquid Handler for automatic fortification with $^{13}C_{12}$-labeled internal standards. In the same procedure, the samples also were pretreated with formic acid (4 mL) and diluted with water (4 mL), before SPE. The extractions were carried out in an automated procedure overnight on a modular SPE workstation. In this procedure, the samples were loaded onto an SPE cartridge containing 540 mg OASIS HLB sorbent at a flow rate of 0.4 mL/min after conditioning of the sorbent with methanol, DCM, and 0.1 M hydrochloric acid in 5% aqueous methanol solution. After complete loading of the sample, the cartridge was dried with pressurized nitrogen at a pressure of 140 kPa for 40 min. Final elution of the sample from the sorbent was made by DCM (12 mL) at a flow rate of 1 mL/min. The final extract was then evaporated to dryness in the vacuum evaporator.

Coextracted lipids were removed during an automated cleanup procedure on the SPE workstation. The cleanup cartridge comprised two layers packed into a 3-mL SPE cartridge. The top layer in the cartridge was activated silica (0.1 g activated at 250°C, overnight), and the bottom was a mixture of silica and concentrated sulfuric acid (2:1 by weight). The dry sample extracts were loaded into racks for the automated SPE cleanup, which involved reconstitution of the samples with hexane (1 mL) and loading onto the cleanup cartridge. This procedure was repeated with two more 1-mL volumes of hexane to ensure complete reconstitution of the samples. Final elution of the target analytes was achieved

Table 1. Concentration [median (range) ng/g lipid] of selected BFRs and CB-153 in archived serum pools from the United States, stratified according to 5-year collection periods.

Compound	1985–1989 (*n* = 9) Median (range)	1990–1994 (*n* = 14) Median (range)	1995–1999 (*n* = 10) Median (range)	2000–2002 (*n* = 7) Median (range)
PBDEs				
BDE-47	5.4 (< 1–44)	28 (3.7–49)	46 (24–68)	34 (29–98)
BDE-85	< 0.5 (< 0.5–1.08)	0.61 (0.50–1.4)	0.78 (0.50–1.9)	0.70 (0.50–1.4)
BDE-99	< 2 (< 2–15)	10 (1.3–18)	13 (9.1–29)	11 (6.8–26)
BDE-100	0.81 (< 0.5–7.3)	4.0 (0.63–7.7)	6.7 (3.8–14)	5.9 (3.5–18)
BDE-153	0.84 (< 0.5–7.3)	1.6 (0.67–15)	4.2 (2.5–16)	7.3 (1.8–17)
BDE-154	< 0.5 (< 0.5–0.94)	< 0.5 (< 0.5–1.07)	0.88 (0.52–1.8)	0.95 (0.50–1.8)
ΣPBDE	9.6 (4.6–74)	48 (7.5–86)	71 (42–120)	61 (47–160)
PBBs				
BB-153	8.0 (2.6–9.4)	5.3 (1.0–8.6)	4.7 (1.9–10)	3.3 (1.4–5.5)
PCBs				
CB-153	90 (48–140)	66 (38–87)	52 (36–110)	35 (19–49)

with an additional 8 mL hexane. The sample extracts were then evaporated to 0.5 mL and quantitatively transferred to GC vials that had been fortified with recovery standards. The samples were finally evaporated to approximately 10 µL in the GC vials using the vacuum evaporator.

Final analytical measurements of target analytes were performed using GC-IDHRMS, as described above and by Sjödin et al. (2003b, in press). All concentration data reported are background corrected for average amount of analyte detected in blank samples (n = 3 per 24 unknowns). The limit of detection when contaminants were detectable in blank samples was defined as three times the standard deviation of the blank samples. A signal-to-noise (S:N) ratio of 10 was used to define the instrumental limit of detection when no contaminants were detectable in the blank samples.

Calculation of population half-lives. The observed decay half-lives for CB-153 and BB-153 were estimated for the population by using a single compartment model with first-order kinetics (Yang and Andersen 1994). The relationship between chemical concentration (C) and time (t) of sampling with first-order kinetics is expressed in Equation 1, where k_{el} represents the decay rate or the elimination rate constant:

$$C_t = C_0 e^{-k_{el} t} \quad [1]$$

The terminal log-linear phase of elimination in this model was used to determine the half-life ($t_{1/2}$) using Equation 2. The elimination rate constant (k_{el}) is defined by Equation 3, where C and t represent the chemical concentration and the time at which the specimen was collected, respectively.

$$t_{1/2} = \frac{0.693}{k_{el}} \quad [2]$$

$$k_{el} = \frac{(\ln C_1 - \ln C_2)}{(t_1 - t_2)} \quad [3]$$

Results

We determined six PBDEs (tetraBDE to hexaBDE), BB-153, and CB-153 in human serum pools collected during 1985–2002. The median and range of concentrations in these pools with respect to the BFRs and CB-153 are shown in Table 1, stratified according to 5-year collection periods. Time of sample collection is plotted against concentration data for BDE-47, the sum of PBDEs (ΣPBDEs), BB-153, and CB-153 in Figure 1.

Correlation coefficients for the individual target analytes and the ΣPBDEs versus the time of sample collection are shown in Table 2.

The concentrations determined for all PBDEs with the exception of BDE-85 increased significantly during the study period, with BDE-47 being the dominating congener. The median level of BDE-47 increased from 5.4 ng/g lipid in 1985–1989 to 34 ng/g lipid for 2000–2002 (Table 1). In contrast, decreasing levels were observed for BB-153 and CB-153 throughout the study period.

The summary parameters for the linear regression analysis of the logarithmic chemical concentration versus time for CB-153 and BB-153 in 40 samples are presented in Table 3. For CB-153, the observed decay rate was determined to be 0.065/year, and the observed decay half-life was 11 years. For BB-153, the observed decay rate was 0.058/year, and the decay half-life was 12 years.

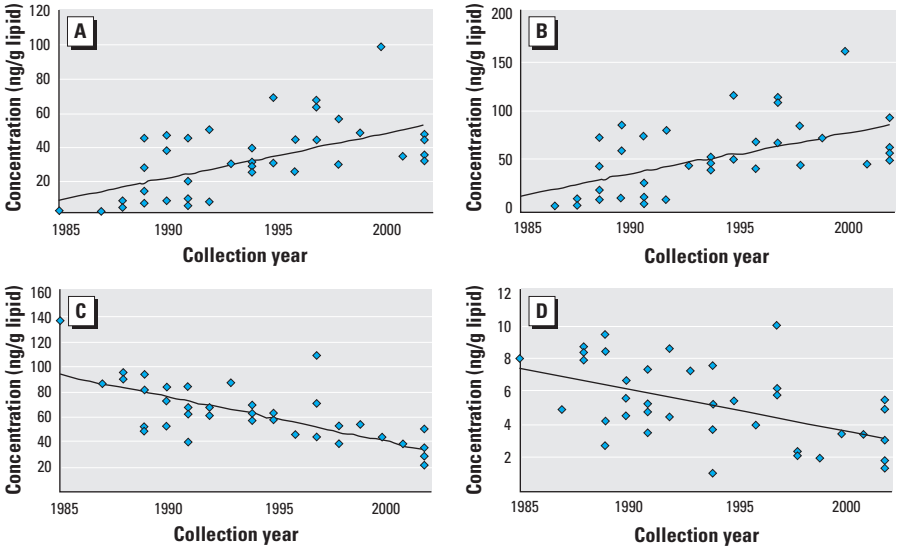

Figure 1. Linear regression trends for concentrations (ng/g lipid) of (*A*) BDE-47, (*B*) the ΣPBDEs, (*C*) CB-153, and (*D*) BB-153 in serum pools plotted against the year of sample collection.

Discussion

Concentrations of PBDEs are well known to have been increasing in Europe from studies conducted in Sweden (Meironyté et al. 1999; Meironyté Guvenius 2002), Norway (Thomsen et al. 2002), and Germany (Schröter-Kermani et al. 2000). More recently, similar findings have been reported for Japan (Akutsu et al. 2003). However, during recent years a decrease in concentration has been indicated in Sweden, probably because of a decrease in use of the lower brominated PBDEs in Europe, as shown by recent use figures [Bromine Science and Environmental Forum (BSEF) 2003]. Estimated use of the pentaBDE mixture in Europe was 150 metric tons/year in 2001. Use of the same product in North America was estimated at 7,100 metric tons in 2001, corresponding to 95% of the total world demand for this product (BSEF 2003).

Information about current and historic concentrations of PBDEs in humans is lacking or limited for North America. However, a recent review in which previously published concentration data on PBDEs were used to construct a time trend, we (Sjödin et al. 2003a) reported increasing levels of PBDEs in North America. The average concentration of BDE-47 in earlier studies was 0.63 ng/g lipid (range, < 0.4–24; n = 12) in serum in 1988 (Illinois; Sjödin et al. 2002); 33 ng/g lipid in breast adipose tissue (range, 7–200; n = 23) in the late 1990s (California Bay, California; She et al. 2002); 83 ng/g lipid (n = 19) in a milk pool in 1997 (New York; Betts 2002); 130 ng/g lipid in a milk pool in 2000 (Austin,

Table 2. Spearman's rank order correlation coefficients and p-levels for the correlation of the measured organohalogen compounds in the United States versus collection year (1985–2002).

Compound	R	p-Value
PBDEs		
2,2´,4,4´-TetraBDE (BDE-47)	0.61	< 0.01*
2,2´,3,4,4´-PentaBDE (BDE-85)	0.22	0.18
2,2´,4,4´,5-PentaBDE (BDE-99)	0.59	< 0.01*
2,2´,4,4,6-PentaBDE (BDE-100)	0.57	< 0.01*
2,2´,4,4´,5,5´-HexaBDE (BDE-153)	0.61	< 0.01*
2,2´,4,4´,5,6-HexaBDE (BDE-154)	0.34	< 0.05
ΣPBDEs	0.55	< 0.01*
PCBs		
2,2´,4,4´,5,5´-HexaCB (CB-153)	–0.70	< 0.01*
PBBs		
2,2´,4,4´,5,5´-HexaBB (BB-153)	–0.51	< 0.01*

*Statistically significant at $p < 0.05$.

Table 3. Regression of the logarithm of CB-153 and BB-153 on time in 40 samples.

	Coefficient	SE	p-Value
CB-153			
Intercept	58	8.2	< 0.001
Time	–0.028	0.0041	< 0.001
BB-153			
Intercept	51	13	< 0.001
Time	–0.025	0.0068	< 0.001

TX, and Denver, CO; Päpke et al. 2001); and 41 ng/g lipid in milk in 2001 (range, 2.9–270; n = 47) (Texas; Schecter 2003). These concentrations compare favorably with those generated in the present study (Table 1). Similarly, the concentrations of the tetraBDEs through hexaBDEs in the U.S. serum pools are significantly increasing over time (Table 2). Furthermore, PBDE levels are substantially higher than those reported for European samples. BDE-47 levels in current (1997–2000) human milk from Sweden range from 1.7 to 2.3 ng/g lipid (Meironyté et al. 1999; Meironyté Guvenius 2002). In people with a high dietary intake of fish from the Baltic Sea (12–20 meals/month) in Sweden, the median PBDE level was 2.2 ng/g lipid (range, 0.96–5.7) for samples collected in 1991 (Sjödin et al. 2000). This can be compared with 0.4 ng/g lipid (< 0.1–2.5) for Swedish people with a negligible consumption of fish (Sjödin et al. 2000). Baltic Sea fish is considered to be a high-risk food with respect to exposure to organohalogen compounds such as PCBs. These studies reveal that Americans who have a median concentration of 34 ng/g lipids (range, 29–98; during 1990–2002) of BDE-47 in their blood were exposed at a much higher degree to lower brominated PBDEs than people in Europe. The exact exposure pathways have not been elucidated; however, they may be similar to other persistent environmental contaminants such as PCBs (Sjödin et al. 2000); that is, the diet may be the major pathway of exposure. On the other hand, direct exposures such as inhalation and/or dermal exposure (Sjödin et al. 1999, 2001) cannot be excluded for chemicals still being used and may have a substantial contribution to the exposures observed.

The serum from which the pools were made was collected by sampling at blood banks and is not representative of the general U.S. population. An important factor to consider in this regard is the large size of our sampling region, covering the southeastern United States and Seattle, Washington. Limited information was available to us about the location where the blood bank collected the samples from which we made the pools. This lack of information prevented us from addressing geographical differences and their effects on the time trend. However, there appears to be increased variability in the concentrations in the pools based on the 2000–2002 pools for BDE-47 from the southeastern United States (n = 4, mean ± SD, 35 ± 7.7 ng/g lipid) and Seattle (n = 3, mean ± SD, 57 ± 35). However, we can conclude that the variability in the concentrations in the pools is large; for example, for both areas combined, the mean ± SD of the BDE-47 concentration (n = 7) was 44 ± 24 ng/g lipids for 2000–2002. One plausible reason for the observed variability may be geographic differences including differences in the diet and lifestyle as well as sex. However, in an earlier Swedish study (Sjödin et al. 2000), no correlation between age of the individuals and PBDE body burden was observed. This may be because PBDEs are currently being used in industry and exposures to the general population have been increasing over time, resulting in similar lifetime exposures independent of the individual's age (Sjödin et al. 2000). Further, the use of pooled samples prevented us from assessing variability individuals including potential effects from the individual's age and/or sex.

In addition to the PBDEs, we measured CB-153 and BB-153 in all the samples. CB-153 is the major PCB congener found in most environmental and human matrices and indicates the total PCB level (Grimvall et al. 1997). We made no attempt to determine the total PCB concentration in our study. BB-153 is the main constituent of technical hexabromobiphenyl (hexaBB). HexaBB has been used and applied as an FR. Although it was banned after an accident in Michigan in the 1970s in which cattle feed was contaminated by this technical product, commercial production of the BB product continued in Europe. To the best of our knowledge, polybrominated biphenyls (PBBs) are no longer commercially produced worldwide.

We found a significant decreasing trend for BB-153 and CB-153 (Table 2, Figure 1). This trend verifies that the levels for these environmental contaminants have been decreasing since the ban of their use in the mid-1970s. The levels of BB-153 are lower than for the main PBDE congener BDE-47, except for 1985–1989 (Table 1). However, in recent years the levels of BDE-47 are comparable or higher than the levels of CB-153 in the serum samples analyzed (Table 1). The highest concentration of CB-153 in the study was close to 140 ng/g lipid weight for a sample collected in 1985. The median concentration for BDE-47 for 2000–2002 was 34 ng/g lipid (range, 29–98 ng/g lipid).

Blanck et al. (2000) estimated the half-life of BB-153 to be in the range of 13–29 years in humans exposed to BB-153 in a dairy feed accident in Michigan in 1973, where animal feeds were accidentally contaminated with PBBs. The half-life of CB-153 in humans occupationally exposed to PCBs has been estimated to be 4–30 years (Ryan et al. 1993; Yakushiji et al. 1984). In another study using a population occupationally exposed to PBDEs, the half-lives of BDE-153 (hexaBDE), BDE-183 (heptaBDE), and BDE-209 (decaBDE) have been estimated to be 680, 110, and 14 days, respectively (Jakobsson et al. 2003). The half-lives in humans for the lower brominated PBDEs (tetraBDEs through pentaBDEs) may be longer because an increase in half-life was observed with decreasing bromination degree for decaBDE, octaBDE, and hexaBDE (Jakobsson et al. 2003); however, this has not yet been demonstrated. In our study, a single-compartment model with first-order kinetics was used to estimate the observed populational half-lives for CB-153 and BB-153. This model is supported by the linear response obtained from the study (Table 3) and when individual human data are used (Blanck et al. 2000; Ryan et al. 1993). Also, animal experiments with PCBs (Clarke et al. 1984; Wyss et al. 1986) and PBB (Fries and Marrow 1978) demonstrated a similar process for elimination. We estimated the populational half-life to be about 11 and 12 years for CB-153 and BB-153, respectively. These estimates compare favorably with earlier findings (Blanck et al. 2000; Ryan et al. 1993; Yakushiji et al. 1984). However, our estimates of the decay half-lives represent not only the elimination of these chemicals from the population but also the population's continuous decrease in exposure to these chemicals over time. Hence, our results are estimates of the population's half-life and not that of the individuals.

The brominated diphenyl ethers are major environmental contaminants in the U.S. general population and represent a health concern based on animal toxicity data. PBDEs cause neurodevelopmental effects in mice dosed with relatively high concentrations of BDE-47 and BDE-99 (Eriksson et al. 2001, 2002; Viberg et al. 2002). Further research is needed to assess whether any adverse effects on human health may be related to the PBDEs at the concentrations reported in this study.

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