High Body Burdens of 2,2′,4,4′-Tetrabromodiphenyl Ether (BDE-47) in California Women

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Following our first report on elevated polybrominated diphenyl ether (PBDE) concentrations in California women, we expanded our investigation to include diverse groups of local women. We analyzed additional adipose and serum samples collected in the late 1990s from San Francisco Bay Area women participating in a breast cancer study and in a reproductive study, respectively. Adipose samples (n = 32) were analyzed by low-resolution mass spectrometry in negative-ion chemical ionization mode, whereas serum samples (n = 50) were analyzed by dual-column gas chromatography with electron capture detection. The results confirmed our earlier findings. Concentrations of 2,2′,4,4′-tetrabromodiphenyl ether (BDE-47) in contemporary California women ranged between 5 and 510 ng/g lipid, with a median (16.5 ng/g lipid) 3–10 times higher than those reported from Europe. In contrast, PBDEs were not measurable in any of 420 archived serum samples collected in the 1960s from San Francisco Bay Area women participating in a study of child development. BDE-47 concentrations did not increase with age or with concentrations of a polychlorinated biphenyl (PCB 153), suggesting other routes of exposure in addition to diet. Rising body burdens of endocrine-disrupting chemicals such as PBDEs may pose a potential public health threat. Key words: adipose tissue, BDE-47, body burdens, California, PBDEs, persistent organic pollutants, polybrominated diphenyl ethers, serum, time trends. Environ Health Perspect 111:1175–1179 (2003). doi:10.1289/ehp.6220 available via http://dx.doi.org/[Online 10 March 2003]

Persistent organic pollutants (POPs) enter the natural environment via a multitude of pathways. Body burdens reflect cumulative exposures to such chemicals and can be used to assess temporal and spatial trends. Body burdens of organochlorine compounds [polychlorinated dibenzo-p-dioxins, polychlorinated biphenyls (PCBs), pesticides] are declining in most of the industrialized countries (Liem et al. 1995; Noren and Meironyte 1998, 2000; Smith 1999) as a result of source reduction measures. Polybrominated diphenyl ethers (PBDEs), on the other hand, show increasing trends worldwide (de Wit 2002). Three industrial formulations of PBDEs are used widely as flame retardants [Bromine Science and Environmental Forum (BSEF) 2001]. Deca-BDE (consisting almost completely of BDE-209) is used mainly in thermoplastics and textiles. In 1999, its use in the United States was estimated at 25,000 metric tons, or 44% of its global use (Hale et al. 2002). Octa-BDE (a mixture of hexa- to octa-BDE congeners) is used in acrylonitrile/butadiene/styrene (ABS) plastics. Its use in the United States was estimated at 1,400 metric tons in 1999, corresponding to about 36% of its global use (Hale et al. 2002). Penta-BDE (a mixture of tetra- and penta-BDE congeners) is used mainly in polyurethane foam. The U.S. market used about 8,000 metric tons in 1999, which is approximately 98% of the global production of penta-BDE (Hale et al. 2002). Although the more brominated formulations are used more extensively worldwide than is penta-BDE, the tetra- and penta-congeners bioaccumulate to a greater degree than do the higher homologues (World Health Organization 1994). In fact, the congener pattern found in biota closely matches the pattern of the penta-BDE formulation (Hale et al. 2002), with 2,2′,4,4′-tetrabromodiphenyl ether (BDE-47) as a dominant congener. Production of all PBDEs has increased over the last 20 years, accompanied by their emergence in environmental and biologic samples (de Boer et al. 1998; de Wit 2002). Hepatotoxicity, embryotoxicity, thyroid, and behavioral effects have been reported in animal studies (Darnerud et al. 2001; McDonald 2002). Of particular concern is the ability of PBDEs to disrupt thyroid hormone balance and to cause behavioral and learning deficits in rodents exposed in utero or postnatally (Eriksson et al. 2001). Elevated PBDE body burdens in women of childbearing age could therefore be an important public health issue.

Concentrations (mean, 86 ng/g lipid) of ΣPBDEs (sum of BDE-47, BDE-99, BDE-100, BDE-153, and BDE-154) in adipose tissues from a group of 23 California women (She et al. 2002) appear to be 3–10 times higher than concentrations reported from other parts of the world (Darnerud et al. 1998; de Wit 2002; Noren and Meironyte 2000; Ohta et al. 2002; Ryan and Patry 2000; Schroeter-Kermani et al. 2000). In addition, concentrations in archived blubber from San Francisco Bay harbor seals demonstrate a 100-fold increase over the last decade (She et al. 2002). In this article, we report on additional PBDE measurements in adipose and serum from diverse groups of California women, in an effort to better understand sources and pathways leading to the observed high levels.

Materials and Methods

Subjects. The PBDE analyses were performed on adipose and serum samples collected in the course of three separate epidemiologic studies. All three studies involved women living in the San Francisco Bay Area of California.

Adipose from the late 1990s. Breast adipose samples from a group of 32 women, residents of the San Francisco Bay Area, were analyzed for PBDEs. This group was a random subsample from a case–control study on breast cancer and organochlorine exposures (Petreas et al. 2000), and it included women with malignancies, ductal carcinoma in situ, and benign breast disease. Participants of the original case–control study were recruited among women undergoing biopsies or lumpectomies at Stanford University Hospital or Kaiser-Oakland Hospital (both in the general San Francisco Bay Area). Eligibility criteria included age between 25 and 65 years, no prior cancer, and not taking tamoxifen or undergoing chemotherapy. Breast adipose tissue (~1 g) was collected during biopsy or lumpectomy. Adipose from the late 1990s.

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breast surgery between 1996 and 1998 and archived at −20°C until analysis. The group of women discussed in this study includes the 23 women whose PBDE concentrations have already been reported (She et al. 2002).

**Serum from the late 1990s.** Between 1997 and 1999, blood was collected from a group of 50 Laotian immigrant women who lived in the San Francisco Bay Area and were participating in a study of organochlorine exposures and menstrual cycle function (Windham et al. 2002). Convenience sampling was used with trained Laotian community field workers, recruiting participants from Asian markets, cultural events, friends, health clinics, and English language classes. Eligibility criteria included reproductive age (19–40 years), recent menstrual period, birth in Southeast Asia, and regular consumption of fish. Blood was drawn at a local clinic and serum was separated and archived at −20°C until analysis.

**Serum from the early 1960s.** For a historic comparison population, archived serum was obtained from a case–control study of cryptorchidism and hypospadias and *in utero* exposure to organochlorine pesticides (OCPs) nested within the historic Child Health and Development Studies (CHDS) cohort. The CHDS (van den Berg 1979), a longitudinal study of 20,000 pregnancies among Northern California Kaiser Foundation Health Plan members, enrolled subjects between 1959 and 1966, a time of unrestricted use of PCBs and OCPs. Mothers were followed during pregnancy, and multiple samples of serum from each pregnancy were collected and archived. A subset of these children was followed for subsequent follow-up examinations. Subjects selected for this study included 155 male infants with hypospadias or cryptorchidism and surviving for 2 years, as well as twice that number of randomly selected controls. Maternal serum from the second or third trimesters of pregnancy was retrieved from archived frozen samples stored at the National Cancer Institute (Frederick, MD). Serum from 420 women was available for this comparison.

**Analytical methods.** Serum and adipose samples were kept frozen below −20°C until analysis. Serum was thawed, and 1 mL was pipetted into a 15-mL test tube. Internal standards [PCB congeners 14, 65, and 166 and tetrachloro-m-xylene (TCMX)] were added before denaturing the proteins with 1 mL acetic acid (California Department of Toxic Substances Control 2003). The analytes in the serum were then extracted four times with 3 mL hexane/dichloromethane (1:1, vol/vol), the eluates were combined and reduced to 75 µL. Twenty-five microliters of a 61.9 µg/µL solution of the recovery standards [pentachloronitrobenzene (PCNB), PCB-30, PCB-204, and PCB-209] were added to the extract to achieve a final concentration of 15.5 µg/µL. Calibration solutions ranged in concentration from about 0.2 µg/µL to 500 µg/µL of BDE-47 and 15.5 µg/µL of PCNB, PCB-30, PCB-204, and PCB-209. Analysis was performed by gas chromatography/electron capture detection (GC/ECD) equipped with 60-m DB-XLB (Agilent Technologies, Wilmington, DE) and Rtx-5ms (Restek Corporation, Bellefonte, PA) capillary GC columns. In previous work (James et al. 2002), 60-m DB-17 and Rtx-5ms columns were used to provide congener separation of the PCBs. However, in this project, we discovered that using the combination of DB-XLB and Rtx-5ms columns provided better resolution of the PCB congeners. By using a long temperature program, the retention time of BDE-47 exceeded the retention time of most PCBs.

Quantitation for BDE-47 was accomplished by using the data obtained on the DB-XLB column. The serum samples were processed in batches of nine. With each batch, we processed 1 mL HPLC-grade water (reagent blank) and 1 mL bovine serum fortified with BDE-47 (among other analytes) to evaluate background contributions from the reagents, precision, and analyte recovery. In addition, samples of pooled human serum were interspersed blindly among the samples (for quality control) to assess accuracy and precision across all serum batches. Total cholesterol and triglycerides were determined enzymatically in a small aliquot of serum at the Clinical and Epidemiological Research Laboratory, Boston Children’s Hospital (Boston, MA). Total lipids were calculated from total cholesterol and triglycerides as described by Phillips et al. (1989), and results were reported as nanograms per gram lipid.

As described in detail previously (She et al. 2002), adipose samples were homogenized and internal standards were added, including 13C12-labeled BDE-77. Although we could have used unlabeled standards for this work, 13C12-labeled BDE-77 allowed us a comparison of instrumental techniques (not reported here). Samples were extracted with 1:1 hexane/dichloromethane, and PBDEs were isolated by passing the extract through a gel permeation chromatographic column and a glass column packed with Florisil in a single automated step (FMS, Waltham, MA). The extracts were concentrated and recovery standards included 13C12-PCB-128, 13C12-PCB-178, and 13C12-α-hexachlorocyclohexane. BDE-47, BDE-99, BDE-100, BDE-153, and BDE-154 were analyzed by low resolution mass spectrometry (Finnigan 4510; Finnigan MAT, San Jose, CA) in the negative ion chemical ionization mode using a DB-5ms column (60 m, 0.25 mm inner diameter, 0.25 µm film thickness; J&W Scientific, Folsom, CA). Methane was used as the reagent gas; the ion source pressure was 0.6 Torr, and the ion source temperature was 100°C. The electron energy was typically 70 eV, and the electron current was kept at 0.3 mA. We monitored m/z 79 and 81, corresponding to bromide. Adipose samples were analyzed in batches of six, with a reagent blank per batch. Samples of certified reference material (SRM 1945, whale blubber; National Institute of Standards and Technology, Gaithersburg, MD) were analyzed to assess accuracy and precision. Lipid content of the adipose samples was determined gravimetrically in an aliquot of the extract, and PBDE results were reported as nanograms per gram lipid.

Statistical analyses to examine correlations between analytes and to compare BDE-47 body burdens among groups and by age were performed using STATA 7 (Stata Corp., College Station, TX).

**Results**

Serum and adipose samples from all three studies (Petreas et al. 2000, 2002; Windham et al. 2002) were originally scheduled for analysis of PCBs and OCPs (She et al. 1997), with the PBDEs added on as a secondary objective after analytical methods were developed. Only 1 mL of serum was available for analysis, limiting our ability to detect low levels. Therefore, only

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**Table 1. Demographic characteristics of the three groups of California women.**

| Group                     | Breast cancer | Reproductive (Laotian) | Child development |
|---------------------------|---------------|------------------------|-------------------|
| No.                       | 32            | 50                     | 420               |
| Years sampled             | 1996–1998     | 1997–1999              | 1959–1967         |
| Age (years)               |               |                        |                   |
| Mean ± SD                 | 47.4 ± 7.7    | 31.4 ± 6.2             | 26.8 ± 6.2        |
| Median (range)            | 48 (28–62)    | 32 (19–40)             | 20 (15–44)        |
| Race (%)                  | White         | 62.5                   | 0                 |
|                           | African American | 25.0                 | 0                 |
|                           | Asian         | 12.5                   | 100               |
|                           | U.S. born (%) | 84.4                   | 0                 |
|                           | Prior pregnancy (%) | 75                  | 88                |

*All participants in this group were pregnant during blood draw; value refers to previous pregnancy.*
BDE-47 could be measured in serum without interference from the blank (signal in the sample should be at least 3x the signal in the blank), resulting in a much higher reporting limit for BDE-47 in serum (10 ng/g lipid) than in adipose tissue (< 0.5 ng/g lipid). BDE-47 was the major congener in adipose samples, and BDE-99, BDE-100, BDE-153, and BDE-154 were also measurable in all adipose samples. Table 1 shows demographic characteristics and Table 2 shows concentrations of BDE-47 and PCB-153 in the three groups of California women.

As shown in Table 1, the 32 women in the breast cancer subsample examined here were 28–62 years of age; most (84.4%) were born in the United States, and 62.5% were white. More than 95% had a college education, and 75% had at least one prior birth. Of the immigrant group, most participants were born in Laos, where they had spent the first part of their lives (mean ± SD, 11.8 ± 6.1 years), and had immigrated to the United States within the previous 2–20 years (14.4 ± 4.5 years), after various lengths of time (5.4 ± 4.8 years) in Thailand, presumably in refugee camps. Half the women had only an elementary school level education, and 18% had completed high school; most (88%) had a prior pregnancy. Among the women from the CHDS, the age ranged from 15 to 44 years (Table 1); 56% had one or more prior pregnancies, and 56% had graduated from high school and 14% from college. Most women were white (63.6%) or African American (26.4%). About 79% were born in the United States, 9.3% were foreign born (about 2% were born in Asia), and the birthplace was unknown for 11.7%.

We analyzed 32 adipose samples and 50 serum samples from the late 1990s and 420 serum samples from the 1960s. All samples were analyzed individually. We had no coelutions on the DB-XLB column with any PCB or OCP expected in human serum (James et al. 2000). Recoveries of internal standards (PCB congeners 14, 65, and 166 and TCMX) were used to gauge overall data quality for all analytes across all serum batches. Recoveries were between 81% and 99%, and no corrections were made to the measurements. In addition, BDE-47 recoveries in fortified bovine serum included with every batch were between 93% and 113%.

As shown in Table 2, BDE-47 was measurable in all adipose samples (100% above the reporting limit), with concentrations ranging from 5.2 ng/g lipid to 196 ng/g lipid, and a mean of 28.9 ng/g lipid. Concentrations in the 1990s serum ranged from < 10 ng/g lipid (reporting limit) to 511 ng/g lipid, with a mean of 50.6 ng/g lipid (median, 16.5 ng/g lipid). Summary statistics were calculated using the reporting limit for all samples at or below that limit. BDE-47 was not measurable in any of the 1960s serum samples. Despite the limitation of an elevated reporting limit, it is clear that none of the 420 serum samples from the early 1960s contained BDE-47 above the reporting limit of 10 ng/g lipid, whereas BDE-47 was measurable in 24 of the 50 serum samples from the late 1990s with the same reporting limit. PCB-153 was measurable in all specimens (adipose and serum) from all three groups of women (Table 2).

The Spearman correlation coefficient ($r$) between BDE-47 concentrations and age for the 32 mostly U.S.-born women (adipose samples) was $r = -0.413$ ($p = 0.019$), indicating a significant negative association. The correlation was not significant for the 50 Laotian serum samples ($r = 0.079$, $p = 0.589$) or for the two 1990s groups combined ($r = 0.058$, $p = 0.606$). In contrast, for the same two 1990s groups, a significant correlation was found for PCB-153 and age ($r = 0.619$, $p < 0.001$). No correlation was found between BDE-47 and PCB-153 ($r = 0.062$, $p = 0.647$) for the same two groups of women combined.

**Discussion**

Noren and Meironyte (1998) reported that concentrations of PBDEs (BDE-28, BDE-47, BDE-66, BDE-85, BDE-99, BDE-100, BDE-153, and BDE-154) in human milk from Sweden were increasing exponentially over time. Several reports on PBDEs in biota followed (Aalae et al. 1999; Darnerud et al. 1998; de Boer et al. 1998; Ryan and Patry 2000; Schroeter-Kermani et al. 2000), including ours (She et al. 2000, 2002), which showed an exponential increase of BDE-47, BDE-99, BDE-100, BDE-153, and BDE-154 in archived blubber from San Francisco Bay harbor seals. In the same study, we reported high concentrations of BDE-47, BDE-99, BDE-100, BDE-153, and BDE-154 in adipose tissues collected from San Francisco Bay Area women in the late 1990s. BDE-47 was the dominant PBDE congener in all of our samples, in agreement with other human studies. Because our current work focuses on BDE-47, we will limit our discussion to that congener in our comparisons with other studies.

Soon after our first study (She et al. 2000), a single composite human milk sample collected in 2000 from the United States showed high concentrations of several PBDE congeners, including BDE-47 (120 ng/g lipid) (Papke et al. 2001), confirming our finding that U.S. levels appeared higher than levels reported in European studies, whose averages ranged within 1–10 ng/g lipid (Darnerud et al. 1998; Noren and Meironyte 2000; Schroeter-Kermani et al. 2000; Thomsen et al. 2002; van Bavel et al. 2002). Conversely, when 12 serum samples collected in 1988 at an Illinois blood bank were analyzed for PBDEs, the median concentration of BDE-47 was 0.6 ng/g lipid, with a range of < 0.4–24 ng/g lipid (Sjödin et al. 2001). According to the authors, these 1988 U.S. levels were considered equivalent to levels measured in Swedish blood collected in 1995 (Sjödin et al. 2001). When we compare these 1988 Illinois serum concentrations (Sjödin et al. 2001) with those we reported in our 1996–1998 California adipose samples (She et al. 2002) and in the composite milk sample (Papke et al. 2001), we can see an increase in U.S. body burdens of BDE-47 over the last 10–15 years. Our newest data support this observation. As shown in Table 2, concentrations of BDE-47 were below the reporting limit (10 ng/g lipid) in all the serum samples from the 1960s, whereas concentrations in 1990s adipose tissues averaged 28.9 ng/g lipid (median, 16.5 ng/g lipid), and concentrations in the late 1990s serum averaged 50.6 ng/g lipid (median, 10 ng/g lipid). A similar increase has been reported for human milk from Canada, where median PBDE levels have increased from 1.7 ng/g lipid in 1992 to 25.4 ng/g lipid in 2001 (Ryan et al. 2002). Although differences in PBDE partitioning in the various matrices examined (adipose, serum, milk) may confound precise comparisons, it is clear that for samples collected in the 1990s from the United States and Canada, body burdens are 3–10 times higher than those reported from Europe (Darnerud et al. 1998; Noren and Meironyte 2000; Schroeter-Kermani et al. 2000; Thomsen et al. 2002; van Bavel et al. 2002) or Japan (Ohta et al. 2002). This observation may be consistent with California regulations mandating that all polyurethane foam and textiles
used in furnishings pass a flammability test (not necessarily requiring use of PBDEs) (State of California 1991, 2000). It is possible that these unique California regulations drive the consumer product market across the United States and, perhaps, Canada. Regional differences have also been reported in sewage sludge, where PBDE concentrations appeared 10 times higher in the United States than in Europe (Hale et al. 2001, 2002).

The Laotian immigrant women in this study had much higher dichlorodiphenyl-dichloroethylene (DDE) and dichlorodiphenyl-trichloroethene (DDT) concentrations (as expected) and quite lower PCB levels in their serum than those measured in the adipose tissue of the mostly U.S.-born women sampled in the same period (Petreas et al. 2002; Windham et al. 2002). It is therefore noteworthy that the Laotians showed serum BDE-47 concentrations equivalent to (or even greater than) adipose concentrations of the contemporary group that was mostly born in the United States. We should note, however, that comparing these two groups is complicated for several reasons: not only are we comparing serum with adipose levels but we are also comparing women of different ages who were born and raised in different continents with different lifestyles. Although we are not familiar with blood/adipose partition coefficients for PBDEs, it is well established that, for most POPs, lipid-adjusted concentrations in serum and adipose tissue are not exactly the same (Archibaque-Engle et al. 1997; Lopez-Carrillo et al. 1999; Mussalo-Rauhama 1991; Needham et al. 1990). As shown in Figure 1, there is little age overlap between the two study populations, and the younger Laotian women appear to have higher and more variable levels of BDE-47 than do the older, mostly U.S.-born women. Because penta-BDE formulations are not widely used in Asia (BSEF 2001), it seems unlikely that these Laotian women were exposed in Laos or in the Thai refugee camps. Thus, it is more likely that practices and lifestyles acquired more recently in the United States are the main exposure pathways. Fish consumption was one of the eligibility criteria for the Laotian women; therefore, this is another source of exposure that may differ from the other populations. These Laotian women consumed fish from the San Francisco Bay in amounts similar to those of an earlier study of anglers fishing in the bay (California Department of Health Services (CDHS) 2001)). Although we do not have data on PBDE levels in bay fish, we have reported an exponential increase in PBDEs in harbor seals (She et al. 2002) from the San Francisco Bay.

In addition, there are fish advisories recommending limited bay fish consumption because of contamination with PCBs and mercury. Similar to PCBs, PBDEs would be expected to accumulate in fatty fish. The anger report (CDHS 2001) noted that Asians are more likely than other ethnic groups to prepare and consume fish in a manner that is likely to increase their exposure to contaminants. Almost all of the Laotian women consumed at least some marine fish, compared with about 30% of the U.S. population (U.S. EPA 1997). On the other hand, given the relatively low PCB body burdens in our Laotian women, fish consumption could not be a major pathway for PBDEs. At this time, we can only speculate on possible explanations for these differences, including different activities or lifestyle factors affecting exposure, age effects on metabolic clearance of BDE-47, or greater BDE-47 partitioning in serum than in adipose tissue.

A lack of a significant increase with age, in contrast to other POPs, has been reported for PBDEs (Darnert et al. 1998; Thomsen et al. 2002), and our data appear to support this. The correlation between BDE-47 and age was not significant for both groups combined. When we examined the two groups separately, the correlation was similarly not significant for serum alone, but a significant negative correlation was seen in adipose tissue. Thomsen et al. (2002) reported higher PBDE concentrations in young children compared with other age groups in Norway, but no differences among age groups corresponding to those seen in our study. We had reported higher ΣPBDE concentrations \( (p < 0.05) \) in the adipose of women younger than 48 years (median age) compared with those older than 48 years in a subset of 23 women (She et al. 2002). Expanding the sample size to 32 women retained the difference between young and old and revealed this significant inverse correlation with age. Given the small number of samples, however, and the presence of outliers, this association may be spurious. Regardless, the important finding is that the association between BDE-47 and age is not positive, a departure from other POPs. When we examined the two 1990s groups of women combined, the concentration of PCB-153 (the most prominent PCB congener in humans) increased with age. On the other hand, there was no correlation between BDE-47 and PCB-153 concentrations in our samples, possibly indicating different exposure pathways for BDE-47 from those for other POPs, which are driven by diet. A strong possibility is inhalation or ingestion of indoor air dust from PBDE-treated consumer products, particularly polyurethane foam. PBDEs have been reported in dust collected with vacuum cleaners from European offices (Leonards et al. 2001) and homes (Knoth et al. 2002). Although BDE-209 was the dominant congener in office and house dust, BDE-47 was also present, ranking second or third in concentration.

Our data indicate the presence of outliers. These high BDE-47 values did not parallel high levels of PCBs or pesticides measured in the same samples (Petreas et al. 2002). High BDE-47 levels were not explained by any of the common POPs predictors such as age, parity, lactation, occupation, country of birth, socioeconomic status, or medical history. Because the study questionnaires were not designed to assess PBDE exposures, questions that might elucidate exposure pathways were not included. Outliers in PBDE distributions have also been reported recently for Swedish blood (van Bavel et al. 2002) and Canadian milk (Ryan et al. 2002). It is possible that these outliers reflect hot spots originating from the relatively recent introduction of PBDEs and its still inconsistent presence in the food web, as well as from the selective use of various PBDE-treated consumer products.

Our analysis showed the emergence of BDE-47 in residents of the San Francisco Bay Area by the late 1990s, compared with the 1960s, confirming similar trends from Canada (Ryan et al. 2002), Germany (Schroeter-Kermami et al. 2000), Sweden (Noreen and Meironyte 1998, 2000), and Norway (Thomsen et al. 2002). Our study has several weaknesses stemming from the fact that the three epidemiologic studies we used were not designed to assess PBDE exposures. Sample volumes were small, raising the reporting level and limiting the numbers of detectable samples, which in turn did not allow analysis of subgroups at higher risk of exposure. In addition, only the most dominant PBDE, BDE-47, could be measured in the serum samples, precluding analysis of PBDE profiles. Nevertheless, BDE-47 concentrations were high, pointing to the need for follow-up studies designed to investigate PBDE exposures. Increasing body burdens, particularly in young women of reproductive age, pose a potential public health threat to future generations. PBDE sources need to be recognized, evaluated, and controlled to minimize exposures. At the same time, the systematic monitoring of

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**Figure 1.** BDE-47 concentrations (ng/g lipid) in serum samples from Laotian women and adipose samples from mostly U.S.-born women. All samples were collected between 1998 and 1999.
body burdens of known and emerging POPs should become a high priority for our public health system (Hooper and McDonald 2000).

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