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Human Exposure to PBDEs: Associations of PBDE Body Burdens with Food Consumption and House Dust Concentrations

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This study was designed to determine the body burden of polybrominated diphenyl ethers (PBDEs) among first-time mothers in the Greater Boston, Massachusetts area and to explore key routes of exposure. We collected breast milk samples from 46 first-time mothers, 2−8 weeks after birth. We also sampled house dust from the homes of a subset of participants by vacuuming commonly used areas. Data on personal characteristics, diet, home furniture, and electrical devices were gathered from each participant using a questionnaire. Breast milk and dust samples were analyzed for PBDEs using gas chromatography/mass spectrometry. PBDE concentrations were log-normally distributed in breast milk and dust. We found statistically significant, positive associations between PBDE concentrations in breast milk and house dust ($r = 0.76$, $p = 0.003$, not including BDE-209), as well as with reported dietary habits, particularly the consumption of dairy products ($r = 0.41$, $p = 0.005$) and meat ($r = 0.37$, $p = 0.01$). Due to low detection rates, it was not possible to draw conclusions about the association between BDE-209 in milk and dust. Our results support the hypothesis that the indoor environment and diet both play prominent roles in adult human exposure to PBDEs.

Introduction

Polybrominated diphenyl ethers (PBDEs) are commonly used as fire retardants in consumer products such as foam cushions, carpets, and televisions. PBDEs are structurally similar to polychlorinated biphenyls (PCBs) and appear to act similarly in the environment, persisting over long periods of time and bioaccumulating in various species (*1*). While there are virtually no data on the effects of PBDEs on human health, results of animal studies suggest reproductive/developmental effects, neurotoxicity and endocrine disruption (*1*−*3*).

Interest in PBDEs increased following the publication of a 1999 Swedish study of PBDEs in breast milk. Analysis of samples stored since 1972 indicated a steep increase in PBDE concentration with a doubling time of 5 years (*4*). Additional studies have observed this trend in the United States (U.S.) and showed that concentrations in the U.S. are at least an order of magnitude higher than in Europe and display considerable between-subject variability (*5*−*7*).

Given the lipophilicity of PBDEs and their presence in consumer products and house dust, suspected routes of human exposure include both diet and the indoor environment. Several studies measured PBDEs in common food products or aggregated diets (*8*−*10*), while others examined PBDEs in house dust (*11*−*18*). Previous estimates of PBDE exposure suggest that incidental ingestion of dust may be important, particularly for children (*15*−*20*), but calculations were based on very uncertain exposure factors for dust ingestion, especially for adults (*21*). Few studies have empirically examined the association between an individual's body burden and dietary habits (*22*−*24*), while only one small study has explored the link between an individual's PBDE body burden and house dust concentrations, finding no association (*13*).

The primary objectives of our study were to characterize PBDE levels in breast milk collected from first time mothers in the Greater Boston (Massachusetts) area and determine whether diet and/or dust are significant determinants of the total absorbed dose of PBDEs.

Materials and Methods

Recruitment of Study Participants. Eligibility for participation was based on a World Health Organization (WHO) protocol for breast milk monitoring (*25*), although we reduced the WHO's 5 year residency requirement to facilitate recruitment. Participants were first-time mothers, 18 years or older, who had lived in the Greater Boston area for at least 3 years at delivery. Participants spoke English or Spanish and had pregnancies that were healthy and singlet. To provide diversity, we recruited participants at three sites: a health center in Lowell, Massachusetts that serves an ethnically diverse, working class community; a private obstetrics office in Cambridge, Massachusetts; and a maternity center in Brookline, Massachusetts. The Cambridge and Brookline facilities serve similar populations, predominantly white and highly educated. Participants were given information about PBDEs and biomonitoring as well as breastfeeding support and education. We provided a small stipend to participants in exchange for milk samples. At the Lowell facility, where existing breastfeeding rates were low, lactation support and manual breast pumps were also distributed to boost breastfeeding rates. The study protocol was approved by the Institutional Review Boards (IRB) at Boston University Medical Center and University of Massachusetts Lowell. All participants gave informed consent prior to enrollment.

Sampling and Analysis of Breast Milk. A single 50 mL breast milk sample was collected from participants 2−8 weeks post-partum between April 2004 and January 2005. Most women used an electric or manual breast milk pump to collect the sample, pumping directly into glass storage jars that had been rinsed with analytical grade solvents and fitted with a Teflon cap liner. Samples were frozen at −20 °C and shipped to ERGO Research Laboratory.

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All analyses were performed following the isotope dilution method. Twelve native standards (^{12}C labeled BDE 17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 183) were obtained from Cambridge Isotope Laboratories (Andover, MA); BDE-209 was obtained from Wellington Laboratories (Guelph, Canada). Six internal ^{13}C labeled standards were obtained from Wellington (BDE 28, 47, 99, 153, 154, 183); BDE-209 was obtained from Cambridge Isotope Laboratories. Solvents were obtained from Merck (n-pentane), Baker (diethyl ether), and Mallinckrodt (ethanol, toluene). Silica gel, alumina oxide, sodium sulfate, and potassium oxalate of the highest purity commercially available were obtained from Merck. Before extraction, the mixture of 7 internal PBDE standards was added to the sample. 5−10 mL of human milk was extracted three times with pentane, after adding 5 mL of water, 1 mL potassium oxalate solution, 10 mL ethanol, and 5 mL ether. The extract was washed with water and dried over sodium sulfate. After solvent evaporation gravimetric lipid determination was performed. The extract was cleaned up by acid treatment and passed through activated silica gel and alumina oxide columns. The final extract was reduced in volume under a stream of nitrogen. The final volume was 50 uL containing ^{13}C labeled BDE-139 for a recovery standard. The measurements were performed using gas chromatography/high-resolution mass spectrometry (HP 5890 coupled with VG Autospec) at RP = 10 000 using a DB 5 (30 m, 0.25 mm ID, 0.1 um film) column for gas chromatographic separation.

Solvents and reagents were tested before the laboratory procedures. All glassware was rinsed by analytical grade solvents prior to use. Silica gel and sodium sulfate were prewashed. Rotary evaporators were not used to reduce the risk of contamination. No plastic equipment was used. For quality control, a laboratory blank and a QC pool of human milk was run with each batch of ten samples. Quantification was only done if sample level was at least twice the blank level. For statistical analysis, half of the detection limit was used for nondetected congeners. Methodologies and QC/QA have been described elsewhere (*26*, *27*).

Sampling and Analysis of House Dust. We recruited Cambridge and Brookline participants for dust sampling. We collected dust as soon after milk sampling as was convenient for participants (1−43 days, median = 18 days). Based on Rudel et al. (*11*), we used a Eureka Mighty-Mite canister vacuum cleaner with a cellulose thimble inserted into a Teflon crevice tool. Study staff vacuumed commonly used rooms, recording the vacuumed surface area (24.7 − 95.9 m^2; median = 62.3 m^2). Pre-extracted soil was run through the vacuum to estimate PBDE levels contributed from the sample collection procedure. Dust samples were transferred to sample jars, frozen at −20 °C and shipped to the Virginia Institute of Marine Science.

Dust samples were sieved to <125 um and a surrogate standard, PCB-204, was added. Blanks were run concurrently with the samples to assess possible introduction of laboratory contamination. Samples were subjected to enhanced solvent extraction with methylene chloride. Large molecular weight biogenic compounds were separated from the PBDEs by chromatography of the extracts on an Envirosep size exclusion column. The resulting fraction of interest was further purified on 2000 mg silica gel solid-phase extraction columns. Following solvent exchange to hexane, PBDEs in the purified extracts were separated on a gas chromatograph, equipped with a 60 m DB-5 column. Helium was used as the carrier gas, and injections were made in the splitless mode with pentachlorobenzene as the internal standard. Data were corrected based on recovery of surrogate PCB-204 in each sample (average recovery = 93%). Identification was accomplished by GC/MS in the full scan electron ionization mode (Varian Saturn 4-D). Quantitation was done by comparison of the sum of the areas of the three major ions of each congener versus that of the internal standard (Cambridge Isotope Laboratories, Andover, MA). BDE-209 was analyzed using a 30 DB-5 column and a JEOL GCMate MS in electron capture negative chemical ionization mode. Decachlorodiphenyl ether was used as the internal standard. m/z 35/37 and m/z 79/81 were monitored for decachlorodiphenyl ether and BDE-209, respectively.

As the concentrations of PBDEs in dust blanks were very low compared with samples, sample results were not blank-corrected. Detection limits varied by dust sample due to the variation in sieved sample masses (see the Supporting Information); for samples with sieved masses of approximately 0.1 gram, the detection limits were 0.5 ug/g for BDE-209 and 0.05 ug/g for other congeners. Nondetected values were treated as zero in statistical analyses.

Questionnaire. We administered a questionnaire designed to collect information on potential routes of exposure to PBDEs as well as general demographic and health parameters. Participants were interviewed in person and each questionnaire took approximately 30 minutes to complete. Questions covered general health, residential history, electronic products, furniture likely to contain foam, estimates of pre-pregnancy food consumption (diet often changes during pregnancy), occupational history, hobbies, recent home renovation, and typical methods of transportation. Dietary serving sizes were defined based on United States Department of Agriculture (USDA) guidelines; photographs of serving sizes of various foods were used to help participants estimate quantities consumed. Participants estimated consumption as never, less than one serving per week, one serving per week, 2−3 servings per week, 4−6 servings per week, one serving daily, or more than one serving per day. Responses were converted to a linear scale for analysis. We examined individual food products as well as total dairy, total meat, and total fish consumption. A dairy fat index estimating the amount of dairy fat consumed per day was constructed based on questionnaire responses and industry established fat content for each product (See the Supporting Information). Potential exposure to PBDEs in electronics was assessed in several ways: the number of computers, televisions, and other electronic devices in homes; the reported hours each item was in use; an index summing all electronics; and an index estimating the total wattage consumed by reported electronics (hypothesizing that wattage may serve as a crude surrogate for size).

Statistical Analysis. We defined ∑PBDE as the sum of congeners found in breast milk: PBDE congeners 17, 28, 47, 66, 85, 99, 100, 138, 153, 154, 183, and 209. Fewer congeners were analyzed in dust: BDE 47, 66, 85, 99, 100, 138, 153, 154, and 209. BDE-209 was only detectable in the larger dust samples and 24% of milk samples. We therefore defined D-∑PBDE as the sum of congeners 47, 66, 85, 99, 100, 138, 153, and 154. As congeners 47, 99, 100, 153, and 154 are major components of the penta commercial mixture (*2*), D-∑PBDE may be considered as roughly penta-like. Associations between PBDE concentrations in breast milk and dust were analyzed for D-∑PBDE and congeners detected in all samples.

Log-normality of PBDE concentrations was assessed using quantile−quantile plots and Shapiro-Wilks tests. Accordingly, PBDE data were log-transformed prior to statistical analyses except for nonparametric tests. We compared PBDE concentrations in breast milk and dust with dietary, occupational, and consumer habits as well as demographic parameters and housing characteristics. Potential associations were explored using scatter plots, stepwise regression, and correlation analysis. A priori hypotheses regarding exposure via diet and dust were explored in more detail using multiple regression to adjust for potential confounding by other dietary or personal factors. Exponentiation of the regression coef-

TABLE 1. Summary Statistics for PBDEs in Breast Milk and Dust

	breast milk (ng/g lipid)				dust (ug/g)			
BDE	median	min	max	% detect	median	min	max	% detect
17	<DL[d]	<DL[d]	0.1	50	NA[c]	NA[c]	NA[c]	NA[c]
28	0.9	0.1	6.0	100	NA[c]	NA[c]	NA[c]	NA[c]
47	13.9	2.0	126.6	100	0.67	0.24	14.61	100
66	0.1	<DL[d]	2.1	93	<DL[d]	<DL[d]	0.29	27
85	0.3	<DL[d]	7.1	98	<DL[d]	<DL[d]	0.79	36
99	2.4	0.4	84.3	100	1.01	0.29	14.80	100
100	2.4	0.4	26.8	100	0.17	<DL[d]	2.78	73
138	0.03	<DL[d]	0.8	67	<DL[d]	<DL[d]	0.08	18
153	3.0	0.4	91.7	100	0.11	<DL[d]	0.56	55
154	0.2	0.04	4.6	100	0.09	<DL[d]	0.46	55
183	0.1	<DL[d]	0.5	72	NA[c]	NA[c]	NA[c]	NA[c]
209	<DL[d]	<DL[d]	10.9	24	<DL[d]	<DL[d]	9.6	45
∑PBDE[a]	30.2	4.3	263.5	100				
D−∑PBDE[b]	28.9	3.9	260.8	100	1.91	0.59	34.4	100

[a] ∑PBDE = Sum of congeners 17, 28, 47, 66, 85, 99, 100, 138, 153, 154, 183, and 209 [b] D-∑PBDE = Sum of congeners 47, 66, 85, 99, 100, 138, 153, and 154 [c] NA = Not analyzed [d] <DL= Below detection limit (See Supporting Information)

ficient provides the fold increase in PBDE concentrations in breast milk per unit of exposure. Statistical analyses were performed using SAS and Microsoft Excel with statistical significance set at 0.05.

Results

Fifty women enrolled in the study and completed the questionnaire. Four women dropped out after giving birth and 46 provided a breast milk sample. Recruitment at the Lowell site was low ($n = 5$). Diversity among the participants was therefore limited; of the 46 participants, 41 were white, and 40 had completed a college education. The age range for the majority of participants was relatively small (see the Supporting Information). Only 12 participants agreed to dust sampling (11 households, since two participants lived together). Participants in the dust study appeared to be similar to the overall study population (see the Supporting Information). Sampled homes were 2−105 years in age (median of 46 years). Six were urban and five suburban, while nine of the 11 homes had pets.

PBDE Concentrations in Breast Milk and Dust. PBDE concentrations in breast milk were log-normally distributed, ranging from 4.3 to 264 ng ∑PBDE /g lipid with a median of 30.2 ng/g lipid; see Table 1 and the Supporting Information. These results appear similar to those found elsewhere in the U.S. and higher than concentrations found in Asia and Europe (*5*, *7*).

The average congener composition of breast milk samples was 52% BDE-47, 17% BDE-153, 13% BDE-99, 11% BDE-100, and 7% other. This overall composition is similar to that reported in previous studies (*5*). While BDE 47 was the dominant congener for the majority of subjects, BDE-153 was the dominant congener in 7% of the participants (see the Supporting Information), an observation that has been noted in previous studies although the cause is unknown (*28*, *29*). BDE-209 was detected in only 11 of the 46 samples.

Dust concentrations were approximately log-normally distributed, ranging from 0.6 to 34.4 ug D-∑PBDE/g with a median of 1.9 ug/g (Table 1 and Supporting Information). Mass of PBDEs per square meter of vacuumed area ranged from 0.28 to 320 ng D-∑PBDE/m^2 with a median of 7.0 ng/m^2, an alternative measure that was highly correlated with dust concentration in ug/g ($r = 0.87$, $p = 0.0001$). Concentrations of BDE-209 in dust ranged from nondetect to 9.6 ug/g (Table 1 and Supporting Information). The median concentration of D-∑PBDE appears similar to other North American studies (*12*−*16*), but higher than elsewhere (*12*, *17*, *18*).

TABLE 2. Associations Between PBDE Concentrations in Breast Milk and Diet[a]

	crude β (95% CI)	adjusted β (95% CI)
all dairy (servings/day)[b]	1.4 (1.1, 1.7)	1.3 (1.0, 1.6)[c]
frozen dairy (servings/day)[b]	3.5 (1.7, 7.4)	3.2 (1.6, 6.6)[c]
dairy fat (ounces/day)[b]	3.7 (1.5, 9.0)	3.6 (1.5, 8.7)[c]
all meat (servings/day)[b]	1.7 (1.1, 2.5)	1.5 (1.0, 2.2)[d]
all fish (servings/day)[b]	1.6 (0.7, 3.5)	1.7 (0.9, 3.4)[e]

[a] The regression coefficients (β) provide the fold-increase in ∑PBDE concentrations (sum of congeners 17, 28, 47, 66, 85, 99, 100, 138, 153, 154, 183, 209) in breast milk (ng/g lipid) per exposure unit. For example, a coefficient of 1.4 indicates a 40% Increase. [b] Serving sizes for dairy varied depending on the individual foods. One ounce equals 28.4 grams. One serving of meat or fish was defined as 2−3 ounces (57−85 grams), in accordance with USDA guidelines. [c] Adjusted for meat and fish. [d] Adjusted for dairy and fish. [e] Adjusted for dairy and meat.

Breast Milk Concentrations and Questionnaire Data. Some questionnaire data were not informative due to limited variability among participants, for example, education, race, and age. Similarly, almost all participants reported sleeping on a foam mattress and having a computer at work. We found no association between ∑PBDE and body mass index or duration of lactation before samples were taken. Usage of electronic equipment was not associated with body burden. Regression results for breast milk concentrations of ∑PBDE as a function of diet are presented in Table 2; the β coefficients provide the fold-increase in breast milk PBDE concentration per unit of exposure. The strongest associations were with dairy consumption, suggesting a 3.2-fold increase (95% CI = 1.6−6.6) in ∑PBDEs per daily serving of frozen dairy products and a 3.6-fold increase (95% CI = 1.5−8.7) per daily ounce of dairy fat. Figure 1 presents the association between ∑PBDEs in breast milk and dairy fat intake ($r = 0.41$ $p = 0.005$). Total meat consumption (including beef, chicken, and pork) was associated with a 1.5-fold increase (95% CI = 1.0−2.2) in ∑PBDEs per daily serving, which was slightly stronger than for beef or chicken separately; the association with pork consumption was weak. Figure 2 presents the association between ∑PBDEs in breast milk and meat consumption ($r = 0.37$ $p = 0.01$). Total fish consumption, including self-caught fish and shellfish, was less clearly associated with body burden than total meat or dairy consumption; the association with consumption of tuna or salmon was higher than for total fish. Inclusion of personal and household characteristics in the models did not substantially affect the regression coefficients for diet. Similarly,

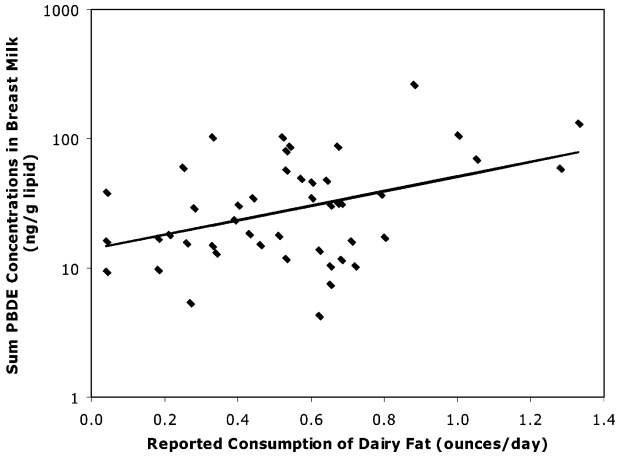

FIGURE 1. Association between ∑PBDE concentration in breast milk and consumption of dairy fat ($r = 0.41$, $p = 0.005$). ∑PBDE is the sum of congeners 17, 28, 47, 66, 85, 99, 100, 138, 153, 154, 183, 209. PBDE concentrations in breast milk (ng/g lipid) were log transformed before regressing. Dairy fat consumption was estimated from reported consumption of dairy products and industry standards for fat content. One ounce equals 28.4 grams.

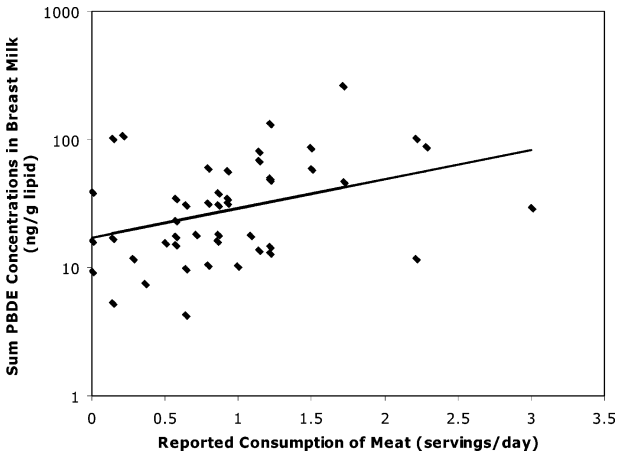

FIGURE 2. Association between ∑PBDE concentration in breast milk and consumption of meat ($r = 0.37$, $p = 0.01$). ∑PBDE is the sum of congeners 17, 28, 47, 66, 85, 99, 100, 138, 153, 154, 183, 209. PBDE concentrations in breast milk (ng/g lipid) were log transformed before regressing. One serving of meat was defined as 2−3 ounces (57−85 grams).

there was no confounding between food categories. One participant, a near vegan since childhood (consuming only small amounts of dairy products), had low PBDE concentrations: 9.5 ng ∑PBDE /g lipid.

Breast Milk Concentrations and House Dust. Concentrations of D-∑PBDE in breast milk and house dust were strongly and positively correlated ($r = 0.76$, $p = 0.003$); see Figure 3. The nonparametric Spearman correlation coefficient was slightly lower ($r = 0.64$, $p = 0.03$). Results were similar when house dust was expressed as mass of D-∑PBDE per area vacuumed ($r = 0.78$, $p = 0.003$). Homes were dichotomized into low or high D-∑PBDE categories based on the median dust concentration. Participants in homes with high levels of D-PBDE in dust had breast milk concentrations 2.6 times higher (95% CI = 1.2, 5.6) than those in homes with low levels. This association was not affected by age, duration of lactation, diet, or other personal characteristics when these covariates were added singly to the regression model. D-∑PBDE concentrations in dust were only weakly correlated with dietary habits, explaining the lack of confounding. Length of time between milk sampling and dust sampling had no impact on the model. The two participants who lived

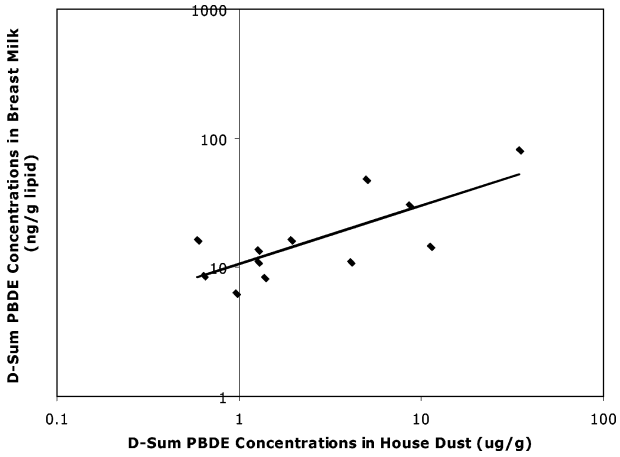

FIGURE 3. Association between D-∑PBDE concentrations in breast milk and house dust ($r = 0.76$, $p = 0.003$). D-∑PBDE is sum of congeners 47, 66, 85, 99, 100, 138, 153, 154. PBDE concentrations in breast milk (ng/g lipid) and dust (ug/g dust) were log transformed before regressing.

in the same household had very similar D-∑PBDE concentrations in breast milk (11.1 and 13.7 ng/g lipid); omitting one from the model had little effect. BDE-47 and -99 were major congeners in breast milk and dust, and they were detected in all samples. The correlation between BDE-47 in breast milk and dust ($r = 0.74$, $p = 0.006$) was similar to the association for D-∑PBDE, while the correlation for BDE-99 was less strong ($r = 0.59$, $p = 0.04$). As only one participant had detectable levels of BDE-209 in both breast milk and dust, no conclusions can be drawn for this congener.

House Dust and Household Characteristics. We found no associations between concentrations of PBDEs in house dust and characteristics of the home, including number of electronic appliances, hours the appliances were operating, or estimated wattage. Data on foam-containing products and heating systems were insufficiently detailed or variable to be informative. Neither PBDE concentrations in house dust nor mass per unit area appeared to depend on the elapsed time since the house was last vacuumed (1−37 days; median = 8 days). The latter result, combined with the lack of dependence of breast milk concentrations on the duration between sampling of milk and dust, may suggest that dust concentrations do not significantly vary on time scales relevant to exposure.

Discussion

Diet. We found statistically significant, positive associations between breast milk concentrations of PBDEs and consumption of dairy products and meat; the association with fish consumption was positive but not significant. Based on limited analysis of different foods, meats were recently estimated to constitute the dominant dietary exposure to PBDEs for U.S. adults (*10*). PBDE serum levels may tend to decrease with years of following a vegan diet (*30*). While earlier studies in Northern Europe (*22*) and Japan (*23*) found associations with fish consumption, the lesser importance of fish in the typical American diet may be one reason for the difference between our results and these previous studies. A recent study in the New York City area reported increased concentrations of PBDEs in people who caught and ate their own fish but the difference was not statistically significant (*24*).

Accurate information on an individual's diet is generally difficult to collect. We attempted to partially mitigate this problem by showing participants pictures of serving sizes. However, dietary habits change over time (including during pregnancy) and there may be substantial variation in the

PBDE concentrations within groups of foods (*10*, *31*). We used fairly broad categories of food and did not consider potential differences in PBDE concentrations caused by cooking (*32*). The strong association for dairy products may in part reflect more accurate measurement: we asked participants about the type of milk they typically drink; the fat content of milk is standardized for the industry; servings of dairy products (e.g., one 8-ounce glass of milk) may be easier to visualize than a serving of meat or fish. We did not ask questions about consumption of plant-based products, some of which may contain significant amounts of PBDEs (*8*, *9*). Accordingly, the results for diet may be affected by exposure measurement error that, if random, will tend to bias results toward the null such that our results likely underestimate the true relationship between diet and PBDEs in breast milk (*33*).

House Dust. Although limited by a small sample size, the strong association between PBDEs in breast milk and house dust (concentration or mass per unit area) indicates that the indoor home environment plays an important role in PBDE exposure. However, it is unclear from this association if dust is on the exposure pathway (e.g., via incidental ingestion) or a marker of exposure from sources in the home, i.e., PBDEs migrate into dust as they migrate into humans. Wilford et al. (*16*) recently found that PBDE dust and air concentrations were correlated in homes. Although recent exposure estimates suggest that dust ingestion may be a more important route of exposure for PBDEs than inhalation (*17*−*20*, *34*), the large uncertainties in these calculations indicate the need for additional research on exposure to PBDEs via dust and inhalation.

We know of only one other study ($n = 10$) that attempted to compare PBDE concentrations in people (measured in breast milk) with dust from their homes (*13*). The authors reported no correlation between the two (by congener or total PBDE) but provided no statistical details. Among potential explanations for the difference in results is the method of dust sampling: Sharp and Lunder analyzed dust from participants' vacuum cleaner bags while we sampled dust using a standardized method. More research comparing the different methods of sampling house dust for PBDEs is needed. For example, use of dust as an exposure measure is generally thought to be improved by taking dust loading into account (*35*, *36*).

If dust does constitute a major source of exposure, the log-normal distribution of PBDE concentrations in breast milk may be due in part to the log-normal distribution in house dust. PBDEs may enter the residential environment by volatilization from polyurethane foam or abrasion from hard plastics (*16*). A few groups have tried, with only modest success, to associate concentrations of PBDEs in house dust with characteristics of buildings and their contents (*9*, *15*−*17*). Similarly, we found no associations between household characteristics or contents and the concentrations of PBDEs in dust or breast milk. As our primary outcome of interest was PBDEs in breast milk, we counted the total number of electronic appliances and foam-containing products in each home and collected the combined dust from several rooms. Associations with PBDE concentrations in dust may be stronger on a room-by-room basis. In addition, counts of foam-containing furniture or electronic appliances may be insufficient for characterizing potential household sources of PBDEs since they ignore both mass and differences in PBDE concentrations between products due to manufacturing or age. Predicting concentrations in dust based on home characteristics remains a major challenge.

A limitation of our study was its small size, particularly the dust sampling. The milk and dust samples were analyzed by different laboratories, but both laboratories have participated in interlaboratory comparisons of PBDEs (e.g., Quasimeme). Additionally, an indoor dust standard reference material (NIST 2585) was analyzed for the full complement of PBDE congeners with results comparable to certified values (*37*). Any analytical variation between the labs is expected to introduce random, non-differential error, potentially biasing results toward the null (*33*).

As study participants were all first-time mothers, the findings of this study may not be generalizable to the overall population. Men and children may be exposed to and accumulate PBDEs differently than women. For example, children are thought to ingest more dust than adults (*21*). In addition, in the households where dust samples were collected, the women were on maternity leave, most likely spending more time at home than the average person. Given geographical differences in usage of commercial PBDE products between the U.S. and the rest of the world, for example, the U.S. dominated the use of the penta product (*2*), exposure patterns in this country may differ from elsewhere. Due to low detection rates, we could not draw conclusions about the association between BDE-209 in breast milk and dust. Additional investigations of exposure to BDE-209 are needed.

The results of our study support the hypothesis that both diet and the indoor environment play prominent roles in adult human exposure to penta-like PBDEs in the U.S. The relative importance of these two routes of exposure may depend, for any given person, on individual exposure factors such as diet and exposure to dust, as well as on the concentrations of PBDEs in the food and dust they encounter. More research is needed to determine if dust is directly on the exposure pathway or if concentrations in dust are a marker of exposure, indicative of other factors that contribute to body burden.

Acknowledgments

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Supporting Information Available

Participant characteristics; PBDE concentrations in milk and dust samples by congener; dietary and dairy fat data. This material is available free of charge via the Internet at http://pubs.acs.org.

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