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Factors Associated with Serum Polybrominated Diphenyl Ether (PBDE) Levels Among School-Age Children in the CHAMACOS Cohort

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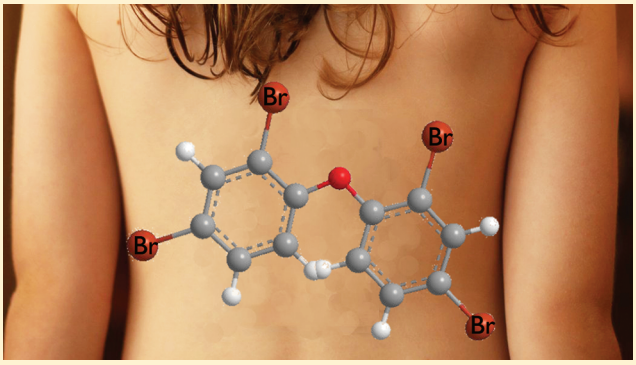
Supporting Information

ABSTRACT: Polybrominated diphenyl ethers (PBDEs) are a class of flame retardants historically used in textiles, furniture, and electronic products. Recent studies have documented widespread PBDE exposure to humans, with higher levels measured in children than adults. We analyzed 10 tri- to hepta-BDE congener levels in blood collected from 7-year old Mexican-American children living in an agriculture community in California (n = 272). The most frequently detected PBDE congeners in child serum were BDE-47, -99, -100, and -153, all of which were measured in >99% of the children. We used multiple linear regression models to examine associations between child total PBDE levels (ng/g lipid) and determinants of exposure. Factors positively associated with higher PBDE levels in the children were total PBDE levels in maternal serum during pregnancy, duration of exclusive breastfeeding, and having no safe places to play in their neighborhood. Child BMI was inversely associated with serum PBDE levels (regression p-values <0.05). Our findings confirm that exposure to the penta-BDE mixture is ongoing, and that Mexican-American children living in California may be experiencing higher PBDE exposure from their environment compared to children sampled from the general U.S. population. Additional research is needed to assess the health impacts of these exposures.



INTRODUCTION

A growing body of research has raised concern about the potential health effects of polybrominated diphenyl ethers (PBDE) flame retardant exposure in children.[1−4] Several studies have documented disruption of thyroid homeostasis, important for normal brain development, in pre- and postnatally exposed animals[5,6] and in humans.[7,8] Consistent with these studies, recent epidemiological evidence suggest adverse neurodevelopmental effects in children associated with pre- and postnatal PBDE exposure.[1−3]

Brominated flame retardants have been used in consumer products for decades. Before their phase out in 2006, the penta-PBDE flame retardant mixture was applied to polyurethane foams found in furniture, child car seats, and related products to meet federal and state flammability standards. The major constituents of the penta-PBDE mixture are BDE-47, -99, and -100. These compounds persist in the environment, and are commonly detected in house dust and human serum.[9,10]

Several studies have reported higher PBDE serum levels among children compared to adults.[11−13] Higher levels in children are likely attributable to increased exposure via breastfeeding and increased ingestion of dust due to frequent hand-to-mouth contact. For example, a Spanish study of 4-year olds (n = 244) found that breastfed infants had significantly higher concentrations of BDE-47 and -99 compared to formula fed children.[14] Further, recent studies have reported significant correlations between levels of PBDE congeners in house dust and both breast milk and human serum.[15,16]

Evidence suggests that California Latino children experience higher levels of PBDE exposure compared to Latino children of other regions.[11] Windham et al. (2010) observed significantly higher adjusted geometric mean PBDE levels in 6- to 9-year-old girls living in California compared to similarly aged girls living in Ohio.[17] In a California study of 2- to 5-year-old children (n = 100), Rose et al. (2010) reported serum PBDE levels 10- to 100-fold higher than a similarly aged population in Mexico and Europe; and 5-times higher than similarly aged children across the U.S.[18] PBDE levels in house dust from California homes are also higher than other regions.[19,20] Higher PBDE levels in California are likely due to the use of these chemicals to comply with the State's furniture flammability regulations.[20,21]

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The Center for the Health Assessment of Mothers and Children of Salinas (CHAMACOS) is a longitudinal birth cohort study investigating environmental exposures and their health effects on children residing in Monterey County, California. In this paper we examine factors associated with PBDE exposure among 7-year old, primarily Latino, children participating in the CHAMACOS cohort study (n = 272). In a previous report, we found that 7-year old Mexican-American children of the CHAMACOS cohort (n = 264) had serum PBDE levels that were on average seven times higher than concentrations measured in 5-year old Mexican children.[11] In the present investigation, we use multiple linear regression models to identify important predictors of these PBDE levels measured in the serum of all the CHAMACOS children (n = 272).

■ MATERIALS AND METHODS

Study Population. Detailed methods for the CHAMACOS study have been described elsewhere.[22,23] Briefly, pregnant women were recruited from six community clinics serving primarily low-income families, between October 1999 and October 2000. Eligible women were ≥18 years old, < 20 weeks gestation, Spanish- or English-speaking, eligible for low-income health insurance, and planning to deliver at the local public hospital. Of the 601 participants enrolled, 526 were followed to delivery of a live-born singleton infant and 353 children were followed through 7 years of age. All study activities were approved by the institutional review board at the University of California, Berkeley. Written, informed consent was obtained from the mothers, and assent was obtained from children at 7 years of age. The Centers for Disease Control and Prevention (CDC) laboratory's role was determined not to constitute engagement in human subjects research.

Study Interviews and Home Visits. Structured interviews with mothers were conducted in English or Spanish by bilingual study staff during pregnancy and when the children were 6 months, and 1-, 2-, 3.5-, 5-, and 7-year(s) old. Information collected included demographics, breastfeeding history, television use (hours viewed per day), maternal smoking (current and during pregnancy), child having lived outside of the United States (yes/no), and neighborhood quality (safety, noise, presence of trash, etc). Height and weight were measured to compute child BMI. Home inspections were conducted at all contacts through 5 years of age (i.e., at 6, 24, and 48 months). Information obtained included quality of housekeeping, resident density, housing disrepair (presence or absence of mold, rotting wood, peeling paint, plumbing leaks), exterior building condition, and number of rooms with wall-to-wall carpeting.[24] Information on housing characteristics at age 7 relied on questionnaire data rather than visual inspection.

At the child's 5-year study visit, we also administered a modified Block food frequency questionnaire (FFQ) to each participant.[25] Information on child-specific dietary intake (e.g., food types, serving size) was used to estimate daily intake of fruit, vegetables, dairy, meat, fish, and total fat using a validated conversion matrix.[25] Because the FFQ was completed at age five, it was not directly concordant with dietary intake information at age 7. We administered an abbreviated version of the FFQ at 7 years, including questions about intake of soda and high calorie processed and fast foods.

Maternal and Child PBDE Serum Sample Collection. Maternal PBDE serum collection methods have been described previously.[11,26,27] Briefly, PBDE congeners were measured in 321 serum samples collected from women during pregnancy (at ∼26 weeks gestation), and 116 samples collected just before delivery. Of the latter, 95 women provided only a delivery serum sample. Twenty-one women provided samples at both time points, resulting in samples from 416 individuals. Total PBDE, BDE-47, BDE-99, BDE-100, and BDE-153 levels (n = 21) were highly correlated between the two sampling time points (Pearson r = 0.98−0.99, p-values <0.001). We used PBDE concentrations from ∼26 weeks gestation, when the majority of measurements were available. For women with only delivery samples available, we back-extrapolated these levels to 26 weeks using linear regression model parameters derived from the 21 matched pairs.[27] Of the maternal samples available, 216 had matching 7-year child serum PBDE levels.

When the children (n = 339) reached 7 years of age, between March 2007 and November 2008, we collected blood from them by venipuncture. Samples were immediately processed and stored at −80 °C until they were shipped on dry ice for analysis.

Serum PBDE Analysis. The CDC (Atlanta, GA) measured PBDEs in serum samples using gas chromatography isotope dilution high resolution mass spectrometry (GC-IDHRMS).[28] Samples were analyzed for 10 tri- to heptabrominated congeners BDE-17, -28, -47, -66, -85, -99, -100, -53, -154, and -183.

PBDE serum concentrations are expressed on a blood lipid basis. Total lipids were determined based on the measurement of triglyceride and total cholesterol in serum using standard enzymatic methods (Roche Chemicals, Indianapolis, IN).[29] For the present analysis, we excluded twins (n = 6), and children lacking 7-year PBDE measurements (n = 59). In addition, two children were missing 7-year lipid measurements, resulting in a final sample size of 272.

The limits of detection (LOD) for PBDE analyses ranged between 0.3 and 5.6 ng/g lipids for all congeners, except for BDE-47, which ranged from 0.4 to 8.0 ng/g lipids using on average 1.7 g (range: 0.2−2.0 g) of serum for analysis. Quality control samples (n = 3) and method blanks (n = 3) were included in each run. Data below the LOD but for which a signal was detected were coded with the concentration obtained. Data below the LOD for which no signal was detected were imputed from a log-normal probability distribution.[30] Several PBDE congeners had low detection frequencies (e.g., BDE-17). These values were extremely low relative to the more frequently detected congeners (e.g., BDE-47) and did not significantly contribute to the total sum of PBDEs. To evaluate total PBDE levels, we summed all 10 congeners by weight.

Child BMI Z-Score. We calculated z-scores for children's body mass index (BMI) (kg/m^2) using sex-specific BMI-for-age percentile data issued in 2000 by the CDC.[31]

Data Analysis. Statistical analysis included computation of descriptive statistics for individual PBDE congeners as well as a measure of total exposure (sum of ten congeners). All values were $\log_{10}$-transformed. We used Pearson correlations, ANOVA and linear regression to assess bivariate associations between child total PBDE levels, BDE-47 and BDE-153 and potential exposure determinants identified a priori, including prenatal maternal PBDE serum levels (log10-transformed), duration of exclusive breastfeeding, reported lack of safe places to play in the neighborhood (as a marker for time spent indoors), child having lived outside the U.S. more than 1 month since birth (yes vs no), number of rooms in the home with wall-to-wall

Table 1. Serum PBDE Levels (ng/g lipid) among 7-Year-Old Children in the CHAMACOS Cohort (n = 272)[a]

	LOD range	DF (%)	GM (95% CI)	min	25th	50th	75th	90th	max
BDE-17	0.3−5.6	35	<LOD	<LOD	<LOD	<LOD	0.9	1.6	11.8
BDE-28	0.3−5.6	88	2.0 (1.8, 2.2)	<LOD	1.3	2.1	3.5	5.6	23.2
BDE-47	0.4−8.0	100	47.5 (43.0, 52.5)	1.9	27.9	46.9	77.5	136	768
BDE-66	0.3−5.6	37	<LOD	<LOD	<LOD	<LOD	0.9	1.5	6.8
BDE-85	0.3−5.6	64	0.9 (0.9, 1.0)	<LOD	<LOD	0.9	1.6	2.6	14.6
BDE-99	0.3−5.6	99.6	11.2 (10.0, 12.4)	<LOD	6.2	10.7	19.3	36.8	218
BDE-100	0.3−5.6	100	10.8 (9.8, 11.9)	0.8	6.0	10.9	16.9	32.8	144
BDE-153	0.3−5.6	100	12.2 (11.1, 13.4)	0.9	6.8	11.5	20.0	32.4	263
BDE-154	0.3−5.6	78	1.2 (1.1, 1.4)	<LOD	0.7	1.1	1.9	3.5	19.0
BDE-183	0.3−5.6	20	<LOD	<LOD	<LOD	<LOD	<LOD	0.9	17.0
sum of 4 PBDE congeners[b]		100	84.8 (77.2, 93.1)	5.8	50.2	84.6	131	229	1300
sum of 10 PBDE congeners[c]		100	91.3 (83.2, 100)	6.9	54.7	91.8	142	242	1390

[a]Abbreviations: DF = detection frequency; LOD = limit of detection. [b]Summed concentrations of 4 PBDE congeners with DFs > 99%: BDE-47; BDE-99; BDE-100; BDE-153. [c]Summed concentrations of 10 PBDE congeners: BDE-17; BDE-28; BDE-47; BDE-66; BDE-85; BDE-99; BDE-100; BDE-153; BDE-154; BDE-183.

carpeting, child's sex, housing quality (e.g., disrepair), and household (e.g., resident density) and demographic characteristics (socio-economic status (SES), maternal smoking, etc.). As we did not record time child spent indoors, we hypothesize that lack of safe places to play outside serves as a proxy for this variable. We used the income-to-poverty ratio to characterize SES continuously.[32] Ratios below 1.00 indicate that the income for the respective family is below the U.S poverty threshold, while a ratio of 1.00 or greater indicates income above the poverty level.[32] We included all variables associated with PBDE levels ($p < 0.20$) in multivariable linear regression models. The Cuzick test was used to test trends across categorical variables with three or more ordered groups (i.e., safe places to play in the neighborhood: "Not a problem"; "Some problem"; "Big problem").[33]

We then constructed three multivariable linear regression models with $\log_{10}$-transformed total PBDE levels, BDE-47 and BDE-153 as dependent variables and all potential exposure determinants identified above as independent variables. For comparison, multivariable linear regression models were also created with concentrations of BDE-99 and -100. (Because associations differed somewhat for BDE-153, we present model results for this congener separately (see below)). In order to control for potential selection bias due to exclusion from analyses and/or loss to follow-up, we ran all final models with and without weights determined as the inverse probability of inclusion in our sample.[34,35] Weights were determined using multiple logistic regression.[34,35] Findings were similar for all of the models described, and therefore results from the unweighted regression analyses are presented.

We also calculated the ratio of BDE-153 to BDE-47. Differences in this ratio can provide insight about penta-BDE exposure patterns because BDE-47 is the primary congener of the penta mixture while BDE-153 may have other sources (i.e., the octa-BDE mixture, used primarily in electronic products). We evaluated the BDE-153 to BDE-47 ratio to determine whether higher ratios were associated with children having more electronic consumer products (DVDs, computers, printers, etc.) in their homes. Because BDE-153 has a longer biological half-life in humans and is relatively enriched in breast milk,[36−38] we also analyzed potential associations between breastfeeding duration and the ratio of BDE-153 to BDE-47. All analyses were conducted using Stata software, version 11 (StataCorp LP, College Station, TX).

RESULTS

Demographic and Household Characteristics. Children were on average 7.0 ± 0.1 years old; 53% were female and 47% were male. At 4 months of age, 28% of the children were exclusively breastfed. Many children (53%) were overweight at 7 years (mean child BMI z-score = 1.1 ± 1.0). The majority of CHAMACOS children consumed >1 serving of meat per day (63%), and <5 servings of fish per month (79%). Average fat intake in this population was 67 g per day at 5 years of age.

At the time of the child's birth, their mothers were on average 25.9 ± 5.1 years of age. Ninety-six percent of the women were of Mexican descent. Eighty-six percent were born in Mexico or another Latin American country, with 46% having lived in the U.S. ≤5 years (mean = 8.2 ± 7.7 years). The CHAMACOS population is economically disadvantaged (i.e., 99.3% within 200% of U.S. poverty threshold), as a result there may not be enough variability in SES to observe potential differences between child's SES and other variables.

Homes of most children had wall-to-wall carpeting in the child's bedroom (83%) and living room (61%) at 7 years of age. On average children had 2.6 (±1.1) televisions in the home and watched 1.9 (±1.1) hours per day. Home resident density was relatively high with 64% having ≥1 person(s) per room (average 1.3 ± 0.7 people per room).

PBDE Levels in Blood. Table 1 presents the geometric mean and distribution of 10 individual and summed PBDE congeners, and the sum of the four congeners detected in >99% of samples (BDE-47, -99, -100, and -153). Total PBDE levels ranged from 6.9 to 1390 ng/g lipid. The congener with the highest serum concentration was BDE-47 (geometric mean (GM) = 47.5 ng/g lipid), followed by BDE-153 (GM = 12.2 ng/g lipid), BDE-99 (GM = 11.2 ng/g lipid) and BDE-100 (GM = 10.8 ng/g lipid). BDE-47 was the dominant congener representing an average of 57% of the total PBDE molar concentration (see Supporting Information (SI) Table S1). The detection frequencies (DFs) of the six less frequently detected congeners (BDE-17, -28, -66, -85, -54, and -83) ranged from 20% to 88% (Table 1). Within the child serum samples, the four most frequently detected PBDE congeners were strongly correlated with each other (Pearson r = 0.63−0.97, p-values < 0.001) (Table 2).

Levels of PBDE congeners in boys and girls were similar, except for BDE-153, which was significantly higher in boys (Table 3 and SI Tables S2 and S3).

Table 2. Pearson Correlations of Individual Congener and Total 7-Year Child PBDE levels (n = 272) and Maternal Prenatal Levels (n = 216) (ng/g lipid)

	Child PBDEs				
	BDE-47	BDE-99	BDE-100	BDE-153	total
Child PBDEs					
BDE-47	1				
BDE-99	0.97[c]	1			
BDE-100	0.94[c]	0.94[c]	1		
BDE-153	0.63[c]	0.66[c]	0.77[c]	1	
total PBDEs[a]	0.98[c]	0.97[c]	0.97[c]	0.77[c]	1
Maternal PBDEs					
BDE-47	0.18[b]	0.14[b]	0.17[b]	0.18[b]	0.18[b]
BDE-99	0.18[b]	0.14[b]	0.17[b]	0.18[b]	0.17[b]
BDE-100	0.17[b]	0.14[b]	0.17[b]	0.17[b]	0.17[b]
BDE-153	0.25[c]	0.22[b]	0.25[c]	0.30[c]	0.26[c]
total PBDEs[a]	0.26[c]	0.24[c]	0.27[c]	0.26[c]	0.26[c]

[a]Summed concentrations of 10 PBDE congeners: BDE-17; BDE-28; BDE-47; BDE-66; BDE-85; BDE-99; BDE-100; BDE-153; BDE-154; BDE-183. [b]p-value <0.05. [c]p-value <0.01.

Prenatal maternal and child BDE-47, -99, and -100 serum concentrations were weakly correlated (Pearson r = 0.14−0.18 (p < 0.05)). The correlation between maternal and child BDE-153 serum concentrations, however, was stronger (Pearson r = 0.30; p < 0.001)(Table 2). It is possible that the stronger correlation observed for BDE-153 may be related to the longer biological half-life of BDE-153 compared to the other congeners.[38] Linear regression results showed a significant positive relationship between the 216 paired prenatal maternal and child total PBDE levels (β = 0.23; p < 0.001).

Table 3 presents maternal and child demographic and potential exposure factors by level of child's total PBDE, BDE-47, and -153 concentrations (n = 272). Univariate results for two congeners from the penta-BDE mixture, BDE-99 and -100, are presented in the SI.

Association of Maternal Characteristics with Child PBDE Levels. We found no associations between child PBDE levels and maternal smoking or SES.

Association of Child-Specific Exposure Determinants with Child PBDE Levels. Levels of total PBDE and BDE-47 were significantly lower among children that were no longer exclusively breastfed at 4 months, and who lived outside the U.S. for at least one month since birth. Negative correlations between total PBDE and BDE-153 levels and child BMI z-scores were statistically significant (Pearson r = −0.12 and −0.37, respectively; p < 0.05). Total PBDE, BDE-47 and BDE-153 levels were significantly higher among children for whom having no safe places to play in the neighborhood was reported as a "big problem" (Cuzick trend test p < 0.01)[33] (Table 3).

Housing Characteristics. We observed a weak but significant positive association between the number of rooms with wall-to-wall carpeting (at the 5-year home visit) and serum BDE-47 levels (Pearson r = 0.13; p < 0.05). We found no other significant relationships between PBDE exposure and housing characteristics or quality (not shown).

Diet. We found no significant relationships between child PBDE levels at 7 years and daily servings of diary or meat, total daily fat intake or fish consumption at five years of age (data not shown).

Multiple Linear Regression Models. Table 4a and 4b present results from multiple linear regression models for total PBDE and BDE-47 levels, and BDE-153 levels, respectively, as a function of potential exposure factors in children. Total child PBDE and BDE-47 levels increased 20.1% (95% CI = 7.9, 32.3) and 20.2% (8.1, 32.2) (p = 0.001), respectively, for each 10-fold increase in maternal prenatal PBDE level after adjustment for child BMI z-score, child having lived outside the U.S., and SES. The child's duration of exclusive breastfeeding was associated with a 4.8% (95% CI = 0.2, 9.6) and 5.5% (0.5, 10.7) (p < 0.05) increase in total PBDE and BDE-47 serum level per month of exclusive breastfeeding. In addition, maternal report of no safe places to play in the neighborhood as "some problem" or "a big problem" was associated with a 41.8% (95% CI = 12.5, 78.6) and 43.8% (12.0, 84.8) (p < 0.01) increase in total PBDE and BDE-47 levels, respectively. We also observed a significant inverse association between total PBDE levels and child's BMI z-score (p = 0.02) (Table 4a). Similarly, results from the multivariable models using BDE-99 and -100 as the dependent variables indicate that maternal prenatal PBDE levels, duration of exclusive breastfeeding and lack of safe places to play were significant determinants for these congeners in children. A potential shortcoming of these models is the lack of maternal breast milk PBDE concentration data for the CHAMACOS cohort. Without these data, it is difficult to confirm that the statistical models accurately reflect the physiological relationship between breastfeeding duration and child PBDE levels. Future studies should examine the contribution of breastfeeding to long-term PBDE body-burden in children. Multiple regression results for BDE-99 and -100 are presented in the SI.

Table 4b presents results from the multiple regression analyses of BDE-153 levels and exposure factors in children. Based on bivariate findings, the final BDE-153 regression model included different covariates than the total PBDE and BDE-47 models, such as child's sex (boy = 1); and did not include number of rooms with wall-to-wall carpeting in the home. Child BDE-153 levels increased 25.3% (95% CI = 14.5, 36.1; p < 0.001) for each 10-fold increase in maternal prenatal PBDE levels after adjustment for child's sex and BMI z-score, child having lived outside the U.S., and SES. Duration of exclusive breastfeeding was associated with a 3.3% (95% CI = −0.7, 7.4; p = 0.11) increase in total PBDE serum level per month of exclusive breast feeding, albeit not significantly. The lack of safe places to play in the neighborhood was associated with a 33.8% (95% CI = 9.0, 64.2; (p < 0.01)) increase in BDE-153 levels. We observed a significant association between serum BDE-153 and child's sex (boy) as well as a strong inverse association with BMI z-score (Table 4b).

Ratio of BDE-153 to BDE-47. The median (95% CI) BDE-153/BDE-47 ratio in our sample was 0.23 (0.22, 0.26), with 6% (n = 15) of children having a ratio >1. We found no associations between the BDE-153/BDE-47 ratio and number of consumer products in the home or time spent watching television. In addition, we found no significant associations between the BDE-153/BDE-47 ratio and breast feeding duration.

Comparison of Prenatal Maternal and Child PBDE Levels. Figure 1 presents median concentrations of total PBDEs and four PBDE congeners in paired CHAMACOS maternal prenatal and 7-year child serum samples (n = 216). Child median total PBDEs, BDE-47, -99, -100, and -153 levels were significantly higher than maternal levels (quantile regression p < 0.001).

Table 3. Maternal, Child and Housing Characteristics in Relation to PBDE Levels (ng/g lipid) in Children at 7 Years (n = 272)

	n (%)	Total PBDEs GM (95% CI) or correlation[a,b]	BDE-47 GM (95% CI) or correlation[a]	BDE-153 GM (95% CI) or correlation[a]
Mother Characteristics				
age at child's birth				
≤25 years	150 (55)	88.0 (77.7, 99.7)	45.3 (39.5, 51.8)	12.2 (10.8, 13.8)
>25 years	122 (45)	95.5 (83.0, 109.8)	50.5 (43.5, 58.6)	12.3 (10.7, 14.1)
maternal education at pregnancy				
≤ 6 years school	120 (44)	79.9 (70.6, 90.3)	41.3 (36.1, 47.3)	10.8 (9.6, 12.2)
7−12 years school	93 (34)	96.7 (81.9, 114.1)	50.5 (42.0, 60.7)	13.1 (11.2, 15.2)
high school grad +	59 (22)	109.5[d] (87.5, 137.0)	57.7[d] (45.9, 72.4)	14.1 (11.0, 18.0)
any maternal smoking during entire pregnancy				
no	258 (95)	90.4 (82.2, 99.5)	46.9 (42.3, 52.0)	12.2 (11.1, 13.4)
yes	14 (5)	108.6 (73.2, 161.2)	61.2 (40.3, 93.0)	12.1 (8.5, 17.4)
any maternal smoking since last interview				
no	257 (95)	90.9 (82.6, 100.0)	47.3 (42.6, 52.4)	12.2 (11.1, 13.4)
yes	15 (5)	99.0 (66.3, 147.9)	52.7 (34.1, 81.4)	12.2 (8.5, 17.7)
monthly family income				
≤$1500 per month	183 (68)	94.9 (84.2, 106.9)	49.6 (43.6, 56.4)	12.4 (11.0, 14.0)
>$1500 per month	87 (32)	83.6 (72.4, 96.5)	43.5 (37.2, 50.9)	11.5 (10.0, 13.2)
income-to-poverty ratio (mean ± SD=0.86 ± 0.4)				
Pearson r	270 (99)	−0.05	−0.04	−0.06
Child Characteristics				
age (mean ± SD=7.0 ± 0.1 years)				
Pearson r	272 (100)	−0.08	−0.07	−0.06
sex				
boy	127 (47)	94.6 (82.3, 108.7)	48.1 (41.5, 55.9)	13.7[d] (12.0, 15.7)
girl	145 (53)	88.5 (78.1, 100.3)	47.0 (41.1, 53.9)	11.0 (9.4, 12.5)
BMI z-score (mean ± SD = 1.1 ± 1.0)				
Pearson r	271 (100)	−0.12[d]	−0.05	−0.37[e]
duration of exclusive breastfeeding (mean ± SD=2.2 ± 2.4 months)				
Pearson r	265 (97)	0.17[e]	0.18[e]	0.10
child exclusively breastfed at 4 months				
no	195 (72)	83.9 (75.6, 93.0)	42.8 (38.2, 47.9)	12.0 (10.7, 13.3)
yes	74 (28)	112.4[e] (92.5, 136.7)	61.4[e] (50.1, 75.3)	12.9 (10.7, 15.5)
child lived outside of U.S. since birth[c]				
no	193 (71)	100.0[e] (164.0, 203.2)	54.5[e] (46.3, 59.4)	12.9 (11.6, 14.5)
yes	79 (29)	73.0 (63.2, 84.3)	37.3 (32.1, 43.4)	10.6 (8.9, 12.5)
no safe places to play in neighborhood				
no problem	189 (70)	82.1 (74.1, 90.9)	42.4 (37.9, 47.5)	11.3 (10.2, 12.5)
some problem	54 (20)	105.9 (87.9, 127.6)	55.7 (46.2, 67.1)	13.6 (10.7, 17.2)
big problem	28 (10)	138.4[e] (89.8, 213.3)	74.2[e] (46.9, 117.5)	16.1[d] (11.2, 23.2)
Housing Characteristics				
wall-to-wall carpet in room where child sleeps				
no	47 (17)	100.1 (81.1, 123.6)	50.5 (40.1, 63.7)	14.6 (12.1, 17.7)
yes	225 (83)	89.5 (80.8, 99.3)	46.9 (42.0, 52.5)	11.8 (10.6, 13.0)
wall-to-wall carpet in living room				
no	105 (39)	85.0 (73.3, 98.7)	43.9 (37.4, 51.6)	12.0 (10.3, 13.9)
yes	167 (61)	95.4 (84.8, 107.5)	50.0 (44.0, 57.0)	12.4 (11.0, 13.9)
no. rooms with wall-to-wall carpet (at 5 years) (mean ± SD=1.6 ± 1.0)				
Pearson r	249 (92)	0.12	0.13[d]	0.05
number of TVs in current home (Mean ± SD=2.6 ± 1.1)				
Pearson r	271 (100)	−0.02	−0.03	−0.02
hours per weekday child watches TV (mean ± SD=1.9 ± 1.1)				
Pearson r	271 (100)	0.0001	0.003	−0.007

[a]Geometric means and 95th confidence interval except when Pearson coefficient (r) is presented. [b]Summed concentrations of 10 PBDE congeners: BDE-17, -28, -47, -66, -85, -99, -100, -153, -154, -183. [c]Child lived more than one month outside of the U.S. since birth. [d]ANOVA or Pearson's correlation p-value <0.05. [e]p-value <0.01.

Table 4a. Results from Multiple Regression Analyses of Log-Transformed Total PBDE and BDE-47 Levels (ng/g lipid) and Exposure Factors in Children (n = 192)[a]

	Total PBDEs[b]		BDE-47	
	% change (95th CI)	p-value	% change (95th CI)	p-value
Maternal prenatal total PBDE serum level (ng/g lipid)	20.1 (7.9, 32.3)	0.001		
Maternal prenatal BDE-47 serum level (ng/g lipid)			20.2 (8.1, 32.2)	0.001
Duration of child's exclusive breastfeeding (months)	4.8 (0.2, 9.6)	0.04	5.5 (0.5, 10.7)	0.03
Lack of safe places to play in neighborhood	41.8 (12.5, 78.6)	0.003	43.8 (12.0, 84.8)	0.005
No. rooms with wall-to-wall carpeting in home[c]	7.8 (−2.9, 19.7)	0.16	9.7 (−2.0, 22.8)	0.11
Child lived outside U.S. since birth (yes)	−20.1 (−36.4, 0.3)	0.05	−20.6 (−38.0, 1.6)	0.07
Child's BMI z-score	−12.1 (−21.1, −2.2)	0.02	−8.7 (−18.7, 2.5)	0.12
SES (income-to-poverty ratio)	−14.0 (−33.3, 10.9)	0.24	−14.7 (−35.2, 12.3)	0.26

[a]Total PBDE model R-squared = 0.19; BDE-47 model R-squared = 0.18. [b]Summed concentrations of 10 PBDE congeners: BDE-17, -28, -47, -66, -85, -99, -100, -153, -154, -183. [c]Carpeting information obtained from home visit when child was 5 years old.

Table 4b. Results from Multiple Regression Analyses of Log-Transformed BDE-153 Levels (ng/g lipid) and Exposure Factors in Children (n = 208).[a]

	BDE-153	
	% change (95th CI)	p-value
Maternal prenatal BDE-153 serum level (ng/g lipid)	25.3 (14.5, 36.1)	<0.0001
Child's sex (boy)	27.0 (5.5, 52.8)	0.01
Duration of child's exclusive breastfeeding (months)	3.3 (−0.7, 7.4)	0.11
Lack of safe places to play in neighborhood	33.8 (9.0, 64.2)	0.006
Child lived outside U.S. since birth (yes)	−16.1 (−31.2, 2.4)	0.08
Child's BMI z-score	−23.9 (−30.7, −16.5)	<0.0001
SES (income-to-poverty ratio)	−14.1 (−31.6, 7.9)	0.19

[a]BDE-153 model R-squared = 0.30.

DISCUSSION

We found that higher levels of primarily penta-BDE congeners (i.e., BDE-47, -99, and -100) in the 7-year old children were positively associated with higher PBDE levels measured in their mothers when they were pregnant, duration of exclusive breastfeeding, and the lack of safe play areas in the child's neighborhood. The number of rooms with wall-to-wall carpeting was marginally associated with higher PBDE levels in the children, as was lower social economic status. Higher child BMI z-scores were inversely associated with child PBDE levels. In bivariate analyses, having lived outside the U.S. was associated with lower PBDE levels; this association remained negative but was nonsignificant in multiple linear regression models. The PBDE serum levels in this low-income population of children were also significantly higher than their mothers[11,27] and are consistent with other studies showing higher levels in children compared to adults.[12,13] These California children had substantially higher PBDE serum levels than other U.S. populations,[17,18] with median BDE-47 levels (47 ng/g lipid) almost two times greater than those of U.S. children 12 to 19 year old (27 ng/g lipid) represented in NHANES (n = 252).[10]

The strong associations of maternal prenatal PBDE levels and duration of exclusive breastfeeding with higher levels in the 7 year-old children suggest that early prenatal and infant exposure to these persistent, bioaccumulative compounds stay in the body during childhood. The association of exclusive

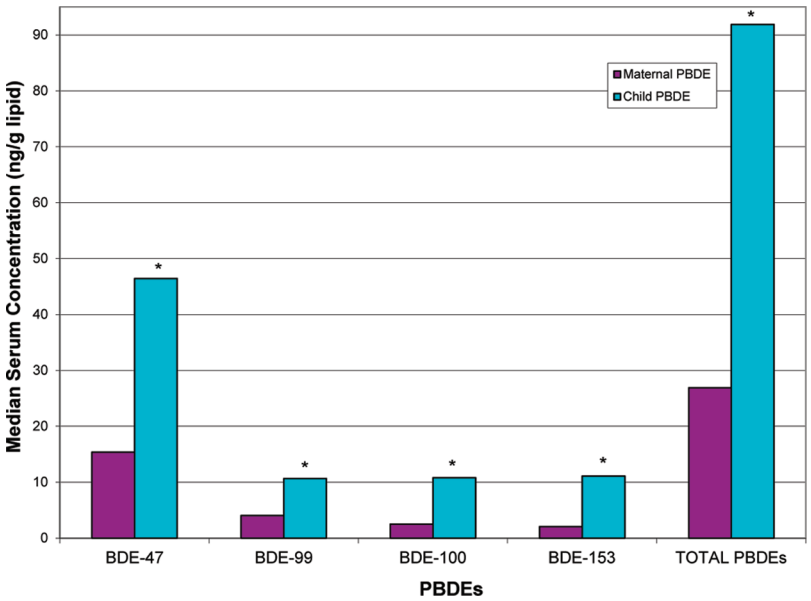

Figure 1. Median levels of total PBDEs and four PBDE congeners in paired CHAMACOS maternal prenatal and 7-year child serum samples (n = 216). * indicated quantile regression p < 0.001.

breastfeeding with the children's PBDE levels is consistent with the chemical and physical characteristics of PBDEs, and other persistent organic pollutants, that results in transfers into breast milk;[36] for example, Schecter et al. (2010) showed that PBDE levels in maternal blood were correlated with breast milk levels, indicating that prenatal maternal blood levels are a marker for later infant exposures during breastfeeding.[37] It is possible these associations are due to the fact that the children and mothers lived in the same homes and shared the same environments. In future analyses, we will utilize physiologically based pharmacokinetic modeling to determine whether predicted prenatal maternal contributions to PBDE levels in school age children are consistent with the statistical models used herein.

In contrast to findings from other U.S. studies,[18,39] none of the dietary intake variables, including daily servings of dairy or meat, total daily fat intake, or fish consumption were associated with child PBDE levels. As diet information was attained via questionnaire, and not using food basket or duplicate diet measurements, exposure misclassification in the diet data used for this study may have led to a bias toward the null.

In general, we found that SES and housing conditions in the home were not associated with child PBDE levels, although we have previously reported significant housing quality problems in this population.[24] Wall-to-wall carpeting, which may use foam padding with flame retardant, was not a significant contributor to child exposures. Overall, SES was fairly uniform in the population, and housing problems were also common; thus, these factors may not have varied enough to ascertain their association with exposures. One of the strongest predictors of exposure was having no safe place to play outside. This population is primarily low-income, and neighborhoods are often perceived as unsafe and there may also be limited access to parks or other outdoor play areas. The lack of safe places to play may be an indicator for more time spent indoors, where most PBDE exposures occur via house dust.[15,40] Because the CHAMACOS cohort is comprised of low income, predominantly Mexican-American immigrant families living in an agricultural community, this cohort of 7-year-old children is unique, and factors predicting PBDE serum levels may not be generalizable to all children living in the U.S. However, the levels in our population agree with other studies showing higher exposure in California children.[17,18]

The inverse association of BMI and PBDE levels in the children is consistent with findings reported by Windham et al (2010)[17] in 6−8 year-old-girls but inconsistent with studies examining persistent organohalogens in adults, where higher BMI is positively associated with higher levels of the contaminants.[27,41] In the CHAMACOS mothers, organohalogen levels were inversely associated with weight gain during pregnancy.[27,41] This pattern is consistent with a hypothesis proposed by Glynn et al. (2003), which suggests that recent weight gain, acting as an additional reservoir for storage of organohalogen intake, dilutes serum concentrations, resulting in lower levels relative to weight gain.[42] At 7 years, the CHAMACOS children are rapidly growing, and it is likely that children with higher BMI have recently gained weight. Thus, our finding is consistent with Glynn et al.'s (2003) proposed pharmacodynamics of organohalogens in serum relative to weight gain (i.e., increase in adipose tissue).[42]

The median BDE-153/BDE-47 ratio of 0.23 (std error = 0.02) that we observed is slightly higher than that found in the commercial penta-BDE mixture (≈ 0.15)[43] but similar to ratios in indoor U.S. dust samples, which range from ∼0.1−0.3.[44−46] The higher BDE-153/BDE-47 ratio in the CHAMACOS children we studied may be due to additional exposures from BDE-153 derived from products treated with the octa-BDE mixture, which was used mainly in electronic products.

We examined potential exposure risk factors (e.g., housing quality) cross-sectionally and cumulatively from earlier ages through age 7 with similar results, indicating the final models were the best descriptive summary of PBDE exposure risk factors at school age. Measuring PBDEs in duplicate diet samples and house dust would have provided a more direct measure of the relative importance of different exposure pathways at age 7. Complete information on the type and condition of all furniture and electrical devices present in the homes was lacking. Additionally, several of the exposure risk factors we examined, such as carpeting, may have been important predictors of the child exposure at younger ages when hand-to-mouth behaviors and crawling on floors were more common. Finally, at the time these measurements were completed, CDC did not have approved methods to measure the higher brominated nona-BDE or deca-BDE (i.e., BDE-209) congeners. Future studies without these limitations are needed to fully assess the sources and pathways of PBDE exposure among California residents.

In summary, we found that CHAMACOS school-age children's PBDE serum levels were higher relative to their mothers and other children in the United States and that early life exposure may contribute to body burdens at school age. The inverse association of PBDE serum levels and BMI suggest complex pharmacodynamics that should be considered when interpreting or modeling flame retardant and other persistent organic pollutant exposure in children. Finally, our finding that the lack of safe places to play was associated with significantly higher flame retardant levels in the children underscore the need for additional research to quantify key residential exposure routes and pathways to school-age children in support of future exposure intervention strategies. Additional research is needed to assess the health impacts of these exposures.

■ ASSOCIATED CONTENT

Supporting Information

A summary of results from bivariate and multiple regression analyses of log-transformed BDE-99 and -100 congener levels and determinants of exposure. LOWESS scatterplots of paired maternal prenatal and 7-year child PBDE levels, summary of child serum lipid levels, beta coefficients from PBDE multiple regression model, child PBDE congener levels by sex, and detailed analytical laboratory methods. This material is available free of charge via the Internet at http://pubs.acs.org.

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Notes

The authors declare no competing financial interest.

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