Polybrominated Diphenyl Ethers, Polychlorinated Biphenyls, and 2,2-Bis(4-chlorophenyl)-1,1-dichloroethene in 7- and 9-Year-Old Children and Their Mothers in the Center for the Health Assessment of Mothers and Children of Salinas Cohort

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S *Supporting Information*

ABSTRACT: We report longitudinal serum concentrations of select persistent organic pollutants (POPs) in children at ages 7 and 9 years and in their mothers prenatally and again when the children were 9 years old. The participating families were enrolled in the Center for the Health Assessment of Mothers and Children of Salinas (CHAMACOS), a longitudinal birth cohort study of low-income Hispanic families residing in the Salinas Valley, California. We observed decreasing concentrations in the mothers with year of serum collection (2009 vs 2011) for six out of seven polybrominated diphenyl ether (PBDE) congeners and for 2,2′,4,4′,5-pentachlorobiphenyl (CB-99; $p < 0.05$). The 9-year-old children had similarly decreasing serum concentrations of all seven PBDE congeners, CB-99, and 2,2′,3,4,4′,5′- and 2,3,3′,4,4′,6-hexachlorobiphenyl (CB-138/158) with year of serum collection (2009 vs 2011; $p < 0.05$). In mixed effect models accounting for weight gain

as the children aged from 7 to 9 years, we observed an annual decrease (−8.3% to −13.4%) in tri- to hexaBDE concentrations ($p < 0.001$), except for 2,2′,3,4,4′-tetrabromodiphenyl ether (BDE-85) and 2,2′,4,4′,5,5′-hexabromodiphenyl ether (BDE-153). The concentrations of these congeners were not associated with time of serum collection and instead showed an −0.9% to −2.6% decrease per kilogram of weight gain during the study period ($p < 0.05$). In the case of tetra- to heptachlorobiphenyls, we observed −0.5% to −0.7% decrease in serum concentration per kilogram of weight gain ($p < 0.05$) and −3.0% to −3.7% decrease in serum concentration per year of aging ($p < 0.05$), except for 2,3′,4,4′,5-pentachlorobiphenyl (CB-118) and 2,2′,4,4′,5,5′-hexachlorobiphenyl (CB-153), which were not associated with time of serum draw. 2,2-Bis(4-chlorophenyl)-1,1-dichloroethene ($p,p′$-DDE) decreased −2.4%/kg of weight gain between the two sampling points ($p < 0.001$). These findings suggest that as children grow, dilution in a larger body size plays an important role in explaining reductions in body burden in the case of traditional POPs such as PCBs and $p,p′$-DDE. By contrast, in the case of PBDEs, reductions are likely explained by reduction in exposure, as illustrated by decreased concentrations in more recent years, possibly amplified by presumed shorter biological half-life than other POPs.
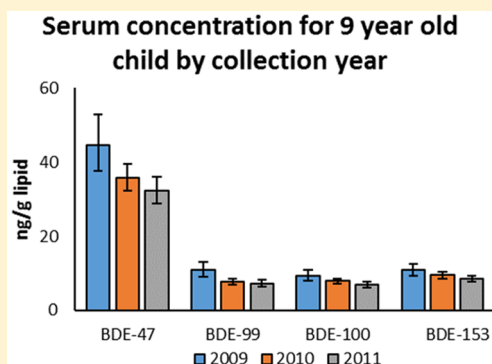
INTRODUCTION

Three commercial formulations of polybrominated diphenyl ethers (PBDEs), identified by their average bromine content, have been in commercial production since the 1970s. The ability of PBDEs to reduce flammability as well as stricter fire safety standards worldwide and increasing use of flammable materials in furniture were all factors that lead to increased use of flame-retardant chemicals, including PBDEs, over the last decades. PBDEs with 4−6 bromines on the diphenyl ether backbone (e.g., technical PentaBDE) were often used as flame retardants in polyurethane foam, commonly present in upholstered furniture and as padding under wall-to-wall

carpets.[1] PBDEs with 8−10 bromines (e.g., technical Octa- and DecaBDE) have been used in hard plastics such as casings of electrical appliances, TVs, and computers.[1] Tetra- through hexaBDE congeners are present in common food items[2] and almost universally detected in human biomonitoring studies in the United States.[3−5] The Penta- and OctaBDE technical mixtures were voluntarily withdrawn from the U.S. market in

Table 1. Percentage Change between Ages 7 and 9 by Year and Change per Kilogram of Weight Gain for Selected PBDEs, PCBs, and p,p'-DDE[a]

analyte	N	change per year			change per kilogram of weight gain		
		% change	95% CI[b]	p-value	% change	95% CI	p-value
		Polybrominated Diphenyl Ethers (PBDEs)					
BDE-28	228	−9.6	−14.4, −4.4	<0.001	−0.4	−1.2, 0.5	0.44
BDE-47	228	−8.3	−13.0, −3.4	<0.001	−0.4	−1.3, 0.5	0.36
BDE-85	228	−4.2	−9.3, 1.1	0.12	−0.9	−1.8, 0.0	0.04
BDE-99	228	−11.6	−16.7, −6.2	<0.001	−0.4	−1.4, 0.6	0.43
BDE-100	228	−10	−14.0, −5.9	<0.001	−0.7	−1.5, 0.2	0.11
BDE-153	228	2.3	−1.6, 6.4	0.25	−2.6	−3.3, −1.9	<0.001
BDE-154	228	−13.4	−17.7, −8.9	<0.001	−0.8	−1.7, 0.0	0.06
		Polychlorinated Biphenyls (PCBs)					
PCB-74	210	−3.7	−6.7, −0.7	0.02	−0.5	−0.9, −0.1	0.02
PCB-99	224	−3.2	−6.1, −0.1	0.04	−0.5	−0.9, −0.1	0.01
PCB-118	209	−2.1	−5.3, 1.2	0.21	−0.7	−1.1, −0.3	<0.001
PCB-138/158	224	−3	−6.0, 0.0	0.05	−0.6	−0.9, −0.2	<0.001
PCB-153	224	−2.9	−5.9, 0.2	0.07	−0.6	−1.0, −0.2	<0.001
PCB-180	224	−3.2	−6.1, −0.1	0.04	−0.6	−0.9, −0.2	0.01
		Persistent Pesticides					
p,p'- DDE	228	−1.7	−6.7, 3.6	0.53	−2.4	−3.4, −1.4	<0.001

[a]For participants with repeated measurements only. [b]95% confidence interval.

2004;[6] while commercial use of DecaBDE ended in 2013,[7] following concerns about their safety to humans due to their potential hormonal activity,[8,9] neurodevelopmental toxicity,[10,11] and decreased infant birth weight.[12]

However, the withdrawal of PBDEs from the market did not lead to an immediate reduction of exposure since historically produced PBDEs are still present in indoor environments, including older furniture and carpets and as residues in house dust. It is also possible that recycling of polyurethane may lead to the reintroduction of PBDEs into new products in our homes. Like exposure to lead, children have higher PBDE exposure than adults.[13,14] This is most likely due to higher frequency of hand-to-mouth behavior in children compared to adults, resulting in higher nondietary ingestion of PBDEs from indoor dust[15] containing PBDEs.[16,17] Breastfeeding during infancy most likely is not a major contributing factor, due to the fact that the highest median 2,2′,4,4′-tetraBDE (BDE-47) concentration were observed at >4−6 years of age in a cohort of Texas children, that is, several years after cessation of breastfeeding. Concentrations then declined until >10−13 years of age, a fact likely explained by dilution with increasing body size and elimination.[18]

The objectives of the current study were to (1) expand the current limited information on the association of PBDE serum concentration and age in young children; (2) compare patterns of PBDE exposure with those of other persistent organic pollutants (POPs) such as polychlorinated biphenyls (PCBs) and 2,2-bis(4-chlorophenyl)-1,1-dichloroethene (p,p'-DDE), which are often present in indoor dust at lower concentrations than PBDEs; (3) explore whether dilution in a larger body size and/or elimination from the body are plausible explanations for decreasing levels of PBDEs and other POPs as the children age; (4) evaluate whether PBDE and other POPs are decreasing in concentration since the phase-out of PentaBDE in 2004; and (5) compare POP concentrations in the longitudinal birth cohort study of low-income Hispanic children and their mothers in California with concentrations in a national sample.[3,19]

■ MATERIALS AND METHODS

Study Population. Details of the CHAMACOS recruitment and follow-up have been published elsewhere.[20,21] Briefly, pregnant women were recruited from six clinics serving low-income families between October 1999 and October 2000 in Monterey County, CA. Inclusion criteria included over 18 years old, less than 20 weeks gestation, eligible for low-income health insurance, planning to deliver at the local public hospital, and Spanish- or English-speaking. Of the participants, 527 (88%) were followed to delivery of a live-born, singleton infant; 294 (56%) of the children provided blood samples at 7 years of age and 264 (50%) at 9 years. Between January 2010 and October 2011, blood samples were collected from additional 9-year-old children (n = 290) and their mothers (n = 294) to supplement the original CHAMACOS cohort. The later recruited subjects were demographically comparable to the original cohort (Table S1). Subjects recruited later in the survey are hereafter referred to as CHAM2, while the original subjects are referred to as CHAM1. Serum samples available for measurements of POPs were collected from the mothers prenatally at 26 weeks gestation (n = 313) and 9 years after birth of the index child (n = 474); samples from the children were collected at 7 years of age (n = 275) and at 9 years (n = 554). The serum collection procedure has been described previously.[22−24] Samples were immediately processed and stored at <−70 °C except when in transit on dry ice to the Centers for Disease Control and Prevention (CDC) in Atlanta, GA, for analysis. All study activities were approved by the institutional review boards at the University of California, Berkeley, and the CDC; written informed consent was obtained from mothers and oral assent from the child starting at age 7. No personally identifiable information on the research subjects was made available to CDC researchers.

Structured Interviews. Interviews with the mothers were conducted in English or Spanish during pregnancy and when the children were 6 months and 1, 2, 3.5, 5, 7, and 9 years old. Collected information included maternal education, age, country of birth, and years in the United States prior to giving
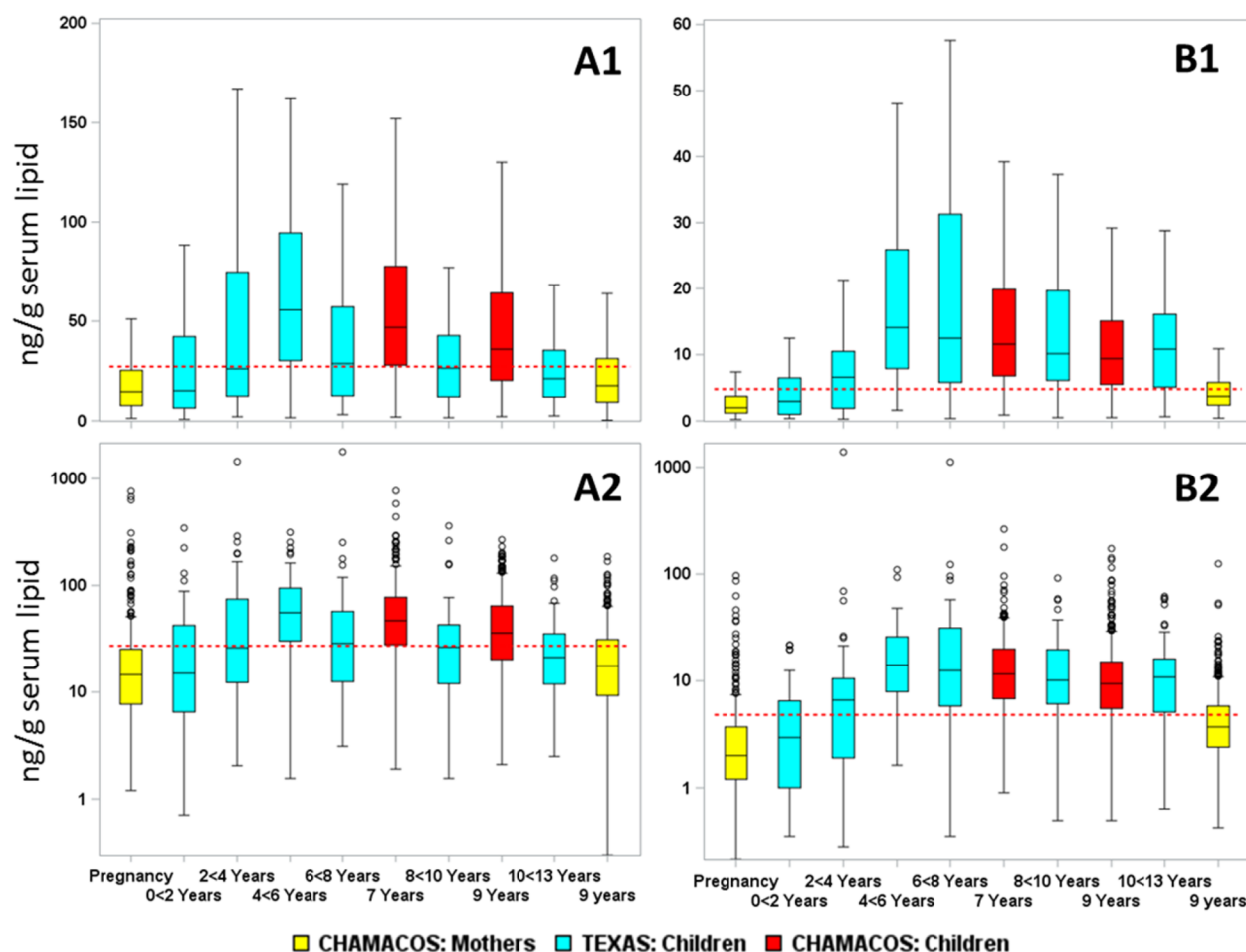
Figure 1. Concentrations (nanograms per gram of serum lipid) of (A) BDE-47 and (B) BDE-153, on a (1) linear and (2) log10 scale. Values for children (7 and 9 years) are given in red; those for their mothers, at week 26 of pregnancy and at age 9 of their children, are shown in yellow. Median and interquartile range (IQR) concentration (box) and outlier range (bars) are shown [outlier range is (25th percentile − 1.5 IQR) and (75th percentile + 1.5 IQR)]. A cohort of Texas children[14] stratified by two-year age groups between <2 years and >10 years are included as a comparison; these values are shown in blue. The red horizontal dashed line represents the National Health and Nutrition Examination Study (NHANES) estimate for age category 12−19 years for survey years 2003/04.[3,19]

birth; family income, parity, and breastfeeding duration and sex of index child (Table S1).[25] Height and weight of mothers and children were measured to compute body mass index.

Measurements of Persistent Organic Pollutants. The analytical methodology has been published elsewhere.[26] Two grams of serum was used for the measurements of PBDEs, PCBs, and p,p′-DDE, and the samples were assigned to 24 sample batches for analysis; each analytical batch contained three quality control and three blank samples composed of bovine serum (Gibco Inc., Grand Island, NY) diluted 1:40 with water; this dilution was made to reduce any target analytes in the blank serum to a level less than 1 order of magnitude lower than the limit of detection (LOD). Bovine serum was tested prior to dilution for native presence of POPs by analyzing volumes from 0.5 to 4 mL and plotting any detectable POPs versus sample size used. A slope of zero for the resulting graphs shows that any detected POPs reflect method background and were not present at a concentration above the LOD in the bovine serum. No POPs were detected in the bovine serum prior to dilution. Measurements of serum POPs were made via gas chromatography/isotope dilution−high-resolution mass spectrometry.[26,27] Analytical data were corrected by subtracting the median blank value. LOD was determined as the higher of

(1) 3 times the standard deviation of the amount present in blanks or (2) the instrumental LOD, defined as the injected amount known to produce a signal/noise ratio >10. Concentrations below the LOD were substituted with LOD/$\sqrt{2}$. The target analytes measured and reported are given in Table 1.

Statistical Methods. Statistical evaluation was limited to PBDE and PCB congeners having greater than 60% detection overall in the study (except maternal 26 weeks) and p,p′-DDE having 100% detection. 2,2′,3,4,4′,5′,6-Heptabromodiphenyl ether (BDE-183) and decabromodiphenyl ether (BDE-209) were also measured, but their detection frequencies were <25% (BDE-183) and <10% (BDE-209), so they were not included in the current data set.

Selected percentiles for the whole data set by sample category (children at ages 7 and 9 and mothers at 26 week of pregnancy and at child age 9) were calculated by the PROC UNIVARIATE procedure (Table S2). The longitudinal design of the study was not considered; differences between sample categories were assessed by Wilcoxon test using the PROC NPAR1WAY procedure.

We assessed the changes in serum concentration (nanograms per gram of lipid) by collection year of child samples at ages 7

Table 2. Child Age 9 Serum Concentration by Collection Year[a]

		year 2009			year 2010			year 2011			ANOVA level of significance		
compd	N	GM[b]	95% CI	N	GM	95% CI	N	GM	95% CI	F	2009 vs 2010	2009 vs 2011	2010 vs 2011
					Polybrominated Diphenyl Ethers (PBDEs)								
BDE-28	67	1.71	1.47−1.99	187	1.45	1.32−1.59	216	1.37	1.47−1.99	**0.06**	**0.06**	**<0.05**	0.45
BDE-47	67	44.7	37.7−53	187	35.8	32.3−39.6	215	32.2	37.7−53	**<0.01**	**<0.05**	**<0.01**	0.17
BDE-85	67	0.92	0.79−1.06	186	0.75	0.69−0.82	213	0.73	0.79−1.06	**<0.05**	**<0.05**	**<0.01**	>0.5
BDE-99	68	10.9	9.02−13.1	187	7.59	6.79−8.5	215	7.2	9.02−13.1	**<0.01**	**<0.01**	**<0.001**	>0.5
BDE-100	65	9.26	7.94−10.8	184	7.86	7.16−8.63	213	6.91	7.94−10.8	**<0.01**	0.07	**<0.01**	0.07
BDE-153	68	10.8	9.33−12.5	181	9.46	8.64−10.4	213	8.47	9.33−12.5	**<0.05**	0.13	**<0.01**	0.10
BDE-154	63	1.02	0.88−1.18	173	0.79	0.72−0.86	202	0.77	0.88−1.18	**<0.01**	**<0.01**	**<0.01**	>0.5
					Polychlorinated Biphenyls (PCBs)								
CB-74	60	0.68	0.61−0.75	185	0.72	0.68−0.77	214	0.7	0.61−0.75	0.49	0.24	>0.5	>0.5
CB-99	68	0.89	0.8−0.99	186	0.87	0.81−0.92	214	0.66	0.8−0.99	**<0.001**	>0.5	**<0.001**	**<0.001**
CB-118	66	0.99	0.9−1.08	186	1.05	0.99−1.11	212	0.9	0.9−1.08	**<0.01**	0.26	0.1	**<0.001**
CB-138/158	67	1.55	1.38−1.74	186	1.53	1.43−1.65	210	1.34	1.38−1.74	**<0.05**	>0.5	**<0.05**	**<0.05**
CB-153	65	1.91	1.69−2.16	187	1.89	1.75−2.04	213	1.66	1.69−2.16	**<0.05**	>0.5	0.06	**<0.05**
CB-180	65	1.15	1−1.32	188	1.18	1.09−1.29	212	1.01	1−1.32	**<0.05**	>0.5	0.13	**<0.05**
					Persistent Pesticides								
p,p′-DDE	66	136	115−163	182	146	131−162	208	128	115−163	0.27	>0.5	>0.5	0.11

[a]The ANOVA p-value comparing collection years is given, as well as the total number of available samples by year. [b]GM, geometric mean.

Table 3. Serum Concentration of Mothers When Their Children Reached 9 Years of Age, by Collection Year[a]

		year 2009			year 2010			year 2011			ANOVA level of significance		
compd	N	GM	95% CI	N	GM	95% CI	N	GM	95% CI	F	2009 vs 2010	2009 vs 2011	2010 vs 2011
					Polybrominated Diphenyl Ethers (Pbdes)								
BDE-28	91	1.4	1.19−1.64	248	1.22	1.11−1.34	214	1.14	1.04−1.25	0.1	0.16	**<0.05**	0.31
BDE-47	90	21.9	18.1−26.6	248	17.6	15.7−19.8	214	16.1	14.5−17.9	**<0.05**	0.06	**<0.01**	0.26
BDE-85	90	0.51	0.44−0.6	245	0.41	0.37−0.45	213	0.42	0.38−0.45	**<0.05**	**<0.05**	**<0.05**	>0.5
BDE-99	90	4.12	3.33−5.09	248	3.28	2.89−3.73	215	3.03	2.69−3.41	**<0.05**	0.07	**<0.05**	0.37
BDE-100	90	4.27	3.59−5.08	247	3.33	3−3.69	211	3.07	2.79−3.38	**<0.01**	**<0.05**	**<0.01**	0.27
BDE-153	91	4.1	3.53−4.78	242	3.67	3.34−4.02	211	3.78	3.47−4.12	0.46	0.21	0.35	>0.5
BDE-154	91	0.52	0.46−0.59	245	0.43	0.4−0.46	211	0.42	0.39−0.45	**<0.05**	**<0.05**	**<0.01**	>0.5
					Polychlorinated Biphenyls (PCBs)								
CB-74	88	0.77	0.68−0.88	234	0.81	0.75−0.87	214	0.75	0.7−0.8	0.37	>0.5	>0.5	0.16
CB-99	90	1.01	0.89−1.15	239	0.96	0.89−1.03	212	0.85	0.79−0.91	**<0.05**	0.49	**<0.05**	**<0.05**
CB-118	91	1.52	1.33−1.74	222	1.36	1.25−1.47	213	1.31	1.22−1.42	0.17	0.15	0.06	>0.5
CB-138/158	91	2.47	2.12−2.87	245	2.14	1.95−2.34	211	2.18	2−2.37	0.26	0.11	0.16	>0.5
CB-153	91	3.44	2.95−4.01	243	3.06	2.8−3.35	211	2.89	2.65−3.14	0.14	0.2	0.05	0.35
CB-180	91	2.4	2.06−2.8	245	2.27	2.07−2.48	211	2.14	1.96−2.33	0.37	>0.5	0.19	0.35
					Persistent Pesticides								
p,p′-DDE	88	290	227−370	231	291	251−337	204	249	217−286	0.27	>0.5	0.28	0.13

[a]The ANOVA p-value comparing collection years is given, as well as the total number of available samples by year.

and 9, as well as mother's concentration sampled at child age 9, by using analysis of variance (ANOVA) models (Tables 2 and 3 and Table S3). To run the model, we first eliminated the outliers, which were defined as above 75th percentile plus 1.5 times the interquartile range (IQR) or below the 25th percentile minus 1.5 times IQR.[28]

Changes in child serum concentration from ages 7 to 9 years of age and their association with increasing body weight were examined by mixed effects models to control for non-independence of repeat samples. Statistical analysis was performed with SAS 9.3 or SAS Enterprise Guide 5.1 (SAS Institute Inc., Cary, NC) and SPSS (IBM, Armonk, NY)

■ RESULTS

Our data on seven PBDE congeners, six PCB congeners, and p,p′-DDE provide useful information to evaluate the association of PBDE serum concentrations and age in young children. All of these POPs had detection frequencies ≥60% in children 7 and 9 years old and in their mothers 9 years after the children's birth. Medians, interquartile range (IQR), and detection frequency are presented in Table S2 and Figure 1. The median concentration for the whole population (CHAM1 and CHAM2) of children decreased significantly for all measured POPs between 7 and 9 years [Table S2 and Figure 1 (BDE-47 and BDE-153) and Figure S1 (CB-153 and p,p′-DDE)]. The
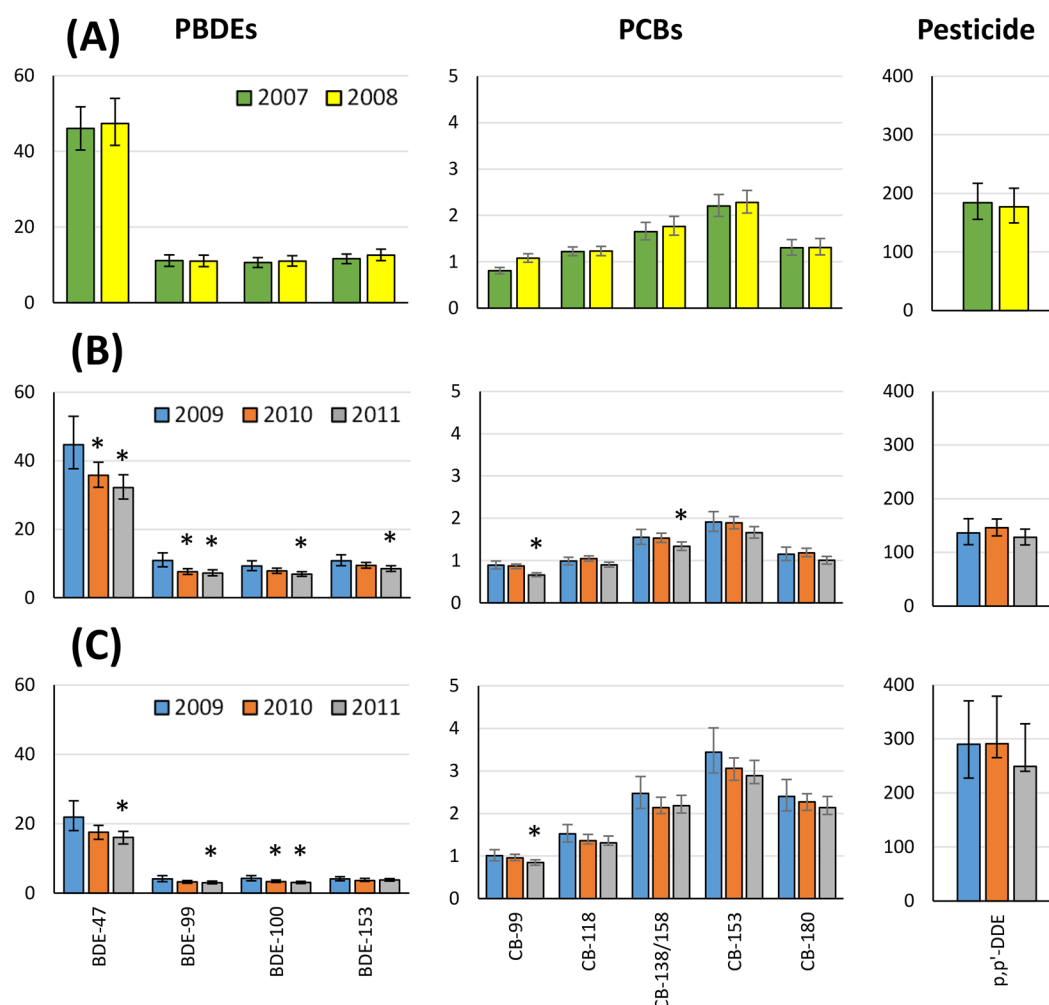
Figure 2. Geometric mean concentration (nanograms per gram of serum lipid) of samples from (A) child age 7, (B) child age 9, and (C) mother at child age 9 by sample collection year. Bars indicate 95% confidence interval. Significant differences compared to first year of sample collection are indicated with asterisks (ANOVA).

percentage reduction from 7 to 9 years was −23% for BDE-47, −25% for 2,2′,4,4′,5-pentabromodiphenyl ether (BDE-99), −19% for BDE-153, −18% for CB-153, and −19% for $p,p′$-DDE. On the other hand, PBDE concentrations were higher (except for BDE-99) while PCB and $p,p′$-DDE serum concentrations tended to be lower (−18.2% to −44.7%) in the mothers 9 years after the child was born compared to concentrations during pregnancy (Table S2 and Figure 1).

Tables 2 and 3 and Table S3 present geometric mean concentrations by collection year for the 7 and 9 year olds and their mothers to compare concentration patterns among these POPs (e.g., PBDE and PCBs) and to evaluate whether PBDE concentrations are declining since the phase-out of PentaBDE in 2004. We observed significantly decreasing concentrations for PBDEs and CB-99 and CB-138/158 by collection year between 2009 and 2011 among the 9-year-old children (Figure 2, Table 2) Similarly, concentrations of PBDEs (except BDE-153) decreased for maternal samples between 2009 and 2011, but among the organochlorines only CB-99 decreased significantly during the same time period (Table 3). By contrast, the concentrations of the 7-year-old children did not differ over the shorter collection period (2007−2008; Figure 2, Table S3).

To assess whether dilution in a larger body size or elimination from the body may explain the decreasing concentrations of PBDEs and other POPs as children age, we present in Table 1 the percent change in congener concentration per kilogram increase in child weight between the blood collections at 7 and 9 years. We observed between −0.4% and −2.6% change for BDE congeners, with significant percent decreases per weight increase for BDE-85 and BDE-153. Similarly, there were significant decreases in congener concentration per kilogram increase in child weight for all PCBs (ranging from −0.5% to −0.7%) and for $p,p′$-DDE (−2.4%). In mixed effect models accounting for weight gain as the children aged from 7 to 9 years, we observed an annual decrease (−8.3% to −13.4%) in tri- to hexaBDEs concentration ($p < 0.001$), except for BDE-85 and BDE-153. These two congeners were not associated with time of serum collection and instead decreased by −0.9% to −2.6%/kg of weight gain during the study period ($p < 0.05$, Table 1). In the case of tetra- to heptachlorobiphenyls, we observed a decrease in serum concentration of −0.5% to −0.7%/kg of weight gain ($p < 0.05$) and a −3.0% to −3.7% decrease in serum concentration per year of aging ($p < 0.05$), except for CB-118 and CB-153, which were not associated with date of serum draw. $p,p′$-DDE

decreased −2.4%/kg of weight gain between the two sampling points ($p < 0.001$, Table 1).

■ DISCUSSION

In this study, we report serum concentrations of select POPs in school-age children and their mothers residing in the Salinas Valley, California. These data expand the current limited information on the association of PBDE serum concentrations and age in young children.

We compared the POP levels in the CHAMACOS children and their mothers with levels reported in a group of Texas children[14] and in a national sample.[3,19] POP concentrations in the CHAMACOS children were generally comparable to a convenience sampling of Texas children, with slightly higher concentration of BDE-47 and similar median concentration of BDE-153 (Figure 1). The BDE-47 concentration in the mothers sampled at the child's age 9 were likewise comparable with the oldest group of Texas children (10 to <13 years), while the BDE-153 concentration is about half that of the Texas children (Figure 1). However, in the Texas children we see a sharp increase in concentration from the youngest age group (<2 years) to an apex concentration at 4 to <6 years and 6 to <8 years for BDE-47 and BDE-153, respectively, which then declined at ages >8 years.[14] Sjodin et al.[14] speculated that this increase up to age 4−6 years was due to the hand-to-mouth behavior of young children exposing them to residential dust, which is known to contain high concentration of PBDEs[16,17,29,30] but lower concentrations of other POPs investigated herein.[31] Subsequent declining concentration may be caused by elimination of PBDEs and/or dilution in a growing body as the child ages and hand-to-mouth behaviors have ended or decreased. The current longitudinal study provides an opportunity to investigate these possibilities to a greater extent than what was possible in the 2014 publication of Sjodin et al.[14]

Among the CHAMACOS children, we observe a similar decline in serum concentration of organochlorine compounds as we see for the PBDEs, although these other POPs (e.g., PCBs and p,p'-DDE) are often present in indoor dust at lower concentrations than PBDEs. For example, CB-153 decreased 18% and p,p'-DDE decreased 19% when the 7- and 9-year-old samplings are compared, while the PBDEs decreased between 22% (BDE-85) and 32% (2,4,4′-tribromodiphenyl ether [BDE-28]). At first this appears inconsistent with the findings of Sjodin et al.[14] However, in the case of traditional organochlorine POPs like PCBs and p,p'-DDE, we observed a significant decreased concentration with weight gain from age 7 to 9 as well as with year of serum draw, which is consistent with a dilution of POPs in a larger body mass as the child ages and grows (Table 1). On the other hand, for the PBDEs we do not see a significant decline with kilogram of weight increase (except for BDE-85 and BDE-153), but we do see on average about a 3-fold larger decrease with each year of aging compared to the traditional POPs (Table 1). The absence of significant associations with weight gain for most PBDE congeners is most likely explained by two factors: a reduction in exposure when hand-to-mouth behavior decreases at an older age and also, potentially, a relatively shorter biological half-life of PBDEs compared with PCBs. The biological half-life of the PBDEs present in commercial pentaBDE (tri- to hexaBDEs) has not yet been measured in humans. However, in electronics recycling workers in Sweden,[32] BDE-183 and BDE-209 have been reported to have biological half-lives of 3 months and 2

weeks, respectively. It can thus be inferred that the lower brominated PBDE congeners may have shorter biological half-lives than PCBs, the half-lives of which are measured in years to decades.[33] The hypothesis that BDE-153 has a longer biological half-life compared with other PBDEs is supported by the observation that in the Texas children[14] the median ratio of BDE-153 to BDE-47 in the youngest age group (<2 years) was 0.18, while the age group >6−8 years had a median ratio of 0.50. This ratio did not change in a consistent direction for children over 8 years of age among the Texas children.[14] In our cohort of CHAMACOS children, the median ratio of BDE-153 to BDE-47 was 0.23 at age 7 and 0.25 at age 9 and significantly different (Wilcoxon two-sided, $p = 0.002$), although, the difference was small in absolute terms. A longer biological half-life of BDE-153 and BDE-85 than that of BDE-183 and BDE-209 could potentially explain why a significant association between weight gain and percent reduction of PBDE serum concentration was observed for these congeners and not others (Table 1). Thus, a longer biological half-life of BDE-153 than other PBDEs would be consistent with the increase in BDE-153 to BDE-47 ratio with age, as observed in the Texas and CHAMACOS cohorts, and with the significant association between declining serum concentration and weight gain from age 7 to 9 in the CHAMACOS children.

The length of the sample collection, extending from March 2007 to November 2008 for collection of the samples from 7-year-old children and from April 2009 to October 2011 from the children and mothers when the children were 9 years of age, provided us with the opportunity to examine changes in concentration by collection year. In the 9-year-old children, we observed significantly decreasing concentrations by collection year for all PBDE congeners ranging from BDE-28 (−20%) to BDE-85 (−35%) and similarly decreasing concentrations of PCBs ranging from CB-118 (−9%) to CB-99 (−26%). This finding is consistent with decreasing exposures during the 19 months of collection of samples from 9-year-old children. No significant change was observed during the shorter collection of samples from 7-year-old children. In the case of the mothers, we did observe decreasing concentrations of all PBDEs except BDE-153, ranging from BDE-85 (−18%) to 2,2′,4,4′,6-pentabromodiphenyl ether (BDE-100) (−28%), but among the PCBs only CB-99 decreased significantly (−9%) during the same time period. These findings are consistent with an overall decline in exposure to POPs in the CHAMACOS cohort, which is consistent with the 2004 phase-out of commercial penta- and octaBDE.

This study has several limitations. It is possible that the later-recruited subjects in the cohort may have had different exposures and/or uncontrolled confounders that may have biased these findings (Table S1). For example, over half of the study population was overweight and/or obese, limiting the generalizability of our findings. However, we observed a significant correlation between decreasing concentrations of PCBs and p,p'-DDE and weight gain. This finding supports the conclusion that for these contaminants, with biological half-lives measured in years, dilution in a growing body size may be the main factor resulting in lower concentrations at older ages. The absence of such a correlation for most PBDEs suggests that lower exposure to PBDEs may be due to reduced dust ingestion from hand-to-mouth behavior at an older age and possibly lower exposures due to reduced use of these chemicals. Strengths of the investigation includes participation of a vulnerable, socioeconomically underserved population with

potentially high exposure to p,p′-DDE due to later phase-out of DDT in Mexico and a longitudinal study design.

Of interest, the geometric mean concentration of BDE-47 in the CHAMACOS 9-year-old children (44.7 ng/g of lipid) is almost twice the geometric mean in the National Health and Nutrition Examination Survey (NHANES) 2003/04 for the 12−19 year age category (28.2 ng/g lipid).[3,19] On the other hand, the geometric mean of BDE-47 in the CHAMACOS mothers when their children were 9 years of age (21.9 ng/g of lipid) was similar to the 2003−2004 NHANES estimate for women over the age of 12 (19.6 ng/g of lipid).[3,19] Looking at the 95th percentile of BDE-47 for 12−19 year olds in 2003/04 NHANES (174 ng/g of lipid),[3,19] we observe that in the case of 7- and 9-year-old children we have 7.3% and 1.6%, respectively, of the children exceeding this level. The fact that the percentage of children in CHAMACOS with higher levels than the 95th percentile of NHANES BDE-47 concentration is not largely different from 5% indicates that we do not have unusual exposures in this socioeconomically underserved population. This observation is reassuring. However, it also suggests that PBDE concentrations in humans will remain elevated in the United States in comparison to other parts of the world for years to come, in large part because of the historical widespread use of these flame retardants in our country.[1]

■ ASSOCIATED CONTENT

Ⓢ Supporting Information

The Supporting Information is available free of charge on the ACS Publications website at DOI: 10.1021/acs.est.7b05460.

> Three tables listing demographic and descriptive statistics and child age 7 geometric mean concentrations by collection year; one figure showing concentrations of CB-153 and p,p′-DDE in the CHAMACOS cohort compared with that of Texas children[14] by age category (PDF)

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Notes

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention (CDC). Use of trade names is for identification only and does not imply endorsement by the CDC, the Public Health Service, or the U.S. Department of Health and Human Services.

The authors declare no competing financial interest.

■ ACKNOWLEDGMENTS

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