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Elevated House Dust and Serum Concentrations of PBDEs in California: Unintended Consequences of Furniture Flammability Standards?

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Studies show higher house dust and body burden levels of PBDE flame retardants in North America than Europe; but little is known about exposure variation within North America, where California's furniture flammability standard affects PBDE use. We compared dust samples from 49 homes in two California communities with 120 Massachusetts homes and with other published studies. Dust concentrations [median (range) ng/g] in California homes of BDE-47, -99, and -100 were 2700 (112-107 000), 3800 (102-170 000), and 684 (<MRL-30 900), respectively, and were 4−10 times higher than previously reported in North America. Maximum concentrations were the highest ever reported in indoor dust. We then investigated whether human serum PBDE levels were also higher in California compared to other North American regions by analyzing the 2003−2004 National Health and Nutrition Examination Survey (NHANES), the only data set available with serum from a representative sample of the U.S. population ($n = 2040$). California residence was significantly associated with nearly 2-fold higher ΣPBDE serum levels [least square geometric mean (LSGM) ng/g lipid, 73.0 vs 38.5 ($p = 0.002$)]. Elevated PBDE exposures in California may result from the state's furniture flammability standards; our results suggest the need for further research in a larger representative sample.

Introduction

Polybrominated diphenyl ethers (PBDEs) are widely used as flame retardants in upholstered furniture and electronics and are released in indoor environments via volatilization or as dust particles (*1*). PBDEs are ubiquitous globally and have been detected in human blood and tissue, marine mammals, sediments, and virtually any matrix taken from anywhere on the planet (*2*). Concentrations in environmental and human samples vary internationally, with much higher serum, breast milk, and house dust levels reported in the U.S. compared with Europe (*3, 4*). Regional variation within the U.S. may result from more stringent furniture flammability standards in California than in other states; however, this possibility has not been evaluated.

Three major PBDE commercial mixtures have been commonly used in consumer products: deca-BDE, octa-BDE, and penta-BDE (*5*). Penta-BDE has been most often mixed into polyurethane foam (PUF) used in furniture, while octa- and deca-BDE are used in electronics and other plastic products (*6*). Penta-BDE is typically about 3−5% by weight in treated foam, and is easily liberated into dust because it is not chemically bound to the foam product. Penta-BDE has been used almost exclusively in the U.S (*6*) and mostly in furniture for sale in California in order to comply with Technical Bulletin 117 (TB117), the state's 1975 performance-based furniture flammability standard (*5, 7*). Regional differences may be somewhat lessened, however, because some TB-117-compliant products are distributed nationwide (*8*), and not all furniture sold in California has complied with the standard (*9*).

Although the effect of California's furniture flammability standard on regional variations in PBDE exposures has not been systematically examined, a few studies have reported serum levels in California, and these results may be compared with serum PBDE levels measured in the National Health and Nutrition Examination Survey (NHANES), a cross-sectional sample representative of the U.S. population. Serum PBDE levels in one California family exceeded the 95th percentile for NHANES (*10, 11*). Separate studies in two groups of California immigrant women—Laotian and Mexican—found serum levels similar to or lower than those of U.S. women in NHANES (*11*−*13*).

House dust has been identified as the primary route of exposure for PBDEs (*1, 3*). An EPA review concluded that 82% of exposure is from incidental ingestion and dermal contact with house dust (*3*). Wu and colleagues (*14*) reported that breast milk PBDE levels in 11 women were correlated with their house dust concentrations. While diet may also contribute to human exposure (*14*), it does not appear to be the major route either in the general population (*15*) or in high fish-consuming subpopulations (*16*).

Concern about human exposure stems from animal studies that consistently show thyroid disruption and adverse neurodevelopmental and reproductive effects following in utero exposures of PBDEs (*17, 18*). In addition, structural and mechanistic similarities with PCBs (*18, 19*), for which extensive human data demonstrate effects on neurodevelopment and other end points (*20, 21*), suggest the relevance of these end points to PBDEs. To date, there are few human health studies of PBDEs, and results are limited and inconsistent (*22, 23*).

While questions remain about the health effects, the toxicology database has been strong enough that use of penta-BDEs and octa-BDEs was banned by the European Union in 2003; and in 2004, U.S. manufacturers discontinued production of these compounds (*24*). Currently, 11 states, including California, have banned the use of penta-BDE and octa-BDE; however, the ubiquity of these chemicals combined with the slow replacement time for products previously manufactured with penta- and octa-BDE suggests that a long-term, substantial exposure reservoir will remain for some time despite PBDE phase-outs (*25*).

In order to investigate whether California flammability standards may result in higher exposures there, we used two distinct data sets to compare penta-BDE concentrations in house dust and in serum in California with the rest of the

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U.S. First, we compared new data on house dust concentrations in 49 California homes with concentrations we previously reported for 120 Massachusetts homes (*4*) and several studies reporting house dust PBDE concentrations in various North American regions. Second, we used data from NHANES to compare serum PBDE levels in California participants and those from other U.S. locations. Currently, no single data set allows for both dust and serum PBDE exposure comparisons on such a large geographic scale. Therefore, we report these findings together because the serum data are most relevant to potential human health outcomes, and the dust data highlight sources of exposure that may contribute to any observed regional differences in serum levels. To our knowledge these regional comparisons provide the first assessment of how California's unique furniture flammability standard may affect regional differences in PBDE exposures within the United States.

Experimental Section

PBDE Dust Measurements. As part of the California Household Exposure Study, dust samples were collected from 49 nonsmoking homes in two Northern California communities: Richmond and Bolinas. The research protocol was approved by Brown University's Institutional Review Board. Richmond is a predominately low income, urban, minority community near transportation corridors and numerous industries including two oil refineries. Bolinas is a rural community north of San Francisco. Sampling protocols and analytical methods have been described in detail elsewhere (*4*). Briefly, dust samples were collected using a Eureka Mighty-Mite vacuum cleaner attached to a Teflon crevice tool, modified to collect dust into a cellulose thimble (Whatman Inc., Clifton, NJ). Samples were collected by vacuuming the surface of rugs, upholstery, wood floors, windowsills, ceiling fans, and furniture in the primary living areas of the home. BDE-47,-99, and -100 were analyzed using gas chromatography/mass spectrometry in selected ion monitoring mode with a method reporting limit (MRL) of 42.0 ng/g. Additional information on analytical methods and QA/QC is provided in the Supporting Information. Three BDE-100 concentrations below the MRL were replaced by one-half the MRL. Differences in dust concentrations between Richmond and Bolinas were assessed using the Wilcoxon rank-sum test.

PBDE Serum Measurements. NHANES uses a complex, multistage sampling framework to produce a sample representative of the noninstitutionalized, civilian U.S. population. As part of the 2003−2004 NHANES survey, a random one-third subset of the participants ($n = 2305$) aged 12 years and above were chosen for PBDE serum analysis. From this subsample, PBDEs were successfully measured in 2040 serum samples. Concentrations for the following 10 PBDE congeners were determined by gas chromatography isotope dilution high resolution mass spectrometry: BDE-17, -28, -47, -66, -85, -99, -100, -153, -154, and -183 (National Center for Environmental Health, CDC, Atlanta, GA). Distributions and percents detected for these congeners have been reported elsewhere (*11*). For this analysis, we selected the six congeners (BDE-28, -47, -99, -100, -153, and -154) that had at least 50% of samples above the LOD. Concentrations below the LOD were substituted by the CDC with a value equal to the congener-specific LOD divided by the square root of two. Since congeners 47, 99, 100, 153, and 154 are the major components of the penta-BDE formulation, with BDEs 47 and 99 accounting for approximately 75% of the total mass, and BDE-28 is a minor component of penta-BDE (*17, 26*), we summed the six congeners to create a summary metric for the penta-BDE formulation (ΣPBDEs). If data for one or more congeners was not reported by the CDC, the participant was coded as missing for ΣPBDEs. Total PBDE concentrations were calculated for 1942 participants and are expressed as ng PBDE per gram serum lipid. Serum PBDE concentrations approximated a log-normal distribution and were log-transformed prior to statistical analyses.

Information pertaining to NHANES participants' county, state, and region of residence (West, Midwest, South, and Northeast) were obtained through the Research Data Center (RDC) (National Center for Health Statistics, Hyattsville, MD). Participants with PBDE serum measurements resided in 29 U.S. counties; four of which were located in California. For confidentiality reasons, the actual survey locations are not disclosed.

Participants from California counties were assigned a "yes" for a binary measure indicating residence in California, versus "no" for participants from counties in other U.S. states. Publicly accessible NHANES data files provide masked variance units (MVUs) to estimate sampling error and to comply with disclosure agreements that prohibit the release of the primary sampling units (PSUs) (*27*). We obtained the true PSUs and stratum information through the RDC and used this information to construct our main variable of interest, residence in California, and to calculate standard errors for all estimates.

We also included these covariates in the serum analysis: age (12−19, 20−39, 40−59, and ≥60 years), sex (male or female), education (≥18 years and not completed high school versus completed high school or <18 years), annual household income (more or less than $20 000), race (non-Hispanic white, non-Hispanic black, Mexican American, or other), and country of origin. (U.S.-born or foreign-born.)

All analyses were conducted in SUDAAN 9.0 (Research Triangle Institute, Cary, NC) and SAS 9.1 (SAS Institute Inc., Cary, NC). SUDAAN calculates variance estimates after incorporating the nonrandom sampling design and the sample population weights, which account for the unequal probability of selection into the survey and the oversampling of certain subgroups. For univariate analyses, geometric means and percentile estimates were calculated with PROC DESCRIPT. Boxplots were constructed using weighted percentile estimates. Differences across groups for categorical data were evaluated using the chi square test. The least-square geometric means (LSGM), which provide geometric mean estimates for a variable after adjustment for other model covariates, were calculated from multivariate regression models.

To obtain the final model, we used backward elimination with a threshold $p < 0.05$ for retaining the variable in the model. We assessed confounding by adding each of the excluded variables back into the model and determining whether the beta coefficient for the main effect changed by >10%. If so, we retained the nonsignificant confounding variable in the model. Participants who were classified in a race/ethnicity category other than Mexican American, non-Hispanic black, or non-Hispanic white ($n = 149$) were included in the descriptive statistics but not in regression analyses. Country of origin and race/ethnicity were not modeled together in multivariate regression models due to the small number of foreign-born non-Hispanic blacks and non-Hispanic whites among California NHANES participants. Alternatively, a four category race/ethnicity variable that distinguished between U.S.-born and foreign-born Mexican Americans was created and used in sensitivity analyses. Results from regression models with ΣPBDEs as the outcome are presented below. Similar models with BDE-47 serum levels as the outcome were constructed and are briefly discussed in the results.

Results

Household Dust. PBDE household dust concentrations in Richmond and Bolinas, California, are presented in Table 1. Median concentrations of BDE-47,-99, and -100 across all

TABLE 1. PBDE House Dust Concentrations in Two California Communities (n = 49)

	Richmond (n = 39)			Bolinas (n = 10)			
	% >MRL[a]	median (ng/g)	range (ng/g)	% >MRL[a]	median (ng/g)	range (ng/g)	p-value[c]
BDE-47	100	3750	112-107 000	100	1260	192-31 100	0.53
BDE-99	100	3830	102-170 000	100	1160	209-44 900	0.68
BDE-100[b]	92.3	756	<MRL- 30 900	100	223	44.5-8720	0.60

[a] Method reporting limit (MRL) = 42 ng/g. [b] Values below the MRL were substituted with 0.5 × MRL. [c] Differences between groups tested using the Wilcoxon rank-sum test.

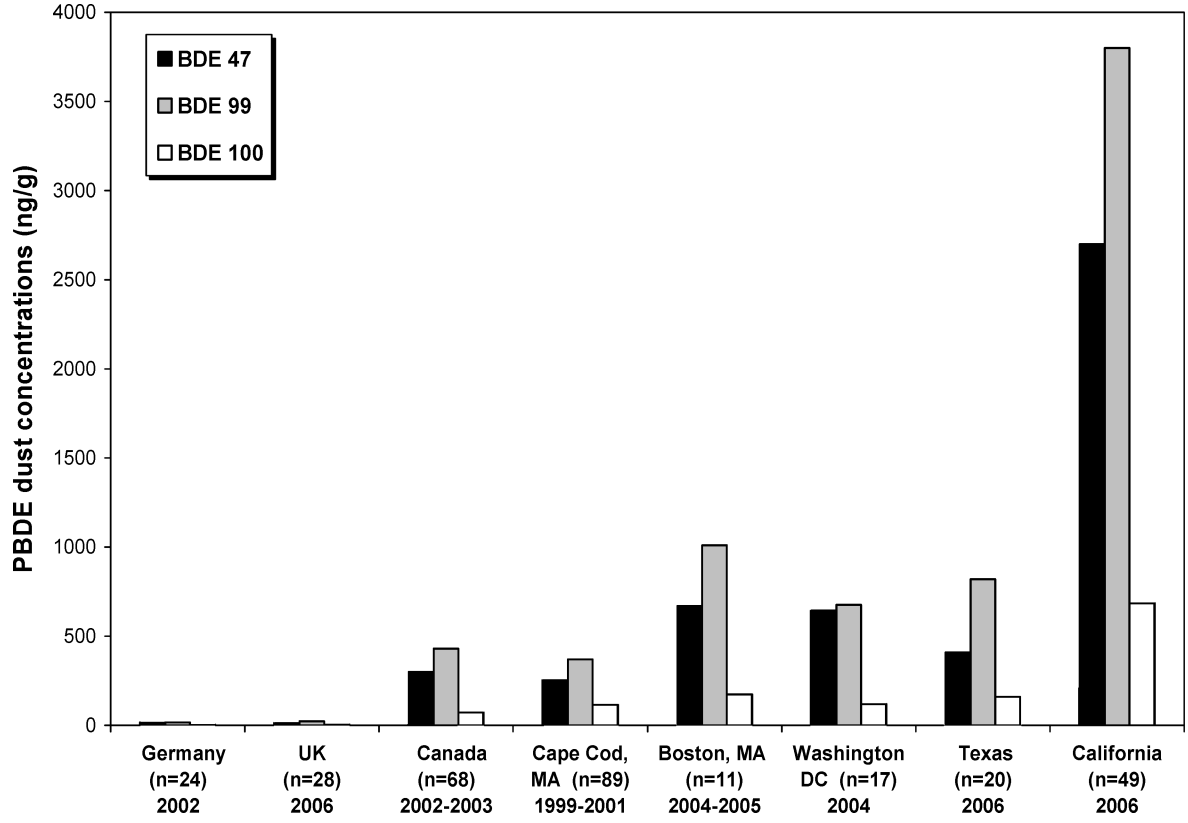

FIGURE 1. Median concentrations (ng/g) of BDE-47, -99, -100 in household dust from different locations. Data from Cape Cod, MA (Rudel et al. 2003, ref *4*) and California collected by the same research group using similar methodology. Data for Germany from Knoth et al. (2002, ref *28*), UK and Texas from Harrad et al. (2008, ref *29*), Canada from Wilford et al. (2005, *1*), Boston, MA from Wu et al. (2007, ref *14*), and Washington DC, from Stapleton et al. (2005, ref *30*). Study location, sample size and year of sample collection are also shown. Adjusted geometric mean estimates, calculated using maximum likelihood estimation for data below the reporting limit, are shown for Cape Cod, MA.

homes (n = 49) were 2700, 3800, and 684 ng/g, respectively. Concentrations were higher in Richmond (n = 39) than Bolinas (n = 10), but these differences were not statistically significant. California PBDE concentrations were also compared with summary measures from previously published studies. Characteristics for our study and comparison studies, including year and location of sampling, sample size, and median dust levels of BDE-47, -99, and -100, are presented in Figure 1. PBDE dust levels in California were markedly higher than previously reported in Europe and North America for all three penta-BDE congeners. Median house dust levels in California were 200 times higher than those reported from Germany (*28*) and United Kingdom (*29*), and 4−10 times higher than levels in Ottawa, Canada (*1*), Cape Cod, MA (*4*), Boston, MA (*14*), Washington, DC (*30*), and Texas (*29*). Maximum dust concentrations (Table 1) in our California study homes were higher than any we were able to identify in the peer-reviewed literature.

Serum. Regional PBDE serum levels were compared across the NHANES sample. Individual BDE congeners and ΣPBDEs varied by U.S. region ($p < 0.05$) with highest levels occurring in the Western region (which includes California) and lowest in the Northeast (Figure 2). The unadjusted medians for the West and California are very similar, with the 95th percentile being highest in California. In adjusted models (described below), the LSGM is slightly higher for California than the West; and the pattern across the four U.S. regions remains the same (results not shown).

Personal characteristics and PBDE serum concentrations of participants living in California versus the rest of the country are presented in Table 2. Of the 2040 NHANES participants, 276 (14%) were from California. California participants were similar to others in the U.S. in age, sex, and income. However, compared to the rest of the U.S., California had lower percentages of non-Hispanic whites and non-Hispanic blacks but a higher percentage of Mexican Americans. California also had higher percentages of foreign-born individuals and those not completing high school.

Four BDE congeners and ΣPBDEs were significantly higher in California residents ($p \leq 0.01$). BDEs 153 and 154 were also higher in California residents, although these differences were not statistically significant. Levels of BDE-47, the

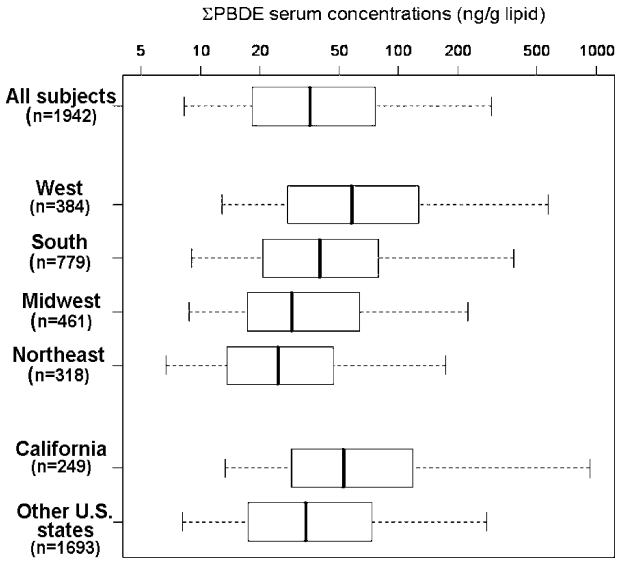

FIGURE 2. Differences in ΣPBDE serum by geographic region within the United States. Boxplots consist of 5th, 25th, 50th, 75th, and 95th percentiles. ΣPBDEs percentile estimates are adjusted for sample design and survey weights.

dominant congener in serum, were approximately 2-fold higher in California residents compared to the rest of the U.S. ($p = 0.003$).

In univariate analyses (results not shown), ΣPBDEs was positively associated with living in California ($p = 0.009$). A significant but nonmonotonic relationship was observed between age and ΣPBDEs ($p = 0.0001$) with highest ΣPBDEs levels observed in adolescents (12−19 years) and lowest levels in the 40−59 years age group. Higher ΣPBDEs were measured in males ($p = 0.008$). Lower household income was positively associated with ΣPBDEs ($p = 0.0002$). Foreign-born individuals had a trend toward lower ΣPBDEs levels ($p = 0.12$), and less educated individuals had a trend toward higher ΣPBDEs levels ($p = 0.11$). Race/ethnicity was not associated with ΣPBDEs ($p = 0.31$). Similar associations were observed with BDE-47 as the model outcome. However, education was inversely associated with BDE-47 ($p = 0.03$), and there were no significant differences by sex. Mexican Americans had significantly higher BDE-47 levels than non-Hispanic whites ($p = 0.01$).

The LSGM concentrations of ΣPBDEs for the multiple regression model are presented in Table 3, and corresponding model coefficients are presented in the Supporting Information (Table S1 (Model 1)). Average serum ΣPBDEs levels for participants residing in California (73.0 ng/g lipid) were approximately 2-fold higher than for participants living in other U.S. states (38.5 ng/g lipid, $p = 0.002$) (Figure S1, Supporting Information) after controlling for age, sex, income, and country of origin (Table 3). Higher serum ΣPBDEs was also significantly associated with adolescents, males, lower income, and being U.S-born. Participants living in low income households (50.4 ng/g lipid) had a significantly higher LSGM than higher income participants (37.7 ng/g lipid, $p < 0.0001$). Foreign-born participants (27.9 ng/g lipid) had a significantly lower LSGM than that of U.S.-born participants (42.1 ng/g lipid, $p = 0.0003$).

Since country of origin and race/ethnicity could not be modeled together due to small subgroup numbers, a multiple regression model including race/ethnicity but not country of origin was conducted as a sensitivity analysis (Supporting Information Tables S1, Model 2) with race/ethnicity recoded to distinguish between foreign-born and U.S.-born Mexican Americans. In this model, foreign-born Mexican Americans had the lowest ΣPBDE serum levels of all racial/ethnic groups (29.4 ng/g lipid), including significantly lower levels than U.S.-born Mexican Americans (43.8 ng/g lipid, $p = 0.0003$) (Supporting Information Table S2). Otherwise, the effect estimate and level of statistical significance for all other covariates, including living in California, and ΣPBDEs was similar between the two models.

Multivariate regression results using the single congener BDE-47 as the outcome were similar to the results for ΣPBDEs. Participants residing in California had an LSGM more than two times greater than those who were living in other U.S. states (41.3 vs 19.5 ng/g lipid, $p = 0.001$). Income, age, and being foreign-born significantly predicted BDE-47 serum levels in the multivariate model, while race and sex did not (results not shown).

Discussion

This is the first study to examine regional variations in PBDE levels in household dust and serum within the U.S. For both media, strong geographic trends were observed with consistently elevated penta-BDE levels in California.

This is also the first study to report penta-BDE house dust levels for multiple homes in California. An earlier report examined PBDE house dust levels in 10 North American regions including California, but only one sample was collected from each location (*31*), thus limiting inferences on regional variation. In our study, California median dust concentrations for the three BDE congeners characteristic of the penta-BDE formulation were 4−10 times higher than levels in other North American regions and approximately 200 times higher than levels from Europe.

The interpretation of comparisons across studies is often limited by differences in sample collection, analytical techniques, and timing of data collection. For example, vacuuming upholstery could produce higher PBDE levels than vacuuming only floors; however, our California dust data and the Cape Cod, Massachusetts, dust data (*4*) were collected and analyzed by the same research group using identical sampling protocols. Similarly, regional comparisons may be confounded by temporal changes in product use and formulation; however, the house dust samples from Texas and U.K (*29*) were collected during the same time as those in California. To strengthen regional comparisons, future studies should examine PBDE house dust concentrations across multiple locations using systematic methods.

To examine regional variation in human serum PBDEs, we were able to analyze NHANES, a U.S. population-based survey with a large sample size and high quality control standards. Results showed a strong association between California residence and higher ΣPBDE levels that persisted even after controlling for race/ethnicity, age, sex, country of origin, and income. On average, ΣPBDE serum levels of California residents were 2 times higher than for residents from other states. Similar geographic trends were observed when serum BDE-47 levels were modeled as the outcome. Serum levels for NHANES California participants were similar to the levels previously reported for two California adults but lower than those of their children (*10*). The LSGM of the foreign-born Mexican American participants examined in our study (29.4 ng/g lipid) was similar to the median PBDE concentration (21 ng/g lipid) among a sample of Mexican American women living in a California agricultural community, most of whom were foreign-born (*12*).

Our analysis is also one of the first studies to examine associations between socioeconomic status (SES) and PBDE exposure. Our results suggest that lower household income is associated with increased serum PBDE exposures. The physical weathering and crumbling of PBDE-treated foam in older furniture, often found in lower income homes, may release greater amounts of penta-BDE compounds into indoor environments (*32*), or cheaper furniture may be

TABLE 2. Personal Characteristics and Serum PBDE Levels for NHANES Participants Living in California vs Other U.S. States ($n = 2040$)

	California ($n = 276$)		other U.S. states ($n = 1764$)		
	frequency (%)[a]	95% CI	frequency (%)[a]	95% CI	p-value[b]
Personal Characteristics[c]					
age (years)					0.12
12−19	15.8	9.7−24.6	13.7	12.0−15.6	
20−39	40.1	29.8−51.4	32.7	28.8−36.9	
40−59	28.3	16.9−43.3	33.7	30.6−36.9	
≥60	15.9	11.2−22.0	19.9	17.3−22.8	
male	53.5	45.1−61.8	47.8	44.7−51.0	0.31
less than high school education	22.9	17.0−28.8	14.9	13.0−16.8	**0.01**
household income <$20 000	26.7	15.2−42.6	25.1	20.8−30.0	0.82
race/ethnicity					**<0.0001**
non-Hispanic white	46.6	33.6−60.1	73.3	64.7−80.6	
non-Hispanic black	4.7	1.9−11.0	12.5	9.1−16.9	
Mexican American	28.0	18.6−39.7	6.1	2.8−12.8	
other	20.8	12.7−32.1	8.0	5.7−11.1	
country of origin					**0.002**
United States	61.2	51.1−70.4	88.2	82.8−92.0	
Mexico	17.2	11.5−24.9	2.9	1.5−5.6	
other	21.6	14.7−30.6	8.9	6.1−12.9	
	geomean[a,d] (ng/g lipid)	95% CI	geomean[a,d] (ng/g lipid)	95% CI	p-value[b]
PBDE Serum Measures[e]					
BDE-28	2.1	1.5−2.7	1.1	0.9−1.3	**0.003**
BDE-47	36.2	25.0−47.4	19.5	16.6−22.4	**0.003**
BDE-99	7.4	5.2−9.6	[f]	[f]	**0.01**
BDE-100	6.0	4.2−7.8	3.8	3.2−4.4	**0.01**
BDE-153	6.8	5.2−8.4	5.6	4.8−6.4	0.18
BDE-154	0.8	0.6−1.0	[f]	[f]	0.05
ΣPBDEs[g]	62.0	44.6−79.4	38.6	33.5−43.7	**0.009**

[a] Estimates are adjusted for survey design and sample weight. [b] Significant ($p < 0.05$) differences between CA and other U.S. states are bolded. [c] Data were missing for education ($n = 3$) and income ($n = 38$). [d] Geomean is the geometric mean concentration. [e] Data were missing for BDE-28 ($n = 53$), BDE-47 ($n = 24$), BDE-99 ($n = 55$), BDE-153 ($n = 1$), BDE-154 ($n = 26$), and ΣPBDEs ($n = 98$). [f] Geometric mean is below the highest limit of detection for individual samples. [g] ΣPBDEs equal to the sum of BDE-28, BDE-47, BDE-99, BDE-100, BDE-153, and BDE-154.

TABLE 3. Adjusted Least Square Geometric Mean (LSGM) Concentrations of ΣPBDE Serum Concentrations (ng/g lipid) by Geographic Location and Other Personal Characteristics ($n = 1771$)[a]

variable	LSGM (95% CI)
geographic location	
California	73.0 (70.7−75.2)[d]
other U.S. states[b]	38.5 (36.4−40.5)
age[d]	
12−19 years[b]	50.9 (48.8−53.0)
20−39 years	43.4 (41.3−45.5)[c]
40−59 years	34.8 (32.7−37.0)[d]
≥60 years	40.4 (35.2−39.4)[d]
sex[d]	
male	44.3 (42.2−46.4)[d]
female[b]	37.3 (35.2−39.4)
household income[d]	
≤$20 000	50.4 (48.3−52.5)[d]
>$20 000[b]	37.7 (35.6−39.8)
country of origin[d]	
U.S.-born[b]	42.1 (40.0−44.2)
foreign-born	27.9 (25.8−30.0)[d]

[a] LSGM estimates are from multivariate regression models adjusted for survey design and sample weights. [b] Referent group. [c] $p < 0.05$. [d] $p < 0.01$.

manufactured in ways that release these chemicals in greater amounts. Our dust findings were consistent with the observation of higher serum PBDE in lower SES groups since we observed higher PBDE dust levels in Richmond (a lower SES community) than Bolinas, although this difference was not statistically significant, possibly due to our small sample size.

Our analysis of the NHANES serum PBDE levels contrasted in some respects from the results of Sjodin and colleagues (*11*). Although we found similar patterns in PBDE levels for sex and age, we found different effects for country of origin and race. After controlling for geographic location, serum PBDE levels of Mexican Americans were not higher than non-Hispanic whites. In fact, foreign-born Mexican Americans had significantly lower serum PBDE levels compared to U.S.-born Mexican Americans and non-Hispanic whites. Sjodin's finding of higher PBDE levels in Mexican Americans may result from the large proportion of Mexican Americans from California surveyed in NHANES. Similarly, his lack of association between country of origin and BDE-47 may be due to the high proportion of foreign-born participants from California in NHANES. Future research should further examine how exposure patterns among foreign-born immigrants change with length of residence in the U.S.

There are several limitations to our study. While NHANES is designed to be a representative sample of the U.S. population, the individuals sampled in California are not intended to be representative of California's population and are sampled from just four of California's 58 counties. Similarly, our dust samples from Richmond and Bolinas in Northern California may not be representative of the entire state. Furthermore, while we build on studies that point to dust as the primary source of human exposure (*1*, *3*), we

were not able to examine direct associations between PBDEs in household dust and body burden in this study. However, the data sources used in this analysis currently provide the most viable way to examine regional variation in PBDE exposure within the U.S.; and the consistent geographic differences we report in PBDE levels in both dust and serum compel additional research in a larger representative population where dust and blood samples can be analyzed from the same cohort. Lastly, while our analysis identified several important predictors of PBDE exposure, most of the variation was unexplained, implying that other unmeasured factors contribute and that determinants of exposure should be furthered investigated.

Given that PBDEs are ubiquitous and exposures differ among subpopulations, it is necessary to evaluate the impact of these exposures on human health end points such as thyroid hormone disruption. In an analysis of NHANES data, higher serum levels of PCBs, which share structural and mechanistic similarities with PBDEs, were associated with significant changes in thyroid hormone levels in the general U.S. population (*21*). Additionally, an increased prevalence of feline hyperthyroidism, which may, in part, be a result of PBDE exposures, has been observed in California cats (*33*). Unfortunately, thyroid hormone levels were measured in previous NHANES cohorts but not in 2003−2004 when PBDE levels were available. Concurrent measurements of PBDE biomarkers and thyroid levels should be a priority in future NHANES cycles.

Our regional analysis of PBDE serum levels adds to prior NHANES analyses that have considered the impacts of public health regulations and policies on population exposure. Prior studies in this vein include an evaluation of urine cotinine, a marker of tobacco smoke exposure, in relation to local regulations about smoking in public places (*34*), and an assessment of blood lead reductions due to the phasing out of lead in gasoline and household paint (*35*). Future studies should continue to monitor penta-BDE body burden, while also tracking exposures to replacement compounds.

Our findings show significantly elevated penta-BDE exposure in house dust and serum in California, which may reflect the unintended consequences of the state's stringent furniture flammability standards (*7*). There may be other explanations for elevated PBDE levels in California. For example, there could be regional differences in diet; however, diet is not considered to be the major source of PBDE exposures (*3*), and dietary differences would not explain the regional differences observed in house dust. These findings raise concern about pending regulations and performance standards that encourage the widespread use of chemical flame retardants, which are toxic or whose safety is uncharacterized. For example, the California agency that promulgated TB117 is on the verge of extending flammability requirements to bed clothing (*36*); and in the past two years, several state and federal initiatives have proposed adopting California's TB117 for furniture flammability (*19*). Although use of penta-BDE has been phased out, new chemicals have been substituted without assessment of their safety or environmental impact, and our findings may foreshadow exposure patterns to be anticipated from these substitutes. Taken together with existing research documenting the distribution of penta-BDEs internationally, these findings suggest the need for more anticipatory assessments of the environmental health impacts of consumer product decisions prior to their implementation.

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Supporting Information Available

Dust analytical methods and QAQC; regression models for ΣPBDEs and pairwise comparisons of ΣPBDEs by race/ethnicity (Tables S1−S2 and Figure S1) This material is available free of charge via the Internet at http://pubs.acs.org.

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