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## Temporal trends and developmental patterns of polybrominated diphenyl ether concentrations over a 15-year period between 1998 and 2013

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### Abstract

Polybrominated diphenyl ethers (PBDEs) were used extensively as flame retardants in furniture containing polyurethane foam until they were phased out of use, beginning in 2004. We examined temporal changes in PBDE concentrations from 1998 to 2013 and characterized patterns of exposure over the early lifecourse among 334 children (903 samples) between birth and 9 years. We examined time trends by regressing PBDE concentration on year of sample collection in age-adjusted models and characterized developmental trajectories using latent class growth analysis (LCGA). Controlling for age, BDE-47 concentrations decreased 5% (95% confidence interval (CI): -9, -2) per year between 1998 and 2013. When considering only postnatal samples, this reduction strengthened to 13% (95% CI: -19, -9). Findings for BDEs-99, 100 and 153 were similar, except that BDE-153 decreased to a lesser extent when both prenatal and postnatal samples were considered (-2%, 95% CI: -7, 0). These findings suggest that, on average, pentaBDE body burdens have decreased since the 2004 phase-out of these chemicals. When examining developmental period, PBDE concentrations peaked during toddler years for the

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majority of children, however, our observation of several unique trajectories suggests that a single measure may not accurately reflect exposure to PBDEs throughout early life.

## Keywords

PBDE; flame retardant; exposure; prenatal; childhood

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## Introduction

In 1970, approximately 37% of adults smoked and household fires attributable to ignition of upholstered furniture from improperly extinguished cigarettes was the leading cause of fire-related deaths in the United States<sup>1</sup>. In response to these statistics, the state of California initiated legislation requiring that companies manufacture fire-safe furniture and in 1975, Technical Bulletin 117 (Cal-117) was ratified, which required that all components of upholstered furniture pass an ‘open flame’ test before entering state commerce<sup>2</sup>. Between 1975 and 2004, polybrominated diphenyl ethers (PBDEs) were the primary flame retardant chemical used to comply with Cal-117. Commercially, PBDEs were used as components of three technical mixtures known as pentaBDE, octaBDE and decaBDE<sup>3</sup>. Beginning in 2004, PBDEs were phased out of use owing to their persistence in the environment and potential for human toxicity<sup>4–7</sup>. The present study focuses on BDEs-47, –99, –100, and –153, which are the predominant congeners in the pentaBDE formulation and are estimated to make up 90% of the human body burden<sup>8</sup>.

The United Nations Environmental Program (UNEP) estimates that 100 000 tons of pentaBDE was manufactured globally between 1975 and 2010<sup>9</sup>, with approximately 85% used in North America<sup>10</sup>, where exposure is ubiquitous and body burdens are the highest in the world<sup>11</sup>. PentaBDE was primarily used in couches, mattresses, carpet padding and other upholstered products<sup>3</sup> and typically comprised approximately 3% (by weight) of the polyurethane foam used in these products<sup>12</sup>. Peak pentaBDE use occurred in 2004 (17 000 tons); considering a product’s first lifespan (~15 years), it is estimated that the majority of pentaBDE-containing products will enter end of life waste streams by 2020<sup>13</sup>.

During production, PBDEs are not chemically bonded to base polymers, thus they have a propensity to migrate away from consumer products and accumulate in the indoor environment<sup>14</sup>. In the United States, human exposure occurs primarily through incidental ingestion of dust, with consumption of meat, fish and dairy products considered secondary sources<sup>15, 16</sup>. Owing to their lipophilic properties, PBDEs accumulate in adipose tissue<sup>17</sup>, readily cross the placenta<sup>18</sup>, and partition into breast milk<sup>19</sup>, placing fetuses and infants at risk for elevated exposure. Estimated half-lives of these congeners in adults range from 1.4 – 2.4 years for BDE-47 and 3.6 – 12.4 years for BDE-153<sup>20</sup>, however, little is known about how the unique exposure pathways (i.e. increased mouthing behaviors), metabolic differences, and other characteristics specific to children influence PBDE body burden.

Previous research has documented higher exposure to pentaBDE congeners among children compared to adults<sup>21</sup>, likely owing to the increased amount of time infants and toddlers spend in close proximity to the floor and the frequency with which young children mouth

fingers, toys and other objects<sup>22</sup>. While several studies have investigated temporal trends in PBDE exposure, the majority of existing research has been conducted in adults and/or has been cross-sectional by design<sup>23–26</sup>. In the present analysis, we aimed to address several of these limitations by investigating both time and age-specific changes in PBDE concentrations over the early lifecourse among a cohort of children that were born in the years spanning the pentaBDE phase-out.

## Materials and Methods

### Study participants

The study sample includes 334 of the 727 children enrolled in the Columbia Center for Children's Environmental Health (CCCEH) Mothers and Newborns birth cohort. As previously described<sup>27</sup>, healthy, non-smoking women living in Northern Manhattan or the South Bronx were enrolled during pregnancy between 1998 and 2006 and followed prospectively. Data analyzed in the present paper were collected between 1998 and 2013 at birth and at age 2, 3, 5, 7 and 9-year follow-up visits, resulting in a total of 903 data points. At each visit, a bilingual (English/Spanish) research worker conducted a structured interview with the mother to ascertain information related sociodemographic and lifestyle factors. Details related to housekeeping behaviors were collected by asking the mother about the frequency with which the home was cleaned with a vacuum, dust mop, damp mop or wet mop. Household material hardship was assessed based on the mothers self-reported ability to afford adequate food, clothing, or housing<sup>28</sup>. Before each visit, mothers were informed about all study procedures and provided written informed consent to participate; after age 7 years, children additionally provided informed assent. Study protocols were approved by the Institutional Review Board of Columbia University; it was determined at the Centers for Disease Control and Prevention (CDC) that the agency was not engaged in human subjects' research.

### Sample collection and laboratory analysis

At the child's birth, umbilical cord blood was collected by study staff, and at age 2, 3, 5, 7 and 9-year visits child venous blood was collected by a pediatric phlebotomist. Following collection, blood was separated and stored at  $-70^{\circ}\text{C}$  at the CCCEH laboratory. Aliquots of all available stored samples from each age period ( $N_{\text{cord}}=327$ ,  $N_{2\text{-years}}=56$ ,  $N_{3\text{-years}}=115$ ,  $N_{5\text{-years}}=42$ ,  $N_{7\text{-years}}=203$ , and  $N_{9\text{-years}}=160$ ) were shipped to the CDC for measurement of 11 PBDE congeners (BDEs: 17, 28, 47, 66, 85, 99, 100, 153, 154, 183, and 209). The present study examines BDEs 47, 99, 100 and 153, which were the most frequently detected congeners across study visits (Table 2). Details of the analytic method have been previously published<sup>29, 30</sup>. Briefly, samples were processed using automatic fortification with internal standards and extracted by automated liquid liquid extraction (Gilson Inc.; Middleton, WI). Analytic determinations were made by gas chromatography isotope dilution high resolution mass spectrometry. Final data were corrected for the median concentration detected in blank samples included in each analytic run (3 blanks per 30 samples). Lipids were co-extracted and removed on a silica: silica/sulfuric acid column using the Rapid Trace equipment (Biotage; Uppsala, Sweden). Total cholesterol and triglyceride levels were determined by standard enzymatic methods using commercially available test kits (Roche Diagnostics;

Indianapolis, IN). Child total plasma lipids were estimated from these measured components using the short formula described by Phillips et al.<sup>31</sup> and cord plasma lipids were estimated using a recently-developed cord blood specific formula [total cord blood lipids =  $2.66 \times$  total cord blood cholesterol + cord blood triglycerides + 0.268, in g lipids/L plasma] (Sjödin A, personal communication).

### Statistical analysis

We examined descriptive statistics and visualized age-specific distributions of BDEs-47, -99, 100, and -153 using histograms and boxplots. We calculated within congener correlations over time, as well as correlations between congeners at each time point. As previously described<sup>32</sup>, we imputed concentrations below the limit of detection (LOD) using a distribution-based multiple imputation method that accounts for sample-specific LOD. Distribution-based methods for imputing non-detected concentrations have been shown to produce unbiased results, even in the presence of a large number of samples (50–70%) with non-detectable concentrations of certain analytes<sup>33</sup>.

We examined temporal trends in exposure by regressing lipid-standardized,  $\log_{10}$ -transformed PBDE concentration on year of sample collection. We built separate models for each congener and used the generalized estimating equations approach with an exchangeable working correlation to account for repeated measures within a child over time. We isolated effects driven by time, rather than age, by adjusting these models for exact age at blood collection, which we included as a time-varying covariate. In addition to examining time as a continuous variable, we investigated the annual percent change in pentaBDE concentration in age-adjusted models stratified by whether samples were collected from 1998 to 2005 (n=457) or 2006 to 2012 (n=446).

To examine trajectories of PBDE exposure over early life, we used latent class growth analysis (LCGA) to empirically estimate discrete groups of children with shared patterns of measured PBDE concentrations (ng/g lipid) from birth through age 9 years<sup>34</sup>. This approach models PBDE concentration as a continuous function of age at the time of blood collection and estimates the probability of trajectory membership for each child. It is well-suited for complicated data structures as it allows for inclusion of all children with PBDE concentrations measured at a minimum of one time point. We  $\log_{10}$ -transformed PBDE concentrations to better approximate a normal distribution and estimated models with varying numbers of groups (1–6) and shapes (linear, quadratic, cubic). We evaluated model fit using the Bayesian Information Criterion (BIC), as well as the magnitude of group membership posterior probabilities.

We performed multinomial logistic regression within the LCGA modeling framework to identify sociodemographic and lifestyle characteristics that predict a child's trajectory assignment. In these models, trajectory membership is treated as the outcome variable and for each covariate the probability of belonging to a given trajectory (versus the persistent low trajectory) is estimated. In addition to date of birth, which we evaluated as a continuous variable in 3-year increments (1998–2000, 2001–2003, 2004–2006), we explored the following variables in bivariate models: ethnicity (African American vs. Dominican), gender (male vs. female), parity (nulliparous vs. multiparous), maternal age at delivery (>24 years

vs. 24 years), maternal level of education (high school vs. less than high school), household material hardship (inability to afford food, housing, or clothing vs. access to all), breastfeeding duration (≥ 12 weeks vs. <12 weeks), presence of a smoker in the home (yes vs. no), and frequency of vacuuming (ever vs. never), dust mopping (ever vs. never), damp mopping (ever vs. never) and wet mopping (ever vs. never). We included variables in congener-specific multivariable models if the p-value from bivariate associations was less than 0.10 for any one of the trajectories across the four congeners. We conducted linear regression analyses using SAS v9.4 (SAS Institute Inc., Cary, North Carolina) and performed LCGA and multinomial logistic regression using the SAS Proc Traj procedure<sup>35</sup>.

## Results

We measured PBDE concentrations in 903 samples collected repeatedly from birth to age 9 years among 334 children born between 1998 and 2006. All children were African American or Dominican. Table 1 presents sociodemographic and lifestyle characteristics of maternal-child pairs included in the analysis. These 334 children did not significantly differ at the p=0.05 level from the fully enrolled cohort (n=727) on any sociodemographic or lifestyle factor examined in this analysis with the following exceptions: children with a measure of PBDEs were more likely to be born to a nulliparous mother (50% vs. 40%), were more likely to live in a household that used a dust mop at the prenatal period (12% vs. 7%), and were less likely to live in a household that used a dust mop at the 7-year period (20% vs. 27%) or a damp mop at the 3-year period (63% vs. 70%). To allow time to age into the later study visits, children with PBDE measures were more likely to be born in 1998–2000 (n=183) compared to 2001–2003 (n=88) and 2004–2006 (n=63).

### PBDE concentrations

Across samples and congeners (BDE-47, -99, -100, -153), LODs for cord and child plasma PBDE concentrations ranged from 0.29 to 11.59 ng/g and 0.45 to 20.20 ng/g lipid, respectively. PBDE concentrations were more frequently detected in child compared to cord plasma samples and at all ages BDE-47 was the most frequently detected congener (Table 2). Geometric mean BDE-47 concentrations were highest in samples collected at age 2 years (38±6 ng/g lipid) and lowest in cord plasma samples (14±1 ng/g lipid); we observed a similar pattern for BDEs-99 and -100, however, while BDE-153 concentrations were also lowest in cord plasma, they peaked at age 7 years (25.1±1.6 ng/g lipid vs. 5.7±0.2 in cord blood). Within congeners, PBDE concentrations measured in cord plasma were poorly correlated with concentrations measured in child plasma, however, concentrations measured between ages 2 and 9 years were moderately to highly correlated (see Supplemental Material, Table S1). Within age periods, BDEs-47, -99 and -100 were moderately to highly correlated (minimum  $R_{\text{Spearman}}$ : 0.76 in cord plasma to maximum  $R_{\text{Spearman}}$  0.96 in 3-year plasma) (see Supplemental Material, Table S2).

### Changes over time

Controlling for child age at blood draw, BDEs-47, -99, -100 and -153 decreased by approximately 5% (95% CI: -9, -2), 7% (-9, -2), 5% (-7, -1), and 2% (-7, 0) per year between 1998 and 2013, respectively (Table 3 and Figure 1). When considering only

samples collected during the postnatal period, which likely reflect direct exposure to PBDEs from the environment rather than from maternal transfer, concentrations decreased by 13% (–19, –9), 13% (–19, –9), 11% (–15, –7), and 11% (–15, –7) per year between 2000 and 2013 for BDEs-47, –99, –100 and –153, respectively. In date-stratified models (1998–2005 vs. 2006–2012), the annual percent decrease in plasma BDE-47 concentration was approximately 5% (–9, 1) for samples collected in 1998–2005, compared to 16% (–21, –10) for samples collected in 2006–2012. Further, plasma BDE-47 concentrations were significantly higher among toddlers who turned 2–3 years old before (GM±SE: 40.2±3.9, n=127) versus after (GM±SE: 20.4±2.9, n=44) 2005. We observed a similar pattern at older ages, such that children who were 7–9 years old in 2005 had plasma BDE-47 concentrations (GM±SD: 29.7±5.1, n=20) that were 42% higher compared to children who were 7–9 year olds in 2011–2012 (12.4±1.9, n=30).

### Early life trajectories

As illustrated by Figure 2, the best fitting LCGA model revealed four trajectories of BDEs-47, 99, and 100. One trajectory was characterized by low PBDE concentrations at all ages ('persistent low'). Two trajectories were defined by high concentrations during childhood, one of which showed a decrease after age 2–3 years ('early postnatal peak') and a second that remained elevated throughout childhood ('sustained postnatal high'). The fourth trajectory was characterized by high prenatal concentrations that decreased after birth ('prenatal high'). Across these three congeners, the majority of children were assigned to the 'persistent low' (34–51%) or 'early postnatal peak' (24–38%) trajectories. We identified three relatively age-invariant trajectories of BDE-153, which we refer to as 'persistent low', 'sustained postnatal moderate', and 'sustained postnatal high'. Congener-specific sample sizes and frequencies for each trajectory are presented in Table 4. Owing to its small size (<10% of the sample), we do not plot the BDE-100 'prenatal high' trajectory, nor do we examine it in regression models; however, we retained the trajectory as it improved LCGA model fit. Across congeners, the mean posterior probability of trajectory membership (0.7–0.9) met or exceeded the widely-accepted threshold for satisfactory group assignment (mean of 0.7), indicating a high likelihood that a child's exposure pattern fit well within his or her assigned trajectory<sup>36</sup> (see Supplemental Material, Table S3).

### Predictors of trajectory assignment

Figure 3 presents odds ratio (OR) estimates from multivariable multinomial models examining determinants of PBDE trajectory membership, which were fit within the LCGA modeling framework. In all models, the 'persistent low' trajectory serves as the reference category. Four children are excluded from these models due to missing information on breastfeeding history. Consistent with changes in concentration over time, year of birth was the most important determinant of trajectory assignment; across congeners, children born later in the cohort were significantly less likely to be assigned to the 'prenatal high' (OR<sub>BDE-47</sub>=0.41, 95% CI: 0.20, 0.82; OR<sub>BDE-99</sub>=0.16, 95% CI: 0.07, 0.37), 'early postnatal peak' (OR<sub>BDE-47</sub>=0.31, 95% CI: 0.16, 0.62; OR<sub>BDE-99</sub>=0.28, 95% CI: 0.13, 0.59, OR<sub>BDE-100</sub>=0.33, 95% CI: 0.18, 0.61), or 'sustained postnatal high' (OR<sub>BDE-47</sub>=0.09, 95% CI: 0.02, 0.42; OR<sub>BDE-99</sub>=0.27, 95% CI: 0.13, 0.58, OR<sub>BDE-100</sub>=0.25, 95% CI: 0.09, 0.64, OR<sub>BDE-153</sub>=0.38, 95% CI: 0.20, 0.69) versus the 'persistent low' trajectory. In addition to



year of birth, the following variables met our criteria (bivariate p-value <0.10) for inclusion in multivariable models: ethnicity, maternal age at delivery, breastfeeding duration, presence of a cigarette smoker residing in the home, dust mopping the home, and damp mopping the home. For time-varying covariates (smoker in the home and household cleaning behaviors), we modeled predictors collected at the prenatal, 3-year and 7-year study visits for the ‘prenatal high’, ‘early postnatal peak’ and ‘sustained postnatal high’ trajectories, respectively.

In general, across trajectories and congeners, African American (versus Dominican) ethnicity, younger maternal age at delivery, longer breastfeeding duration, and living in a household with an active smoker were associated with higher odds of assignment to the ‘prenatal high’, ‘early postnatal peak’ or ‘sustained postnatal high’ trajectories versus the ‘persistent low’ trajectory (see Figure 3). With regard to cleaning behaviors, dust mopping was associated with lower odds of assignment to the ‘sustained postnatal high’ BDE-47 trajectory; however, this association was imprecisely estimated given the relatively low prevalence of dust mopping in the cohort (20% at the 7-year visit). In contrast, while dust mopping was not associated with the ‘sustained postnatal moderate’ or ‘postnatal high’ trajectories of BDE-153, children in households that used a damp mop were more likely to be assigned to these groups.

## Discussion

In the present analysis, we measured plasma PBDE concentrations over a 15-year period. Given our relatively large sample size and the frequency of repeated measures, this study provides one of the most comprehensive PBDE exposure assessments that has been conducted among children to date. Controlling for age, we found that concentrations of congeners in the pentaBDE technical mixture, which was phased out of U.S. commerce in 2004, significantly decreased between 1998 and 2013.

Several previous studies have examined temporal trends in PBDE concentrations with inconsistent findings. For example, while Zota et al. found geometric mean pentaBDE concentrations decreased by 65% between 2008/2009 and 2011/2012 among two small samples of pregnant women living in California<sup>26</sup>, Hurley et al. found that serum pentaBDE concentrations increased marginally among 1253 older women (40–94 years) living in California between 2011 to 2015<sup>23</sup>. Notably, it is difficult to compare our findings to previous research investigating temporal trends, as the majority of studies have examined adult populations and/or have been limited to relatively short time frames that did not span the pentaBDE phase-out. When examining changes in concentration across age, we found that average plasma pentaBDE concentrations were consistent with other U.S.-based longitudinal<sup>37–41</sup> and cross-sectional<sup>21, 24, 42–50</sup> studies of children, except that we detected slightly lower concentrations of BDEs-47, –99 and –100 at older ages and lower concentrations of BDE-153 at all ages (Supplemental Material, Table S4 and Figure S5).

In addition to examining average changes over time and age, we used LCGA to identify children with similar developmental patterns of PBDE exposure over early life. While this method is used extensively in psychology and the social sciences (i.e. criminology,





high' trajectory, which may reflect differences in cleaning behaviors or other cultural differences between African American and Dominican households.

Consistent with previous research demonstrating breastfeeding as a pathway of PBDE exposure<sup>15</sup>, children who were breastfed 12 weeks or longer were more likely to be assigned to the sustained postnatal high trajectory. Unexpectedly, breastfed children were also more likely to have high prenatal BDE plasma concentrations. In this cohort, breastfeeding (<12 weeks vs. ≥12 weeks) was associated with indicators of low socioeconomic status, such as material hardship (OR=1.66, 95% CI: 1.20, 2.82). It is possible that breastfeeding is serving as an indicator of unmeasured cultural or socioeconomic factors associated with PBDEs, such as the use of second-hand or deteriorating household furniture, which may be more likely to contain (due to older age) and leach (due to greater wear and tear) PBDEs. Children born into households with an active smoker were also more likely to be assigned to the 'prenatal high' trajectory. The direction of this finding is consistent with the results of a U.S.-based study that detected higher hand wipe PBDE concentrations among young children living in homes with an active smoker<sup>59</sup>. It is unlikely that cigarettes are a direct source of PBDEs; however, similar to our breastfeeding findings, it is possible that smoking may serve as an indicator of unmeasured socioeconomic factors related to PBDE exposure.

With regard to cleaning behaviors, children in households that used a dust mop were less likely to have high concentrations of BDEs-47, 99 and 100 throughout childhood. In contrast, children in households that reported using a damp mop were more likely to have moderate or high BDE-153 concentrations throughout childhood. While unexpected, this later finding is consistent with results from the Spain-based INMA cohort, which found more frequent housekeeping (>1 times /week, including sweeping, vacuuming, dusting, and mopping) was associated with significantly higher serum concentrations of BDE-153, but not the other congeners, among pregnant women<sup>60</sup>.

The U.S. Environmental Protection Agency recommends that parents dust, wet mop and use a vacuum with a high efficiency particulate air (HEPA) filter to reduce children's exposure to flame retardants in dust<sup>61</sup>; however, given our inconsistent findings related to cleaning behaviors, further research, including household intervention studies, is needed to better understand what behavioral modifications are most effective for reducing exposure.

Strengths of our study include the large sample size, variation in both the chronological date and child age of blood collection, and the rich set of prospectively collected covariate data, including information on cleaning behaviors. Specific strengths of LCGA include the ability to retain all children with data at a minimum of one follow-up period, as well as the ability to model variation in the age at blood draw within follow-up periods. Moreover, future research can employ LCGA modeling to investigate the impact of exposure timing on distal health outcomes among children. Importantly, although our data met the generally accepted posterior probability threshold for trajectory assignment, it is possible that some children were misclassified, which may have biased our findings towards the null.

Concurrent with the voluntary 2004 industry phase-out of pentaBDE, New York State passed an environmental law that codified the prohibition of pentaBDE production and

use<sup>62</sup>. Despite these regulatory changes, PBDEs continue to leach from existing consumer products and migrate into house dust. Indeed, in the present study, we detected PBDE concentrations in approximately 80% of cord plasma samples collected between 1998 and 2006, and 100% of child (ages 2–9 years) samples collected between 2000 and 2013. Moreover, our finding of lower plasma BDE-47 concentrations among children who were 7–9 years old in 2011–2012 versus 2005 suggests that while pentaBDE concentrations have been decreasing since their 2004 phase-out, they continue to be detectable in the blood of young children nearly 10 years following their removal from U.S. commerce.

Our findings of several unique PBDE trajectories may inform future research studies as well as interventions designed to target specific windows of peak exposure. Importantly, in the United States, the majority of furniture and other household items containing polyurethane foam are disposed of in landfills. For example, approximately 1.3 million tons of carpet/padding, furniture, and other bulky items were disposed of in California landfills in the year 2004 alone<sup>63</sup>. With more PBDE-containing items entering end-of-life waste streams in the coming decades, shifts in environmental contamination patterns due to leaching from outdoor reservoirs may trigger a transition in human exposure pathways from dust to dietary sources (fatty fish, seafood, meat, dairy)<sup>64</sup>. As time since the pentaBDE phase-out elapses, monitoring landfills, surrounding environmental media, and wildlife will be critical for understanding shifts in exposure pathways and reducing human exposure.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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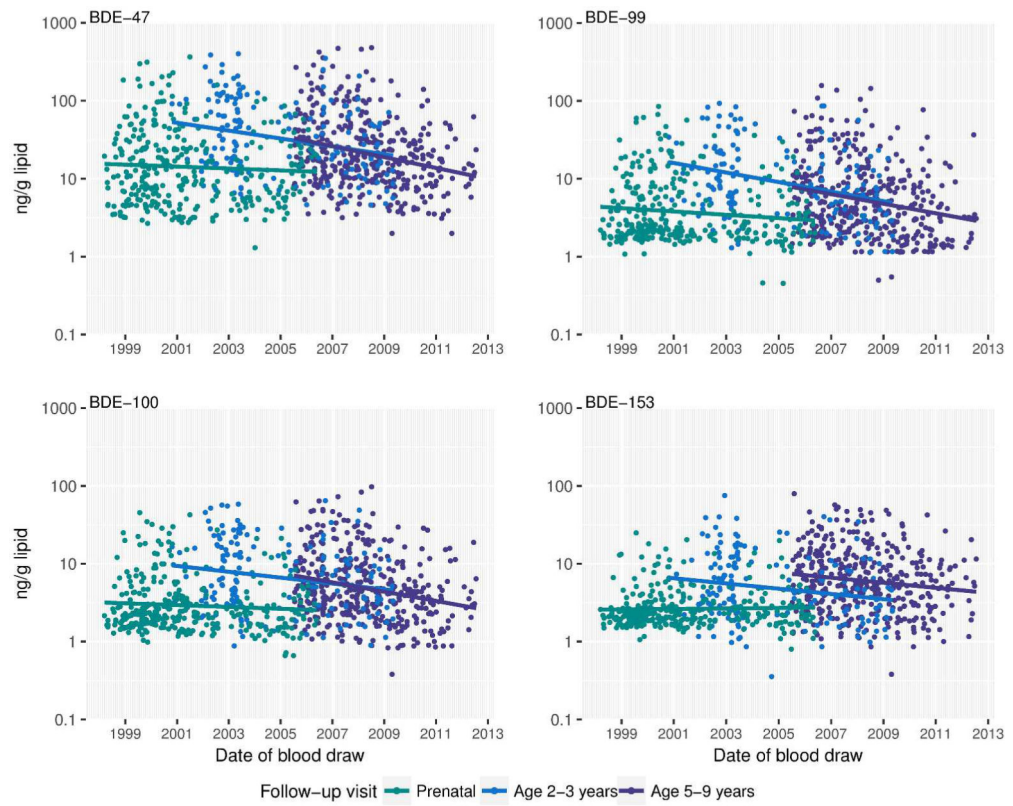
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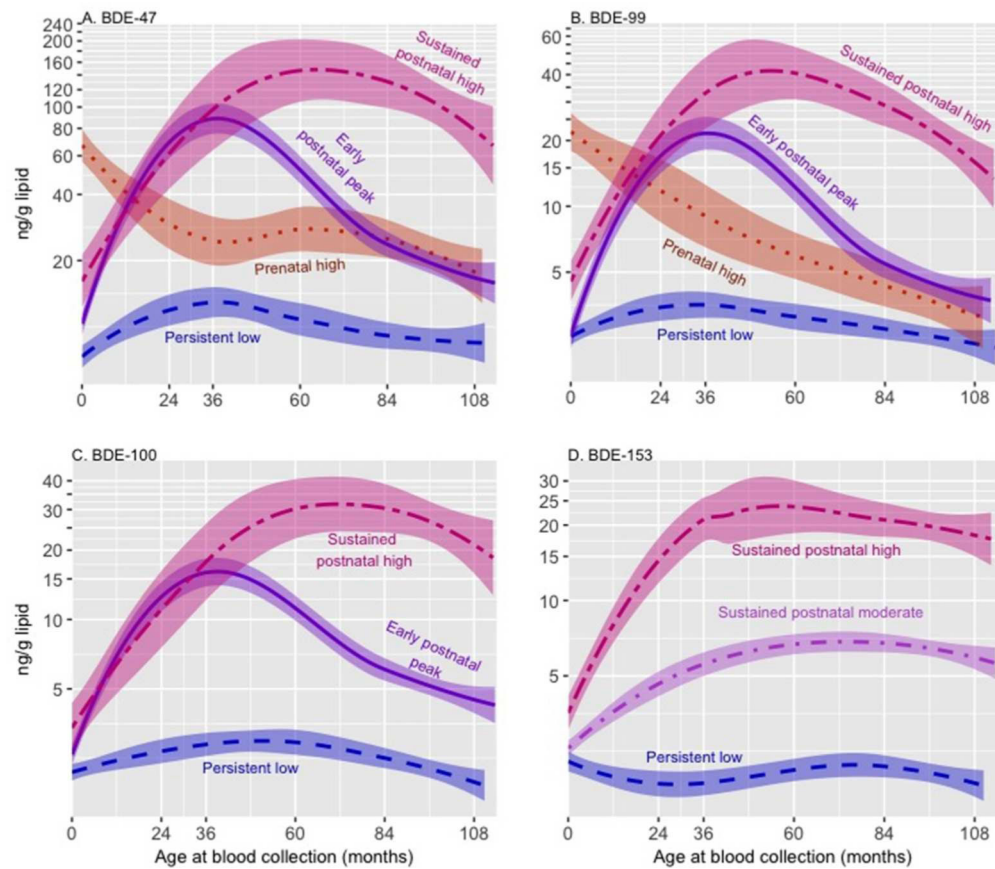
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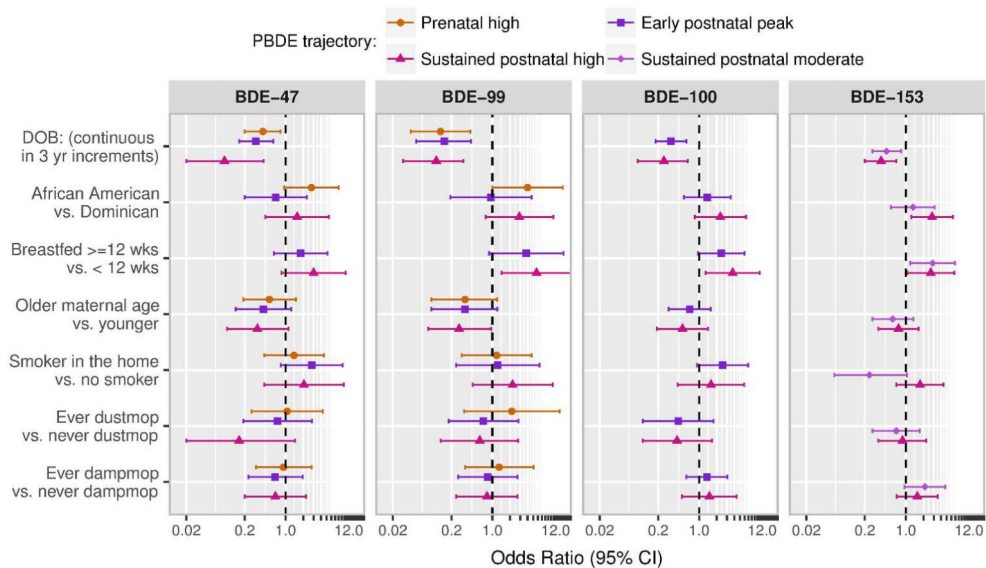




**Figure 1.** Changes in age-adjusted plasma PBDE concentrations (ng/g lipid) between 1998 and 2013 (n=903 samples from 334 children). Prenatal concentrations were measured in cord plasma.



**Figure 2.** Trajectories of plasma PBDE concentrations (ng/g lipid) from birth through 9 years (n=334) estimated using latent class growth analysis; bands represent 95% confidence intervals. The 'persistent low' trajectory serves as the reference category.



**Figure 3.** Odds ratios (ORs) from multivariable multinomial models examining determinants of PBDE trajectories over early life. The ‘persistent low’ trajectory serves as the reference category. OR from models examining breastfeeding as a predictor of the prenatal high trajectory are not plotted due to the small number of breastfed children that were assigned to this trajectory and resulting wide confidence intervals.

**Table 1.**

Characteristics of maternal-child pairs (n=334).

	n (%)
Child birth: 1998–2000	183 (55)
Child birth: 2001–2003	88 (26)
Child birth: 2004–2006	63 (19)
African American	124 (37)
Dominican	210 (63)
Maternal age ≥ 24 years <sup>a</sup>	161 (48)
Maternal <H.S. education <sup>a</sup>	117 (35)
Nulliparous <sup>a</sup>	168 (50)
Child sex (female)	182 (54)
Breastfed < 12 weeks	217 (66)
Smoker in home	
Prenatal	114 (34)
3 years	71 (21)
7 years	49 (15)
Material hardship	
Prenatal	129 (39)
3 years	96 (31)
7 years	104 (36)
Ever vacuum home	
Prenatal	57 (17)
3 years	51 (17)
7 years	70 (22)
Ever dust mop home	
Prenatal	41 (12)
3 years	52 (16)
7 years	67 (20)
Ever damp mop home	
Prenatal	190 (57)
3 years	209 (63)
7 years	202 (60)
Ever wet mop home	
Prenatal	174 (58)
3 years	194 (61)
7 years	194 (64)

<sup>a</sup>At delivery

Abbreviations: high school (H.S.)

**Table 2.**

Summary of PBDE concentrations measured in umbilical cord and child plasma between birth and age 9 years (n=903 samples from 334 children).

	Cord (n=327)	Age 2 (n=56)	Age 3 (n=115)	Age 5 (n=42)	Age 7 (n=203)	Age 9 (n=160)
<b>BDE-47</b>						
GM±GSD (pg/g serum)	30.8±1.9	139.4±21.0	133.1±13.4	98.6±15.0	90.2±6.8	77.8±6.4
GM±GSD (ng/g lipid)	14.1±0.9	37.8±5.8	32.1±3.1	25.6±3.8	23.2±1.7	18.1±1.4
<LOD (n, %)	66 (20)	0 (0)	1 (1)	1 (2)	5 (2)	1 (1)
Non-reportable (n, %)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
<b>BDE-99</b>						
GM±GSD (pg/g serum)	8.7±0.4	42.8±7.8	34.6±3.6	23.9±3.9	23.1±1.8	19.5±1.6
GM±GSD (ng/g lipid)	3.7±0.2	18.1±11.7	8.2±0.9	6.1±1.0	5.8±0.5	4.4±0.4
<LOD (n, %)	161 (49)	1 (2)	9 (8)	7 (17)	40 (20)	30 (19)
Non-reportable (n, %)	1 (0.3)	13 (23)	9 (8)	0 (0)	0 (0)	0 (0)
<b>BDE-100</b>						
GM±GSD (pg/g serum)	6.7±0.3	26.6±3.9	27.5±2.5	23.3±3.5	20.7±1.4	17.6±1.4
GM±GSD (ng/g lipid)	2.9±0.2	7.2±1.1	6.6±0.6	6.0±0.9	5.3±0.4	4.0±0.3
<LOD (n, %)	191 (58)	0 (0)	5 (4)	4 (10)	17 (8)	12 (8)
Non-reportable (n, %)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (0.6)
<b>BDE-153</b>						
GM±GSD (pg/g serum)	5.7±0.2	18.0±2.5	20.4±1.8	23.4±3.7	25.1±1.6	23.7±1.8
GM±GSD (ng/g lipid)	2.6±0.1	4.8±0.7	4.9±0.4	6.1±1.0	6.4±0.4	5.5±0.4
<LOD (n, %)	204 (62)	1 (2)	7 (6)	4 (10)	12 (6)	9(6)
Non-reportable (n, %)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	2 (1)
Total lipids (mg/dL)	236±11 <sup>a</sup>	385±70 <sup>b</sup>	433±77 <sup>b</sup>	398±62 <sup>b</sup>	406±79 <sup>b</sup>	450±87 <sup>b</sup>

Abbreviations: GM: geometric mean, GSD: geometric standard deviation, LOD: limit of detection; PBDE: polybrominated diphenyl ether.

<sup>a</sup>Estimated using: total cord blood lipids = 2.66 × cord blood total cholesterol + cord blood triglycerides + 0.268, in g lipid/L plasma

<sup>b</sup>Estimated using: total blood lipids = 2.27 × total cholesterol + triglycerides + 0.623, in g lipid/L plasma

**Table 3.**

Change in cord or child plasma PBDE concentrations (ng/g lipid) over time in GEE models adjusting for age at sample collection.

	Percent change/year (95% CI) <sup>a</sup>	N children <sup>b</sup>	N observations <sup>b</sup>
Cord & child samples (1998–2013)			
BDE-47	−4.5 (−8.8, −2.3)	334	903
BDE-99	−6.7 (−8.8, −2.3)	334	880
BDE-100	−4.5 (−6.7, −0.9)	334	902
BDE-153	−2.3 (−6.7, 0.0)	334	901
Child samples only (2000–2013)			
BDE-47	−12.9 (−18.7, −8.8)	288	576
BDE-99	−12.9 (−18.7, −8.8)	285	554
BDE-100	−10.9 (−14.9, −6.7)	288	575
BDE-153	−10.9 (−14.9, −6.7)	281	574

<sup>a</sup>Percent change calculated using:  $((1-10^{\beta}) \times 100)$ , where the  $\beta$  coefficient is estimated by regressing  $\log_{10}$ -transformed PBDE concentration on year of sample collection.

<sup>b</sup>Sample size varies due to non-reportable PBDE results (see Table 2)



**Table 4.**

Sample size of each PBDE exposure trajectory, N (%)

	<b>Persistent low</b>	<b>Prenatal high</b>	<b>Early postnatal peak</b>	<b>Sustained postnatal moderate</b>	<b>Sustained postnatal high</b>
BDE-47	113 (34)	68 (20)	116 (35)	NA	37 (11)
BDE-99	148 (44)	49 (15)	81 (24)	NA	56 (17)
BDE-100	155 (46)	NA	117 (35)	NA	32 (10)
BDE-153	88 (26)	NA	NA	185 (55)	61 (18)

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