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Determinants of prenatal exposure to polybrominated diphenyl ethers (PBDEs) among urban, minority infants born between 1998 and 2006☆

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ABSTRACT

Polybrominated diphenyl ethers (PBDEs) are environmentally persistent chemicals that structurally resemble legacy pollutants, such as polychlorinated biphenyls (PCBs). PBDEs were added to consumer products for over 30 years, before being phased out due to evidence of toxicity. We examined temporal changes in prenatal exposure to PBDEs, as well as other sources of variation. We measured PBDEs in umbilical cord plasma from 327 minority infants born in New York City between 1998 and 2006. We used linear regression to examine changes in concentrations over time and in relation to lifestyle characteristics collected during pregnancy. We detected BDE-47 in 80% of samples with a geometric mean concentration of 14.1 ng/g lipid. Ethnicity was the major determinant of PBDE exposure; African American infants had 58% higher geometric mean cord plasma concentrations of BDE-47 ($p < 0.01$) compared to Dominican infants. Notably, African American mothers were more likely to be born in the United States, which itself was associated with 40% ($p < 0.01$) higher concentrations. We observed small decreases in PBDE concentrations by date of birth and no difference before and after their phase-out in 2004. Final multivariable models explained 8–12% of variability in PBDE concentrations depending on the congener. Our finding that prenatal exposure to PBDEs decreased only modestly between 1998 and 2006 is consistent with the persistent properties of PBDEs and their ongoing release from existing consumer products.

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Following a 1975 fire-safety law passed by the state of California, companies across the United States began adding polybrominated diphenyl ethers (PBDEs) to couches, chairs and other upholstered products, including those designed for infants and children (Abbasi et al., 2015; Stapleton et al., 2011a). Industry has primarily relied on three commercial formulations, which each consist of several PBDE congeners that vary in the number and location of bromine atoms around a diphenyl ether backbone (UNEP, 2012). Over time, PBDEs migrate away from consumer products and enter house dust (Frederiksen et al., 2009). In the United States, human exposure occurs primarily through incidental ingestion of dust, with consumption of meat, fish and dairy products considered secondary sources (Lorber, 2008). PBDEs are classified as persistent organic pollutants (POPs). Structurally, they resemble several organohalogenated pollutants with known human toxicity, including polybrominated biphenyls (PBBs) and polychlorinated biphenyls (PCBs) (UNEP, 2012). PBDEs are persistent, bioaccumulative and capable of long-range transport once released into the environment (Law et al., 2014). Due to their lipophilic properties, PBDEs bioaccumulate in adipose tissue, cross the placenta, and partition into breastmilk (Frederiksen et al., 2009; Lorber, 2008). As previously reviewed (Fromme et al., 2016; Linares et al., 2015), research examining human exposure has documented associations between PBDEs and endocrine disruption, reproductive problems and neurodevelopmental deficits. Owing to these health and environmental concerns, the three major commercial formulations were

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phased out of production in the United States between 2004 (PentaBDE, OctaBDE) and 2013 (DecaBDE) (Corportation, 2005; USEPA, 2010). Despite these phase-outs, existing consumer products that are infrequently replaced, such as upholstered furniture, continue to release PBDEs, therefore, it is likely that only minimal reductions in human body burden will occur as these products are progressively removed from the indoor environment. In this study, we examined time trends and other potential determinants of prenatal exposure to PBDEs, including ethnicity, in a low-income, minority cohort of infants born between 1998 and 2006.

1. Methods

1.1. Study population

We conducted this study among a subset of 727 participants enrolled in the Columbia Center for Children's Environmental Health (CCCEH) Mothers and Newborns birth cohort, which was designed to examine sub-clinical health effects associated with prenatal exposure to several environmental chemicals. Women with a singleton pregnancy were recruited from two prenatal clinics in Northern Manhattan between 1998 and 2006. Women were excluded if they were younger than 18 or older than 35 years, started prenatal care after the 20th week of pregnancy, were active smokers, had a history of drug abuse, or had diabetes, hypertension or known HIV infection (Perera et al., 2006). All study protocols were approved by the Institutional Review Board of Columbia University; it was determined that the Centers for Disease Control and Prevention were not engaged in human subjects' research. Before each study visit, mothers were informed about all study procedures and provided written informed consent to participate.

1.2. Data collection

At the prenatal visit, trained research workers conducted structured interviews to ascertain information on demographic and several lifestyle factors, including material hardship and environmental tobacco smoke exposure. We defined material hardship as the inability to afford food, clothing or housing, and assessed environmental tobacco smoke (ETS) exposure by maternal report of smokers in the home in combination with cord blood cotinine concentrations as previously described (Rauh et al., 2004). We assessed home cleaning habits by asking question about the frequency of vacuuming and mopping. After birth, we abstracted data related to the pregnancy, delivery, and newborn from hospital medical records.

1.3. Umbilical cord blood collection and PBDE analysis

At delivery, umbilical cord blood was collected by study staff and transported to the CCCEH laboratory where samples were processed and stored in multiple aliquots at −70 °C. Staff at the CDC measured 11 PBDE congeners (BDE-17, -28, −47, −66, −85, −99, −100, −153, −154, −183, and 209) in stored umbilical cord plasma (Jones et al., 2012; Sjodin et al., 2004). Briefly, after fortification with internal standards, plasma samples were extracted using a Gilson 215 liquid handler (Gilson Inc., Middleton, WI) and lipids were removed on a Rapid Trace modular SPE work station (Biotage, Uppsala, Sweden). Final analytic concentrations were determined by gas chromatography isotope dilution high-resolution mass spectrometry on a DFS instrument (ThermoFisher, Bremen, Germany). Blanks (N = 3) were processed with every 30 samples and the median blank value was subtracted from the final result. Serum lipids (total cholesterol and triglycerides) were measured using commercially available test kits (Roche Diagnostics, Indianapolis, IN) and total blood lipids were estimated using a recently developed cord blood-specific formula [total cord blood lipids = 2.657 × total cord blood cholesterol + cord blood triglycerides + 0.268, in g lipids/L plasma] (A Sjodin, unpublished data, November 2016].

1.4. PBDE predictors

We reviewed published literature to identify potential determinants of PBDE exposure related to: 1) the mother (ethnicity, country of birth, residential history, age at delivery, education, relationship status, parity, time since previous pregnancy, and employment during pregnancy); 2) the newborn (sex, birthweight, gestational age); and 3) the household (material hardship, cleaning habits, environmental tobacco smoke) for which we had collected information during the prenatal period.

1.5. Statistical analysis

1.5.1. Distribution-based multiple imputation of non-detected concentrations

We focused our analysis on BDEs-47 (detection frequency: 80%), −99 (50%), −100 (42%) and −153 (38%), which were the most frequently detected congeners. We natural-log transformed concentrations to better approximate a normal distribution and examined each as a continuous, lipid-adjusted variable. We imputed concentrations below the limit of detection (LOD) using a distribution-based multiple imputation approach (R function available in Supplemental Information Table 1 and LODs available in Supplemental Information Table 2). Specifically, to account for each sample's unique LOD, which is proportional to the available plasma volume and lipid content, we randomly drew a value from a congener-specific distribution of detected data for which the LOD was less than or equal to that of the non-detected concentration. We repeated this imputation procedure 10 times and pooled parameter estimates for all subsequent analyses using the Multiple Imputation by Chained Equations (MICE) R package.

1.5.2. Predictors of cord plasma PBDE concentrations

We examined descriptive statistics for each potential determinant identified from our review of the literature. To evaluate whether children without a measure of PBDEs were different from those with a measure of PBDEs, we conducted student's t, Wilcoxon-Mann-Whitney, and Pearson's chi-square tests to identify differences in predictors among children included versus excluded for not having a measure of PBDEs.

We used linear regression to model the association between the child's date of birth and natural-log transformed PBDE concentrations (ng/g lipid). We treated date of birth as a continuous variable and examined units of both days and years. In addition to time, we examined effect estimates and p-values from models between PBDE concentrations and other potential predictors, which we included in congener-specific multivariable models if the univariate p-value was less than 0.1. We visualized our findings by plotting geometric mean PBDE concentrations within levels of each predictor, with continuous variables dichotomized at the median.

A woman's ancestry, geography, language, religion, lifestyle and cultural traditions shape her ethnicity. In turn, these factors may influence individual determinants of PBDE exposure. For example, PBDEs sorb to dust particles and ethnicity may influence home cleaning habits. Therefore, to better understand the impact of ethnicity on exposure, we examined potential determinants of cord blood PBDE concentrations among African American and Dominican participants in separate models. These models included the same predictors examined in unstratified models. We further

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