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Exposure to Persistent Organic Pollutants and Birth Characteristics: The Upstate KIDS Study

Griffith Bell[a], **Neil Perkins**[a], **Germaine Buck Louis**[b], **Kurunthachalam Kannan**[c], **Erin Bell**[d], **Chongjing Gao**[c], **Edwina Yeung**[a]

[a.]Division of Intramural Population Health Research, *Eunice Kennedy Shriver* National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, MD

[b.]Dean's Office, College of Health and Human Services, George Mason University, Fairfax, VA

[c.]Wadsworth Center, New York State Department of Health, and Department of Environmental Health Sciences, School of Public Health, State University of New York at Albany, Albany, NY

[d.]Department of Environmental Health Services, University at Albany, State University of New York, Albany, NY.

Abstract

Background: Prenatal exposure to persistent organic pollutants (POPs) may be associated with obesogenic effects in offspring. Our study is the first to investigate associations between concentrations of POPs from newborn dried blood spots (DBS) and birth characteristics.

Methods: Concentrations of 10 polychlorinated biphenyl congeners (PCBs), polybrominated diphenyl ether-47 (PBDE-47), and p,p´-dichlorodiphenyldichloroethylene (*p,p´*-DDE) were measured from DBSs collected at birth from 2065 singleton infants. DBS samples were pooled in groups of five and assayed together in order to reach limits of detection. Differences in risk of large for gestational age (LGA, defined as >90th percentile of birthweight for sex and gestational age), small for gestational age (SGA, <10th), and preterm birth (gestational age <37 weeks) were estimated using logistic regression per unit (ng/ml) increase in concentration of each chemical, adjusting for individual level covariates, including maternal age, race/ethnicity, prepregnancy BMI, education, parity, smoking, and infant sex while assuming a gamma distribution and using multiple imputation to account for pools.

Results: There were 215(11.3%) singletons born LGA, 158(7.5%) born SGA, and 157(7.6%) born preterm. Higher concentrations of POPs were positively associated with slightly higher risk of LGA and higher birth weight.

Conclusions: Relationships between POPs measured in newborn DBS and birth size were mixed. Pooled analysis methods using DBS could address challenges in limits of detection and costs for population-based research.

Corresponding author: Edwina Yeung, Epidemiology Branch, Division of Intramural Population Health Research, *Eunice Kennedy Shriver* National Institute of Child Health and Human Development, 6710B Rockledge Dr, MSC 7004, Bethesda, MD 20817, tel: 301-435-6921, edwina.yeung@nih.gov.

Introduction

Prenatal exposure to persistent organic pollutants (POPs), such as polychlorinated biphenyls (PCBs), *p,p'*-DDE and polybrominated diphenyl ethers (PBDEs) during pregnancy, is associated with adverse outcomes in offspring.[1–7] Maternal exposure to POPs is of concern, since many POPs have long half-lives and can pass from the mother via placental transfer to the developing fetus, as well as through breast milk to infants.[7–10] While many studies have reported associations between higher maternal concentrations of some POPs and lower birthweight - a crude proxy for fetal growth - findings remain equivocal.[1,2,7,11–18] possibly due to varying study protocols, heterogeneity in exposure levels between populations, or declining temporal concentrations in pregnant women. Still other studies have suggested that postnatal exposure is associated with larger body size and an increased risk of obesity in children.[2,11,19–21] Studies have also examined a wide range of pollutants, with observed associations varying by congener.[13] While several studies have measured POPs in maternal or cord blood, we are unaware of studies that measure POPs directly in the blood of newborn infants. Our study is the first to examine the cross-sectional relationship between direct measures of POPs concentrations in newborn blood samples and birth outcomes.

In our study, we examine fetal exposure to specific POPs through the use of dried blood spots (DBS) collected as part of a state newborn screening program. Newborn screening programs have collected DBS from newborns in the U.S. for over 40 years with the explicit purpose of screening for various genetic disorders and public health research.[22–25] As such, newborn DBS represent a potentially valuable population-level resource for quantifying POPs, metals and other persistent chemicals in relation to long-term health outcomes as well as for tracking population exposures over time.[22,23,26–31] Methods for quantifying POPs in DBS have been developed and found comparable across different blood matrices.[22] With parental consent, we utilized banked DBSs to assess POPs and birth characteristics.[23,32]

Methods

Design and Study Population

Our referent cohort is the Upstate KIDS Study, a population-based longitudinal study of mothers and their children who were born in New York State (excluding New York City) between 2008–2010.[33] In keeping with the Study's original aim to assess infertility treatment on children's growth and development, the cohort oversampled on infants conceived with infertility treatment. Using a matched exposure design with infertility treatment as the exposure, all singleton infants whose birth certificate noted the use of infertility treatment for conception were frequency matched to infants without such designation on residence in the State's regional perinatal systems at a ratio of 1:3. Recruitment occurred at approximately 4 months after delivery and parental consent to use DBS from the New York State Newborn Screening Program was solicited at 8 months. Along with consent, our current analysis required a whole DBS circle being available for measure (n=2610).[32] We further excluded 545 twins due to complex associations with birthweight and gestational age, leaving 2065 singletons of the original 3905 (67%) for analysis. We previously determined that maternal and infant characteristics were similar between newborns with samples available and their counterparts.[32]

Exposure Measurements

Whole-blood concentrations of select PCB congeners, *p,p'*-DDE and PBDE were measured using DBS samples collected by the newborn screening program activities. Blood samples were collected from heel-pricks of infants on standardized Whatman filter paper cards within 48 hours after birth. Cards were then dried and sent to the New York State Department of Health where they were stored at 4°C and archived by receipt date in polyethylene bags for follow-up testing. Following parental consent, banked DBS cards were retrieved from cold storage for analysis. Using published laboratory protocols for the analysis of POPs, *p,p'*-DDE, one PBDE (congener # 47) and 11 PCBs (congeners # 8, 15, 18, 28 ,52, 70, 95, 101, 138, 153, and 180) were quantified (ng/mL) using gas chromatography-DFS high resolution mass spectrometry.[28,30] Individual blood spots provided insufficient volume for assays, so pools of 5 were used to obtain the minimum required sample volume for analysis. The pools were constructed based on the original study aims. Thus, samples were stratified by plurality and fertility treatment (i.e., each pool of 5 DBS samples included infants with the same plurality and fertility treatment status), but otherwise individuals were unconstrained on values of other variables. Methods for analysis of pooled data have been developed and are further described in the statistical methods section.[34] As previously described[26,30], DDE had a limit of detection of 0.02 ng/ml, PBDE-47 of 0.003 ng/ml, PCBs 138, 153, and 180 of 0.002 ng/ml, PCBs 18 and 28 of 0.004 ng/ml, and all remaining PCBs (8, 15, 52, 70, 95, 101) of 0.008 ng/ml. Further details of the analytical methods have been reported elsewhere.[26,30] The percent of pooled DBS below LOD were <1% for DDE, and PCBs 8, 18, 101, and 153. All other chemicals had 2–4% below LOD except for PCB-15 which had 40% below LOD.

Outcome Measurements

Birthweight was obtained from birth certificates. Gestational age on birth certificates was estimated based on all available perinatal information including ultrasound data and the number of full weeks from the mother's last menstrual period to the birth of the infant. Size for gestational age was defined based on a representative U.S. national reference population of singletons.[35] Newborns who were in the $<10^{th}$ or $>90^{th}$ percentiles of weight for their gestational age were categorized as small or large for gestational age, respectively. In analyses of SGA and LGA, infants who were appropriate for gestational age (AGA) were used as the reference category ($10–90^{th}$ percentile). Infants with birthweights below 2,500 g were classified as low birthweight. Preterm birth was defined as birth before 37 weeks gestational age. Birth weight z-scores were calculated according to World Health Organization Child Growth Standards.[36] Birth length was reported on the baseline maternal questionnaire. Ponderal index was calculated as [birthweight (g)/length (cm^3) × 100].[37]

Statistical analysis

We examined distributions of POPs and covariates that were obtained from the maternal baseline questionnaire and birth certificates, and included maternal age (years), pre-pregnancy body mass index (weight in kg/height in m^2), race (white or non-white), smoking during pregnancy (yes/no), college education or more (yes/no), prior parity (yes/no), gestational weight gain (kg) and infant sex (male/female). Covariates were selected a priori

based on subject matter knowledge of associations with both the exposures and the outcomes. To ensure that the models were not over-adjusted, models were also run with minimal adjustment for maternal age.

Individual concentrations of each analyte were imputed using individual level covariates and the pooled pollutant measurement for that individual. Pooled values of pollutants retained the right skewness seen in individual level measurements; thus, a gamma distribution was assumed for the imputation.[38] The gamma distribution is similar in shape to lognormal but has been assumed in the pooling literature because of the convenient property where if the individual concentrations follow a gamma distribution, then the pools also follow a gamma distribution which lognormal lacks.[34,38–42] After employing multiple imputation (100 imputations) to obtain individual level pollutant exposures from pooled samples, we used linear and logistic regression to examine the associations between a 0.1-ng/mL increase in each POP and continuous outcomes and binary birth characteristics, respectively.[34,40] Values of pollutants that were below the limits of quantification were replaced by a near-zero constant, as consistent with EPA guidance. Models failed to converge for PCB-180 (likely due to higher levels of near-zero data for this pollutant) and the pollutant was dropped from the analysis. Pooling was accounted for in all analyses. To assess departures from linearity, analyses were performed with quartiles of chemical concentrations. Analyses were conducted in R version 3.3.2 (R Core Team, Vienna, Austria).

Results

Study population.

Infants in our study had mothers who averaged 30.8 (SD 6.0) years of age, were slightly overweight (mean BMI 26.9 (SD 6.7) kg/m^2) and gained an average of 17.3 kg (SD 1.1) during pregnancy (Table 1). The majority of mothers had at least a college degree (55.9%), were white (89.7%), and were nulliparous (66.5%). Infants' mean gestational age and birthweight were 38.8 weeks (SD 1.8) and 3399 grams (SD 552), respectively. The distribution of chemical concentrations from the pools are shown in Table 1 and from individual concentrations assuming a gamma distribution in Supplemental Table 1.

PCBs.—We observed small differences in birth weight associated with higher levels of several PCBs. In Table 2, per 0.1 ng/mL higher concentrations of PCB congeners 52 and 95 were associated with a slightly higher odds of large for gestational age birth (PCB-52 OR 1.02, 95% CI: 1.00, 1.03; PCB-95 OR 1.03, 95% CI 1.00, 1.05) after adjustment for confounders. Supplemental Table 2 shows similar odds ratios in models minimally adjusted for maternal age. Higher concentrations of these PCBs were associated also with lower odds of preterm birth (PCB-52 OR 0.92, 95% CI: 0.85, 0.99; PCB-95 OR 0.79; 0.65–0.96). Higher concentrations of PCB congeners 52, 70, and 138 were associated with slightly reduced odds of low birthweight (<2500g) (PCB-52 OR 0.98, 95% CI: 0.97, 0.99; PCB-70 OR 0.69, 95%CI: 0.53, 0.89; PCB-138 OR 0.98, 95% CI: 0.98, 0.99), which is consistent with the overall pattern of higher concentrations of PCBs being associated with larger birth size. Higher levels of PCB congeners 8, 52, 70, and 95 were associated with higher

continuous birthweights. (Table 3) However, there were no differences in ponderal index or continuous gestational age.

PBDE and p,p'-DDE.—Higher levels of PBDE-47 were associated with somewhat lower odds of large for gestational age (PBDE-47 OR 0.87, 95% CI: 0.74, 1.01), in contrast with results from PCBs. (Table 2) We did not observe associations between infant concentrations of *p,p'*-DDE and birth characteristics, although a one ug/L higher concentration of *p,p'*-DDE was positively associated with a slightly higher birthweight. (Table 3) Quartile results supported the linear assumption made (Supplemental Table 3).

Discussion

In this population-based birth cohort study, we observed that higher concentrations of some POPs as measured in newborn DBS were positively associated with birth weight after adjustment for confounders. For several PCB congeners, we observed increased odds of being born large for gestational age, against the context of decreased risks of being born preterm or low birth weight. Being born at a higher birthweight may have potential implications for future health, as higher birthweights and large for gestational age birth have been shown to be important risk factors for neonatal morbidity, as well as obesity and metabolic disorders later in life.[43,44] Our study used well-validated and innovative pooling methods to identify potential risks of several PCBs which had previously been less commonly studied. We believe this to be the first study to examine the relationship between POPs as measured in DBS and birth characteristics.

Studies of POPs measured in other biologic media such as serum or plasma have shown mixed results in relation to birthweight. These mixed results may be due to varying levels of exposure, heterogeneous study designs, measurement of a variety of chemical classes at different times in the reproductive cycle, in various specimens such as maternal blood[1,15,18,45–48], umbilical cord blood[6,8,49–52], placental tissue[50], and breast milk.[7,9] It is unclear which tissue or timing of exposure might best reflect the potential risk for adverse outcomes and in which direction.[53] Our use of a novel measurement of POPs concentrations in whole blood collected from DBS makes it somewhat difficult to compare our results with other studies utilizing other biologic media such as maternal blood, given the limited information on the partitioning of EDCs between maternal and infant blood. We are unaware of other studies that examine the association between POPs and birth characteristics using DBS. Numerous studies have documented fetal exposure to POPs through placental transfer.[53] Concentrations of POPs in infant cord blood are correlated with concentrations in maternal serum and are thought to be a proxy of fetal exposure.[53] However, some POPs that are detected in maternal serum are not readily detectable in cord blood and, therefore, fetuses' internal dose remained unknown.[50,52,53] Studies of background levels of POPs and birthweight have shown equivocal associations with birthweight in a variety of different PCB congeners.[1,3,5,7,11–15,18,21,53–55] To better compare with other studies, we examined each chemical individually rather than grouping by chemical composition. We made no assumptions about the direction of association, or potential interactions in mixtures. Our findings are similar to a study by Lignell, et al 2013, which found positive associations between prenatal exposure to PCBs and birthweight in a Swedish population with low-level

background exposure.[7] Lignell et al. did not examine PCB congeners 52 or 95, but did find a positive association with PCB-138, which we also identified. We observed a reduced risk of being born large for gestational age at higher levels of PBDE-47 exposure, which is consistent with effects of PBDE on the upper end of the birthweight distribution.[7] Newborns with higher concentrations of PBDE-47 had lower birthweight in our study (−34.8g, 95% CI: −1119.1, 49.6); however, there was imprecision in this estimate. Our study found stronger associations between POPs and low birthweight and birthweight in grams, than for SGA and birthweight z-score, which account for gestational age. We found little association between POPs and gestational age, however gestational age was generally slightly higher with increasing concentration of POPs. Higher levels of PCB-70 were associated with the largest increase in gestational age and the greatest increase in mean birthweight, but size measures incorporating gestational age showed less difference. This may indicate that some increases in birth size may be been in part related to longer gestational age."

While biological mechanisms linking these specific PCB congeners to LGA are unclear, numerous mechanistic *in vivo* and *in vitro* studies support the potential of POPs and endocrine disrupting chemicals (EDCs) to act as obesogens.[56–58] POPs can induce hormonal and epigenetic modifications leading to metabolic changes in the maternal uterus, programming the developing fetus towards a propensity for adipogenesis.[57] In *in vitro* models, some POPs have been shown to affect regulation of peroxisome proliferator activated receptor gamma (PPARy), the master regulator of adipogenesis.[58] Studies have also suggested that exposure to some POPs promote inflammation, mitochondrial dysfunction, lipid peroxidation, and oxidative stress.[56,57] POPs can also alter leptin signaling and modulate differentiation of adipocytes, impacting insulin sensitivity and adiposity.[59,60] While there is evidence that POPs can alter epigenetic programming, specific mechanisms linking epigenetic changes to obesogenic effects are not well characterized and require further study.[57] Given that POPs may exhibit different biological effects, we chose to examine each pollutant individually, making no assumptions about interaction or mixtures. While dioxin-like PCBs have been a source of health concern, our results showed that some non-dioxin like PCBs may also be of interest. Our study was able to address several limitations from prior studies of fetal POPs exposure with birth characteristics. First, through the use of newborn DBS, our study measures the circulating concentrations of pollutants directly from the newborns, which may be a better measure of fetal exposure than maternal measures. Measurement of POPs concentrations in pregnant women can be influenced by changes in blood volume expansion that occurs during pregnancy.[46] While statistical techniques (e.g., lipid adjustment) have been developed to account for blood volume expansion in analysis, if interpreted without caution these can lead to biased estimates of effect.[61] Second, our study used a pooled sampling strategy to allow analysis of samples that might not reach LOD if analyzed individually. Participants often choose to enroll in research studies because they desire their data and experience to be used to improve the health of their communities.[32] Through use of these pooled data techniques, our study improves the likelihood that all data collected from research participants can be used and helps keep ethical obligations to participants and their communities.[32]

Our study has several limitations. As SGA and intra-uterine growth restriction have maternal, placental, and fetal risk factors, it is unclear how different newborn/fetal blood

concentrations of POPs assessed at birth are from maternal blood concentrations and placental concentrations, and whether fetal blood concentrations are a direct cause of outcomes or a proxy for concentrations in other tissues. Also, because samples were taken from newborns, we were unable to estimate the developmental window-specific periods during which the fetus was exposed. However, POPs have long half-lives, and our measurements may represent cumulative exposure across the pregnancy. Some studies have hypothesized that the POPs exposure in early pregnancy (or even prior to pregnancy) may be a key window to determine health effects.[13] Maternal concentrations of POPs can vary across different developmental windows in pregnancy; however, POPs exposure is unlikely to be eliminated across the pregnancy.[46] Several studies have shown that concentrations of POPs in maternal serum can be fairly stable throughout pregnancy.[46,62] In studies of POPs in early pregnancy or prior to pregnancy, however, some POPs have been associated with lower birthweights and SGA birth.[13] It is possible that exposure to some POPs later in pregnancy may have the opposite effect, associating with larger birthweights and LGA. Early childhood exposure to POPs has been associated with increased risk of childhood obesity; however, these effects may differ by congener and further study is needed to test this developmental window hypothesis.[7,19,21,56,57] Exposure to POPs may occur through consumption of certain foods, and our results may be residually confounded by greater food consumption, despite adjustment for BMI. Our study used pooled sampling techniques and not individual-level pollutant data. While there is a loss of information when pooling, point estimates should closely resemble those obtained from individual level concentrations when reasonable distributional assumptions hold (e.g. symmetric or skewed).[34,38–40,42] Of note, most existing methods require pools be formed based on outcome status. However, we utilized and adapted methods to analyze our pools of mixed outcome and covariate status (wherein members of a pool may have different outcomes statuses).[34] Methods for analysis of pooled data have been demonstrated to have low bias; however, they are still subject to potential measurement error or biases consistent with an observational design. As our study is exploratory and hypothesis generating, we made multiple comparisons to identify potential associations for further study. Our study found more associations than would be expected by chance alone.

To our knowledge, this is the first large population-based birth cohort study to examine the relationship between POPs measured in newborn blood spots in relation to birth characteristics. Newborn DBS are widely collected, representing a potential proxy data source for maternal exposure to environmental pollutants during pregnancy, and may be useful when maternal markers are unavailable. Further study of newborn DBS in other populations may help clarify the relationships between POPs and birth weight. We observed that at background levels, many chemicals were associated with differences in birth characteristics, however in general the associations were of small magnitude and most congeners showed no relationship to birth size. Our findings provide evidence that neonatal exposure to POPs may be associated with larger birth size; however, further evidence is needed to confirm these findings and we recommend caution in interpretation of our results. Infant dried blood spots represent an important and underutilized source of data on neonatal exposure to environmental pollutants.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1.

Description of the Upstate KIDS Study (n=2065)

Maternal characteristics	Mean (SD) or n (%)	Min, Max
Age (years)	30.8 (6.0)	(14, 51)
Non-white race	213 (10.3%)	
College education	1155 (55.9%)	
Pre-pregnancy BMI (kg/m^2)	26.9 (6.7)	(14.1, 67.2)
Smoked during pregnancy	242 (11.7%)	
Nulliparous	1364 (66.5%)	
Gestational weight gain (kg)	17.3 (1.1)	(11.8, 19.1)
Infant Characteristics		
Gestational age (weeks)	38.8 (1.75)	(26, 42)
Birthweight (g)	3399.4 (552.3)	(720, 5255)
Male infant sex	1087 (52.6%)	
Low birthweight (<2500g)	106 (5.1%)	
Small for gestational age	158 (7.7%)	
Large for gestational age	215 (10.4%)	
Preterm (gestational age <37 weeks)	157 (7.7%)	
Pollutant concentrations (ng/mL), pooled level data		
p,p'-DDE	0.207 (0.202)	(0.013, 3.023)
PBDE-47	0.243 (0.275)	(0.0001, 2.092)
PCB-8	0.250 (0.292)	(0.0001, 2.788)
PCB-15	0.054 (0.155)	(0.0001, 1.989)
PCB-18	0.184 (0.119)	(0.0001, 0.926)
PCB-28	0.128 (0.117)	(0.00002, 0.977)
PCB-52	0.082 (0.068)	(0.0001, 0.588)
PCB-70	0.061 (0.050)	(0.00001, 0.644)
PCB-95	0.106 (0.080)	(0.0001, 0.625)
PCB-101	0.111 (0.084)	(0.0001, 0.702)
PCB-138	0.080 (0.090)	(0.0001, 0.918)
PCB-153	0.109 (0.107)	(0.0001, 0.804)

Mean (SD) when units indicated or n (%) otherwise

Table 2.

Adjusted odds ratios and their 95% confidence interval for each birth outcome measure per 0.1 ng/mL increase in chemical concentration

Outcome	Odds Ratio	Lower 95% CI	Upper 95% CI	p-value	Odds Ratio	Lower 95% CI	Upper 95% CI	p-value	Odds Ratio	Lower 95% CI	Upper 95% CI	p-value	Odds Ratio	Lower 95% CI	Upper 95% CI	p-value
	Large for gestational age (>90th percentile)				Small for gestational age (<10th percentile)				Low birthweight (<2500g)				Preterm birth (gestational age <37 weeks)			
pp'-DDE	0.993	0.972	1.014	0.48	0.99	0.96	1.02	0.46	0.98	0.94	1.01	0.15	0.99	0.97	1.02	0.62
PBDE-47	0.988	0.975	1.001	0.08	0.99	0.98	1.00	0.14	1.00	0.98	1.02	0.87	0.99	0.98	1.00	0.17
PCB8	1.01	0.99	1.03	0.25	0.99	0.98	1.00	0.19	1.00	0.97	1.04	0.82	1.00	0.98	1.01	0.44
PCB15	1.00	1.00	1.01	0.49	0.99	0.996	0.999	0.03	1.00	0.99	1.00	0.48	1.00	0.99	1.00	0.004
PCB18	0.99	0.98	1.01	0.22	0.99	0.96	1.03	0.69	1.01	0.98	1.05	0.42	0.99	0.98	1.01	0.46
PCB28	1.00	0.99	1.01	0.72	0.99	0.98	1.01	0.42	0.99	0.98	1.01	0.30	0.99	0.98	1.00	0.29
PCB52	1.02	1.00	1.03	0.02	0.99	0.98	1.01	0.42	0.98	0.97	0.99	0.0003	0.99	0.98	1.00	0.02
PCB70	1.00	0.99	1.02	0.66	0.98	0.96	1.00	0.06	0.69	0.53	0.89	0.005	0.99	0.97	1.00	0.04
PCB95	1.03	1.00	1.05	0.02	0.99	0.98	1.01	0.36	1.00	0.97	1.03	0.84	0.98	0.96	1.00	0.03
PCB101	1.01	0.99	1.02	0.55	1.00	0.98	1.02	0.84	0.99	0.96	1.01	0.32	0.99	0.96	1.01	0.37
PCB138	0.99	0.99	1.00	0.10	0.99	0.98	1.00	0.10	0.98	0.98	0.99	0.0007	0.99	0.98	1.00	0.06
PCB153	1.01	0.99	1.03	0.12	0.99	0.97	1.01	0.48	0.98	0.96	1.01	0.15	0.99	0.98	1.01	0.27

All models adjusted for maternal age, pre-pregnancy BMI, race/ethnicity, smoking, education, parity, and infant sex

Table 3.

Adjusted mean changes and their 95% confidence interval for each birth outcome measure per 0.1 ng/mL increase in pollutant concentration

	Birthweight (grams)				Birth weight z-score				Ponderal index (kg/m^3)				Gestational age (weeks)			
	M.D.	Lower 95% CI	Upper 95% CI	p value	M.D.	Lower 95% CI	Upper 95% CI	p value	M.D.	Lower 95% CI	Upper 95% CI	p value	M.D.	Lower 95% CI	Upper 95% CI	p value
pp'-DDE	13.5	−0.81	28.0	0.06	0.0002	−0.02	0.02	0.99	−0.0008	−0.01	0.01	0.88	0.03	−0.01	0.07	0.15
PBDE-47	0.3	−9.9	10.5	0.95	−0.01	−0.03	0.007	0.26	0.002	−0.006	0.01	0.60	0.01	−0.02	0.04	0.40
PCB8	13.6	4.3	22.9	0.004	0.007	−0.009	0.02	0.41	0.002	−0.005	0.009	0.54	0.02	−0.01	0.05	0.23
PCB15	6.6	−5.8	19.0	0.30	0.009	−0.01	0.03	0.42	−0.001	−0.01	0.008	0.84	0.006	−0.03	0.05	0.75
PCB18	−1.0	−30.6	28.7	0.95	−0.03	−0.07	0.009	0.14	−0.002	−0.02	0.01	0.79	0.007	−0.06	0.07	0.82
PCB28	2.3	−20.4	24.9	0.84	−0.004	−0.04	0.03	0.81	−0.001	−0.02	0.01	0.86	0.03	−0.03	0.09	0.35
PCB52	38.6	5.2	72.0	0.02	0.03	−0.04	0.09	0.45	0.007	−0.02	0.04	0.61	0.07	−0.04	1.07	0.21
PCB70	43.4	−3.7	90.5	0.07	−0.003	−0.08	0.08	0.95	0.031	−0.004	0.07	0.08	1.00	−0.05	2.05	0.19
PCB95	36.4	5.6	67.3	0.02	0.004	−0.05	0.06	0.89	0.005	−0.02	0.03	0.70	0.06	−0.03	1.05	0.18
PCB101	25.6	−5.5	56.7	0.11	−0.007	−0.06	0.04	0.79	0.009	−0.01	0.03	0.41	0.08	−0.02	1.07	0.11
PCB138	11.9	−15.4	39.1	0.39	−0.009	−0.05	0.03	0.70	0.006	−0.01	0.02	0.51	0.04	−0.04	1.02	0.23
PCB153	10.3	−13.5	34.0	0.40	−0.005	−0.04	0.03	0.82	0.005	−0.02	0.03	0.65	0.01	−0.07	0.08	0.90

Adjusted for maternal age, pre-pregnancy BMI, race/ethnicity, smoking, education, parity, and infant sex

M.D., mean difference