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# Prenatal Exposure to Polychlorinated Biphenyls and Body Fatness in Girls

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## Abstract

Polychlorinated biphenyls (PCBs) are synthetic, organochlorine compounds previously used in industrial processes. Although banned in 1980's across Europe, these chemicals persist in the environment and are associated with adverse health outcomes in children. We investigated the association between *in utero* concentrations of PCBs and girls' body fatness. Concentrations of various PCB congeners (PCB 118, PCB 138, PCB 153, PCB 170, and PCB 180) were measured in maternal serum samples collected in the early 1990's. Body fatness was measured in the daughters at 9 y of age using body mass index (BMI) and dual-energy x-ray absorptiometry (DXA) for percent body fat. Using multivariable linear regression, we explored associations between prenatal PCB congener concentrations and body fatness outcomes. Among 339 mother-daughter dyads, the median and interquartile range (IQR) for PCB congeners ranged between 15.0 ng $g^{-1}$ (11.0–20.8) for PCB 118 to 64.6 ng $g^{-1}$ (48.6–86.3) for PCB 153. Among daughters, the median was 27.5% (21.7–34.6) for percent body fat, 39.6% (36.4–43.5) for percent trunk fat, 4.9 kg $m^{-2}$ (3.5–7.0) for fat mass index and 18.1 kg $m^{-2}$ (16.3–20.6) for body mass index. Multivariable-adjusted regression analyses showed little or no association between prenatal PCB concentrations with daughters' body fatness measures. Prenatal concentrations of PCB congeners were not strongly associated with measures of body fatness in girls.

### Keywords

ALSPAC; polychlorinated biphenyl compounds; weight; body fat; children

## 1. Introduction

Polychlorinated biphenyls (PCBs) are synthetic, organochlorine compounds previously used in industrial processes as insulators and coolants in electrical equipment (Ross, 2004).

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PCBs comprise 209 congeners differing by chlorination degree and substitution pattern (Ross, 2004). PCB manufacture was restricted in the 1970s, and ultimately banned in most countries, including the United Kingdom (UK) and the United States (US) (Ross, 2004; European Commission, 2016). Nevertheless, PCB exposure is still a concern because of continued use in existing closed electrical and hydraulic systems, the chemical stability of PCBs and persistence in the environment, and the adverse health outcomes related to even low-level exposure (USDHHS 2000; Cupul-Uicab et al., 2013; 2016). The half-life for PCB 138 is approximately three, and for highly chlorinated congeners, (e.g., PCBs 153, 180) 7–9 y (Grandjean et al., 2008).

Humans can be exposed to PCBs from inhalation or ingestion of contaminated sources (USDHHS, 2000). Maternal ingestion of PCBs may lead to fetal exposure fetus through the placenta and the infant through breastmilk (Blanck et al., 2002; Karmaus et al., 2002b; DiVall, 2013; Agay-Shay et al., 2015). PCBs are endocrine disrupting compounds that mimic the action of natural hormones such as thyroid hormone and estrogens; thus, early exposure can profoundly affect growth and development (Dirinck et al., 2011; DiVall, 2013; Agay-Shay et al., 2015). Prenatal PCB exposure has been associated with preterm birth and lower birthweight (Guo et al., 1995; Patandin et al., 1999; Ribas-Fito et al., 2001); however, evidence for an association between prenatal exposure and body fatness later in life is conflicting (Blanck et al., 2002; Hatch et al., 2010; DiVall, 2013). Moreover, sexually dimorphic responses have been observed (Gladen et al., 2000; Karmaus et al., 2002a). Among girls, prenatal exposures to PCBs have been positively associated with childhood weight in at least two studies (Gladen et al., 2000; Agay-Shay et al., 2015). In contrast, another study observed that prenatal PCB exposure above 5 ppb was associated with reduced weight adjusted for height in girls (Blanck et al., 2002). Given these inconsistencies, our aim was to use data from a prospective birth cohort, the Avon Longitudinal Study of Parents and Children (ALSPAC), in the UK, to investigate the association between prenatal serum PCB concentrations and body fatness in 9 year-old girls.

## 2. Materials and methods

### Population

ALSPAC is a prospective birth cohort that enrolled 14,541 pregnant women residing in Avon, UK with expected delivery dates between April 1991 and December 1992 (Golding et al., 2001; Ness, 2004; Boyd et al., 2013; Fraser et al., 2013). The cohort included 14,062 live births. ALSPAC's goal was to study the effects of various factors, including genetics, lifestyle, and environment, on the health and development of children. The study website contains details of all the data that is available through a fully searchable data dictionary and variable search tool (http://www.bris.ac.uk/alspac/researchers/our-data/). Participants in the current study were from an ancillary study of puberty including singleton, female participants at the age of 13 y in 2004–2005. Two valid assessments of pubertal status between the ages of 8 and 13 were returned by 3,682 girls. From this group, a nested case-control study of 448 mother-daughter dyads was designed to explore the effects of prenatal environmental exposures on selected health outcomes (Christensen et al., 2011). Ethical approval for the study was obtained from the ALSPAC Ethics and Law Committee and

the Local Research Ethics Committees. Informed consent for the use of data collected via questionnaires and clinics was obtained from participants following the recommendations of the ALSPAC Law and Ethics Committee, the Local Research Ethics Committees and the Centers for Disease Control and Prevention (CDC) Institutional Review Board at the time. Consent for biological samples was collected in accordance with the Human Tissue Act (2004).

## Data Collection

At enrollment, pregnant mothers self-reported demographic, health, and lifestyle information. After birth, mothers reported birth characteristics for their children and birth weight and gestational age were abstracted from obstetric records. Detailed information has been collected longitudinally on the children in research clinics and using guardian- and self-reported questionnaires (Golding et al., 2001; Golding and Team, 2004).

Daughters' total and regional body fat, lean mass, and bone mass were measured using a Lunar Prodigy Dual X-ray Absorptiometry (DXA) scanner (GE Medical Systems Lunar, Madison, WI, USA) at age 9 y. Our body fatness outcomes included DXA-total body fat percentage (%BF), DXA-trunk fat percentage (%TF) [(trunk fat (g)/total fat (g)) *100], fat mass index (FMI) [total fat mass (kg)/height $(m)^2$] and body mass index (BMI) [weight (kg)/height $(m^2)$]. The current analysis included 339 girls who completed DXA scans at 9 y and had maternal serum PCB congener concentrations.

## Laboratory Analyses

Pregnant mothers provided a single prenatal blood sample at enrollment, which was processed and frozen for later analysis. Median gestational age at collection was 15 weeks with an interquartile range (IQR) of 10–28 weeks. Serum samples were transported to the National Center for Environmental Health (NCEH) at the Centers for Disease Control and Prevention (CDC) in Atlanta, GA for analysis. Samples were analyzed for PCB congeners using solid phase extraction followed by gas chromatography isotope dilution high resolution mass spectrometry (Sjodin et al., 2004). Laboratory analyses included low- and high-concentration pooled standards, reagent blanks, and study samples. Values for measurements below the limit of detection (LOD) were calculated by dividing the LOD by the square root of 2. PCB congeners 138 and 158 could not be distinguished from each other and were considered a single congener concentration (referred to as PCB 138) in all analyses.

## Statistical Analyses

Based on the literature, we chose to analyze data for concentrations of five PCB congeners (PCB 118, PCB 138, PCB 153, PCB 170, and PCB 180) (Karmaus et al., 2002a; Dirinck et al., 2011; Cupul-Uicab et al., 2013; Agay-Shay et al., 2015) expressed as lipid-adjusted concentrations (ng $g^{-1}$ lipid). We plotted laboratory data, looked for outliers and calculated measures of central tendency and distributions for demographic characteristics. To account for the sampling scheme used for participant selection in the nested case-control study, we constructed stratum-weighted linear regression models, weighting (weight=15.1) the girls who attained menarche at 11.5 y or older (a random sample of the ALSPAC girls who

attained menarche ≥ 11.5 y of age) and assigned a weight of 1 to girls who attained menarche at <11.5 y (all who attained menarche <11.5 y in ALSPAC are included) (Richardson et al., 2007). Regression models were used to evaluate the associations between each maternal PCB congener with daughter's body fatness measures after adjustment for maternal pre-pregnancy BMI (continuous), breastfeeding status (any v. none), and birthweight (continuous). Additional covariates were considered, including maternal age (continuous), race (Caucasian v. other as there are few non-Caucasians in ALSPAC), smoking status (yes/no during pregnancy), parity (continuous), gestational age at blood collection (continuous), previous live birth (yes/no), and daughters' preterm delivery status (gestational age <37 wk, yes/no). We also explored nonlinear relationships by adding quadratic or square root terms for congeners to models. None of these improved model fit or led to meaningful changes in the relationship between body fatness and PCBs; thus, they were not retained in final models. Lastly, we considered potential effect modification by maternal educational status by including these variables and their cross-product terms with each of the PCBs in their respective models. Maternal educational status was coded into three categories (low, medium, high). For analysis, not attaining General Certificates of Secondary Education (GCSEs, at 16 y of age) was coded as "low", obtaining GCSEs as "medium," GCSEs and/or vocational training with additional education (e.g., University) was considered "high." Statistical analyses were performed using Statistical Analysis Systems (SAS version 9.3) software.

## 3. Results

Table 1 presents median maternal prenatal PCB levels (ng $g^{-1}$ lipid) by sample demographic characteristics. Overall, the sample was primarily Caucasian (95.0% of mothers), the majority of mothers were normal weight before pregnancy, and nearly half reported a previous live birth. The majority of mothers did not smoke during pregnancy and more than 75% reported breastfeeding.

PCB analytes were detected in 98% of samples. Concentrations (ng $g^{-1}$) of PCB 153 were highest (median 64.6; IQR: 48.6–86.3) and PCB 118 were lowest (median 15.0; IQR: 11.0–20.8). Concentrations of congeners tended to be higher among older, more educated mothers and non-smokers. Among daughters, the median was 27.5% (21.7–34.6) for %BF, 39.6% (36.4–43.5) for %TF, 4.9 kg $m^{-2}$ (3.5–7.0) for FMI, 18.1 kg $m^{-2}$ (16.3–20.6) for BM, and 35.8 kg (31.0–42.4) for weight.

Overall, there was no clear evidence that prenatal PCB levels were associated with daughters' body fatness measures at age 9 (Table 2). There was one modest and some marginal inverse associations with wide confidence intervals seen between prenatal PCB concentrations and measures of daughters' body fatness for PCBs 180, 170 and 153.

## 4. Discussion

PCBs mimic the action of natural hormones such as estrogens and thyroid hormones (McKinney and Waller, 1994) and are theorized to promote adipogenesis and lipid accumulation, even at low-levels, through interactions with various receptors (e.g., sex

steroid or corticosteroid receptors) or inducing epigenetic modifications in obesity-related genes (McKinney and Waller, 1994; Dirinck et al., 2011; DiVall, 2013). Though it is biologically plausible that PCB exposure could affect body fatness, the literature presents mixed results. Differences in the populations and PCB congeners examined, use of summary measures and the timing of assessments may in part explain the divergent results across studies. In a study including a subset of the U.S. Collaborative Perinatal Project, where mothers were recruited between 1959–1965, researchers evaluated a summary measure of PCB congeners and observed no clear association between total PCBs with BMI among 1915 children aged 0 to 7 (Cupul-Uicab et al., 2013). In a population of both girls and boys aged 10 to 15, a positive association was observed for a summary measure of PCB exposure with increased weight, but the association was limited to girls (Gladen et al., 2000). In another study of both girls and boys, prenatal exposures to PCB congeners 138 and 180 were positively associated with being overweight at age 7 (Agay-Shay et al., 2015). Conversely, an inverse association was found between prenatal PCB exposures measured between 1976–1979 and weight (Blanck et al., 2002). Concentrations above 5 parts per billion (substantially higher than our population) were associated with reduced weight adjusted for height among 308 daughters aged 5 to 24 (Blanck et al., 2002). Tang-Peronard and colleagues (Tang-Peronard et al., 2014) examined the association between prenatal exposure to PCBs in blood collected between 1997–2000 with youth obesity at 5 and 7 y among 656 mother-child dyads in the Faroe Islands. PCB congeners 138, 153, and 180 were summed for analysis. Prenatal PCB exposure was positively associated with change in BMI between 5 and 7 y and with obesity at age 7, but only among daughters of overweight mothers.

The commercial production of PCBs in the United States was restricted in the early 1970s and ultimately banned by the US Environmental Protection Agency in 1979 (Ross, 2004). Due to the decrease in production, strict regulation of use, and cleanup of contaminated sites, the overall US population has limited exposure to PCBs in fish and other foods and relatively low PCB blood levels (Ross, 2004). PCBs are persistent in the environment and can bioaccumulate; therefore, to decrease the risk of prenatal PCBs exposure, pregnant women could decrease their consumption of living organisms, such as fish, proximal to environmental sources.

This study has limitations. PCBs were measured once during pregnancy and daughters' levels were not measured postnatally. Our study population's demographic characteristics somewhat limit the generalizability of our findings. Data from the US National Health and Nutrition Survey (NHANES) 2003–2004 cycle for females reported higher median (95% confidence interval) concentrations of PCB 118 of 36 ng $g^{-1}$ (30–43), PCB 138 of 98 ng $g^{-1}$ (85–115), PCB 153 of 138 ng $g^{-1}$ (113–149), PCB 170 of 40 ng $g^{-1}$ (34–44), and PCB 180 of 110 ng $g^{-1}$ (94–123)(CDC, 2018), than observed in our study population. Our analyses were conducted on a sample of mother-daughter dyads selected for an ancillary study of pubertal development and a subset of daughters completed DXA scans. We used weighted linear regression models to adjust for the sampling scheme for this analysis. Our sample was relatively representative of the overall cohort, although mothers tended to be older and more educated (Supplemental Table 1). We previously reported that the maternal characteristics for girls included in the ancillary sample were similar to the group of girls enrolled in the

cohort (Christensen et al., 2011; Hartman et al., 2017). Finally, data were not available to analyze PCBs among mothers of boys.

## 5. Conclusion

In this study, prenatal concentrations of selected PCB congeners were not strongly associated with body fatness in girls.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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**Table 1.**

Frequency distribution and lipid adjusted maternal serum concentrations (ng $g^{-1}$ lipid) of selected polychlorinated biphenyl (PCB) analytes in a sample of British girls (n=339)

| | Frequency n (%) | PCB 118 Median (IQR)[1] | PCB 138 Median (IQR) | PCB 153 Median (IQR) | PCB 170 Median (IQR) | PCB 180 Median (IQR) |
|---|---|---|---|---|---|---|
| Overall | 339 (100) | 15.0 (11.0–20.8) | 41.2 (30.5–54.3) | 64.6 (48.6–86.3) | 19.0 (14.5–25.6) | 45.7 (33.6–61.4) |
| Maternal pre-pregnancy BMI | | | | | | |
| Underweight (<18.5) | 15 (4.4) | 15.2 (8.0–24.5) | 52.3 (42.2–66.2) | 84.8 (76.9–103.7) | 24.6 (22.1–27.0) | 61.6 (56.3–86.9) |
| Normal (18.5–24.9) | 217 (64.0) | 15.3 (11.1–20.7) | 42.6 (31.0–55.2) | 68.3 (52.1–88.1) | 20.3 (15.8–26.4) | 48.5 (36.6–62.1) |
| Overweight (25.0–29.9) | 52 (15.3) | 17.0 (11.0–21.9) | 40.8 (28.7–51.2) | 59.0 (45.1–78.2) | 16.1 (13.8–21.5) | 37.8 (30.0–55.6) |
| Obese (≥30.0) | 23 (6.8) | 15.5 (12.2–20.5) | 35.1 (26.7–50.6) | 54.3 (40.7–72.2) | 13.2 (11.9–18.8) | 31.5 (25.4–44.9) |
| Missing | 32 (9.4) | 12.8 (10.6–16.3) | 35.5 (28.2–49.1) | 56.4 (41.6–72.1) | 16.4 (11.6–21.6) | 37.4 (25.4–50.3) |
| Maternal education[2] | | | | | | |
| Low | 51 (15.0) | 14.2 (10.3–19.8) | 37.1 (30.4–52.1) | 59.7 (45.8–81.8) | 17.9 (13.6–22.4) | 43.9 (31.2–59.3) |
| Medium | 107 (31.6) | 13.5 (10.1–18.3) | 35.4 (28.2–47.7) | 56.2 (44.4–74.3) | 16.7 (13.2–21.5) | 38.0 (30.1–49.7) |
| High | 161 (47.5) | 17.7 (13.0–23.1) | 47.6 (36.4–58.3) | 74.4 (57.8–95.6) | 21.4 (16.9–27.9) | 54.4 (40.6–66.6) |
| Missing | 20 (5.9) | 12.3 (10.0–15.3) | 35.0 (27.4–51.2) | 52.7 (37.8–72.1) | 15.6 (10.0–20.4) | 35.9 (21.7–48.9) |
| Maternal Race | | | | | | |
| White | 322 (95.0) | 15.0 (11.0–20.8) | 41.3 (30.5–54.1) | 64.6 (48.7–86.3) | 19.0 (14.6–25.6) | 45.9 (34.2–61.4) |
| Nonwhite | 7 (2.1) | 20.4 (12.0–25.5) | 50.1 (30.4–70.7) | 78.1 (41.5–103.9) | 17.9 (10.3–26.9) | 45.7 (23.0–61.7) |
| Missing | 10 (2.9) | 12.5 (12.2–15.8) | 36.6 (30.4–52.5) | 59.1 (47.1–74.4) | 17.3 (13.6–21.1) | 40.0 (33.3–50.4) |
| Maternal age at delivery | | | | | | |
| <25 y | 64 (18.9) | 10.6 (8.8–13.8) | 29.7 (23.5–37.6) | 44.2 (35.0–54.8) | 13.2 (10.1–16.0) | 29.0 (22.6–37.5) |
| 25–29 y | 129 (38.1) | 14.3 (11.3–19.6) | 38.4 (31.0–47.2) | 59.8 (48.1–74.1) | 17.4 (14.4–21.2) | 40.7 (33.3–50.4) |
| ≥30 y | 146 (43.1) | 18.6 (14.2–25.0) | 52.1 (40.4–63.5) | 81.9 (65.1–105.5) | 23.8 (19.2–30.1) | 59.2 (46.8–73.8) |
| Maternal Smoking (any smoking during pregnancy) | | | | | | |
| Yes | 69 (20.4) | 12.7 (9.4–17.3) | 38.3 (28.1–52.5) | 60.8 (46.0–78.6) | 17.9 (13.4–22.6) | 40.2 (31.1–59.3) |
| No | 270 (79.6) | 15.9 (12.0–21.5) | 41.9 (31.2–54.3) | 65.4 (49.3–88.1) | 19.4 (14.7–26.0) | 46.8 (35.3–62.1) |
| Previous Live Birth | | | | | | |
| Yes | 166 (49.0) | 15.0 (10.8–21.5) | 41.1 (31.2–54.3) | 66.9 (50.0–86.3) | 20.0 (15.7–26.2) | 48.5 (36.6–62.1) |

| | Frequency n (%) | PCB 118 Median (IQR)[1] | PCB 138 Median (IQR) | PCB 153 Median (IQR) | PCB 170 Median (IQR) | PCB 180 Median (IQR) |
|---|---|---|---|---|---|---|
| No | 160 (47.2) | 15.9 (12.0–20.9) | 43.9 (30.5–55.6) | 64.6 (46.9–89.3) | 18.3 (13.7–25.3) | 44.0 (31.7–61.5) |
| Missing | 13 (3.8) | 10.8 (9.8–12.2) | 34.5 (26.3–39.3) | 59.7 (37.1–64.6) | 17.7 (10.3–19.1) | 43.6 (23.9–44.3) |
| Low Birth Weight (<2,500 g at delivery) | | | | | | |
| Yes | 16 (3.2) | 20.9 (14.5–25.8) | 53.4 (46.1–63.4) | 84.0 (61.4–106.6) | 20.7 (17.6–31.4) | 53.1 (38.2–76.9) |
| No | 323 (96.8) | 14.8 (10.9–20.4) | 41.0 (30.4–53.9) | 63.7 (48.1–85.5) | 19.0 (14.4–25.0) | 45.2 (33.3–60.8) |
| Preterm Delivery (<37 wk gestation) | | | | | | |
| Yes | 11 (5.2) | 17.2 (12.2–20.9) | 48.7 (39.3–67.9) | 69.7 (62.8–120.8) | 19.4 (18.4–35.7) | 46.8 (39.0–89.7) |
| No | 328 (96.8) | 14.9 (11.0–20.7) | 41.2 (30.4–54.1) | 64.1 (48.3–86.2) | 18.9 (14.4–25.2) | 45.6 (33.3–61.1) |
| Ever Breastfed | | | | | | |
| Yes | 262 (77.3) | 15.1 (11.3–22.0) | 42.7 (31.2–56.9) | 67.8 (50.0–90.0) | 20.1 (15.6–26.5) | 47.3 (35.7–62.3) |
| No | 58 (17.1) | 13.1 (9.8–18.8) | 36.3 (27.9–48.3) | 54.4 (44.0–75.8) | 15.8 (13.1–21.9) | 36.2 (29.2–51.9) |
| Missing | 19 (5.6) | 16.1 (14.2–19.1) | 40.4 (33.8–52.5) | 60.5 (53.0–81.8) | 16.1 (14.3–21.9) | 41.2 (33.3–57.8) |
| Menarche (y) | | | | | | |
| ≥11.5 | 181 (53.4) | 14.8 (11.3–20.8) | 44.3 (31.4–55.2) | 68.6 (50.0–88.1) | 20.0 (15.4–26.4) | 48.1 (36.1–62.3) |
| <11.5 (early) | 158 (46.6) | 15.3 (10.8–20.7) | 39.6 (30.2–52.8) | 60.3 (47.7–84.5) | 17.9 (13.9–24.4) | 44.0 (31.5–59.3) |

[1] IQR=interquartile range

[2] Basic level of General Certificates of Secondary Education completed around 16 y of age

**Table 2.**

Adjusted Regression coefficients (β)[*,**] for associations between selected prenatal polychlorinated biphenyls concentrations and measures of body fatness in girls at age 9

| Analyte (ng g-1) | DXA-total body fat (%) | | | DXA-Fat Mass Index (kg/m$^2$) | | | DXA-trunk fat (%) | | | BMI (kg/m$^2$) | | | Weight (kg) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | β | 95% CI | p | β | 95% CI | p | β | 95% CI | p | β | 95% CI | p | β | 95% CI | p |
| **PCB 118** | | | | | | | | | | | | | | | |
| **Model 1** * | −0.006 | −0.108–0.095 | 0.90 | −0.005 | −0.035–0.025 | 0.74 | −0.002 | −0.059–0.063 | 0.95 | −0.013 | −0.049–0.023 | 0.49 | −0.033 | −0.126–0.060 | 0.49 |
| **Model 2**** | −0.005 | −0.104–0.094 | 0.92 | −0.005 | −0.034–0.024 | 0.73 | −0.011 | −0.049–0.071 | 0.72 | −0.014 | −0.049–0.021 | 0.45 | −0.029 | −0.120–0.061 | 0.53 |
| **PCB 138** | | | | | | | | | | | | | | | |
| **Model 1** * | −0.037 | −0.085–0.011 | 0.13 | −0.010 | −0.025–0.004 | 0.14 | −0.014 | −0.043–0.014 | 0.33 | −0.013 | −0.030–0.004 | 0.14 | −0.035 | −0.079–0.009 | 0.12 |
| **Model 2**** | −0.038 | −0.085–0.009 | 0.11 | −0.011 | −0.025–0.003 | 0.12 | −0.010 | −0.039–0.019 | 0.49 | −0.013 | −0.030–0.003 | 0.12 | −0.032 | −0.075–0.011 | 0.14 |
| **PCB 153** | | | | | | | | | | | | | | | |
| **Model 1** * | −0.029 | −0.061–0.003 | 0.08 | −0.008 | −0.017–0.002 | 0.11 | −0.016 | −0.035–0.003 | 0.10 | −0.009 | −0.020–0.003 | 0.13 | −0.026 | −0.055–0.004 | 0.09 |
| **Model 2**** | −0.028 | −0.060–0.004 | 0.08 | −0.008 | −0.017–0.001 | 0.10 | −0.012 | −0.032–0.007 | 0.21 | −0.009 | −0.021–0.002 | 0.11 | −0.024 | −0.053–0.005 | 0.11 |
| **PCB 170** | | | | | | | | | | | | | | | |
| **Model 1** * | −0.093 | −0.208–0.022 | 0.11 | −0.026 | −0.060–0.008 | 0.13 | −0.066 | −0.135–0.003 | 0.06 | −0.028 | −0.069–0.012 | 0.17 | −0.091 | −0.197–0.014 | 0.09 |
| **Model 2**** | −0.095 | −0.210–0.021 | 0.11 | −0.026 | −0.061–0.008 | 0.12 | −0.055 | −0.126–0.015 | 0.12 | −0.029 | −0.070–0.013 | 0.17 | −0.078 | −0.183–0.028 | 0.15 |
| **PCB 180** | | | | | | | | | | | | | | | |
| **Model 1** * | −0.044 | −0.090–0.003 | 0.06 | −0.012 | −0.025–0.002 | 0.09 | −0.031 | −0.059– −0.003 | **0.03** | −0.013 | −0.029–0.004 | 0.13 | −0.038 | −0.080–0.004 | 0.08 |
| **Model 2**** | −0.045 | −0.091–0.001 | 0.06 | −0.012 | −0.026–0.001 | 0.07 | −0.027 | −0.055–0.001 | 0.06 | −0.013 | −0.030–0.003 | 0.11 | −0.035 | −0.077–0.007 | 0.10 |

*Per unit (ng g$^{-1}$ lipid) increase in analyte. Model 1 adjusts for sampling design, continuous prepregnancy BMI (kg m$^{-2}$), and maternal education category (n=297 with complete data for DXA variables, 290 for BMI)

**Model 2 for each PCB analyte is additionally adjusted for breastfeeding status (n=282 with complete data for DXA variable, 275 for BMI)