Polybrominated diphenyl ethers (PBDEs) are chemical additives used as flame retardants in commercial products. PBDEs are bioaccumulative and persistent and have been linked to several adverse health outcomes.

**Objectives:** This study leverages an ongoing pregnancy cohort to measure PBDEs and PBDE metabolites in serum collected from an understudied population of pregnant women late in their third trimester. A secondary objective was to determine whether the PBDEs or their metabolites were associated with maternal thyroid hormones.

**Methods:** One hundred forty pregnant women > 34 weeks into their pregnancy were recruited into this study between 2008 and 2010. Blood samples were collected during a routine prenatal clinic visit. Serum was analyzed for a suite of PBDEs, three phenolic metabolites (i.e., containing an –OH moiety), and five thyroid hormones.

**Results:** PBDEs were detected in all samples and ranged from 3.6 to 694 ng/g lipid. Two hydroxylated BDE congeners (4’-OH-BDE 49 and 6-0H-BDE 47) were detected in > 67% of the samples. BDEs 47, 99, and 100 were significantly and positively associated with free and total thyroxine (T4) levels and with total triiodothyronine levels above the normal range. Associations between T4 and PBDE levels remained after controlling for smoking status, maternal age, race, gestational age, and parity.

**Conclusions:** PBDEs and OH-BDEs are prevalent in this cohort, and levels are similar to those in the general population. Given their long half-lives, PBDEs may be affecting thyroid regulation throughout pregnancy. Further research is warranted to determine mechanisms through which PBDEs affect thyroid hormone levels in developing fetuses and newborn babies.

**Keywords:** flame retardants, OH-BDEs, PBDEs, pregnancy, thyroid hormones. Environ Health Perspect 119:1454–1459 (2011). http://dx.doi.org/10.1289/ehp.1003235 [Online 29 June 2011]
Materials and Methods

Participant recruitment. Participants were recruited from within an ongoing observational prospective cohort study assessing the joint effect of social, environmental, and host factors on pregnancy outcomes [the Healthy Pregnancy, Healthy Baby (HPHB) Study] (Burgette and Reiter 2010; Miranda et al. 2010; Swamy et al. 2011). The HPHB study enrolls pregnant women from the Duke Obstetrics Clinic and the Durham County Health Department Prenatal Clinic. Women receiving prenatal care at these sites were eligible to participate if they were at least 18 years of age, English literate, between 18 and 28 weeks of gestation at study enrollment, lived in Durham County, were planning on delivering at Duke University Medical Center, and did not have a multiple gestation or any known fetal genetic or congenital abnormalities. All patients > 34 weeks of gestation enrolled in the HPHB study and who were routine patients at the Durham County Health Department’s Prenatal Clinic at the Lincoln Community Health Center (Durham, NC) between September 2008 and June 2010 were eligible to participate in the present study.

The choice of this clinic ensured a predominantly African-American study population. Women were approached during their routine third trimester laboratory visit (35–36 weeks) for participation in this study. Of all women approached for this study, > 97% agreed to participate. After women gave informed consent, samples were collected during the third trimester of pregnancy (> 34 weeks of gestation). All aspects of this study were carried out in accordance with a human subjects research protocol approved by the Duke University Institutional Review Board.

Sample collection. Consenting women filled out a short questionnaire that documented information regarding their personal, diet, and lifestyle characteristics. Two tubes of blood were collected during the clinic visit: one 4.5-mL tube (BD Vacutainer plasma separation with lithium heparin) and one 10-mL tube (BD Vacutainer serum separator). The tubes were allowed to sit for several hours in a refrigerator and were later centrifuged (within 24 hr) at 3,500 RMP for 5 min to isolate the plasma and serum, respectively. The 10-mL tubes (~ 4–5 g serum) were stored at –20°C until analysis. The smaller tubes were sent to the clinical laboratory at Duke University Hospital for thyroid hormone analysis within 8 hr of centrifugation.

Thyroid hormone analysis and lipid measurement. Plasma samples were analyzed by the clinical laboratory at Duke University Hospital (Durham, NC). TSH was measured using the Access Total T3 assay (Baxter Diagnostics, Fullerton, CA). Lipid content of the serum was determined using an enzymatic method based on measurements of serum cholesterol and triglycerides (Covaci et al. 2006).

Chemicals. Information on the specific internal, recovery, and quantitative standards used can be found in Supplemental Material, p. 2 (http://dx.doi.org/10.1289/ehp.1003235).

Sample extraction. Serum samples (approximately 3–5 g) were extracted using a previously published method (Stapleton et al. 2008). More information regarding sample extraction can be found in Supplemental Material, p. 2 (http://dx.doi.org/10.1289/ehp.1003235).

Sample analysis. Extracts were analyzed for 27 PBDE congeners using a method reported by Stapleton et al. (2008). A subset of 57 serum extracts was spiked with 10 ng [13C6, 6-OH-BDE 47] and [13C6, 6-OH-BDE, 6′-OH-BDE] to the dried extracts. Dried extracts were reconstituted in 100 µL methanol and analyzed using liquid chromatography tandem mass spectrometry (LC/MS-MS) for phenolic metabolites including 2,4,6-tribromophenol (246 TBP), 4′-hydroxy-2,2′,4′,5′-tetrabromodiphenyl ether (4′-OH-BDE-49), 6′-hydroxy-2,2′,4′,5′-tetrabromodiphenyl ether (6′-OH-BDE-49), 6-hydroxy-2,2′,4′,5′-tetrabromodiphenyl ether (6′-OH-BDE-49), 6-hydroxy-2,2′,4′,5′-tetrabromodiphenyl ether (6′-OH-BDE-49), 6-hydroxy-2,2′,4′,5′-tetrabromodiphenyl ether (6′-OH-BDE-49), 6-hydroxy-2,2′,4′,5′-tetrabromodiphenyl ether (6′-OH-BDE-49), and alpha-, beta- and gamma hexabromocyclododecane (HBCD). More information on the LC/MS-MS method can be found in Supplemental Material, pp. 2–3 (http://dx.doi.org/10.1289/ehp.1003235).

Quality control. Bovine serum (Invitrogen, Gaithersburg, MD) was used for quality assurance to test performance of the extraction method for PBDEs and HBCD. Further details on the calculation of method detection limits (MDLs) and internal standard and SRM recoveries can be found in Supplemental Material (http://dx.doi.org/10.1289/ehp.1003235).

Statistical analysis. Statistical analyses were performed using Stata 11 (StataCorp, College Station, TX). Values below MDL were assigned a value equal to half the detection limit for statistical analyses. Summary statistics were computed for thyroid hormones, individual and total PBDEs, and phenolic metabolites, using only those compounds with detection frequencies ≥ 50%. Correlations among PBDEs, metabolites, and thyroid hormones were examined using Spearman rank-order correlation.

The distributions of thyroid hormone, PBDEs, and metabolites were assessed for normality (Shapiro–Wilkes) and log-transformed if they were log-normally distributed. We implemented multiple linear regression analysis to assess the association between thyroid hormone levels and PBDE metabolite levels, controlling for maternal characteristics known to influence thyroid hormone levels, including smoking status (two categories of nonsmoker and ever-smoked before 28 weeks of gestation), race (non-Hispanic black or other), age (three categories: 18–19, 20–24, and 25–39 years), gestational age at blood draw (weeks), and parity (operationalized as whether or not the infant was firstborn). In addition, we used logistic regression (adjusted for the same covariates) to examine whether PBDE or PBDE metabolite levels were associated with TT3 levels above the normal range in the general population (80–178 ng/dL) or FT3 levels below the normal range (0.52–1.21 ng/dL) and ordered logistic regression to estimate associations of PBDEs/metabolites with low, normal, or high hormone TT3 levels (normal range 5.5–10.8 µg/dL). For TSH, the normal range in the general population was defined as 0.34–5.66 µIU/mL. Few observations fell outside the normal range for TT3 (2.2–3.8 pg/mL), precluding further analysis.

Results

Population characteristics. Between September 2008 and June 2010, 140 pregnant women enrolled in this study. Women were all residents of Durham County, North Carolina. Characteristics of the 137 participants are presented in Table 1. Eighty percent of the women were non-Hispanic white, 11% non-Hispanic black, and 12% Hispanic. Smoking status was assessed for 127 participants. Of these, 34% were nonsmokers, 62% smoked before 28 weeks of gestation, and 4% smoked after 28 weeks of gestation.

Table 1. Cohort characteristics (n = 137).

| Characteristic       | n (%) |
|----------------------|-------|
| Maternal race        |       |
| Non-Hispanic white   | 12 (9) |
| Non-Hispanic black   | 110 (80) |
| Hispanic             | 12 (9) |
| Other                | 3 (2)  |
| Maternal age (years) |       |
| 18–19                | 32 (23) |
| 20–24                | 69 (50) |
| 25–39                | 36 (26) |
| Parity               |       |
| First birth          | 70 (51) |
| Third birth          | 36 (28) |
| Fourth birth         | 19 (14) |
| Five or more         | 4 (3)  |
| Male infant sexa     | 62 (46) |
| Maternal education   |       |
| Less than high school| 19 (14) |
| High school diploma  | 72 (53) |
| More than high school| 46 (34) |
| Not married          | 127 (93) |
| Smoking              |       |
| Nonsmoker            | 90 (66) |
| Smoked before 28 weeks of gestation | 47 (34) |

*Total n = 133 because of missing values. Percentages may not sum to 100 because of rounding.
of the women in the study were non-Hispanic black, and 23% were < 20 years of age. Note that oversampling of non-Hispanic blacks is an intentional component of the parent HPHB Study. This was the first pregnancy for roughly half of the women enrolled in this study, and more than 87% completed high school. Of all the women participating in this study, only one reported a personal history with thyroid problems; she was not excluded from this study.

**Thyroid hormones.** Thyroid hormone data were available for only 137 of the 140 women enrolled (Table 2); however, we had a total of 136 thyroid hormone measurements per individual hormone type because of problems with the laboratory measurement of some hormones in a few samples. TSH, FT₄, and FT₃ were log-normally distributed, whereas TT₄ was normally distributed. TT₃ was not normal or log-normally distributed and was log-transformed for regression analysis. With the exception of one participant with a TSH level of 0.31 µIU/mL, all serum samples were found to have TSH levels within normal ranges. Pregnancy can affect thyroid hormone levels, and normal ranges presented here are for the general population. Approximately 10% of the serum samples (n = 13) had FT₄ levels below normal ranges, and one was just above normal at 1.22 ng/dL. For measurement of TT₄, 28% of the serum samples had levels below the normal range, and five samples had TT₄ levels above normal. Five samples had FT₃ levels below the normal range, and two were higher than normal. In contrast, approximately 63% of the serum samples displayed TT₃ levels higher than normal, whereas the remaining 37% were within normal ranges.

**PBDEs.** PBDE data are available for 137 of the 140 women enrolled (Table 2). Eight of the 27 PBDE congeners measured were found to be above MDLs in the serum samples analyzed. Attempts were made to measure BDE-209; however, laboratory blank contamination with BDE-209 resulted in all values below the MDL. Total PBDEs (defined as the sum of BDEs 47, 99, 100, and 153) were log-normally distributed; however, individual PBDEs were not normal or log-normally distributed. At least one of the BDE congeners was detected in all samples, and total PBDE concentrations ranged from 3.6 to 694 ng/g lipid (Table 2).

The most abundant congener was 2,2′,4,4′-tetrabromodiphenyl ether (BDE-47), which ranged in concentration from < 2.0 to 297 ng/g lipid, with a geometric mean value of 16.5 ng/g lipid. BDE-47 contributed 50% of the total PBDE burden, on average. The second most abundant BDE congener was 2,2′,4,4′,5′-pentabromodiphenyl ether (BDE-153) and the most commonly detected (~ 96% of samples), with concentrations ranging from < 1.2 to 67.6 ng/g lipid. BDE-153 was approximately 20% of the total PBDE burden, on average, although in one individual serum sample BDE-153 was the only congener detected. On average, 2,2′,4,4′,5′-pentabromodiphenyl ether (BDE-99) and 2,2′,4,4′,6-pentabromodiphenyl ether (BDE-100) represented 17% and 14% of the total PBDE burden, respectively.

**HBCD and phenolic metabolites.** A subset of 57 samples was further analyzed for isomers of HBCD (alpha, beta, and gamma), 246 TBP, and four different hydroxylated PBDE congeners by liquid chromatography tandem mass spectrometry. HBCD was < 0.17 ng/g lipid in all serum extracts; however, alpha-HBCD was detected (0.18–0.21 ng/g lipid) in the standard reference material (SRM 1958: National Institute of Standards and Technology, Gaithersburg, MD) used for quality assurance/quality control, demonstrating that the method did recover HBCD. The measured concentration in SRM 1958 ranged from 64 to 76% of the reported value (Keller et al. 2010).

**Assocations between PBDEs, metabolites, and thyroid hormones.** The PBDEs were all highly correlated (r = 0.39–0.95) (Table 3). Total PBDEs and individual BDE congeners were highly correlated with 4′-OH-BDE-49 (r = 0.38–0.62; Figure 1) and 6-OH-BDE-49 and 6-OH-BDE-47 were detected in 72% and 67% of the samples, respectively. 246 TBP was detected at the highest concentrations (< 1.4–151 ng/g lipid); however, values were above MDL in only 38% of the samples. Total OH-BDE levels (4′-OH-BDE-49 plus 6-OH-BDE-47) ranged from < 0.03 to 13.3 ng/g lipid, with a geometric mean value of 0.28 ng/g lipid.

### Table 2. Thyroid hormone levels and PBDE and metabolite concentrations (nanograms per gram lipid) measured in serum from pregnant women.

| Variable                  | MDL          | Detection frequency (%) | Geometric mean (95% CI) | Min       | Max       | 25th     | 50th     | 75th     | 95th     |
|---------------------------|--------------|-------------------------|-------------------------|-----------|-----------|----------|----------|----------|----------|
| TSH (µU/mL)               | 100.00       | 1.25 (1.15–1.37)        | 0.31                    | 5.38      | 0.89      | 1.28     | 1.76     | 2.91     |
| FT₄ (ng/dL)               | 98.53        | 6.1 (5.6–6.7)           | 0.3                    | 11.9      | 5.2       | 6.9      | 8.5      | 10.4     |
| FT₃ (ng/dL)               | 100.00       | 0.67 (0.64–0.69)        | 0.40                    | 1.22      | 0.59      | 0.67     | 0.75     | 0.89     |
| FT₄ (ng/mL)               | 100.00       | 195 (188–204)           | 100                    | 411       | 165       | 180      | 221      | 349      |
| FT₃ (ng/mL)               | 100.00       | 2.71 (2.65–2.77)        | 1.85                    | 4.25      | 2.47      | 2.71     | 2.97     | 3.39     |

**PBDEs** (n = 137)

| BDE-28 | 1.2–3.0 | 36.69 | NA | < 1.2 | 16.89 | 0.60 | 0.60 | 2.58 | 6.00 |
| BDE-47 | 2.0–4.5 | 94.89 | 16.5 (13.64–19.98) | < 2.0 | 297.45 | 8.98 | 18.87 | 30.64 | 114.36 |
| BDE-66 | 1.2      | 2.19  | NA | < 1.2 | 3.93  | 0.60 | 0.60 | 0.60 | 0.60 |
| BDE-99 | 2.0–4.5 | 64.23 | 4.72 (3.74–5.94)   | < 2.0 | 249.08 | 1.00 | 5.50 | 12.59 | 49.83 |
| BDE-100 | 1.2     | 219.06 | 4.19 (3.51–5.00)   | < 1.2 | 107.45 | 2.27 | 4.61 | 7.20 | 25.85 |
| BDE-153 | 1.2     | 16.06 | NA | < 1.2 | 10.49 | 0.60 | 0.60 | 0.60 | 0.60 |
| BDE-153 | 1.2     | 96.35 | 5.93 (5.09–6.92)   | < 1.2 | 67.55  | 3.02 | 5.55 | 9.82 | 22.33 |
| BDE-154 | 1.2     | 48.18 | NA | < 1.2 | 52.89 | 0.60 | 0.60 | 2.40 | 7.59 |

| Total PBDEs | 3.59 | 693.95 | 20.02 | 36.56 | 64.58 | 228.16 |

| Phenolic metabolites (n = 57) |
|------------------------------|
| 246 TBP | 1.4–2.5 | 38.18 | NA | < 1.4 | 150.74 | 1.65 | 2.43 | 15.14 | 119.71 |
| 4′-OH-BDE-49 | 1.01–0.03 | 71.93 | 0.11 (0.07–0.16) | < 0.01 | 3.92 | 0.02 | 0.12 | 0.27 | 2.32 |
| 6-OH-BDE-47 | 0.01–0.03 | 66.67 | 0.17 (0.10–0.29) | < 0.01 | 10.79 | 0.02 | 0.19 | 0.57 | 5.82 |

Abbreviations: Max, maximum; Min, minimum; NA, not available (detection frequency was < 50%).

**αOH-BDEs** variable includes BDE-47, BDE-99, BDE-100, and BDE-153 because each of these had > 50% detection rates. In creating this summed variable of the four PBDEs, observations that were below detection limit were included as one-half MDL. For 246 TBP, total n = 55. **αOH-BDEs** includes 4′-OH-BDE-49 and 6-OH-BDE-47 because each of these had > 50% detection rates.

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Results of the multivariable model (Table 4) show that TT₄ is significantly and positively associated with BDE-47, BDE-99, and ∑BDEs (r = 0.32–0.50, p < 0.05), but was not significantly associated with BDE-153. Logged FT₃ is significantly and positively associated with BDE 47, BDE-153, and ∑BDE (r = 0.05, p < 0.05) but was not significantly associated with BDE-99 or BDE-100.

These results hold equally well in logistic regression models (results not shown). In these models, PBDEs and OH-BDEs were logged base 2 transformed so that their estimated odds ratios (ORs) are interpreted as the change in odds associated with a 2-fold increase in the particular PBDE or OH-BDE. Adjusted ordinal logistic regression model ORs for normal versus low or high vs normal TT₄ levels with a 2-fold increase in exposure were 1.42 [95% confidence interval (CI), 1.08–1.86, p = 0.01] for BDE-47; 1.37 (95% CI, 1.01–1.84, p = 0.04) for BDE-100; 1.25 (95% CI, 1.00–1.57, p = 0.05) for BDE-99; and 1.45 (95% CI, 1.04–2.01, p = 0.03) for ∑BDEs. Likelihood ratio tests of the proportionality of odds assumption did not find evidence to reject the null hypothesis that the odds were the same across outcome categories. For each 2-fold increase in BDE-47, the odds of having low FT₃ decreased by 40% (OR = 0.60; 95% CI, 0.39–0.93, p = 0.02) and by 49% for ∑BDEs (OR = 0.51; 95% CI, 0.27–0.97, p = 0.04). Although BDEs modeled as a continuous variable, high TT₄ levels (178 ng/dL) were positively associated with FT₄ (OR = 1.30; 95% CI, 1.00–1.69, p = 0.04) and inversely associated with 4’-OH-BDE-49 (OR = 0.51; 95% CI, 0.30–0.86, p = 0.01) and ∑OH-BDEs (OR = 0.72; 95% CI, 0.48–1.07, p = 0.10).

Discussion

The PBDE levels and congener distributions in this pregnancy cohort were similar to levels reported among the general U.S. population in the 2003–2004 National Health and Nutrition Examination Survey (NHANES) (Sjodin et al. 2008). This similarity was unexpected, because PBDEs have been phased out from use since 2004, and there was a 4- to 7-year time gap between the sample collections for the two cohorts. One might expect to see declining levels of PBDEs in human tissues over the preceding 4–7 years. Our data suggest that levels are stable; however, some caveats must be noted with this comparison. The NHANES study observed higher PBDE levels in non-Hispanic whites relative to non-Hispanic whites, and decreasing PBDE levels with age; therefore, levels may be comparable because our cohort was composed primarily of non-Hispanic black females ages 18–22 years. Geometric mean BDE-47 levels in NHANES (2003–2004) for 12- to 19-year-olds (28.2 ng/g lipid), 20- to 39-year-olds were 0.72; 95% CI, 0.48–1.07, p = 0.10).
(21.5 ng/g lipid), and non-Hispanic blacks (24.3 ng/g lipid) were higher than the geometric mean level measured for our cohort (16.5 ng/g lipid). Thus, our results are consistent with decreasing PBDE levels over time within race and age groups.

Several phenolic metabolites were also identified in a subset of these samples (Table 2). Previous studies have observed these phenolic compounds in both human tissues and in laboratory animals exposed to PBDE mixtures, suggesting they are in fact metabolites of PBDEs (Athanasiadou et al. 2008; Hakkinen and Letcher 2003; Qiu et al. 2007, 2009; Wan et al. 2010). Qiu et al. (2009) reported mean levels of 0.8, 0.3, and 0.3 ng/g lipid for 246 TBP, 4′-OH-BDE-49, and 6-OH-BDE-47, respectively, in maternal blood samples (n = 4) collected in 2003-2004. These levels are similar to the median levels we report here of 2.5, 0.11, and 0.17 ng/g lipid for 246 TBP, 4′-OH-BDE-49, and 6-OH-BDE-47, respectively. Although 246 TBP is a known metabolite of PBDEs, it is also produced and used as a flame retardant in phenolic and epoxy resin itself (Anderson et al. 2006). Thus, there can be more than one source of exposure to this compound in human tissues.

All PBDEs and OH-BDEs were highly correlated, except 6-OH-BDE-47 and BDE-99. This weaker relationship may be attributable to the fact that 6-OH-BDE-47 is not a likely metabolite of BDE-99 and/or may have marine natural sources (Malmvärn et al. 2005). In addition, BDE-99 may be metabolized much more rapidly than BDE-47 and BDE-153 (Chen et al. 2006; Hakkinen and Letcher 2002; Lupton et al. 2009; Stapleton et al. 2004b, 2009).

Positive associations between PBDEs and FT₄ and TT₃, levels have been observed in nonpregnancy cohorts in previous studies (Meeker et al. 2009; Turney et al. 2008). Unfortunately, we were not able to control for potential confounders [e.g., polychlorinated biphenyls (PCBs), organochlorine pesticides] in this study. However, both Turney et al. (2008) and Chevrier et al. (2010) were able to control for these confounders, and they demonstrated that associations between PBDEs and thyroid hormones remained. In addition, PCBs have been associated with decreases in T₄, not increases as observed in our study (Brouwer et al. 1998; Wang et al. 2005).

Positive associations between PBDEs and T₃ observed are opposite to those typically observed in laboratory animal exposure studies, where exposed animals experience decreases in circulating T₃ and T₄ levels (Fernie et al. 2005; Tomiy et al. 2004; Zhou et al. 2001). A previous study examining PBDEs in pregnant women found no significant associations between PBDEs and T₄; however, the authors did observe an inverse association between PBDEs and TSH (Chevrier et al. 2010). Low TSH levels are generally associated with higher T₄ levels via feedback mechanisms regulated by the pituitary (Hulbert 2000); thus, the results observed here and by Chevrier et al. (2010) suggest a similar trend with respect to the influence of PBDEs on thyroid hormone regulation during pregnancy. Differences in TSH associations between the two studies may be related to the fact that African-American women in general have lower TSH levels (Walker et al. 2005). Both cohorts displayed very similar PBDE levels, with geometric mean BDE-47 levels of 15.3 and 16.9 for their cohort and our cohort, respectively. However, as noted by Chevrier et al. (2010), different methods for measuring thyroid hormones may also be a factor in different trends observed. Here we used a chemiluminescent immunoassay for measuring unbound (i.e., free) thyroid hormones (which may be a limitation), whereas Chevrier et al. (2010) used an equilibrium dialysis method. Diurnal variations in thyroid hormone secretions may also be a factor.

Our findings suggest an inverse association between 4′-OH-BDE-49 and TT₃, but our analysis was limited by the small sample number (n = 56). Most animal exposure studies have observed significant decreases in T₄ with exposure to PBDEs, but not T₃. However, a large proportion of this cohort (63%) did have TT₃ levels that were above normal levels for an average adult, which is likely linked to their late stage of pregnancy. Previous in vitro studies have demonstrated that hydrolylation of the PBDE congener results in significantly increased binding affinities for thyroid hormone transporters found in serum (Marchesini et al. 2008; Meerts et al. 2000). Specifically, 4′-OH-BDE-49 has been shown to have a high binding affinity to the thyroid hormone serum transporter transthyretin (Ucan-Martin et al. 2009). Thus, it may be possible that PBDE metabolites are competing for space on the thyroid hormone transporters, resulting in more free (i.e., unbound) T₄. However, TT₃ was also positively associated with PBDEs. An alternate explanation may be related to the effect of PBDEs and/or their metabolites on deiodinase activity. Previous in vivo and in vitro studies in fish models have suggested that deiodinase enzymes (DIs) may be metabolizing PBDEs via a dehalogenation pathway (Noyes et al. 2010; Stapleton et al. 2004a). DIs serve to metabolize T₃ to T₄ in peripheral tissues, supplying the main source of T₄ that binds to thyroid nuclear receptors to activate transcriptional processes (Kohlert 1999). If DI activity was inhibited by PBDEs and/or their metabolites, it might result in an increase in circulating T₃ levels and a decrease in T₄ levels. Here we have reported on the levels of PBDEs in a pregnancy cohort consisting primarily of African-American women and report on the associations between OH-BDEs and thyroid hormones. Although there are some limitations to this study (e.g., analysis during the third trimester of pregnancy, method used for measuring thyroid hormones), the results do support previous studies that have observed associations between PBDEs and thyroid hormones. PBDEs have a very long half-life in the human body, and PBDE levels measured in the third trimester are likely to reflect levels throughout pregnancy. Consequently, it is possible that PBDEs may be affecting thyroid hormone regulation throughout pregnancy, including the first trimester when the fetus relies solely on maternal thyroid hormone supply. More research is needed to determine the mechanisms by which PBDEs affect thyroid hormone regulation during pregnancy and how this, in turn, affects the development of the fetus.

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