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Associations between sociodemographic characteristics and exposures to PBDEs, OH-PBDEs, PCBs, and PFASs in a diverse, overweight population of pregnant women

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Abstract

Exposures to persistent organohalogen chemicals during pregnancy are associated with adverse health effects. Low-income, minority women with pre-existing co-morbidities may be particularly vulnerable to these exposures, but have historically been understudied. We aimed to characterize exposures to multiple chemical classes among a sample of ethnically diverse, lower income, overweight or obese pregnant women. Serum concentrations of polybrominated diphenyl ethers (PBDEs) and their hydroxylated metabolites (OH-PBDEs), polychlorinated biphenyls (PCBs), and poly- and perfluoroalkyl substances (PFASs) were measured in 98 pregnant women (California; 2011–2013). Aggregate exposures were evaluated using correlational clustering, a “chemical burden” score, and principal components analysis (PCA). Associations between sociodemographic characteristics and individual and aggregate exposures were evaluated using multivariable linear regression. Clustering and PCA both produced four groupings: (PC1) PBDEs/OH-PBDEs, (PC2) PCBs, (PC3) PFNA/PFOA/PFDa, (PC4) PFHxS/PFOS. Race/ethnicity and pre-pregnancy BMI

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Conflicts of Interest

All coauthors have no conflicts of interest to declare.

were associated with PBDEs, OH-PBDEs and PC1. Maternal age was associated with PCBs and PC2. Parity was associated with PBDEs, OH-PBDEs and PC2. Poverty was negatively associated with PCBs whereas food insecurity was positively associated with PFOS. We observed variations in sociodemographic profiles of exposures by chemical class and weak across-class correlations. These findings have implications for epidemiologic studies of chemical mixtures and for exposure reduction strategies.

Keywords

chemical mixtures; flame retardants; health disparities; obesity; PCBs; Perfluorinated compounds

Introduction

Persistent organohalogen compounds are widespread industrial chemicals that disturb normal functioning of numerous hormonal and homeostatic processes in humans (Diamanti-Kandarakis et al. 2009). Due to their ecological and biological persistence, as well as their relative toxicity to humans, environmental exposure to chemicals such as polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs), and certain long-chained poly- and perfluoroalkyl substances (PFASs) have been largely phased-out or banned in the United States. Despite decreased production, these legacy chemicals are still widely detected in the U.S. population (Sjödén et al. 2013; Jain 2014), including pregnant women (Woodruff et al. 2011; Mitro et al. 2015). Pregnancy is a critical window for exposure to organohalogen chemicals. PBDEs, PCBs, and PFASs can cross the placental barrier to a developing fetus (Mitro et al. 2015) and have been detected in various biological media, including cord blood, amniotic fluid, and breast milk (Barr et al. 2007). Furthermore, prenatal exposure has been associated with a range of adverse health effects to both women and children, including pregnancy complications and neurodevelopmental harm in childhood (Wang et al. 2016).

In the U.S., almost all pregnant women have detectable concentrations of one or more PBDE, PCB and PFAS chemicals in their serum (Woodruff et al. 2011); although, more recent biomonitoring data suggest maternal concentrations of certain organohalogen classes are declining (Wang et al. 2011; Hurley et al. 2016; Parry et al. 2018). Not all population groups in the U.S. may be uniformly experiencing exposure declines since sources of exposure can vary by sociodemographic, geographic, occupational and lifestyle factors. Nationally, serum organohalogen concentrations have differed by race/ethnicity and poverty status in U.S. women of childbearing age (U.S. Environmental Protection Agency [EPA] 2017). Biomonitoring studies of organohalogens during pregnancy have also shown economically-disadvantaged, minority, and urban communities have higher overall concentrations (James et al. 2002; Borrell et al. 2004; Wolff et al. 2005; Vrijheid et al. 2012). In addition to chemical stressors, these communities are also disproportionately burdened by non-chemical stressors (Morello-Frosch et al. 2011; Sexton and Linder 2011; Evans et al. 2014).

Prior research has primarily focused on evaluating sociodemographic determinants of individual organohalogen concentrations in pregnant populations; however, multiple

chemical classes have been measured simultaneously in pregnant women (Woodruff et al. 2011; Braun et al. 2014), raising the need to better characterize cumulative exposures. Few studies have attempted to characterize profiles of environmental mixtures during pregnancy (Robinson et al. 2015; Lee et al. 2017). Only one recent U.S. study we are aware of has examined determinants of chemical mixtures during pregnancy (Kalloo et al. 2018).

This study aims to examine variability in concentrations of 52 persistent organohalogen chemicals from four chemical classes (PBDEs, hydroxylated PBDE metabolites (OH-PBDEs), PCBs, PFASs) and combined mixtures of these individual chemicals in a unique population of racially/ethnically diverse, low-income, overweight, pregnant women from Northern California.

Methods

Study population and exclusion criteria

The Maternal Adiposity, Metabolism, and Stress (MAMAS) study was an eight-week intervention designed to determine whether certain stress-reduction techniques could decrease maternal stress and increase healthy weight gain during pregnancy (on www.clinicaltrials.gov) and has been described in detail elsewhere (Coleman-Phox et al. 2013; Vieten et al. 2018).

Women were eligible for the study if they were 8–23 weeks pregnant, 18–45 years old, English-speaking, annual household income of up to 500% of the federal poverty level, and a self-reported pre-pregnancy body mass index (BMI) between 25–40 kg/m². Subsequent medical record confirmation of pre-pregnancy BMI revealed seven participants with a pre-pregnancy BMI between 23.0 and 25.0 kg/m², and three participants with a pre-pregnancy BMI between 40.0 and 42.1 kg/m²; however, all participants were retained in the intervention arm. Participants were recruited in the San Francisco Bay area at locations that provided prenatal care and/or social services. Women were excluded if they had polycystic ovarian syndrome and were treated with Metformin, active substance abuse, recent weight loss, chronic corticosteroid use, pre-existing diabetes, an eating disorder, a positive early pregnancy diabetes screening test, multiple gestation, or a history of gastric bypass surgery. The 215 participants were assigned to either a control group or a mindfulness-based stress reduction and healthy eating behavioral intervention. Enrolled women completed questionnaires at baseline (10 to 24 weeks gestation) from 2011–2013 and were followed until 6–9 months postpartum. The MAMAS study was approved by the University of California, San Francisco (UCSF) Committee on Human Research and the California Pacific Medical Center, University of California, Berkeley and Contra Costa Regional Medical Center and Health Centers Institutional Review Boards. All participants provided informed consent.

Only the women in the intervention arm provided biological samples and are therefore the focus of the present analysis. There were 105 participants who met study inclusion criteria, but seven participants lacked baseline biological samples or data on multiple maternal characteristics, leaving 98 women for analysis.

Maternal organohalogen measurements

A 10 mL fasting maternal blood sample was collected at the baseline visit (between 10–24 weeks of gestation) in an additive-free red top tube (BD Vacutainer) by trained UCSF staff. Blood was allowed to clot for 1h, then placed on ice for a subsequent 1h. Samples were centrifuged at 1300g for 10 min at 4°C and 1 mL serum was aliquoted into five vials. Samples were stored in an –80°C freezer for up to three months. Serum was analyzed for individual PBDEs, OH-PBDEs, PCBs, and PFASs at the Environmental Chemistry Laboratory of the Department of Toxic Substances Control (Berkeley, CA, USA).

Detailed analytical laboratory methods, including sample extraction, instrumentation and procedures, validation, and quality control have been described on this study population in detail elsewhere (Zota et al. 2018). Briefly, serum sample extraction and analytical methods were performed for 19 PBDEs, 8 OH-PBDEs, 15 PCBs, and 11 PFASs based on commonly used techniques (Zota et al. 2011; Parry et al. 2018). We present on aggregated linear and branched isomers of both PFOS and PFOA. PBDE congener, OH-PBDE metabolite, and PCB congener concentrations were measured using gas chromatography/ high-resolution mass spectrometry. An online solid phase extraction liquid chromatography tandem mass spectrometry method was employed to determine concentrations of PFAS analytes in maternal serum (Wang et al. 2011). Serum lipid analysis was conducted at Boston Children's Hospital. A common method (Phillips et al. 1989) was used to calculate total serum lipids based on measured total cholesterol, triglycerides, and phospholipids. Each analyte's method detection limit (MDL) was defined as three times the standard deviation of the concentrations of blank analytes; further information on sample MDLs are published elsewhere (Zota et al. 2018).

A multiple imputation method was employed for values below the MDL using a log-normal probability distribution with parameters calculated using maximum likelihood estimation (Helsel 1990; Baccarelli et al. 2005; Zota et al. 2011). Lipid-soluble chemical concentrations (PBDEs and PCBs) were normalized using total serum lipids (ng/g lipid), whereas hydroxylated PBDE and PFAS analytes were reported as wet-weight concentrations (ng/mL).

Participant characteristics

Participant sociodemographic and behavioral information was collected through a questionnaire administered at baseline, which has been described in detail elsewhere (Vieten et al. 2018). Gestational age at enrollment, pre-pregnancy BMI, and parity were initially self-reported, then confirmed via medical records for all women. Gestational age at enrollment and expected delivery date were self-reported and later confirmed via medical records. For most participants, expected delivery date was calculated based on last menstrual period and ultrasound measurements in early pregnancy. A 24-hour dietary recall was administered by trained interviewers at baseline.

A number of potential participant characteristics were considered for statistical analysis based on a review of the literature. Sociodemographic characteristics include race/ethnicity (White or Other, African American, Latina), poverty status (annual household income

>100%, 100% of the 2011 federal poverty level), highest educational attainment (high school graduate, > high school graduate), and marital status (single/separated/divorced, married/in a relationship/engaged). Dietary measures include food insecurity and the Alternative Healthy Eating Index modified for Pregnancy (AHEI-P) scale assessed at baseline. Food insecurity was determined by the ten-point U.S. Household Food Security Survey Module (US Department of Agriculture 2012) and then dichotomized into marginal-to-high food security (i.e., food secure households) and low-to-very low food security (i.e., food insecure households). The Alternative Healthy Eating Index modified for Pregnancy (AHEI-P) is a 90-point, multicomponent scale based on the 2010 AHEI (US Department of Agriculture 2010), which categorizes the quality intake of fruits, vegetables, meat, and nutrients, but excludes AHEI components that are generally avoided during pregnancy (e.g., alcohol consumption). We also considered the following behavioral and biological characteristics for our statistical models: gestational age (weeks at enrollment), pre-pregnancy BMI (kg/m^2), BMI at baseline (kg/m^2), parity (continuous), smoking status (current/former smoker versus never smoked), and lipid concentrations (g/mL).

Maternal adiposity was considered in our analysis because lipophilic organohalogens bioaccumulate and are retained in fat tissue. We chose pre-pregnancy BMI over baseline BMI in models because it temporally precedes natural gestational weight gain and, therefore, more likely reflects long-term chemical exposures of lipophilic organohalogens. Additionally, baseline visits for participants varied across a 14-week window when greater weight gain may occur. BMI at baseline visit was highly correlated with pre-pregnancy BMI (Spearman $r=0.89$; $p<0.001$).

Statistical analysis

Descriptive statistics, including geometric means (GM) and geometric standard errors (GSE), were calculated for individual and total sum chemical concentrations. Of the 52 organohalogen chemicals available, individual chemicals were further examined for the subset of chemicals that were detected in at least half of the participants (i.e., detection frequency [DF] $\geq 50\%$), which includes four PBDE congeners (BDE-47, -99, -100, -153), two OH-PBDE metabolites (5-OHBDE-47, 6-OHBDE-47), three PCB congeners (PCB-138, -153, -180), and five PFAS analytes (perfluorohexane sulfonate [PFHxS], perfluorooctane sulfonate [PFOS], perfluorooctanoic acid [PFOA], perfluorononanoic acid [PFNA], and perfluorodecanoic acid [PFDeA]). We also calculated summary exposure metrics. For chemicals with a $\geq 50\%$ DF, we summed congener or analyte concentrations for Σ PBDEs, Σ OH-PBDEs, Σ PCBs, and Σ PFASs. Prior to our statistical analysis, we natural log-transformed serum concentrations given the distributions were skewed and non-normal.

Relationship between maternal characteristics and chemical concentrations were initially examined using bivariate linear regression models, and associations with a p -value <0.20 in any models were chosen for further “exploratory” analysis (Supplementary Tables 1 and 2). Subsequently, retained variables were fitted into a model and backwards elimination was employed, with a significance level of $p<0.05$. Final multivariable linear regression models were used to evaluate chemical class-specific participant characteristics identified by backward elimination and individual maternal serum organohalogen concentrations.

Regression coefficients and 95% confidence intervals (CIs) are provided, as well as an adjusted R-squared to measure the proportion of variation explained by included variables. Both descriptive statistics and multivariable linear regression models for Σ PBDEs, Σ OH-PBDEs, Σ PCBs, and Σ PFASs were also examined. To examine the effect of lipid normalization on our results, we ran additional sensitivity analyses using wet-weight concentrations of PBDEs and PCBs and adding log-adjusted total serum lipids as an independent covariate in our final models. Test for effect modification by income or race/ethnicity on chemical concentrations was determined by adding cross product terms into final models. To further explore the role of poverty status on the relationship between potential participant characteristics and chemical exposures, we also examined the association of race/ethnicity and chemical concentrations after stratifying by poverty status and while controlling for maternal age, pre-pregnancy BMI, and parity (not shown).

We used three methods of grouping chemicals: hierarchical clustering, a “chemical burden” sum score approach, and principal components analysis (PCA). We chose these unsupervised approaches (i.e., not informed by an outcome) for their variable reduction strategies which account for multicollinearity, and to identify exposure groups based on co-occurrence. We examined Spearman correlations between all individual chemicals and their chemical classes, and classified these correlations as high [(+/-) 0.60 to 1.00], moderate [(+/-) 0.40 to 0.59], or weak [0.00 to (+/-) 0.39]. An agglomerative hierarchical clustering approach was performed using the value of “1 minus the correlation coefficient (r)” to calculate the dissimilarity index, which ultimately allows for grouping of the most correlated terms to determine profiles of chemical exposures in the cohort. A heat map was produced to visualize groupings. To examine the “chemical burden” to each study participant, a sum count score was calculated for each participant by adding the number of highly detected chemicals that had concentrations in the top 25th percentile of each chemical’s distribution (potential range in each participant: 0–14). For example, an individual identified with the top 25th percentile serum concentrations of two PFAS, two PBDEs, one OH-PBDE, and two PCB analytes would be assigned a “chemical burden” sum count score of seven. A cumulative frequency plot of the sum score by all participants was produced to visually examine if certain participants had high exposures to multiple chemicals within and across chemical classes. To account for the large number of correlated exposure terms and further understand the correlation structure of our data, we employed PCA, which categorizes correlated variables into artificial factors while maximizing the amount of variance in the dataset. We used log-transformed values for each individual chemical with a 50% DF. Principal components (PCs) were chosen based on eigenvalues > 1.0 and visual analysis of a scree plot. Varimax rotation was employed to produce optimal components. An individual chemical with a factor loading >0.50 was considered to be a critical chemical of a component. Using backward selection, linear regression analysis was used to determine if any participant characteristics were associated with each principal component. All significant variables were fitted in final multivariable linear regression models.

All statistical analyses were conducted in SAS version 9.3 (SAS Institute, Cary, NC) or in R software version 3.3 (<https://cran.r-project.org/>).

Results

The study sample had substantial racial and ethnic heterogeneity, with an equal distribution of Latina women (33.7%), non-Hispanic African American women (32.7%), and non-Hispanic White women or women from other races/ethnicities (33.7%) (Tables 1 and 2). Almost half of the women (47.8%) were living at or below the 2011 federal poverty level. Participants were more likely to have at least a high school education (65.3%) and to be in a relationship, engaged or married (66.3%). By design, all of the participants were overweight and 44.9% had an obese pre-pregnancy BMI (≥ 30 kg/m²). Almost 40% of participants were categorized as having low or very low food security (i.e., food insecure), and half scored below the 50th percentile for the AHEI-P, indicating a poor quality diet.

Fourteen of the 52 chemicals were detected in 50% of the population with BDE-47, -99, -153, PCB-153, PFNA, PFOS, PFOA, and PFHxS detected in >85% of participants. Hierarchical clustering of all chemicals within and across chemical classes revealed high within-chemical class and low across-chemical class correlation (Figure 1). BDE-47, -99, and -100 were highly correlated with each other, and were weakly-to-moderately correlated with BDE-153. OH-PBDEs were highly correlated with each other, and strongly-to-moderately correlated with parent PBDE congeners. PCB-153 and PCB-180 were highly correlated with each other, and moderately correlated with PCB-138. Moderate to strong positive correlations were seen among some PFAS analytes, with two clustered groupings of (a) PFHxS and PFOS, and (b) PFDeA, PFNA and PFOA.

In multivariable analysis, African American women had higher GM concentrations of BDE-100 and -153, whereas Latina women had lower GM concentrations of 6-OHBDE-47 and Σ OH-PBDEs, compared to women who were White or of other races/ethnicities (Table 3). A higher pre-pregnancy BMI was positively associated with higher PBDE and OH-PBDE concentrations in unadjusted models; however, after adjusting for race/ethnicity, education, and parity, the association only remained significant for BDE-100. Higher educational attainment was positively associated with higher exposure to PBDEs and OH-PBDEs in unadjusted models, and this association remained significant for BDE-99 in adjusted models. After adjustment, parity was inversely associated with BDE-100, 5-OHBDE-47, and Σ OH-PBDEs exposures. Results were similar in exploratory models (Supplemental Table 1).

Maternal age and/or poverty status were significantly associated with individual and aggregate PCB concentrations, accounting for 31 to 47% of the variance in multivariable linear regression models (Table 4). For final adjusted models of PCBs, an increase in maternal age was associated with a significant increase in log maternal serum concentrations of PCB-138, -153, -180, and Σ PCBs. After controlling for maternal age, women living at or below the poverty level had significantly lower GM concentrations of PCB-138 and Σ PCBs, compared to women living above the poverty level. There was little change in results when we expanded our final models to include additional participant characteristics such as race/ethnicity, education, pre-pregnancy BMI, gestational age, marital status, and/or AHEI-P score (Supplemental Table 2), with the exception of PCB-180 becoming significantly associated with poverty status. Log serum PFOS concentrations were significantly higher in

women living in food insecure households, compared to women who had marginal to high food security (Table 4 and Supplemental Table 2). No additional participant characteristics were associated with any PFAS analytes in final and exploratory models. When total serum lipids were adjusted for in final models for wet weight concentrations of PBDEs and PCBs, the magnitude and direction of results were similar (Supplemental Tables 3 and 4). We did not observe evidence of effect modification of poverty status for associations between race/ethnicity and environmental exposures (data not shown).

Figure 2 shows each participant's "chemical burden" sum score, by chemical class, for chemicals with a concentration greater than the 75th percentile concentration of the distribution. A median of 5 chemicals (range: 3–10 chemicals) were present; at least three of the four chemical classes examined were present in each participant's serum at concentrations in the top 25th percentile relative to the study population. No association was observed between the "chemical burden" sum score and participant characteristics (not shown). When PCA was conducted, four PC groupings were determined: (PC1) PBDEs and OH-PBDEs, (PC2) PCBs, (PC3) PFNA, PFOA, and PFDeA, and (PC4) PFHxS and PFOS; these four PCs explained 70% of the variance (Table 5). In multivariable analyses (Table 6), race/ethnicity and pre-pregnancy BMI were significantly positively associated with PC1 loadings (PBDEs and OH-PBDEs). Maternal age was positively associated with PC2 (PCBs), and parity was inversely associated with PC2. There were no significant participant factors of either PC3 (PFNA, PFOA, PFDeA) or PC4 (PFHxS and PFOS) loadings.

Discussion

In our analysis of a unique pregnancy cohort of overweight, lower-income women from California, we found a variation in sociodemographic characteristics associated with individual organohalogen chemicals and their chemical classes. We explored associations between select maternal characteristics and both individual chemicals and aggregate groupings. In individual, total sum and aggregate analyses, African American women had increased maternal serum PBDE and OH-PBDE concentrations, and Latina women had lower PBDE/OH-PBDE concentrations. Other significant factors of PBDE/OH-PBDE included higher pre-pregnancy BMI and higher educational attainment, while parity was associated with lower levels. PCB concentrations were positively associated with maternal age, similar to other pregnancy cohorts (Wolff et al. 2005; Ibarluzea et al. 2011). In this lower income cohort, those living at or below the poverty level had significantly lower PCB concentrations, supporting results from national estimates (U.S. EPA 2017).

Five PFAS analytes were highly detected in the population, with PFNA, PFOS, PFHxS, and PFOA at 98% DF. PFAS GM concentrations were similar to, or lower than, other North American pregnancy or population-based cohorts (Louis et al. 2016; Morello-Frosch et al. 2016; Lewin et al. 2017; Starling et al. 2017; U.S. EPA 2017). Interestingly, we found that participants from households that were food insecure had significantly higher maternal serum PFOS concentrations than those who were food secure. In general, diet is an important contributor to PFAS concentrations in pregnant women (Halldorsson et al. 2008; Sagiv et al. 2015; Manzano-Salgado et al. 2016). Poorer dietary quality and food availability, as measured by food insecurity, may be indicators for higher consumption of PFAS-

contaminated foods. PFAS compounds have been detected in food packaging materials (Yuan et al. 2016; Schaidt et al. 2017), can migrate into food items (Tittlemier et al. 2007), and have been associated with fast food consumption (Harris et al. 2017) and takeaway containers (Bonorow et al. 2019). Given the variability in concentrations across food sources, future consideration should be given to account for dietary quality. Exposure to PFASs through a variety of sources, including diet, consumer products, and drinking water, further complicate the ability to identify patterns using biomonitoring data alone. Based on both hierarchical clustering and PCA, two groupings emerged: (1) sulfonic (PFHxS and PFOS) and carboxylic acids (PFDeA, PFNA, and PFOA). PFHxS and PFOS are long-chained perfluoroalkyl sulfonate analogues with the longest half-lives in human serum (Olsen et al. 2007), which may increase the likelihood of bioaccumulation. PFOS and PFHxS have been associated with certain exposure sources including aqueous film forming foams, microwaveable food, and waterproof clothing (Rotander et al. 2015; Wu et al. 2015; Sienbenaler et al. 2017). Sources of exposure to carboxylic acids are diverse, though indirect sources include oil- and water-repellant applications of fluorotelomer alcohols and polyfluoroalkyl phosphate esters (D'eon and Mabury 2011). Certain long-chain PFAS chemicals have been linked to adverse childhood neurodevelopment (Chen et al. 2013; Wang et al. 2015), decreased fetal growth (Lam et al. 2014), immune suppression (NTP 2016), metabolic (Starling et al. 2017; Liu et al. 2018) and carcinogenic (Benbrahim-Tallaa et al. 2014) effects. Within our study population, maternal PFASs, including PFOS, PFOA, and Σ PFASs, were associated with biomarkers of inflammation (Zota et al. 2018). Given the ubiquity of PFAS exposure sources and continued detection of both long-chained and newer shorter-chained PFASs in the environment and biomonitoring studies, further characterization of PFAS exposures and their sources is warranted.

In utero PBDE exposure continues to be a concern for numerous adverse health outcomes, including sufficient evidence of a causal effect on childhood neurodevelopmental outcomes (Lam et al. 2017). In our study, we found four PBDE congeners at detectable concentrations in >75% of the study population, with BDE-47 detected in all participants. Two lower brominated hydroxy-BDEs metabolites were also found in at least half of the study population, albeit at lower concentrations. It is possible our high detection rate of the lower brominated PBDE congeners was a result of their longer half-life in the body and higher absorption rate compared to the higher brominated PBDE congeners (Costa et al. 2014). Median PBDE concentrations in this cohort were either higher (Morello-Frosch et al. 2016) or similar (Parry et al. 2018) to other Northern California cohorts, and higher than the general US population (U.S. EPA 2017). As a result of California 2006 ban and EPA's 2014 phase out of PBDEs, concentrations of PBDEs in California pregnant women have been declining (Zota et al. 2016; Parry et al. 2018). While exposure is decreasing overall, our results showing African American women with elevated PBDE levels suggest persistence of racial/ethnic disparities in exposures. Similarly elevated maternal PBDE/OH-PBDE concentrations were seen among African American women when compared to Latina women in three separate pregnancy cohorts (Horton et al. 2013; Cowell et al. 2017; Parry et al. 2018). As noted previously (Zota et al. 2010; James-Todd et al. 2016), racial disparities may be related to differences in exposure to PBDE-contaminated dust released from treated furniture and poorer housing quality, as seen in a study of dust levels in California homes

(Whitehead et al. 2013). An alternative explanation for the observed differences in body burdens by race/ethnicity may be due to differences in PBDE metabolism by race/ethnicity.

In our cohort restricted to overweight and obese pregnant women, we still found pre-pregnancy BMI was significantly associated with increased maternal PBDE concentrations after lipid adjustment. Adiposity is linked to higher PBDE concentrations, as PBDEs are lipophilic and stored in adipose tissue. Growing evidence also suggest these chemicals have an obesogenic effect, and may play a causal role in adipogenesis (Janesick and Blumberg 2016). In our same study population, Zota et al. (2018) found obesity modified the association between certain persistent organohalogens and biomarkers of inflammation during pregnancy. Given the proportion of women entering pregnancy as overweight or obese has been increasing in the U.S. (Deputy et al. 2018), additional research is needed to examine the interplay of obesity and chemical concentrations during pregnancy, and their impact on health.

We detected the three most common non-dioxin-like PCB congeners (PCB-138, -153, -180) in more than half of the population, with PCB-153 measured in most serum samples. PCB-153 and PCB-180 were highly correlated with each other, and moderately correlated with congener 138. It is not surprising that age was positively associated with higher PCB concentrations in our population, reflecting the bioaccumulative nature of these highly lipophilic compounds. Younger women are less likely to have been exposed to PCB-containing consumer products as production and use have been phased out since 1979. Our study also indicates that women living at or below poverty had lower PCB levels, similar to other studies examining socioeconomic status and PCB concentrations (Borrell et al. 2004; Vrijheid et al. 2012). Underlying justification of socioeconomic differences in PCB concentrations have been poorly explained. One explanation is socioeconomic differences in fish consumption, which is a strong predictor of PCB concentrations (Xue et al. 2014). Further exploration into this potential disparity is warranted.

We used three unsupervised approaches to group chemicals into mixtures, all with differing purposes to characterize multiple chemical exposures in our population. Our use of a maternal “chemical burden” sum score revealed all participants had all four chemical classes present at concentrations in the top 25th percentile relative to the study population in their blood during pregnancy. Our use of this metric which characterized across-class chemical profiles during pregnancy is supported by conclusions of concomitant multi-chemical exposures from other pregnancy cohorts (Woodruff et al. 2011; Robinson et al. 2015; Fisher et al. 2016; Kalloo et al. 2018). Hierarchical clustering and PCA had similar results for grouping chemical exposures. Although the “chemical burden” sum score identified women who had relatively high concentrations of multiple chemicals in their serum, the apparent lack of cross-chemical-class groupings suggest that exposure sources and metabolic pathways vary across chemical classes. These unsupervised methods to aggregate chemical exposures rely on statistical associations such as Euclidian distance or correlations to determine groupings. A recent study, which used unsupervised methods to examine predictors of chemical mixtures during pregnancy, found that determinants of chemical mixtures were similar to the determinants of individual chemicals. They also found evidence of racial/ethnic differences among certain chemical groupings (Kalloo et al. 2018). To better

understand the association between chemical mixtures and adverse health outcomes, we aim to employ supervised methods in future epidemiologic analyses. A previous study by our research group has already reported an association between prenatal chemical mixtures and inflammation biomarkers using weighted quantile sum regression (Zota et al. 2018). Our current study identifies potential characteristics associated with chemical mixtures which can better inform future epidemiologic analyses and development of exposure reduction strategies.

Our study had a number of strengths and limitations. We were able to examine the chemical burden of an understudied population with multiple social and biological stressors. This historically understudied population of low-income, overweight, pregnant women may be uniquely vulnerable to environmental toxicants since their social positions, existing co-morbidities, and life stage may independently and synergistically amplify the adverse health effects of environmental toxicants. We also employed multiple dietary measures not commonly used in environmental health studies, including food insecurity and AHEI-P. Given our results with PFOS and food insecurity, future exploration of the relationship between dietary quality and chemical concentrations during pregnancy is warranted. We were also able to rely on biological measurements and saw consistency when utilizing multiple aggregation techniques.

Our analysis was limited to evaluating sociodemographic characteristics, so sources and timing of maternal exposure to organohalogen compounds were unknown. As this was a cross-sectional analysis, participant characteristics and chemical measurements were gathered simultaneously during pregnancy, and therefore, no causal associations can be inferred due to lack of temporality. Given each persistent organohalogen has a differing half-life in the body, our cross-sectional design and use of single-spot measurements may further introduce bias. For example, it is possible that our two PFAS groupings were based on differences in biological persistence rather than common sources of exposure. As with any biological samples, serum organohalogen biomarker measurements are subject to non-differential exposure misclassification. Given the population studied is restricted to lower-income overweight/obese pregnant women from California, and because the original study design was intended to test an intervention, the results will likely have low generalizability. With the study population comprised of women in their second trimester, it is also feasible that the results may not reflect the association among other pregnant women at different times in their pregnancy, a phenomenon referred to as left truncation. Lastly, the study population's small sample size limited our ability to perform more in-depth analyses on specific subgroups, may have reduced the precision of exposure groupings, and could have led to chance findings.

In summary, our study identified sociodemographic characteristics of both individual organohalogen chemicals and chemical groupings in an overweight/obese pregnancy cohort, adding to the growing body of literature on chemical stressors during pregnancy. Further, evidence of racial/ethnic disparities in PBDE levels, and food insecurity associated with higher PFOS levels, were identified in pregnant women, underscoring the need to target disproportionately impacted communities for environmental public health intervention.

Future population health studies should utilize multiple statistical methods to understand environmental chemical exposures and their health effects across the life course.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Hierarchically-clustered Spearman correlations of individual and total sum organohalogen concentrations in maternal serum (N=98)

Note: Strong correlation = (+/-) 0.60 to 1.00, moderate correlation = (+/-) 0.40 to 0.59, and weak correlation = 0.00 to (+/-) 0.39.

Figure 2.
Frequency of “chemical burden” sum count score in 98 maternal serum samples (N=14 chemicals)

Baseline maternal serum PBDE (ng/g lipid) and OH-PBDE (ng/mL) concentrations among MAMAS participants by select characteristics (N=98)

Table 1

	N (%)	GM (GSE), PBDEs (ng/g lipid)				GM (GSE), OH-PBDEs (ng/mL)			
		BDE-47	BDE-99	BDE-100	BDE-153	ΣPBDEs	5-OH-BDE-47	6-OH-BDE-47	ΣOH-PBDEs
Total population	98 (100)	32.55 (2.76)	8.32 (0.64)	4.88 (0.44)	8.35 (0.76)	57.35 (4.48)	0.004 (0.001)	0.004 (0.001)	0.01 (0.002)
MDL (% > MDL)		0.05 (100.0)	0.03 (88.8)	0.01 (79.6)	0.01 (90.8)	---	0.003 (50.0)	0.003 (54.1)	---
White/Other	33 (33.67)	27.82 (2.80)	6.94 (0.72)	4.11 (0.51)	8.06 (1.30)	50.64 (4.84)	0.003 (0.001)	0.004 (0.001)	0.01 (0.002)
African American	32 (32.65)	44.53 (7.88)	10.90 (1.70)	7.10 (1.34)	13.34 (1.67)	79.79 (12.79)	0.01 (0.003)	0.01 (0.003)	0.02 (0.01)
Latina	33 (33.67)	28.12 (3.90)	7.67 (0.95)	4.03 (0.53)	5.50 (0.79)	47.15 (5.91)	0.003 (0.001)	0.002 (0.0003)	0.005 (0.001)
27 years at enrollment	51 (52.04)	36.63 (3.57)	9.37 (0.85)	5.83 (0.60)	8.27 (1.12)	63.31 (5.91)	0.004 (0.001)	0.004 (0.001)	0.01 (0.002)
> 27 years at enrollment	47 (47.96)	28.64 (3.98)	7.31 (0.90)	4.02 (0.59)	8.45 (1.02)	51.51 (6.48)	0.003 (0.001)	0.004 (0.001)	0.01 (0.002)
2011 poverty level	44 (47.83)	35.13 (4.74)	8.90 (1.11)	5.38 (0.74)	8.29 (1.13)	60.59 (7.61)	0.005 (0.001)	0.003 (0.001)	0.01 (0.002)
> 2011 poverty level	48 (52.17)	30.12 (3.51)	7.69 (0.80)	4.61 (0.59)	9.13 (1.13)	55.00 (5.88)	0.003 (0.001)	0.005 (0.001)	0.01 (0.002)
HS graduate	34 (34.69)	39.20 (5.69)	10.68 (1.41)	5.52 (0.84)	8.89 (1.32)	67.46 (9.10)	0.01 (0.002)	0.004 (0.001)	0.01 (0.003)
> HS graduate	64 (65.31)	29.50 (3.01)	7.29 (0.66)	4.57 (0.51)	8.08 (0.93)	52.60 (4.95)	0.003 (0.001)	0.004 (0.001)	0.01 (0.002)
Single or other status	33 (33.67)	30.79 (3.56)	8.21 (0.87)	4.60 (0.61)	8.36 (1.30)	54.97 (5.92)	0.004 (0.001)	0.004 (0.001)	0.01 (0.002)
In-relationship	65 (66.33)	33.49 (3.79)	8.37 (0.86)	5.03 (0.60)	8.35 (0.94)	58.59 (6.11)	0.004 (0.001)	0.004 (0.001)	0.01 (0.002)
Marginal/high food security	58 (61.70)	31.34 (3.53)	7.98 (0.80)	4.85 (0.58)	8.72 (1.08)	56.56 (5.84)	0.004 (0.001)	0.005 (0.001)	0.01 (0.002)
Low/very low food security	36 (38.30)	34.06 (4.74)	8.93 (1.14)	4.77 (0.72)	7.69 (1.13)	58.13 (7.59)	0.004 (0.001)	0.003 (0.001)	0.01 (0.002)
<50th %ile score, AHEI-P	47 (49.47)	34.28 (3.83)	8.78 (0.80)	4.98 (0.63)	8.00 (1.15)	59.25 (6.19)	0.004 (0.001)	0.003 (0.001)	0.01 (0.002)
50th %ile score, AHEI-P	48 (50.53)	30.58 (4.03)	8.02 (1.01)	4.74 (0.65)	8.58 (1.03)	55.01 (6.68)	0.003 (0.001)	0.004 (0.001)	0.01 (0.002)
< 14 weeks gestational age ²	25 (25.51)	27.18 (4.07)	7.66 (0.90)	3.69 (0.59)	6.86 (1.20)	47.72 (6.40)	0.003 (0.001)	0.002 (0.001)	0.01 (0.002)
14 weeks gestational age ²	73 (74.49)	34.63 (3.48)	8.56 (0.81)	5.37 (0.57)	8.94 (0.94)	61.07 (5.69)	0.004 (0.001)	0.005 (0.001)	0.01 (0.002)
< 30 kg/m ² BMI ³	54 (55.10)	28.18 (3.03)	7.43 (0.73)	3.97 (0.46)	8.23 (0.85)	50.38 (4.90)	0.003 (0.001)	0.003 (0.001)	0.01 (0.001)
30 kg/m ² BMI ³	44 (44.90)	38.86 (5.04)	9.56 (1.13)	6.30 (0.83)	8.51 (1.35)	67.23 (8.22)	0.006 (0.002)	0.005 (0.001)	0.02 (0.004)
Nulliparous	46 (46.94)	34.04 (4.40)	8.75 (1.00)	5.46 (0.75)	7.91 (1.27)	60.11 (7.40)	0.005 (0.001)	0.005 (0.001)	0.01 (0.003)
Multiparous	52 (53.06)	31.30 (3.47)	7.96 (0.83)	4.42 (0.52)	8.76 (0.84)	55.01 (5.44)	0.004 (0.001)	0.003 (0.001)	0.01 (0.002)
Never smoker	49 (52.69)	32.63 (4.14)	8.52 (0.96)	5.06 (0.65)	7.35 (0.92)	56.25 (6.60)	0.004 (0.001)	0.003 (0.001)	0.01 (0.002)
Current or former smoker	44 (47.31)	32.97 (3.94)	8.41 (0.91)	4.72 (0.64)	9.54 (1.39)	59.56 (6.59)	0.004 (0.001)	0.004 (0.001)	0.01 (0.003)

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¹Maternal serum concentrations were log-transformed
²Measured at baseline (range: 10–24 weeks gestation)
³Pre-pregnancy BMI confirmed with medical records.

Abbreviations: GM = geometric mean; GSE geometric standard error of the mean; AHEL-P = Alternative Healthy Eating Index modified for Pregnancy.

Table 2:

Baseline maternal serum PCB (ng/g lipid) and PFAS (ng/mL) concentrations among MAMAS cohort participants by select characteristics (N=98)

	N (%)	GM (GSE) ¹ , PCBs (ng/g lipid)					GM (GSE) ¹ , PFASs (ng/mL)				
		PCB-138	PCB-153	PCB-180	ΣPCBs	PFNA	PFDeA	PFOS	PFOA	PFHxS	ΣPFASs
Total population	98 (100)	2.26 (0.21)	3.95 (0.25)	2.08 (0.21)	8.88 (0.61)	0.58 (0.03)	0.18 (0.01)	2.95 (0.16)	1.21 (0.09)	0.54 (0.04)	5.94 (0.27)
MDL (% > MDL)		0.01 (56.1)	0.01 (87.8)	0.01 (57.1)	---	0.07 (100.0)	0.12 (69.4)	0.27 (100.0)	0.05 (98.0)	0.04 (99.0)	---
White/Other	33 (33.67)	2.89 (0.46)	4.24 (0.55)	2.55 (0.41)	10.41 (1.40)	0.51 (0.05)	0.17 (0.02)	2.74 (0.26)	1.19 (0.14)	0.52 (0.07)	5.48 (0.44)
African American	32 (32.65)	1.95 (0.32)	4.01 (0.35)	1.89 (0.22)	8.44 (0.83)	0.67 (0.06)	0.18 (0.02)	2.77 (0.26)	1.32 (0.13)	0.48 (0.06)	5.78 (0.45)
Latina	33 (33.67)	2.05 (0.30)	3.64 (0.40)	1.86 (0.26)	7.95 (0.90)	0.58 (0.05)	0.19 (0.03)	3.37 (0.33)	1.12 (0.19)	0.63 (0.09)	6.62 (0.50)
27 years at enrollment	51 (52.04)	1.59 (0.16)	3.05 (0.22)	1.33 (0.11)	6.28 (0.45)	0.57 (0.04)	0.19 (0.02)	2.99 (0.25)	1.14 (0.14)	0.57 (0.06)	6.04 (0.42)
> 27 years at enrollment	47 (47.96)	3.31 (0.45)	5.23 (0.48)	3.38 (0.38)	12.92 (1.19)	0.60 (0.04)	0.17 (0.02)	2.91 (0.21)	1.28 (0.11)	0.51 (0.06)	5.83 (0.34)
2011 poverty level	44 (47.83)	1.82 (0.22)	3.64 (0.31)	1.72 (0.19)	7.55 (0.67)	0.60 (0.05)	0.18 (0.02)	2.93 (0.22)	1.23 (0.14)	0.56 (0.06)	6.03 (0.35)
> 2011 poverty level	48 (52.17)	3.03 (0.40)	4.52 (0.44)	2.68 (0.32)	11.08 (1.10)	0.55 (0.04)	0.18 (0.02)	2.93 (0.26)	1.17 (0.13)	0.52 (0.06)	5.80 (0.43)
HS graduate	34 (34.69)	1.69 (0.23)	3.28 (0.29)	1.57 (0.20)	7.01 (0.66)	0.61 (0.06)	0.19 (0.03)	3.00 (0.25)	1.24 (0.16)	0.51 (0.07)	6.09 (0.39)
> HS graduate	64 (65.31)	2.64 (0.30)	4.37 (0.36)	2.42 (0.25)	10.06 (0.88)	0.57 (0.04)	0.18 (0.02)	2.92 (0.21)	1.19 (0.11)	0.56 (0.06)	5.86 (0.36)
Single or other status	33 (33.67)	2.30 (0.37)	4.20 (0.50)	2.50 (0.36)	9.54 (1.15)	0.67 (0.07)	0.19 (0.03)	3.03 (0.24)	1.38 (0.13)	0.49 (0.06)	6.14 (0.42)
In-relationship	65 (66.33)	2.24 (0.25)	3.83 (0.29)	1.89 (0.19)	8.56 (0.70)	0.54 (0.03)	0.18 (0.02)	2.91 (0.21)	1.13 (0.12)	0.57 (0.06)	5.84 (0.35)
Marginal/high food security	58 (61.70)	2.34 (0.30)	4.10 (0.34)	2.21 (0.25)	9.39 (0.86)	0.57 (0.04)	0.18 (0.02)	2.66 (0.18)	1.14 (0.11)	0.51 (0.05)	5.51 (0.31)
Low/very low food security	36 (38.30)	2.09 (0.29)	3.77 (0.38)	1.89 (0.23)	8.14 (0.84)	0.59 (0.05)	0.17 (0.02)	3.47 (0.34)	1.30 (0.16)	0.59 (0.08)	6.65 (0.54)
<50th %ile score, AHEI-P	47 (49.47)	2.15 (0.26)	3.88 (0.31)	1.83 (0.20)	8.38 (0.71)	0.59 (0.05)	0.16 (0.02)	2.98 (0.24)	1.15 (0.13)	0.49 (0.06)	5.86 (0.40)
50th %ile score, AHEI-P	48 (50.53)	2.37 (0.33)	4.06 (0.41)	2.35 (0.31)	9.45 (1.03)	0.57 (0.04)	0.19 (0.02)	2.96 (0.23)	1.24 (0.13)	0.60 (0.07)	6.05 (0.37)
< 14 weeks gestational age ²	25 (25.51)	1.89 (0.38)	3.72 (0.52)	1.97 (0.33)	8.23 (1.18)	0.63 (0.07)	0.20 (0.03)	3.05 (0.26)	1.37 (0.24)	0.56 (0.07)	6.22 (0.54)
14 weeks gestational age ²	73 (74.49)	2.41 (0.24)	4.04 (0.29)	2.12 (0.20)	9.11 (0.70)	0.57 (0.03)	0.18 (0.02)	2.92 (0.20)	1.15 (0.09)	0.53 (0.05)	5.85 (0.31)
< 30 kg/m ² BMI ³	54 (55.10)	2.14 (0.27)	4.44 (0.39)	2.44 (0.29)	9.47 (0.90)	0.58 (0.05)	0.18 (0.02)	3.01 (0.23)	1.19 (0.13)	0.52 (0.05)	5.99 (0.39)
30 kg/m ² BMI ³	44 (44.90)	2.42 (0.33)	3.43 (0.30)	1.70 (0.19)	8.20 (0.79)	0.58 (0.04)	0.18 (0.02)	2.87 (0.24)	1.22 (0.12)	0.57 (0.07)	5.88 (0.36)
Nulliparous	46 (46.94)	2.34 (0.34)	3.79 (0.39)	1.95 (0.26)	8.91 (0.97)	0.60 (0.04)	0.18 (0.02)	3.12 (0.27)	1.36 (0.13)	0.56 (0.06)	6.41 (0.36)
Multiparous	52 (53.06)	2.19 (0.25)	4.10 (0.32)	2.20 (0.22)	8.85 (0.75)	0.56 (0.04)	0.18 (0.02)	2.81 (0.20)	1.08 (0.12)	0.52 (0.06)	5.55 (0.38)
Never smoker	49 (52.69)	2.29 (0.28)	3.88 (0.36)	1.96 (0.23)	8.61 (0.82)	0.57 (0.04)	0.21 (0.02)	3.01 (0.25)	1.20 (0.14)	0.55 (0.06)	6.11 (0.42)
Current or former smoker	44 (47.31)	2.17 (0.32)	4.04 (0.38)	2.18 (0.27)	9.12 (0.94)	0.58 (0.04)	0.16 (0.02)	2.81 (0.22)	1.20 (0.12)	0.51 (0.06)	5.61 (0.35)

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¹Maternal serum concentrations were log-transformed
²Measured at baseline (range: 10–24 weeks gestation)
³Pre-pregnancy BMI confirmed with medical records.

Abbreviations: GM = geometric mean; GSE = geometric standard error of the mean; AHEIP = Alternative Healthy Eating Index modified for Pregnancy; BMI = body mass index.

Table 3.

Associations between log-transformed maternal serum PBDE (ng/g lipid) and OH-PBDE (ng/mL) concentrations and participant characteristics

	African American ¹		Latina ¹		>HS graduate ²		Pre-pregnancy BMI		Parity		Adj R-squared
	β (95% CI)	p	β (95% CI)	p	β (95% CI)	p	β (95% CI)	p	β (95% CI)	p	
ln-BDE-47	0.37 (-0.04, 0.78)	0.07	-0.04 (-0.44, 0.36)	0.85	0.26 (-0.09, 0.61)	0.15	0.04 (-0.001, 0.08)	0.05	-0.13 (-0.29, 0.04)	0.14	0.15
ln-BDE-99	0.36 (-0.01, 0.73)	0.06	0.02 (-0.34, 0.38)	0.92	0.36 (0.04, 0.69)	0.03	0.02 (-0.01, 0.06)	0.16	-0.12 (-0.27, 0.03)	0.12	0.15
ln-BDE-100	0.45 (0.03, 0.87)	0.04	-0.03 (-0.44, 0.38)	0.89	0.15 (-0.21, 0.52)	0.40	0.06 (0.02, 0.10)	0.004	-0.19 (-0.36, -0.01)	0.04	0.21
ln-BDE-153	0.47 (0.03, 0.91)	0.03	-0.41 (-0.83, 0.02)	0.06	0.13 (-0.25, 0.50)	0.50	0.01 (-0.03, 0.05)	0.56	-0.06 (-0.24, 0.12)	0.50	0.17
ln-ΣPBDEs	0.37 (-0.001, 0.75)	0.05	-0.12 (-0.48, 0.25)	0.53	0.24 (-0.08, 0.57)	0.13	0.03 (-0.001, 0.07)	0.06	-0.13 (-0.28, 0.02)	0.09	0.17
ln-5-OH-BDE-47	0.97 (-0.02, 1.96)	0.05	-0.23 (-1.20, 0.74)	0.64	0.54 (-0.31, 1.40)	0.21	0.07 (-0.03, 0.16)	0.16	-0.41 (-0.81, -0.001)	0.05	0.14
ln-6-OH-BDE-47	0.70 (-0.10, 1.49)	0.08	-0.93 (-1.70, -0.16)	0.02	0.13 (-0.56, 0.81)	0.71	0.05 (-0.03, 0.12)	0.19	-0.28 (-0.60, 0.05)	0.10	0.21
ln-ΣOH-PBDEs	0.58 (-0.08, 1.24)	0.08	-0.85 (-1.49, -0.21)	0.01	0.31 (-0.26, 0.88)	0.28	0.04 (-0.02, 0.11)	0.17	-0.31 (-0.58, -0.04)	0.02	0.24

Categorical reference groups:

¹ White or other race/ethnicity² high school graduate.

* Bolded value indicates p<0.05

Table 4.

Associations between log-transformed maternal serum PCB (ng/g lipid) and PFAS (ng/mL) concentrations and participant characteristics

	Maternal age (years)		Poverty level ¹		Low/very low food security ²		Adj R-squared
	β (95% CI)	p	β (95% CI)	p	β (95% CI)	p	
ln-PCB-138	0.08 (0.05, 0.11)	<0.0001	-0.35 (-0.67, -0.02)	0.04			0.31
ln-PCB-153	0.06 (0.04, 0.08)	<0.0001	-0.10 (-0.32, 0.13)	0.40			0.29
ln-PCB-180	0.10 (0.07, 0.12)	<0.0001	-0.25 (-0.50, 0.01)	0.06			0.47
ln- Σ PCBs	0.08 (0.06, 0.09)	<0.0001	-0.23 (-0.44, -0.02)	0.03			0.47
ln-PFNA					0.04 (-0.18, 0.25)	0.74	0.001
ln-PFDeA					-0.07 (-0.41, 0.28)	0.71	0.002
ln-PFOS					0.27 (0.04, 0.50)	0.02	0.05
ln-PFOA					0.13 (-0.19, 0.45)	0.43	0.007
ln-PFHxS					0.14 (-0.20, 0.47)	0.42	0.007
ln- Σ PFAS					0.19 (-0.003, 0.38)	0.05	0.04

Categorical reference groups:

¹ >2011 federal poverty level

² marginal/high food security

* Bolded value indicates p<0.05

Maternal serum chemical loading on the first four principal components, based on an eigenvalue of >50 for varimax rotated components

Table 5.

Analyte	Component 1	Component 2	Component 3	Component 4
BDE-47				
BDE-99				
BDE-100				
BDE-153				
5-OHBDDE-47				
6-OHBDDE-47				
PCB-138				
PCB-153				
PCB-180				
PFNA				
PFDeA				
PFOS				
PFOA				
PFHxS				
Variance Explained:	28%	18%	16%	8%

Table 6.

Associations between principal components and participant sociodemographic characteristics.

Predictor	PC1 (BDE-47,-99,-100, -153, 5-OHBDDE-47, 6-OHBDDE-47)	PC2 (PCB-138, -153, -180)	PC3 (PFNA, PFDeA, PFOA)	PC4 (PFOS, PFHxS)
	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)
White or Other (Ref)	1	1	1	1
African American	0.62 (0.16, 1.09)	-0.07 (-0.43, 0.30)	0.28 (-0.24, 0.81)	0.10 (-0.41, 0.61)
Latina	-0.10 (-0.56, 0.36)	0.01 (-0.35, 0.37)	-0.06 (-0.57, 0.45)	0.48 (-0.18, 0.99)
Maternal age (years)	-0.01 (-0.05, 0.02)	0.13 (0.10, 0.16)	0.005 (-0.04, 0.04)	0.02 (-0.17, 0.06)
Parity	-0.17 (-0.38, 0.03)	-0.24 (-0.40, -0.08)	0.02 (-0.21, 0.24)	-0.20 (-0.42, 0.03)
Pre-pregnancy BMI (kg/m ²)	0.05 (0.01, 0.10)	-0.01 (-0.05, 0.02)	-0.01 (-0.05, 0.04)	0.01 (-0.04, 0.06)

* Bolded value indicates $p < 0.05$.

Abbreviations: PC = principal component.