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Variability and predictors of serum perfluoroalkyl substance concentrations during pregnancy and early childhood

Samantha L. Kingsley[a,*], Melissa N. Eliot[a], Karl T. Kelsey[a,b], Antonia M. Calafat[c], Shelley Ehrlich[d], Bruce P. Lanphear[e,f], Aimin Chen[g], Joseph M. Braun[a]

[a] Department of Epidemiology, Brown University, School of Public Health, Providence, RI, USA
[b] Department of Laboratory Medicine & Pathology, Brown University, Providence, RI, USA
[c] Division of Laboratory Sciences, National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, GA, USA
[d] Division of Biostatistics and Epidemiology, Cincinnati Children's Hospital Medical Center, University of Cincinnati, College of Medicine, Cincinnati, OH, USA
[e] Child and Family Research Institute, BC Children's and Women's Hospital, Vancouver, British Columbia, Canada
[f] Faculty of Health Sciences, Simon Fraser University, Burnaby, Canada
[g] Department of Environmental Health, University of Cincinnati, Cincinnati, OH, USA

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ABSTRACT

Exposure to poly- and perfluoroalkyl substances (PFAS), ubiquitous and persistent environmental contaminants, could be associated with adverse health outcomes, but there are limited longitudinal data assessing patterns and predictors of exposure during childhood. We quantified concentrations of eight different PFAS in sera collected from women during pregnancy and children at delivery and ages 3 and 8 years in 367 mother-child pairs enrolled in a prospective cohort from 2003 to 2006. In general, median childhood PFAS concentrations increased from birth to age 3 and then decreased by age 8. Maternal serum PFAS concentrations during pregnancy were strongly correlated with cord serum concentrations ($0.76 < r < 0.94$), but were weakly correlated with childhood concentrations ($0.12 < r < 0.30$). Several sociodemographic factors were associated with maternal PFAS concentrations, including income, race, and parity. In children, serum PFAS concentrations were associated with maternal age at delivery, race, parity, and child age. Breastfeeding duration was positively associated with childhood PFAS concentrations at ages 3 and 8 years. In addition, stain repellant use was associated with higher perfluorooctanoic acid and perfluorohexane sulfonic acid concentrations at age 8 years. Serum PFAS concentrations are higher during early childhood, a potentially sensitive period of development, and were highest among breastfed children.

1. Introduction

Poly- and perfluoroalkyl substances (PFAS) are a group of synthetic fluorinated chemicals widely used in industrial and consumer products, including some stain and water resistant coatings, non-stick cookware, food container coatings, floor polish, fire-fighting foam, carpets, apparel, upholstery and industrial surfactants (Agency for Toxic Substances and Disease Registry, 2015; DeWitt, 2015; European Food Safety Authority, 2008). The strong carbon-fluoride bond makes them resistant to environmental degradation. Some PFAS have biological half-lives in humans ranging from 3.8 to 8.5 years, and are ubiquitous in the environment and human sera from both children and adults (Andersen et al., 2013; Apelberg et al., 2007; Darrow et al., 2013; Fei et al., 2010; Fromme et al., 2010; Grandjean et al., 2012; Halldorsson et al., 2012; Hamm et al., 2010; Inoue et al., 2004; Jain, 2013a; Javins et al., 2013; Lee et al., 2013; Olsen et al., 2007; Whitworth et al., 2012; Zhang et al., 2013).

Production of PFAS began in the 1950s and peaked in the 1990s; in the United States, voluntary industry phase-out of perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA) began in the early 2000s (Bowman, 2015; Environmental Protection Agency, 2000; Fromme et al., 2009; Kato et al., 2011b). Humans are primarily exposed to PFAS through food and water, and, to a lesser extent, via dust exposure (ingestion and inhalation) (Fromme et al., 2009; Lorber and Egeghy, 2011). Human exposures to PFAS have attracted considerable attention in recent years due to PFAS widespread use, persistence in humans and the environment, and association with a variety of health outcomes in humans (Agency for Toxic Substances and Disease Registry, 2015; Antignac et al., 2013; Braun, 2016; Cardenas et al., 2017; DeWitt, 2015; Emmett et al., 2006; Environmental Protection

* Correspondence to: Brown University School of Public Health, Box G-S121-2, Providence, RI 02912, USA.
E-mail address: Samantha_Kingsley@brown.edu (S.L. Kingsley).

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Agency, 2013; European Food Safety Authority, 2008; Fleisch et al., 2017; Lopez-Espinosa et al., 2011; Maisonet et al., 2012; Mora et al., 2017; Office of Health Assessment and Translation, National Toxicology Program, 2016; Shoeib et al., 2005; Starling et al., 2017). Additionally, the detection of PFAS in cord blood strongly shows that these chemicals can cross the placenta and reach the developing fetus (Beesoon et al., 2011; Glynn et al., 2012; Kim et al., 2011; Porpora et al., 2013).

There is limited knowledge about the patterns and determinants of PFAS exposure in mother – child dyads and even less about longitudinal patterns in childhood (Winkens et al., 2017). Longer duration of breastfeeding and higher parity are associated with reduced PFAS concentrations in mothers (Berg et al., 2014; Brantsaeter et al., 2013; Jain, 2013b; Kato et al., 2014; Lauritzen et al., 2016; Manzano-Salgado et al., 2016; Mondal et al., 2014; Morck et al., 2015; Ode et al., 2013; Sagiv et al., 2015) and higher PFAS concentrations in children (Mondal et al., 2014; Papadopoulou et al., 2016; Wu et al., 2015). Prior studies have also identified age (Berg et al., 2014; Harris et al., 2017; Manzano-Salgado et al., 2016; Morck et al., 2015; Sagiv et al., 2015), race (Harris et al., 2017; Kato et al., 2014), contaminated drinking water (DeWitt, 2015; Frisbee et al., 2009; Herrick et al., 2017; Worley et al., 2017), and some features of diet (Berg et al., 2014; Brantsaeter et al., 2013; Halldorsson et al., 2008; Manzano-Salgado et al., 2016; Wu et al., 2015) as predictors of PFAS concentrations in women and children. However, these studies have either focused solely on maternal PFAS concentrations (Berg et al., 2014; Brantsaeter et al., 2013; Halldorsson et al., 2008; Jain, 2013b; Kato et al., 2014; Lauritzen et al., 2016; Manzano-Salgado et al., 2016; Sagiv et al., 2015) or only measured PFAS once in women and children (Berg et al., 2014; Brantsaeter et al., 2013; Harris et al., 2017; Jain, 2013b; Lauritzen et al., 2016; Manzano-Salgado et al., 2016; Morck et al., 2015; Sagiv et al., 2015). Few studies have examined PFAS concentrations in newborns and young children (Harris et al., 2017; Morck et al., 2015; Papadopoulou et al., 2016; Pinney et al., 2014; Schecter et al., 2012; Wu et al., 2015; Ye et al., 2017).

Exposure to PFAS during gestation or childhood is of particular concern because biologically protective mechanisms are not fully developed. Thus, it is important to understand the longitudinal patterns of PFAS concentrations in pregnant women and their children and identify predictors of PFAS exposure. The purpose of this study was to characterize the patterns and predictors of serum PFAS concentrations during pregnancy, at delivery, and during childhood.

2. Methods

2.1. Study participants

The Health Outcomes and Measures of the Environment (HOME) Study is a prospective pregnancy and birth cohort that enrolled pregnant women living in the Cincinnati, Ohio metropolitan area from March 2003 to January 2006. Participant recruitment and eligibility criteria have been previously described (Braun et al., 2017). About 37% of the 1263 women eligible were enrolled (n = 468) in the study. Of those enrolled, we restricted our analyses to women who delivered singleton births and had a gestational PFAS measurement (n = 367), and to singleton children who had at least one PFAS measurement at birth, 3-year, or 8-year visits (n = 333). The institutional review board (IRB) of Cincinnati Children's Hospital Medical Center (CCHMC) approved the study protocol. The Centers for Disease Control and Prevention (CDC) deferred to CCHMC IRB as the IRB of record. Women provided written informed consent to participate in the study for themselves and their children after research assistants explained the study protocols.

2.2. Serum PFAS concentrations

We collected venous blood samples from mothers at prenatal clinic appointments at 16 and 26 weeks of gestation, and within 48 h of delivery. Children's samples included venous cord blood at birth and venous blood at the 3-year and 8-year clinic visits. We measured serum concentrations of eight PFASs: PFOA, PFOS, perfluorononanoic acid (PFNA), perfluorohexane sulfonic acid (PFHxS), 2-(N-methylperfluorooctane sulfonamido) acetic acid (Me-PFOSA-AcOH, also known as MeFOSAA), 2-(N-ethyl-perfluorooctane sulfonamido) acetic acid (Et-PFOSA-AcOH, also known as EtFOSAA), perfluorodecanoic acid (PFDeA, also known as PFDA), and perfluorooctane sulfonamide (PFOSA, also known as FOSA) at the CDC laboratory. As previously described, concentrations of all PFAS chemicals were quantified in 100 µL of serum using online solid phase extraction coupled to high performance liquid chromatography-isotope dilution with tandem mass spectrometry (Kato et al., 2011a, 2014). Low-concentration and high-concentration quality control (QC) samples, reagent blanks, and serum blanks were analyzed with the study samples. The analytical methods for the maternal and child samples were the same as those used to quantify PFAS concentrations in NHANES samples for the 2009–2010 and 2011–2012 NHANES cycles, respectively (Centers for Disease Control and Prevention, 2011, 2013). The limits of detection (LOD) were 0.1 ng/mL (PFOA, PFHxS, PFDA, FOSA, EtFOSAA); 0.2 ng/mL (PFOS); 0.082 ng/mL (PFNA); and 0.087 ng/mL (MeFOSAA). We assigned concentrations below the LOD to a value of LOD /√2 (Supplemental Table S1).

2.3. Predictors of PFAS concentrations

We examined demographic, perinatal, and environmental factors - assessed with questionnaires, medical chart reviews, and biomarkers - as predictors of gestational and childhood serum PFAS concentrations. We obtained child sex (male, female) and parity (0, 1, > 1) from medical charts at delivery. Trained research staff administered standardized questionnaires at each study visit to assess demographic and lifestyle characteristics. These characteristics included: maternal age (years), maternal race (non-Hispanic white, non-Hispanic black, other), household income (per $10,000), marital status (married, unmarried), fish consumption during pregnancy (< once a month, 1–3 times a month, 1–3 times a week, > four times a week) and at ages 3 and 8 years (< once a month, > once a month), and duration of any breastfeeding (months, up to 3 years for index child).

Several prior studies have found that smoking, or tobacco smoke exposure biomarkers, is associated with serum PFAS concentrations in other populations (Jain, 2013b; Lauritzen et al., 2016; Sagiv et al., 2015). Based on this, we included serum cotinine as a potential predictor of maternal PFAS concentrations. In addition, if serum cotinine was associated with PFAS concentrations, then we would want to adjust for smoking or secondhand tobacco smoke exposure in studies examining the health effects of PFAS, since tobacco smoke exposure is associated with many health outcomes. Serum cotinine concentrations were measured in samples taken at 16 and 26 weeks of gestation and the two log10-transformed measures were averaged. Childhood serum cotinine concentrations were measured annually from 1 to 4 years of age, and multiple $\log_{10}$-transformed measures were averaged and used in analyses. At the 8-year visit, we administered questionnaires asking about stain repellent use in the last 3 years (any, none), frequency of non-stick cookware use (almost daily, sometimes, rarely, never), and frequency of microwave popcorn consumption (any, none).

2.4. Statistical analyses

We calculated the geometric mean (GM), range, interquartile range (IQR), and median of each PFAS at each visit and used boxplots of natural log-transformed serum PFAS concentrations to examine longitudinal patterns of PFAS exposure. When available, we used the 16-week serum PFAS concentrations to assess gestational exposure; if the 16-week sample was missing, we used 26-week concentrations, or birth concentrations if both were missing. Serum PFAS concentrations were