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Contribution of diet and other factors to the observed levels of selected perfluoroalkyl acids in serum among US children aged 3-11 years☆

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ABSTRACT

Data from National Health and Nutrition Examination Survey for 2013–2014 for children aged 3–11 years (N = 639) were analyzed to evaluate the contribution of diet and other factors in variability associated with the observed levels of seven perfluoroalkyl acids in serum, namely, 2(N-methyl-perfluorooctane sulfonamide) acetic acid (MPAH), perfluorodecanoic acid (PFDE), perfluorononanoic acid (PFNA), perflurorohexane sulfonic acid (PFHxS), linear isomer of PFOA (NPFOA), linear isomer of PFOS (NPFOS), and monomethyl isomer of PFOS (MPFOS). Diet accounted for a low of 18.6% of the total explained variance in the adjusted levels of NPFOA and a high of 72.3% for PFNA. Consumption of meat other than fish and poultry was associated with increased levels of NPFOS ($\beta = 0.00035$, $p < 0.01$) and MPFOS ($\beta = 0.00027$, $p = 0.02$). However, consumption of fish was associated with decreased levels of PFDE ($\beta = -0.00058$, $p = 0.01$). Consumption of eggs was associated with higher levels of PFDE ($\beta = 0.00105$, $p = 0.04$). Higher levels of PFHxS were associated with consumption of fruits and juices ($\beta = 0.00019$, $p = 0.03$). Exposure to environmental tobacco smoke in indoor environments other than home was associated with 12.6% increase in the levels of NPFOA. Boys had higher adjusted geometric mean (AGM) than girls for MPAH (0.88 vs. 0.70 ng/mL, $p = 0.04$) and NPFOS (2.73 vs. 2.27 ng/mL, $p = 0.04$). Non-Hispanic white had higher AGMs than Hispanics for MPAH (0.15 vs. 0.07, $p < 0.01$), for NPFOA (1.98 vs. 1.64 ng/mL, $p < 0.01$), and MPFOS (1.39 vs. 1.18 ng/mL, $p = 0.03$). Non-Hispanic white also had higher AGM than non-Hispanic Asians and others for PFHxS (0.99 vs. 0.63 ng/mL, $p < 0.01$) and NPFOA (1.98 vs. 1.53 ng/mL, $p < 0.01$).

1. Introduction

Perfluoroalkyl acids (PFAA), used in a wide variety of consumer products (Okada et al., 2013), are present everywhere. Selected PFAAs have been found to be present in water sediments (Wang et al., 2017), tap water (Lu et al., 2017), and agricultural soil and grains near industrial parks (Liu et al., 2017a). They have been detected in maternal serum samples (Bjerregaard-Olesen et al., 2017), breast milk (Cariou et al., 2015), and follicular fluids (Petro et al., 2014; McCoy et al., 2017). Adverse effects of prenatal exposure to PFAAs have been investigated by quite a few researchers (Alkhalawi et al., 2016; Kobayashi et al., 2017; Callan et al., 2016; Goudarzi et al., 2016; Okada et al., 2013). Breast feeding has been considered to be an excretion route for mothers (Cariou et al., 2015; Mondal et al., 2014). Exposure to PFAAs have been reported to adversely affect glomerular filtration rate and serum uric acid levels among adolescents aged 12–19 years (Kataria et al., 2015), clinical measures of reproductive health (McCoy et al., 2017), glycemic health (Lin et al., 2009), cardiovascular disease (Shankar et al., 2012), levels of total and non-high-density cholesterol (Nelson et al., 2010), and thyroid homeostasis (Ji et al., 2012; Lopez-Espinosa et al., 2012; Knox et al., 2011; Lin et al., 2012; Melzer et al., 2010; Jain, 2013).

Exposure to PFAAs can occur via contaminated food (Ostertag et al., 2009; Schuetze et al., 2010), food packaging and non-stick cookware (Tittlemier et al., 2006), water (Holzer et al., 2008), indoor air (https://www.atsdr.cdc.gov/pfc/sources_of_exposure.html) and indoor and outdoor dust (Fromme et al., 2009; Kubwabo et al., 2005). Infants can be exposed to PFAAs via breast milk (Cariou et al., 2015). Occupational exposure to PFAAs and subsequent transfer to family members has been documented by Fu et al. (2015). Makey et al. (2017) investigated the possibility of airborne PFAA precursors as a source of exposure to PFAAs in 50 maternal sera samples collected in 2007–2008 from participants in Vancouver, Canada.

PFAAs are used as multi-purpose surfactants or water/oil repellents (Lu et al., 2017). Some of the major PFAAs are: perfluorooctanoic acid (PFOA), perfluorooctane sulfonate (PFOS), perfluorohexane sulfonate

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(PFHxS), and perfluorononanoic acid (PFNA). Elimination half-life of PFOS, PFOA, and PFHxS in serum among humans is 5.4 years, 3.8 years, and 8.5 years respectively (Olsen et al., 2007) and as such, they bio-accumulate over time. Bio-accumulative potential of PFHxS and PFOS was found to be higher than other PFAAs (Fu et al., 2015).

Jain (2014a) used data from NHANES for the period 2003–2008 and contribution of diet and selected risk factors on the serum levels of PFOA, PFOS, PFHxS, and PFNA among US population aged > = 12 years was investigated. Dietary factors accounted for 10.4–21.2% of the explained variability depending on the PFAA. In a Canadian study (Tittlemier et al., 2007), diet was found to account for more than 60% of the total exposure to PFAAs. In an intake study in the population of Flanders, Belgium (Cornelis et al., 2012), dietary intake including from vegetables dominated total intake of PFOA and PFOS. Liu et al. (2017b) reported fish, shellfish, red meat and poultry to be associated with increased PFAAs concentrations in plasma, whereas grains and soy products were found to be inversely associated with PFAAs.

While NHANES data on PFAAs for those aged > = 12 years have been released in the public domain since 2003–2004 NHANES cycle, it was only for 2013–2014 that PFAA data for children aged 3–11 were released. Consequently, this study was undertaken to assess the contribution of diet and demographic factors to the observed levels of selected PFAAs in serum.

2. Materials and methods

2.1. Availability of data for PFAASs

Data for children aged 3–11 years were available for observed levels of 14 PFAAs in serum (https://wwwn.cdc.gov/Nchs/Nhanes/2013-2014/SSPFAC_H.htm), namely, perfluorooctane sulfonamide (PFSA), 2(N-methyl-perfluorooctane sulfonamide) acetic acid (MPAH), 2(N-ethyl-perfluorooctane sulfonamide) acetic acid (EPAH), perfluorodecanoic acid (PFDE), perflurorbutane sulfonic acid (PFBS), perfluoroheptanoic acid (PFHP), PFNA, perfluoroundecanoic acid (PFUA), perfluorododecanoic acid (PFDO), PFHxS, linear isomer of PFOA (NPFOA), branch isomer of PFOA (BPFOA), linear isomer of PFOS (NPFOS), and monomethyl isomer of PFOS (MPFOS). However, percent observations at or above the limit of detection for EPAH, PFBS, PFDO, PFHP, PFSA, PFUA, and BPFOA were too low (Table 1) to do a meaningful data analysis. Consequently, data were analyzed for seven PFAAs, namely, MPAH, MPFOS, NPFOA, NPFOS, PFDE, PFHxS, and PFNA.

Table 1
Percent observations at or above the limit of detection with 95% confidence intervals for selected perfluoroalkyl acids and substances (PFAAs) for children aged 3–11 years. Data from National Health and Nutrition Examination Survey 2013–2014.

PFAS	> = LOD (95% CI)
2(N-ethyl-perfluorooctane sulfonamido) acetic acid	3.4 (1.1 – 5.8)
2(N-methyl-perfluorooctane sulfonamido) acetic acid[a]	53.2 (42.8 – 63.6)
Monomethyl branch isomers of PFOS[a]	100 (100 – 100)
Linear perfluorooctanoate[a]	99.8 (99.3 – 100.3)
Linear perfluorooctane sulfonate[a]	100 (100 – 100)
Perfluorobutane sulfonic acid	4.9 (1.9 – 8.0)
Pefluorodecanoic acid[a]	47.3 (39.1 – 55.5)
Perflurododecanoic acid	0 (0 – 0)
Perfluoroheptanoic acid	19.2 (12.8 – 25.6)
Perfluorohexane sulfonic acid[a]	99.9 (99.7 – 100.1)
Perfluorononanoic acid[a]	99.9 (99.6 – 100.1)
Perfluorooctane sulfonmide	3.2 (1.0 – 5.5)
Perfluoroundecanoic acid	27.5 (21.5 – 33.5)
Branch isomers of perfluorooctanoate	28.2 (20.3 – 36.1)

[a] Selected for data analysis.

Table 2
Unweighted sample sizes by gender and race/ethnicity used in unadjusted and adjusted analyses.

	Unadjusted Analysis		Adjusted Analysis	
	N	%	N	%
Total	639	100	526	100.0
Boys	343	53.7	285	54.2
Girls	296	46.3	241	45.8
Non-Hispanic White	166	26.0	138	26.2
Non-Hispanic Black	160	25.0	135	25.7
Hispanic	220	34.4	180	34.2
Non-Hispanic Asian	49	7.7		
Other	44	6.9		
Non-Hispanic Asian and others			73	13.9

The total sample size available for analysis was 639 (Table 2) but because of the missing values for independent variables, the total sample size available for doing the adjusted analyses was limited to 526 (Table 2).

2.2. Data on exposure to environmental tobacco smoke

Data on exposure to environmental tobacco smoke (ETS) inside children's home (https://wwwn.cdc.gov/Nchs/Nhanes/2013-2014/SMQFAM_H.htm) were available for two variables, namely, number of persons smoking inside the home and if so, number of days they smoked inside home during the last 7 days. A variable ETS_H was generated to indicate the exposure to ETS at home (no, yes). Data were also available for exposure to ETS (https://wwwn.cdc.gov/Nchs/Nhanes/2013-2014/SMQSHS_H.htm) during the last 7 days when inside a restaurant, taking ride in a car, when inside somebody else's home, and when in another indoor area. A variable indicating exposure to ETS (no, yes) from one or more other indoor environments was generated for use in the analysis.

2.3. Data on dietary variables

Jain (2014a) used data from NHANES individual food files for the survey period 2003–2008 to evaluate the association of 17 food groups with the levels of selected PFAAs. These food groups were: cheese, milk and milk products other than cheese, fish, poultry, eggs, dry beans, grain products, fruits and juices, dark green vegetables, tomatoes, vegetables other than tomatoes and dark green vegetables, fats and oils, sugars and sweets, non-alcoholic beverages, alcoholic beverages, and non-carbonated water. For this study, since alcoholic beverages were not an applicable choice, data in grams consumed for other 16 food groups were used to assess the association between consumption of these foods with 7 PFAAs listed above. Data were available at https://wwwn.cdc.gov/Nchs/Nhanes/2013-2014/DR1IFF_H.htm. Data on total intake (https://wwwn.cdc.gov/Nchs/Nhanes/2013-2014/DR1TOT_H.htm) of calories, fats, and caffeine during the last 24 h were also used.

2.4. Treatment of observations below the limit of detection (LOD)

When percent observations > = LOD are at least 60%, all observation below LOD are usually substituted as LOD/Sqrt(2) as proposed by Hornung and Reed (1990). However, depending up on the percent observations below the LOD, substituting observations below LOD as LOD/Sqrt(2) may adversely affect estimation of location and dispersion parameters (Jain et al., 2008). Instead, use of maximum likelihood procedure with 5 imputations as proposed by Lubin et al. (2004) was recommended by Jain et al. (2008) and Jain and Wang (2008) when the sample size was at least 100 and percent observations below LOD were < 70%. Consequently, a decision was made to use maximum likelihood procedure as proposed by Lubin et al. (2004) with