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Associations between serum per- and polyfluoroalkyl substances and asthma morbidity in the National Health and Nutrition Examination Survey (2003–18)

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Abstract

Background: Per- and polyfluoroalkyl substances (PFAS) are a class of chemicals widely used in manufacturing and are highly resistant to degradation, so they accumulate in the environment. Serum concentrations of these so-called forever chemicals have been associated with impairment of innate and adaptive immune responses. The relationship between serum PFAS levels and asthma morbidity has not been studied.

Objective: We tested the association between serum PFAS concentration and asthma exacerbations.

Methods: We performed secondary analysis of data from the National Health and Nutrition Examination Survey (NHANES, 2003–18). We fit multivariable logistic regression models to estimate odds ratios and 95% CIs for asthma exacerbation in the prior 12 months, given serum concentrations of PFAS. Models were adjusted for relevant covariates.

Results: Of 1101 participants with self-reported current asthma and available serum PFAS data, we observed that higher serum perfluorooctanoic and perfluorodecanoic acids were associated with greater odds of asthma attacks in the previous 12 months (respectively, adjusted odds ratio 1.16, 95% CI 1.01, 1.33; and adjusted odds ratio 1.21, 95% CI 1.03, 1.43). After stratification by age, the association between perfluorooctanoic acid and asthma attacks was significant in the 12–18-year-old group only (adjusted odds ratio 1.56, 95% CI 1.06, 2.31). No significant relationships were observed between PFAS and asthma-related emergency department visits. After correction for multiple comparison testing, none of the associations reached the threshold of significance.

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Conclusion: The role of these bioaccumulative forever chemicals in susceptibility to asthma attacks warrants further examination in longitudinal studies. (J Allergy Clin Immunol Global 2023;2:100078.)

Keywords

PFAS; infection; asthma; viral

INTRODUCTION

Asthma is influenced by a complex interaction of genetic and environmental factors. Viral respiratory infections are the most common trigger for acute exacerbations of asthma, and modulation of the immune response to infection has been associated with increased frequency and severity of exacerbations.[1] There is increasing interest in the health risks of per- and polyfluoroalkyl substances (PFAS), a class of >4000 synthetic compounds used in manufacturing of items like nonstick cookware, water-repellant fabrics, and fire extinguisher foams that contaminate food and drinking water sources. Perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) are known immunotoxins in humans with effects on the innate and adaptive immune responses.[2] The US National Toxicology Program concluded that PFOS and PFOA were associated with reduced antibody responses to vaccines,[3,4] impaired nuclear factor kappa–light-chain enhancer of activated B cells (aka NF-kB) signaling,[5,6] and suppression of natural killer cell activity.[2,7] Despite being phased out of production, such legacy PFAS remain relevant to human health because of their extreme stability in the environment and persistence in the human body. Other PFAS types, including perfluorononanoic acid and perfluorodecanoic acid (PFDA), have been reported to be associated with similar impaired immune responses[3,8] and higher rates of lower respiratory tract infections in children.[9] The relationship between PFAS exposure and asthma prevalence is inconsistent, with some reporting a significant positive association[10–13] and others finding no association.[9,14–16] It is unknown whether a relationship exists between PFAS exposure and worsened asthma severity or control. Given the role of acute respiratory infections in triggering exacerbations of asthma, we hypothesized that PFAS exposure was associated with increased asthma morbidity.

To test our hypothesis, we performed an analysis of data from 8 cycles (2003–18) of the National Health and Nutrition Examination Survey (NHANES). Serum PFAS measurement was obtained from a random one-third subsample of participants 12 years and older during each cycle. Current asthma, asthma attacks, and asthma-related emergency room or urgent care visits in the prior 12 months were assessed by self-report. Those reporting a history of emphysema, chronic bronchitis, or chronic obstructive pulmonary disease were excluded from the analysis. All analyses were conducted by Stata v17.0 (StataCorp, College Station, Tex). We fit multivariable logistic regression models to estimate odds ratios and 95% CIs for asthma exacerbation in the prior 12 months, given serum concentrations of PFAS. Seven PFAS types were collected in all NHANES cycles between 2003 and 2018. Of these, 6 types were found in concentrations at or above the limit of detection in ≥50% of samples and were included in the analysis. Separate models were generated for each PFAS type: PFOS, PFOA, perfluorohexane sulfonic acid, perfluorononanoic acid, 2-(*N*-methyl-PFOSA) acetic acid,

and PFDA. Serum PFAS data were $\log_2$ transformed to approximate a normal distribution. Models were adjusted for age, sex, race, ethnicity, family income-to-poverty ratio, body mass index, and serum cotinine concentration. A correction for multiple comparison testing was added, calculated as 0.05 divided by 6, the number of PFAS types tested. Detailed information is available in this article's Methods section in the Online Repository at www.jaci-global.org.

RESULTS AND DISCUSSION

A total of 1101 individuals were identified as having current asthma and available serum PFAS data, although as a result of sample limitations in the 2013–14 cycle, PFOS and PFOA data were available for only 935 participants. Forty-nine percent of this population reported having an asthma attack in the prior 12 months, and 15% reported an asthma-related emergency department/urgent care visit in the prior 12 months. Demographic characteristics and mean serum PFAS concentrations are presented in Table I. Serum PFOS, PFOA, and perfluorohexane sulfonic acid concentrations were higher in male participants than in female participants. Non-Hispanic White participants and those with higher family income-to-poverty ratio had higher serum levels of PFOS and PFOA compared to other race/ethnicity and income groups. We observed high correlation between PFOA and PFOS ($r = 0.79$) and low to moderate correlations between other PFAS types, with correlation coefficients ranging from 0.11 to 0.66 (Table II).

We observed positive associations between asthma attacks in the past 12 months and serum PFAS concentrations in the adjusted models, with the strongest associations observed with PFOA and PFDA (Table III). After stratification by age, PFDA remained statistically significant among those >18 years of age but not in those ≤18 years. In the overall population and the adolescent group, PFOA was positively associated with odds of asthma attacks. Stratification by sex revealed that the association between PFDA and higher odds of asthma attacks remained significant only for male subjects (Table IV). However, no associations passed the significance threshold when corrected for multiple testing. No significant associations were observed between PFAS concentrations and emergency department/urgent care visits in the adjusted models.

In this analysis of a representative sample of the US population, we observed that serum concentrations of PFDA and PFOA were positively associated with asthma attacks in individuals 12 years and older after adjustment for covariates. Among adolescents, but not adults, every doubling of serum PFOA concentration was associated with a 1.6-fold increase in the odds of having had an asthma attack in the previous 12 months. Among adults, but not adolescents, every doubling of serum PFDA concentration was associated with a 1.2-fold increase in the odds of asthma attacks. However, no associations remained statistically significant after correction for multiple testing.

PFOA and PFDA have been associated with immune suppression in previous studies,[2,3,8,9] and PFOA is also linked to increased frequency of lower respiratory tract infections,[9,17] the most common trigger for acute asthma exacerbations. These findings support the biological plausibility of PFAS as a potential environmental risk factor for asthma exacerbation.

Susceptibility to exposure-related health effects may be dependent on PFAS type as well as age, biological sex, and other individual-level factors. Previous studies have observed that children are particularly vulnerable to the health effects of PFAS,[18–20] which is in line with our findings showing a significant association between PFOA and asthma attacks among adolescents but not adults. Biological sex may also mediate the relationship between some PFAS and asthma morbidity. Consistent with our observations, multiple studies have reported higher serum PFAS concentrations in male compared to female subjects, suggesting potential sex-based differences in exposure or clearance of PFAS from the body.[21,22]

Our analysis is limited by the cross-sectional nature of the data and by potential recall bias inherent to survey data. As a result of limitations in the data collected in the survey, we were unable to control for some relevant covariates, including asthma severity, allergic sensitization, and air pollution exposure. Despite the large size of the NHANES data set, a relatively small number of participants had both current asthma (9%) and serum PFAS measurements (random one-third subsample). Further, the prevalence of asthma-related emergency department/urgent care visits in the prior 12 months was only 15%; the low number of events may have limited our ability to detect an association with serum PFAS levels. Different results may have been observed in a larger or more severe asthma population. It is possible that PFAS could be a marker for other unmeasured risk factors for poorly controlled asthma, though our analysis adjusted for most of the well-described potentially confounding variables. Our analysis was limited to only the PFAS types measured in the NHANES study; it is unknown whether an association exists between asthma morbidity and unmeasured PFAS types, including newer compounds like the ammonium salt of hexafluoropropylene oxide dimer acid, or GenX, which was created to replace legacy PFAS types. Finally, our study outcome was based on self-reported asthma attacks and emergency department visits, but we cannot definitively conclude that these events were triggered by infection. However, given that most asthma exacerbations are mediated by respiratory infection, we interpret our findings as supporting a potential relationship between PFAS-induced immune suppression and increased likelihood of asthma exacerbation.

In conclusion, we found that higher serum concentrations of PFOA and PFDA were associated with higher odds of asthma attacks in the previous 12 months in a nationally representative sample. Further mechanistic studies and prospective cohort studies are needed to determine whether and how PFAS exposure contributes to increased susceptibility to asthma exacerbations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations used

NHANES	National Health and Nutrition Examination Survey
PFAS	Per- and polyfluoroalkyl substances
PFDA	Perfluorodecanoic acid
PFOA	Perfluorooctanoic acid
PFOS	Perfluorooctane sulfonate

REFERENCES

1. Papadopoulos NG, Christodoulou I, Rohde G, Agache I, Almqvist C, Bruno A, et al. Viruses and bacteria in acute asthma exacerbations—a GA^{2}LEN-DARE systematic review. Allergy 2011;66:458–68. [PubMed: 21087215]
2. US Department of Health and Human Services; National Toxicology Program (NTP). Immunotoxicity associated with exposure to perfluorooctanoic acid (PFOA) or perfluorooctane sulfonate (PFOS). September 1, 2016. Available at: https://ntp.niehs.nih.gov/go/749926.
3. Grandjean P, Andersen EW, Budtz-Jorgensen E, Nielsen F, Molbak K, Weihe P, et al. Serum vaccine antibody concentrations in children exposed to perfluorinated compounds. JAMA 2012;307:391–7. [PubMed: 22274686]
4. Grandjean P, Heilmann C, Weihe P, Nielsen F, Mogensen UB, Budtz-Jorgensen E. Serum vaccine antibody concentrations in adolescents exposed to perfluorinated compounds. Environ Health Perspect 2017;125:077018. [PubMed: 28749778]
5. Corsini E, Avogadro A, Galbiati V, dell'Agli M, Marinovich M, Galli CL, et al. *In vitro* evaluation of the immunotoxic potential of perfluorinated compounds (PFCs). Toxicol Appl Pharmacol 2011;250:108–16. [PubMed: 21075133]
6. Corsini E, Sangiovanni E, Avogadro A, Galbiati V, Viviani B, Marinovich M, et al. *In vitro* characterization of the immunotoxic potential of several perfluorinated compounds (PFCs). Toxicol Appl Pharmacol 2012;258:248–55. [PubMed: 22119708]
7. Brieger A, Bienefeld N, Hasan R, Goerlich R, Haase H. Impact of perfluorooctanesulfonate and perfluorooctanoic acid on human peripheral leukocytes. Toxicol In Vitro 2011;25:960–8. [PubMed: 21397682]
8. Timmermann CAG, Jensen KJ, Nielsen F, Budtz-Jorgensen E, van der Klis F, Benn CS, et al. Serum perfluoroalkyl substances, vaccine responses, and morbidity in a cohort of Guinea-Bissau children. Environ Health Perspect 2020;128:87002. [PubMed: 32772733]
9. Impinen A, Longnecker MP, Nygaard UC, London SJ, Ferguson KK, Haug LS, et al. Maternal levels of perfluoroalkyl substances (PFASs) during pregnancy and childhood allergy and asthma related outcomes and infections in the Norwegian Mother and Child (MoBa) cohort. Environ Int 2019;124:462–72. [PubMed: 30684804]
10. Humblet O, Diaz-Ramirez LG, Balmes JR, Pinney SM, Hiatt RA. Perfluoroalkyl chemicals and asthma among children 12–19 years of age: NHANES (1999– 2008). Environ Health Perspect 2014;122:1129–33. [PubMed: 24905661]
11. Kvalem HE, Nygaard UC, Lodrup Carlsen KC, Carlsen KH, Haug LS, Granum B. Perfluoroalkyl substances, airways infections, allergy and asthma related health outcomes—implications of gender, exposure period and study design. Environ Int 2020;134:105259. [PubMed: 31733527]
12. Dong GH, Tung KY, Tsai CH, Liu MM, Wang D, Liu W, et al. Serum polyfluoroalkyl concentrations, asthma outcomes, and immunological markers in a case– control study of Taiwanese children. Environ Health Perspect 2013;121:507–13. [PubMed: 23309686]
13. Averina M, Brox J, Huber S, Furberg AS, Sorensen M. Serum perfluoroalkyl substances (PFAS) and risk of asthma and various allergies in adolescents. The Tromso study Fit Futures in Northern Norway. Environ Res 2019;169:114–21. [PubMed: 30447498]

14. Beck IH, Timmermann CAG, Nielsen F, Schoeters G, Johnk C, Kyhl HB, et al. Association between prenatal exposure to perfluoroalkyl substances and asthma in 5-year-old children in the Odense Child Cohort. Environ Health 2019;18:97. [PubMed: 31730470]
15. Jackson-Browne MS, Eliot M, Patti M, Spanier AJ, Braun JM. PFAS (per- and polyfluoroalkyl substances) and asthma in young children: NHANES 2013–2014. Int J Hyg Environ Health 2020;229:113565. [PubMed: 32485600]
16. von Holst H, Nayak P, Dembek Z, Buehler S, Echeverria D, Fallacara D, et al. Perfluoroalkyl substances exposure and immunity, allergic response, infection, and asthma in children: review of epidemiologic studies. Heliyon 2021;7:e08160. [PubMed: 34712855]
17. Dalsager L, Christensen N, Halekoh U, Timmermann CAG, Nielsen F, Kyhl HB, et al. Exposure to perfluoroalkyl substances during fetal life and hospitalization for infectious disease in childhood: a study among 1,503 children from the Odense Child Cohort. Environ Int 2021;149:106395. [PubMed: 33508532]
18. Bulka CM, Avula V, Fry RC. Associations of exposure to perfluoroalkyl substances individually and in mixtures with persistent infections: recent findings from NHANES 1999–2016. Environ Pollut 2021;275:116619. [PubMed: 33578314]
19. Granum B, Haug LS, Namork E, Stolevik SB, Thomsen C, Aaberge IS, et al. Prenatal exposure to perfluoroalkyl substances may be associated with altered vaccine antibody levels and immune-related health outcomes in early childhood. J Immunotoxicol 2013;10:373–9. [PubMed: 23350954]
20. Impinen A, Nygaard UC, Lodrup Carlsen KC, Mowinckel P, Carlsen KH, Haug LS, et al. Prenatal exposure to perfluoralkyl substances (PFASs) associated with respiratory tract infections but not allergy- and asthma-related health outcomes in childhood. Environ Res 2018;160:518–23. [PubMed: 29106950]
21. Graber JM, Alexander C, Laumbach RJ, Black K, Strickland PO, Georgopoulos PG, et al. Per and polyfluoroalkyl substances (PFAS) blood levels after contamination of a community water supply and comparison with 2013–2014 NHANES. J Expo Sci Environ Epidemiol 2019;29:172–82. [PubMed: 30482936]
22. Kato K, Wong LY, Jia LT, Kuklenyik Z, Calafat AM. Trends in exposure to polyfluoroalkyl chemicals in the US population: 1999–2008. Environ Sci Technol 2011; 45:8037–45. [PubMed: 21469664]

Clinical implications:

Self-reported asthma attacks were associated with serum concentrations of the persistent environmental chemicals called PFAS in a US representative sample. The role of these bioaccumulative forever chemicals in susceptibility to asthma attacks warrants further study.

TABLE I.

Serum PFAS concentrations and demographic characteristics

	Legacy PFAS (ng/mL)			Other PFAS types (ng/mL)				
Characteristic	**No. (%)**	**PFOS**	**PFOA**	**No. (%)**	**PHs**	**PFNA**	**Me-PFOSA-AcOH**	**PFDA**
Overall	935	8.0 (11.5)	2.5 (2.7)	1101	1.3 (1.8)	0.8 (0.7)	0.2 (0.2)	0.2 (0.1)
Sex								
Male	397	10.7 (13.9)	3.1 (3)	453	1.8 (1.9)	0.9 (0.8)	0.3 (0.3)	0.2 (0.2)
Female	538	6.5 (9.5)	2.2 (2)	648	1.0 (1.3)	0.7 (0.6)	0.2 (0.2)	0.2 (0.2)
Race/ethnicity								
Non-Hispanic White	378	9.2 (11.4)	2.9 (2.7)	446	1.4 (1.9)	0.8 (0.7)	0.3 (0.3)	0.2 (0.2)
Non-Hispanic Black	271	8.5 (11.8)	2.3 (2.4)	321	1.3 (1.7)	0.8 (0.8)	0.2 (0.2)	0.2 (0.2)
Mexican American	130	6.6 (12.9)	2.3 (2.6)	146	1.2 (1.7)	0.7 (0.6)	0.2 (0.2)	0.2 (0.1)
Other Hispanic	83	7 (9.7)	2.7 (2.8)	98	1.2 (1.5)	0.9 (0.7)	0.1 (0.2)	0.2 (0.2)
Other race, or multiracial	73	4.4 (8)	1.8 (2)	90	0.9 (1.4)	0.7 (0.6)	0.1 (0.2)	0.2 (0.2)
Age								
≤18 years	302	8.1 (11.5)	2.5 (2.9)	338	1.4 (2.0)	0.8 (0.7)	0.2 (0.2)	0.2 (0.2)
>18 years	633	8.0 (11.6)	2.5 (2.6)	763	1.2 (1.6)	0.8 (0.7)	0.2 (0.2)	0.2 (0.2)
Body mass index								
<30 kg/m^2	540	8.8 (14.1)	2.7 (2.8)	633	1.4 (1.8)	0.8 (0.7)	0.2 (0.2)	0.2 (0.2)
≥ 30 kg/m^2	395	7.1 (10.2)	2.2 (2.1)	468	1.2 (1.5)	0.7 (0.7)	0.2 (0.2)	0.2 (0.2)
Family income-to-poverty ratio								
<1	238	6.8 (9.9)	2.1 (1.9)	281	1.2 (1.4)	0.7 (0.7)	0.2 (0.2)	0.2 (0.2)
≥1 to <3	353	7.5 (12.2)	2.4 (2.7)	405	1.2 (1.6)	0.7 (0.8)	0.2 (0.2)	0.2 (0.2)
≥3	344	9.6 (11.7)	2.9 (2.8)	415	1.4 (1.9)	0.9 (0.7)	0.2 (0.2)	0.2 (0.2)
Serum cotinine (ng/mL)								
Below LOD	219	7.7 (10.8)	2.4 (2.4)	276	1.3 (1.9)	0.7 (0.8)	0.1 (0.2)	0.2 (0.2)
Above LOD	716	8.3 (11.9)	2.6 (2.7)	825	1.3 (1.7)	0.8 (0.8)	0.2 (0.2)	0.2 (0.2)

Data are presented as medians (interquartile ranges).

LOD, Level of detection; *Me-PFOSA-AcOH*, 2-(*N*-methyl-PFOSA) acetic acid; *PFHxS*, perfluorohexane sulfonic acid; *PFNA*, perfluorononanoic acid.

TABLE II.

Weighted Pearson correlation coefficients for PFAS types (N = 1101)

	PFOS*	PFOA*	PFHxS	PFNA	PFDA	Me-PFOSA-AcOH
PFOS*	1.0					
PFOA*	.79	1.0				
PFHxS	.31	.31	1.0			
PFNA	.35	.49	.21	1.0		
PFDA	.31	.38	.11	.66	1.0	
Me-PFOSA-AcOH	.29	.32	.17	.24	.20	1.0

Me-PFOSA-AcOH, 2-(*N*-methyl-PFOSA) acetic acid; *PFHxS*, perfluorohexane sulfonic acid; *PFNA*, perfluorononanoic acid.

*Sample size 935.

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TABLE III.

Adjusted odds ratios (aORs) with 95% CIs for asthma attacks and emergency care visits for asthma in the past 12 months by serum $\log_2$ PFAS concentrations (ng/mL), overall and stratified by age group

	Overall			Age ≤18 years			Age >18 years		
Type of PFAS	**No.**	**aOR (95% CI), overall**	***P***	**No.**	**aOR (95% CI)**	***P***	**No.**	**aOR (95% CI)**	***P***
Asthma attack in past 12 months									
PFOS	859	1.08 (0.95, 1.22)	.22	279	1.21 (0.91, 1.59)	.18	580	1.07 (0.93, 1.23)	.33
PFOA	859	1.16 (1.01, 1.33)	.04	279	1.56 (1.06, 2.31)	.03	580	1.12 (0.95, 1.32)	.16
PFHxS	1010	1.05 (0.93, 1.17)	.43	312	1.11 (0.90, 1.37)	.35	698	1.05 (0.91, 1.21)	.52
PFNA	1010	1.11 (0.96, 1.28)	.17	312	1.05 (0.79, 1.41)	.73	698	1.13 (0.95, 1.34)	.17
Me-PFOSA-AcOH	1010	1.10 (0.99, 1.23)	.08	312	1.24 (0.95, 1.61)	.11	698	1.09 (0.96, 1.23)	.17
PFDA	1010	1.21 (1.03, 1.43)	.02	312	1.24 (0.86, 1.79)	.24	698	1.23 (1.02, 1.48)	.03
Emergency care visit for asthma in past 12 months									
PFOS	859	0.99 (0.79, 1.23)	.90	262	0.91 (0.61, 1.34)	.61	580	1.03 (0.79, 1.33)	.85
PFOA	859	1.03 (0.77, 1.38)	.85	262	0.76 (0.46, 1.26)	.29	580	1.10 (0.78, 1.55)	.58
PFHxS	1010	1.02 (0.86, 1.20)	.85	292	0.97 (0.71, 1.33)	.85	698	1.06 (0.85, 1.33)	.59
PFNA	1010	1.00 (0.78, 1.28)	.99	292	0.65 (0.41, 1.04)	.07	698	1.06 (0.78, 1.44)	.70
Me-PFOSA-AcOH	1010	0.96 (0.78, 1.18)	.70	292	1.01 (0.67, 1.52)	.96	698	0.96 (0.75, 1.23)	.76
PFDA	1010	1.09 (0.86, 1.39)	.48	292	0.91 (0.58, 1.47)	.70	698	1.10 (0.82, 1.48)	.51

Adjusted for age, sex, race, ethnicity, body mass index, income-to-poverty ratio, and serum cotinine.

Me-PFOSA-AcOH, 2-(*N*-methyl-PFOSA) acetic acid; *PFHxS*, perfluorohexane sulfonic acid; *PFNA*, perfluorononanoic acid.

TABLE IV.

Adjusted odds ratios (ORs) with 95% CIs for asthma attacks and emergency care visits for asthma in the past 12 months by serum $\log_2$ PFAS concentrations (ng/mL), overall and stratified by sex

	Overall			Male			Female		
Type of PFAS	**No.**	**Adjusted OR (95% CI)**	***P***	**No.**	**Adjusted OR (95% CI)**	***P***	**No.**	**Adjusted OR (95% CI)**	***P***
Asthma attack in past 12 months									
PFOS	859	1.08 (0.95, 1.22)	.22	371	1.18 (0.95, 1.48)	.13	488	1.01 (0.85, 1.19)	.93
PFOA	859	1.16 (1.01, 1.33)	.04	371	1.23 (0.94, 1.61)	.13	488	1.10 (0.90, 1.34)	.35
PFHxS	1010	1.05 (0.93, 1.17)	.43	421	1.00 (0.80, 1.25)	.99	589	1.06 (0.92, 1.23)	.41
PFNA	1010	1.11 (0.96, 1.28)	.17	421	1.17 (0.91, 1.52)	.22	589	1.07 (0.87, 1.32)	.51
Me-PFOSA-AcOH	1010	1.10 (0.99, 1.23)	.08	421	1.21 (0.99, 1.49)	.07	589	1.03 (0.90, 1.18)	.68
PFDA	1010	1.21 (1.03, 1.43)	.02	421	1.36 (1.05, 1.75)	.02	589	1.14 (0.92, 1.43)	.23
Emergency care visit for asthma in past 12 months									
PFOS	859	0.99 (0.79, 1.23)	.90	371	1.14 (0.70, 1.86)	.59	488	0.91 (0.67, 1.24)	.54
PFOA	859	1.03 (0.77, 1.38)	.85	371	1.45 (0.64, 3.30)	.37	488	0.88 (0.62, 1.25)	.47
PFHxS	1010	1.02 (0.86, 1.20)	.85	421	1.15 (0.69, 1.93)	.59	589	0.97 (0.81, 1.15)	.70
PFNA	1010	1.00 (0.78, 1.28)	.99	421	1.12 (0.64, 1.97)	.69	589	0.98 (0.74, 1.28)	.86
Me-PFOSA-AcOH	1010	0.96 (0.78, 1.18)	.70	421	1.37 (0.90, 2.10)	.14	589	0.82 (0.63, 1.06)	.13
PFDA	1010	1.09 (0.86, 1.39)	.48	421	1.39 (0.86, 2.25)	.18	589	0.99 (0.74, 1.34)	.97

Adjusted for age, sex, race, ethnicity, body mass index, income-to-poverty ratio, and serum cotinine.

Me-PFOSA-AcOH, 2-(*N*-methyl-PFOSA) acetic acid; *PFHxS*, perfluorohexane sulfonic acid; *PFNA*, perfluorononanoic acid.