HHS Public Access

Author manuscript

J Expo Sci Environ Epidemiol. Author manuscript; available in PMC 2019 May 27.

Published in final edited form as:

J Expo Sci Environ Epidemiol. 2019 March ; 29(2): 172–182. doi:10.1038/s41370-018-0096-z.

Per- and polyfluoroalkyl substances (PFAS) blood levels after contamination of a community water supply and comparison with 2013-14 NHANES

Judith M. Graber, PhD[1,2], **Cora Alexander, MPH**[1,3], **Robert J. Laumbach, MD**[3,4], **Kathleen Black, PhD**[4], **Pamela Ohman Strickland, PhD**[1], **Panos G. Georgopoulos, PhD**[2,3], **Elizabeth G. Marshall, PhD**[1], **Derek G Shendell, D. Env**[3], **Donald Alderson, MS**[5], **Zhongyuan Mi, MPH**[2], **Michael Mascari, MPH**[1], and **Clifford P. Weisel, PhD**[2,3]

[1]Rutgers School of Public, Department of Biostatistics and Epidemiology

[2]Rutgers Environmental and Occupational Health Science Institute – Division of Exposure Science and Epidemiology

[3]Rutgers School of Public Health, Department of Environmental and Occupational Health

[4]Rutgers Environmental and Occupational Health Science Institute – Clinical Research and Occupational Medicine

[5]Rutgers University Biostatistics and Epidemiology Services Center (RUBIES), Rutgers Biological and Health Sciences

Abstract

Introduction: Per- and polyfluoroalkyl substances (PFAS), including perfluorononanoic acid (PFNA) and perfluorooctanoic acid (PFOA), were detected in the community water supply of Paulsboro New Jersey in 2009.

Methods: A cross-sectional study enrolled 192 claimants from a class-action lawsuit, not affiliated with this study, who had been awarded a blood test for 13 PFAS. Study participants provided their blood test results and completed a survey about demographics; 105 participants also completed a health survey. Geometric means, 25th, 50th, 75th and 95th percentiles of exposure of PFNA blood serum concentrations were compared to that of the 2013–2014 NHANES, adjusted for reporting level. Associations between PFNA, PFOA, PFOS, and PFHxS and self-reported health outcomes were assessed using logistic regression.

Results: PFNA serum levels were 285% higher in Paulsboro compared with U.S. residents. PFNA serum levels were higher among older compared with younger, and male compared to female, Paulsboro residents. After adjustment for potential confounding, there was a significant

Corresponding author Judith M. Graber, PhD, Associate Professor, Department of Epidemiology, Rutgers School of Public Health, Environmental and Occupational Health Sciences Institute, 170 Frelinghuysen Road, Rm. 320, Piscataway, NJ 08854, Office: 848-445-0190, Cell: 207-441-3862, Fax: 732-445-0116, graber@eohsi.rutgers.edu.

Declaration of competing financial interests

The authors declare they have no actual or potential competing financial interests.

association between increased serum PFNA levels and self-reported high cholesterol (OR: 1.15, 95% CI: 1.02, 1.29).

Discussion/Conclusion: Further investigation into possible health effects of PFAS exposure in Paulsboro and other community settings is warranted. Since exposure has ceased, toxicokinetics of PFAS elimination should be explored.

Introduction

Per- and polyfluoroalkyl substances (PFAS) are anthropogenic persistent organic pollutants (POPs) of emerging health and environmental concern because of their resistance to degradation, tendency for global transport, and ability to bioaccumulate. PFAS are highly heat resistant, hydrophobic, and extremely durable. They are used in the manufacturing and production of many consumer products. Unlike many other POPs, PFAS are soluble in water, facilitating community-level exposures from contaminated community water supplies and private wells. (1, 2) Their presence in the environment is a result of consumer product use, industrial use, emissions, and disposal. They are found in drinking water, the food chain, household dust, and air. (3, 4) PFAS contamination of community water supplies has been associated with use of fire-fighting foams at military installations and civilian airports, major industrial sites and, to a lesser extent, wastewater treatment plants. (2, 5)

PFAS are detected in human populations globally. (6) There is a growing consensus that PFAS pose a threat to human and environmental health. (7) Greater than 99% of NHANES participants age 12 and older have detectable levels of one or more PFAS in serum. (8) The most commonly detected compounds are perfluorooctanoic acid (PFOA), perfluorooctane sulfonic acid (PFOS), perfluorohexane sulfonic acid (PFHxS) and perfluorononanoic acid (PFNA). In the US, beginning in the early 2000's the major manufacturers began to voluntarily phase out PFOA and PFOS in facility emissions and product content. The US Environmental Protection Agency (EPA) has established health advisory levels for PFOA and PFOS in drinking water at 70 parts per trillion, with the aim of protecting sensitive populations. (9) Use of PFOS was banned by the European Union in 2008 and PFOA will be phased out by 2020. (10) Serum levels of both PFOA and PFOS have declined in the general population, however serum levels of other PFAS which have not been phased out, including PFNA, have increased. (8)

The toxicities of PFOA and PFOS have been studied more extensively than that of other PFAS. In 2016, the Persistent Organic Pollutants Review Committee to the Stockholm Convention on Persistent Organic Pollutants reached a consensus agreement that exposure to PFOA is likely to lead to significant adverse human health and environmental effects. (1) The International Agency for Research on Cancer Research classifies PFOA as a possible human carcinogen. (11)

PFAS, including PFNA and PFOA, were detected in the community water supply of Paulsboro, New Jersey, in 2009. During 2011 and 2012, the New Jersey Department of Environmental Protection (NJ DEP) monitored PFAS in raw and finished drinking water in some New Jersey communities. Paulsboro had the highest groundwater concentration of PFNA in the state at 96 ng/L (12), and PFNA was detected in finished water with levels as

high as 72 ng/L. PFOA was also present at 26 ng/L. Further monitoring done in 2013 and 2014 found elevated PFNA levels in 45 samples from six distribution systems. These had mean and maximum values of 36 ±34 ng/L and 93 ng/L. Samples from both the source water for the Paulsboro water system and from well water in nearby communities had mean levels of 44 ±49ng/L and a maximum value of 150 ng/L (data available from NJ DEP upon request). These values provide only a snapshot of the potential exposure to the residents prior to remediation of the community water supply with granular activated carbon treatment, which began in April 2014. In 2015, the New Jersey Drinking Water Quality Institute recommended Maximum Contaminant Level (MCL) drinking water levels of 14, 13, and 13ng/L for PFOA, PFOS, and PFNA, respectively (13) with the PFNA recommendation adopted by the New Jersey Department of Protection as a drinking water standard (MCL-Maximum contaminant level) in September 2018.. (13)

A growing number of epidemiologic studies have reported associations between increased PFNA serum levels and adverse health effects across the life spectrum, including disrupted lipid homeostasis, reduced immune response, adverse birth outcomes, and attention deficits. The C8 Study, which enrolled people in communities near a chemical plant in the Mid-Ohio Valley (USA) with PFAS contamination of the drinking water, included an exposure-response relationship between higher serum PFNA and lower levels of sex hormones and insulin-like growth factor 1 (IGF-1) in boys and girls. Like sex hormones, IGF-1 also plays an important role in growth and sexual maturation. (14) Exposure to PFAS has been associated with increased levels of cholesterol and the liver enzymes in children and adults. (4, 15) For example, a population-based, longitudinal Swedish study reported that increased serum PFNA was associated with changes in liver biomarkers markers including lowered mean bilirubin (−1.56 μmol/l, 95% CI −1.93 to −1.19). (16) In adult women, but not men, serum PFNA has also been associated with increased levels of intima-media complex (IM-GSM), a marker of arterial lipid infiltration. (17) Associations between PFNA exposure and a decrease in the early humoral immune response to vaccination boosters have been reported. (18) Cord blood PFNA concentrations have been associated with learning disorders, inattention, impulsivity/hyperactivity, and oppositional defiant disorder, but not with neurobehavioral symptoms of attention deficit disorder. (4)

Paulsboro New Jersey is an industrial town. In 2014 the population was 6,097 and the median household income was under $41,061, well below that of the US and New Jersey (in 2014 $53,657 and $76,126, respectively).(19) Fifty-six percent of the population were female and 45% classified themselves into a race-ethnic group other than non-Hispanic white. (12) A manufacturing facility near Paulsboro that used PFNA in their industrial processes from the 1970’s until 2010 was identified as the only likely source of the water contamination. In response to the PFAS water contamination in Paulsboro, a class-action lawsuit awarded qualified Paulsboro residents one test for serum levels of 13 PFAS, including PFNA. This study used the results of the lawsuit blood tests to estimate current PFAS serum levels in the lawsuit participants, compare those results to those of US residents, as measured by the National Health and Nutrition Examination Survey (NHANES), and assess associations between PFAS serum concentrations and self-reported health outcomes.

Materials/Subjects and Methods

Study Population – Paulsboro, New Jersey

We conducted a cross-sectional study of current or former Paulsboro residents who had received PFAS blood-test results as claimants in the 2016 class-action lawsuit. The lawsuit eligibility included residing in Paulsboro New Jersey for at least 12 months prior to April 19, 2014, when remediation of the community water supply was initiated.

Six study enrollment sessions were held in the Paulsboro area between November 14, 2016 and January 21, 2017 during weekdays, weekday evenings, and on weekends. Community outreach conducted before and during these sessions included: mailing a recruitment flyer to all residential addressees in Paulsboro (obtained through a commercial vendor); sending the flyer and conducting in-person outreach to local faith-based and community organizations and businesses (e.g. barber shops, nail salons, and groceries); emailing the flyer to clients of two attorneys involved with the lawsuit; and posting information about the study on local social media sites.

At each enrollment session, study personnel obtained informed consent from eligible adults who had in-hand a copy of their and/or their children/legal guardians' PFAS blood results obtained from the lawsuit. Enrolled adults also completed a brief survey about the participant's age, sex, race and number of years living in Paulsboro. After enrollment, blood test results and completed surveys were scanned into secure laptops. Two study technicians entered the laboratory and survey data independently. The dual entries were compared electronically. Discrepancies were resolved using the source data.

The Health Outcomes Survey

Participants were invited to complete an additional online survey that typically took 12 to 15 minutes to complete and included questions about their current and previous health conditions. Participants were surveyed using the following question stem: "Have you ever been diagnosed by a doctor with any of the following health conditions?" For any 'yes' responses, year of first diagnosis was asked. This question was asked for each of the following 32 outcomes (in eight categories), selected based on a review of PFAS epidemiological literature:

- Cardiovascular: high blood pressure, high cholesterol, coronary artery disease, stroke, other cardiovascular;
- Autoimmune disease: Lupus, type I diabetes, inflammatory bowel disease, ulcerative colitis, Crohn's disease, multiple sclerosis, rheumatoid arthritis, other autoimmune disease;
- Liver conditions: hepatitis, enlarged liver, fatty liver disease, cirrhosis, other liver disease;
- Neurological conditions: Alzheimer's disease, Parkinson's disease, Lou Gehrig's disease (AML), other neurological diseases;
- Thyroid conditions: hypothyroidism, hyperthyroidism, other thyroid disease;

- Renal disease: chronic kidney disease, end-stage renal disease, other kidney disease;
- Cancer: Any diagnosis, for positive responses, type was asked;
- Reproductive issues (among women only): pregnancy induced hypertension/pre-eclampsia, other pregnancy problems.

After frequencies of responses were assessed, ‘cardiovascular’ was further categorized into ‘cardiovascular conditions except high cholesterol’ and ‘high cholesterol’.

Paulsboro PFAS Blood Level Assessment

PFAS serum testing was conducted for the class action settlement claimants, independent of this study. To the best of our knowledge, in-home phlebotomy was contracted to a local laboratory and the blood samples were labeled with a class member ID (assigned by the settlement class administrator). Serum was then sent to SGS Axys Analytics in British Columbia, Canada (henceforth ‘Axys Laboratories’). Axys Laboratories is an environmental laboratory and is therefore not CLIA (Clinical Laboratory Improvement Amendments) certified. To our knowledge, the analytical methods used by Axys Laboratories are comparable to those used by NHANES for analyzing environmental blood data. In brief, quantitative detection of PFAS was conducted using online solid phase extraction coupled to high performance liquid chromatography-turboion spray ionization-tandem mass spectrometry (online SPE-HPLC-TIS-MS/MS). (20) After testing for 13 PFAS, results were returned to the settlement class administrator, identified, and mailed to the participant. The reports did not include interpretation of the PFAS serum levels. The study team had no role in the blood collection, testing or dissemination of results. As outreach to the community for the study began, concerned claimants made the study team aware of the lack of interpretation of the blood results. Team investigators therefore developed an informational flyer to address this significant gap. The flyer explained technical aspects of the results and the difference between a clinical blood test and the PFAS serum levels. The flyer was distributed by the claimants’ legal representatives, on the Rutgers University website, and at our community report-back meeting after the study was completed.

Study population: the National Health and Nutrition Examination Survey (NHANES)

The NHANES is an ongoing cross-sectional survey administered by the U.S. Centers for Disease Control and Prevention (CDC), Center for Health Statistics in two-year cycles. Participants for NHANES are selected to be representative of the US non-institutionalized population using a multistage cluster sample design. (21) Serum is tested for PFAS among one-third of NHANES participants age 12 and older. NHANES documentation includes detailed descriptions of the laboratory methods. For NHANES, PFOA and PFOS were analyzed as the sum of the respective linear and branched polymers.

Statistical analysis

To assess whether the results of Paulsboro PFAS serum levels in the current study would be generalizable to all Paulsboro residents, the distribution of participants’ age, sex, and race was compared to that of the 2014 U.S. census for Paulsboro using Chi-squared tests. (22)

The median age of the 186 study participants was 47.0 years old, significantly older than residents of Paulsboro reported by the in 2014 US census (35.3; $p < 0.001$). The distribution of the sample by sex and race was similar. Because of the difference in the age distribution between the Paulsboro sample and the intercensal estimates, post-stratification weights were developed using standard methods. These weights were applied to the analyses comparing Paulsboro PFAS data to the 2014 intercensal estimates, in order for the results to be generalizable to the town population. (23)

To compare the serum concentrations to the US population, demographic characteristics of the Paulsboro sample including age, sex, and race-ethnicity were compared with those from the NHANES 2013–2014 cycle. Since NHANES restricts PFAS testing to participants age 12 and older (8), Paulsboro data for the age category (0 to 11) were excluded from these analyses. Demographic characteristics were categorized to be consistent with published NHANES results (age: 12 to 19, 20 to 39, 40 to 59, and ≥60 years; race/ethnicity: Hispanic, non-Hispanic black, non-Hispanic white). Years that study participants lived in the town of Paulsboro was categorized as less than 10; 10 to 19; 20 to 34 and 35 or more.

Inconsistencies between PFAS acronyms used by NHANES and Axys Laboratories were resolved by email communication with an Axys Laboratories senior scientist. NHANES acronyms are used henceforth in this report (see Supplemental Table 1). For NHANES data analysis, we used the published sample weights designed for the one-third subset of the full survey for all effect and variance estimates. These weights account for unequal selection probabilities of the complex sampling design and planned oversampling of specific population subgroups.

Prevalence and 95% confidence intervals (95% CIs) for detectable PFAS in the Paulsboro study sample were calculated and compared with those from the NHANES 2013–2014 cycle. NHANES reporting for PFAS includes a limit of detection (LOD) for each PFAS, for example 0.10 μg/L for PFNA. In contrast, rather than an LOD, Axys Laboratories results displayed a reporting limit (RL) which varied per analyte and for some PFAS, per test result. The majority of PFAS analytes had a RL of 0.50 μg/L. Three analytes (PFBS, PFHxS, and PFOS) had a RL of 1.0 μg/L. For some test results, a sample detection limit (SDL) was displayed instead of an RL. SDLs were always higher than the RL and lower than the reported PFAS serum level. For comparability of the samples with NHANES LOD, we considered NHANES results below the Axys Laboratories RL to be below the LOD. As such, we only included those NHANES values above the Axys Laboratories RL in our comparison of NHANES and Paulsboro PFAS serum concentrations.

Four PFAS were detected in most (>70%) Paulsboro serum samples (PFNA, PFOA, PFOS and PFHxS). The correlations among those four PFAS serum levels were evaluated using unweighted Spearman correlation coefficients. The weighted geometric means and 25^{th}, 50^{th}, 75^{th} and 95^{th} percentiles of exposure were calculated for the Paulsboro and NHANES samples. For this comparison, the NHANES serum concentrations are presented based on both the NHANES LOD and the restriction to only the samples detected at the higher RL from the Paulsboro serum test results (described above). For all analyses, PFAS values below the analytic method LOD were calculated as the LOD divided by the square root of

two. For the PFNA serum levels, we further stratified the geometric means and percentiles by age group, and for the Paulsboro sample by years lived in Paulsboro.

Associations between serum levels and demographic characteristics were assessed for all participants (n=186) using generalized linear regression. Demographic characteristics assessed in the models were age (continuous and categorical: 0 to 19 years; 20 to 39 years; 40 to 59 years; 60 and older), gender, and race-ethnicity (as grouped above). Interaction between age and each of the other characteristics was assessed by including cross products of these terms in the model.

Study participants age 19 and older were eligible to participate in the longer health survey. The distribution of demographic characteristics was compared between those who did and did not complete the health survey using Mantel-Haenszel chi square tests (gender, race-ethnicity) and T-tests (age, years lived in Paulsboro). Median serum levels of PFNA, PFOA, PFOS and PFHxS were compared using 2-sided Wilcoxon rank sum tests. Associations between serum PFAS concentrations and the health outcomes was limited to conditions reported by 20 or more respondents in order to have sufficient sample size to conduct multivariable analyses. As such, multivariable analysis was not feasible for most of the health outcomes, including: autoimmune conditions (n=19), cancer (n=14), female reproductive conditions (n=12), liver conditions (n=6), neurological conditions (n=14), thyroid conditions (n=10), and renal disease (n=2). We therefore assessed associations between PFAS serum levels for ‘high cholesterol’ (n=29) and ‘cardiovascular conditions except high cholesterol’ (n=39). For each of these two outcomes, separate models were explored for each of PFAS, as well as in a model that included all four (PFNA, PFOA, PFOS, PFHxS). For these analyses, the untransformed, log transformed continuous, and quadratic forms of each PFAS were compared in saturated models. Log transformation improved the model fit for PFOA and was therefore used for modeling that exposure. In all models, age and BMI contributed significantly to the model fit while gender, race-ethnicity and smoking status at enrollment did not. Effect modification was assessed between each PFAS and gender, age, and race-ethnicity by entering cross-product terms into the full model and considered present if the cross-product term p-value was significant (α=0.05). The contribution of each covariate to the models [age, gender (reference = female), race-ethnicity (reference = non-Hispanic white), BMI (continuous), and smoking status (current, former, never (reference=never))] was assessed in nested models by the model fit using the likelihood ratio test, and by observing whether there was a change in the odds ratio of more than 10%. We conducted all analyses using SAS software, version 9.4; code is available by request to the corresponding author. This study was approved by the Rutgers Institutional Review Board.

Results

Study participants

Of the 6,000 persons eligible for the resident class lawsuit, 1,329 had their blood drawn for PFAS serum levels and 192 enrolled in our study. Six participants were excluded because most demographic data were missing. For one participant, 2 PFAS laboratory reports were received; the updated PFAS test result replaced the initial report (as per Axys Laboratory

instructions). For the study participants included in the analysis, there were no significant differences between the weighted proportion of the Paulsboro and NHANES participants by age, sex or race-ethnicity (Table 1).

Prevalence of detectable serum PFAS levels

All 186 Paulsboro study participants had at least one PFAS detected in their serum. The 165 enrollees over age 12 were included in the analysis comparing Paulsboro PFAS serum levels to those of NHANES participants. Three of the 13 measured PFAS—perfluorobutanesulfonic acid (PFBS), PFHxA, and perfluoro-n-pentanoic acid (PFPeA) —were not detected in any study participants. Four were detected in most participants – with a weighted prevalence of over 70% (PFHxS, PFOS, PFNA, and, PFOA). The other PFAS were detected in 1 to 11 percent of participants. The percent of test results with SDL rather than RL reported were 1.30% for PFOA (range: 0.50, 7.56 μg/L), 1.9% for PFHxS (range: 1.00, 2.02 μg/L), 4.4% for PFOS (range: 1.00, 4.78 μg/L), and 23.3% for PFNA (range: 0.50, 11.00 μg/L). The serum levels of the four PFAS were significantly correlated with each other; Spearman correlation coefficients ranging from 0.58 (PFOS with PFHxS) to 0.78 (PFNA with PFOA).

The prevalence of detectable concentrations of measured PFAS was similar between the Paulsboro and U.S. samples with the exceptions of PFOA, PFOS and PFNA (Table 2). There was a statistically significantly higher detection rate for PFOA, PFOS, and PFNA among the Paulsboro sample compared with the NHANES adjusted sample. Geometric mean levels of PFHxS and PFOS were similar between the Paulsboro and NHANES samples. For the Paulsboro sample, the geometric mean for PFOA was 46% higher than NHANES, while the geometric mean of PFNA was 285% higher (Table 3). In both the Paulsboro and NHANES samples, serum levels of PFOS were significantly higher than the other PFAS. Of note, the 95th percentile of exposure for PFNA in the Paulsboro study participants was over five times that of the U.S. population, a statistically significant difference.

Comparison of Paulsboro and NHANES serum PFNA levels by demographics

The geometric mean of PFNA serum levels was higher among the older age groups for both the Paulsboro and NHANES samples. The ratio between the PFNA levels in the NHANES and Paulsboro samples increased with age and was less than 3 in those children and young adults (age group 12 to 19 years= 2.90, 20 to 39 years = 2.70), while over 4 among older adults (age group 40 to 59 years = 4.57; age ≥ 60 years = 5.80) (Table 4).

PFNA serum levels among the 21 children age 11 years and younger in the Paulsboro study (geometric mean: 2.44 μg/L, 95% CI: 1.69, 3.51) were similar to those of children age 12 to 17. Due to small sample size, 95% CIs for the 95th percentile were not calculated for this age group; the 75th percentile was 3.31 μg/L (95% CI: 2.19, 4.43; Supplemental Table 2).

Geometric mean PFNA serum levels were similar between men and women in the NHANES data, accounting for the higher LOD (0.92 μg/L, 95% CI: 0.86, 1.00 and 0.90 μg/L, 95% CI: 0.86, 0.94). However, among Paulsboro participants the level was significantly higher among men compared with women (4.32 μg/L 95 %CI: 3.51, 5.31, and 2.95 μg/L 95 %CI: 2.48, 3.52). In both samples the point estimates were highest among non-Hispanic white and

lowest among Hispanic participants, but the difference was not statistically significant in the Paulsboro sample, possibly due to the small number of Hispanic participants in the latter (n=7; Table 4). In generalized linear models assessing the associations between PFNA serum levels and demographic characteristics, a significant interaction was seen between age and gender (p=0.0137) such that the association was significant among females only. For every increase year of age among females there was a 1.5% (95% CI: 0.7, 2.3; p=0.001) increase in log unit of PFNA serum concentration, but this association was not significant in males (p=0.3686; Table 5).

Health outcomes

Of the 149 study participants age 19 and older, 70.5% (n=105) completed by the health survey. These adults provided health information on 14 children age 12 to 19, too few to assess health outcomes for that age group. There were no significant differences between the adults who completed the survey and those who did not in terms of mean age (49.9 vs. 52.3 year; p=0.39), or race-ethnicity (p=0.18). A slightly higher proportion of women completed the health survey than men (65.1 vs 59.8%, p=0.10). Median PFAS serum levels did not differ significantly between those who did and did not take the survey for PFNA (p=0.1526), PFOA (p=0.4913), and PFHxS (p=0.6973), but for PFOS the median serum levels were lower among those who took the health survey and this difference was borderline significant (7.25 μg/L sd 4.50, and 8.37 μg/L sd 5.57, respectively p=0.0597). No statistically significant interactions were detected. There were no significant associations of PFAS and cardiovascular conditions other than high cholesterol (Table 6). For all PFAS, there was a positive association between increased serum levels and self-reported physician diagnosed high cholesterol. After adjusting for age and BMI, for every 1 μg/L increase in blood PFNA concentration, the odds of having high cholesterol increased significantly by 16% (OR: 1.16, CI: 1.03, 1.29). A borderline significant association was also seen for the associations between high serum cholesterol and PFOS (OR=1.10, 95% CI: 0.99, 1.22) and PFHxS (OR=1.07, 95% CI: 0.99, 1.12; Table 6). When the four most prevalent the PFAS were included in the model, the association between PFNA and high cholesterol remained positive, but was attenuated and no longer statistically significant.

Discussion

More than two years after the remediation of the Paulsboro New Jersey community water supply, serum concentrations of PFNA and PFOA were significantly higher than that of the US population. Disturbingly, the overall geometric mean for serum PFNA for Paulsboro was significantly greater than that 95th percentile of that seen in the 2013–2014 NHANES. To our knowledge, this is the first epidemiological study conducted in a community with PFNA-contaminated drinking water. When PFAS were discovered in the Paulsboro community water supply in 2009, the measured levels of PFNA far exceeded the 2007 NJ Department of Environmental Protection advisory level of 40 ng/L for PFOA, and the subsequent 2018 drinking water standard for PFNA of 13ng/L. (13)

The geometric mean serum concentration of PFNA among the oldest age group of Paulsboro residents was two and half times that of the youngest, in contrast to lack of association with

age in both the analysis of 2013–2014 NHANES and multi-cycle NHANES. (8) However, a report from a 2008 nationally representative cross-sectional sample of adults in Spain did show a significant increase in geometric mean serum concentrations from the youngest to oldest group, but with a smaller difference than that observed in Paulsboro (<29 years = 0.192 μg/L vs. >50 years old 1.124 μg/L; p <0.01). Geometric mean serum levels of PFNA in that study (overall geometric mean = 0.96 μg/L, 95% CI: 0.89, 1.03) were very similar to this study's NHANES sample results (0.961 μg/L, 95% CI: 0.87,0.96). (1) The higher levels in older age in Paulsboro may reflect elimination differences by age with higher exposure burden over time.

PFNA serum concentrations were significantly higher among males than females in the Paulsboro sample. In a study of four 2-year NHANES cycles from 1999 to 2008, males had significantly higher PFNA geometric mean serum concentrations of PFNA compared with females, adjusted for age and race (p<0.01). (8) Other studies have also reported a higher blood or urine levels of PFNA among males, suggesting the possibility of sex-related differences in exposure, (e.g., water consumption, occupational exposure, or food consumption) or elimination. Possible contributing factors to lower levels of PFNA (and other PFAS) among woman compared to men include breast feeding, parity, and menstruation. While a Norwegian mother and child cohort did not see evidence of associations between menstrual cycle length and PFAS concentration, oral contraception use in the past year was associated with long cycle length and increased serum levels of PFNA and one other PFAS. (24, 25) A report from a Spanish birth cohort reported that plasma concentrations of PFOA and PFNA decreased with parity. (26)

The PFAS blood levels used for this study were from samples collected approximately two and half years after the remediation of the Paulsboro community water supply, but the extent to which serum PFAS concentrations reported here underestimate pre-remediation levels is not known. Estimates of the half-life of PFNA (a 9-carbon chain compound) in humans have not been published, to our knowledge. The half-lives for elimination of 8-carbon chain compounds, including PFOA and PFOS, from human serum have been estimated at between 3 and 8 years. (27–29) The half-life of PFNA is likely longer than that of PFOA and PFOS, because the rate of elimination generally decreases with increasing carbon chain length. As such, the PFNA serum concentrations reported here are expected to be lower than the serum concentration, and therefore the body burden, of the participant s in 2014 when remediation of the town drinking water system began.

Among the study participants who completed the health survey (n=105), a significant association between PFNA and self-reported high cholesterol level was observed after adjusting for potential confounding factors. Over 400 epidemiologic studies have evaluated human PFAS exposure and health effects. Positive associations have been observed between increased serum or urine concentrations of several PFAS and adverse health outcomes including increases in serum lipids, liver damage, thyroid disease, immune suppression, reproductive toxicity, and developmental disorders. Most studies that examined the association between serum or urine concentrations of PFOA and PFOS have shown positive associations with serum lipids, including cholesterol. Few published studies have assessed the associations between PFNA exposure and serum lipid levels. (30) A cross-sectional

study in China reported a positive association between total cholesterol with PFOA and PFNA after adjusting for age, gender, and BMI (test for trend: PFOA p=0.015; PFNA p=0.002). (31) In the US population, an analysis of the 2003–2004 NHANES data found a positive and significant relationship between total cholesterol (measured per mg/dL increase per μg/L of each PFAS) with PFOS (0.27 mg/dl, 95% CI: 0.05 to 0.48) and PFOA (1.22 mg/dl, 95% CI: 0.04 to 2.40) but not with PFNA (2.01 mg/dl, 95% CI: −1.16 to 5.18). However, a significant linear trend was seen across increasing quartiles of PFNA concentrations (p=0.04). (15) In a study of maternal serum lipids in a Norwegian cohort, one of the few significant findings was that total cholesterol was positively associated with PFOS (log-transformed as a continuous variable) such that for each ln-unit increase in PFOS, there was an increase of 8.96 mg/dL (95% CI: 1.70, 16.22) in total cholesterol. Both PFOS and PFNA concentrations were positively associated with increased HDL serum levels (PFOA: 4.39 mg/dl increase in HDL per ln-μg/L (95% CI: 2.37, 6.42); PFNA: 2.84 mg/dl increase in HDL per ln-μg/L (95% CI: 0.97, 4.71).

The study presented here had several limitations. The serum PFAS concentrations were obtained in the context of a legal settlement which raises concerns about potential selection bias. The PFAS serum testing was conducted by an established environmental laboratory which used different reporting standards then NHANES. Rather than reporting an LOD as is used by NHANES, a higher RL was provided with the PFAS results. While the current study adjusted for this by setting all NHANES results below the Paulsboro sample RL, the reported prevalence estimates from this community may not be directly compared to other community exposures where blood collection has been done for a research study and standardized reporting protocols were used.

Self-selection into this study may have resulted in either under- or over-estimation of serum blood levels for those exposed in Paulsboro and surrounding areas. Our respondents were on average older than all Paulsboro residents. If the levels of PFAS exposure experienced by this population are indeed associated with adverse health outcomes, then participation may have been higher among residents with exposure-related illness. If so, the study participants may represent the upper end of the distribution of exposure in Paulsboro. Conversely, higher exposed individuals with associated health outcomes may not have attended our recruitment sessions due to disabling conditions. If so, the study participants may represent the lower end of the distribution of exposure in Paulsboro. It would have been informative to compare the PFAS blood levels and demographic characteristics of the 186 people in the study with those of the 1,329 people who had their blood tested as part of this lawsuit. However, the demographic characteristics and PFAS serum concentrations for the claimants are not publicly available. In addition, for the potential for self-selection based on health outcomes, there is also the potential for self-selection on the basis of blood serum concentrations, as potential participants received their results prior to considering whether to participate in this study.

Another limitation of the study is that health outcome ascertainment was by self-report. We used standardized questions to minimize reporting errors. (32) Participants were asked to report whether they had been diagnosed by a doctor with any of 32 health conditions within eight groups. Due to the small sample size and low prevalence of many of these conditions,

most health outcomes were grouped, for example hypothyroidism and hyperthyroidism were analyzed in the “thyroid conditions” group. Some of the conditions grouped together likely have multiple independent causal pathways. If PFAS exposure were a component of one or more of those pathways, the contribution would need to be very large to have seen a significant odds ratio. For less common outcomes like cancer, it would not be possible to observe any association. The relatively small sample size also impacted our ability to assess PFAS prevalence and distribution among children. Two studies that have published PFAS serum concentrations among children with similar age ranges had PFAS detection limits in serum that were are much lower than those in our study. (33, 34) As such, these are not directly comparable to our findings. Future studies of this and other community PFAS exposures should seek to understand the extent and impact of PFAS exposure among children.

The cross-sectional nature of this study means that reverse causality in the association between serum PFNA levels and self-reported high cholesterol is possible. The study design and lack of in-depth exposure assessment means that it is not appropriate to use the data from this study to quantify the relationship between exposure due to the PFAS water contamination and PFAS blood levels.

This study also has some notable strengths, including that this is one of few studies that examined the impact of chronic high-level exposure to PFNA outside an occupational setting. The findings are useful for describing PFAS blood levels in this highly exposed population and for comparing PFAS serum levels to other exposure events.

Conclusion and Recommendations

More than two years after the remediation of the community water supply to remove PFAS, PFNA serum levels among Paulsboro residents were more than three times higher than that of the US population. A statistically significant and positive association between self-reported high cholesterol and PFNA exposure was observed, consistent with reports that other PFAS may affect blood lipid levels. As part of the study outreach, the authors provided information to the community about PFAS exposure and an interpretation of the PFAS laboratory results. Members of PFAS exposed communities should be informed of their past exposure, and they should share this information with their health care providers. The authors have informed physicians in the Paulsboro area about the PFNA exposure and referred them to the ATSDR Interim Guidance for responding to patients with PFAS exposure. (35)

The duration and intensity of PFNA contamination in the community water supply of Paulsboro is not known. PFAS are chemicals of environmental and health concern and further investigation into possible health effects of PFAS exposure in Paulsboro, and other community settings, is warranted. As well, since exposure has ceased, the toxicokinetics of PFNA elimination should be explored.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments, including grant information

This work was funded by the Rutgers Center for Environmental Exposure and Disease (CEED) pilot grant program NIEHS Center grant project number 5P30ES005022–29. We would like to thank the many people who made this project possible, including current and former residents of Paulsboro NJ who participated in this study, as well as: Kerry Butch of the CEED Community Outreach and Engagement Core, Brian Buckley PhD, Executive Director of Laboratories at EOHSI and the many EOHSI staff members and Rutgers SPH students who assisted with study implementation, including Shahnaz Alimokhtari, Omkar Bhawmik, Taylor Black, Clarimel Cepeda, Jennifer Gilman, Marta Hernandez, Prerna Malik, Amber Minnick, Alan Perez, Parita Ratnani, and Darsey Schulaka.

References

1. Bartolome M, Gallego-Pico A, Cutanda F, Huetos O, Esteban M, Perez-Gomez B, et al. Perfluorinated alkyl substances in Spanish adults: Geographical distribution and determinants of exposure. The Science of the total environment 2017;603–604:352–60.
2. Hu XC, Andrews DQ, Lindstrom AB, Bruton TA, Schaider LA, Grandjean P, et al. Detection of Poly- and Perfluoroalkyl Substances (PFASs) in U.S. Drinking Water Linked to Industrial Sites, Military Fire Training Areas, and Wastewater Treatment Plants. Environ Sci Technol Lett 2016;3(10):344–50. [PubMed: 27752509]
3. Oakes KD, Benskin JP, Martin JW, Ings JS, Heinrichs JY, Dixon DG, et al. Biomonitoring of perfluorochemicals and toxicity to the downstream fish community of Etobicoke Creek following deployment of aqueous film-forming foam. Aquat Toxicol 2010;98(2):120–9. [PubMed: 20206387]
4. Rappazzo KM, Coffman E, Hines EP. Exposure to Perfluorinated Alkyl Substances and Health Outcomes in Children: A Systematic Review of the Epidemiologic Literature. International journal of environmental research and public health 2017;14(7).
5. Dobraca D, Israel L, McNeel S, Voss R, Wang M, Gajek R, et al. Biomonitoring in California firefighters: metals and perfluorinated chemicals. Journal of occupational and environmental medicine / American College of Occupational and Environmental Medicine 2015;57(1):88–97.
6. Lindstrom AB, Strynar MJ, Libelo EL. Polyfluorinated compounds: past, present, and future. Environ Sci Technol 2011;45(19):7954–61. [PubMed: 21866930]
7. Zhao Z, Xie Z, Moller A, Sturm R, Tang J, Zhang G, et al. Distribution and long-range transport of polyfluoroalkyl substances in the Arctic, Atlantic Ocean and Antarctic coast. Environmental pollution 2012;170:71–7. [PubMed: 22771353]
8. Kato K, Wong LY, Jia LT, Kuklenyik Z, Calafat AM. Trends in exposure to polyfluoroalkyl chemicals in the U.S. Population: 1999–2008. Environ Sci Technol 2011;45(19):8037–45. [PubMed: 21469664]
9. US Environmental Protection Agency (EPA). PFAS Laws and Regulations 2018 [Available from: https://www.epa.gov/pfas/pfas-laws-and-regulations.
10. Official Journal of the European Union. Commission Regulation (EU) 2017/1000 of 13 June 2017 amending Annex XVII to Regulation (EC) No 1907/2006 of the European Parliament and of the Council concerning the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) as regards perfluorooctanoic acid (PFOA), its salts and PFOA-related substances [Available from: https://eur-lex.europa.eu/legal-content/EN/TXT/PDF/?uri=CELEX:32017R1000&from=EN.
11. International Agency on Cancer Research. List of Classifications by cancer sites with sufficient or limited evidence in humans, Volumes 1 to 114* Last updates Nov. 2015 [Available from: http://monographs.iarc.fr/ENG/Classification/Table4.pdf.
12. Post GB, Louis JB, Lippincott RL, Procopio NA. Occurrence of perfluorinated compounds in raw water from New Jersey public drinking water systems. Environ Sci Technol 2013;47(23):13266–75. [PubMed: 24187954]
13. Federal and NJ State Primary and Secondary Drinking Water Standards as of September 2018 2015 [Available from: https://www.state.nj.us/dep/watersupply/pdf/dw-standards.pdf.
14. Lopez-Espinosa MJ, Mondal D, Armstrong BG, Eskenazi B, Fletcher T. Perfluoroalkyl Substances, Sex Hormones, and Insulin-like Growth Factor-1 at 6–9 Years of Age: A Cross-Sectional Analysis

within the C8 Health Project. Environ Health Perspect 2016;124(8):1269–75. [PubMed: 26794451]

15. Nelson JW, Hatch EE, Webster TF. Exposure to polyfluoroalkyl chemicals and cholesterol, body weight, and insulin resistance in the general U.S. population. Environ Health Perspect 2010;118(2):197–202. [PubMed: 20123614]
16. Salihovic S, Stubleski J, Karrman A, Larsson A, Fall T, Lind L, et al. Changes in markers of liver function in relation to changes in perfluoroalkyl substances - A longitudinal study. Environ Int 2018;117:196–203. [PubMed: 29754000]
17. Lind PM, Salihovic S, van Bavel B, Lind L. Circulating levels of perfluoroalkyl substances (PFASs) and carotid artery atherosclerosis. Environmental research 2017;152:157–64. [PubMed: 27771570]
18. Kielsen K, Shamim Z, Ryder LP, Nielsen F, Grandjean P, Budtz-Jorgensen E, et al. Antibody response to booster vaccination with tetanus and diphtheria in adults exposed to perfluorinated alkylates. J Immunotoxicol 2016;13(2):270–3. [PubMed: 26181512]
19. DeNavas-Walt C PBD. Income and Poverty in the United States: 2014. Report Number P60–252 2015 [Available from: https://www.census.gov/library/publications/2015/demo/p60-252.html.
20. US Centers for Disease Control and Prevention (CDC). Laboratory Procedure Manual [Available from: https://wwwn.cdc.gov/nchs/data/nhanes/2013-2014/labmethods/PFAS_H_MET.pdf.
21. USDHHS. Centers fo Disease Control and Prevention. About the National Health and Nutrition Examination Survey (NHANES) 2018 [Available from: https://www.cdc.gov/nchs/nhanes/about_nhanes.htm.
22. US Census Bureau. American Fact Finder 2014 [Available from: https://factfinder.census.gov/faces/tableservices/jsf/pages/productview.xhtml?src=CF.
23. Lee SE FRN. Analyzing Complex Survey Data: Sage Publications Inc.; 2005.
24. Singer AB, Whitworth KW, Haug LS, Sabaredzovic A, Impinen A, Papadopoulou E, et al. Menstrual cycle characteristics as determinants of plasma concentrations of perfluoroalkyl substances (PFASs) in the Norwegian Mother and Child Cohort (MoBa study). Environmental research 2018;166:78–85. [PubMed: 29879567]
25. Rush EL, Singer AB, Longnecker MP, Haug LS, Sabaredzovic A, Symanski E, et al. Oral contraceptive use as a determinant of plasma concentrations of perfluoroalkyl substances among women in the Norwegian Mother and Child Cohort (MoBa) study. Environ Int 2018;112:156–64. [PubMed: 29274593]
26. Manzano-Salgado CB, Casas M, Lopez-Espinosa MJ, Ballester F, Martinez D, Ibarluzea J, et al. Variability of perfluoroalkyl substance concentrations in pregnant women by socio-demographic and dietary factors in a Spanish birth cohort. Environ Int 2016;92–93:357–65.
27. Olsen GW, Burris JM, Ehresman DJ, Froehlich JW, Seacat AM, Butenhoff JL, et al. Half-life of serum elimination of perfluorooctanesulfonate,perfluorohexanesulfonate, and perfluorooctanoate in retired fluorochemical production workers. Environ Health Perspect 2007;115(9):1298–305. [PubMed: 17805419]
28. Li Y, Fletcher T, Mucs D, Scott K, Lindh CH, Tallving P, et al. Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water. Occup Environ Med 2018;75(1):46–51. [PubMed: 29133598]
29. Zhang Y, Beesoon S, Zhu L, Martin JW. Biomonitoring of perfluoroalkyl acids in human urine and estimates of biological half-life. Environ Sci Technol 2013;47(18):10619–27. [PubMed: 23980546]
30. US Department of Health and Human Servcies, Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Perfluoroalkyls Draft for Public Comment June 2018 2018.
31. Fu Y, Wang T, Fu Q, Wang P, Lu Y. Associations between serum concentrations of perfluoroalkyl acids and serum lipid levels in a Chinese population. Ecotoxicology and environmental safety 2014;106:246–52. [PubMed: 24863755]
32. Centers for Disease Control and Prevetion. Behavioral Risk Factor Surveillance System (BRFSS) [Available from: http://www.cdc.gov/BRFSS/.

33. Schecter A, Malik-Bass N, Calafat AM, Kato K, Colacino JA, Gent TL, et al. Polyfluoroalkyl compounds in Texas children from birth through 12 years of age. Environ Health Perspect 2012;120(4):590–4. [PubMed: 22182702]

34. Ye X, Kato K, Wong LY, Jia T, Kalathil A, Latremouille J, et al. Per- and polyfluoroalkyl substances in sera from children 3 to 11 years of age participating in the National Health and Nutrition Examination Survey 2013–2014. Int J Hyg Environ Health 2018;221(1):9–16. [PubMed: 28993126]

35. Centers for Disease Control and Prevetion, National Center for Environmental Health, Agency for Toxic Substances and Disease RegistrAn Overview of Perfluoroalkyl and Polyfluoroalkyl Substances and Interim Guidance for Clinicians Responding to Patient Exposure Concerns. Revised on 6/7/2017 [Available from: https://www.atsdr.cdc.gov/pfc/docs/pfas_clinician_fact_sheet_508.pdf.

Table 1.

Demographic characteristics of the Paulsboro participants age 12 and older including age, sex, and race-ethnicity were compared with those from the NHANES 2013–2014 cycle.

	Paulsboro (n=165)			NHANES (n=1,599)	
	n	%	weighted %[a]	n	weighted %[b]
Age group					
12 to 19	16	8.6	15.7	251	14.8
20 to 39	28	15.1	27.0	381	26.2
40 to 59	70	37.6	34.3	460	35.6
≥ 60	51	27.4	21.5	507	22.4
Sex					
Male	68	36.6	44.4	835	46.2
Female	97	52.2	54.2	764	52.7
Race					
Hispanic	15	8.1	6.9	366	8.2
NH White	102	54.8	58.0	250	53.6
NH Black	44	23.7	32.5	641	35.7
Other	1	0.5		342	1.5
Years lived in Paulsboro					
1 to 9	41	22.0	28.9	--	--
10 to 19	51	27.4	34.8	--	--
20 to 34	29	15.6	13.8	--	--
≥35	44	23.7	21.2	--	--

[a] Study participants were significantly older than the Paulsboro population, weights adjust the sample to reflect the age distribution of Paulsboro New Jersey according to the 2014 US census.

[b] NHANES published sample weights.

Table 2.

Prevalence of detectable PFAS in the 165 Paulsboro study participants age 12 and older compared with NHANES 2013–2014 cycle results using both NHANES published level of detection (LOD) and that restricted to only the samples detected at the higher LOD in the Paulsboro serum test results

PFAS	Paulsboro Percent (95% CI)	NHANES Adjusted using Paulsboro data LOD[a] Percent (95% CI)[b]	NHANES Using NHANES LODs Percent (95% CI)
PFDeA	8.3 (3.9, 12.7)	11.4 (8.7, 14.2)	80.1(74.8, 85.5)
PFDoA	1.3 (0, 3.4)	0.6 (0.2, 0.9)	14.0 (7.1, 20.8)
PFHpA	1.1 (0, 3.3)	0.5 (0, 1.2)	12.5 (9.7, 15.2)
PFHxS	70.6 (62.3, 78.9)	68.0 (63.0, 73.1)	98.9 (98.4, 99.5)
PFNA	98.6 (96.9, 100)	73.8 (67.9, 79.6)	98.8 (98.0, 99.5)
PFOA	98.2 (95.8, 100)	94.2 (92.9, 95.5)	96.5 (95.1, 97.9)
PFOS	98.1 (96.2, 100)	93.9 (92.3, 95.5)	97.1 (95.9, 98.2)
PFUA	10.7 (6.0, 15.3)	6.1 (4.1, 8.0)	41.3 (34.2, 48.5)

[a]The PFAS was defined as detectable if the level reported was above the reporting limits of the Paulsboro test result: PFDeA, PFHpA, PFHxS, PFNA, PFUA, PFDoA = minimum 0.50 μg/L; PFOS and PFOA = 1.0 μg/L; NHANES adjusted comparisons includes sets all NHANES results below the RL to non-detectable.

Table 3.

Geometric mean and selected percentiles of selected per- and polyfluoroalkyl substances (PFAS) concentrations in serum (μg/L) for the population 12 years of age and older in Paulsboro New Jersey compared with U.S. NHANES 2013–2014 (n=165)

PFAS		n	Geometric Mean (95% CI)	25th (95% CI)	50th (95% CI)	75th (95% CI)	95th (95% CI)
PFHxS	Paulsboro	124	2.03 (1.84, 2.25)	1.33 (1.20, 1.45)	2.02 (1.67, 2.38)	2.77 (2.49, 3.05)	4.70 (3.63, 5.76)
	NHANES	1432	2.18 (2.02, 2.36)	1.31 (1.24, 1.38)	1.93 (1.76, 2.10)	3.10 (2.74, 3.45)	6.46 (4.48, 8.44)
PFOA	Paulsboro	165	3.03 (2.70, 3.40)	1.94 (1.67, 2.22)	2.98 (2.43, 3.53)	4.69 (3.87, 5.51)	8.80 (6.91, 10.70)
	NHANES	2080	2.08 (1.91, 2.26)	1.35 (1.28, 1.43)	2.05 (1.89, 2.21)	3.06 (2.74, 3.37)	5.57 (4.63, 6.51)
PFOS	Paulsboro	164	5.37 (4.75, 6.06)	3.09 (2.57, 3.60)	5.66 (4.73, 6.59)	9.28 (7.93, 10.62)	14.76(11.62, 17.90)
	NHANES	2098	5.39 (4.98, 5.84)	3.20 (2.88, 3.51)	5.34 (4.95, 5.72)	8.76 (8.08, 9.43)	18.48 (15.26, 21.70)
PFNA	Paulsboro	165	3.50 (3.04, 4.04)	2.01 (1.60, 2.42)	3.89 (3.18, 4.60)	5.99 (4.47, 7.51)	12.41 (9.68, 15.13)
	NHANES	1599	0.91 (0.87, 0.96)	0.58 (0.57, 0.59)	0.78 (0.75, 0.81)	1.18 (1.09, 1.28)	2.22 (1.90, 2.54)

Table 4.

Geometric mean and selected percentiles of perfluorononanoic acid (PFNA) concentrations in serum (in ug/ml) for the population 12 years of age and older in Paulsboro New Jersey compared with U.S. NHANES 2013–2014, stratified by age, sex and race-ethnicity (n=165)

		n	Geometric Mean (95% CI	25th (95% CI)	50th (95% CI)	75th (95% CI)	95th (95% CI)
Age group (years)							
Paulsboro	12 to 19	16	2.43 (1.74, 3.40)	1.41 n/a[a]	2.12 (0.68, 3.55)	4.06 (2.12, 6.01)	6.22 (n/a)[a]
NHANES	12 to 19	251	0.84 (0.68, 1.04)	0.51 (0.48, 0.54)	0.69 (0.59, 0.78)	1.07 (0.56, 1.57)	2.58 (n/a)[a]
Paulsboro	20 to 39	28	2.13 (1.64, 2.79)	1.18 (0.57, 1.79)	2.13 (1.09, 3.17)	3.78 (2.37, 5.18)	4.35 (n/a)[a]
NHANES	20 to 39	381	0.79(0.74, 0.83)	0.54 (0.52, 0.55)	0.68 (0.64, 0.72)	0.89 (0.84, 0.95)	1.64 (1.40, 1.89)
Paulsboro	40 to 59	70	4.30 (3.60, 5.13)	2.42 (1.45, 3.40)	4.97 (3.97, 5.96)	7.54 (6.17, 8.91)	11.92 (9.53, 14.30)
NHANES	40 to 59	460	0.94 (0.87, 1.01)	0.61 (0.54, 0.67)	0.80 (0.70, 0.90)	1.23 (1.10, 1.37)	2.22 (1.72, 2.72)
Paulsboro	≥ 60	51	6.15 (4.94, 7.66)	3.78 (2.38, 5.18)	6.26 (3.84, 8.68)	10.45 (7.58, 13.33)	16.84 (n/a)[a]
NHANES	≥ 60	507	1.06 (0.98, 1.16)	0.70 (0.65, 0.76)	0.94 (0.87, 1.01)	1.37 (1.23, 1.51)	2.60 (1.57, 3.64)
Sex							
Paulsboro	Male	68	4.32 (3.51, 5.31)	2.42 (1.52, 3.33)	4.18 (3.45, 4.91)	7.15 (4.80, 9.50)	14.27 (10.39, 18.14)
NHANES	Male	835	0.92 (0.86, 1.00)	0.60 (0.58, 0.61)	0.79 (0.74, 0.84)	1.19 (1.06, 1.33)	2.25 (1.93, 2.57)
Paulsboro	Female	97	2.95 (2.48, 3.52)	1.58 (1.17, 2.00)	2.66 (1.65, 3.66)	5.43 (3.96, 6.89)	11.12 (7.95, 14.29)
NHANES	Female	764	0.90 (0.86, 0.94)	0.57 (0.56, 0.58)	0.77 (0.74, 0.79)	1.16 (1.07, 1.26)	2.17 (1.69, 2.64)
Race							
Paulsboro	Hispanic	15	3.13 (1.99, 4.94)	1.40 (n/a)[a]	2.96 (0.68, 5.23)	5.45 (1.82, 9.08)	8.56 (n/a)[a]
NHANES	Hispanic	366	0.77 (0.71, 0.83)	0.51 (0.50, 0.53)	0.65 (0.59, 0.71)	0.87 (0.79, 0.95)	1.85 (1.40, 2.30)
Paulsboro	NH Black	44	3.28 (2.49, 4.31)	1.60 (0.81, 2.39)	3.79 (2.76, 4.82)	5.07 (2.46, 7.69)	10.08 (4.92, 15.24)
NHANES	NH Black	641	0.93 (0.87, 0.98)	0.61 (0.56, 0.65)	0.80 (0.75, 0.85)	1.22(1.10, 1.33)	2.16 (1.68, 2.64)
Paulsboro	NH White	102	3.69 (3.09, 4.41)	2.09 (1.50, 2.68)	4.11 (3.01, 5.21)	6.39 (4.75, 8.02)	13.75 (10.88, 16.62)
NHANES	NH White	250	1.00 (0.89, 1.12)	0.59 (0.56, 0.62)	0.83 (0.67, 0.99)	1.42 (1.12, 1.72)	2.83 (2.24, 3.41)
Years Lived in Paulsboro							
	1 to 9	41	2.43 (1.93, 3.06)	1.30 (0.83, 1.78)	2.24 (0.98, 3.50)	4.14 (2.87, 5.40)	5.95 (n/a)[a]
	10 to 19	51	3.17 (2.44, 4.12)	1.60 (0.89, 2.30)	2.89 (1.83, 3.95)	6.18 (3.91, 8.46)	11.86 (8.07, 15.65)
	20 to 34	29	4.56 (3.69, 5.62)	3.52 (2.13, 4.91)	4.96 (4.01, 5.91)	5.84 (4.35, 7.32)	9.59 (n/a)[a]

	n	Geometric Mean (95% CI	25th (95% CI)	50th (95% CI)	75th (95% CI)	95th (95% CI)
	≥35	44	5.73 (4.52, 7.26)	2.79 (1.57, 4.01)	6.36 (4.05, 8.66)	9.78 (6.96, 12.60)	15.82(n/a)[a]

[a]Confidence intervals were not calculated for numerators of less than 5

Table 5.

Associations between serum concentration of PFNA and demographic characteristics for all participants (n=186)

	Estimate	95% CI	P-value
Intercept	0.571	(0.125, 0.909)	0.0100
Age for each gender*			
Female	0.015	(0.007, 0.023)	<.0001
Male	0.237	−(0.282, 0.757)	0.3686
Race-ethnicity (ref=NH White)			
Black	−0.063	(−0.324, 0.198)	0.0630
Other	−0.284	(−0.620, 0.053)	0.0979

* The estimates are for the change in PFNA for each year of age among males and females. [The model includes terms for the main effect of age and gender as well an interaction term for these variables, as such the estimates for age groups shown accounts for the interaction (p=0.0137)]

Table 6.

Associations between serum concentrations of the four PFAS and self-reported[a] high cholesterol and any other cardiovascular condition, assessed using logistic regression models[b] (n=105)

	PFNA		PFOA		PFOS		PFHxS[b]		All	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Outcome = High Cholesterol										
PFAS										
PFNA	1.16	(1.03, 1.29)							1.10	(0.94, 1.29)
PFOA			1.12	(0.94, 1.35)					1.03	(0.94, 1.14)
PFOS					1.10	(0.99, 1.22)			1.08	(0.94, 1.24)
PFHxS[c]							1.07	(0.99, 1.16)	1.04	(0.96, 1.13)
Age	1.03	(0.99, 1.06)	1.03	(1.00, 1.07)	1.03	(0.99, 1.07)	1.07	(1.02,1.12)	1.05	(0.99, 1.10)
BMI	0.96	(0.90, 1.03)	0.98	(0.92, 1.04)	0.99	(0.92, 1.05)	1.01	(0.93,1.10)	0.99	0.91, 1.09)
Outcome = Other Cardiovascular Conditions										
PFAS										
PFNA	1.04	(0.93, 1.17)							1.05	(0.89, 1.25)
PFOA			0.97	(0.90, 1.05)					0.95	(0.84, 1.08)
PFOS					1.08	(0.98, 1.21)			1.10	(0.95, 1.27)
PFHxS[c]							1.02	(0.94,1.10)	1.00	(0.92, 1.09)
Age	1.07	(1.03, 1.12)	1.08	(1.04, 1.13)	1.07	(1.03, 1.11)	1.10	(1.05,1.16)	1.09	(1.03, 1.15)
BMI	1.09	(1.02, 1.18)	1.10	(1.02, 1.18)	1.11	(1.03, 1.19)	1.14	(1.04,1.25)	1.15	(1.04, 1.28)

[a] All health outcomes are self-reported as a positive response to the question “Have you ever been diagnosed by a doctor with any of the following health conditions?”

[b] All variables were entered as continuous variables

[c] PFHxS was entered all models in the quadratic form

J Expo Sci Environ Epidemiol. Author manuscript; available in PMC 2019 May 27.