PFAS Exposure Assessments Final Report

INFORMATION TO PROTECT OUR COMMUNITIES

Findings Across Ten Exposure Assessment Sites

National Center
for Environmental Health
Agency for Toxic Substances
and Disease Registry

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About ATSDR

The Agency for Toxic Substances and Disease Registry (ATSDR) is a federal public health agency of the U.S. Department of Health and Human Services (HHS). ATSDR works with other agencies and state, tribal and local governments to protect communities from harmful health effects related to exposure to natural and manmade hazardous substances. For more information about ATSDR, visit https://www.atsdr.cdc.gov/

Abbreviations

9Cl-PF3ONS	9-chlorohexadecafluoro-3-oxanone-1-sulfonic acid
11Cl-PF3OUdS	11-chloroeicosafluoro-3-oxaundecane-1-sulfonic acid
AFFF	aqueous film forming foam, also known as “A triple F”
ASTHO	Association of State and Territorial Health Officials
ATSDR	Agency for Toxic Substances and Disease Registry
CDC	Centers for Disease Control and Prevention
DONA	4,8-dioxa-3H-perfluorononanoic acid
EA	exposure assessment
EMEG	environmental media evaluation guide
EPA	U.S. Environmental Protection Agency
EtFOSAA	N-ethyl perfluorooctanesulfonamidoacetic acid
FOD	frequency of detection
FtS 4:2	fluorotelomer sulfonic acid 4:2
FtS 6:2	fluorotelomer sulfonic acid 6:2
FtS 8:2	fluorotelomer sulfonic acid 8:2
HA	health advisory
HFPO-DA (GenX)	hexafluoropropylene oxide dimer acid
HHS	U.S. Department of Health and Human Services
LOD	limit of detection
MeFOSAA	N-methyl perfluorooctanesulfonamidoacetic acid
µg/L, or ug/L	micrograms per liter (same as parts per billion or 1,000 parts per trillion)
ng/g	nanograms per gram (same as parts per billion or micrograms per kilogram)
N-EtFOSA	N-ethyl perfluorooctanesulfonamide
N-EtFOSE	N-ethyl perfluorooctanesulfonamidoethanol
N-MeFOSA	N-methyl perfluorooctanesulfonamide
N-MeFOSE	N-methyl perfluorooctanesulfonamidoethanol
n-PFOA	linear isomer of PFOA
n-PFOS	linear isomer of PFOS
NDAA	National Defense Authorization Act
NHANES	National Health and Nutrition Examination Survey
NYSDOH	New York State Department of Health
PADOH	Pennsylvania Department of Health
PEATT	PFAS Exposures Assessment Technical Tools
PFAS	per- and polyfluoroalkyl substances
PFBA	perfluorobutanoic acid
PFBS	perfluorobutane sulfonic acid
PFDA	perfluorodecanoic acid
PFDoA	perfluorododecanoic acid
PFDS	perfluorodecane sulfonic acid
PFDoS	perfluorododecanesulfonate

PFHpA	perfluoroheptanoic acid
PFHpS	perfluoroheptane sulfonic acid
PFHxA	perfluorohexanoic acid
PFHxS	perfluorohexane sulfonic acid
PFNA	perfluorononanoic acid
PFNS	perfluorononane sulfonic acid
PFOA	perfluorooctanoic acid
PFOS	perfluorooctane sulfonic acid
PFOSA	Perfluorooctanesulfonamide
PFPeA	perfluoropentanoic acid
PFPeS	perfluoropentane sulfonic acid
PFTA	perfluorotetradecanoic acid
PFTrA	perfluorotridecanoic acid
PFUnA	perfluoroundecanoic acid
ppt	parts per trillion (same as 1 nanogram per liter)
Sb-PFOA	branched isomers of PFOA
Sm-PFOS	branched isomers of PFOS

Executive Summary

Background and Purpose

PFAS (or per- and polyfluoroalkyl substances) are a family of synthetic chemicals that have been used in industry and consumer products since the 1940s. There are thousands of different PFAS. The exposure assessments (EAs) described in this report evaluated some of the most commonly studied PFAS, such as perfluorooctanoic acid (PFOA), perfluorooctane sulfonic acid (PFOS), perfluorohexane sulfonic acid (PFHxS), perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA), perfluoroundecanoic acid (PFUnA), and N-methyl perfluorooctanesulfonamidoacetic acid (MeFOSAA).

PFAS do not occur naturally but are widespread in the environment. They have been found in soil, water, air, and in animals and plants. Most PFAS (including PFOA, PFOS, PFHxS, PFNA, PFDA, and PFUnA) are either very resistant to breaking down or degrade into other PFAS that do not break down further in the environment. Major exposure routes for PFAS include drinking contaminated water and eating contaminated food, but exposure may also occur through other routes (e.g., ingestion of contaminated dust or inhalation of PFAS in air). Once PFAS enter people's bodies, some of them (including PFOA, PFOS, PFHxS, and PFNA) can remain in the body for long periods and can be measured in the blood years after exposure. Most people in the United States have been exposed to PFAS. At least one PFAS was detected in more than 99% of National Health and Nutrition Examination Survey (NHANES) samples collected for the 1999-2000 survey cycle.

The Centers for Disease Control and Prevention (CDC) and the Agency for Toxic Substances and Disease Registry (ATSDR) conducted EAs in the following 10 communities that are near current or former military bases and were found to contain PFAS levels in the past exceeding the Environmental Protection Agency's (EPA) 2016 health advisory (HA) of 70 parts per trillion (ppt) for PFOA and PFOS combined::

- Westhampton Beach and Quogue Area, New York (NY pilot EA)
- Montgomery and Bucks Counties, Pennsylvania (PA pilot EA)
- Hampden County, Massachusetts (Westfield EA)
- Berkeley County, West Virginia (Berkeley County EA)
- New Castle County, Delaware (New Castle County EA)
- Spokane County, Washington (Airway Heights EA)
- Lubbock County, Texas (Lubbock County EA)
- Fairbanks North Star Borough, Alaska (Moose Creek EA)
- El Paso County, Colorado (Security-Widefield EA)
- Orange County, New York (Orange County EA)

The two pilot EAs conducted in NY and PA were implemented by state agencies under a cooperative agreement, and the remaining eight EAs were implemented by ATSDR. This report (1) summarizes and compares individual site results from all 10 EA sites, (2) explores the relationship between previously measured PFAS levels in drinking water and currently measured PFAS levels in blood across all 10 EA sites, and (3) analyzes combined data across the eight ATSDR-led EA sites; these analyses were only possible for the ATSDR-led subset of sites due to differences in methods and data availability for the

state-led pilot EA sites. Individual reports were published detailing the findings from each of the EA communities.[1]

Possibly as early as the 1970s, Air Force and Air National Guard bases used aqueous film forming foam (AFFF) containing PFAS for firefighter training and for responses to fire events. Over time, the PFAS from the AFFF entered the ground, moved to offsite locations, and affected nearby municipal wells, private wells, or surface drinking water supplies. At all EA sites, exposures were mitigated through corrective actions, which included inactivating contaminated wells, switching to new water sources, installing filtration and treatment systems, or providing alternative drinking water supplies. Final mitigation was achieved in each community at different times between 2014 and 2019. Based on information available to ATSDR, all households in affected areas across the EAs now have a drinking water supply with PFAS concentrations that meet or are below current federal and state guidelines for PFAS in drinking water. ATSDR does not recommend that community members who get drinking water from any of the affected public water systems use alternative sources of water. For affected private wells, ATSDR recommends community members continue to use the alternative sources of water or filtration systems provided to them.

These EAs assessed PFAS levels in the blood and urine of some residents in each community living near the current or former military bases where public water systems or private wells had PFAS levels above EPA's HA. Participant selection was based on methods prescribed in the EA study protocol and aimed at collecting data that were generalizable to each sampling frame (areas within the site communities where known or expected PFAS exposure occurred). Test results were compared to PFAS levels in a nationally representative sample (NHANES). Tap water and indoor dust samples from a subset of participating households were also analyzed. These EA results will help participants and their communities better understand their PFAS exposure, allow ATSDR to provide recommendations to reduce exposure, and inform public health efforts related to protecting communities from sources of PFAS other than contaminated drinking water supplies.

ATSDR will use the data collected from these EAs to help inform future studies of PFAS exposures.

Pilot EA Activities

Two of the 10 EAs were conducted through a cooperative agreement with the Association of State and Territorial Health Officials (ASTHO). ASTHO funded the Pennsylvania Department of Health (PADOH) and the New York State Department of Health (NYSDOH) to conduct pilot biomonitoring studies using the CDC/ATSDR PFAS Exposures Assessment Technical Tools (PEATT). ATSDR developed the PEATT to help state, local, tribal, and territorial health departments conduct PFAS biomonitoring activities, with the assumption that drinking water is the primary source of PFAS exposure. The PEATT includes a protocol for identifying a representative sample of exposed households, risk communication materials, questionnaires, and EPA's water sampling protocol to help characterize PFAS exposure in communities. The goal of the two pilot EAs was to gather biomonitoring data in two communities and identify possible

[1] Individual reports are available online at https://www.health.pa.gov/topics/Documents/Environmental%20Health/PEATT%20Pilot%20Project%20Final%20Report%20April%2029%202019.pdf (PA pilot EA), https://www.health.ny.gov/environmental/investigations/drinkingwaterresponse/docs/westhampton_quogue_group_level_blood_testing (NY pilot EA), and https://www.atsdr.cdc.gov/pfas/activities/assessments/sites.html (ATSDR-led EAs).

ways of improving the PEATT as a tool for states and territories to use when measuring and evaluating community exposures to PFAS in drinking water.

In 2018, PADOH and NYSDOH invited randomly selected households in communities affected by PFAS to participate in the pilot EAs. In total, blood and exposure histories were collected from 396 participants from 196 households. A small subset of follow-on urine and environmental sampling was also conducted in Pennsylvania. New York opted out of conducting the PEATT expansion which provided funding for the urine and environmental sampling. The methods and results of the two pilot assessments informed the protocol for subsequent PFAS EAs. For the purposes of this report, only the blood data from the pilot EAs are combined with the other EA data for selected statistical analyses.

ATSDR-led EA Activities

In 2018, the National Defense Authorization Act (NDAA) authorized CDC/ATSDR to complete EAs in communities near military installations to assess PFAS drinking water exposures. In response, ATSDR conducted eight additional PFAS EAs after the completion of the pilot EAs. ATSDR invited households who met the eligibility criteria to participate in these EAs. To be eligible to participate, household residents must have (1) lived in an affected area and received their drinking water from a contaminated water source for at least 1 year before exposures were mitigated (these residents have the greatest likelihood of past exposures to PFAS via their water source), (2) been greater than three years old at the time of sample collection, and (3) not been anemic or had a bleeding disorder that would prevent giving a blood sample.

Between September 2019 and October 2020, 1,988 eligible people (1,791 adults and 197 children) from 1,094 households participated in EA sample collection events where ATSDR:

- administered exposure history questionnaires
- collected blood and urine samples
- collected tap water and dust samples from a randomly selected subset of homes
- tested for 7 PFAS in blood, 14 in urine, 18 in water, and 33 in dust (note blood analysis was conducted for all participants while urine, water, and dust analyses were conducted for subsets of participants)[2]
- measured PFHxS, PFOS, PFOA, PFNA, PFDA, and PFUnA across all media
- mailed individual biological and environmental results to participants

These EAs were designed to estimate geometric mean ("average") concentrations of PFOS in blood for each community with a precision goal of at least 15%. The target sample size needed to meet precision goals was informed by findings from the pilot EAs. The precision is a measure of how wide the confidence interval is around the estimated geometric mean. ATSDR met the precision goal for PFOS at all eight sites. Precision was calculated for all measured species, but the target sample size was based on PFOS precision alone. The EAs were also designed to estimate PFAS levels in blood that were generalizable to the population as a whole in each EA community, but ATSDR explored the potential for any participation bias—that is, substantive differences between those who chose to participate and

[2] The laboratory reports branched and linear isomers of PFOA and PFOS in blood and urine. ATSDR reports on the sum of the individual isomer concentrations of PFOA and PFOS.

those who did not. For precision estimates for each PFAS from each EA and for further discussion on participation bias, refer to each EA site's report.

The ATSDR-led EA samples were collected and analyzed in accordance with ATSDR's *Exposure Assessment Protocol: Biological and Environmental Sampling of PFAS* (EA protocol) to ensure their quality. For the pilot EAs, investigators followed similar procedures outlined in ATSDR's PEATT although differences in the information collected through participant surveys did not allow ATSDR to combine data from the pilot and ATSDR-led EAs for some statistical analyses.

Analyses Conducted for this Report

All EAs

This report summarizes community PFAS blood levels, measured in serum, for the group of residents who participated in all 10 EAs. The blood data for each of the EAs are compared to nationally representative samples of the U.S. population. Specifically, ATSDR compared each EA dataset to NHANES data collected by CDC, controlling for differences in the age distributions of the two populations. ATSDR looked at two NHANES survey periods (2015–2016 and 2017–2018). For all EAs, ATSDR also explored the relationship between observed past PFAS drinking water levels at each site and corresponding blood levels.

In this report, we refer to the two pilot PFAS data collection efforts as "pilot EAs" and the remaining eight EAs as the "ATSDR-led EAs." "All EAs" refer to both the pilot and ATSDR-led EAs. When we write blood levels of PFAS, we are referring to the measurement of PFAS in the serum fraction of the blood.

ATSDR-led EAs

For the eight ATSDR-led EAs only, this report also evaluates PFAS levels by age, race/ethnicity, sex, number of years living in the community, drinking water consumption patterns, and other exposure parameters. In addition, the urine sample results from a subset of participants are presented, as well as the results from the dust and tap water samples. To assess possible relationships between blood levels and various demographic and exposure variables, ATSDR used statistical models on the combined data across the eight sites. Univariate statistics, which evaluate one variable at a time, were used as a tool to examine the data broadly and find patterns within the data. Multivariate regression modeling was used to simultaneously account for multiple variables and to control for potential confounding factors.[3] While this same type of modeling was conducted for individual sites, combining the data across sites allows for a more robust analysis. It allows ATSDR to further evaluate the associations between measured blood levels and various exposure variables. A potential limitation in combining data from different populations is that the overall variability in the data is increased, which may actually reduce statistical power or produce potentially spurious results. However, the overarching goal of the EAs was to examine the strength of the association of PFAS levels in drinking water measured across sites while controlling for other exposure variables as potential confounders.

See the "Methods" section of this report for details on ATSDR's analytical approaches.

[3] A confounding variable is a factor that may distort or mask the relationship between a potential predictor and measure of exposure. For example, age may confound the relationship between blood PFAS levels and how long someone lived in their home, because older people tend to have lived in their homes longer than younger people.

Key Findings

Main conclusions from the EAs are presented below, which describe

- How PFAS blood levels measured at all EA sites compare to national levels (Findings 1–3),
- Why PFAS blood levels measured across all 10 EA sites are believed to be strongly associated with past drinking water exposures (Finding 4),
- Possible associations between PFAS blood levels and other exposure variables based on analysis of combined data from the eight ATSDR-led EAs (Finding 5), and
- Urine (Finding 6) and environmental sampling (Findings 7 and 8) results from the eight ATSDR-led EAs.

Findings for the 10 individual EAs can be found in the separately published EA-specific reports.

Finding 1. Average age-adjusted PFHxS blood levels are higher than national levels in all 10 EA communities.

At all 10 EA sites, the age-adjusted geometric mean (i.e., average) PFHxS levels were statistically higher ($p<0.05$) in EA participants compared to national levels (CDC's NHANES 2017–2018). Of all the PFAS analyzed in blood, PFHxS levels had the largest elevations when compared to national levels. Across EA sites, geometric mean blood PFHxS levels ranged from 2.4 to 61 times national levels. The highest age-adjusted geometric mean blood level for PFHxS was 65.6 micrograms per liter (µg/L), observed in Airway Heights, WA.

Finding 2. Average age-adjusted PFOS and PFOA blood levels are higher than national levels in most EA communities.

Average PFOS was statistically elevated in 8 of 10 EAs at 1.2 to 9.2 times national levels, and average PFOA was statistically elevated in 7 of 10 EAs at 1.2 to 6.3 times national levels. The highest age-adjusted geometric mean PFOS (39.1 µg/L) and PFOA (8.9 µg/L) blood levels also were observed in Airway Heights, WA.

Finding 3. Other average age-adjusted PFAS blood levels were higher than national levels in some but not all EA communities.

PFNA levels were also statistically elevated in 4 of 10 EAs, and blood levels at these EA sites ranged from 1.4 to 2.2 times national levels with a maximum adjusted geometric mean of 0.903 µg/L. The remaining PFAS (PFDA, PFUnA, and MeFOSAA; see abbreviation list for full list of PFAS names) were detected at lower frequencies. Adjusted geometric mean blood PFDA and PFUnA were statistically elevated at one site (New Castle, DE) at levels that were 1.4 and 1.7 times NHANES 2017–2018 levels, respectively. Blood MeFOSAA levels were not statistically elevated at any sites.

Finding 4. Elevated blood levels of PFHxS, PFOS, and PFOA may result from past drinking water contamination.

Multiple lines of evidence from the EAs support the finding that past exposure to contaminated drinking water may have contributed to the elevated blood levels.

- *The highest PFAS blood levels were observed in participants who lived in communities with the highest historical PFAS drinking water levels.* The strongest evidence linking blood PFAS levels to drinking water data is the consistent association observed with maximum historic

concentrations of PFAS measured in drinking water at all EA sites. Drinking water measurements were provided by affected water systems or the Air Force (in the case of private well sites) for PFHxS, PFOS, and PFOA. The individual drinking water measurements were statistically associated with corresponding PFAS measured in blood in the combined data from all 10 EA sites. In other words, residents of households that had the highest contamination for PFHxS, PFOS, and PFOA in their drinking water generally had higher blood levels for these substances.

- *Three of the PFAS (PFHxS, PFOS, and PFOA) with statistically elevated blood levels were previously detected in drinking water supplies at all EA sites.* PFAS were first detected in drinking water supplies at EA sites between 2009 and 2017. We do not know if contamination began earlier because no earlier data are available. From 2014 through 2019, each site had reduced PFAS levels below EPA's 2016 HA in each drinking water supply. Because of the long biological half-lives of PFAS (2.1 to 35 years), past drinking water exposures may have contributed to the EA participants' elevated blood PFAS levels observed sometimes years later. Of the PFAS measured in participants' blood, PFHxS has the longest estimated half-life, which, combined with the relatively high concentrations of PFHxS previously measured in drinking water in these communities, may be why PFHxS blood levels exceeded national levels by the largest margin.
- *Correlation across PFAS in blood suggests a common exposure source.* PFHxS, PFOS, and PFOA were highly correlated in EA participants' blood (Pearson correlation coefficient, *r* between 0.83 and 0.86 for the combined data from all 10 EAs). This means that, typically, residents who had elevated blood PFHxS levels also had elevated blood PFOS and blood PFOA levels. This correlation suggests a common exposure source, such as drinking water, though other sources of exposure may also have contributed to the observed blood levels. It also suggests a common contamination source for drinking water, such as AFFF, which contained these three PFAS in historical formulations.
- *Drinking water consumption patterns were shown to be predictors of blood PFAS levels.* Regression modeling conducted across the eight ATSDR-led EAs further supports this finding:
    - In univariate and multivariate models, a consistent predictor of participant blood PFHxS, PFOS, and PFOA levels was how long the resident had lived in the sampling frame during the past 20 years. Those who lived in the area longest had higher PFHxS, PFOS, and PFOA blood levels—and also likely drank the contaminated water for the longest period.
    - After controlling for other variables in multivariate models, personal drinking water consumption rates were associated with blood PFHxS levels.
    - In univariate and multivariate analyses, adults who used at least one filter or treatment device in their homes and adults who reported not drinking tap water at all (i.e., only reported drinking bottled water) on average had statistically lower blood levels of PFHxS, PFOS, and PFOA when compared to those reporting no filter or treatment device.
    - In multivariate analyses, as the number of days since drinking water mitigation increased, average blood PFOS and PFOA levels decreased in adults.

Finding 5. PFAS blood levels varied with different demographic and exposure characteristics of the participant population.

As highlighted below, the multivariate analyses conducted for the combined data set for the eight ATSDR-led EAs (adults and children) revealed statistically significant associations between PFAS blood levels and some of the demographic and exposure variables examined. While the EAs were not designed to quantify other exposures as potential predictors for PFAS blood levels, the findings from these EAs

show other factors that may influence PFAS blood levels, many of which have been documented in previous studies. Because these EAs were not designed to characterize non-drinking water exposures, the strength of these associations varied, and the results of these analyses should be interpreted with caution. Some of these associations may be due to chance as we are testing many associations at once. Some of the variables were associated with blood levels of those PFAS elevated in drinking water (PFHxS, PFOS, and PFOA) and others associated with PFNA or PFDA blood levels only.

- *Age*. Blood levels of PFHxS, PFOS, PFOA, and PFNA were statistically higher in older adult participants, and the size of the effect was stronger in females. In children between 3 and 17 years of age, blood PFHxS and PFOA levels decreased for every additional year in age.
- *Sex*. Male adults had statistically higher blood levels of PFHxS, PFOS, PFOA, and PFNA than females, and the difference between males and females was larger in younger adults. In children, blood levels in males were higher than females for PFOS, PFOA, PFNA, and PFDA.
- *Race/ethnicity.* Race and ethnicity were only associated with blood PFNA levels in multivariate models for adults and children. Compared to those who identified as "White, non-Hispanic," some groups had higher PFNA blood levels, while others had lower PFNA levels.
- *Cleaning frequency*. Adults who reported cleaning their homes more frequently had higher PFNA blood levels than adult participants who reported cleaning their homes a few times per year or less.
- *Soil contact*. Children who reported coming in contact with soil more frequently had higher levels of PFOS, PFNA, and PFDA.
- *Eating locally grown produce*. Adults and children who reported eating locally grown fruit and vegetables had higher blood PFDA levels than those who did not.
- *Consuming local dairy*. Adults who reported drinking locally produced milk 'rarely' or more frequently had higher blood PFHxS and PFOA levels compared to participants who reported never drinking locally produced milk
- *Using stain-resistant products*. Participants who reported using stain-resistant products a few times per year or more frequently had blood levels of PFNA that were higher than participants who never used them.
- *Number of children*. Female adults who reported having children had lower blood PFHxS levels than females who did not have children.
- *Breastfeeding and infant formula consumption*. In child participants, every additional month of reported breastfeeding was associated with an increase in blood PFDA levels. Every additional month of formula consumption was associated with an increase in blood PFNA levels.

Finding 6. PFHxS, PFHxA, and PFBA were detected in urine at low concentrations.

ATSDR analyzed 204 of the urine samples collected (189 adults and 15 children) as part of the eight ATSDR-led EA. PFHxS, PFHxA, and PFBA were the only PFAS detected in any of the 204 urine samples analyzed from eligible participants. Concentrations of biologically persistent PFAS are expected to be higher in serum than in urine, as was observed in this assessment. This trend is also evident in other biomonitoring studies in the general population and in communities with known PFAS exposures.

- PFBA was detected in 79 of the 204 urine samples that were analyzed (38%). This PFAS was measured in participants from six different sites and at individual sample concentrations ranging from 0.1 µg/L to 2.2 µg/L. Note, there are challenges in measuring trace levels of PFBA,

including selectivity of the analytical instrumentation and potential for external contamination. Therefore, we advise caution when interpreting the PFBA results.

- PFHxS was detected in 9 urine samples (4%) and detected individual sample concentrations ranged from 0.1 µg/L to 0.4 µg/L. PFHxS is not generally found in urine. Detection of PFHxS suggests recent exposure or elevated PFHxS levels in blood. All PFHxS detections were in the Airway Heights, WA location, which had the highest measured PFHxS concentrations in blood and among the most recent exposures to PFAS in drinking water prior to mitigation of the sites evaluated.
- PFHxA was detected in two urine samples (1%) and only among participants from the Lubbock County, TX EA.

ATSDR did not analyze all participants' urine samples because none of the species were detected in more than 60% of the samples analyzed at any site.

Finding 7. Almost all tap water samples collected during the ATSDR-led EAs were below all federal and state guidelines for PFAS in drinking water in place at the time samples were collected.

Only two of the 176 tap water samples (101 unfiltered and 75 filtered) from 117 randomly selected EA participant households were above federal or state guidelines for PFAS in drinking water at the time of the ATSDR-led EAs. The two samples exceeded ATSDR's environmental media evaluation guide (EMEG) for PFOS in drinking water. One sample came from a whole-house filter that may not have been properly maintained and the other from an unfiltered tap sample not used for drinking water; ATSDR followed up individually with the first homeowner to recommend filter replacement. The findings suggest that past drinking water contamination may be the source of PFAS in the serum, rather than current drinking water levels.

Finding 8. Patterns and levels of PFAS in dust measured in households participating in the ATSDR-led EAs are comparable to those reported in selected other U.S. studies.

No nationally representative comparison values are available, but geometric mean and median concentrations for PFAS measured in dust collected in the subset of participating households (n=117) were within the range of levels reported in a few published studies of other U.S. communities (with or without known PFAS contamination). Eighteen PFAS were detected with a frequency greater than 60%. Of the PFAS measured in both household dust and blood, PFNA and PFDA were statistically correlated with the same PFAS measured in participants' blood.

Limitations

There are several limitations associated with this assessment.

- Although similar data were collected for ATSDR's two pilot EAs and the eight ATSDR-led EAs, the methods are slightly different (different recruitment methods, PEATT included mix of private well and public water participants in the same community, different questions on questionnaire and different variable categories for some questions, different PFAS measured in blood, differences in inclusion of urine and environmental measurements). Combined analysis of exposure variables was therefore only possible for the eight ATSDR-led EAs. Data are compared and trends presented where possible for the two pilot sites.

- Participants in each EA community may not be fully representative of the entire community. The sampling recruitment method used for each EA was designed to measure blood PFAS concentrations that were generalizable to residents from each EA community who were exposed to contaminated drinking water. The household response rates varied from 1.6% to 18% for the ATSDR-led EAs. Participant characteristics were different than those of each area's overall population. Generally, participants were older than the corresponding EA community, and at some sites the racial and ethnic distribution of participants was different. Few children under the age of 18 participated in the EAs.
- The recruitment targets for the EAs meant that, at some sites, all households within the sampling frame were invited to participate rather than only inviting randomly selected households to participate. For all sites, identical efforts were made to recruit the selected households to participate in the EAs. For logistical reasons, a maximum of 3,000 households were invited to participate from any one of the EA communities. At four sites (WV, WA, TX, and AK), the number of households was not large enough to reach the target participation rate, even when inviting all households within the sampling frame. Because identical procedures were used to recruit participants across all sites, this difference is not expected to have a significant impact on the results.
- Recruitment and field data collection for three of the ATSDR-led EAs were conducted after the start of the COVID-19 pandemic. Recruitment activities were underway for the Orange County, NY EA in March of 2020 when the work was paused due to the pandemic. All field activities for the AK, CO, and NY (Orange County) EAs were also conducted during the pandemic. It is possible that the participation rates for these sites would have been higher if work had been completed prior to the pandemic.
- Measurement of blood, urine, and environmental PFAS concentrations for EA participants may improve the understanding of exposure in these communities but will not provide discrete information about all sources of exposure. Additionally, identifying every potential confounding exposure is not possible. Samples were collected over a three-year period from 2018 through 2020. Although multivariate regression models explained a moderate portion of variability of participants' blood PFAS levels, other factors not identified could influence the relationships reported in this report (see "Statistical Analysis" section for details).
- These EAs were not designed to investigate health outcomes. Without additional information about exposure-response relationships, the results of the EAs cannot be used to assess current or past health problems or predict the future occurrence of disease. PFAS found in a person's blood or urine means that exposure has occurred. The presence of PFAS in blood or urine does not tell us how, where, when, or for how long a person was exposed to PFAS. Exposure to PFAS does not mean adverse health effects will result, either now or in the future.
- We know that the family of PFAS includes thousands of different chemicals. Due to analytical limitations, the EAs were only able to assess exposure to a targeted list of PFAS for which testing methods were available at the time of the EAs. We are not able to draw conclusions about exposure to PFAS for which we did not have the analytical ability to measure.
- While we know that people can be exposed to PFAS through a variety of pathways, these EAs focused specifically on exposure through drinking water. While we made some attempt to characterize PFAS in environmental media, the data were not sufficient to draw strong conclusions about exposure via other pathways (food, incidental ingestion, lactational/gestational exposure, inhalation, etc.)

- These EAs collected data from a single point in time. Therefore, our ability to comment on how exposure may have changed over time is limited. It is likely that blood concentrations of PFAS in the past were higher than those measured during the EAs.
- There are challenges in measurement of trace levels of PFBA in urine, including selectivity of the analytical instrumentation and potential for external contamination. Therefore, we advise caution when interpreting the PFBA results in urine.
- These EAs did not directly assess participants' tap water consumption prior to the reduction of PFAS in the various drinking water supplies in the EA communities.
- The dust results are exploratory and should be interpreted with caution. They are based on a limited set of samples, and in some cases those samples are based on a small sample mass.
- The analytical method used to measure PFAS in drinking water samples collected as part of the EAs has detection limits of 2–5 ppt for individual PFAS. While these detection limits are approaching the lowest achievable detection limits with current technology, they are higher than EPA's interim health advisory levels for PFOA and PFOS that were released in 2022 (after completion of all EA sampling activities). In this report, we retain comparisons to EPA's 2016 health advisory which was in place at the time of sample collection as well as comparisons to ATSDR's EMEGsfor PFAS in drinking water.

Recommendations

Although the exposure contribution from PFAS in drinking water in all EA communities has been reduced to levels below EPA's 2016 health advisory, there are actions stakeholders and community members can take to further reduce exposures to PFAS and protect public health.

Recommendations for water providers:

1. Based on the PFAS drinking water test results from sites with public water supplies, ATSDR does not recommend an alternate source of drinking water at this time. However, operators of affected public water systems should continue to monitor concentrations of PFAS in drinking water delivered to EA communities and appropriately maintain treatment systems to ensure that concentrations of PFAS remain below existing or new Federal and state guidelines for specific PFAS in drinking water. Results of PFAS monitoring should be shared with community members through appropriate communication channels.
2. The Air Force is encouraged to continue providing bottled water and/or water filtration systems for households with private wells with PFAS concentrations above relevant state or Federal guidelines unless a different alternative source of drinking water that meets all guidelines has been provided. Testing should continue to be made available for private wells for PFAS if new data indicate they may be impacted by PFAS-containing groundwater. Households with private wells that receive bottled water and/or have water filtration systems installed specifically to treat water to remove PFAS should continue to use these alternative sources of water.

Recommendations for residents in affected areas:

1. Become familiar with Consumer Confidence Reports for information on each system's water quality.

2. Private well owners living in areas affected by PFAS should consider having their wells tested for PFAS if testing has not been conducted before. To learn more about testing wells for PFAS in your community, visit the resources listed in EA-specific reports.
3. Based on test results, consider installing a home water treatment system to further lower levels of PFAS in drinking water. The global public health organization NSF International has developed a test method to verify a water filter's ability to reduce PFOA and PFOS to below the health advisory levels set by the EPA or individual states. NSF International-approved devices can be found at: https://info.nsf.org/Certified/DWTU/. Click on "reduction devices" at the bottom of the page for PFOA and PFOS. Any treatment systems installed should be operated and maintained according to manufacturer recommendations to ensure proper operation and removal of PFAS from water.
4. Nursing mothers should continue breastfeeding. Based on current science, the known benefits of breastfeeding outweigh the potential risks for infants exposed to PFAS in breast milk.
5. When possible, eliminate or decrease potential exposure to PFAS in consumer products such as stain-resistant products and food packaging materials. To learn more visit: https://www.fda.gov/food/chemical-contaminants-food/questions-and-answers-pfas-food
6. Pay attention to advisories about food consumption, such as local fish advisories.
7. Discuss any health concerns or symptoms with your health care provider. Share results of PFAS blood testing with your health care provider and make them aware of ATSDR resources for clinicians (https://www.atsdr.cdc.gov/pfas/resources/info-for-health-professionals.html). Follow the advice of your health care provider and the recommendations for checkups, vaccinations, prenatal care, and health screening tests.
8. Follow the advice of your child's health care provider and the recommendations for well child checkups, vaccinations, and recommended health screening tests. Consult https://health.gov/myhealthfinder to help identify those vaccinations and tests. Pregnant women should follow their healthcare provider's recommendations for prenatal care.
9. For additional information about environmental exposures and children's health, contact the Pediatric Environmental Health Specialty Units, a nationwide network of experts in reproductive and children's environmental health (https://www.pehsu.net/).

Recommendations for future work/action:

1. Federal and state regulatory agencies can consider the EA findings about PFAS serum levels relative to maximum measured historical concentrations of PFAS in drinking water for policy development.
2. ATSDR recommends expanded monitoring for PFAS in drinking water in communities beyond those included in the EAs to improve our ability to identify and respond to communities affected by PFAS. The EAs have clearly shown that communities with exposure to PFAS through their drinking water have elevated blood concentrations of some PFAS when compared to the national population. At the EA sites, the PFAS concentrations in water were known and actions were taken to reduce exposure. There may be other unidentified communities with similar levels of exposure because monitoring data for PFAS in drinking water are not routinely available. EPA's Unregulated Contaminant Monitoring Rule 5 will require sample collection from some public water systems for 29 PFAS between 2023 and 2025. . This information will be helpful as a snapshot of potential contamination at a point in time, but regular monitoring for

PFAS in drinking water would allow public health authorities to take swift action in the event PFAS were detected in a water supply.

3. ATSDR will continue to share information about ongoing research related to the potential for health effects following PFAS exposure (such as the Multi-Site Study) with EA communities. ATSDR will keep the EA communities updated with any changes to ATSDR's clinician guidelines for PFAS. Understanding the relationship between PFAS exposure and health outcomes will allow communities and governmental agencies to make better decisions about how to protect public health. The information from the health studies can be applied to communities across the nation, including the communities where the EAs were conducted. More information about ATSDR's Multi-Site Study can be found at https://www.atsdr.cdc.gov/pfas/activities/studies/multi-site.html

For More Information

If you have questions or comments or want more information on the EAs or for other PFAS related questions, call 800-CDC-INFO or email pfas@cdc.gov. For more information on the work CDC/ATSDR is doing to address PFAS exposure, visit ATSDR's PFAS website: https://www.atsdr.cdc.gov/pfas/.

Background and Purpose

The Centers for Disease Control and Prevention (CDC) and the Agency for Toxic Substances and Disease Registry (ATSDR) conducted exposure assessments (EAs) in communities near current or former military bases that are known to have had per- and polyfluoroalkyl substances (PFAS) in their drinking water. In 2018, the National Defense Authorization Act (NDAA) authorized CDC/ATSDR to complete EAs in communities near military installations to assess PFAS drinking water exposures.

EA participants were recruited among residents living near military bases who received drinking water from a public water system or private well that had PFAS levels above state or federal guidelines. For more information and a map of the EA sites see the "Methods" section of this report. For more information about activities at each EA site, see individual reports at https://www.atsdr.cdc.gov/pfas/activities/assessments/sites.html.

The EAs involved a combination of collecting exposure history questionnaires, biological samples (blood and urine), and environmental samples (tap water and household dust). ATSDR collected biological samples and administered questionnaires at eight locations between September 2019 and October 2020. ATSDR also collected environmental samples in a subset of randomly chosen participant homes. These ATSDR-led EAs were partly informed by two pilot studies conducted in 2018 by the Pennsylvania Department of Health (PADOH) and the New York State Department of Health (NYSDOH) in coordination with the Association of State and Territorial Health Officials (ASTHO) and CDC/ATSDR. This report (1) summarizes and compares individual site results across all 10 EA sites, (2) explores the relationship between PFAS levels in past drinking water and in blood across all 10 EA sites, and (3) analyzes combined data across the eight ATSDR-led EA sites; these analyses were only possible for the ATSDR-led subset of sites due to differences in methods and data availability for the state-led pilot EA sites.

The results of the EAs

- tell us the amount of PFAS in the blood of individual participants and each EA community and how these levels compare to the general U.S. population,
- tell us the amount of PFAS in the urine of a subset of individual participants and each EA community and how these levels compare to the general U.S. population,
- provide a better understanding of environmental factors that are associated with PFAS exposure,
- provide information that may be used to stop or reduce PFAS exposure,
- produce information that public health professionals can use to help communities affected by PFAS, and
- inform future studies looking at the effect of PFAS exposure on human health.

The EAs do not look at what types of health problems are associated with exposure and are not meant to determine if PFAS levels in blood or urine are risk factors for illness now or later in life. Additionally, the EAs do not tell us exactly how or where people were exposed or when or how long PFAS exposure lasted.

ATSDR's *Exposure Assessment Protocol: Biological and Environmental Sampling of PFAS,* called the PFAS EA protocol for short [ATSDR 2019a], provides additional background, describes the criteria for selecting communities for the eight ATSDR-led EAs, and highlights the procedures ATSDR used in conducting

those EAs. For the two pilot EAs, investigators followed procedures outlined in ATSDR's *PFAS Exposures Assessment Technical Tools (PEATT)* [ATSDR 2018].

What Are PFAS?

PFAS are synthetic chemicals used in many industries and consumer products since the 1940s. They have been used in nonstick cookware; water-repellent clothing; stain-resistant fabrics and carpets; cosmetics; firefighting foams; and products that resist grease, water, and oil [Buck et al. 2011; Gluge et al. 2020; Wang et al. 2017]. Exposure to PFAS has been associated with increased cholesterol, decreased vaccine response in children, changes in liver enzymes, small decreases in infant birth weights, increased risk of high blood pressure or pre-eclampsia in pregnant women, and increased risk of kidney and testicular cancer [ATSDR 2021a].

There are thousands of different PFAS. These assessments evaluated some of the most commonly studied PFAS, which include perfluorooctanoic acid (PFOA), perfluorooctane sulfonic acid (PFOS), perfluorohexane sulfonic acid (PFHxS), perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA), perfluoroundecanoic acid (PFUnA), and N-methyl perfluorooctanesulfonamidoacetic acid (MeFOSAA). The manufacture and import of PFOA, precursor chemicals that can break down to PFOA, and related higher homologue chemicals have been mostly phased out in the United States. However, existing stocks of PFOA might still be used, and there might be PFOA in some imported articles. PFOS manufacture in the United States has not been reported to the EPA since 2002; however, there are some limited ongoing uses of PFOS. These PFAS with long perfluoroalkyl chains are no longer produced in the United States because of concerns over their high persistence, tendency to bioaccumulate, and potential risks to human health and the environment. Other countries may still manufacture and use them, but U.S. manufacturers have replaced these compounds with shorter chained PFAS or chemicals with alternative chemistries, such as GenX (HFPO-DA), which typically have shorter biological half-lives. Some of the PFAS discussed in this report, such as MeFOSAA, are considered precursors that can degrade in the environment or in people to other PFAS [ATSDR 2021a; Wang et al. 2017].

PFAS do not occur naturally, but, due to human use, they are widespread in the environment. PFAS can be released into the environment during their production, use, or disposal. PFAS have been found in air, water, soil, sediment, animals, and plants. Most PFAS (including PFOA, PFOS, PFHxS, PFNA, PFDA, and PFUnA) are either very resistant to breaking down or degrade into other PFAS that do not break down further. Certain PFAS will therefore remain in the environment indefinitely. Once PFAS enter people's bodies, some of them (including PFOA, PFOS, PFHxS, and PFNA) can remain in the body for long periods and can be measured in the blood years after exposure. Most people in the United States have been exposed to PFAS. At least one PFAS was detected in more than 99% of NHANES samples (1999–2000 survey cycle) [Calafat et al. 2007a]. Exposure can occur via contaminated drinking water for which ingestion is believed to be the primary exposure route. Studies have shown that showering, bathing, and swimming in water containing PFAS at levels seen in EA communities are not expected to be an important contributor to PFAS exposure relative to the contribution from drinking water, but the contribution from other routes of exposure has not been fully evaluated [Sunderland 2019].

ATSDR's PFAS EAs focused on communities with known exposures via contaminated drinking water. However, residents may have had additional exposures to PFAS, such as the following [Sunderland 2019]:

- eating food packaged in materials containing PFAS (e.g., popcorn bags, fast food containers, pizza boxes)
- eating fish or shellfish caught in PFAS-contaminated waters
- using consumer products such as stain-resistant carpeting and water-repellent clothing
- eating garden vegetables grown with PFAS-contaminated water or soil
- accidentally swallowing PFAS-contaminated soil
- drinking infant formula mixed with PFAS-contaminated water
- consuming breastmilk from women exposed to PFAS
- gestational exposure to PFAS
- working in industries that manufacture, process, or use products containing PFAS
- background exposure to PFAS due to their ubiquitous nature

ATSDR asked EA participants about these types of potential exposures to evaluate whether these exposures might influence PFAS levels in the EA communities.

After PFAS enter the human body, some PFAS can remain there for a long time—sometimes years. Most studies estimate a half-life of PFHxS between 4.7 and 8.5 years, although some have estimated half-lives as long as 35 years [ATSDR 2021a]. Most half-life estimates for PFOS are between 3.3 and 7.4 years, with a maximum of 27 years [ATSDR 2021a]. For PFOA, most studies estimate the half-life between 2.1 and 3.9 years with a maximum of 10.1 years [ATSDR 2021a].

The body of science about PFAS exposure and health effects is growing rapidly. Some, but not all, scientific studies have shown that exposure to certain PFAS may be linked to harmful health effects [ATSDR 2021a]. While these EAs did not examine specific health outcomes associated with PFAS exposure, EA findings might help inform future studies on how PFAS exposure affects human health.

Why the Selected Sites?

The EA sites are located near military installations with identified PFAS drinking water contamination from use of products such as aqueous film forming foam (AFFF). When selecting the EA sites, ATSDR considered the extent of PFOA and PFOS contamination in drinking water supplies, the duration over which exposure may have occurred, and the number of potentially affected residents.[4] By choosing a range of exposure scenarios, the lessons learned can be applied to communities facing similar PFAS drinking water exposures.

The following are the selected sites shown in Figure 1:

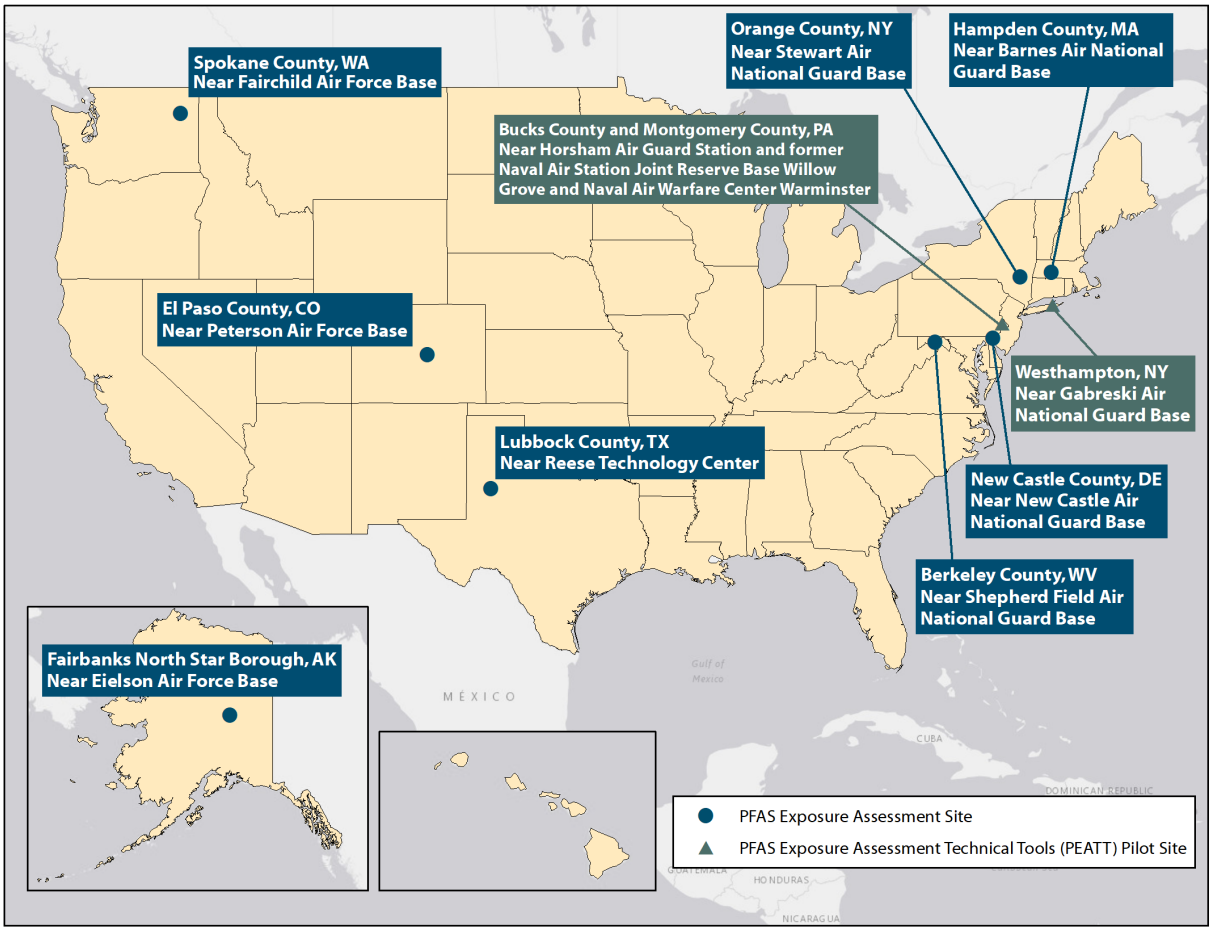
- Westhampton Beach and Quogue Area, New York (NY Pilot EA)
- Montgomery and Bucks Counties, Pennsylvania (PA Pilot EA)
- Hampden County, Massachusetts (Westfield EA)

Throughout this report, we reference the two pilot EAs ("pilot EAs") and the "ATSDR-led EAs" that followed. The pilot EAs were conducted in 2018 using methods described in PEATT [ATSDR 2018]. The ATSDR-led EAs were conducted in 2019-2020 following ATSDR's EA Protocol [ATSDR 2019a]. Because the pilot EAs and ATSDR-led EAs were conducted independently using slightly different methods, distinctions are made throughout this report when referring to the pilot EAs (2 sites), the ATSDR-led EAs (8 sites), or both (10 sites).

[4] PFHxS data were not available for all sites evaluated so were not considered in the site selection process even though drinking water contaminated by AFFF often reported higher concentrations of PFHxS than PFOA or PFOS.

- Berkeley County, West Virginia (Berkeley County EA)
- New Castle County, Delaware (New Castle County EA)
- Spokane County, Washington (Airway Heights EA)
- Lubbock County, Texas (Lubbock County EA)
- Fairbanks North Star Borough, Alaska (Moose Creek EA)
- El Paso County, Colorado (Security-Widefield EA)
- Orange County, New York (Orange County EA)

Figure 1. Map of PFAS EA site locations



PFAS and precursors that degrade to other PFAS measured in these EAs were used in historical AFFF formulations. Two types of PFAS-containing AFFF were manufactured before 2016 [ITRC 2020]. Both formulations contained PFAS or PFAS precursors, the use of which resulted in the release of PFOS, PFHxS, PFOA, PFHxA, and many other PFAS into the environment. Possibly as early as the 1970s, these military bases used AFFF containing PFAS for their firefighter training and responses to fire events. Over time, the PFAS from the AFFF moved off site in groundwater and contaminated nearby drinking water sources.

Table 1 describes the characteristics of each EA site. PFAS were first detected in each community water supply from 2009 to 2017, although we do not know when they first entered water sources in each EA community.

At seven sites, community members received drinking water from public water systems, at two sites community members relied on private wells, and at one site community members received drinking water from a combination of a public water system and affected private wells. For public water system sites, testing was initially conducted by the affected public water system, most often to fulfill requirements of the U.S. Environmental Protection Agency's (EPA's) Third Unregulated Contaminant Monitoring Rule (UCMR 3) [EPA 2017]. In one instance, a water system tested for PFAS in 2009 prior to inclusion of PFAS in UCMR 3. For private well sites, testing was conducted by the Air Force or a state health agency.

At all sites, the sum of PFOA and PFOS levels in historic drinking water samples were above the EPA's 2016 health advisory (HA) of 70 parts per trillion (ppt). The highest reported sampling results detected in drinking water across sites were 2,900 ppt for PFOA, 3,100 ppt for PFOS, and 1,500 for PFHxS. These results were detected at three separate sites. The maximum levels for PFOA and PFHxS were detected in public water systems. The maximum level for PFOS was detected from a private well.

At all sites, exposures were mitigated through various corrective actions, which included inactivating contaminated water sources or installing filtration and treatment systems. The final mitigation actions were taken between 2014 and 2019. The information available to ATSDR indicates that all households in affected areas across the EA sites had a drinking water supply that met or was below federal and state guidelines for PFAS during the time of sample collection, with the possible exception of households with private wells who have declined water testing and/or water treatment systems.

Table 1. Characteristics of EA sites

Site	Contaminated Drinking Water Supply	Date PFAS First Detected in Drinking Water	Maximum PFAS in Drinking Water (ppt)			Final Mitigation Date*	EA Data Collection Dates (Blood Draw)
			PFHxS	PFOS	PFOA		
NY EA Pilot	Suffolk County Water Authority	2013/2014	149	2,440	230	2016	April–October, 2018
PA EA Pilot	Horsham Water and Sewer Authority (HWSA); Warrington Township Water and Sewer Department (WTWSD); Warminster Municipal Authority (WMA); and Private Wells	HWSA: June 2014 WTWSD: October 2014 WMA: November 2013 Private Wells: September 2014	1,323	2,615	655	July 2016	May–September, 2018
Westfield, MA	City of Westfield Municipal Water Supply	February 2013	170	160	43	January 20, 2016	September 4–17, 2019
Berkeley County, WV	City of Martinsburg and Berkeley County Water Supply Systems	February 2014	105	114	46	May 19, 2016	September 24–October 7, 2019
New Castle County, DE	Artesian Water; Municipal Services Commission (MSC) Water Systems	Artesian: July 2013; MSC: September 2009	680; 1,400	1,800; 2,300	140; 440	July 18, 2016; August 5, 2014	October 16–27, 2019
Airway Heights, WA	Airway Heights Water Department	May 2017	1,500	1,200	320	June 8, 2017	November 4–14, 2019
Lubbock County, TX	Private Wells	September 2017	1,450	998	2,900	September 20, 2019†	February 26–March 4, 2020
Moose Creek, AK	Private Wells	May 2015	1,395‡	3,100	250	December 28, 2017†	August 18–25, 2020
Security-Widefield, CO	Security Water District (SWD); Widefield Water and Sanitation District (WWSD); and Security Mobile Home Park (SMHP)	SWD: January 2014 WWSD: November 2013 SMHP: February 2016	590	210	90	November 10, 2016	September 15–28, 2020
Orange County, NY	City of Newburgh Public Drinking Water Supply	December 2013	70	170	27	May 2, 2016	October 23–29, 2020

*The date shown was used to determine potential EA participant eligibility.

†For private wells sites, actual mitigation dates varied depending on when the Air Force detected PFAS levels above EPA HAs. The last known mitigation date is shown.

‡PFHxS levels from private wells were not available. This value is estimated by applying a PFHxS to PFOS ratio of 0.45 based on samples collected during the EA.

Methods

ATSDR's PFAS EA protocol details the approaches used to recruit participants, collect samples, administer exposure history questionnaires, and evaluate data for the ATSDR-led EAs. This protocol built on the work of the two pilot EAs conducted by PADOH and NYSDOH.

This section briefly describes the methods for the two pilot EAs and how the EA protocol methods were applied to the other eight EA sites. Because the pilot and ATSDR-led EAs were conducted independently using slightly different methods (e.g., different exposure questionnaires), the pilot EA data cannot be combined with the data from the ATSDR-led EAs for statistical analyses. The Results and Discussion section compares blood levels across all 10 EA sites, distinguishes where pilot EA findings are presented and where they are not, and presents more qualitative comparisons of pilot EA and ATSDR-led EA findings where possible.

Table 2, at the end of this section, provides more details on the number of participants enrolled and the final number of samples collected across all EA sites. Table 3 lists the PFAS measured in the EA's biological and environmental samples.

Pilot EAs

Through a cooperative agreement with ASTHO, funding was provided to the PADOH and NYSDOH to conduct pilot biomonitoring studies using the ATSDR PEATT [ATSDR 2018]. Subject matter experts from ATSDR and CDC's National Center for Environmental Health developed the PEATT to help state, local, tribal, and territorial health departments conduct PFAS biomonitoring activities, with the assumption that drinking water is the primary source of PFAS exposure. The PEATT includes a protocol for statistically based sampling, risk communication materials, questionnaires, and EPA's water sampling protocol to help characterize PFAS exposure in communities. A goal of the two pilot EAs was to measure and evaluate community exposures to PFAS in drinking water. Another goal was to identify possible improvements of the PEATT for future PFAS biomonitoring. The methods and results of the two pilot EAs informed the protocol for the ATSDR-led EAs.

In 2018, PADOH conducted a pilot EA in Montgomery and Bucks Counties, PA, near the Horsham Air Guard Station and former Naval Air Warfare Center Warminster. The affected area had an estimated population of 84,184 people and 32,595 households. PADOH collected blood samples from 235 community members (209 adults and 26 children) from 118 randomly selected households and analyzed the blood samples for 11 PFAS. PADOH released a final report of findings in April 2019. In the summer of 2019, CDC and ASTHO expanded the PA PEATT pilot EA to include further testing of the original EA participants and potential environmental exposures in the community. The follow-up testing included PFAS analyses in urine collected from 186 participants and analyses in environmental samples (tap water and dust) collected from 10% of households (n=14). The results of these urine and environmental samples are not included in this report. Additional information and details on PADOH's PFAS activities, including the pilot EA, can be found at:
https://www.health.pa.gov/topics/envirohealth/Pages/PFAS.aspx

In 2018, NYSDOH conducted a pilot EA in Westhampton Beach and Quogue Area near the Gabreski Air National Guard Base. The affected area had an estimated 2,125 households, 30% of which were expected to be year-round residences. NYSDOH collected blood samples from 161 community members from 78 randomly selected households and analyzed the blood samples for 11 PFAS. NYSDOH released a

final report in June 2019. Additional information and details on NYSDOH PFAS activities, including the pilot EA, can be found at:
https://www.health.ny.gov/environmental/investigations/drinkingwaterresponse/

Recruitment for the ATSDR-led EAs

The NDAA authorized CDC/ATSDR to evaluate PFAS exposure in communities near current or former military bases that are known to have had PFAS in their drinking water. ATSDR selected eight sites across the country to study. These EAs built upon the CDC/ATSDR-funded PFAS EA pilot work mentioned above. Unless otherwise indicated, the following text about recruitment applies to these ATSDR-led EAs.

Sampling Frames

Each EA targeted a specific geographic area, called the sampling frame or sampling area—an area where known or expected PFAS exposure occurred. For the six communities served by public water supplies, the sampling frame was defined by service boundaries of an entire public water system, a portion of a public water system's service area, or portions of multiple public water systems that had PFAS levels above state or federal guidelines. For the two communities served by private wells, the sampling frame was defined by the areas with impacted private drinking water wells. Based on a review of land parcel data from each community, ATSDR identified 38,581 households across the eight sampling frames. Additional information on each sampling frame is described in site-specific reports [ATSDR 2021b; ASTDR 2022a-g]. How people living within the sampling frames were chosen to participate in the EAs is described in the subsections that follow.

Participant Eligibility

Residents from each sampling frame who met the following criteria were eligible to participate in the ATSDR-led EAs:

- Lived within the sampling frame for at least one year before PFAS exposures were mitigated at the site.
- Were at least 3 years old at the time of recruitment. This age criterion was used because national reference values are not available for children under the age of three.
- Did not have bleeding disorders and were not anemic, unless they confirmed with their doctor the ability to safely provide a blood sample.

People potentially exposed to PFAS occupationally, such as firefighters, active-duty military, and veterans, were able to participate if their households were selected to participate. Participants did not receive incentives and paid no costs to participate.

Participant Recruitment

ATSDR's recruitment strategy was aimed at achieving the EA PFAS protocol recruitment target of 395 participants at each site. ATSDR invited a maximum of 3,000 households per EA community. This cap was based on the response rates being observed within the EA communities. If the sampling frame size was smaller than 3,000 households, all households were invited (Berkeley County, WV; Airway Heights, WA; Lubbock County, TX; Moose Creek, AK). For sampling frames larger than 3,000 (New Castle County, DE; Security-Widefield, CO; Orange County, NY), 3,000 households were randomly selected from the total household pool. For the Westfield (MA) EA, recruitment targets were reached with the random selection of 1,349 households.

Measuring PFAS in the blood of people from randomly selected households allowed ATSDR to estimate exposure to PFAS from public drinking water for the entire community (the sampling frame) in the affected area, even those who were not tested. At some sites, all households were recruited to participate to meet recruitment goals. In those cases, everyone had an equal chance to participate. The overall design was aimed at collecting data that were generalizable to individual sampling frames.

Results from randomly selected participants can provide information about community-level exposures when it is not possible to invite the entire sampling frame. Every household (or cluster) within a sampling frame or random pool had an equal chance of being selected, and all members of selected households who met eligibility criteria were invited to participate. This means that a single household may have multiple participants.

Recruitment was done through a combination of mailings, phone calls, and door-to-door visits to households. Each household for which ATSDR had a phone number received a minimum of three recruitment call attempts. In each attempt, ATSDR called all working phone numbers (cell phone and landline) associated with a household. For calls that went to voicemail, ATSDR staff left messages encouraging residents to call back to schedule appointments. Door-to-door visits occurred after each household had received an initial outreach letter and/or at least one recruitment call attempt.

After recruitment, 2,294 residents from 1,198 households across the EA sites scheduled appointments for biological sampling and completing a questionnaire. Not all enrollees ended up participating in data collection activities.

Data Collection and Analysis for the ATSDR-led EAs

Each EA involved collecting three types of data: questionnaires, biological samples (blood and urine), and environmental samples (tap water and household dust). The ATSDR project team collected biological samples at a location within or near each sampling frame and administered questionnaires either in person or by telephone between September 2019 and October 2020. ATSDR also collected environmental samples in a subset of randomly chosen participant homes.[5] All data met the stringent quality control requirements for sample collection and analysis.

Before any data collection, ATSDR obtained written consent from the participants. The purpose of the consent process was to ensure participants were fully aware of the purpose of the EA, sample collection

[5] ATSDR collected environmental samples at the same time as biological data collection with the exception of Orange County, NY. Because of growing evidence of a COVID-19 surge, ATSDR delayed in-home tap water and dust until June 2021.

procedures, benefits and risks of participating, and privacy protections. Copies of consent forms are included in the PFAS EA Protocol.

ATSDR project staff handled all data collected in accordance with the *Standard Operating Procedures of PFAS Exposure Assessment Data Management* [ATSDR 2019b]. These procedures have very strict requirements for handling any personally identifiable information. ATSDR project staff protected this information to the extent required by federal and state law. All signed consent forms were mailed to and are securely archived at ATSDR headquarters. Questionnaire data were collected using dedicated encrypted laptops with no internet access, and these data were transferred at program completion to ATSDR's secure data network. All information provided by participants was kept confidential, and no personally identifiable information appears in any of ATSDR's public reports for this site.

Biological Sampling and Questionnaire Administration

Of the 2,294 residents who scheduled EA data collection appointments, 2,046 (89%) participated in the EAs. ATSDR administered exposure history questionnaires to these individuals: 1,829 for adults 18 and older, and 217 for children between the ages of 3 and 17. ATSDR used one questionnaire for adults and another for children. Both addressed topics relevant to PFAS exposure, such as residential and work histories, drinking water habits, and use of PFAS-containing consumer products.

Phlebotomists collected blood samples from 2,033 participants. ATSDR processed the blood samples in the field, aliquoting the serum portion of the blood.

After the sampling was complete and upon further review of each participant's residential history, ATSDR determined that 45 participants had not lived in the EA sampling frames for at least one full year before the individual site's mitigation date, and therefore were not eligible for the assessment. Questionnaire and biological data for these participants were excluded from the data evaluation, but ATSDR sent them their individual results. This means that a total of 1,988 blood samples (1,791 adults and 197 children) were considered in the community statistics. These samples were collected from participants residing in 1,094 unique households. This represents an overall household participation rate of 6.5% (i.e., 6.5% of the 16,768 recruited households had at least one person participate in the EA). See Table 2.

Urine samples were collected from 2,036 participants (1,828 adults and 208 children). Per the EA protocol, 10% (204) of the urine samples were randomly selected for initial analysis. These 204 samples were collected from participants (189 adults and 15 children) who resided in 192 unique households.

CDC's National Center for Environmental Health laboratory analyzed the serum portion of blood and urine samples for the suite of PFAS measured in the 2015–2016 National Health and Nutrition Examination Survey (NHANES) [CDC 2019]. As part of NHANES, CDC takes biological samples and tests them for chemicals, including PFAS, from a representative sample of 5,000 people across the country during each two-year cycle. All laboratory analyses followed established procedures for quality assurance and control according to the Center's methodology.

During the consent process, participants were given the option to allow ATSDR to store biological samples for potential future PFAS analysis. Blood and urine samples from participants who provided this consent are being stored frozen at CDC for potential future analysis.

Environmental Sampling

ATSDR attempted to recruit approximately 10% of participating households participating in the eight ATSDR-led EAs for environmental sampling. In total, ATSDR invited 254 households from which 126 environmental sampling appointments were scheduled.

ATSDR collected tap water and dust samples from 117 households that kept their environmental sampling appointments. Nine households were unavailable to complete their scheduled environmental sampling appointment or were not eligible. At each participating household, ATSDR collected a drinking water sample from the kitchen tap. If point-of-use filtration was in place, ATSDR project staff attempted to collect a sample before and after filtration. Tap water samples were collected and analyzed in accordance with EPA's Method 537.1: Determination of Selected Per- and Polyfluorinated Alkyl Substances in Drinking Water by Solid Phase Extraction and Liquid Chromatography/Tandem Mass Spectrometry [Shoemaker and Tettenhorst 2018].

Project staff also collected a composite dust sample from the floor at a minimum of three locations inside each selected home: the primary living space as identified by the homeowner (e.g., living room, family room, television room), the kitchen, and the bedroom in which participants reported spending the most time. Dust collection was intended to generate more information about the contribution of non-drinking-water exposures to overall PFAS exposure. Participants were instructed not to vacuum carpeting or sweep floors for five days prior to the scheduled visit. Adapting methods described in Scher et al. [2018], ATSDR collected dust samples using a high-volume air sampler connected to an open-faced 37-millimeter filter cassette with an 0.8 micron filter. A wooden 2 square foot (ft^2) sampling template was used to mark off each sampling area. ATSDR project staff attempted to collect at least 1 gram of dust in the open-faced cassettes from each home by vacuuming the same 2 ft^2 surface at least four times with the cassette (vertically, horizontally, and in circles). Samples were taken preferentially from mats, carpets, and area rugs. Household dust samples were analyzed in accordance with SGS AXYS Method MLA-110 (revision 01, version 06), *Analytical Procedure for the Analysis of Per- and Polyfluoroalkyl Substances (PFAS) in Aqueous Samples, Solids and Solvent Extracts by LC-MS/MS* [SGS AXYS 2019].

The environmental samples collected during the EA were consumed in the analytical process and are not available for potential future analysis.

Statistical Analysis

The EA Protocol describes the statistical methods used for the EAs. Briefly, the data objectives of each ATSDR-led EA were to (1) estimate geometric mean concentrations of PFAS in the sampling frame population (with a precision target of at least 15% and a 5% level of significance for PFOS), (2) compare community level data to national levels, and (3) explore relationships between questionnaire data and measured biological and environmental data.

ATSDR processed the PFAS sampling results in two ways before performing statistical analyses:

- First, ATSDR substituted all non-detect observations with a value equal to the limit of detection (LOD) divided by the square root of 2. (A non-detect result means the sample did not contain enough PFAS to be reliably measured by this project's highly sensitive laboratory methods.) This substitution method is consistent with that applied in CDC's NHANES. Note that each site report appendix provides the results of a sensitivity analysis exploring alternate substitution approaches.
- Second, ATSDR calculated the total PFOA and total PFOS concentrations measured in each blood and urine sample. The laboratory reports two different measurements for PFOA and PFOS. For PFOA, the laboratory reports the amount of branched PFOA (Sb-PFOA) measured in the sample separate from the amount of linear PFOA (n-PFOA) in the same sample. ATSDR summed these values and performed statistical analyses using total PFOA results. Similarly, ATSDR calculated total PFOS by summing the linear PFOS (n-PFOS) and branched PFOS (Sm-PFOS) concentrations. These same summation methods are applied to NHANES data.

For blood and urine, ATSDR first calculated summary statistics for each PFAS (i.e., frequency of detection, maximum detected concentration, geometric mean, and 95% confidence intervals around the geometric mean). Percentiles (25^{th}, 50^{th} [median], 75^{th}, 90^{th}, and 95^{th}) were calculated and presented in EA-specific reports. The protocol specified that geometric means would be

Statistical Terms

Geometric mean: The geometric mean is a type of average and provides an estimate of the central point of a set of numbers. It is often used for environmental data that exhibit a skewed distribution (e.g., a data set with several values that are much higher than the rest of the results). The geometric mean is less influenced by high values than an arithmetic mean.

Percentiles (25th, 50th, 75th, 90th, 95th): A percentile provides additional information about the distribution of a data set and represents the value below which a certain percentage of the data fall. For example, a 95th percentile of 25 micrograms per liter (µg/L) indicates that 95% of results fall below this concentration.

Confidence intervals: A confidence interval provides information about the reliability of a statistic. In this EA, ATSDR estimated geometric means for the PFAS blood levels measured among EA participants. The 95% confidence interval around the geometric mean represents the range within which the true population mean is expected to lie. More specifically, if we hypothetically repeated the assessment 100 times, 95 times out of 100 the mean of the sampling frame population would fall within this range.

Precision: Precision provides information on the reproducibility of a study and is associated with sample size. The larger the sample size the higher the precision. In the context of this EA, precision was estimated based on the width of confidence intervals around the geometric mean. A wide confidence interval indicates low precision while a narrow confidence interval suggests high precision.

calculated if >=60% of samples had detections. Geometric means were calculated as the measures of central tendency because of the lognormal distribution of blood and urine measurements. Note that many of the statistics could not be calculated for urine due to the low detection frequency.

> A **p-value** helps determine the significance of the results of a statistical test, such as the difference between two means. The lower the p-value the more likely the observed difference is not due chance alone. In this report, a p-value of less than 0.05 ($p<0.05$) is described as *statistically significant*.

ATSDR conducted all statistical analyses in SAS (release 9.4, SAS Institute, Cary, NC) using complex survey procedures (e.g., SURVEYMEANS, SURVEYREG). To do this, ATSDR assigned household IDs to all participants and calculated summary statistics while accounting for clustering at the household level. Each site-specific report provides more information on clustering and further details on the statistical methods used for each EA including how results from each EA compared to the assumptions used to estimate the target sample size of 395 participants.

Data Analysis Across All 10 EA Sites

ATSDR compared PFAS blood levels measured in each EA to national levels following similar procedures as in each ATSDR-led site specific report. To control for differences in the age distribution, geometric means for each EA were adjusted to the age distribution of the U.S. population during NHANES 2015–2016 (i.e., for the EA sample population 12 years and older). ATSDR then statistically compared the age-adjusted geometric means for all 10 EAs to NHANES 2015–2016 (as reported in individual EA reports) and to the more recently released NHANES 2017–2018 PFAS data.

ATSDR also explored the relationship between PFAS blood levels at all 10 EA sites and 1) maximum levels of PFAS in drinking water supplies and 2) the time between when PFAS exposures ended and when biological samples were collected.

Data Analysis Across ATSDR-led EA Sites

ATSDR used information gathered in the EA exposure questionnaire to understand and quantify how demographic data and other exposure characteristics relate to PFAS measurements in blood. These analyses were only possible for the ATSDR-led subset of sites due to differences in methods and data availability for the state-led pilot EA sites. Individual reports were published detailing the findings from each of the EA communities.

ATSDR combined the data from the eight ATSDR-led sites and conducted univariate and multivariate statistical analyses. ATSDR relied on self-reported information, such as age, race/ethnicity, sex, length of residency in the sampling frame, tap water and food consumption patterns, and work/school history. All numerical responses were treated as continuous variables. In some cases, categorical variables were collapsed when there were too few responses (<10) in a given category.

Univariate statistics were used to evaluate one variable at a time, mostly as a tool to examine the data broadly and find patterns within the data. Multivariate regression modeling was used to account for multiple variables simultaneously to control for potential confounding factors. While this same type of modeling was conducted for individual sites, combining the data across sites allows for a more robust analysis because of a larger number of total participants. It allows ATSDR to further evaluate the strength of the associations between measured blood levels and various exposure variables. However, the overarching goal of the EAs was to examine the strength of the association of PFAS levels in drinking

water measured across sites while controlling for other exposure variables as potential confounders. Since PFAS drinking water levels were generally similar within a given EA site, this analysis was only possible when combining the data across ATSDR-led EAs.

For all univariate and multivariate analyses, ATSDR modeled log transformed (logarithm base 10 or $\log_{10}$) blood PFAS concentrations. When conducting statistical analyses on data combined from multiples EAs, ATSDR treated each EA site as a stratum to control for differences in variability across sites, and applied weights to control for differences in sampling rates across sites. ATSDR did not adjust for multiple comparisons. The variables presented in Appendix C were considered for univariate and multivariate regressions. When developing multivariate models, a backwards stepwise approach was used. In instances where variables were very similar or correlated with one another, only one variable that represented the strongest predictor (based on univariate analyses) or that was determined by expert judgement to be the most relevant to exposure was used during model selection. A variable representing "site" was not included in multivariate modeling because drinking water levels tended to primarily vary by site. To explore sex-specific associations (e.g., having biological children [yes/no], having breastfed children [yes/no], duration of breastfeeding), ATSDR also evaluated multivariate models separately for males and females.

Appendix B provides more detail on the SAS survey procedures used for the statistical analyses.

For urine, ATSDR compared community-level EA data to national-level data from the 2013–2014 NHANES compiled by Calafat et al. [2019], the only nationally representative data available for PFAS in urine. ATSDR relied on two sample t-tests for these comparisons, using a p-value of less than 0.05 to identify statistically significant differences. ATSDR did not conduct detailed statistical analyses for urine data because of low frequencies of detection. ATSDR analyzed a subset of urine samples and found that, for all PFAS, the frequency of detection was <60%. Since no geometric mean PFAS concentration exceeded the NHANES 95th percentile, ATSDR did not analyze the rest of the samples, in accordance with the EA protocol.

For tap water data, ATSDR compared PFAS levels measured with and without filtration to EPA's 2016 HA value (70 ppt for PFOA and PFOS combined) for PFAS in drinking water, ATSDR's environmental media evaluation guides (EMEGs) for PFAS in drinking water, and to state health guidelines or standards. For dust, ATSDR calculated summary statistics and discussed results in context of those published in selected peer-reviewed literature. ATSDR also evaluated correlations between PFAS levels measured in household dust and blood collected from participants residing in homes where dust samples were collected.

Table 2. Summary of recruitment and data collection efforts across all 10 EA sites

Recruitment	PA Pilot	NY Pilot	Westfield, MA	Berkeley County, WV	New Castle County, DE	Airway Heights, WA	Lubbock County, TX	Moose Creek, AK*	Security-Widefield, CO*	Orange County, NY*
Households invited	600	800	1,349	2,922	3,000	2,516	701	317	3,000	3,000
Participation rate	20%	NA	18%	5.6%	4.5%	6.7%	14%	15%	6.3%	1.6%
Biological sampling										
Individuals enrolled	235	161	514	334	244	391	260	93	384	74
Households enrolled	119	78	260	186	151	194	106	46	200	55
Environmental sampling										
Households invited	--	NA	40	33	42	39	30	30	30	10
Households enrolled	14		19	19	14	20	14	13	20	7
Completed questionnaires	235	161	472	285	216	349	219	89	355	61
Adults	209	143	415	254	204	302	195	79	321	59
Children	26	18	57	31	12	47	24	10	34	2
Blood samples included in community statistics	235	161	459	275	214	333	214	88	346	59
Adults	209	143	410	247	203	286	190	79	318	58
Children	26	18	49	28	11	47	24	9	28	1
Households	118	--	247	165	134	168	96	48	188	48
Urine samples										
Collected			471	283	216	346	219	86	354	61
Adults	209	NA	415	254	204	301	195	76	324	59
Children	26		56	29	12	45	24	10	30	2
Analyzed			47	27	22	34	22	9	36	7
Adults	24		46	24	18	32	20	8	34	7
Children	(total)		1	3	4	2	2	1	2	0
Households			44	23	20	33	20	9	36	7
Dust samples collected and analyzed	14	NA	17	19	13	19	12	13	18	6
Tap water samples collected and analyzed			24	27	20	26	16	19	34	11
Filtered	14	NA	8	10	7	7	10	11	17	5
Unfiltered	(total)		16	17	13	19	6	8	17	6

*EA conducted during the COVID-19 pandemic.

Table 3. List of PFAS measured for in blood, urine, tap water, and dust*

PFAS Abbreviation	PFAS Chemical Name	Measured in Blood?	Measured in Urine?	Measured in Water?	Measured in Dust?
PFBS	perfluorobutane sulfonic acid	P	✓ P	✓ P	✓ P
PFPeS	perfluoropentane sulfonic acid				✓ P
PFHxS	perfluorohexane sulfonic acid	✓ P	✓ P	✓ P	✓ P
PFHpS	perfluoroheptane sulfonic acid				✓ P
PFOS	perfluorooctane sulfonic acid	✓ P	✓	✓ P	✓ P
n-PFOS	sodium perfluoro-1-octanesulfonate	✓	✓ P		
Sm-PFOS	mixture of sodium perfluoro-5-methylheptane sulfonate isomers	✓	✓ P		
PFNS	perfluorononane sulfonic acid				✓ P
PFDS	perfluorodecane sulfonic acid				✓ P
PFDoS	perfluorododecanesulfonate				✓ P
PFBA	perfluorobutanoic acid		✓ P		✓ P
PFPeA	perfluoropentanoic acid		✓ P		✓ P
PFHxA	perfluorohexanoic acid		✓ P	✓ P	✓ P
PFHpA	perfluoroheptanoic acid	P	✓ P	✓ P	✓ P
PFOA	perfluorooctanoic acid	✓ P	✓	✓ P	✓ P
n-PFOA	ammonium perfluorooctanoate	✓	✓ P		
Sb-PFOA	mixture of perfluoro-5-methylheptanoic acid isomers	✓	✓ P		
PFNA	perfluorononanoic acid	✓ P	✓ P	✓ P	✓ P
PFDA	perfluorodecanoic acid	✓ P	✓ P	✓ P	✓ P
PFUnA	perfluoroundecanoic acid	✓ P	✓ P	✓ P	✓ P
PFDoA	perfluorododecanoic acid	P		✓ P	✓ P
PFTrA	perfluorotridecanoic acid			✓ P	✓ P
PFTA	perfluorotetradecanoic acid			✓ P	✓ P
PFOSA	perfluorooctanesulfonamide	P			✓ P
N-MeFOSA	N-methylperfluorooctanesulfonamide				✓ P
MeFOSAA	N-methyl perfluorooctanesulfonamidoacetic acid	✓ P		✓ P	✓ P
N-MeFOSE	N-methylperfluorooctanesulfonamidoethanol				✓ P
N-EtFOSA	N-ethylperfluorooctanesulfonamide				✓ P
N-EtFOSAA	N-ethyl perfluorooctanesulfonamidoacetic acid			✓ P	✓ P
N-EtFOSE	N-ethylperfluorooctanesulfonamidoethanol				✓ P
FtS 4:2	fluorotelomer sulfonic acid 4:2				✓ P
FtS 6:2	fluorotelomer sulfonic acid 6:2				✓ P
FtS 8:2	fluorotelomer sulfonic acid 8:2				✓ P
HFPO-DA (GenX)	hexafluoropropylene oxide dimer acid		✓ P	✓	✓ P
DONA	4,8-dioxa-3H-perfluorononanoic acid		✓ P	✓	✓ P
9Cl-PF3ONS	9-chlorohexadecafluoro-3-oxanone-1-sulfonic acid		✓ P	✓	✓ P
11Cl-PF3OUdS	11-chloroeicosafluoro-3-oxaundecane-1-sulfonic acid			✓	✓ P

P=two pilot EAs (urine and water samples collected at PA pilot site only); ✓= eight ATSDR-led EAs

*Urine and environmental sampling results from the pilot EA are not included in the statistical analyses in this report.

Results and Discussion

This section presents the overall findings of the EAs. First the general characteristics of the participants in the ATSDR-led and pilot EAs are highlighted. For the ATSDR-led sites, a brief overview is presented of how the demographic characteristics of those participating in the EAs compared to the overall population in the sampling frame.

Next, PFAS levels measured in blood across all 10 EA sites are compared to national levels. The results of a pooled analysis of blood levels and relationships to past drinking water exposure patterns (PFAS levels in drinking water and time since mitigation) are then presented across all 10 EA sites. Correlations among the PFAS measured in blood of all EA participants are then presented.

The remainder of the Results section focuses on the statistical modeling and trend analyses for the combined data across the ATSDR-led EAs. First, analysis results of pooled exposure history questionnaire data are summarized. Most analyses reflect the entire participant population, but some pertain to subsets of that population. This is because separate exposure history questionnaires were administered to adults and children and because some questions on the adult questionnaire only applied to females. The section steps through the various modeling results, the outcomes of which informed the key findings of this report. The pilot EA sites are not included in these analyses because, as noted previously, the differences in questionnaire structure restrict the ability to merge the datasets. Lastly, this section summarizes the urine, tap water, and household dust measurements collected across all ATSDR-led sites. The results of the similar urine and environmental data collected for one of the two pilot sites (PA) are not included here.

Throughout this section, the observed trends are further evaluated using insights from the broader scientific literature on PFAS drinking water exposures. For more information on findings of the individual EAs, see the reports [ATSDR 2021b; ATSDRa-g] and consumer summaries published for each of the 10 EAs.

Profile of EA Participants

ATSDR-led EAs

EA participants responded to exposure history questions and reported information on many characteristics, such as their age, sex, race/ethnicity, residential and occupational history, and drinking water consumption. Table 4 provides an overview of the mix of responses across the eight ATSDR-led EAs. More details for specific sites can be found in the individual EA reports.

For these EAs, the average age of participants was 51.9 years, and 80% of the participants identified themselves as White, non-Hispanic. Of EA participants, 54% identified as female, 46% identified as male, and 90% were adults, aged 18 years or older. The age cutoff is important because adults were administered a different exposure history questionnaire with more detailed questions. Among the adult participants, 70% reported living in their current homes for more than 10 years.

Adults were also asked about their current primary source of drinking water: 6.9% said private well, and 31% said bottled water. Adults reported drinking an average of 7.2 8-ounce cups of water a day at home, and 61% said they currently use some type of filtering or treatment device for their drinking water. Examples include filters on refrigerators, pitchers, and faucets; whole-house carbon filtration systems; and reverse osmosis treatment systems. The questionnaire asked adults for their occupational

histories over the past 20 years; 12% reported holding one or more jobs with potential PFAS exposures (e.g., firefighting, military, aviation).

Pilot EAs

As detailed in the individual pilot EA reports, PADOH and NYSDOH collected similar information for the pilot EA participants, although the questionnaires did not collect exactly the same demographic information. Participant counts (adults and children) were 235 and 161 for the PA and NY pilots, respectively. The distribution of males and females was similar to other EA sites, with 44% males and 56% females for the PA pilot and 42% males and 58% females for the NY pilot.

For the PA pilot, the age distribution was 5.1% children aged 3-11 years, 8.1% aged 12-19 years and 87% aged 20 years or older. 54% of participants reported living in their current homes for more than 20 years and 82% reported living at their current address for more than 10 years.

For the NY pilot, 11% of participants were children aged 3-11 years, 18% aged 20-39 years, 30% aged 40-59 years and 40% aged 60 and older. 19% of participants reports living in their current homes for 10-19 years, 22% for 20-29 years, and 20% for more than 30 years.

Race and ethnicity data were not reported in the pilot EAs.

Table 4. Characteristics of EA participants for ATSDR-led EAs*

Characteristics	Westfield, MA	Berkeley County, WV	New Castle County, DE	Airway Heights, WA	Lubbock County, TX	Moose Creek, AK	Security-Widefield, CO	Orange County, NY	Total
Adults and children	N=459	N=275	N=214	N=333	N=214	N=88	N=346	N=59	N=1,988
Age (years)									
<18	11%	10%	5.0%	14%	11%	10%	8.1%	1.7%	9.9%
18 to <50	32%	28%	20%	32%	29%	25%	28%	20%	28%
50+	57%	61%	75%	54%	60%	65%	64%	78%	62%
Sex									
Male	47%	47%	46%	46%	45%	55%	44%	44%	46%
Female	54%	53%	54%	54%	55%	45%	56%	56%	54%
Race and ethnicity†									
White, non-Hispanic	90%	82%	84%	83%	64%	86%	71%	75%	80%
Not White or Hispanic	10%	18%	16%	17%	36%	14%	29%	24%	20%
Adults only									
Years lived at current address									
<10	27%	28%	23%	53%	25%	20%	25%	24%	30%
10 to <20	32%	39%	26%	32%	20%	42%	26%	26%	30%
20 to <30	17%	15%	21%	8.0%	26%	16%	24%	21%	18%
30+	23%	17%	30%	7.0%	29%	22%	25%	29%	22%
Current primary drinking water source									
Public water system	74%	68%	83%	64%	--	41%‡	68%	74%	63%
Bottled water	25%	32%	17%	36%	49%	22%	32%	26%	31%
Private well	--	--	--	--	48%	38%	--	--	6.9%
Average tap water consumption while living at current home (8-ounce cups per day)									
0	14%	9.0%	4.0%	9%	11%	10%	14%	7.0%	11%
>0 to <2	5.6%	6.0%	10%	4.0%	4.0%	8.0%	4.1%	0%	5.4%
2 to <4	20%	17%	17%	11%	21%	14%	13%	12%	16%
4 to <6	21%	16%	26%	17%	16%	10%	19%	28%	19%
6 to <8	14%	16%	13%	13%	10%	11%	14%	12%	13%
8+	26%	35%	30%	46%	38%	47%	36%	40%	35%

Characteristics	Westfield, MA	Berkeley County, WV	New Castle County, DE	Airway Heights, WA	Lubbock County, TX	Moose Creek, AK	Security-Widefield, CO	Orange County, NY	Total
Current use of filter or treatment device									
One or more filter/treatment device(s)	58%	75%	64%	48%	68%	58%	60%	57%	61%
None	42%	25%	36%	52%	32%	42%	40%	43%	39%
Occupational exposures to PFAS in the past 20 Years									
One or more occupational exposure(s)	7.9%	11%	14%	13%	2.0%	29%	13%	11%	12%
None	92%	89%	86%	87%	98%	71%	87%	89%	88%

* The sums of percentages for different fields in this table do not always add up to 100%, because of rounding.

† ATSDR collapsed categories for race and ethnicity for all analyses because of the few responses across categories.

‡ Many participants reported that their current primary drinking water source was delivered water or water from a tank at their homes. These responses were coded as “public water system” for this EA.

Comparison of EA Participants' Demographics to Sampling Frame Demographics

The ATSDR-led EAs were designed to estimate PFAS levels in blood that were generalizable to the corresponding sampling frame as a whole. Given the response rates at each site, which ranged from 1.6% (Orange County, NY) to 18% (Hampden County, EA), each site report explored the potential for participation bias—that is, substantive differences between those who chose to participate and those who did not.

ATSDR used 2010 Census data [USCB 2010] to compare demographic profiles of site participants with the profile of all residents in the corresponding sampling frame. Details of each site's analysis are presented in site-specific reports. Across sites, the comparison revealed the following:

- **Age distribution.** The EA participants generally included a higher proportion of older adults (age 50+ years) and a lower proportion of younger adults (18–50 years) than the corresponding sampling frame populations. Specifically, at all eight sites, EA participants were statistically older than the sampling frame population. The greatest difference was observed at the Orange County, NY EA where 78% of the EA participants reported being 50 or older, but 21% of the sampling frame population falls in this age range [ATSDR 2022g]. ATSDR chose 50 years as a cutoff for older and younger adults based on the median age of menopause in the United States, which may affect exposure profiles. Similarly, at this same site 1.7% of the EA participants reported being under 18, though the proportion of children in the sampling frame is 31%.

- **Race/ethnicity.** At five of eight EA sites, the racial/ethnic composition of the EA participants was statistically different than the sampling frame population. In general (seven of eight EA sites), each sampling frame population was composed of populations that were majority "White alone" according to Census data. However, at four sites, the proportion of EA participants who identified as "White alone" was significantly greater than the proportion of the corresponding sampling frame population. Two sites had statistically fewer Black participants; two sites had statistically fewer Hispanic participants; and one site had statistically more participants who identified as more than one race. For this comparison, combined race and ethnicity were not available at the block level from the Census. Therefore, only ethnicity and certain race categories were compared because of the small number of respondents at some of the EA sites.

Although each population invited to participate was likely representative of the sampling frame, the population that ultimately enrolled in each EA was generally older and often contained more White, non-Hispanic individuals. The effect of age and race/ethnicity on PFAS blood levels and its implications on a site's community statistics is further explored in each site-specific report. However, in general, the overrepresentation of older participants in the EAs tend to bias estimates of community PFAS blood levels higher. This is because PFAS blood levels tend to increase with age. To help account for the bias caused by an older sample of EA participants, ATSDR calculated geometric means that were age-adjusted to each community's sampling frame population. ATSDR focused its analyses and interpretations on these age-adjusted results. Differences in the racial/ethnic composition of EA participants are less likely to have an effect on the findings reported here because ATSDR did not often find significant associations between race/ethnicity and blood PFAS levels. Therefore, ATSDR did not calculate community level statistics adjusted for race and ethnicity. Refer to each site's individual report for these sampling frame-adjusted community statistics.

PFAS Measured in Blood Across the 10 EAs

This section summarizes PFAS levels that ATSDR measured from the 1,988 blood samples provided by eligible participants across the eight EAs and the 396 participants from the two pilot EAs. At least one PFAS was detected in the blood of nearly all EA participants (>99%). Because of the widespread use of PFAS, such high detection frequencies are common in the general U.S. population [CDC 2019]. Five PFAS (PFHxS, PFOS, PFOA, PFNA, PFDA) were frequently detected in the blood of EA participants (detection frequencies above 60%). PFUnA and MeFOSAA were detected in fewer than 60% of the EA participant samples; however, at some sites, detection frequencies of these two PFAS were higher.
Results are summarized in tables, bar graphs, and 'box and whisker' plots (see text box).

Comparison of EA Participants' PFAS Blood Levels to the National Population

This section compares PFAS levels measured among EA participants to levels found in the U.S. general population. Since the age distribution of EA participants differs from the age distribution of NHANES, ATSDR calculated geometric means adjusted to the age distribution of the U.S. population in NHANES 2015–2016. Because there were very few participants under 30 years old at many sites, these age-adjusted calculations may still not be fully adjusted to the 2015–2016 NHANES population at every site; however, the age-adjusted geometric means reported here allow for a more even comparison across sites controlling for differences in age distribution.

Table 5 shows the statistical comparison of age-adjusted geometric means from each EA site to the data available from the 2015–2016 and the 2017–2018 NHANES surveys [CDC 2019 and 2021]. Each EA site report and the NY pilot EA presented comparisons with the latest NHANES survey at the time of the EAs, which was the 2015–2016 survey. The PA pilot EA compared to the 2013-2014 NHANES survey period. Since that time, geometric means for blood PFAS levels from the 2017-2018 survey have become available. Comparisons against this newer survey are described below. Figure 2 and Figure 3 present a visual depiction of these comparisons. Note that the data in these figures are arranged by geometric mean for each PFAS so the order of sites differs from figure to figure. Table A1 through A8 in Appendix A show additional summary statistics for the eight ATSDR-led EA sites.

Of the PFAS analyzed in blood, PFHxS levels had the largest elevations when compared to national levels. At all 10 EA sites, the age-adjusted geometric mean PFHxS levels among participants was statistically higher than national levels from both NHANES surveys. Across sites, blood PFHxS levels ranged from 2.4 to 61 times NHANES 2017–2018 levels. The age-adjusted geometric mean blood PFOS and PFOA levels were also generally statistically elevated across EA sites. PFOS was elevated in 8 of 10 EAs at 1.2 to 9.2 times NHANES 2017–2018 levels, and PFOA was statistically elevated in 7 of 10 EAs, at 1.2 to 6.3 times. The highest age-adjusted geometric mean blood levels for PFHxS (65.6 µg/L), PFOS (39.1 µg/L), and PFOA (8.9 µg/L) were all observed in Airway Heights, WA. PFNA levels were also statistically elevated in 4 of 10 EAs, and blood levels at these EA sites ranged from 1.4 to 2.2 times NHANES 2017-2018 levels with a maximum adjusted geometric mean of 0.903 µg/L in New Castle, DE.

PFHxS has been detected in legacy AFFF formulations and may be present in impacted drinking water supplies because of degradation of other PFAS. The estimated half-life of PFHxS is longer than the estimated half-lives for PFOS and PFOA, which may explain why blood PFHxS levels were more elevated than levels of other PFAS [ATSDR 2021a].

The remaining PFAS (PFDA, PFUnA, and MeFOSAA) were not measured at the two pilot EAs. Adjusted geometric mean blood PFDA and PFUnA were statistically elevated at one of the eight ATSDR-led sites

(New Castle, DE) at levels that were 1.4 and 1.7 times NHANES 2017–2018 levels, respectively. Blood MeFOSAA levels were not statistically elevated at any sites.

All PFAS measured in blood for this EA have been phased out of production in the United States. Following this phase-out, national blood levels for most PFAS have been steadily declining since 2000 [CDC 2019]. Differences between geometric mean EA blood levels, collected in 2019 or 2020, and the NHANES 2019-2020 geometric mean (not yet available) could be greater than the differences between geometric mean EA blood levels and the NHANES 2017–2018 geometric mean presented here.

ATSDR compiled blood PFAS levels for the three most prevalent PFAS (PFHxS, PFOS, and PFOA) from other PFAS biomonitoring studies to provide further context on the current EA blood levels (Appendix A, Table A9). Average PFAS blood levels from all EA sites were below those measured in occupationally exposed manufacturing workers (e.g., Olsen et al. 2003, Steenland and Woskie 2012). While all EA geometric means reported here were age-adjusted to the national age distribution for a more direct comparison, the geometric means from other studies in Table A9 were not age-adjusted. Even so, below are some observations:

- For PFHxS, age-adjusted average blood levels from four EA sites were higher than those observed in other communities with contaminated drinking water: Portsmouth, New Hampshire; Little Hocking, Ohio; and Decatur Alabama [NH DPHS 2016; Frisbee et al. 2009; Olsen et al. 2003]. At two other sites, PFHxS blood levels were within the range (4.1–6.4 µg/L) of these other communities; and at four sites, PFHxS levels were below the range observed in other communities.
- For PFOS, age-adjusted average blood levels from four EA sites were within the range (8.6–39.8 µg/L) of those observed in other communities with contaminated drinking water, and six EA sites were below the range observed in these communities.
- For PFOA, age-adjusted average blood levels from two EA sites were within the range (3.1–227.6 µg/L) of those observed in other communities with contaminated drinking water, and eight EA sites were below the range observed in these communities.

Table 5. Geometric means and 95% confidence intervals for blood PFAS levels in µg/L age-adjusted to match NHANES 2015–2016

Site	PFHxS	PFOS	PFOA	PFNA	PFDA	PFUnA	MeFOSAA
PA Pilot EA	**5.99* (5.09-7.05)**	**9.43* (8.27-10.8)**	**2.85* (2.56-3.18)**	**0.692* (0.646-0.742)**	NA§	NA§	NA§
NY Pilot EA	**2.62* (2.31-2.97)**	**5.64* (5.17-6.15)**	1.38‡ (1.24-1.53)	**0.581† (0.540-0.625)**	NA§	NA§	NA§
Westfield, MA	**4.02* (3.58–4.52)**	**5.29* (4.89–5.73)**	**1.77* (1.66–1.89)**	0.418‡ (0.390–0.447)	0.148† (0.139–0.158)	NA§	NA§
Berkeley County, WV	**2.96* (2.45-3.57)**	**5.06† (4.37-5.86)**	1.33‡ (1.23-1.44)	0.347* (0.308-0.391)	0.134* (0.123-0.147)	NA§	NA§
New Castle County, DE	**11.5* (9.05–14.7)**	**13.5* (11.2–16.3)**	**3.74* (3.31–4.24)**	**0.903* (0.831–0.980)**	**0.279* (0.252–0.309)**	**0.208†, ¶ (0.184–0.235)**	0.130¶ (0.108–0.146)
Airway Heights, WA	**65.6* (55.8–77.1)**	**39.1* (33.9–45.0)**	**8.91* (7.84–10.1)**	**0.694* (0.615–0.783)**	0.200‡ (0.179–0.214)	NA§	NA§
Lubbock County, TX	**4.93* (3.39–7.19)**	3.58* (3.10–4.14)	**1.94* (1.60–2.34)**	0.169* (0.151–0.188)	0.124* (0.114–0.135)	NA§	NA§
Moose Creek, AK	**9.13* (6.55–12.7)**	**14.6* (11.6–18.4)**	**1.75† (1.56–1.98)**	0.275* (0.238–0.317)	NA§	NA§	0.126¶ (0.107–0.150)
Security-Widefield, CO	**8.08* (6.88–9.50)**	**5.15† (4.48–5.91)**	**1.82* (1.65–2.02)**	0.245* (0.223–0.270)	0.119* (0.108–0.131)	NA§	0.122¶ (0.110–0.136)
Orange County, NY	**3.56* (3.00–4.22)**	4.76 (4.23–5.35)	1.32‡ (1.17–1.49)	0.293* (0.259–0.331)	0.171† (0.158–0.186)	0.126¶ (0.113–0.141)	NA§
NHANES 2015-2016	1.18 (1.08-1.30)	4.72 (4.40-5.07)	1.56 (1.47-1.66)	0.577 (0.535-0.623)	0.154 (0.140-0.169)	NA§	NA§
NHANES 2017-2018	1.08 (0.996-1.18)	4.25 (3.90-4.62)	1.42 (1.33-1.52)	0.411 (0.364-0.464)	0.193 (0.178-0.209)	0.125 (0.115-0.135)	0.130 (0.121-0.140)

NA = not applicable; µg/L = micrograms per liter; **bolded** values are statistically higher than NHANES 2017-2018.

*Statistically significantly difference from NHANES 2015-2016 and 2017-2018 ($p<0.05$).

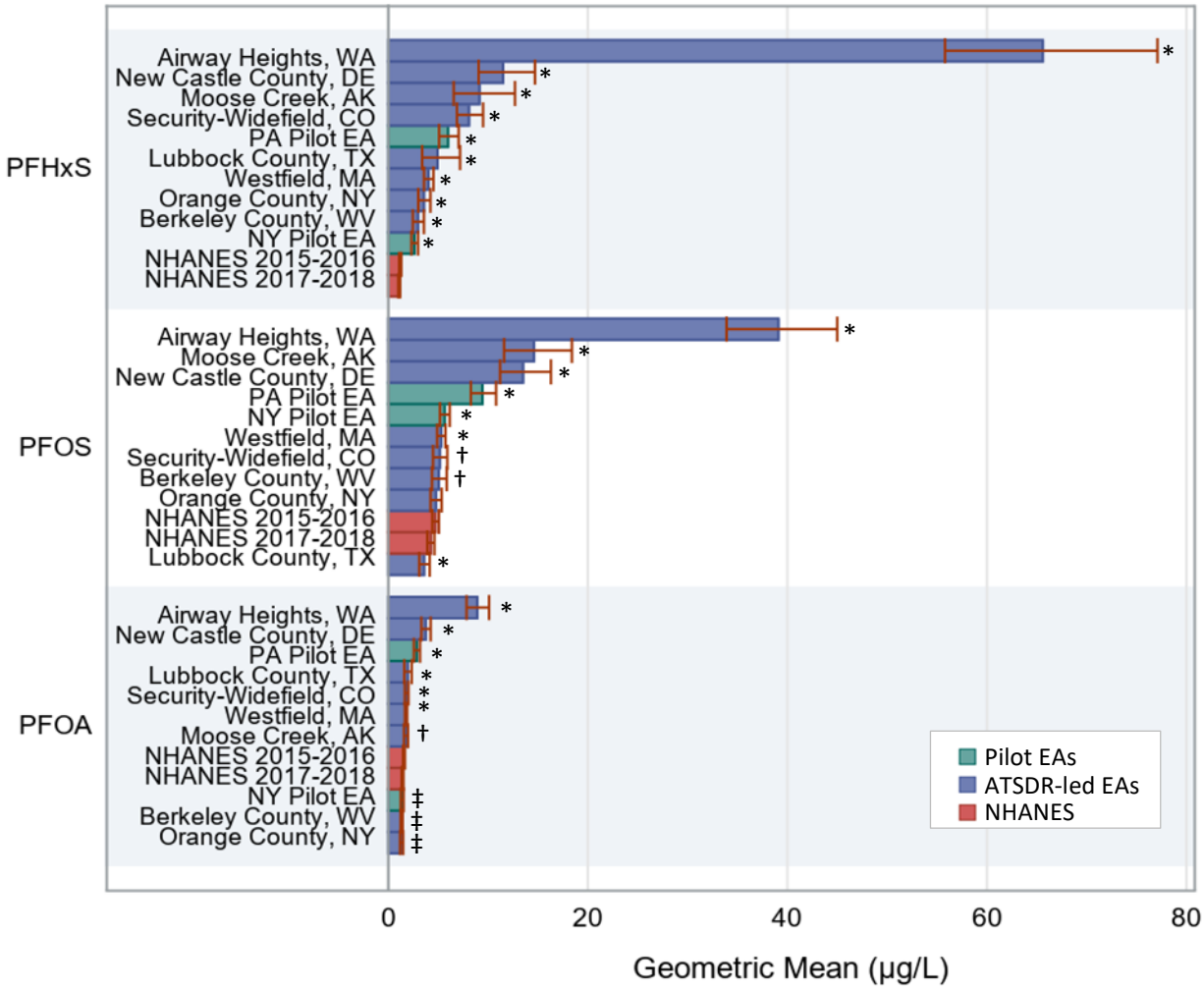
† Statistically significant difference from NHANES 2017-2018 only ($p<0.05$).

‡ Statistically significant difference from NHANES 2015-2016 only ($p<0.05$).

§ Per the protocol, geometric means were not calculated for PFAS detected in less than 60% of samples.

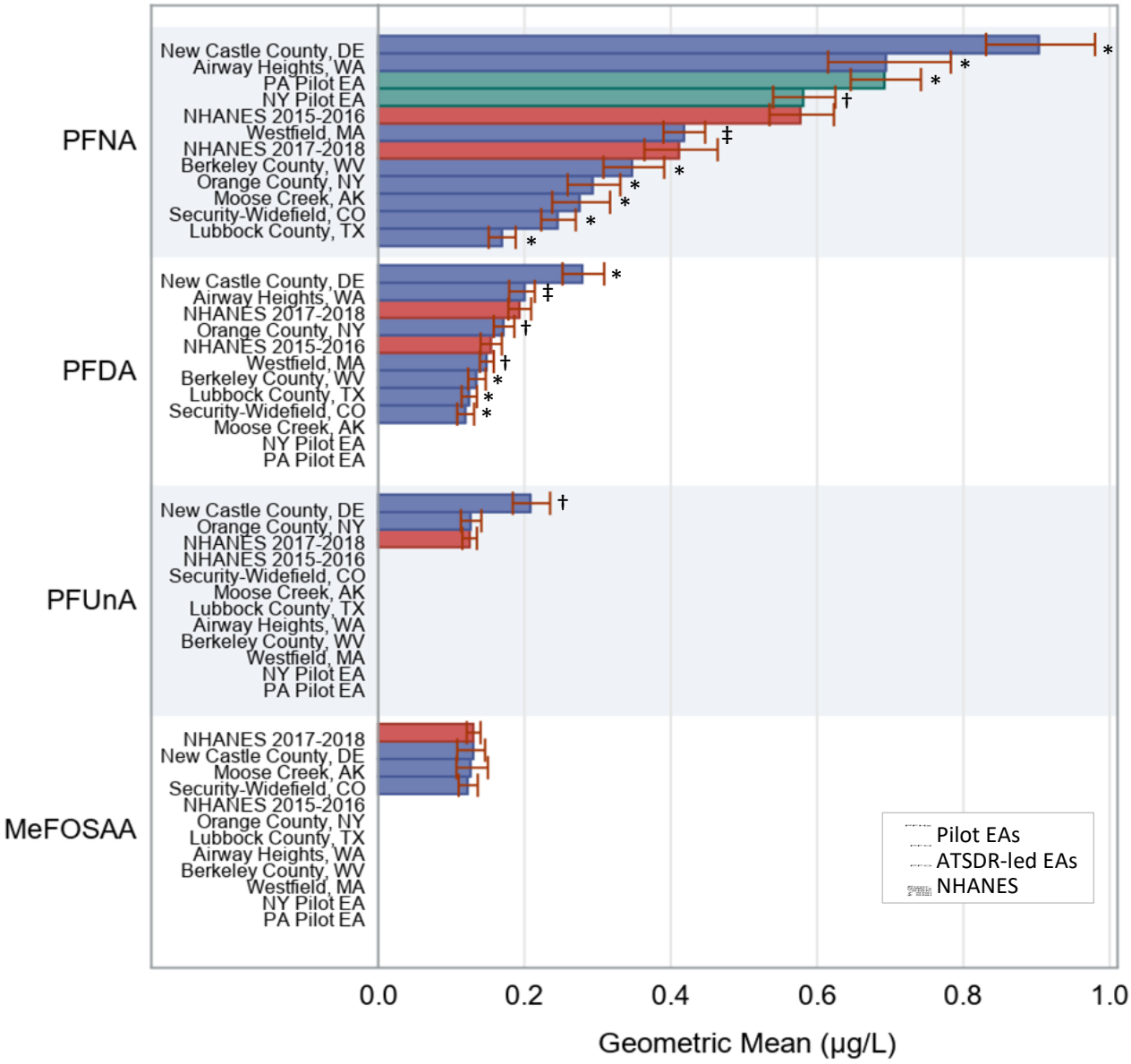
¶ No statistical comparison could be made with NHANES 2015-2016 because NHANES 2015-2016 did not calculate a geometric mean for this PFAS because this PFAS was detected in less than 60% of NHANES 2015-2016 sample.

Figure 2. Geometric means and 95% confidence intervals for blood PFHxS, PFOS, and PFOA levels in micrograms per liter (µg/L) age-adjusted to NHANES 2015–2016 across EA sites compared to national levels



Error bars represent 95% confidence intervals. Note that overlapping confidence intervals do not mean that differences are not statistically significant.
**Statistically significantly difference from NHANES 2015-2016 and 2017-2018 (p<0.05).*
† Statistically significant difference from NHANES 2017-2018 only (p<0.05).
‡ Statistically significant difference from NHANES 2015-2016 only (p<0.05).

Figure 3. Geometric means and 95% confidence intervals for blood PFNA, PFDA, PFUnA, and MeFOSAA levels in micrograms per liter (µg/L) age-adjusted to NHANES 2015–2016 across EA sites compared to national levels



Error bars represent 95% confidence intervals. Note that overlapping confidence intervals do not mean that differences are not statistically significant.
**Statistically significantly difference from NHANES 2015-2016 and 2017-2018 (p<0.05).*
† Statistically significant difference from NHANES 2017-2018 only (p<0.05).
‡ Statistically significant difference from NHANES 2015-2016 only (p<0.05).

Comparison of PFAS Blood Levels to Drinking Water Levels and Mitigation Date

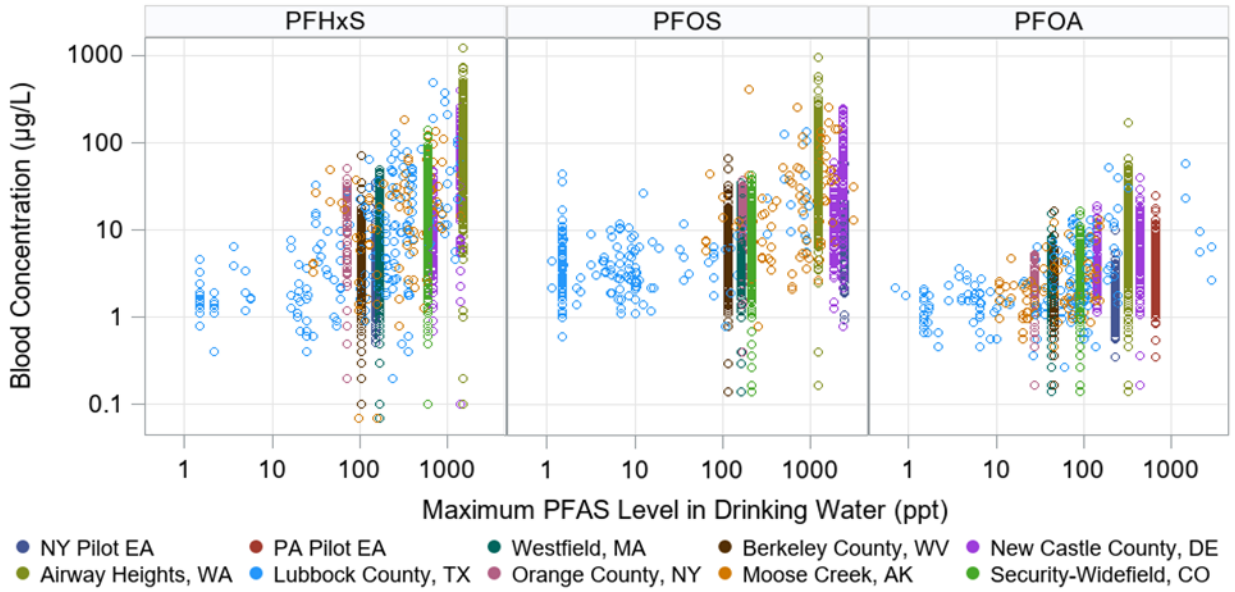
PFAS were first detected in drinking water supplies at EA sites between 2009 and 2017, though contamination likely began before the first detections at each site. Among the site documents ATSDR reviewed, the highest sampling result from active drinking water supplies ranged from 70 to 1,500 ppt for PFHxS, 114 to 2,615 ppt for PFOS, and 27 to 2,900 ppt for PFOA. From 2014 through 2019, each site had reduced PFAS levels below EPA's 2016 HA in each drinking water supply. However, these PFAS have very long biological half-lives (2.1 to 35 years). Therefore, even though drinking water PFAS exposures across sites were significantly reduced from 2014 to 2019, past drinking water exposures were likely a contributing factor to the EA participants' elevated blood PFAS levels observed sometimes years later.

ATSDR considered the relationship between observed drinking water levels at all 10 EA sites and corresponding blood levels. Drinking water testing data were obtained from either the affected public water supplies in each community or from private well testing conducted by the Air Force. For public water system sites, the maximum values shown in Table 1 were assigned to the site's EA participants. For the New Castle County EA, significant differences in both the PFAS blood levels and final mitigation dates from the two affected water systems led ATSDR to assign participants from each water system different maximum PFAS drinking water levels. For the Security-Widefield EA, multiple drinking water systems were contaminated but site-specific analyses did not show significant differences between PFAS blood levels in participants from each water systems, so all participants were assigned the same maximum drinking water level and mitigation date. For the two private well sites (Lubbock, TX and Moose Creek, AK), individual households were assigned the maximum value measured from potentially multiple rounds of testing by the Air Force. In some instances, drinking water values were not available for households at private well sites. This analysis was only conducted for PFHxS, PFOS, and PFOA based on the availability of drinking water data.

Figure 4 shows PFAS blood levels in participants at each site by corresponding PFAS drinking water l evels. An increasing relationship between drinking water levels and blood levels of all three PFAS measured in drinking water can be seen. The increase in blood PFAS levels appears to begin starting at about 70 to 100 ppt in drinking water. However, few of the EA sites contained participants who received drinking water with lower levels of PFAS (<70 ppt).

PFHxS had the strongest correlation with corresponding drinking water levels. ATSDR used Pearson correlation coefficients (r) for this analysis. An r of 0 means two data sets are uncorrelated, an r of 1 means two data sets are exactly correlated (i.e., they rise and fall in proportional amounts), and an r of negative 1 means the two datasets are exactly inversely correlated (i.e., when one data set rises the other falls proportionately). Across all 10 EA sites, the correlation between PFHxS in drinking water and blood was 0.62, and for every 1% increase in drinking water concentration, there was a corresponding 0.57% increase in blood PFHxS level. Similarly, drinking water levels and blood levels were correlated for both PFOS (r=0.48) and PFOA (r=0.44). For every 1% increase in PFOS and PFOA, there was a corresponding 0.46% and 0.48% increase in blood levels, respectively.

Figure 4. Maximum PFAS level in drinking water and corresponding blood levels by site



A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.

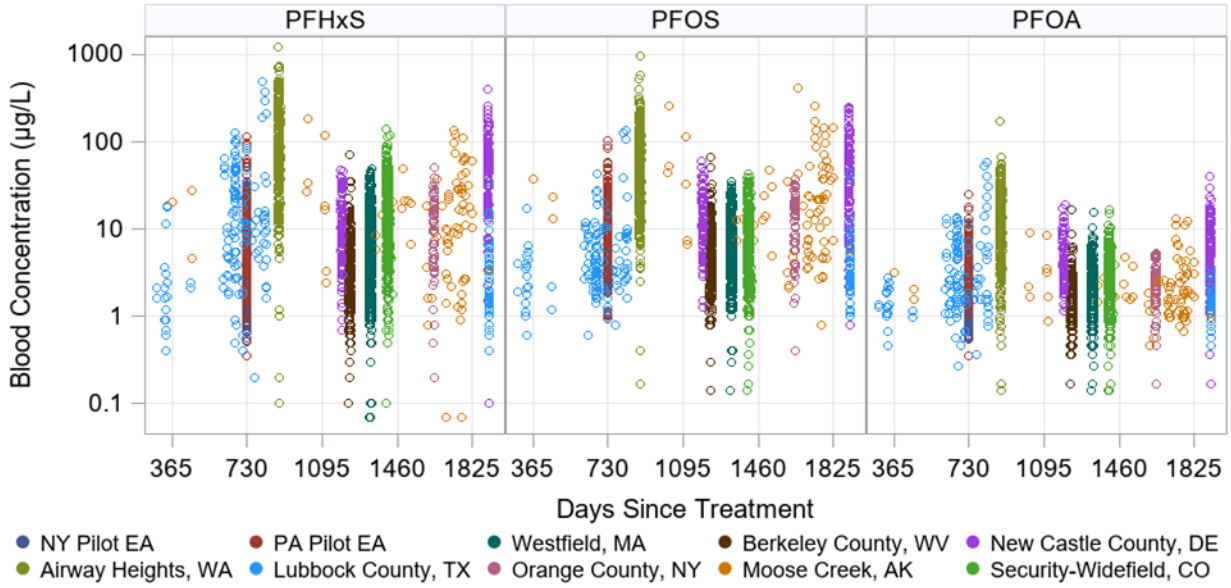
PFAS remain in the body for a long period of time; however, blood PFAS levels are expected to decrease as the time since the last exposure increases. Therefore, comparisons in blood PFAS levels between sites may be affected by 1) final mitigation dates at each site (from August 2014 to September 2019),2) the degree of historical water contamination, and 3) date of biological sample collection for each EA participant (from April 2018 through October 2020). To account for this, ATSDR considered the time between when participants provided biological samples during the EA and the final mitigation dates for each site. The mitigation date corresponds to the final date of the water treatment installation or intervention by the Air Force or public water system when exposure no longer exceeded the EPA 2016 HA. For public water system sites, the difference between the final mitigation dates shown in Table 1 and when the EA participant provided biological samples was calculated. For private well sites, individual household levels mitigation dates were provided by the Air Force for most households and this date was used instead of the Table 1 value, when available.

Figure 5 shows that the time between when a participant provided their biological sample and the last mitigation date at a site was not statistically correlated with blood PFHxS (r = -0.04) or blood PFOS levels (r= -0.05*)*. Across sites, blood PFOA may have a weak negative correlation with the days since final mitigation (r = -0.16), but this result does not appear consistent within all sites. For example, in both Moose Creek, AK and New Castle, DE, an increasing relationship is observed despite large differences in final mitigation dates across participants within each site.

The lack of clear associations between blood PFAS levels and final mitigation date may be due to varying definitions of final mitigation across the EA sites. To properly account for both drinking water levels and the time between biological sample and the end of exposures would require a pharmacokinetic model that takes into account the biological half-life of each PFAS. In either case, these results suggest that elevated blood PFHxS, PFOS, and PFOA levels were due to PFAS-contaminated drinking water. These results were robust even though each EA site mitigated its drinking water exposures at different points in time (from August 2014 to September 2019), and the date of biological sample collection for each EA

participant varied (from April 2018 through October 2020). ATSDR concludes that the maximum observed PFAS levels in drinking water is a much stronger predictor of blood PFAS levels and is evidence of a drinking water source of exposure across EA sites.

Figure 5. Days since mitigation in drinking water and corresponding blood levels by site



A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.

Correlations Among PFAS in Blood

ATSDR also evaluated correlations between PFAS in blood in the 2,384 participants from the 10 EAs. This analysis determined whether any PFAS tended to have similar patterns in the blood of EA participants, which might suggest similar sources and exposure pathways. Table 6 shows the Pearson correlation coefficients for the five most frequently detected PFAS.

PFHxS, PFOS, and PFOA blood levels showed the strongest correlations (Table 6). All pairings among these chemicals had Pearson correlation coefficients close to 1 (r = 0.83–0.86). PFNA was strongly correlated with PFDA (r = 0.70) and to a lesser degree PFOS (r = 0.67) and PFOA (r = 0.62). PFDA had the weakest correlations with the other PFAS (r = 0.16–0.36).

The correlation pattern among PFAS observed here was generally consistent with patterns observed across individual sites. While statistically significant correlations were often observed across all pairs of PFAS, the correlations among PFHxS, PFOS, and PFOA were always significant and consistently the strongest, suggesting similar or common background sources or exposure pathways. PFHxS and PFOS, and to a lesser extent PFOA, have many common exposure sources, as these compounds are often found together in consumer products. While correlations between PFAS have been observed in other studies [NH DPHS 2016; ATSDR 2013; CDC 2019], the correlations observed between these three PFAS in the blood results from these 10 EAs are much higher than those observed in the general U.S. population (r between 0.46 and 0.66) [Calafat et al. 2007b]. Instead, the high correlation between PFHxS, PFOS, and PFOA is consistent with those found in the blood of people living in communities with contaminated drinking water [ATSDR 2013], providing further evidence that drinking water was likely a contributing

source of exposure among EA participants. In addition, the correlations between PFHxS, PFOS, and PFOA in these EAs study are much higher than the correlations observed for PFNA and PFDA, two compounds that were not generally found at elevated levels in affected water systems or private wells, providing further evidence of a distinct exposure pathway for these three compounds.

Table 6. Pearson correlation coefficients between PFAS in blood (log)*

	PFHxS	PFOS	PFOA	PFNA	PFDA
PFHxS	1	0.85	0.86	0.44	0.16
PFOS	0.85	1	0.83	0.67	0.36
PFOA	0.86	0.83	1	0.62	0.34
PFNA	0.44	0.67	0.62	1	0.70
PFDA	0.16	0.36	0.34	0.70	1

* All correlations are significant ($p<0.001$).

PFAS Blood Levels by Demographics and Other Exposure Characteristics for ATSDR-led EAs

This section examines how the demographic and exposure history information from the questionnaire for the eight ATSDR-led EAs related to blood PFAS levels. Since different questionnaires were administered to adult and child participants, responses for these populations were analyzed separately. Furthermore, some questions were applicable only to female adult participants and are therefore also presented separately. Appendix C (Tables C1 and C2) summarizes all adult and child questionnaire responses.

ATSDR used univariate and multivariate models to evaluate the relationships between questionnaire data from the eight ATSDR-led EAs and blood PFAS levels. ATSDR developed models for the five PFAS that were detected in over 60% of samples (PFHxS, PFOS, PFOA, PFNA, and PFDA). Data from the two pilot EAs are not included in these analyses because the questionnaires were different and more limited. Table 7 summarizes the demographic and exposure characteristics that were statistically significant in each adult multivariate model, and Table 8 summarizes the demographic and exposure characteristics that were statistically significant in each child multivariate model. The discussion that follows focuses on variables that were significant for PFHxS, PFOS, and PFOA, the compounds that were elevated in drinking water across sites. Appendix C provides a complete set of results for all five PFAS.

Table 7. Summary of significant variables (p<0.05) in multivariate regression models in adult participants in ATSDR-led EAs

Parameter	PFHxS			PFOS			PFOA			PFNA			PFDA		
	All Adult	Adult Female	Adult Male	All Adult	Adult Female	Adult Male	All Adult	Adult Female	Adult Male	All Adult	Adult Female	Adult Male	All Adult	Adult Female	Adult Male
Age (continuous)	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Sex (categorical)	✓	NA	NA	✓	NA	NA	✓	NA	NA	✓	NA	NA	—	NA	NA
Age × sex (continuous)*	✓	NA	NA	✓	NA	NA	✓	NA	NA	✓	NA	NA	—	NA	NA
Race/ethnicity (categorical)	—	—	—	—	—	—	—	—	—	✓	✓	✓	—	—	—
Years in sampling frame in the past 20 years [Residency duration] (continuous)	✓	✓	✓	✓	✓	✓	✓	✓	✓	—	—	—	—	—	—
Drinking water source [bottled, private well, public] (categorical)	—	—	—	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Drinking water filter use [any filter, no filter, bottled water only,] (categorical)	✓	✓	—	✓	✓	✓	✓	✓	✓	—	—	—	—	—	—
Drinking water consumption (continuous)	✓	—	✓	—	—	—	—	—	—	—	—	—	—	—	—
Max PFAS level in drinking water (continuous)	✓	✓	✓	✓	✓	✓	✓	✓	✓	NA	NA	NA	NA	NA	NA
Days since mitigation (continuous)	—	—	—	✓	✓	✓	✓	✓	✓	—	—	—	✓	—	✓
Cleaning frequency (categorical)	—	—	—	—	—	—	—	—	—	✓	✓	—	—	—	—
Stain-resistant product use (categorical)	—	—	—	—	—	—	—	—	—	✓	—	—	—	—	—
Local milk consumption (categorical)	✓	—	—	—	—	—	✓	—	—	—	—	—	—	—	—
Local fruit and vegetable consumption (categorical)	—	—	—	—	—	—	—	—	—	—	—	—	✓	—	✓
Childbirth (yes/no)	NA	✓	NA	NA	—	NA	NA	—	NA	NA	—	NA	NA	—	NA

✓ = statistically significant, '—' = not statistically significant, NA = not applicable

*This variable is an interaction term, which means the effect of one variable on serum PFAS levels depends on the value of another.

Table 8. Summary of significant variables (p<0.05) in multivariate regression models in child participants from ATSDR-led EAs

Parameter	PFHxS	PFOS	PFOA	PFNA	PFDA
Age (continuous)	✓	—	✓	—	—
Sex (categorical)	—	✓	✓	✓	✓
Race/ethnicity (categorical)	—	—	—	✓	—
Years in sampling frame [Residency duration] (continuous)	✓	—	✓	—	—
Drinking water consumption at home (continuous)	—	—	—	✓	—
Drinking water PFAS level (categorical)	✓	✓	✓	NA	NA
Days since mitigation (continuous)	✓	✓	✓	—	—
Local fruit and vegetable consumption (categorical)	—	—	—	—	✓
Frequency of contact with soil (categorical)	—	✓	—	✓	✓
Months of formula use (continuous)	—	—	—	✓	—
Months of breastfeeding (continuous)	—	—	—	—	✓

✓ = statistically significant, '—' = not statistically significant, NA = not applicable

The following subsections briefly summarize results for these topics. All other results are presented in Appendix C, as described below.

- Tables C1 and C2 present response frequencies for all questions included in the adult and child questionnaire, respectively. These tables also present geometric means and 95% confidence intervals around geometric means stratified by the response options (e.g., statistics are presented separately for males and females) for PFHxS, PFOS, PFOA, PFNA, and PFDA. Blood levels of MeFOSAA and PFUnA were not detected at a high enough frequency to present meaningful results.
- Tables C3 and C4 present univariate modeling results for all questions in the adult and child questionnaire for the same five PFAS, as data allow. Data are presented only when a category had at least 10 responses. Some categories were collapsed to meet this threshold.
- Tables C5–C24 present multivariate modeling results for PFHxS, PFOS, PFOA, PFNA, and PFDA. Multivariate models, including the goodness-of-fit measure, R-squared or R^2, are presented separately for all adults, male adults only, female adults only, and children. The closer the R^2 value is

Goodness of Fit Measure

R-squared or R^2 is a statistical measure used to evaluate how well a mathematical model explains the measured data by looking at the differences between the observed PFAS concentrations and values predicted by the model.

- An R^2 of 1 means the model completely predicts the observed PFAS concentrations, so that there are no differences between the model and the PFAS concentrations and 100% of the PFAS concentrations are explained by the model.
- An R^2 of less than 1 means that there are measurements scattered higher and/or lower than the model predictions and there are differences between the two.

to 1, the more the variables in the model explain the variability in blood PFAS levels. Across all adult models, R^2 values ranged from 0.03 to 0.41. Across all child models, R^2 values ranged from 0.14 to 0.62. ATSDR modeled male and female adults separately to explore sex-specific differences including the potential effect of childbirth and breastfeeding on female blood PFAS levels. The variables considered in male-only and female-only models were limited to those that were significant in final all-adult models.

- Figures C1–C47 present boxplots for unadjusted blood levels by each demographic and exposure characteristic included in the statistical analyses.

Variability describes the spread or dispersion of data values. If the values are similar to each other there is little variability, if the values are spread out there is more variability.

Multivariable regression can help us understand how much of the variability in PFAS blood levels can be explained by the combination of factors in the model such as age, sex, and length of residency, among others. If the model does not explain a large portion of the variability, that means there are other unknown factors influencing PFAS levels in blood.

Blood PFAS Levels and Age

Because many studies have found that older people have higher blood PFAS levels, ATSDR investigated how EA participants' ages related to their blood levels. At seven of eight sites, age was statistically associated with blood PFAS levels after controlling for other variables. As Figure 6 illustrates, this association remained in the combined dataset where blood levels for PFHxS, PFOS, PFOA, PFNA, and PFDA increased with participant age for adults, and PFHxS and PFOA levels decreased with age for children.

For adults, ATSDR's univariate analysis showed that blood PFHxS, PFOS, PFOA, PFNA, and PFDA were higher in older individuals than in younger individuals, and this finding was statistically significant. Figure 6 shows the correlation between age and blood PFAS levels was strongest for PFHxS. The univariate analysis indicates that on average, blood PFHxS levels across participants from eight EA sites increased 2.6% for every year of participant age in adults. This suggests a 29% increase in blood PFHxS levels for every 10 years of age in adult participants. The calculated increases for PFOS (2.4% per year of participant age), PFOA (1.3% per year of participant age), PFNA (1.5% per year of participant age), and PFDA (0.6% per year of participant age) were lower.

ATSDR's multivariate analysis—which accounted for various confounding factors—provided further perspective on this trend, showing that the age dependence was generally stronger for women than men among adults for PFHxS, PFOS, PFOA, and PFNA. For example, the all-adult model for PFHxS (Appendix C, Table C5) suggests a 3.0% increase in blood PFHxS for every additional year of participant age in female participants, and a 1.1% increase in blood PFHxS levels for every additional year of participant age in males, when controlling for other characteristics; these findings were statistically significant. Similar results were observed in the

What are confounders?

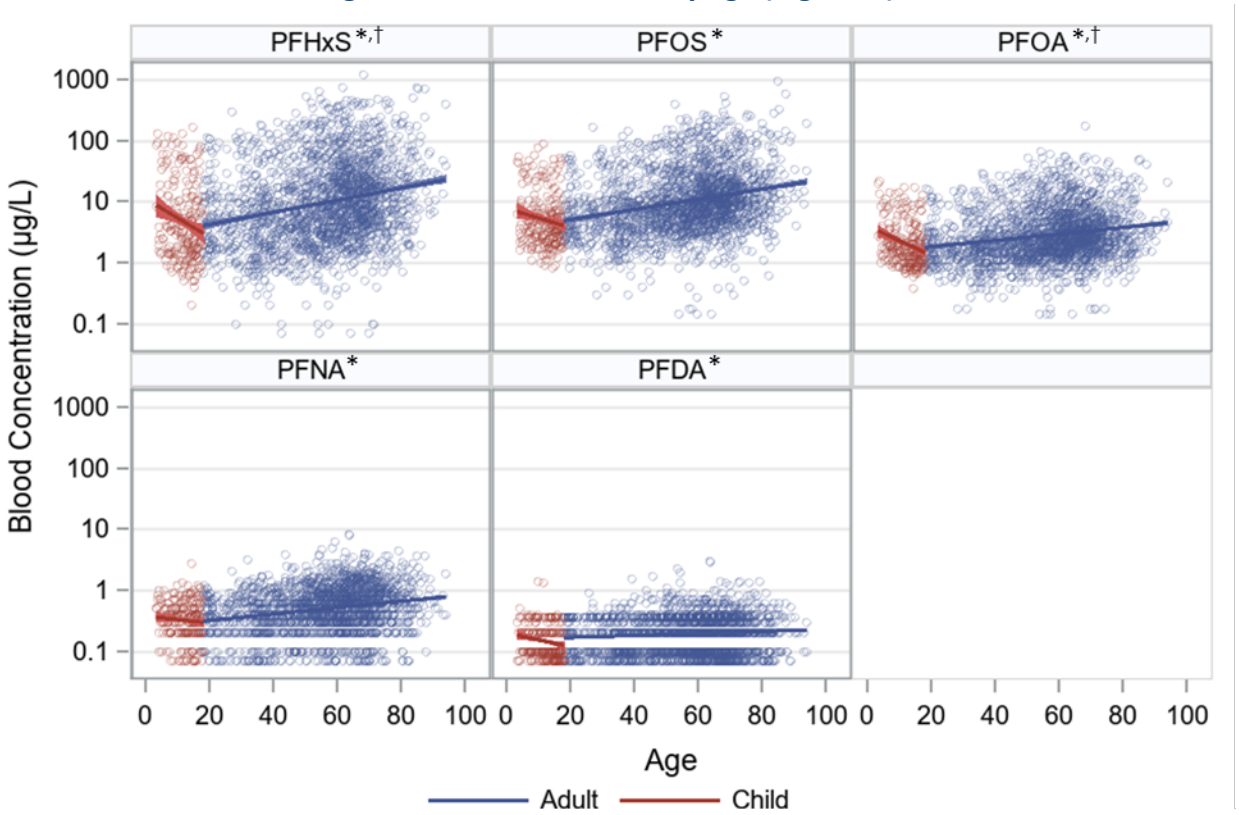
Confounding is a distortion in the estimated relationship between a potential predictor and measure of exposure due to the presence of a third variable—called a confounder. In order for confounding to occur, that third variable must be associated with both the predictor (or independent variable) and the measure of exposure (or dependent variable). For example, age can act as a confounder on the estimated strength of association between length of residence in the sampling frame and blood PFAS levels.

By adjusting for these types of confounding variables in multivariate statistical models, ATSDR can calculate less biased estimates of the relationships between dependent and independent variables of interest.

stratified male-only and female-only models. Age also remained a significant predictor of blood levels for PFOS (2.5% per year in females and 1.3 per year in males, Table C8), PFOA (1.7% per year in females and 0.6% per year in males, Table C11), and PFNA (1.6% per year in females and 1.0% in males, Table C14) in all-adult multivariate models.

As Figure 6 shows, blood PFHxS and PFOA levels decreased with increasing participant age in children (participants under 18), and this trend was statistically significant in both univariate and multivariate analyses. Multivariate models showed a 10.4% decrease in blood PFHxS levels and a 5.9% decrease in blood PFOA levels for every additional year in child participant age.

Figure 6. PFAS blood level by age (log scale)



A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant trend (p<0.05) in adults in univariate regressions.*
†Statistically significant trend (p<0.05) in children in univariate regressions.
Note: Age was statistically significant in adult multivariate regressions for PFHxS, PFOS, PFOA, PFNA, and PFDA; and age was statistically significant in child multivariate regressions for PFHxS and PFOA.

As previously discussed, blood PFAS levels were statistically higher in older adults than younger adults. In children, the opposite trend was observed for blood PFHxS and PFOA, which were higher in younger children compared to older children. Differences in the associations between blood PFAS levels and age in adults and children have been observed in other studies [ATSDR 2013; NH DPHS 2016; CDC 2019]. Generally, increasing blood levels in adults are due to the long biological half-lives of PFAS and changes in excretion (e.g., menopause) with increasing age. The half-life of a chemical is the amount of time it takes for 50% of the substance to be eliminated from the body. Most studies estimate a half-life of

PFHxS between 4.7 and 8.5 years, although some have estimated half-lives as long as 35 years [ATSDR 2021a]. Most half-life estimates for PFOS are between 3.3 and 7.4 years, with a maximum of 27 years [ATSDR 2021a]. For PFOA, most studies estimate the half-life between 2.1 and 3.9 years with a maximum of 10.1 years [ATSDR 2021a]. In the presence of continued exposures that exceed clearance rates, PFAS will accumulate in the human body over time. On the other hand, PFAS blood levels in children may be inversely associated with age due to multiple factors including early life exposures and growth dilution. Early-life exposures may have occurred since PFAS can cross the placenta and are found in breast milk [ATSDR 2021a]. In addition, hand-to-mouth touching and spending more time closer to the floor with settled dust is much greater in toddlers than in older children. As a child grows, these early-life exposure factors diminish. Additionally, large increases in body size lower blood levels despite increasing or constant PFAS body burdens. This process is known as growth dilution [Koponen et al. 2018].

Blood PFAS Levels by Sex

ATSDR investigated how blood PFAS levels vary between males and females because previous research has shown that, all other factors being equal, adult males tend to have higher blood PFAS levels than adult females. At seven of eight sites, sex was statistically associated with blood levels for at least one PFAS after controlling for other variables. ATSDR's univariate analyses showed that PFAS levels were indeed higher in adult males than in adult females in the combined dataset by 27% for PFOS and by 12% for PFNA in univariate analyses (Figure 7).

The all-adult multivariate models showed a significant difference between males and females for PFHxS, PFOS, PFOA, and PFNA, and the difference was larger in younger people. For example, 30-year-old males had higher blood PFHxS, PFOS, PFOA, and PFNA levels than 30-year-old females by 94.5%, 87.5%, 49.6%, and 34.9%, respectively. For 50-year-old males, this difference was reduced to 34.6% for PFHxS, 48.9% for PFOS, 18.9% for PFOA, and 17.5% for PFNA compared to 50-year-old females.

How to read a box and whisker plot: A box and whisker plot illustrates a summary of the data using different statistical measures. See the image below for how to interpret the figures throughout this report.

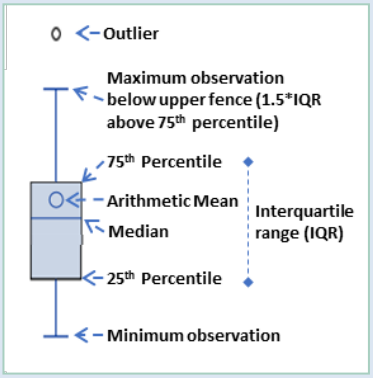
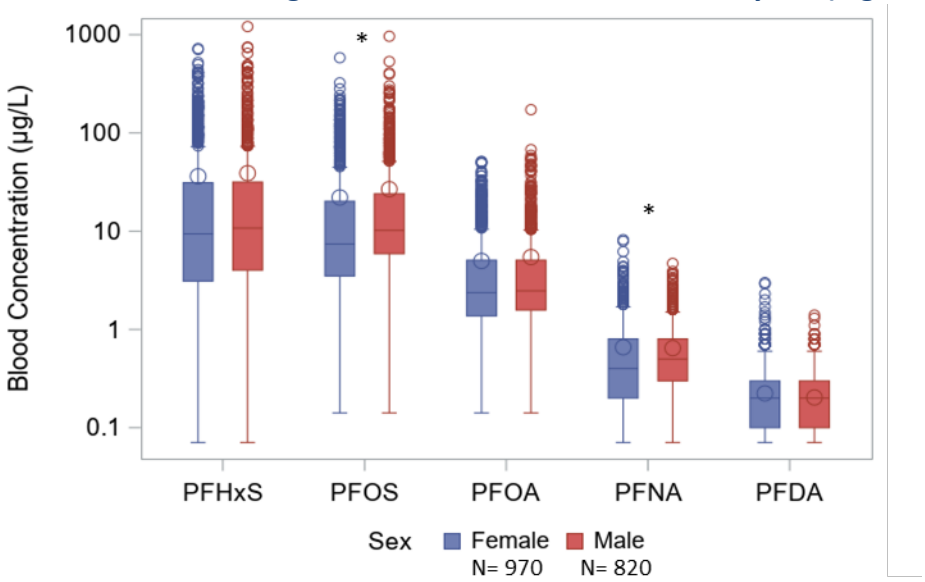


Figure 7. PFAS blood level in adults by sex (log scale)

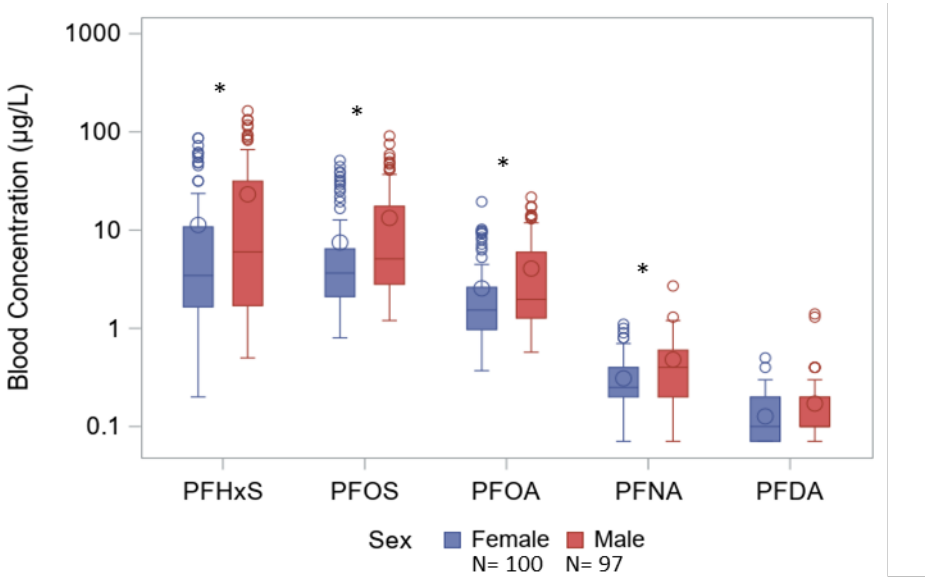


See 'How to read a box and whisker plot' earlier in the PFAS in Blood section.
A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant difference (p<0.05) in univariate regressions.*
Note: Sex was statistically significant in multivariate regressions for PFHxS, PFOS, PFOA, and PFNA.

Figure 8 shows that in children, PFAS blood levels were also elevated in males compared to females. Specifically, blood levels in male children were 67% higher for PFHxS, 69% higher for PFOS, 45% higher for PFOA, and 59% higher for PFNA. In multivariate models, sex continued to be statistically associated with PFOS, PFOA, and PFNA blood levels, while associations with PFHxS were no longer significant. Multivariate models also showed a significant relationship with PFDA blood levels. Specifically, blood l evels in male children were higher than female children for PFOS by 26%, PFOA by 23%, PFNA by 44%, and PFDA by 14%.

Sex-based differences have been observed in other studies in communities with contaminated drinking water and for the general U.S. population [e.g., ATSDR 2013; NH DPHS 2016; CDC 2019]. Sex-based differences are likely due to additional excretion routes in females including through menstrual fluid, breastfeeding, pregnancy, and renal clearance rate differences [ATSDR 2021a]. Some of these factors could contribute to the differences seen in children since children could be as old as 17.

Figure 8. PFAS blood level in children by sex (log scale)



See 'How to read a box and whisker plot' earlier in the PFAS in Blood section.
A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant difference (p<0.05) in univariate regressions.*
Note: Sex was statistically significant in multivariate regressions for PFOS, PFOA, PFNA, and PFDA are significant in multivariate regressions.

Blood PFAS Levels and Tap Water Consumption

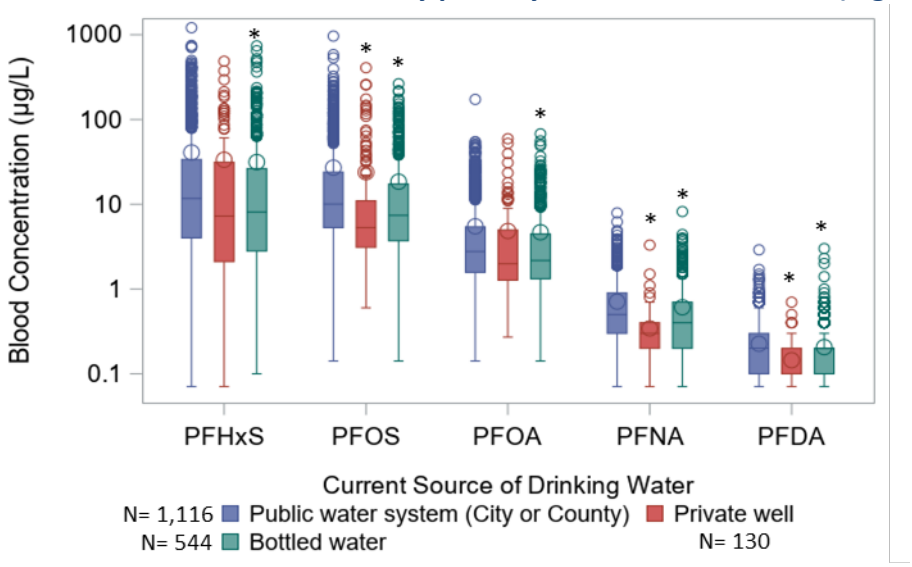
ATSDR conducted these EAs to learn more about how exposure to PFAS-contaminated drinking water affects blood PFAS levels. This relationship is complicated because EA participants were likely exposed to PFAS not only in contaminated drinking water but also in various consumer products and food items unrelated to the water.

ATSDR investigated several questions from the adult and child questionnaires to characterize relationships between blood PFAS levels and consumption of PFAS-contaminated drinking water. These questions are about the drinking water source, amount of tap water consumed at home or school, and residential history. In some cases, data trends may have been affected by subtleties in the wording of exposure history questions. Notably, these questions pertained to current drinking water practices. It is uncertain whether these responses would have applied to past drinking water practices. Despite this limitation, at individual sites, PFAS levels were often associated with at least one exposure characteristics related to tap water consumption.

Drinking water source. For adults, ATSDR first considered participants' primary drinking water source. Adult participants were asked, "What is your current main source of drinking water in your home?" At five of eight sites, water source was associated with blood PFAS levels after controlling for other variables. In the combined dataset, 62% of responses were public water system, 30% were bottled water, and 7.3% were private well. Those who identified as primarily drinking from private wells were from either the Lubbock, TX or Moose Creek, AK EAs. All other EAs consisted of sites with contaminated public water systems or 'tap water'. Those who indicated bottled water as their current primary source of drinking water consisted of participants from all eight EAs.

Among PFAS analyzed in blood, all PFAS were statistically associated with current main drinking source in adults. Compared to adults who reported drinking primarily from tap water, adults who reported drinking mainly bottled water had blood levels that were 18% lower for PFHxS, 20% lower for PFOS, 15% lower for PFOA, 16% lower for PFNA, and 13% lower for PFDA (Figure 9). In multivariate models, when controlling for other variables, these associations were no longer statistically significant. Adults who reported drinking primarily from a private well had lower blood levels for PFOS (32%), PFNA (50%), and PFDA (34%) in univariate analyses. However, in multivariate models, the association with blood PFOS was reversed, and adults who primarily reported drinking from private wells at home had blood levels that were 111% higher than those who reported drinking from the public water system. Adults who reported drinking primarily from private wells also had 42% higher blood PFOA levels, 44% lower PFNA levels, and 29% lower PFDA levels. Multivariate models stratified by sex showed that these relationships remained consistent in both adult males and females.

Figure 9. PFAS blood level in adults by primary current water source (log scale)



See 'How to read a box and whisker plot' earlier in the PFAS in Blood section.
A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant difference (p<0.05) in univariate regressions with "Public water system".*
Note: Current Source of Drinking Water was statistically significant in multivariate regressions for PFOS, PFOA, PFNA, and PFDA

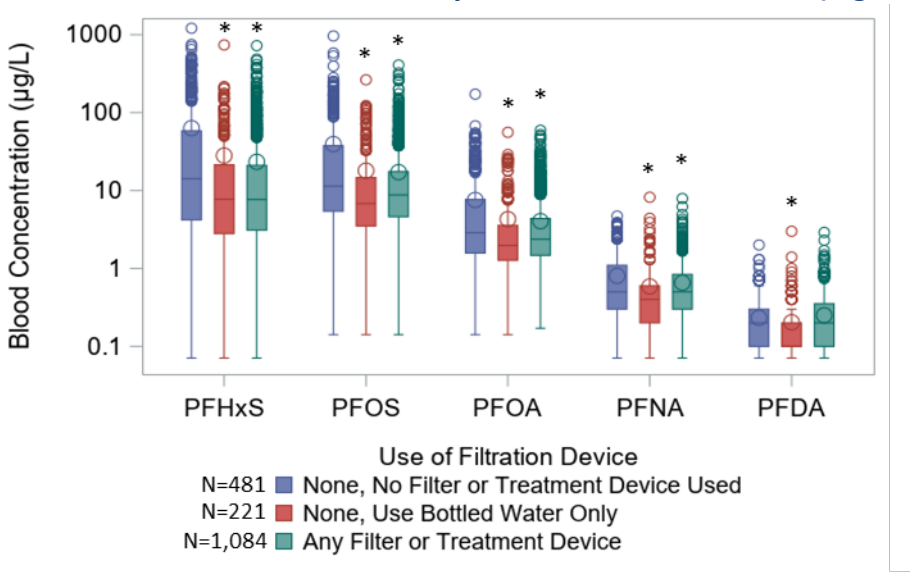
The lack of significant differences in multivariate models between bottled water drinkers and tap water drinkers was an unexpected result, which ATSDR believes may be a result of how the question was worded—particularly the word "current." ATSDR also asked participants about any changes to their drinking water habits in the past year. However, since drinking water exposure was generally mitigated over a year prior to when the EA survey was conducted, changes in drinking water behavior within the past year would not affect drinking water exposure. It is possible that participants who reported currently drinking bottled water in the past year drank tap water during the period of contamination, but the extent to which that occurred is not known. In contrast, the statistical differences in blood PFAS levels between those whose primary source of drinking water is private wells compared to those whose source is a public water system may simply reflect differences in the drinking water levels in the two EA sites with private well contamination compared to the other sites. Due to these considerations, ATSDR's

data analysis did not rely heavily on answers to this question when interpreting associations between PFAS levels and exposure characteristics.

Use of filtration device. In adults, ATSDR also considered relationships between blood PFAS levels and current use of drinking water filtering devices and water treatment devices. At four of eight sites, use of a filtration device was statistically associated with PFAS blood levels after controlling for other variables. As Figure 10 shows, 61% of adult participants reported using a filter or treatment device on the tap water that they drink at home, 27% of adult participants reported no filter or treatment device on the tap water that they drink at home, and 12% of adult participants reported not drinking tap water at home at all. In ATSDR's univariate analyses, adult participants who reported using any filter or treatment device on the tap water that they drink at home on average had statistically lower blood levels of PFHxS (34%), PFOS (33%), PFOA (21%), and PFNA (14%) than those who reported drinking tap water without any filter or treatment device. Similarly, adult participants who reported not drinking tap water at all (i.e., only reported drinking bottled water) on average had statistically lower blood levels of PFHxS (51%), PFOS (53%), PFOA (38%), PFNA (34%), and PFDA (28%) than adult participants who reported drinking tap water without any filter or treatment device.

These results remained statistically significant in multivariate models. In all-adult multivariate models, participants who reported using a filter or treatment device on average had blood PFAS levels that were 28% lower for PFHxS, 25% lower for PFOS, and 19% lower for PFOA. Similarly, adult participants who reported not drinking tap water at all (i.e., only reported drinking bottled water) on average had statistically lower blood levels of PFHxS (45%), PFOS (36%), and PFOA (35%) than participants who reported drinking tap water without any filter or treatment device. Multivariate models stratified by sex revealed that, with the exception of models for PFHxS, which remained significant in the female-only models, the associations between water source and other PFAS were not significant.

Figure 10. PFAS blood level in adults by filter or treatment device (log scale)

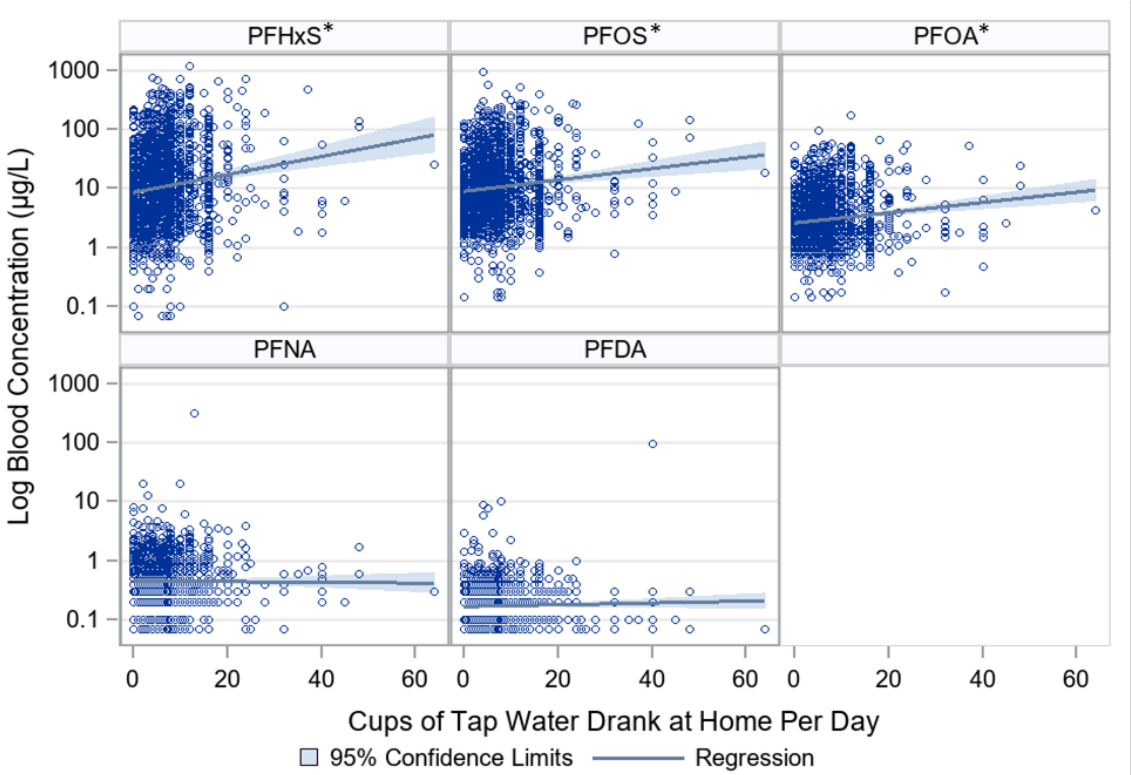


See 'How to read a box and whisker plot' earlier in the PFAS in Blood section.
A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant difference (p<0.05) in univariate regressions with "None, no Filter or Treatment device Used".*
Note: Current Source of Drinking Water was statistically significant in multivariate regressions for PFHxS, PFOS, and PFOA.

Consumption rates. ATSDR also considered participants' self-reported tap water consumption rates. Adult participants were asked, "During the time you [or your child] lived in a home served by the water source identified above, on average how many 8-ounce cups of water or beverages prepared with tap water did you drink while at home per day?" At three of eight sites, drinking water consumption rates were statistically associated with PFAS after controlling for other variables. ATSDR's univariate analyses revealed a statistically significant relationship between blood PFAS levels in adults and the amount of tap water consumed (Figure 11). For every additional cup of tap water an adult reported drinking at home per day, PFHxS, PFOS, and PFOA blood levels increased by 2.5%, 1.2%, and 1.4%. In multivariate models, only the relationship with PFHxS remained significant and for every cup of water an adult reported drinking, blood PFHxS levels increased by 2%. Multivariate models stratified by sex revealed that this relationship was primarily observed in males.

As can be seen in Figure 11, a subset of participants reported consumption rates that fall above the higher end values (95th percentile) reported in EPA's Exposure Factors Handbook of 3,292 milliliters per day (approximately 14 cups) [EPA 2019]. This relatively small percentage of participants may have overestimated their drinking water consumption, but this is not expected to alter conclusions.

Figure 11. PFAS blood level in adults by tap water consumption at home (log scale)



A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant trend (p<0.05) in univariate regressions.*
Note: Tap Water Consumption at Home was statistically significant in multivariate regressions for PFHxS and PFOS.

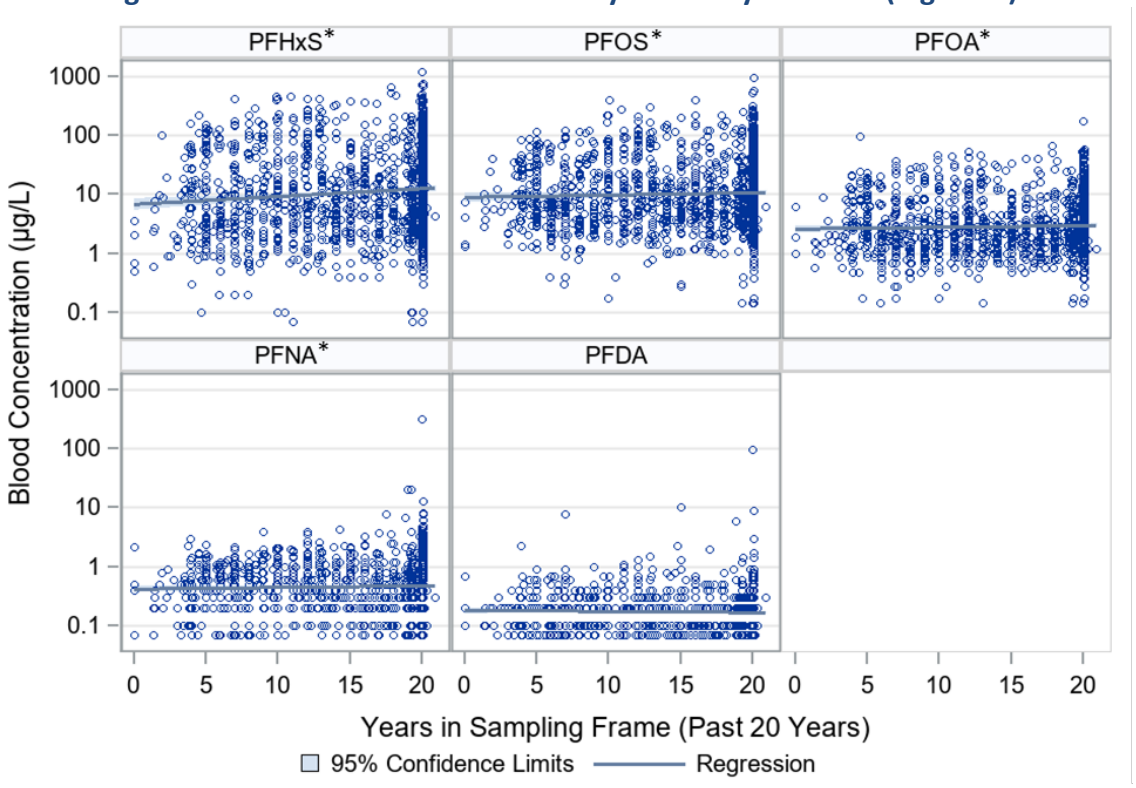
In children, the amount of tap water drank at home was not statistically significant in univariate models. In multivariate models, the number of cups of water a child (or his or her parents) reported drinking at home was only statistically associated with blood PFNA levels. After controlling for other variables, every additional cup of water drank at home was associated with a 3% reduction in blood PFNA levels.

Length of residency. For adults, ATSDR also considered the length of residency. The exposure history questionnaire asked adults where they had lived for the past 20 years. ATSDR calculated the total amount of time participants reported living in the sampling frame over this period. These responses can serve as a proxy for potential exposure to PFAS-contaminated drinking water in the community. That is, the longer the residency within the sampling frame, the greater the likelihood of past PFAS exposure from contaminated drinking water. At seven of eight sites, length of residency duration was statistically associated with blood PFAS levels after controlling for other variables.

Figure 12 shows the relationship between reported residence duration in the sampling frame for the past 20 years and blood PFAS levels. A consistent statistical relationship was observed where blood levels increased with the number of years participants lived in the sampling frame for PFHxS (7.0% per year), PFOS (4.3% per year), PFOA (2.2% per year), and PFNA (1.8% per year). The multivariate analysis showed PFHxS, PFOS, and PFOA still had significant relationships with residency duration when controlling for other variables. For every additional year that an adult participant lived in the sampling

frame, blood PFHxS increased by 6.0%, blood PFOS increased by 3.2%, and blood PFOA by 2.3%. These associations were significant in both male-only and female-only models for all three compounds.

Figure 12. PFAS blood level in adults by residency duration (log scale)



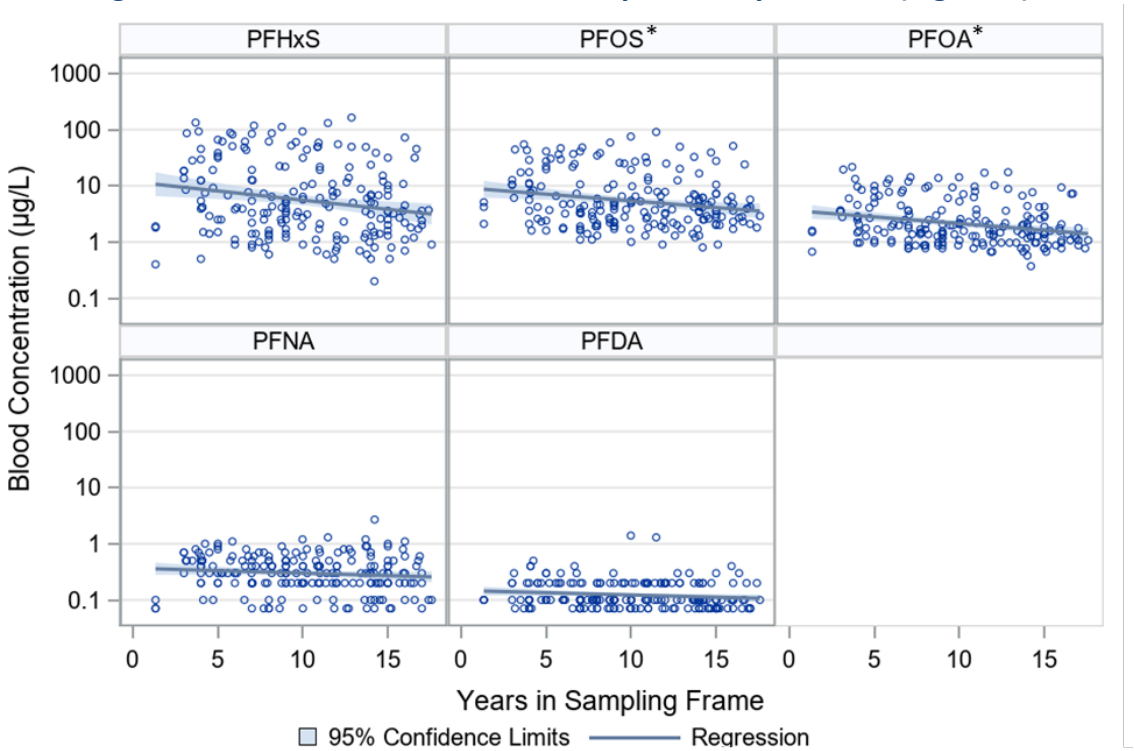
A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant trend ($p<0.05$) in univariate regressions.*
Note: Years in Sampling Frame was statistically significant in multivariate regressions for PFHxS, PFOS, and PFOA.

In children, the length of residency duration was also associated with blood levels of PFOS and PFOA, but the association was in the opposite direction as expected (Figure 13). For every additional year a child lived within the sampling frame, blood PFOS levels decreased by 4.6% and blood PFOA levels decreased by 5.1%. This initial negative association is likely due to the correlation between residency duration and age in children—older children lived in the sampling frame longer—and the negative association between PFAS blood levels and age in children. After controlling for age and other variables, multivariate analyses showed that for every additional year a child reported living in the sampling frame, there was a 10% increase in blood PFHxS levels and a 4.2% increase in blood PFOA levels.

ATSDR found the length of residency in a site's sampling frame was one of the most consistent predictors of blood PFAS levels. ATSDR considered residency duration to be a suitable surrogate for drinking water exposures because only residents who lived in the sampling frame before each system's eligibility date would have had any exposure to the PFAS-contaminated drinking water, and because of the likelihood that exposure would increase with the number of years that EA participants lived in the area. However, since older adults tended to live in the sampling frame longer, this variable was often correlated with age in adults. Because of this, it was unclear from univariate models alone whether the

association between the time someone lived in the sampling frame and PFAS blood levels was primarily due to age. After controlling for other variables, the multivariate statistical analysis found that residency duration remained a strong predictor of blood PFHxS, PFOS, and PFOA levels, providing further evidence of a drinking water exposure source.

Figure 13. PFAS blood level in children by residency duration (log scale)

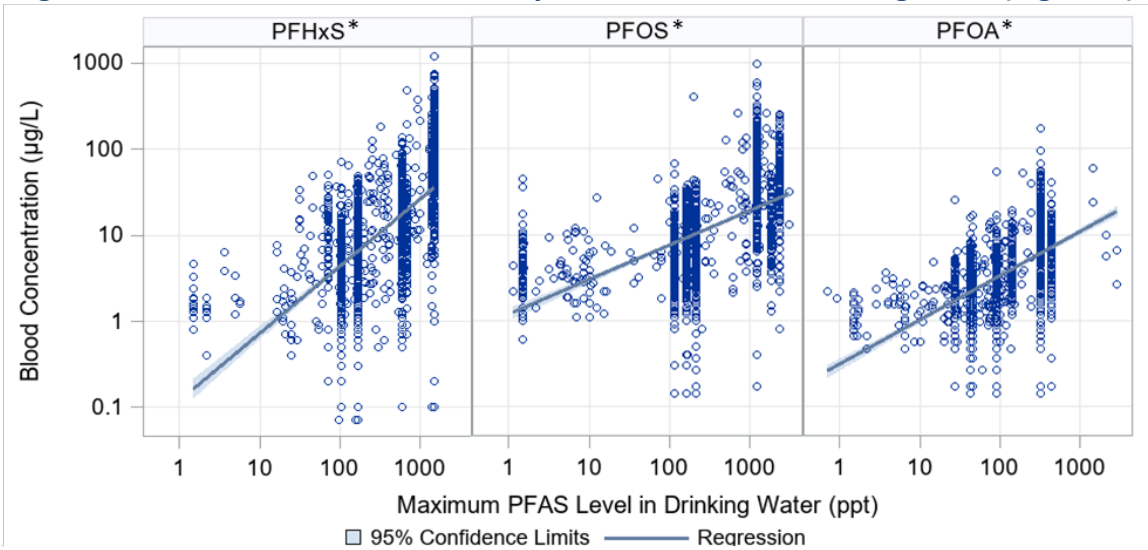


A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant trend ($p<0.05$) in univariate regressions.*
Note: Years in Sampling Frame was statistically significant in multivariate regressions for PFHxS and PFOA.

Drinking water testing data. ATSDR also considered historic drinking water testing data obtained from each site. Maximum values were obtained from public water systems or private well testing data as described above. This analysis was only conducted for PFHxS, PFOS, and PFOA.

In univariate models for adults, the $\log_{10}$ of maximum PFHxS, PFOS, and PFOA drinking water concentrations were statistically associated with corresponding blood PFAS levels (Figure 14). Comparisons were made only between like PFAS. For example, the effect of PFHxS drinking water concentrations were only compared with blood PFHxS levels. For each 1% increase in maximum PFHxS drinking water concentration, blood PFHxS levels increased on average by 0.56%. For each 1% increase in maximum PFOS drinking water concentration, blood PFOS levels increased on average by 0.46%. For each 1% increase in maximum PFOA drinking water concentration, blood PFOA levels increased on average by 0.48%. These associations remained statistically significant in the multivariate analyses where for each 1% increase in maximum PFHxS, PFOS, and PFOA drinking water concentration, there was a corresponding increase in blood PFHxS (0.6%), PFOS (0.5%), and PFOA (0.5%), respectively. Multivariate models stratified by sex show that this association was observed in both males and females.

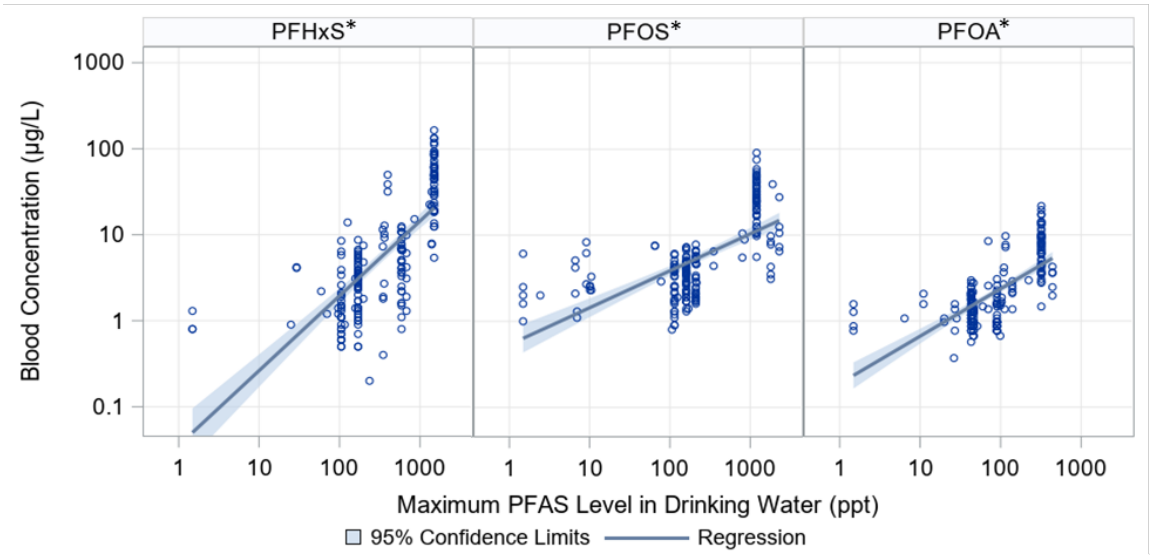
Figure 14. PFAS blood levels in adults by maximum PFAS in drinking water (log scale)



A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant trend ($p<0.05$) in univariate regressions.*
Note: Maximum PFAS Level in Drinking Water was statistically significant in multivariate regressions for PFHxS, PFOS, and PFOA.

In univariate models for children, the $\log_{10}$ of maximum PFHxS, PFOS, and PFOA drinking water concentrations were statistically associated with corresponding blood PFAS levels (Figure 15). For each 1% increase in maximum PFHxS, PFOS, and PFOA drinking water concentrations, there was a corresponding increase in blood PFHxS (0.89%), PFOS (0.51%), and PFOA (0.60%) levels. These associations remained significant in multivariate models where for each 1% increase in maximum PFHxS, PFOS, and PFOA drinking water concentration, the corresponding blood levels increased on average by 0.9%, 0.5%, and 0.5%, respectively.

Figure 15. PFAS blood levels in children by maximum PFAS in drinking water (log scale)



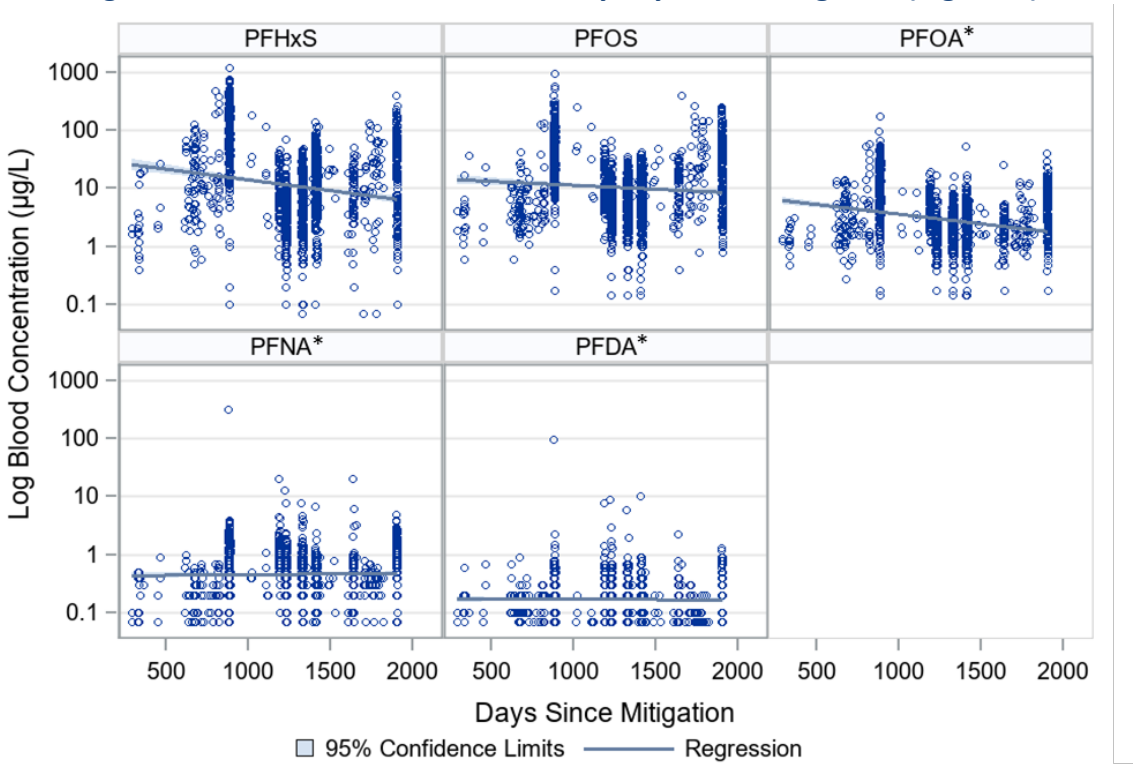
A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant trend (p<0.05) in univariate regressions.*
Note: Maximum PFAS Level in Drinking Water was statistically significant in multivariate regressions for PFHxS, PFOS, and PFOA.

At most sites, all three of these PFAS (PFHxS, PFOS, and PFOA) were detected in the drinking water supplies, and these compounds were also highly correlated in blood measurements. Therefore, one explanation for the high correlation among these compounds in the blood is that the EA participants had a common exposure profile for PFHxS, PFOS, and PFOA, such as drinking water. However, the correlations alone cannot be used to identify the underlying source or combination of sources that contributed most to exposure.

Days Since Mitigation. ATSDR also considered the time between when participants provided biological samples during the EA and the final mitigation dates for each site. This value was calculated as previously described for both public water system and private well sites.

In univariate analyses for adults, the greater the number of days that had passed since the drinking water in a household had been mitigated, the statistically lower the blood PFOA levels (0.042% per day), and the higher the blood PFNA (0.027% per day) and PFDA blood levels (0.025% per day). After controlling for other variables with multivariate analyses, the number of days since mitigation was statistically related to a decrease in blood PFOS (0.03% per day) and PFOA (0.05% per day) levels, and an increase blood PFDA (0.02% per day) levels in adults. Multivariate models stratified by sex show that this association was observed in both males and females for PFOS and PFOA, and only in males for PFDA.

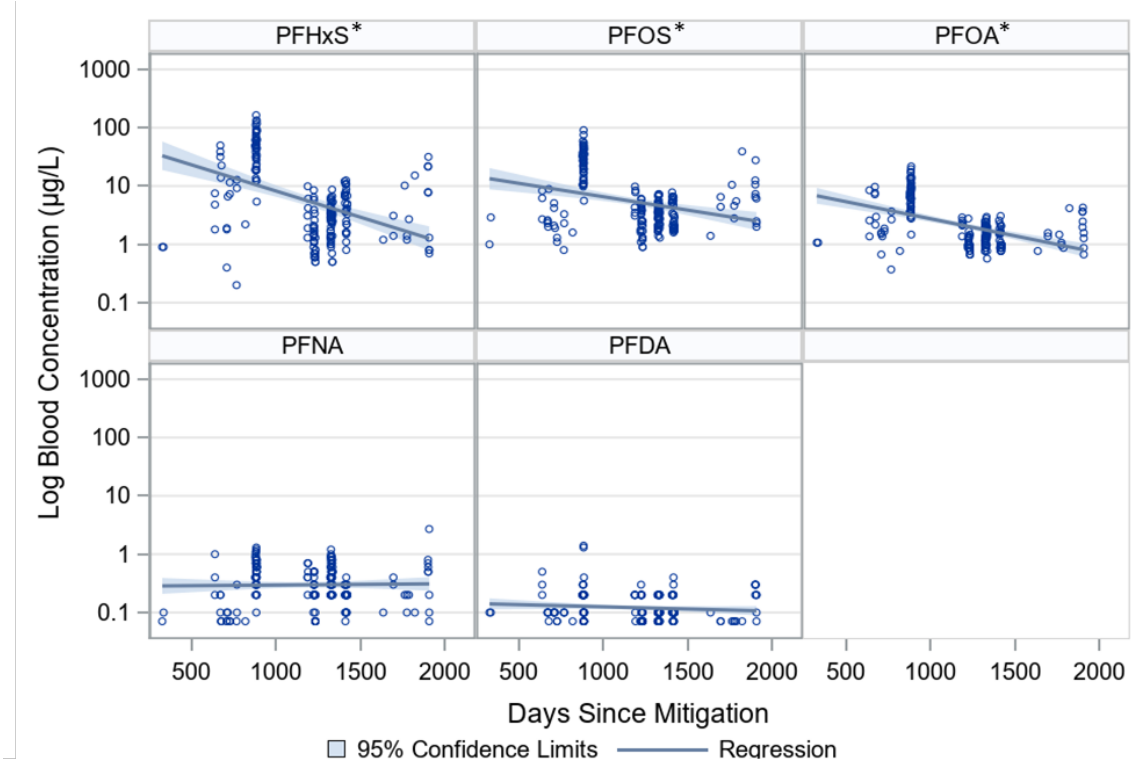
Figure 16. PFAS blood levels in adults by days since mitigation (log scale)



A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant trend ($p<0.05$) in univariate regressions.*
Note: Maximum PFAS Level in Drinking Water was statistically significant in multivariate regressions for PFOS, PFOA, and PFDA.

In children (Figure 17) the number of days since mitigation was associated with decreased levels of PFHxS (0.2% per day), PFOS (0.1% per day), and PFOA (0.1% per day). These associations remained significant in multivariate analyses after controlling for other variables. For each day that passed since mitigation, blood PFHxS, PFOS, and PFOA levels decreased by 0.1%.

Figure 17. PFAS blood levels in children by days since mitigation (log scale)



A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant trend ($p<0.05$) in univariate regressions.*
Note: Maximum PFAS Level in Drinking Water was statistically significant in multivariate regressions for PFHxS, PFOS, and PFOA.

Finally, an exposure history question pertained to whether adult participants drank tap water while at work. However, because identifying whether a participant's place of employment was in the sampling frame was difficult, ATSDR did not evaluate the data for drinking water consumption patterns at work.

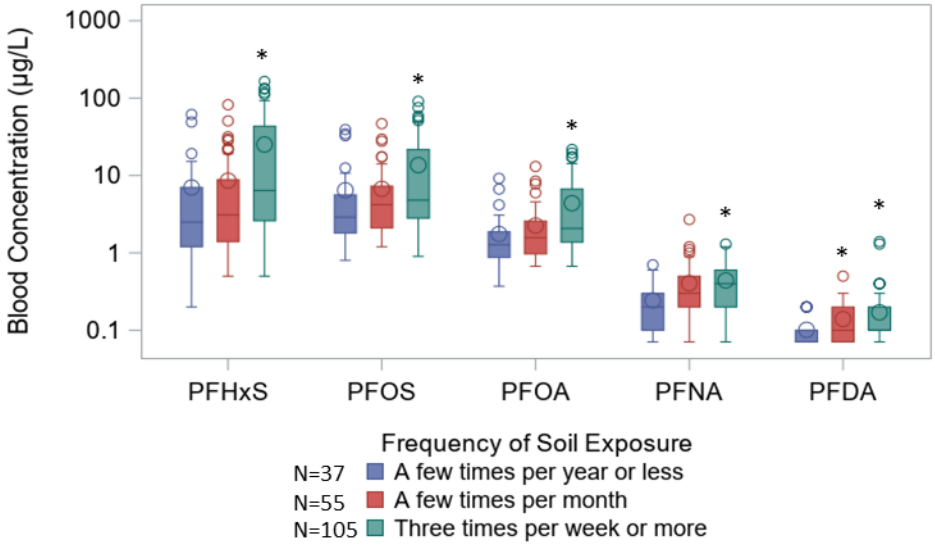
Blood PFAS Levels and Soil Exposure

Adult and child participants were asked how often they play in or touch soil or dirt in the sampling frame. At two of eight sites, soil exposure was statistically associated with blood PFAS levels in adults after controlling for other variables. However, in the combined dataset no statistically significant relationship was observed for self-reported soil contact frequency and blood PFAS levels among adults in multivariate models.

In univariate models for children, participants who reported coming in contact with soil three times a week or more had higher blood PFHxS (173%), PFOS (74%), PFOA (77%), PFNA (67%), and PFDA (35%) l evels than those who reported coming in contact with soil a few times per years or less (Figure 18). Children who reported coming in contact with soil a few times per month had 24% higher blood PFDA levels. In multivariate models for children, the association with soil contact and blood PFOS, PFNA, and PFDA remained significant. Children who reported coming in contact with soil three times a week or more had 41% higher blood PFOS levels, 49% higher blood PFNA levels, and 23% higher blood PFDA levels than those who reported coming in contact with soil a few times per year or less. Similarly,

children who reported coming in contact with soil a few times per month had 40% higher blood PFOS levels, and 21% higher blood PFDA levels.

Figure 18. PFAS blood level in children by frequency of soil exposure (log scale)



See 'How to read a box and whisker plot' earlier in the PFAS in Blood section.
A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant difference (p<0.05) in univariate regressions with "A few times per year or less".*
Note: Frequency of Soil Exposure was statistically significant in multivariate regressions for PFOS, PFNA, and PFDA.

Blood PFAS Levels and Consumption of Selected Local Food Items

Some PFAS accumulate in plants, fish, and animals. The questionnaire asked adult and child EA participants how often they consume locally grown fruits and vegetables, locally caught fish, and milk from animals in the sampling frame. Blood PFHxS, PFOS, and PFOA levels were not associated with consumption of locally caught fish, or locally grown fruits and vegetables, but were associated with consumption of locally produced milk.

At no individual sites was consumption of locally produced milk statistically associated with blood PFAS levels. In the combined data set, blood PFAS levels were not associated with consumption of locally produced milk in univariate analyses for adults. However, in multivariate analyses for adults, blood PFHxS levels were higher by 78% and blood PFOA by 38% among adults who reported ever drinking locally produced milk compared to participants who reported never drinking locally produced milk.

In children, univariate models indicated that participants who reported ever consuming locally produced milk had 41% lower blood PFOS levels, 41% lower blood PFOA levels, and 23% lower blood PFNA levels than those who never reported having consumed locally produced milk. Since only 13 children reported ever consuming locally produced milk, these results should be interpreted with caution.

Blood PFAS Levels and Childbirth (adult females and children only)

PFAS have been demonstrated to pass through the placental barrier and into the developing fetus during gestation, and have been measured in maternal serum, cord blood, breast milk [Cariou et al. 2015], placenta [Chen et al. 2017], fetal tissue [Mamsen et al. 2019], and neonates [Wang et al. 2014]. These studies suggest gestation, birth, and breastfeeding as excretion pathways for mothers and gestation and breastfeeding as potential exposure pathways for infants. The adult questionnaire asked female participants whether they had any biological children, and if so, how many. At one of eight sites having biological children (yes/no) was statistically associated with blood PFAS levels after controlling for other variables. At a different site, the number of biological children ("continuous") was statistically associated with blood PFAS levels. The child questionnaire asked participants their birth order. Most female adults (79%) reported having biological children. Having children (yes/no) was not statistically associated with blood PFAS levels in univariate models. However, in multivariate models, female adults who reported having children had 24% lower blood PFHxS levels than females who did not have children. In contrast, the number of children was not statistically associated with blood PFAS levels in multivariate models.

In children, the birth order was not statistically associated with PFAS levels in multivariate models.

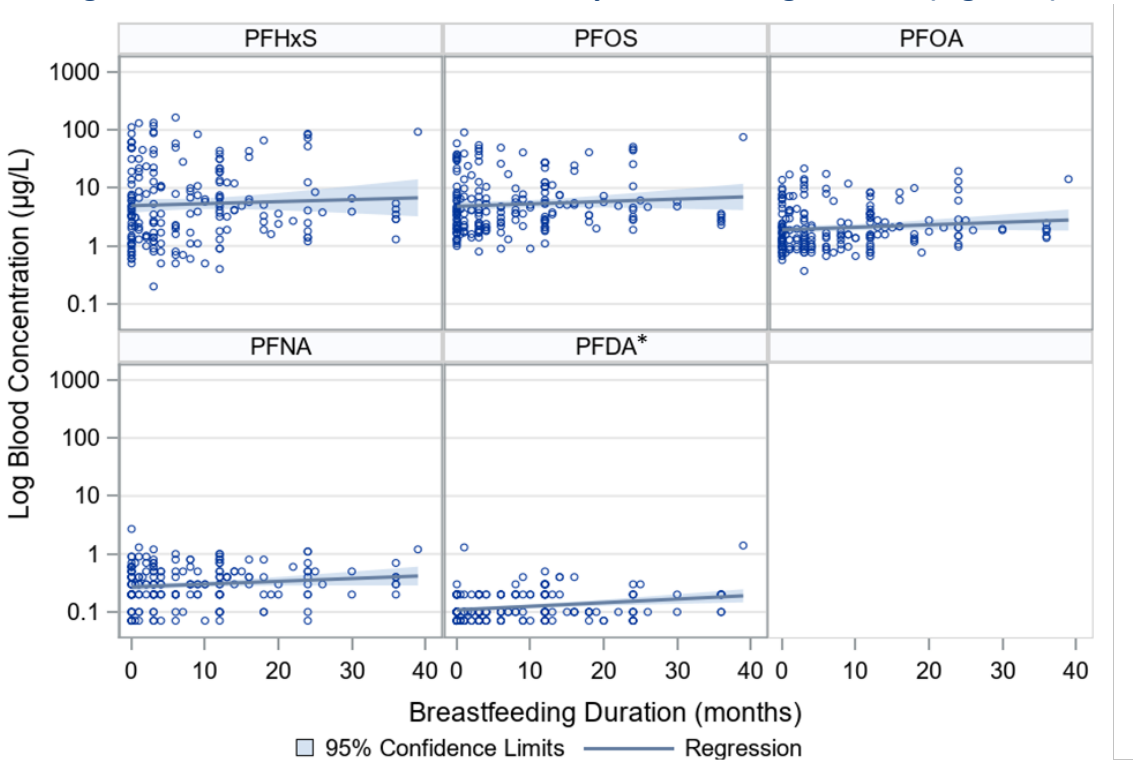
Blood PFAS Levels and Breastfeeding

During breastfeeding, some PFAS in the breast milk might be transferred from mother to child. Therefore, breastfeeding might reduce PFAS levels in mothers and increase PFAS levels in their breastfed children [Kim 2020; Kingsley 2018]. Accordingly, the adult and child exposure history questionnaires included questions about breastfeeding. A question was also included for children about their consumption of formula and if the formula was made using tap water.

Among adult female participants across EAs, 52% reported that they had breastfed a child. Neither having ever breastfed a child (yes/no) nor the total duration of breastfeeding was associated with PFAS blood levels in multivariate models.

Among children, 76% of participants in the EA were breastfed. Having ever breastfed or not (yes/no) was statistically associated with blood PFDA levels in children, but no other PFAS. Children who reported being breastfed had blood PFDA levels that were 26% higher than children who did not. In addition, each month of reported breastfeeding was associated with an increase of 1.3% in blood PFDA levels in univariate models. This association remained statistically significant in multivariate models where every additional month of reported breastfeeding led to an increase in blood PFDA levels of 1.1%.

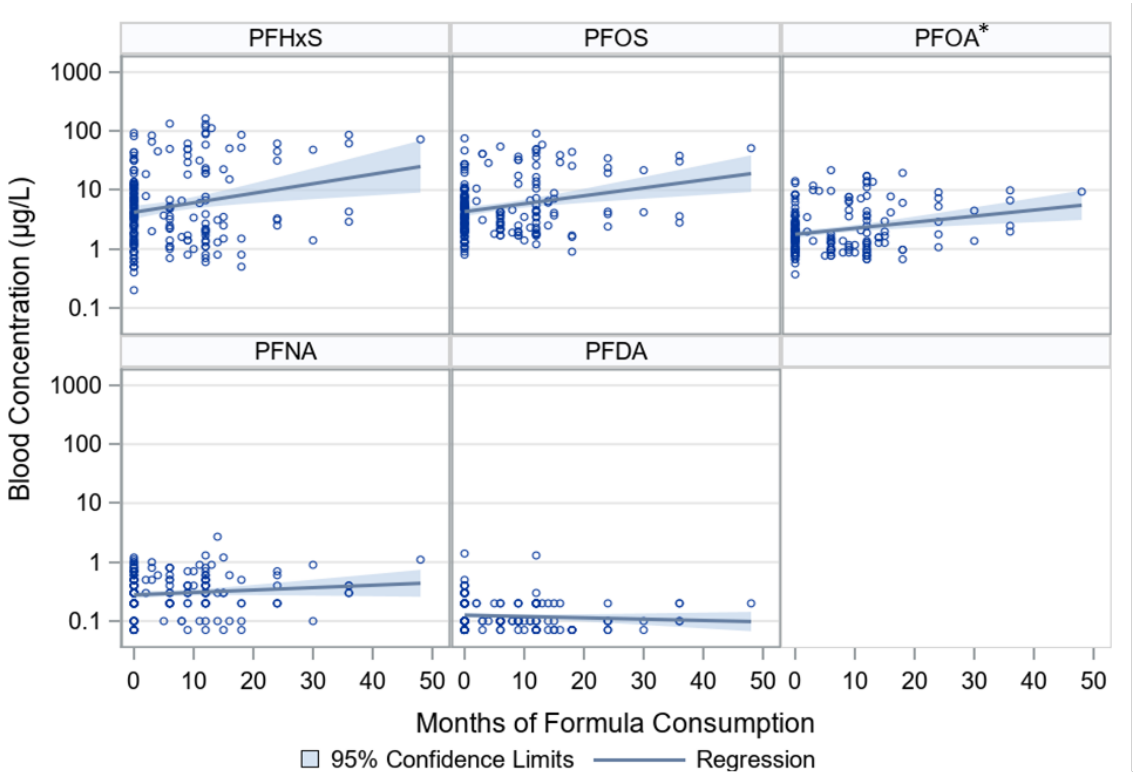
Figure 19. PFAS blood level in children by breastfeeding duration (log scale)



A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant trend ($p<0.05$) in univariate regressions.*
Note: Breastfeeding Duration was statistically significant in multivariate regressions for PFDA.

Approximately half of the children in the EA (48%) consumed infant formula reconstituted with tap water (some of these children were also breastfed). In univariate analyses, for every additional month a child consumed infant formula, blood PFOA levels increased by 1.9%. However, in multivariate models, formula consumption was only associated with blood PFNA levels. Specifically, for every additional month of formula consumption blood PFNA increased by 1.3%.

Figure 20. PFAS blood level in children by formula duration (log scale)



A log10 scale is used to allow easier visualization of the wide range of measured blood levels, as it uses equal spacing for each factor of 10 increase.
**Statistically significant trend ($p<0.05$) in univariate regressions.*
Note: Tap Water Consumption at School was statistically significant in multivariate regressions for PFNA.

Blood PFAS Levels and Other Variables

Through the exposure history questionnaires, ATSDR gathered information on several other behaviors and possible contributing factors to PFAS exposures. The variables listed below were not statistically associated with blood levels of PFHxS, PFOA, and PFOS among EA participants in multivariate analyses. In some cases, statistical associations were seen for blood levels of PFNA or PFDA.

- **Race/Ethnicity.** Adult and child participants were asked to provide information about their race and ethnicity. After controlling for other variables, race/ethnicity was not a significant predictor of blood PFAS levels at any of the eight sites. However, there were not enough participants in different race and ethnicity categories to support robust statistical analyses at individual sites. Here, ATSDR conducted a more detailed analysis of race and ethnicity. Race and ethnicity were only statistically associated with blood PFNA levels in multivariate models for adults and children. Compared to those who identified as "White, non-Hispanic", some groups had higher PFNA blood levels, while others had lower PFNA levels.
- **Blood donation frequency.** Adult participants were asked how often they donate blood or plasma, because frequent blood and plasma donations might result in decreasing blood PFAS levels. At three of eight sites, blood donation was statistically associated with decreased blood PFAS levels after controlling for other variables. However, blood donation was not significant in multivariate models in adults for the combined dataset.

- **Cleaning Frequency.** Adult participants were asked about the frequency at which they clean their homes. At two of eight sites, cleaning frequency was statistically associated with blood PFAS levels after controlling for other variables. At one site, increased cleaning frequency was associated with increased blood PFAS levels, while at the other site cleaning was associated with decreased blood PFAS levels. In multivariate analyses, in the combined dataset, adults who reported cleaning their homes more frequently had statistically higher PFNA blood levels.
- **Flooring.** Adult participants were asked about the type of flooring in their living rooms, kitchens, and bedrooms. Carpet has been linked to increased PFAS exposure because PFAS-containing stain- and grease-repelling coatings are often applied to carpet [Beesoon et al. 2012]. At one of eight sites, carpet in any room in the house was associated with increased PFAS blood levels after controlling for other variables. However, the presence of carpet in EA participants' homes was not statistically associated with blood PFAS levels in the combined dataset.
- **Locally grown produce.** In multivariate analyses in adults, only blood PFDA was statistically associated with consumption of locally grown fruits and vegetables. Adults who reported eating locally grown fruits and vegetables three times per week or more had 23% higher blood PFDA levels, and those who reported eating locally grown fruits and vegetables a few times per month had 21% higher blood levels than those who did not. Multivariate models stratified by sex showed that this association was primarily observed in males.
- **Stain-resistant product use.** Many stain-resistant products used to treat fabrics and carpet have been formulated with PFAS. The exposure history questionnaire asked adult EA participants how frequently they used these products, because such uses may be associated with PFAS exposures. At one of eight sites, use of stain-resistant products was statistically associated with increased blood PFAS levels after controlling for other variables. In multivariate models for the combined dataset, stain-resistant product use was only associated with elevated blood PFNA levels.
- **Fast food consumption.** PFAS may be present in fast food take-away containers and food packaging. Consumption of fast food may serve as an additional source of PFAS exposure. However, among EA participants, reported frequency of fast food consumption was not statistically associated with blood PFAS levels in multivariate models. In recent years, fast food packaging has likely been reformulated to contain shorter chain PFAS compounds. This shift may make it more challenging to link the longer chain PFAS exposure measured in the EAs to fast food consumption.
- **Kidney disease.** The exposure history questionnaire asked about kidney disease because it can affect blood PFAS levels [Barry et al. 2013; Watkins 2013]. At three of eight sites, kidney disease was statistically associated with blood PFAS levels after controlling for other variables. At one site, kidney disease was associated with increased PFHxS levels; at two other sites, kidney disease was associated with decreased PFAS levels (i.e., PFHxS for one site and PFOS for the other). However, kidney disease was not associated with PFAS blood levels in the combined dataset. Note also that kidney disease was self-reported and there may be misclassification with this variable.
- **Occupational exposure.** Workers can be exposed to PFAS through job tasks that involve manufacturing or working with PFAS. Adult participants were asked about their occupational history over the past 20 years. At one of the eight sites, reported exposure to PFAS at work was statistically associated with increased blood PFAS levels after controlling for other variables. At a different site, exposure at work was statistically associated with decreased blood PFAS.

However, occupational exposure to PFAS was not significant in multivariate models in adults for the combined dataset.

PFAS in Urine at ATSDR-led EAs

The EA protocol called for ATSDR to initially analyze 10% of the urine samples collected at a given site. The protocol indicated that ATSDR would analyze all participants' urine samples if initial analyses showed geometric mean urine concentrations of any PFAS to be greater than the NHANES 95th percentile values. Previous work indicated that PFAS are not often detected in urine. A 10% sample was included here to verify if PFAS are present in urine for residents in areas with PFAS in drinking water. For the purposes of this comparison, ATSDR relied on the most recently published PFAS urine data from the 2013-2014 NHANES. Of the 17 PFAS measured in the 2,682 urine samples included in this NHANES data set, only PFBA and PFHxA were detected in more than 1.5% of the NHANES urine samples [Calafat et al. 2019].

Across the EA sites, ATSDR randomly selected 206 participants' urine samples for analysis. Two people were later determined to have not met the EA's eligibility criteria and results from their samples were excluded. The urine samples summarized below were provided by 189 adults and 15 children, and these individuals lived in 192 unique households. Since no PFAS were detected in more than 60% of the analyzed samples for a given site, geometric means were not calculated for any PFAS in urine and ATSDR did not analyze the remainder of the urine samples.

PFHxS, PFHxA, and PFBA, were the only PFAS detected in any of the 204 urine samples analyzed from eligible participants.

- PFHxS was detected in nine of the urine samples (4%) that were analyzed from EA participants and only among individuals from the Airway Heights, WA EA. Detected concentrations ranged from 0.1 µg/L to 0.4 µg/L. Participants from this EA site also had the highest average PFHxS concentrations measured in blood across all sites and among the most recent exposures to PFAS in drinking water prior to mitigation of the sites evaluated. Geometric mean comparison values were not available from NHANES for this PFAS since PFHxS was detected in <0.1% of national samples [Calafat et al. 2019]. The NHANES 95th percentile for PFHxS is below the limit of detection. Note that PFHxS is not generally found in urine [Calafat et al., 2019].
- PFHxA was detected in two of the urine samples (1%) that were analyzed and only among participants from Lubbock, TX. Geometric mean comparison values were not available from NHANES for this PFAS since PFHxA was only detected in 23% of samples [Calafat et al. 2019]. Like PFHxS, the NHANES 95th percentile for PFHxA is below the limit of detection. PFHxA was not measured in the blood of EA participants.
- PFBA was detected in 79 of the urine samples (38%) that were analyzed. This PFAS was measured in participants from six different sites and at concentrations ranging from 0.1 µg/L to 2.2 µg/L. PFBA was detected in 32 urine samples (16%) at concentrations greater than the corresponding NHANES 95th percentile of 0.300 µg/L. A geometric mean comparison value is not available from NHANES because PFBA was only detected in 13% of samples (Calafat et al., 2019). Note, there are challenges in measurement of trace levels of PFBA, including selectivity of the analytical instrumentation and potential for external contamination [Abraham et al. 2021]. Therefore, we advise caution when interpreting the PFBA results.

Information on urinary concentrations of PFAS in humans is limited, yet it may be important to understand exposure to short-chain and alternative PFAS. Because urine is the primary route of excretion for many PFAS, urinary concentrations may reflect more recent exposures than do serum concentrations. Some PFAS were detected in serum but not in urine. These seemingly contradictory results highlight the importance of using the appropriate biomonitoring matrix for exposure assessment. Concentrations of biologically persistent compounds (like some PFAS) are expected to be higher in serum than in urine, as was observed in this assessment. This trend is also evident in other biomonitoring studies in the general population and in communities with known PFAS exposures [Calafat et al. 2019].

PFAS in Tap Water at ATSDR-led EAs

ATSDR collected a total of 176 tap water samples from the 117 randomly selected EA participant households and analyzed these samples for PFAS. At each participating household, ATSDR collected a drinking water sample from the kitchen tap. If point-of-use filtration was in place (e.g., a refrigerator filter, an under-the-sink reverse osmosis system), ATSDR attempted to collect samples both before and after filtration. Across the eight EA sites, ATSDR collected 101 unfiltered samples and 75 filtered samples. Each sample was analyzed for 19 different PFAS. Detection limits were 2 ppt for all PFAS, except for HFPO-DA or GenX (5 ppt).

Detailed information on tap water sample results for each site are provided in the site-specific reports. Table 9 presents a summary of PFAS detection frequencies and reported concentration ranges for filtered and unfiltered samples that were collected at each site. There were no PFAS detected in any of the filtered or unfiltered tap water samples collected at the Airway Heights, WA and Orange County, NY sites. Seven PFAS were detected in samples collected at the other six sites (i.e., PFHxS, PFOS, PFOA, PFBS, PFHpA, PFHxA, and HFPO-DA [GenX]). PFAS were detected most frequently among unfiltered samples collected at the two sites where participants were on private wells (i.e., Lubbock County, TX and Moose Creek, AK).

Across all sites, the maximum measured concentrations in drinking water were 67 ppt for PFHxS, 46 ppt for PFOS, 14 ppt for PFOA, 12 ppt for PFBS, 9.2 ppt for PFHpA, 57 ppt for PFHxA, and 7.8 ppt for HFPO-DA (GenX). No other PFAS were detected in any of the drinking water samples collected as part of the EAs. All measured concentrations from these samples were below EPA's 2016 HA of 70 ppt for PFOA and PFOS combined, ATSDR's EMEG for PFAS in drinking water (with the exception of one filtered sample and one unfiltered sample, discussed below), as well as all other available state-specific standards or guidelines in place at the time that this report was published. This includes ATSDR's recommended EMEGs for PFAS in drinking water of 140 ppt for PFHxS, 14 ppt for PFOS, 21 ppt for PFOA, and 21 ppt for PFNA; the Massachusetts Department of Environmental Protection who published a drinking water standard in 2020 of 20 ppt for total PFOA, PFOS,PFNA, PFHxS, PFHpA, and PFDA ("PFAS6"); and New York State who published a maximum contaminant level of 10 ppt for PFOS and PFOA.

Two samples had PFOS measured above ATSDR's EMEG for PFOS. The sample with 46 ppt of PFOS represents an unfiltered sample in a household where only filtered water was used for drinking water. PFOS was not detected in the filtered water sample collected from this household. The sample with 33 ppt of PFOS was from a sample of filtered water from inside a home that had a self-maintained whole house filtration system. It is suspected that the filter in use had not been maintained appropriately and was serving as a source of PFAS into otherwise clean water. That one filtered sample had measurable concentrations of six PFAS while all other samples (both filtered and unfiltered) collected from the

Berkeley County, WV site had non-detectable levels for all PFAS other than PFBS. ATSDR followed up individually with that homeowner to recommend replacement of the filter in use at that household. See additional discussion of this observation below as possible filter maintenance issues were seen at more than one site. The next highest concentration of PFOS measured in drinking water was 11 ppt.

At some households, PFAS were measured at higher concentrations in filtered samples than corresponding unfiltered samples collected from the same household. This occurred, for example, at households from the Westfield EA and the Moose Creek EA. In Westfield, PFOS, PFOA, and PFHxS were detected in a sample collected after a refrigerator filter but not in the corresponding unfiltered sample from the kitchen tap. A similar scenario was observed at a household from the Moose Creek EA. Numerous PFAS were also detected in a filtered sample collected from a household with a whole house filtration system from the Berkeley County EA. Note that there was no corresponding unfiltered sample for this household, but PFAS were not detected in the eight other filtered samples collected from households with whole house filtration systems in this EA. It is not clear why PFAS were detected in filtered samples in these examples, but one possible explanation could be related to filter maintenance. This trend was not observed across the other paired filtered and unfiltered samples but highlights the importance of properly maintaining filtration systems.

Table 9. Summary of PFAS detections in tap water samples collected at EA sites (in ppt)*

EA Site	Sample Type	PFHxS		PFOS		PFOA		PFBS		PFHpA		PFHxA		HFPO-DA	
		FOD	Range of Detects	FOD	Range of Detects	FOD	Range of Detects	FOD	Range of Detects	FOD	Range of Detects	FOD	Range of Detects	FOD	Range of Detects
Westfield, MA	Unfiltered (n=16)	0	—	0	—	0	—	0	—	0	—	0	—	0	—
	Filtered (n=8)	1/8	3.6	1/8	3.5	1/8	2.0	0	—	0	—	1/8	7.2	0	—
Berkeley County, WV	Unfiltered (n=17)	0	—	0	—	0	—	11/17	2.0–3.3	0	—	0	—	0	—
	Filtered (n=10)	1/10	63	1/10	33	1/10	13	3/10	2.0–3.1	1/10	5.5	1/10	8.8	0	—
New Castle County, DE	Unfiltered (n=13)	0	—	0	—	3/13	4.2–9.6	3/13	3.5–3.8	3/13	6.0–9.2	3/13	30–37	1/13	7.8
	Filtered (n=7)	0	—	0	—	0	—	0	—	0	—	2/7	2.0–6.2	0	—
Airway Heights, WA	Unfiltered (n=19)	0	—	0	—	0	—	0	—	0	—	0	—	0	—
	Filtered (n=7)	0	—	0	—	0	—	0	—	0	—	0	—	0	—
Lubbock County, TX	Unfiltered (n=6)	2/6	66–67	0	—	2/6	14	2/6	12	2/6	4.3–4.5	2/6	47–48	0	—
	Filtered (n=10)	2/10	7.2–36	0	—	2/10	4.3–4.6	2/10	8.5–11	2/10	2.5	3/10	3.9–54	0	—
Moose Creek, AK	Unfiltered (n=7)	7/7	4.9–43	4/7	2.3–46	5/7	2.8–9.6	7/7	2.1–6.1	1/7	3.2	7/7	2.3–7.5	0	—
	Filtered (n=11)	1/11	5.8	1/11	2.7	1/11	3.0	1/11	2.2	0	—	1/11	3.0	0	—
Security-Widefield, CO	Unfiltered (n=17)	0	—	1/17	3.0	0	—	0	—	4/17	2.1–2.5	7/17	4.8–57	0	—
	Filtered (n=17)	1/17	3.5	1/17	11	1/17	3.0	1/17	2.1	1/17	2.3	4/17	3.9–51	0	—
Orange County, NY	Unfiltered (n=6)	0	—	0	—	0	—	0	—	0	—	0	—	0	—
	Filtered (n=5)	0	—	0	—	0	—	0	—	0	—	0	—	0	—

FOD= frequency of detection, ppt= parts per trillion, '—' = not detected

Note: Detection limits are 2ppt for all PFAS, except for HFPO-DA (5 ppt)

* PFNA, PFDA, PFUnA, PFDoA, PFTrA, PFTA, MeFOSAA, EtFOSAA, DONA, 9Cl-PF3ONS, and 11Cl-PF3OUdS were not detected in any tap water samples and therefore are not shown.

PFAS in Household Dust at ATSDR-led EAs

ATSDR collected dust samples from the same 117 randomly selected participant households where tap water samples were collected and analyzed these samples for PFAS. These samples were taken from multiple locations in each household, including the primary living space as identified by the homeowner (e.g., living room, family room, television room), the kitchen, and the bedroom in which participants reported spending the most time. When necessary, additional sampling was performed in other rooms to allow ATSDR to collect the proper amount of dust for testing.

Detailed summary statistics for each site (i.e., frequency of detection, geometric means, 95% confidence intervals around the geometric means, and percentiles) are provided in the site-specific reports. Table 10 presents geometric means for PFAS that were measured in dust in at least 60% of samples collected at a given site. Note that per the study protocol, ATSDR only calculated geometric means for PFAS detected in 60% or more of the samples. Several PFAS were not detected at this frequency at any site and therefore not included in this table (i.e., PFDoS, PFDS, PFHpS, PFNS, PFOSA, PFPeS, PFHpS, N-EtFOSA, N-MeFOSA, N-EtFOSA, FtS 8:2, FtS 4:2, HFPO-DA, ADONA, 9CL-PF3ONS, and 11CL-PF3OUdS).

Table 10. Geometric means for dust samples collected at EA sites in nanograms per gram

PFAS	Westfield, MA	Berkeley County, WV	New Castle County, DE	Airway Heights, WA	Lubbock County, TX	Orange County, NY	Moose Creek, AK	Security-Widefield, CO
Households	*n=17*	*n=19*	*n=13*	*n=19*	*n=12*	*n=6*	*n=13*	*n=18*
PFBS	NA*	NA*	NA*	NA*	NA*	3.50	NA*	3.25
PFHxS	5.3	NA*	NA*	NA*	NA*	NA*	2.55	3.53
PFOS	12.9	15.9	10.9	14.4	5.42	12.4	8.35	12.2
PFBA	NA*	NA*	14.1	NA*	NA*	NA*	NA*	11.0
PFPeA	NA*	NA*	6.09	NA*	NA*	NA*	NA*	NA*
PFHxA	8.2	8.51	10.1	8.00	3.56	6.16	2.56	6.54
PFHpA	5.9	5.50	7.41	NA*	NA*	NA*	2.23	3.51
PFOA	15.2	15.1	13.3	8.88	4.77	10.7	4.06	7.99
PFNA	3.8	3.85	7.87	NA*	NA*	NA*	3.06	6.70
PFDA	4.3	NA*	9.19	NA*	NA*	NA*	1.98	3.92
PFUnA	2.0	NA*	7.63	NA*	NA*	NA*	1.07	NA*
PFDoA	2.4	NA*	5.88	NA*	NA*	NA*	1.42	NA*
PFTrA	NA*	NA*	5.18	NA*	NA*	NA*	NA*	NA*
PFTA	NA*	NA*	3.85	NA*	NA*	NA*	NA*	NA*
MeFOSAA	NA*	NA*	NA*	NA*	NA*	NA*	1.83	2.35
N-MeFOSE	NA*	NA*	NA*	NA*	NA*	NA*	17.1	26.8
EtFOSAA	6.8	8.64	7.94	NA*	NA*	16.1	2.92	3.08
FtS 6:2	NA*	NA*	NA*	NA*	NA*	NA*	12.2	NA*

NA = not applicable

*Per the EA protocol, geometric means were not calculated for PFAS detected in less than 60% of samples.

PFOS, PFOA, and PFHxA were detected in at least 60% of the dust samples collected at all eight EA sites. Geometric means for PFOS ranged from 5.42 nanogram per gram (ng/g) to 15.9 ng/g while means for PFOA ranged from 4.06 ng/g to 15.2 ng/g. Geometric means for PFHxA ranged from 2.56 ng/g to 10.1 ng/g. Of note, New Castle County, DE and Moose Creek, AK had the greatest number of PFAS detected

in at least 60% of samples. Thirteen of the PFAS measured in dust were detected at this frequency in each of these communities.

To provide some context to the results summarized above, average levels of PFAS measured at each site were compared to average dust levels reported in other U.S.-based studies (with or without known PFAS contamination). This includes evaluations of indoor dust collected at 30 homes in the greater Boston area [Fraser et al. 2013], 124 homes in California [Wu 2015], 15 U.S. homes [Karásková et al. 2016], and 19 homes in Minnesota cities with PFAS-contaminated soil and drinking water [Scher et al. 2018]. Across these studies and in this EA, PFOA and PFOS were consistently reported at the highest concentrations. Geometric mean concentrations ranged from 24 to 45 ng/g for PFOA and 27 to 35 ng/g for PFOS [Fraser et al. 2013; Wu et al. 2015]. Two of the studies did not report geometric means; for these studies, median concentrations were reported at 9 ng/g and 51 ng/g for PFOA and 14 ng/g and 67 ng/g for PFOS [Karásková et al. 2016 and Scher et al. 2018, respectively]. Geometric mean concentrations for PFOA and PFOS measured in the eight EA sites were generally similar to or lower than what was reported from these four studies. The same holds true for PFNA. PFHxS was only detected in 60% or more of the dust samples collected in three EA sites. Geometric concentrations from these communities were comparable to that reported in the four studies described above. Details on these studies (e.g., geometric means, ranges) can be found in Appendix A, Table A10.

While these results suggest that PFAS measured in the dust samples collected at the EA Sites were found at similar or lower levels than reported elsewhere in the United States, note that the studies referenced here do not necessarily provide representative comparisons and are provided only for additional context. The sample collection methods and analytical methods were also not consistent among these studies.

ATSDR also evaluated the correlation between PFAS measured in dust and blood. This analysis included analytical data from the 117 dust samples summarized above and from the 200 blood samples collected from participants residing in the same homes as where the dust samples were collected. Using log-transformed data, ATSDR calculated Pearson correlation coefficients for the PFAS measured in at least 60% of the dust samples and that were also measured in blood. Data were log-transformed for this analysis since both dust and blood concentrations were found to be log-normally distributed.

Table 11 presents Pearson correlation coefficients for PFAS measurements in household dust and PFAS measurements in the blood of participants from the same households. Note that PFUnA and MeFOSAA were detected in fewer than 60% of dust samples and therefore excluded from the table.

Table 11. Pearson correlation coefficients between PFAS in dust (log) and blood (log)*

	PFHxS	PFOS	PFOA	PFNA	PFDA
PFHxS	0.13 *(p=0.06)*	—	—	—	—
PFOS	—	0.12 *(p=0.08)*	—	—	—
PFOA	—	—	0.09 *(p=0.19)*	—	—
PFNA	—	—	—	0.28* (*p<0.0001)*	—
PFDA	—	—	—	—	0.19* *(p=0.009)*

* Statistically significant (p<0.05)

Statistical correlations (p<0.05) between dust and blood PFAS concentrations were only observed for two PFAS, PFNA and PFDA. However, Pearson correlation coefficients for these comparisons ranged from 0.28 to 0.19, indicating only weak correlation. Measurements of PFHxS, PFOS, and PFOA in dust were not statistically correlated with the same PFAS measured in blood. For these PFAS, correlation coefficients ranged from 0.09 to 0.12, similarly indicating weak correlation. Given these weak correlations, the contribution of dust to EA participant PFAS levels appears to be low compared to drinking water contributions.

The dust results presented here are exploratory and should be interpreted with caution. In some cases, those samples are based on a small sample mass. The target sample mass for this study was 1 gram, but this target was not always met. Results based on less than 1 gram of dust have higher detection limits, which is a potential source of bias.

Key Findings

Main conclusions from the EAs are presented below, which describe

- How PFAS blood levels measured at all EA sites compare to national levels (Findings 1–3),
- Why PFAS blood levels measured across all 10 EA sites are believed to be strongly associated with past drinking water exposures (Finding 4),
- Possible associations between PFAS blood levels and other exposure variables based on analysis of combined data from the eight ATSDR-led EAs (Finding 5), and
- Urine (Finding 6) and environmental sampling (Findings 7 and 8) results from the eight ATSDR-led EAs.

Findings for the 10 individual EAs can be found in the separately published EA-specific reports.

Finding 1. Average age-adjusted PFHxS blood levels are higher than national levels in all 10 EA communities.

At all 10 EA sites, the age-adjusted geometric mean (i.e., average) PFHxS levels were statistically higher (p<0.05) in EA participants compared to national levels (CDC's NHANES 2017–2018). Of all the PFAS

analyzed in blood, PFHxS levels had the largest elevations when compared to national levels. Across EA sites, geometric mean blood PFHxS levels ranged from 2.4 to 61 times national levels. The highest age-adjusted geometric mean blood level for PFHxS was 65.6 micrograms per liter (µg/L), observed in Airway Heights, WA.

Finding 2. Average age-adjusted PFOS and PFOA blood levels are higher than national levels in most EA communities.

Average PFOS was statistically elevated in 8 of 10 EAs at 1.2 to 9.2 times national levels, and average PFOA was statistically elevated in 7 of 10 EAs at 1.2 to 6.3 times national levels. The highest age-adjusted geometric mean PFOS (39.1 µg/L) and PFOA (8.9 µg/L) blood levels also were observed in Airway Heights, WA.

Finding 3. Other average age-adjusted PFAS blood levels were higher than national levels in some but not all EA communities.

PFNA levels were also statistically elevated in 4 of 10 EAs, and blood levels at these EA sites ranged from 1.4 to 2.2 times national levels with a maximum adjusted geometric mean of 0.903 µg/L. The remaining PFAS (PFDA, PFUnA, and MeFOSAA) were detected at lower frequencies. Adjusted geometric mean blood PFDA and PFUnA were statistically elevated at one site (New Castle, DE) at levels that were 1.4 and 1.7 times NHANES 2017–2018 levels, respectively. Blood MeFOSAA levels were not statistically elevated at any sites.

Finding 4. Elevated blood levels of PFHxS, PFOS, and PFOA may result from past drinking water contamination.

Multiple lines of evidence from the EAs support the finding that past exposure to contaminated drinking water may have contributed to the elevated blood levels.

- *The highest PFAS blood levels were observed in participants who lived in communities with the highest historical PFAS drinking water levels.* The strongest evidence linking blood PFAS levels to drinking water data is the consistent association observed with maximum historic concentrations of PFAS measured in drinking water at all EA sites. Drinking water measurements were provided by affected water systems or the Air Force (in the case of private well sites) for PFHxS, PFOS, and PFOA. The individual drinking water measurements were statistically associated with corresponding PFAS measured in blood in the combined data from all 10 EA sites. In other words, residents of households that had the highest contamination for PFHxS, PFOS, and PFOA in their drinking water generally had higher blood levels for these substances.
- *Three of the PFAS (PFHxS, PFOS, and PFOA) with statistically elevated blood levels were previously detected in drinking water supplies at all EA sites.* PFAS were first detected in drinking water supplies at EA sites between 2009 and 2017. We do not know if contamination began earlier because no earlier data are available. From 2014 through 2019, each site had reduced PFAS levels below EPA's 2016 HA in each drinking water supply. Because of the long biological half-lives of PFAS (2.1 to 35 years), past drinking water exposures may have contributed to the EA participants' elevated blood PFAS levels observed sometimes years later. Of the PFAS measured in participants' blood, PFHxS has the longest estimated half-life, which, combined with the relatively high concentrations of PFHxS previously measured in drinking water in these communities, may be why PFHxS blood levels exceeded national levels by the largest margin.
- *Correlation across PFAS in blood suggests a common exposure source.* PFHxS, PFOS, and PFOA were highly correlated in EA participants' blood (Pearson correlation coefficient, *r* between 0.83

and 0.86 for the combined data from all 10 EAs). This means that, typically, residents who had elevated blood PFHxS levels also had elevated blood PFOS and blood PFOA levels. This correlation suggests a common exposure source, such as drinking water, though other sources of exposure may also have contributed to the observed blood levels. It also suggests a common contamination source for drinking water, such as AFFF, which contained these three PFAS in historical formulations.

- *Drinking water consumption patterns were shown to be predictors of blood PFAS levels.* Regression modeling conducted across the eight ATSDR-led EAs further supports this finding:
    - In univariate and multivariate models, a consistent predictor of participant blood PFHxS, PFOS, and PFOA levels was how long the resident had lived in the sampling frame during the past 20 years. Those who lived in the area longest had higher PFHxS, PFOS, and PFOA blood levels—and also likely drank the contaminated water for the longest period.
    - After controlling for other variables in multivariate models, personal drinking water consumption rates were associated with blood PFHxS levels.
    - In univariate and multivariate analyses, adults who used at least one filter or treatment device in their homes and adults who reported not drinking tap water at all (i.e., only reported drinking bottled water) on average had statistically lower blood levels of PFHxS, PFOS, and PFOA when compared to those reporting no filter or treatment device.
    - In multivariate analyses, as the number of days since drinking water mitigation increased, average blood PFOS and PFOA levels decreased in adults.

Finding 5. PFAS blood levels varied with different demographic and exposure characteristics of the participant population.

As highlighted below, the multivariate analyses conducted for the combined data set for the eight ATSDR-led EAs (adults and children) revealed statistically significant associations between PFAS blood levels and some of the demographic and exposure variables examined. While the EAs were not designed to quantify other exposures as potential predictors for PFAS blood levels, the findings from these EAs show other factors that may influence PFAS blood levels, many of which have been documented in previous studies. Because these EAs were not designed to characterize non-drinking water exposures, the strength of these associations varied, and the results of these analyses should be interpreted with caution. Some of these associations may be due to chance as we are testing many associations at once. Some of the variables were associated with blood levels of those PFAS elevated in drinking water (PFHxS, PFOS, and PFOA) and others associated with PFNA or PFDA blood levels only.

- *Age*. Blood levels of PFHxS, PFOS, PFOA, and PFNA were statistically higher in older adult participants, and the size of the effect was stronger in females. In children between 3 and 17 years of age, blood PFHxS and PFOA levels decreased for every additional year in age.
- *Sex*. Male adults had statistically higher blood levels of PFHxS, PFOS, PFOA, and PFNA than females, and the difference between males and females was larger in younger adults. In children, blood levels in males were higher than females for PFOS, PFOA, PFNA, and PFDA.
- *Race/ethnicity*. Race and ethnicity were only associated with blood PFNA levels in multivariate models for adults and children. Compared to those who identified as "White, non-Hispanic," some groups had higher PFNA blood levels, while others had lower PFNA levels.

- *Cleaning frequency*. Adults who reported cleaning their homes more frequently had higher PFNA blood levels than adult participants who reported cleaning their homes a few times per year or less.
- *Soil contact*. Children who reported coming in contact with soil more frequently had higher levels of PFOS, PFNA, and PFDA.
- *Eating locally grown produce*. Adults and children who reported eating locally grown fruit and vegetables had higher blood PFDA levels than those who did not.
- *Consuming local dairy*. Adults who reported drinking locally produced milk 'rarely' or more frequently had higher blood PFHxS and PFOA levels compared to participants who reported never drinking locally produced milk.
- *Using stain resistant products*. Participants who reported using stain-resistant products a few times per year or more frequently had blood levels of PFNA that were higher than participants who never used them.
- *Number of children*. Female adults who reported having children had lower blood PFHxS levels than females who did not have children.
- *Breastfeeding and infant formula consumption*. In child participants, every additional month of reported breastfeeding was associated with an increase in blood PFDA levels. Every additional month of formula consumption was associated with an increase in blood PFNA levels.

Finding 6. PFHxS, PFHxA, and PFBA were detected in urine at low concentrations.

ATSDR analyzed 204 of the urine samples collected (189 adults and 15 children) as part of the eight ATSDR-led EA. PFHxS, PFHxA, and PFBA were the only PFAS detected in any of the 204 urine samples analyzed from eligible participants. Concentrations of biologically persistent PFAS are expected to be higher in serum than in urine, as was observed in this assessment. This trend is also evident in other biomonitoring studies in the general population and in communities with known PFAS exposures.

- PFBA was detected in 79 of the 204 urine samples that were analyzed (38%). This PFAS was measured in participants from six different sites and at individual sample concentrations ranging from 0.1 µg/L to 2.2 µg/L. Note, there are challenges in measuring trace levels of PFBA, including selectivity of the analytical instrumentation and potential for external contamination. Therefore, we advise caution when interpreting the PFBA results.
- PFHxS was detected in 9 urine samples (4%) and detected individual sample concentrations ranged from 0.1 µg/L to 0.4 µg/L. PFHxS is not generally found in urine. Detection of PFHxS suggests recent exposure or elevated PFHxS levels in blood. All PFHxS detections were in the Airway Heights, WA location, which had the highest measured PFHxS concentrations in blood and among the most recent exposures to PFAS in drinking water prior to mitigation of the sites evaluated.
- PFHxA was detected in two urine samples (1%) and only among participants from the Lubbock County, TX EA.

ATSDR did not analyze all participants' urine samples because none of the species were detected in more than 60% of the samples analyzed at any site.

Finding 7. Almost all tap water samples collected during the ATSDR-led EAs were below all federal and state guidelines for PFAS in drinking water in place at the time samples were collected.

Only two of the 176 tap water samples (101 unfiltered and 75 filtered) from 117 randomly selected EA participant households were above federal or state guidelines for PFAS in drinking water at the time of the ATSDR-led EAs. The two samples exceeded ATSDR's EMEG for PFOS in drinking water. One sample came from a whole-house filter that may not have been properly maintained and the other from an unfiltered tap sample not used for drinking water; ATSDR followed up individually with the first homeowner to recommend filter replacement. The findings suggest that past drinking water contamination may be the source of PFAS in the serum, rather than current drinking water levels.

Finding 8. Patterns and levels of PFAS in dust measured in households participating in the ATSDR-led EAs are comparable to those reported in selected other U.S. studies.

No nationally representative comparison values are available, but geometric mean and median concentrations for PFAS measured in dust collected in the subset of participating households (n=117) were within the range of levels reported in a few published studies of other U.S. communities (with or without known PFAS contamination). Eighteen PFAS were detected with a frequency greater than 60%. Of the PFAS measured in both household dust and blood, PFNA and PFDA were statistically correlated with the same PFAS measured in participants' blood.

Limitations

There are several limitations associated with this assessment.

- Although similar data were collected for ATSDR's two pilot EAs and the eight ATSDR-led EAs, the methods are slightly different (different recruitment methods, PEATT included mix of private well and public water participants in the same community, different questions on questionnaire and different variable categories for some questions, different PFAS measured in blood, differences in inclusion of urine and environmental measurements). Combined analysis of exposure variables was therefore only possible for the eight ATSDR-led EAs. Data are compared and trends presented where possible for the two pilot sites.
- Participants in each EA community may not be fully representative of the entire community. The sampling recruitment method used for each EA was designed to measure blood PFAS concentrations that were generalizable to residents from each EA community who were exposed to contaminated drinking water. The household response rates varied from 1.6% to 18% for the ATSDR-led EAs. Participant characteristics were different than those of each area's overall population. Generally, participants were older than the corresponding EA community, and at some sites the racial and ethnic distribution of participants was different. Few children under the age of 18 participated in the EAs.
- The recruitment targets for the EAs meant that, at some sites, all households within the sampling frame were invited to participate rather than only inviting randomly selected households to participate. For all sites, identical efforts were made to recruit the selected households to participate in the EAs. For logistical reasons, a maximum of 3,000 households were invited to participate from any one of the EA communities. At four sites (WV, WA, TX, and AK), the number of households was not large enough to reach the target participation rate, even when inviting all households within the sampling frame. Because identical procedures were used

to recruit participants across all sites, this difference is not expected to have a significant impact on the results.

- Recruitment and field data collection for three of the ATSDR-led EAs were conducted after the start of the COVID-19 pandemic. Recruitment activities were underway for the Orange County, NY EA in March of 2020 when the work was paused due to the pandemic. All field activities for the AK, CO, and NY (Orange County) EAs were also conducted during the pandemic. It is possible that the participation rates for these sites would have been higher if work had been completed prior to the pandemic.
- Measurement of blood, urine, and environmental PFAS concentrations for EA participants may improve the understanding of exposure in these communities but will not provide discrete information about all sources of exposure. Additionally, identifying every potential confounding exposure is not possible. Samples were collected over a three-year period from 2018 through 2020. Although multivariate regression models explained a moderate portion of variability of participants' blood PFAS levels, other factors not identified could influence the relationships reported in this report (see "Statistical Analysis" section for details).
- These EAs were not designed to investigate health outcomes. Without additional information about exposure-response relationships, the results of the EAs cannot be used to assess current or past health problems or predict the future occurrence of disease. PFAS found in a person's blood or urine means that exposure has occurred. The presence of PFAS in blood or urine does not tell us how, where, when, or for how long a person was exposed to PFAS. Exposure to PFAS does not mean adverse health effects will result, either now or in the future.
- We know that the family of PFAS includes thousands of different chemicals. Due to analytical limitations, the EAs were only able to assess exposure to a targeted list of PFAS for which testing methods were available at the time of the EAs. We are not able to draw conclusions about exposure to PFAS for which we did not have the analytical ability to measure.
- While we know that people can be exposed to PFAS through a variety of pathways, these EAs focused specifically on exposure through drinking water. While we made some attempt to characterize PFAS in environmental media, the data were not sufficient to draw strong conclusions about exposure via other pathways (food, incidental ingestion, lactational/gestational exposure, inhalation, etc.)
- These EAs collected data from a single point in time. Therefore, our ability to comment on how exposure may have changed over time is limited. It is likely that blood concentrations of PFAS in the past were higher than those measured during the EAs.
- There are challenges in measurement of trace levels of PFBA in urine, including selectivity of the analytical instrumentation and potential for external contamination. Therefore, we advise caution when interpreting the PFBA results in urine.
- These EAs did not directly assess participants' tap water consumption prior to the reduction of PFAS in the various drinking water supplies in the EA communities.
- The dust results are exploratory and should be interpreted with caution. They are based on a limited set of samples, and in some cases those samples are based on a small sample mass.
- The analytical method used to measure PFAS in drinking water samples collected as part of the EAs has detection limits of 2–5 ppt for individual PFAS. While these detection limits are approaching the lowest achievable detection limits with current technology, they are higher than EPA's interim health advisory levels for PFOA and PFOS that were released in 2022 (after completion of all EA sampling activities). In this report, we retain comparisons to EPA's 2016

health advisory which was in place at the time of sample collection as well as comparisons to ATSDR's EMEGs for PFAS in drinking water.

Recommendations

Although the exposure contribution from PFAS in drinking water in all EA communities has been reduced to levels below EPA's 2016 health advisory, there are actions stakeholders and community members can take to further reduce exposures to PFAS and protect public health.

Recommendations for water providers:

1. Based on the PFAS drinking water test results from sites with public water supplies, ATSDR does not recommend an alternate source of drinking water at this time. However, operators of affected public water systems should continue to monitor concentrations of PFAS in drinking water delivered to EA communities and appropriately maintain treatment systems to ensure that concentrations of PFAS remain below existing or new Federal and state guidelines for specific PFAS in drinking water. Results of PFAS monitoring should be shared with community members through appropriate communication channels. State and/or federal guidelines/regulations for PFAS can change; these recommendations are based on guidelines/regulations available at the time of the publication of this report.
2. The Air Force is encouraged to continue providing bottled water and/or water filtration systems for households with private wells with PFAS concentrations above relevant state or Federal guidelines unless a different alternative source of drinking water that meets all guidelines has been provided. Testing should continue to be made available for private wells for PFAS if new data indicate they may be impacted by PFAS-containing groundwater. Households with private wells that receive bottled water and/or have water filtration systems installed specifically to treat water to remove PFAS should continue to use these alternative sources of water.

Recommendations for residents in affected areas:

1. Become familiar with Consumer Confidence Reports for information on each system's water quality.
2. Private well owners living in areas affected by PFAS should consider having their wells tested for PFAS if testing has not been conducted before. To learn more about testing wells for PFAS in your community, visit the resources listed in EA-specific reports.
3. Based on test results, consider installing a home water treatment system to further lower levels of PFAS in drinking water. The global public health organization NSF International has developed a test method to verify a water filter's ability to reduce PFOA and PFOS to below the health advisory levels set by the EPA or individual states. NSF International-approved devices can be found at: https://info.nsf.org/Certified/DWTU/. Click on "reduction devices" at the bottom of the page for PFOA and PFOS. Any treatment systems installed should be operated and maintained according to manufacturer recommendations to ensure proper operation and removal of PFAS from water.
4. Nursing mothers should continue breastfeeding. Based on current science, the known benefits of breastfeeding outweigh the potential risks for infants exposed to PFAS in breast milk.

5. When possible, eliminate or decrease potential exposure to PFAS in consumer products such as stain-resistant products and food packaging materials. To learn more visit: https://www.fda.gov/food/chemical-contaminants-food/questions-and-answers-pfas-food
6. Pay attention to advisories about food consumption, such as local fish advisories.
7. Discuss any health concerns or symptoms with your health care provider. Share results of PFAS blood testing with your health care provider and make them aware of ATSDR resources for clinicians (https://www.atsdr.cdc.gov/pfas/resources/info-for-health-professionals.html). Follow the advice of your health care provider and the recommendations for checkups, vaccinations, prenatal care, and health screening tests.
8. Follow the advice of your child's health care provider and the recommendations for well child checkups, vaccinations, and recommended health screening tests. Consult https://health.gov/myhealthfinder to help identify those vaccinations and tests. Pregnant women should follow their healthcare provider's recommendations for prenatal care.
9. For additional information about environmental exposures and children's health, contact the Pediatric Environmental Health Specialty Units, a nationwide network of experts in reproductive and children's environmental health (https://www.pehsu.net/).

Recommendations for future work/action:

1. Federal and state regulatory agencies can consider the EA findings about PFAS serum levels relative to maximum measured historical concentrations of PFAS in drinking water for policy development.
2. ATSDR recommends expanded monitoring for PFAS in drinking water in communities beyond those included in the EAs to improve our ability to identify and respond to communities affected by PFAS. The EAs have clearly shown that communities with exposure to PFAS through their drinking water have elevated blood concentrations of some PFAS when compared to the national population. At the EA sites, the PFAS concentrations in water were known and actions were taken to reduce exposure. There may be other unidentified communities with similar levels of exposure because monitoring data for PFAS in drinking water are not routinely available. EPA's Unregulated Contaminant Monitoring Rule 5 will require sample collection from some public water systems for 29 PFAS between 2023 and 2025. This information will be helpful as a snapshot of potential contamination at a point in time, but regular monitoring for PFAS in drinking water would allow public health authorities to take swift action in the event PFAS were detected in a water supply.
3. ATSDR will continue to share information about ongoing research related to the potential for health effects following PFAS exposure (such as the Multi-Site Study) with EA communities. ATSDR will keep the EA communities updated with any changes to ATSDR's clinician guidelines for PFAS. Understanding the relationship between PFAS exposure and health outcomes will allow communities and governmental agencies to make better decisions about how to protect public health. The information from the health studies can be applied to communities across the nation, including the communities where the EAs were conducted. More information about ATSDR's Multi-Site Study can be found at https://www.atsdr.cdc.gov/pfas/activities/studies/multi-site.html

For More Information

If you have questions or comments or want more information on the EA sites, call 800-CDC-INFO or email pfas@cdc.gov. For more information on the work CDC/ATSDR is doing to address PFAS exposure, visit ATSDR's PFAS website: https://www.atsdr.cdc.gov/pfas/. For other EA or PFAS-related questions, email pfas@cdc.gov.

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