

Letter Health Consultation

Evaluation of Perfluoroalkyl Substances (PFAS) in Public and Private Drinking
Water Wells

FORMER NAVAL AIR STATION JOINT RESERVE BASE (NASJRB) AND
AIR RESERVE STATION (ARS)

HORSHAM, MONTGOMERY COUNTY, PENNSYLVANIA

EPA FACILITY ID: PAD987277837

Embargoed Do not distribute

MARCH 13, 2020

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Division of Community Health Investigations
Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

An ATSDR health consultation is a verbal or written response from ATSDR to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

You may contact ATSDR toll free at

1-800-CDC-INFO

or

visit our home page at: <https://www.atsdr.cdc.gov>

Embargoed Do not distribute

LETTER HEALTH CONSULTATION

Evaluation of Perfluoroalkyl Substances (PFAS) in Public and Private Drinking
Water Wells

FORMER NAVAL AIR STATION JOINT RESERVE BASE (NASJRB) AND
AIR RESERVE STATION (ARS)

HORSHAM, MONTGOMERY COUNTY, PENNSYLVANIA

EPA FACILITY ID: PAD98727637

Embargoed Do not distribute

Prepared by
U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry
Division of Community Health Investigations



March 13, 2020

Paul Leonard
Acting Director, Superfund and Emergency Management Division
United States Environmental Protection Agency (USEPA)
1650 Arch Street, MS: 3HS00
Philadelphia, PA 19103

Dear Mr. Leonard:

On September 24, 2014, the United States Environmental Protection Agency (EPA) Region 3 Superfund and Emergency Management Division asked the Agency for Toxic Substances and Disease Registry (ATSDR) to evaluate past and current exposures to per- and polyfluoroalkyl substances (PFAS) in public and private drinking water sources near military bases in Bucks and Montgomery Counties, Pennsylvania. These bases include:

- The former Naval Air Warfare Center located in Warminster Township, Bucks County, Pennsylvania (Warminster site).
- The former Naval Air Station Joint Reserve Base (NASJRB) and the active Air Reserve Station (ARS) Willow Grove located in Horsham, Montgomery County (Willow Grove site).

The source of PFAS in groundwater near these bases is assumed to be the past use of aqueous film-forming foam (AFFF) at these locations. PFAS contamination in groundwater was discovered at the Willow Grove site in 2011 as part of ongoing environmental investigations and offsite in 2014 when public drinking water supplies were sampled for unregulated contaminants. In 2014 through 2016, PFAS-contaminated public water supply wells near the bases were taken out of service and/or treatment installed on the impacted wells. Private well owners above a level of concern were provided bottled water and offered hookups to public water. In January 2016, ATSDR produced a public health evaluation of public and private drinking water supplies for the nearby Warminster site. This Willow Grove public health evaluation focuses on exposures to community members in Horsham and Warrington Townships near the Willow Grove site.

Community members potentially affected by the PFAS in drinking water contamination near the Willow Grove site include

- Approximately 46,000 users of public water supplies in Horsham and Warrington Townships,
- Hundreds of users of private drinking water wells in the area, and
- Civilian and uniformed service personnel and their families who may have resided or worked on the site.

We do not have information on the specific formulation of the AFFF foam used at the Willow Grove site. Perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) are the primary

PFAS detected in the drinking water at the Willow Grove site. Several other PFAS have been detected at this site, some of which have similar health endpoints as PFOA and PFOS. Analytical methods are insufficient to identify and measure the full range of people's exposure to PFAS. Toxicological and epidemiological data are only available to support the development of health-based screening values for some of the PFAS that have been detected in water and in people's bodies. At this time, ATSDR has developed provisional minimal risk levels (MRLs) for four PFAS chemicals (PFOS, PFOA, perfluorohexanesulfonic acid (PFHxS) and perfluorononanoic acid (PFNA)). To evaluate the potential risk for cumulative exposures, ATSDR calculated estimated exposure doses for the maximum levels detected of these four PFAS chemicals in the public and private drinking water supplies at this site and compared these exposure doses to the provisional MRLs for each contaminant. To put these estimated exposure doses in perspective, ATSDR also calculated margins-of-exposure (MOEs) for the four PFAS with provisional MRLs.¹

In the absence of data, chemical component-based approaches are used in risk assessment of chemical mixtures. Component chemicals, that are judged to be toxicologically similar, are evaluated by dose additive risk assessment methods that include the hazard index (HI), relative potency factors, and toxicity equivalency factors. These methods are based on potency weighted dose addition and assume that there are no greater than or less than additive interactions among the chemicals in the dose region of interest. ATSDR used an HI approach to evaluate the combined exposure of the four PFAS with provisional MRLs.² There are other PFAS without these screening levels, some of which were detected at this site. ATSDR does not have a method at this time to evaluate the health effects of the combined exposures to mixtures of PFAS compounds beyond PFOA, PFOS, PFNA, and PFHxS.

Conclusions

After reviewing the available information and considering all factors that may contribute to the health effects of PFAS exposures, ATSDR reached six conclusions for the site. These conclusions are limited by several uncertainties relating to the human health risks from PFAS exposures. Because of these uncertainties, ATSDR used a conservative approach, including several lines of evidence (see the attachment to this letter for details) to evaluate the public health implications of exposures to PFAS at this site.

Conclusion 1: Past Exposures to Public and Private Drinking Water

Before actions began in 2014 to reduce exposures, drinking public and private well water contaminated with PFAS at this site could have increased the risk for harmful effects for community members.

Other sources of PFAS exposure (e.g., from food, consumer products, fish that may be contaminated with PFAS near the site, etc.) could increase the risk of harmful effects beyond the risk from the drinking water exposures alone.

¹ The MOE is the effect level, developed from animal studies, used to derive the ATSDR provisional MRL divided by the dose from exposure to the drinking water.

² The hazard index approach assumes dose additivity to assess the non-cancer health effects of a mixture. The HI approach requires an established health effect screening level (such as an MRL or Reference Dose) for each chemical in the mixture. For this evaluation, the hazard index is the sum of the HQs for each of the four PFAS with ATSDR provisional MRLs.

The cancer risk from past exposure to a mixture of PFAS is uncertain. The cancer risk from exposure to PFOA alone in the drinking water is estimated at low to very low.

Basis for Conclusion The estimated exposure doses for PFOA, PFOS, PFNA, and PFHxS (based on maximum concentrations) from drinking the public and private water supplies were below effect levels found in animal studies. However, some of the estimated doses were well above their respective provisional ATSDR MRLs, indicating a potential for health concern, especially for PFOS.

Research in humans suggests that high levels of certain PFAS may lead to increased cholesterol levels, changes in liver enzymes, decreased vaccine response in children, increased risk of high blood pressure or pre-eclampsia in pregnant women, small decreases in infant birth weights, and increased risk of kidney or testicular cancer. Developmental (particularly in animals) and immune effects are among the most sensitive and common endpoints across multiple PFAS.

The combined exposures to a mixture of PFOS, PFOA, PFHxS, and PFNA could have increased the risk for developmental and immune effects above what might be expected from exposure to any of these PFAS alone. For other PFAS and other health endpoints, the scientific information is far less certain.

Food, consumer products, and mixtures of PFAS in the drinking water are all possible contributors to a person's overall PFAS exposure and body burden. Some pre-existing risk factors could increase the risk of harmful effects.

ATSDR's calculated MOEs support that past exposures to PFAS in the public and private drinking water supplies at this site were of concern. ATSDR's margin of exposure calculations indicate that the most exposed population using the maximum level of PFOS and PFOA detected in Horsham public water, Warrington public water, or a private well, and drinking more than average amounts of water on a daily basis, may have an increased risk of harmful effects.

Statistically representative blood testing of community members by the Pennsylvania Department of Health (PADOH) revealed the following:

- PFOA, PFOS, PFHxS, and PFNA blood levels are elevated as compared to national averages.
- Among the 11 PFAS tested for, only four compounds (PFOS, PFOA, PFHxS and PFNA) were consistently detected in the blood of community members from this area.
- PFOS was detected in all 235 participants.
- Overall, 75, 81, 94, and 59 percent of the study participants had levels exceeding the national average for PFOA (1.94 µg/L), PFOS (4.99 µg/L), PFHxS (1.35 µg/L) and PFNA (0.68 µg/L), respectively.
- The levels of PFOA, PFOS, PFHxS and PFNA among different age groups within the community differed significantly.

ATSDR estimates a very low to low increased risk of cancer from exposure to the maximum PFOA concentrations in the public and private drinking water supplies.

- EPA has developed an oral slope factor for PFOA to evaluate the cancer risk, based on testicular cancer from a rat study. This estimated cancer risk must be viewed with caution because the EPA oral slope factor has not been fully adopted and other cancers that were elevated in epidemiological studies of PFOA exposure were not evaluated.
- EPA does not have oral slope factors for other PFAS. Therefore, ATSDR cannot calculate the estimated cancer risk from other PFAS exposures, and the actual cancer risk from PFAS mixtures is uncertain.

Epidemiologic data suggest a link between PFOA exposure and elevated rates of kidney, prostate, and testicular cancer. Other information about cancers and PFAS exposures are uncertain.

- Animals given PFOA have shown higher rates of liver, testicular, and pancreatic tumors.
- Additional studies are needed to confirm the link between PFOA and other PFAS exposures and cancer to say they are the cause.
- A causal link based on human studies between cancer and PFOS exposures remains uncertain. Animal studies have found limited but suggestive evidence of PFOS exposure and increased incidence of liver, thyroid, and mammary tumors.

PADOH continues to evaluate cancer incidence rates for this community using data reported to the Pennsylvania Cancer Registry.

- The state has not found a consistent pattern in cancers in adults or children in the site area.
- PADOH's most recent update presents elevated incidence rates for some types of cancers in the area with drinking water contamination compared to Montgomery and Bucks counties and the Commonwealth of Pennsylvania.
- The current results do not indicate consistently higher incidence rates in all service areas for any cancer type, or consistently higher incidence rates for a given cancer in both sexes.

Conclusion 2: Unique Considerations for Private Well Water Users

Some private well users may have had some of the highest offsite PFAS drinking water exposure levels, increasing the possibility of harmful effects.

Basis for Conclusion Individual private well samples had a variety of PFAS concentrations. The maximum levels of PFAS found in the private drinking water wells exceeded the maximum levels in the offsite public water supplies. Furthermore, maximum detections in private well water represent actual exposure levels. This is not the case in a public water system, where mixing from multiple water sources occurs.

The private drinking water wells that are located closer to the site appear to have

higher levels of PFAS. The northeast and southwest sides of the sites showed the greatest number of private drinking water wells with PFOA and PFOS exceeding the EPA health advisory (HA).

- Approximately 26% of private drinking water wells (166 of the 640 private wells sampled) had levels of PFOA and PFOS above the EPA HA.
- In total, 48% (309 of 640 private wells sampled) had either PFOS or PFOA levels above the ATSDR intermediate child Environmental Media Evaluation Guide (EMEG)³ values.
- Approximately 11% (67 of the 640 private wells sampled) had PFHxS levels above the ATSDR intermediate child EMEG and
- 0.6% (4 of the 640 private wells sampled) had PFNA levels above the ATSDR intermediate child EMEG.

Individuals whose private drinking water wells were not sampled (specifically those within the site area who did not grant access for PFAS sampling) may be at risk for health effects from ongoing exposures.

In the PADOH blood sampling, the small number of participants who reported using private wells as their drinking water source had higher levels of PFOA, PFOS and PFNA in their blood compared to those using public water as their drinking water source. However, this was based on only a small number of private well users and the difference was not statistically significant.

Conclusion 3: Current Exposures to Public and Private Water Supplies Post Mitigation

Consuming water from public and private drinking water supplies after mitigation actions were implemented is not expected to cause harm to community members.

Basis for Conclusion

Since 2014, a number of actions have been implemented to significantly reduce the levels of PFAS in the public water supplies, including installing carbon filtration of affected public drinking water supply wells and taking some affected public water supply wells off-line.

- Based on the actions taken by the Navy, the Air National Guard, EPA, and the public drinking water providers and ongoing monitoring of the drinking water initiated in 2014, current exposures to PFOS and PFOA are below the EPA HA and in most cases below the ATSDR EMEG values derived from the ATSDR MRLs.
- Other PFAS are either detected at low levels or not detected.

In August 2016, Horsham Township voluntarily collected residential tap water samples (109 public water users), to assist in determining the effectiveness of the employed short-term measures including carbon filtration of the public drinking water wells and taking wells off-line.

³ATSDR EMEGs are environmental concentrations derived from ATSDR MRL doses that represent amounts in water, soil, and air to which humans may be exposed during a specified period of time without experiencing adverse health effects.

- While the average PFOS and PFOA levels were below the ATSDR child EMEGs, PFOS concentrations (maximum of 0.028 µg/L) exceeded the ATSDR child EMEG in 27 samples (24% of the samples).
- The Horsham tap water results did not exceed the ATSDR adult EMEGs for PFOS or PFOA.
- Since the tap water sampling in 2016, Horsham Township has taken additional measures for the public drinking water wells (including interconnections, purchasing water, and new filtration). These measures have reduced the system-wide combined average (in 2018) for PFOA and PFOS to approximately 0.004 µg/L, below ATSDR's adult and child EMEGs.

Tap water samples have not been collected in Warrington Township.

- Warrington Township public water users are now served by the North Wales Water Authority (NWWA).
- Monthly monitoring of finished drinking water performed at the NWWA Forest Park Water Treatment Plant from 2016-2019 found PFOS from non-detect to 0.0025 µg/L and PFOA from non-detect to 0.0045 µg/L.
- Maximum detected results for PFOS and PFOA at NWWA's Forest Park water treatment plant are currently well below the EPA HA and ATSDR EMEG values.

Private drinking water well users with PFOS and PFOA above the EPA HA have been provided bottled water or were connected to the public water supply.

- The Navy, Air Guard and local water utilities have completed the connections to public water for the majority of the private well water users with PFOS and PFOA contamination above the EPA HA.
- As of December 2019, on the Navy side only 7 properties still need a connection to the public water supply, and 2 of these properties have scheduled hookups.
- As of December 2019, on the Air Guard side, 16 properties were not yet connected (at one of these properties, the well is no longer in use).

Private drinking water well users with PFOS and PFOA below the EPA HA but above 0.04 µg/L are part of the Navy and Air Guard resampling plans.

Conclusion 4: On-Base Drinking Water Exposures

Based on the available limited sampling information, there was a past exposure pathway to PFAS in drinking water for civilian employees, service members, and their families on-base.

Basis for Conclusion ATSDR acknowledges a past exposure pathway for civilian employees, service members, and their families at this location. The maximum levels of PFAS detected in *on-base* drinking water supply wells (not delivered water at the tap) exceeded ATSDR's EMEGs for PFOS and PFOA for adults and children. In addition, the

maximum levels observed in on-base supply wells were higher than the levels seen in *off-base* public water supply and private drinking water wells.

- Historical on-base sampling and drinking water use information is very limited. ATSDR is lacking specific information about the populations that used the drinking water in the past and the distribution system on-base (such as potential blending of water sources).
- Therefore, ATSDR's ability to assess the public health implications of past on-base exposures to PFAS is limited.
- Consistent with the off-base contaminated drinking water supplies, the on-base drinking water wells are not currently used for consumption, and bottled water is used on-base for drinking water.

Conclusion 5: Surface Water and Biota Pathways

Community members may have been exposed to PFAS from non-drinking water sources in the site area, including the consumption of fish that may be contaminated with PFAS and incidental ingestion of PFAS while playing in the nearby creeks. Surface water ingestion is not likely to result in a significant additional exposure. The levels of PFAS in native (not stocked) local fish are not known.

Basis for Conclusion PFAS are present in surface waters surrounding the Willow Grove site, and community members (including children) may have recreational contact with these surface waters.

- Swallowing small amounts of PFAS during recreational activities in the nearby creeks is not likely to result in a significant exposure. Available data suggests that absorption of PFAS through the skin is not expected to be a major exposure route.
- PFAS can bioaccumulate in native (non-stocked) fish. People consume fish from local surface water bodies.
- Fish sampling for PFAS has not occurred nearby the Willow Grove site.

Conclusion 6: Breastfeeding

Based on available scientific information, ATSDR concludes that the health and nutritional benefits of breastfeeding outweigh the risks associated with PFAS in breast milk.

Basis for Conclusion Community members, particularly mothers who were exposed to PFAS in public and private drinking water supplies in the past, have expressed concern over the health implications of PFAS exposures to infants who breastfeed. Developmental effects are the most sensitive adverse health effects resulting from early life exposure to some PFAS. Studies have shown infants are exposed during pregnancy, through the mother to the fetus (maternal transfer) and occur to the nursing infant during breastfeeding. However, breastfeeding provides clear health and nutritional benefits, including protection from some illnesses and infections and reductions in the risks of developing asthma and sudden infant death syndrome.

In general, the Centers for Disease Control and Prevention recommends breastfeeding, despite the potential presence of chemical toxicants in breast milk. Given what we know about PFAS exposure, the benefits of breastfeeding outweigh any risks. However, the science on the health effects of PFAS exposure on mothers and children continues to expand. A woman's decision to breastfeed is an individual choice, made after consideration of many different factors (many unrelated to PFAS exposure) and in consultation with her healthcare providers. Information developed by ATSDR to guide doctors (see https://www.atsdr.cdc.gov/pfas/docs/pfas_clinician_fact_sheet_508.pdf) can aid in this decision-making process.

Considering the many health benefits of breastfeeding for mother and child, ATSDR recommends that nursing mothers continue to breastfeed. ATSDR recommends that a nursing mother who has specific concerns should consult her healthcare provider. ATSDR is available to consult with any healthcare provider, if needed.

Recommendations

To continue to protect the public health of residents drinking public and private drinking well water near the site, ATSDR recommends:

- The Navy and the Air National Guard should continue efforts to characterize the extent of the PFAS groundwater contamination near the site, as well as continue the ongoing private drinking well monitoring.
- The Navy, Air National Guard, and local authorities should continue outreach efforts for private drinking well owners within the study area that were not sampled for PFAS.
- The Navy and the Air National Guard, in coordination with EPA, PADEP, and the public drinking water authorities, continue implementing a long-term remedy to permanently mitigate public exposures to contaminated public and private drinking water sources at this site.
- Sampling of public water supplies should continue to ensure the effectiveness of the treatment systems. The treatment system should be adjusted, as necessary, to prevent exposure above the EPA HA and to reduce exposure to other PFAS.
- Community members with private drinking wells with water at or above the EPA HA for PFOS and PFOA should continue to reduce their exposure by using bottled water for drinking and cooking, until connections are made to the public water supply.
- The Navy and Air National Guard should characterize other non-drinking water environmental exposure pathways to PFAS in the area (e.g., biota sampling and off-site surface soil). Although federal and/or Pennsylvania screening values for PFAS in biota are not currently available, other state screening values are available for guidance.

- Community members choosing to further reduce their exposures to PFAS via home filtration systems should use only filters certified for the removal of PFAS and ensure their systems are maintained and the water tested to verify effective PFAS removal.
- Nursing mothers should continue to breastfeed. ATSDR recommends that a nursing mother who has specific concerns should consult her health-care provider. ATSDR is available to consult with any health-care provider, if needed. For formula-fed infants, caregivers are encouraged to use pre-mixed baby formula or reconstitute dry formula using alternative water sources that do not contain PFAS.
- PFAS levels in the public and private water supplies have been reduced. However, given the long half-life of several of these compounds and ongoing consumer product exposures to PFAS other than PFOS and PFOA, individuals in this community may still have a PFAS body burden greater than the general population.

ATSDR encourages any resident with long-term exposures or elevated PFAS in their blood to reduce exposures from all sources of PFAS. Community members should take steps to reduce their potential background sources of PFAS exposure, including avoiding or limiting the use of products containing PFAS. Examples of products that may contain PFAS include non-stick cookware, stain resistant carpets, water repellent clothing, cleaning products, make-up and personal care products.

Public Health Action Plan

In coordination with our agency partners of EPA, the Navy, Air National Guard, PADOH, local health departments, PADEP, and the local water authorities,

- ATSDR will continue to engage in national PFAS public health activities. As part of the Multi-Site Health Study, CD/ATSDR awarded funding in 2019 to RTI International and PADOH to look at PFAS exposures and human health effects in Montgomery and Bucks Counties. The multi-site study will collect information about the immune response, lipid metabolism, kidney function, thyroid disease, liver disease, glycemic parameters, and diabetes.
- ATSDR will continue attending public meetings to discuss the process of preparing this health consultation and community concerns upon the community's request. ATSDR will make presentations, develop handouts, and provide additional assistance as necessary to notify community members of the findings of this health consultation. ATSDR will notify the Navy, Air National Guard, EPA, local water authorities, and state and local health departments regarding the findings of this report and work to evaluate the protectiveness of mitigation action plans.
- ATSDR will continue to work with health partners to share and update health education information related to PFAS with community members and area health professionals as needed.

- The assessment of private drinking water wells in this document evaluates the maximum private well concentrations of PFAS. ATSDR is available, if requested, to evaluate and discuss individual private well sampling results and what the exposures mean for each individual family. Further, ATSDR will consider targeted health education outreach for the families who refused private drinking water well sampling or were not sampled and to those homes with the highest private drinking water well concentrations in the past.
- ATSDR will continue to partner with PADOH on their biomonitoring efforts in the community and to engage interested agency and community stakeholders in communications about ATSDR's PFAS exposure assessment and health study activities.
- ATSDR will continue to review and evaluate data, including private drinking water, surface water, and fish sampling data, provided for this site upon request.
- ATSDR will work with the Navy and the Air National Guard as they continue efforts to characterize the nature and extent of the PFAS groundwater contamination near the site, including ongoing private drinking water well monitoring and outreach efforts for private drinking well owners near the site that were not yet sampled for PFAS.
- ATSDR will continue to work with the EPA, the Navy and the Air National Guard as they implement a long-term remedy to permanently mitigate public exposures to contaminated public and private drinking water sources at this site.
- ATSDR recognizes that additional information is needed about the types of PFAS in AFFF and the type of AFFF used at Willow Grove. Standard laboratory methods capable of detecting a broader range of PFAS in environmental samples are also needed. As more information becomes available, ATSDR will incorporate it into future assessments of exposure to PFAS from sites associated with the use of AFFF.

Limitations

We must deal with several limitations and uncertainties when evaluating human health implications from PFAS exposures in drinking water. Because of these limitations and until better methods are developed, ATSDR used a conservative approach to evaluate the possibility for harmful health effects. ATSDR used a weight-of-evidence approach considering multiple exposures and factors. These included consideration of past body burdens, length of exposure, multiple PFAS in the water, contributions from other non-water sources, and similarity of health effects for various PFAS, all sources or factors that could contribute to overall health effects of PFAS exposures. Specifically:

- The amount of time and the levels at which people in the community were exposed to PFAS in their drinking water (private, public, and on-base water supplies) are uncertain. Depending on movement of the PFAS in the groundwater, ATSDR can reasonably assume that exposures to some residents may have occurred for many years. However, historical sampling data for PFAS are not available. Sampling of the off-site wells did not occur until 2014. Public water sampling occurred at the entry point to the distribution system for each well and may not reflect exposure at the tap, due to blending in the public water wells before

distribution.

- ATSDR's evaluation for this community is based primarily on the drinking water sampling results of six analyzed PFAS. However, because the type of AFFF used at the Willow Grove site and its specific PFAS formulation are unknown, the water sampling for six PFAS may not capture the full spectrum of exposures. Identification of new PFAS species in AFFF-impacted groundwater is ongoing. Little is known about the newly discovered PFAS.
- The health consequences of PFAS in the body are uncertain. Significant uncertainty remains about the lowest concentration at which toxic effects might occur in people exposed to PFAS for many years. Therefore, people exposed for many years could be at increased health risk.
- Toxicological information is insufficient to understand and evaluate the public health significance of exposure to PFAS contaminants at the levels and combinations seen in drinking water at this site. Much of the toxicological information regarding the effects of PFAS is based on animal studies. Doses of PFOA, PFOS, PFHxS, and PFNA in animal studies are higher than the levels found in the environment to which humans are exposed. Humans and experimental animals differ in how their bodies absorb and react to PFAS. This leaves questions about the relevance of animal effects to humans. Currently, toxicological information is limited on how all the different PFAS compounds detected in the water interact or if they have similar modes of potential toxicity.
- The science on the health effects of PFAS exposure on mothers and children continues to expand. Studies have shown infants are exposed during pregnancy through the mother to the fetus (maternal transfer), and exposures occur to the nursing infant during breastfeeding.
- Analytical methods are insufficient to identify and measure the full range of people's exposure to PFAS. Toxicological and epidemiological data is only available to support the development of health-based screening values for some of the PFAS that have been detected in water and in people's bodies. With the exception of the hazard index approach for PFOA, PFOS, PFHxS, and PFNA, there is not a broadly accepted scientific method to quantitatively evaluate the possible health effects of combined exposures to PFAS. Not all PFAS share the same health outcomes. Therefore, ATSDR evaluated the scientific literature to determine what health effects from the chemicals in the PFAS mixture found in the drinking water at this site might have similar health endpoints.

Attachment A details the supporting information for this evaluation and the conclusions. If you have further questions, please contact me at 215-814-3141 or via email at lkw9@cdc.gov.

Sincerely,

Lora Siegmann Werner, MPH
Agency for Toxic Substances and Disease Registry
Division of Community Health Investigations, Eastern Branch
Regional Director, Region 3

Attachment A: Data Review and Health Evaluation

Purpose

This letter health consultation provides an evaluation of the public health implications of past and current exposures to per- and polyfluoroalkyl substances (PFAS) contamination in public and private drinking water sources near the Willow Grove site. The document includes recommendations to the public to protect their health and the Navy and the Air National Guard to further characterize and reduce exposures near the site. ATSDR provided a public health evaluation of public and private drinking water supplies for the nearby Warminster site in January 2016 (ATSDR 2016b).

Contents

Purpose	12
Background and Statement of Issues	15
Site Background	15
ATSDR Evaluation Process	19
Exposure Pathway Analysis	19
Exposure and Health Effects	19
Identifying Chemicals of Concern	20
Drinking Water Exposure Pathway	21
Surface Water and Biota Pathways	22
Background Exposures to PFAS	22
Screening Analysis: Comparison to Health-based Comparison Values	23
Environmental Data	26
Horsham Water and Sewer Authority (HWSA) Public Water Supply Sampling	27
HWSA Past Exposures	27
HWSA Current Exposures	31
Warrington Township Water and Sewer Department (WTWSD) Sampling	34
WTWSD Past Exposures	35
WTWSD Current Exposures	37
Private Drinking Water Wells Sampling	37
Private Drinking Water Wells Past Exposures	37
Private Drinking Water Wells Current Exposures	41
On-Base Drinking Water Sampling	41
Surface Water and Biota Environmental Data	43
Exposure Assessment: Dose Calculations (Non Cancer)	44

Exposure Dose Calculations: HWSA Past Exposures	46
Exposure Dose Calculations: WTWSD Past Exposures	49
Exposure Dose Calculations: Private Drinking Water Wells Past Exposures	52
Estimated Excess Cancer Risk Evaluation	55
Public Health Implications	57
Exposures to PFOA and PFOS (individually).....	59
Exposures to Perfluorononanoic acid (PFNA).....	61
Exposures to Perfluorohexanesulfonic acid (PFHxS).....	62
Exposures to Perfluoroheptanoic acid (PFHpA).....	63
Exposures to Perfluorohexanoate (PFHxA).....	63
Exposures to Other PFAS	63
Exposure to a Mixture of PFAS	63
Susceptible Populations: Pre-existing Conditions and Developmental Effects	65
Cancer Effects	66
Cancer Incidence Data Reviews for this Community	68
PADOH PFAS Exposures Assessment Technical Tools Pilot Biomonitoring Project.....	70
Limitations and Uncertainties of Human Health Risks from PFAS Exposures.....	71
Multiple Exposure Sources	72
Lack of Historical Exposure Data	72
Incomplete Information on AFFF used at Willow Grove and Specific PFAS Formulations	72
Limited Information and Methods to Assess Public Health Implications	72
Community Concerns.....	73
Breastfeeding.....	73
Water Filtration	75
Biomonitoring	76
Acknowledgements	76
References	77

Embargoed Do not distribute

The following is a list of tables that support the evaluation in this attachment:

- Table A-1. PFAS detected in the public and private drinking water supplies near the Willow Grove site.
- Table A-2. Summary of Horsham public water well sampling results for PFAS (based on UCMR3 sampling results June 2014 – December 2015).
- Table A-3. Summary of Horsham tap water sampling results in µg/L (2016).
- Table A-4. Horsham Raw and Treated Supply Well PFAS Sampling Results (2018).

Table A-5. Summary of Warrington public water supply well sampling results for PFAS (based on UCMR3 sampling results November 2014 –December 2015).

Table A-6. Summary of Willow Grove private residential drinking water well sampling results for PFAS (as of April 2017).

Table A-7. On-base water supply well sampling results for PFOA and PFOS ($\mu\text{g/L}$), by building location.

Table A-8. Horsham public water: past PFOS environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Table A-9. Horsham public water: past PFOA environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Table A-10. Horsham public water: past PFHxS environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Table A-11. Horsham public water: past PFNA environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Table A-12. Horsham public water: Hazard Index for combined past exposures to PFOS, PFOA, PFHxS, and PFNA.

Table A-13. Warrington public water: past PFOS environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Table A-14. Warrington public water: past PFOA environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Table A-15. Warrington public water: past PFHxS environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Table A-16. Warrington public water: Hazard Index for combined past exposures to PFOS, PFOA, and PFHxS (PFNA= non-detect).

Table A-17. Private drinking water: past PFOS environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Table A-18. Private drinking water: past PFOA environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Table A-19. Private drinking water: past PFHxS environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Table A-20. Private drinking water: past PFNA environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Table A-21. Private drinking water: Hazard Index for combined past exposures to PFOS, PFOA, and PFHxS (PFNA=non-detect).

Table A-22. Estimated cancer risk calculations for PFOA in public and private drinking water.

Table A-23. PFAS and possible effects on organ systems.

The following is a list of figures that support the evaluation in this attachment:

Figure A-1. Willow Grove site layout.

Figure A-2. Map of public water systems in the site area.

Figure A-3. Public water sampling results by PFAS type (July 2014-December 2015).

Figure A-4. Average Horsham tap water PFOS sampling results by neighborhood (2016).

Figure A-5. Average Horsham tap water PFOA sampling results by neighborhood (2016).

Figure A-6. Private drinking water well sampling results near the Willow Grove site for PFOA and PFOS (as of September 2019).

Figure A-7. Private drinking well sampling results near the Willow Grove site by PFAS type (September 2014-April 2017).

Figure A-8. On-base water supply well sampling results for PFOA and PFOS (combined), by building location (June 2014-May 2016).

Background and Statement of Issues

PFAS are a class of man-made chemicals and only tested for in larger public drinking water supplies as of 2014. Although there are no federal regulatory levels for PFAS in drinking water, the EPA has issued a non-regulatory health advisory for PFOA and PFOS and some states have determined their own regulatory levels. PFAS have been used for decades as ingredients to make products that resist heat, oil, stains, grease, and water. Commercial and consumer products containing these compounds were introduced in the 1950s. PFAS have been used and found in a variety of products such as aqueous film-forming foam (AFFF), nonstick cookware, stain-resistant carpets, fabric coatings, some food packaging, makeup, and personal care products. PFAS have also been found in many industrial applications such as floor care and cleaning products (ATSDR 2018).

In 2013, EPA released the third Unregulated Contaminant Rule (UCMR3) that included sampling for PFAS. Under the UCMR3, EPA required all public water systems (serving >10,000 people) and a nationally representative sample of small systems (serving <10,000 people) to sample for up to 30 contaminants (including PFAS) that are not currently regulated. The six PFAS included in the UCMR3 sampling were perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), perfluorohexanesulfonic acid (PFHxS), perfluorobutane sulfonate (PFBS) and perfluoroheptanoic acid (PFHpA) (EPA 2015).

In 2014, EPA Region 3 asked ATSDR to evaluate past and current exposures to PFAS in public and private drinking water sources near military bases in Bucks and Montgomery Counties, Pennsylvania. The source of PFAS in groundwater near these bases is assumed to be the past use of aqueous film-forming foam (AFFF) at these locations. PFAS contamination in groundwater was discovered at the Willow Grove site in 2011 as part of ongoing environmental investigations and offsite in 2014 when public drinking water supplies were sampled for unregulated contaminants.

Site Background

The 1,200-acre site is comprised of the former Naval Air Station Joint Reserve Base (NASJRB) and current Air Reserve Station (ARS) at Willow Grove in Horsham, Montgomery County and Warrington Township, Bucks County, Pennsylvania (jointly referred as NASJRB-ARS) (Figure A-1). The installations are co-located within one perimeter, approximately 25 miles north of Philadelphia. Aircraft operations, including aviation and reserve training began at the site during the 1920s when the facility was named Pitcairn Airfield. NASJRB operations started in 1942 when the Navy acquired Pitcairn Airfield and established a classified anti-submarine program at this location. After World War II, the Navy used the site for reserve training, and jet training began in 1949. The Air Force base began operations in 1958. In 1994, the air station's name changed to NASJRB at Willow Grove to reflect the integration and joint operations of the Navy, Marine Corps, Air Force, Army Reservists, and the Pennsylvania Air National Guard (Horsham Land Redevelopment Authority 2016).

In 2001, the base employed 1,571 active-duty individuals, 993 members of the National Guard, 3,500 members of the Reserves, and 778 civilians with approximately 1,700 staff stationed on-base.

About 230 people resided on the bases year-round: around 30 people resided in single family dwellings and around 200 resided in barracks. Additionally, there were five officer family units, 200 enlisted family units, and 250 unaccompanied enlisted units as well as a daycare center on base for 96 children. The Willow Grove Branch Medical Clinic was also located onsite and provided primary care, medical support, preventive medicine, and occupational health services to 20,000 active duty reserve, retired personnel, and their family members (ATSDR 2002). Training activities and associated facility operations generated hazardous materials. These were stored and disposed of at the site (EPA 2016a).

In 1995, the NASJRB was added to the EPA National Priorities List (NPL) under the Comprehensive Environmental Response Compensation and Liability Act (CERCLA, also known as Superfund) because of groundwater contamination with volatile organic compounds (VOCs). In 2002, ATSDR produced a public health assessment evaluating drinking water, soil, surface water, sediment, and fish data from the site (ATSDR 2002). This public health evaluation pre-dated the site monitoring and detection of PFAS.

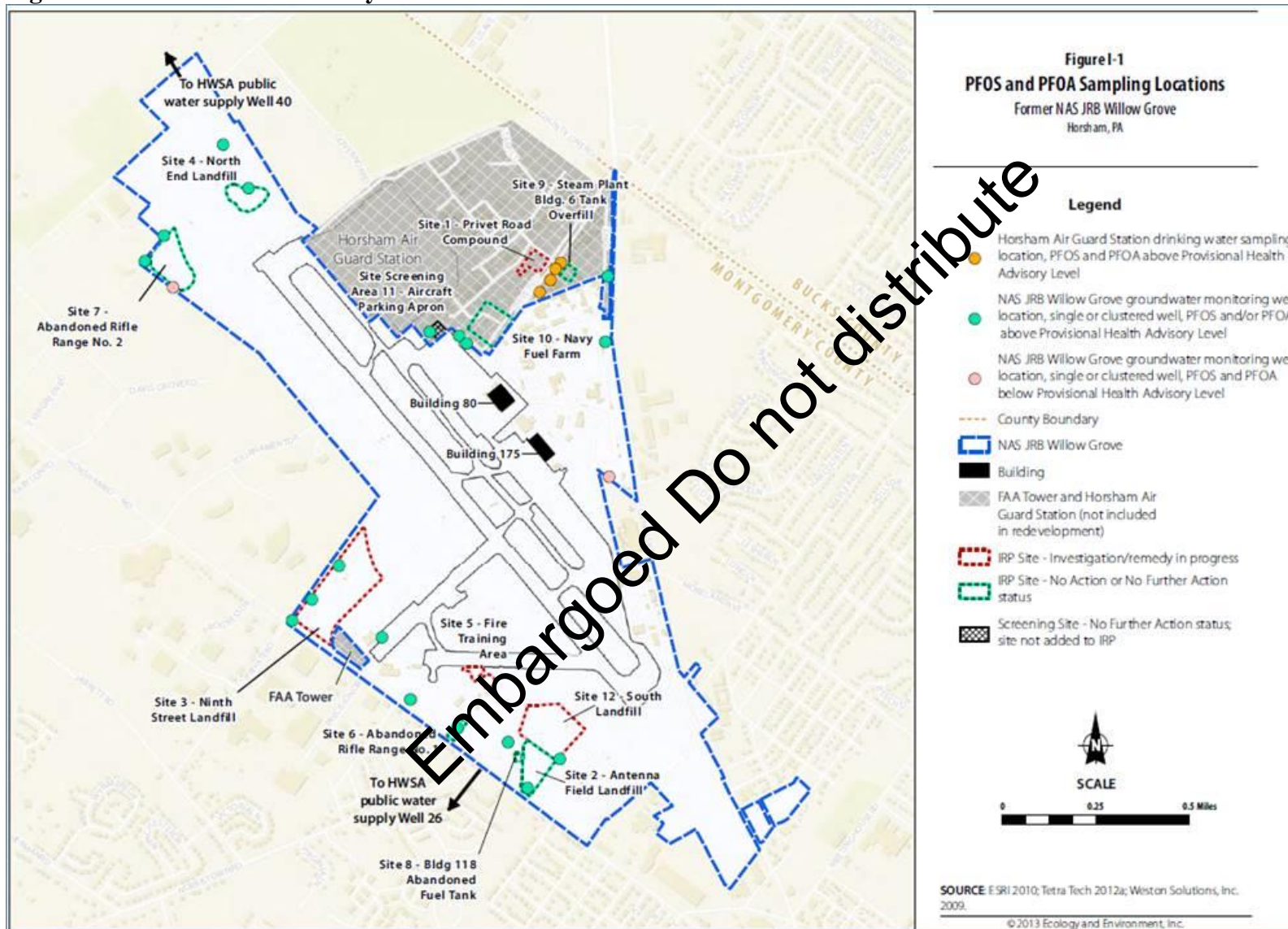
In 2005, base closure for NASJRB was initiated, under the DoD Base Realignment and Closure Program. The base officially disestablished in 2011, and Navy and Marine Corps squadrons and units moved to McGuire Air Force Base. The Navy transferred approximately 45 acres to the Air Force between 2009 – 2011, including two drinking water wells and other surplus facilities. As of 2018, the Navy continues to own their portion of the property and provide the necessary maintenance and security services until further property transfers occur. The 111th Fighter Wing of the Pennsylvania Air National Guard remains at their present site along with Army Reserve and Army National Guard units on the former U.S. Air Force Reserve Center facility. Currently, activities at the site include providing materials, facilities, services, and training in direct support of all units assigned to the stations (Horsham Land Redevelopment Authority 2016).

The Navy and Air National Guard continue to investigate and cleanup the site, under a Federal Facility Agreement between EPA and Pennsylvania Department of Environmental Protection (PADEP). Cleanups have been completed at several locations on the site including some of the former landfill locations (Sites 2 and 4), rifle ranges (Sites 6 and 7), and other sites (Sites 8-11). Investigations are ongoing at several other sites: Privet Road Compound (Site 1), Ninth Street Landfill (Site 3), Fire Training Area (Site 5), and South Landfill (Site 12). In 2011, limited groundwater sampling was conducted as part of the Site 5 Record of Decision. This sampling identified PFOS and PFOA as a potential concern to be re-evaluated during the Five-Year Review (Tetra Tech 2013). Figure A-1 below shows how the site is sub-divided and the on-site sampling locations for PFAS. The Navy and Air National Guard are currently further delineating the potential PFAS sources areas on-base.

Additional information on the Navy and Air National Guard sampling and site work can be found on their Administrative Records pages, at the following links:

- 1). Navy Administrative Record for Willow Grove at:
https://bracpmo.navy.mil/brac_bases/northeast/reserve_base_willow_grove/documents.html
and
- 2). Air National Guard Administrative Record for Willow Grove at:
<http://afcec.publicadmin-record.us.af.mil/search.aspx>

Figure A-1. Willow Grove site layout.



*Source: EPA. This site layout is used to show the potential source locations on-site. However, the PFOA and PFOS information on this map is based on the provisional health advisory for PFOA and PFOS.

In 2014, parallel to the ongoing investigations at the site, the Horsham Water and Sewer Authority (HWSA) sampled the public drinking water supply for PFAS. In 2014, Warrington Township Water and Sewer Department (WTWSD) voluntarily accelerated sampling for UCMR3, due to the detections of PFAS on-site and in nearby Horsham (EPA, USNAVY & PADEP 2015). For EPA's UCMR3 sampling nationwide, approximately 1% of the 5,000 public water systems monitored from 2013 to 2015 had combined PFOA and PFOS concentrations above the EPA Health Advisory (HA) of 0.07 µg/L (EPA 2016b). The primary PFAS in the public and private drinking wells near the site are PFOS and PFOA.

AFFF containing PFAS has been commercially available since the mid-1960s or 1970s (Prevedouros et al 2006; ATSDR 2018; NRL 2015). It is unknown when AFFF was first used at the site. Per the Navy's PFC Source Evaluation Report for NASJRB Willow Grove, all AFFF storage tanks and associated piping were emptied, cleaned, and closed in place with the residual AFFF transported off site for disposal in 2011. The former firefighting training area (Site 5) was used from 1942 to 1976 and is a likely source of PFOS and PFOA detected in the down gradient HWSA wells. Other potential source areas at the site include runways, plane crash sites, and hangar areas (EPA, USNAVY, PADEP 2015). In addition, an F-14 Tomcat crashed in an isolated 10-acre area north of the site in June 2000 and is referred to as the Aircraft Mishap Site. The Navy investigated the crash area because fuel and oil were released to the environment. The Navy conducted remedial actions at the location (Foster Wheeler 2000).

In 2014, the Navy developed a plan for additional on-site sampling for the Navy portion of the site to understand the extent of contamination and further identify sources of PFOS and PFOA. The Navy contracted with EPA to begin sampling private drinking water wells in the area. The Air National Guard is conducting parallel efforts on the Air National Guard portion of the Willow Grove site and in the nearby community. The Navy and Air National Guard established an interagency agreement with EPA to provide residents whose private drinking water wells were at or above EPA's former Provisional Health Advisory Levels (former PHALs), and subsequently for private drinking water wells at or above EPA's HA, with bottled water to use for drinking and cooking. Efforts are currently underway to connect these locations to public water. A subset of additional private drinking water wells with lower levels of PFAS (previously within 25% of the PFOS or PFOA former PHALs and now below the EPA HA but have a combined level of PFOA and PFOS above 0.640 µg/L) are being monitored through quarterly resampling. The Navy, the Air National Guard, EPA, HWSA and WTWSD are currently implementing a long-term plan to address concentrations of PFOA and PFOS above the EPA HA in drinking water sources near the site (EPA, USNAVY & PADEP 2015). HWSA and WTWSD, with support from the Navy and Air National Guard, have installed treatment systems on the public water wells impacted by PFAS contamination near the site.

The EPA HA is based on a lifetime exposure (70 years) and assumes a 20% relative source contribution of contaminant exposure from drinking water. The EPA HA of 0.07 µg/L for PFOS and PFOA combined was released in 2016. The EPA HA replaces the EPA former PHALs of 0.2 µg/L for PFOS and 0.4 µg/L for PFOA in drinking water that were based on short-term exposures. In the derivation of the EPA HA, the application of uncertainty factors offers a margin of safety and protection to the most sensitive populations, which is fetuses during pregnancy and breastfed infants (EPA 2016b; EPA 2016b).

ATSDR Evaluation Process

Exposure Pathway Analysis

ATSDR's health evaluations are exposure driven. Exposure might occur by eating food, breathing air, skin contacting with a substance, or drinking a substance containing the contaminant. A release does not always result in exposure. ATSDR evaluates site conditions to determine if people could have been (a past scenario), are (a current scenario), or could be (a future scenario) exposed to site-related contaminants. ATSDR also considers the route a substance takes from its source (where it is released to the environment) to points of exposure (where people contact the substance). This is an exposure pathway. An exposure pathway has five elements:

- 1) a source of contamination (e.g., spill or release)
- 2) an environmental media and transport mechanism (e.g., groundwater)
- 3) a point of exposure (e.g., tap water)
- 4) a route of exposure (e.g., drinking)
- 5) a receptor population (e.g., people potentially or actually exposed)

When all five elements are present, the exposure pathway is termed a completed exposure pathway. When evaluating exposure pathways, ATSDR identifies whether exposure to contaminated media (such as drinking water) has occurred, is occurring, or might occur. ATSDR also identifies an exposure pathway as completed, potential or eliminates the pathway from further evaluation. Exposure pathways are complete if all five elements of a human exposure pathway are present. A potential pathway occurs when one or more pathway elements cannot be proved or disproved. A pathway is eliminated if at least one element is missing (ATSDR 2005).

Exposure and Health Effects

At sufficient exposure levels, chemicals in the environment can cause harmful health effects. The type and severity of effects are influenced by complex factors that include the following:

- concentration (how much)
- the frequency or duration of exposure (how often and how long)
- the way the chemical enters the body
- combined exposure to other chemicals

Age, gender, nutritional status, genetics, health status, and other characteristics can affect how a person's body responds to an exposure and whether the exposure harms their health. When a completed exposure pathway is identified, ATSDR evaluates chemicals in that pathway by comparing exposure levels against screening values. The screening values are developed from available scientific findings about exposure levels and health effects. They reflect an estimated contaminant concentration that is not expected to cause adverse health effects for a given chemical, assuming a standard daily contact rate (such as amount of water consumed) and body weight. To be conservative and protective of public health, screening values are generally based on contaminant concentrations many times lower than levels at which no effects were observed in experimental animals or human studies. ATSDR does not use screening values to predict the occurrence of adverse health effects, but rather to serve as a health protective first step in the evaluation process.

Identifying Chemicals of Concern

Screening values include ATSDR's health-based comparison values (CVs).⁴ ATSDR develops CVs to screen environmental contamination for further evaluation. If contaminant concentrations are above these CVs, ATSDR reviews exposure variables (such as duration and frequency), the toxicology of the contaminant, and epidemiology studies for likelihood of possible health effects. During this part of the evaluation process, ATSDR estimates site-specific exposure doses and compares those to health guideline values. This health guideline comparison allows ATSDR to evaluate possible public health effects for relevant site-specific conditions. Health guidelines are developed based on data drawn from the epidemiologic and toxicological literature. Uncertainty factors (sometimes known as safety factors) are applied to ensure that the guidelines protect human health.

ATSDR's minimal risk levels (MRLs) and EPA's reference doses (RfDs) and cancer slope factors are the health guidelines most commonly used in the screening process. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without a detectable risk of adverse non-cancer health effects over a specified duration of exposure. An oral slope factor is an EPA derived estimate of the increased cancer risk from oral exposure to a dose of 1 mg/kg-day for a lifetime.

ATSDR MRLs are intended to serve as a tool to help public health professionals identify populations potentially at risk for health effects from exposure to a particular chemical. It is important to note that MRLs are a screening tool that help identify exposures that could be *potentially* hazardous to human health. Exposure above the MRLs does not mean that health problems will occur. Instead, it may act as a signal to health assessors to look more closely at a particular site where exposures may be identified. MRLs do not define regulatory or action levels for ATSDR.

The way an MRL is calculated depends on the type and quality of data available. MRLs can be set for three different time periods including acute (about 1 to 14 days), intermediate (from 15-364 days), and chronic (exposure for more than 365 days). ATSDR has developed over 400 human health MRLs. MRLs are developed for health effects other than cancer. For PFAS, ATSDR has developed provisional MRLs for PFOS, PFOA, PFHxS, and PFNA based on intermediate duration animal studies. ATSDR is using these intermediate MRLs to also screen and evaluate chronic exposures (ATSDR 2018).

ATSDR Minimal Risk Levels (MRLs)

- ATSDR MRLs are presented as a daily dose (mg/kg/day) and are an estimate of the amount of a chemical a person can eat, drink, or breathe each day without a detectable risk to health.
- MRLs serve as a screening values to help identify exposures that could be potentially hazardous to human health. Exposure above the MRLs does not mean that health problems will occur.
- MRLs are not intended to be used as public water standards or as a regulatory/action value.
- Because scientific information is often limited to animal studies, uncertainty (safety) factors are applied to MRL calculations resulting in lower, more conservative risk values.

⁴ ATSDR prioritizes use of CVs from ATSDR or other federal agencies. If these are not available, ATSDR may use CVs from other sources such as states. As the state of science on compounds progresses, more values may become available, and some values are revised from their current values.

MRLs undergo a rigorous review process. First, they are reviewed by ATSDR's expert toxicologists, an expert panel of external peer reviewers, and then an interagency MRL workgroup that includes participation from other federal agencies. Along with ATSDR, the other federal organizations on the MRL workgroup include CDC's National Center for Environmental Health (NCEH), the National Toxicology Program (NTP), National Institute of Occupational Safety and Health (NIOSH), and EPA. ATSDR's MRLs are then submitted for public comment (ATSDR 2018).

Per ATSDR public health assessment guidance, when no federal CVs were available, ATSDR used applicable state values to screen the data from this site for further evaluation. For contaminants for which there were no CVs (either federal or state), ATSDR automatically retained these contaminants for further evaluation in this analysis. ATSDR considered if the exposure to multiple PFAS and other sources of exposure would also have an impact on health. It is important to note that CVs can change over time as new toxicological data and information become available. The following sections describe the evaluation process in more detail, focusing first on who was potentially exposed (the exposure pathway analysis). We then consider the chemicals identified for further evaluation (the screening analysis) and finally discuss the public health implications of exposure (ATSDR 2005).

Drinking Water Exposure Pathway

Exposures to contaminants in drinking water can include ingestion exposure (from drinking water, cooking with water, incidental ingestion during showering), dermal exposure (from bathing, showering or dishwashing), and inhalation exposure (from bathing or showering).

ATSDR considers ingestion of the water the most significant exposure pathway at this site. PFAS do not easily volatilize during bathing and showering, and absorption of PFAS through skin is slow or limited. Breathing in PFAS (outside the occupational setting) and absorbing PFAS through the skin are not significant routes of exposure due to the physical characteristics of this chemical class (Prevedouros 2006, ATSDR 2018). Therefore, dermal and inhalation exposures via household use of PFAS contaminated drinking water were not evaluated further in this document.

Persons exposed to drinking water at this site include: adults and children offsite that consumed the PFAS contaminated public and private drinking water, and workers and service members on the base that consumed PFAS contaminated water. Pregnant women who consumed PFAS contaminated water on or offsite could have transmitted their exposure to their developing fetus in utero and to infants by breastfeeding.

Willow Grove PFAS Exposure Pathway Summary

Drinking Water: Completed (past, current, future); Evaluated

Surface Water: Completed (past, current, future); Not evaluated due to physical characteristics of PFAS

Soil: Potential (past, current, future); Not evaluated due to lack of data

Background Exposures (including Dermal): Completed (past, current, future); Not evaluated due to lack of data

Ambient Air: No completed pathway; Not evaluated

Due to the actions of the Navy and Air National Guard, current exposures in the public and private drinking water supplies to PFOA and PFOS are below the EPA HA. Concurrent exposures to exceedances of PFNA (in private wells) and PFHxS (in private and public water supply wells) above comparison values have been addressed by these actions as well. The potential exposure to levels of PFAS in public and private water supplies above the EPA HA are a past completed exposure pathway at this site. For private drinking water users whose wells have not been tested, there may be a current completed pathway of concern.

The next step in the ATSDR evaluation process is to compare the drinking water sampling data to health-based CVs, including Environmental Media Evaluation Guides (EMEGs) derived from the ATSDR provisional MRLs for PFOS, PFOA, PFHxS, and PFNA. See the Screening Analysis: Comparison to Health-based Comparison Values section later in the document for details.

Surface Water and Biota Pathways

Exposure to PFAS from non-drinking water sources, including the consumption of native (non-stocked) fish that may be contaminated with PFAS near the site and incidental ingestion of PFAS while playing in the nearby creeks, could add to the body burden and the potential risk. PFAS are present in surface waters surrounding the Willow Grove site, and community members (including children) may have recreational contact with these surface waters. Therefore, this is a completed exposure pathway at the Willow Grove site. However, swallowing small amounts of PFAS during recreational activities in the nearby creeks is not likely to result in a significant exposure.

Absorption of PFAS through the skin is not a major exposure route due to the physical characteristics of PFAS. These same characteristics cause most PFAS to remain in the water column and not separate into sediment or become volatilized into the air, thereby reducing the likelihood of exposures via contact with creek sediment or breathing while swimming. Given the physical properties of this chemical class and the limited ingestion and dermal contact exposures that occur during recreational activities with surface water, ATSDR considers exposure from this pathway to be minor. Therefore, ATSDR did not evaluate the surface water pathway further in this document.

PFAS can bioaccumulate in native fish and wild game. This would be less of a concern for stocked fish. People consume fish from local surface water bodies in the site area. Therefore, biota pathway is a potential past, current and future completed exposure pathway at this site as well. Some states, such as Minnesota and Michigan, have issued guidance on fish sampling for PFOS and fish consumption advisories based on PFOS levels in fish fillet samples (Minnesota 2018; Michigan 2018). Community members near the site may have been exposed currently and in the past to PFAS in fish near the site. However, currently fish sampling has not occurred nearby the Willow Grove site. Therefore, ATSDR is not able to further evaluate this potential exposure pathway at this time. ATSDR is available to evaluate future fish sampling data collected near the site in consultation with our partners in Pennsylvania, if any such data are collected.

Background Exposures to PFAS

ATSDR acknowledges this community's background exposures to PFAS as a component of their overall PFAS exposures. However, further evaluation of this exposure pathway is outside the scope of this document. We do not have enough information to identify the individual exposure sources and to estimate the background exposure levels. Those sources might include PFAS-contaminated food, hand-to-mouth transfer from surfaces previously treated with PFAS-containing stain protectants (e.g., carpet, which is most significant for infants and toddlers), personal care and

cleaning products, or eating food packaged in material containing PFAS. Oral exposure through food and water is considered the major exposure pathway for the general population. Exposure to PFAS is widespread in the population. There are multiple sources of these compounds in air, indoor dust, food, water, and consumer products. Studies show that nearly all people have some PFAS in their blood regardless of age (Wu, X.M. et.al 2015; Kato, K. et al, 2011; Calafat, A.M. et al 2007).

Background levels of 12 different PFAS in the blood serum of the United States population are monitored at regular intervals through the National Health and Nutrition Examination Survey (NHANES) (CDC 2019). PFOA, PFOS, PFNA, and PFHxS were detected in the blood of 95-99.8% of samples of people's blood collected in the U.S. (Kato, K et al, 2011). In the U.S. general population, the mean serum PFOA and PFOS concentrations in 2015-2016 were 1.56 and 4.72 ng/mL, respectively. As summarized in Kato K et al (2011), the "NHANES 1999-2008 PFAS biomonitoring data are consistent with reduced population exposure to PFOS because of efforts of industry and government. For PFHxS, the data suggest reduced exposure from 1999 to 2006, but increased exposure during 2007-2008. The NHANES data also suggest that during 2003-2008, PFOA exposure, although significantly lower than in 1999-2000, has remained essentially unchanged while for PFNA, human exposure during 1999-2008 has shown an upward trend." Additional time series analyses and further assessment of sociodemographic factors, which likely influence lifestyle choices, by PFAS type are important to continue to understand different PFAS exposures and trends in the U.S. population.

Due to their chemical structure, PFAS are very stable in the environment and are resistant to biodegradation, photooxidation, direct photolysis, and hydrolysis (ATSDR 2018). Similar to other communities impacted by PFAS in their drinking water, community members near the site exposed to the highest level of PFAS may have higher PFAS serum levels than the general population. As discussed later in the document, the Pennsylvania Department of Health (PADOH) implemented a pilot biomonitoring program to study PFAS levels in the blood of community members from this area. Preliminary results of this pilot study were released in December 2018, and a final report was released in April 2019 (PADOH 2019). The work done by PADOH served as a pilot for CDC/ATSDR's PFAS exposure assessments and contributed to the overall body of knowledge on PFAS exposure. PADOH expanded the pilot exposure assessment to include urine and additional environmental sampling in late 2019.

Screening Analysis: Comparison to Health-based Comparison Values

Following identification of a completed/potential exposure pathway, ATSDR conducted a screening analysis of detected chemicals against available health-based CVs. Due to the lack of available comparison values for some PFAS, not all of the CVs used to screen the data were from ATSDR or other federal agency sources. As the state of science on these compounds progresses, more values may become available, and CVs can be lowered or raised in the future. In the interim, state and international health-based values were also used to screen the data. ATSDR has not fully evaluated the toxicological basis for these state and international health-based screening values.

In general, CVs are conservative and include uncertainty or safety factors that account for the most sensitive populations as well as uncertainties in translating effects observed in laboratory animal experiments to potential human health effects. Applying uncertainty factors results in a more conservative CV. Most animal studies have tested doses of PFAS that are higher than the levels found in the environment to which humans are exposed. Because the scientific information on a

hazardous substance is often limited to animal studies and might not include human studies, uncertainty factors might be applied as part of the calculations resulting in lower, more conservative values. Uncertainty factors help us account for differences among what is observed in animals, as compared to humans; or when we do not know certain things about how a chemical might affect a sensitive population (for example, the very young, or people who might have other health problems); or when we do not have complete information about the exposure levels causing health effects.

Adverse health effects are not expected to occur if an exposure concentration is below a CV. However, an exposure concentration at or above the CV does not automatically mean adverse effects will occur. Rather, it means that there is a need to conduct a site-specific exposure evaluation. The health risk for an individual depends on individual human factors (e.g. personal habits, occupation, and overall health), and site-specific environmental exposure factors (e.g. duration and amount of exposure). Therefore, the comparison values are not used to predict the occurrence of adverse health effects without looking at site-specific conditions. Contaminants for which there were no health-based CVs (either federal or state) were retained for further evaluation and discussed further in the document, per the ATSDR Public Health Assessment Guidance Manual (ATSDR 2005).

ATSDR used the provisional ATSDR intermediate MRLs for PFOA, PFHxS, and PFNA to calculate Environmental Media Evaluation Guide (EMEG) values to screen the public and private drinking water results. ATSDR EMEGs represent concentrations of substances in water, soil, and air to which humans may be exposed during a specified period of time (acute, intermediate or chronic) without experiencing adverse health effects. Acute exposures are defined as those of 14 days or less; intermediate exposures are those lasting 15 days to 1 year; and chronic exposures are those lasting longer than 1 year. ATSDR EMEGs can be calculated for substances for which ATSDR has developed MRLs that are presented in Toxicological Profiles using exposure assumptions. The EMEGs for drinking water are calculated based on the following equation (ATSDR 2005):

$$\text{ATSDR Drinking Water Environmental Media Evaluation Guide (EMEG) } (\mu\text{g/L}) = \frac{\text{ATSDR Provisional MRL (mg/kg/day)} / 1000 \times \text{Body Weight (kg)}}{\text{Drinking Water Ingestion Rate (L/day)}}$$

To calculate EMEG values, ATSDR used default exposure assumptions defined by specific age groups with corresponding exposure doses for each age group (ATSDR 2005). Body weights were selected based on data from NHANES 1999 – 2006, as reported in the USEPA Exposure Factors Handbook (EPA 2011). For child EMEG values, ATSDR used a daily drinking water ingestion rate of 1.1 liters per day and a body weight of 7.8 kilograms (kgs) or approximately 17 pounds. To calculate an adult EMEG, ATSDR applied a daily drinking water ingestion rate of 3.09 liters per day and a body weight of 80 kgs or approximately 176 pounds.

The federal and state health-based screening values for the PFAS compounds utilized in this assessment are further described below. Table A-1 summarizes PFAS exceeding CVs in the public and private drinking water supplies near the Willow Grove site.

Table A-1. PFAS detected in the public and private drinking water supplies near the Willow Grove site.

Contaminant	Health-Based CV (µg/L) *	Detected above Available CV	Detected above Available CV	Detected above Available CV
		Horsham Public Water Supply±	Warrington Public Water Supply	Private Drinking Water Wells
PFOS	0.014** ATSDR intermediate child EMEG**	Yes	Yes	Yes
PFOA	0.021 ATSDR intermediate child EMEG**	Yes	Yes	Yes
PFHpA	No CV available	-	-	-
PFHxS	0.14 ATSDR intermediate child EMEG**	Yes	Yes	Yes
PFHxA	No CV available	-	-	-
PFNA	0.021 ATSDR intermediate child EMEG**	No	No	Yes
PFBS	2 Minnesota Chronic Health-Based Value	No	No	No

* The current CVs in drinking water are based on the best available scientific information. However, as new studies and information become available CVs can change.

** ATSDR intermediate Child Environmental Media Evaluation Guide (EMEG) is calculated from the ATSDR provisional intermediate Minimum Risk Levels and assumes a body weight of 7.8 kg and ingestion of 1.1 liters per day. Please visit the following for more information on EMEGs for PFAS: https://www.atsdr.cdc.gov/pfas/mrl_pfas.html

±In addition to the UCMR3 PFAS constituents (PFOA, PFOS, PFHxS, PFNA, PFBS, and PFHpA), in 2015 HWSA also analyzed water samples for the following PFAS: PFHxA, PFDA, PFDoA, PFTA, PFTDA, and PFUA. See Table A-2 for specifics.

Perfluorooctane sulfonic acid (PFOS)

ATSDR used the child EMEG value of 0.014 µg/L as a screening value for PFOS in public and private drinking water wells.

Perfluorooctanoic acid (PFOA)

ATSDR used the child EMEG value of 0.021 µg/L as a screening value for PFOA in public and private drinking water wells.

Perfluoroheptanoic (PFHxS)

ATSDR used the child EMEG value of 0.14 µg/L as a screening value for PFHxS in public and private drinking water wells.

Perfluorononanoic acid (PFNA)

ATSDR used the child EMEG value of 0.021 µg/L as a screening value for PFNA in public and private drinking water wells.

Perfluorobutane sulfonate (PFBS)

Currently, ATSDR does not have a CV for drinking water exposures to PFBS. In 2017, Minnesota developed a health-based CV for PFBS of 2 µg/L for chronic non-cancerous health effects. The Minnesota health-based CV for PFBS is based on a chronic reference dose of 0.00043 mg/kg/day. The critical effects endpoints observed in laboratory animals were kidney epithelial and tubular/ductal hyperplasia, and the co-critical effects were focal papillary edema and necrosis in the kidney. The Minnesota health-based CV includes an uncertainty factor of 300 (3 for interspecies extrapolation, 10 for interspecies variability, 3 for database insufficiencies, and 3 for the use of a subchronic study) (Minnesota Department of Health 2017). ATSDR used this Minnesota CV to screen the PFBS results in this document.

Perfluorohexanoic Acid (PFHxA)

PFHxA was only sampled for in the Horsham public drinking water supply. Currently, ATSDR does not have a CV for drinking water exposures to PFHxA. Very limited information is available relating to the health effects of ingesting PFHxA. One study evaluated the chronic oral (ingestion) toxicity of PFHxA in laboratory animals (Klaunig et al. 2015). Exposure to female rats to 200 mg/kg/day resulted in hematological alterations (decreases in red blood cells, hemoglobin levels, and increases in reticulocyte counts), renal effects (tubular degeneration, necrosis, increased urine volume, and reduced specific gravity), and liver effects (necrosis). No adverse alterations (No Observed Adverse Effect Levels, or NOAELs) were observed at 30 mg/kg/day (females) or at 100 mg/kg/day (males). One major uncertainty related to this study is that serum PFHxA levels were not measured. As previously mentioned, PFHxA has only limited animal and human studies, has not been studied as extensively as PFOA or PFOS, and the only identified chronic study has limitations. PFHxA has not been studied as extensively as PFOS, PFOA, PFHxS, and PFNA (the PFAS with ATSDR provisional MRLs) and the only identified chronic study has limitations. This PFAS is short-chained and not sulfonated, indicating it less bioaccumulative (EPA 2018b). The health effects of many short-chained PFAS have not been fully researched. Given the lack of information, ATSDR cannot make any health conclusions for PFHxA at this time.

Perfluoroheptanoic acid (PFHpA)

Currently, ATSDR does not have a CV for drinking water exposures to PFHpA. Very limited information is available in the scientific literature (in either human or animal studies) relating to the health effects of exposure to PFHpA. ATSDR identified several human studies (e.g., for cardiovascular disease, serum lipids, immune responses, etc.) that were either limited or found no association. No animal or other studies for PFHpA were identified to allow ATSDR to make a comparison between the exposure dose from drinking water to effect levels (i.e., NOAELs or LOAELs). Therefore, we cannot make any health conclusions for PFHpA at this time.

Environmental Data

This section describes the PFAS concentrations in drinking water collected for the public and private drinking water supplies and the comparison of these values to the selected health screening CVs. Provided below are detailed description of PFAS in drinking water supplies for past and

current exposures for HWSA, WTWSD, and private drinking water wells. This section also includes a summary of the available surface water and biota environmental data.

Horsham Water and Sewer Authority (HWSA) Public Water Supply Sampling

The HWSA currently serves approximately 7,300 domestic accounts, and HWSA estimates this represents approximately 22,900 people served. The HWSA provides service mainly to Horsham Township (19044) and small portions of Ambler (19002) and Hatboro (19040). The HWSA is served by 15 wells as well as interconnections with other nearby water utilities. HWSA is comprised of two zones - a high-pressure zone and a low-pressure zone. The high zone has two storage tanks supplied by three wells and two interconnections. The low zone has three storage tanks served by 11 wells and an interconnection with Aqua Pennsylvania Southeastern Division. All of the HWSA wells with PFOS and/or PFOA detections above the EPA HA were located in the low-pressure zone. HWSA's low-pressure zone serves the northeastern and southeastern portions of the Township and comprises approximately two-thirds of the system. The Willow Grove site is entirely located in the low-pressure zone with the boundary between the two zones falling generally at the far western edge of the base. Figure A-2 provides a map depicting the site area and public water service areas, including the high and low pressure zones in the HWSA system. (Note, the nearby Former Naval Air Center Warminster and the Warminster Municipal Water Authority are evaluated in an earlier ATSDR health report and are also included on this map.)

The wells in each zone pump simultaneously to fill tanks and demand is generally met by water in those tanks. The elevated tanks in the low zone generally supply water to a certain area of the system. Each tank will have different PFAS concentrations depending on which wells are supplying water to them. If water is moved between the zones due to demand, it usually occurs via gravity from the high zone to the low zone. If a property near a well has a demand surge at the same time that well is pumping, it is possible that the water at the tap at that property would have a higher blend of water from that nearby source. Prior to 1996 the system did not have pressure zones which means customers located in the current high-pressure zone may have received water from wells in the low-pressure zone (ATSDR 2011).

The maximum measured concentrations of PFOS (1.0 µg/L in well #40) and PFOA (0.29 µg/L in well #40) in the HWSA public drinking water system were detected in June 2014. However, the duration of this concentration in the water distribution system is not known, and we do not know if higher concentrations occurred in the past. More detailed analyses of the system would be needed to estimate historical PFAS concentrations at specific housing areas. These analyses would involve looking at the water-distribution system operating conditions, historical monthly well pumping records, and customer consumption information in more detail. Given the mixing within HWSA and zones, there is some uncertainty about past exposure levels at individual residential taps.

HWSA Past Exposures

Sampling data for PFAS in the HWSA system is not available prior to 2014. This represents an important data gap and limitation in ATSDR's exposure assessment. ATSDR reviewed the PFAS sampling results from the HWSA through December 2015 (Table A-2 and Figure A-3).

The HWSA system consists of 15 public water supply wells. In June 2014, the 14 out of 15 HWSA water supply wells active at that time were sampled for PFAS (PFOA, PFOS, PFHxS, PFNA, PFBS, and PFHpA), as part of the UCMR-3.

Figure A-2. Map of public water systems in the site area.

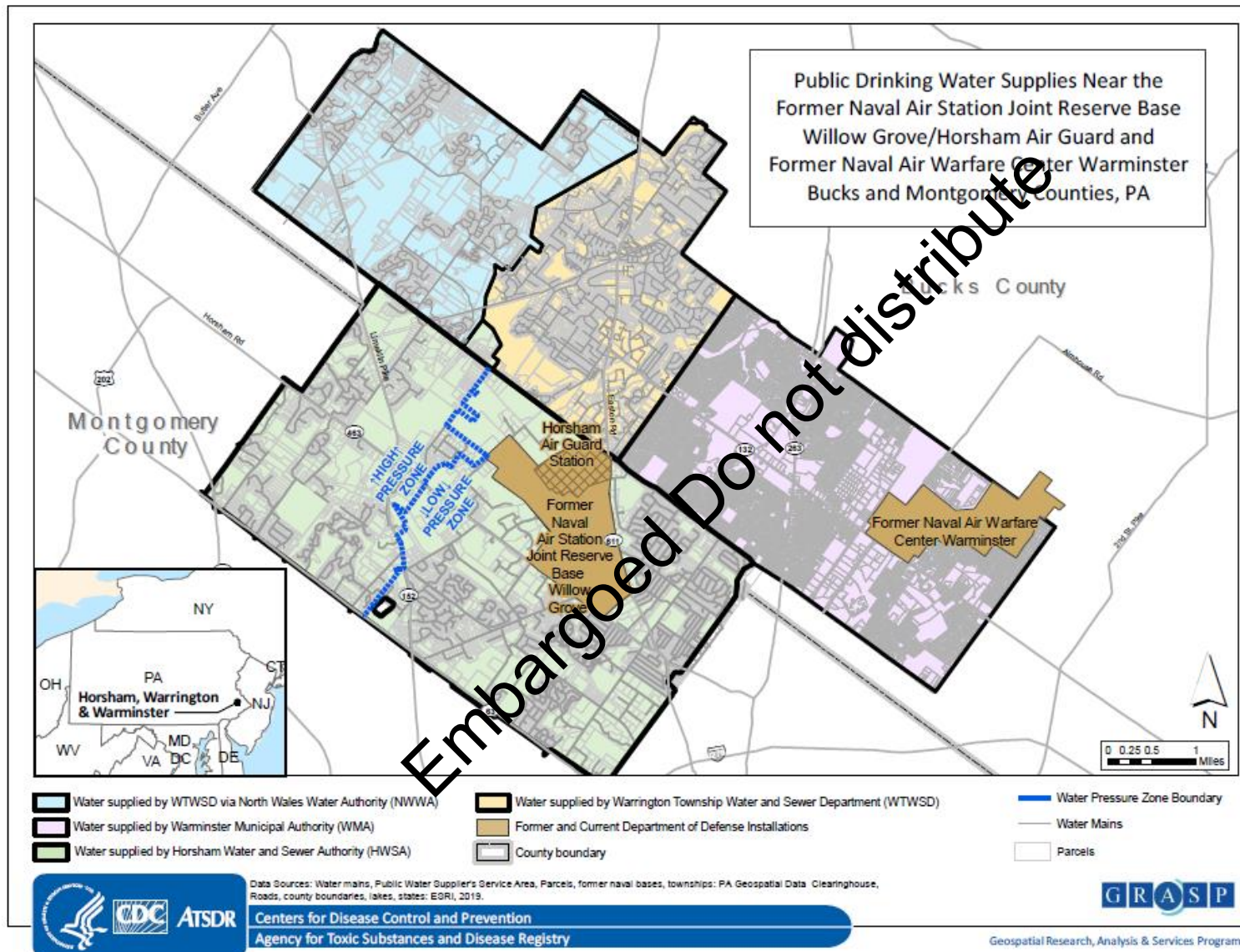
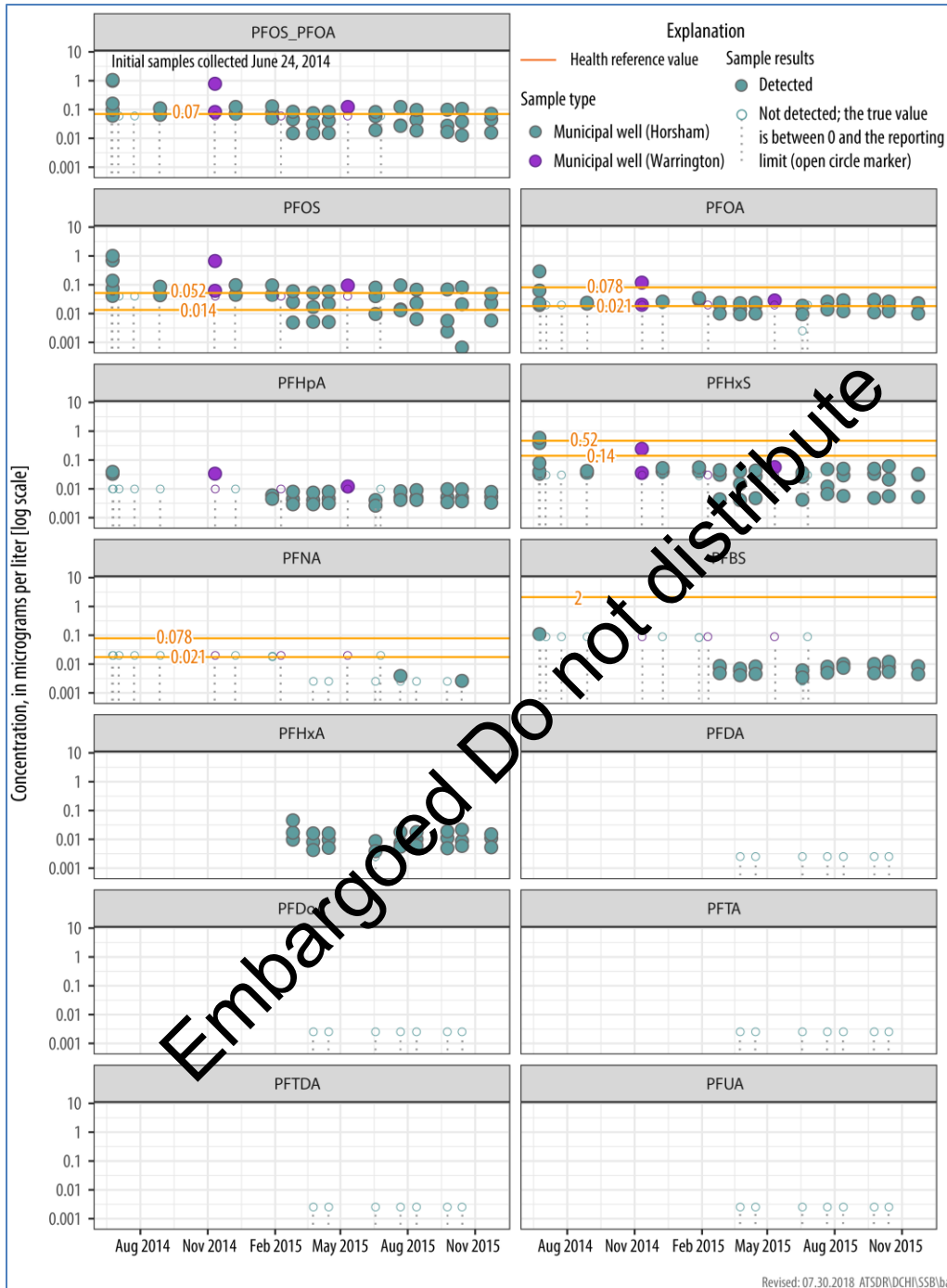


Figure A-3. Horsham public water sampling results by PFAS type (June 2014 – December 2015).



Notes: PFOS= perfluorooctane sulfonate; PFOA= perfluorooctanoic acid; PFHpA = perfluoroheptanoic acid; PFHxS = perfluorohexane sulfonate; PFHxA = perfluorohexanoic acid; PFNA = perfluorononanoic acid; PFBS = perfluorobutane sulfonate; PFDA = perfluorodecanoic acid; PFDoA = perfluorododecanoic acid; PFTA = perfluorotetradecanoic acid; PFTDA= perfluorotetradecanoic acid); PFUA = perfluoroundecanoic acid. PFDA, PFDoA, PFTA, PFTDA, and PFUA results only in Horsham public water wells (all non-detect)

Health reference/comparison values: PFOA – ATSDR intermediate EMEG of 0.021 µg/L (children) and 0.078 µg/L (adults); PFOS ATSDR intermediate EMEG of 0.014 µg/L (children) and 0.052 µg/L (adults); PFHxS- ATSDR intermediate EMEG of 0.14 µg/L (children) and 0.52µg/L (adults); PFNA = ATSDR intermediate EMEG of 0.021 µg/L (children) and 0.078 µg/L (adults); PFBS = 2 µg/L (Minnesota Health-Based Comparison Value).; no CVs for PFHpA, PFHxA, PFDA, PFDoA, and PFTA. The current CVs in drinking water are based on the best available scientific information. However, as new studies and information become available CVs can change.

Table A-2. Summary of Horsham public water well sampling results for PFAS (based on sampling results June 2014 – December 2015).

Contaminant	Health-Based Comparison Value (CV)(µg/L)	Detection Limit (µg/L)	Minimum	Minimum Detected Value (µg/L)	Max Detected Value (µg/L)	Mean (µg/L)†	Wells Above CV
PFOS	0.014 ATSDR intermediate child EMEG	0.0025-0.04	ND	0.0025	1.0	0.068	5
PFOA	0.021 ATSDR intermediate child EMEG	0.0025-0.02	ND	0.0096	0.29	0.024	4
PFHpA	No CV	0.0025-0.01	ND	0.0026	0.039	0.0066	-
PFHxS	0.14 ATSDR intermediate child EMEG	0.0025-0.03	ND	0.0041	0.59	0.045	2
PFHxA	No CV	0.0025	ND	0.0028	0.046	NA‡	-
PFNA	0.021 ATSDR intermediate child EMEG	0.002-0.025	ND	0.0026	0.0039	NA‡	0
PFBS	2 MN chronic Health-Based Value	0.0025-0.09	ND	0.0032	0.11	0.009	0

Notes: Table presents sampling data, provided by HWSA from the 14 municipal wells. The maximum PFOS and PFOA concentrations were 1.0 µg/L and 0.29 µg/L. The maximum combined PFOA and PFOS in individual wells was 1.06 µg/L (Well 40). Wells 10, 17, 21 are equipped with temporary treatment systems and were restored to service to the public supply on 4/11/17, 1/12/17 and 1/13/17 respectively. Wells 26 & 40 are equipped with permanent treatment systems and were restored to service to the public supply on 3/16/17 and 4/12/17, respectively. HWSA wells 26 and 40, which were taken off-line in October 2014, remain off-line. The HWSA wells with detections of PFHxS above the ATSDR child EMEG of 0.14 µg/L were well #'s 26 and 40. The current CVs in drinking water are based on the best available scientific information. However, as new studies and information become available CVs can change.

† ATSDR used the detection limit to substitute for non-detect values.

‡ The percent of non-detects in these datasets were high (>90% non-detects) and therefore a mean could not be calculated

ND= Non-detect; NA = Not available; PFOS = Perfluorooctane sulfonate; PFOA = Perfluorooctanoic acid; PFHpA = Perfluoroheptanoic acid; PFHxA= Perfluorohexanoate; PFHxS = Perfluorohexylsulfonic acid; PFNA = Perfluorononanoic acid; PFBS = Perfluorobutanesulfonic acid; EMEG= ATSDR Environmental Media Evaluation Guide

Two HWSA public wells (wells #26 and well #40) had levels of PFOS greater than the EPA former PHAL of 0.2 µg/L. In 2014, HWSA well # 26 and #40 were taken off-line and customers were alerted. The two contaminated wells generally supplied about 25% of the water for the system;

however, there were times that the two contaminated wells supplied as much as 35% of the water for the system.

According to the 2014 consumer confidence report for the HWSA, the average level of PFOS reported was 0.06 µg/L, the average level of PFHxS was 0.037 µg/L, and the average level of PFOA was non-detect (Horsham Water and Sewer 2014).

In addition to the PFAS included in the UCMR-3 analyte list, in 2015, HWSA also analyzed for six additional PFAS (PFHxA, PFDA, PFDoA, PFTA, PFTDA, and PFUA). With the exception of PFHxA and PFNA, these PFAS were not detected above the method reporting limit of 0.0025 µg/L during the sampling event. PFHxA was detected in a range of non detect to 0.046 µg/L, and PFNA was detected in a range of non detect to 0.0039 µg/L. ATSDR does not have a CV for PFHxA. The PFNA detections did not exceed ATSDR’s intermediate child EMEG CV.

In May 2016, following the release of the EPA HA for PFOA and PFOS, wells #s 10, 17, and 21 were also taken out of service. One of these three wells was shut down as a cautionary and voluntary decision, based on detections above the EPA’s HA (well #10). The other two wells (well #s 10 and 21) generally had levels below the HA but had tested above the EPA HA once and were shut down as a precaution. The other nine wells that now supply public drinking water across the township have showed levels of PFOS and PFOA below the EPA HA and below ATSDR’s EMEGs.

The results showed PFOS and PFOA were detected above the ATSDR child EMEG in five public HWSA wells. The maximum PFOS and PFOA concentrations were 1.0 µg/L and 0.29 µg/L, respectively. The maximum combined PFOA and PFOS in an individual well was 1.06 µg/L (Well # 40). PFBS was detected at maximum concentration 0.11 µg/L in the HWSA system, which is below the Minnesota CV of 2 µg/L. PFNA was detected at a maximum of 0.0039 µg/L, which is below the ATSDR child EMEG. PFHxS was detected in HWSA public water sampling in two wells (maximum value of 0.59 µg/L in HWSA well #40), exceeding the ATSDR child EMEG value.

HWSA Current Exposures

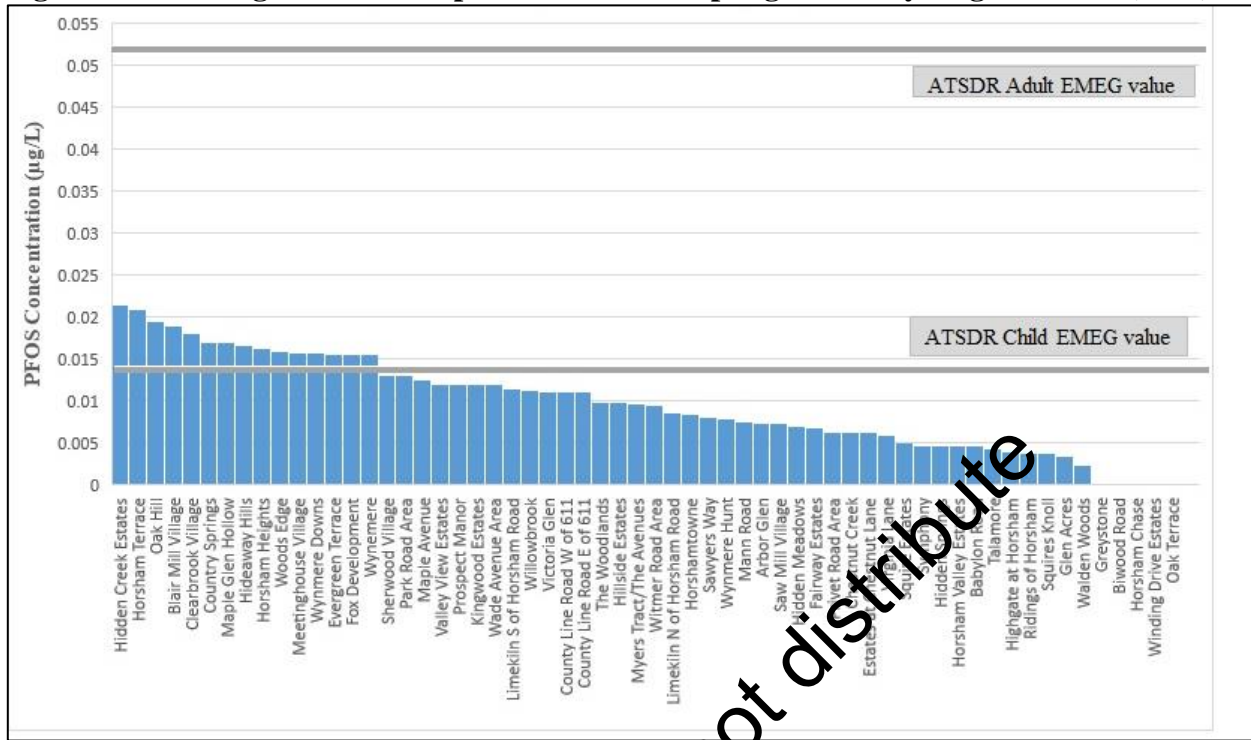
In August 2016, Horsham Township voluntarily and proactively collected water samples from 109 public water users at point-of-use locations (i.e., residential tap samples). The purpose of this sampling was to assist in determining the effectiveness of the employed short-term measures including carbon filtration of the public drinking water wells and taking public wells off-line. Table A-3 below provides a summary of the tap water sampling data in Horsham and Figures A-4 and A-5 provide an overview by neighborhood sampled (Horsham Township 2016).

Table A-3. Summary of Horsham Tap Water Sampling Results (2016).

	Horsham Tap Water Results PFOS	Horsham Tap Water Results PFOA	Horsham Tap Water Results PFOS+PFOA
Average (µg/L)	0.0102	0.0073	0.0168
Maximum (µg/L)	0.028	0.015	0.041 (in a given tap sample)
# Samples > child EMEG	27 (24%)	0	
# Samples > adult EMEG	0	0	

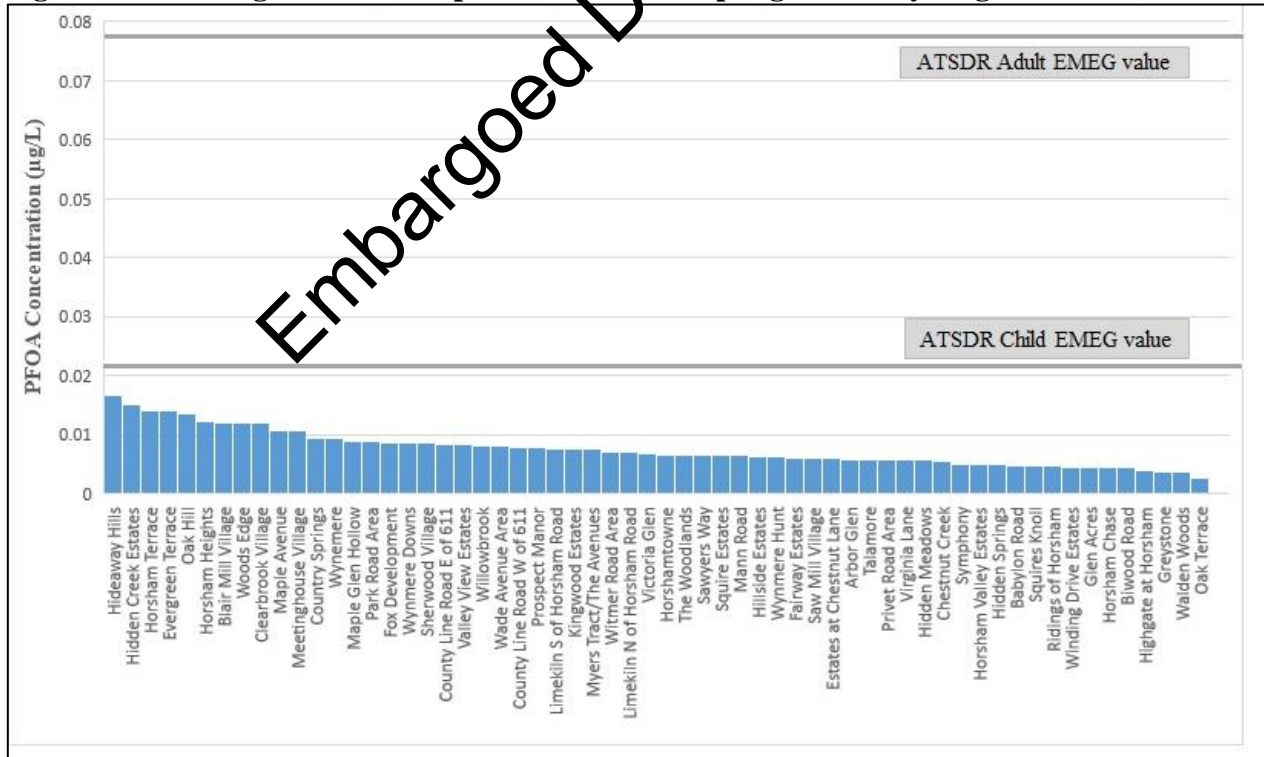
EMEG= ATSDR Environmental Media Evaluation Guide for PFOA and PFOS
µg/L= micrograms per liter

Figure A-4. Average Horsham tap water PFOS sampling results by neighborhood (2016).



EMEG= ATSDR Environmental Media Evaluation Guide for PFOA and PFOS

Figure A-5. Average Horsham tap water PFOA sampling results by neighborhood (2016).



EMEG= ATSDR Environmental Media Evaluation Guide for PFOA and PFOS

The 2016 tap water sampling results indicated that the average PFOS and PFOA levels were below the EPA HA in the Horsham Township’s public water supply (Horsham Water and Sewer 2016). The average PFOS and PFOA results were 0.0102 µg/L and 0.0073 µg/L, respectively. The maximum PFOS and PFOA results were 0.028 µg/L for PFOS and 0.015 µg/L for PFOA. Tap water results exceeded the ATSDR child EMEG for PFOS in 27 samples (or ~25% of the samples). The tap results did not exceed the ATSDR adult EMEGs for PFOS or PFOA (Horsham Township 2016).

Since the tap water sampling in 2016, Horsham Township has taken additional steps and measures to reduce levels of PFAS in the water system, including: installed additional interconnections with neighboring water supplier; increased purchases of water from other sources; and currently installing permanent filters to replace the temporary filters on wells 10, 17 and 21 and new filters on five additional wells. Due to these measures, the system-wide average for PFOA and PFOS was 0.004 µg/L in 2018. Table A-4 describes the Horsham public water PFAS sampling results by water supply well (Horsham Township 2018).

Table A-4. Horsham Raw and Treated Supply Well PFAS Sampling Results in µg/L (2018).

Well #	Dates in Service in 2018	Contaminant	Raw Water Avg.	Raw Water Min.	Raw Water Max.	Treated Water Avg.	Treated Water Min.	Treated Water Max.	MCL	MCLG	HAL
7	1/1/2018 - 12/31/2018	PFOS	0.0063	0.0032	0.0088	NA	NA	NA	NA	NA	0.07
7	1/1/2018 - 12/31/2018	PFOA	0.0087	0.0036	0.0120	NA	NA	NA	NA	NA	0.07
7	1/1/2018 - 12/31/2018	Combined	0.0149	0.0068	0.0098	NA	NA	NA	NA	NA	0.07
9	9/24/2018 - 12/31/2018	PFOS	0.0021	ND	0.0069	NA	NA	NA	NA	NA	0.07
9	9/24/2018 - 12/31/2018	PFOA	0.0030	ND	0.0070	NA	NA	NA	NA	NA	0.07
9	9/24/2018 - 12/31/2018	Combined	0.0051	ND	0.0139	NA	NA	NA	NA	NA	0.07
10	1/1/2018 - 1/25/2018 & 10/10/2018-12/31/2018	PFOS	0.0325	0.0240	0.0480	ND	ND	ND	NA	NA	0.07
10	1/1/2018 - 1/25/2018 & 10/10/2018-12/31/2018	PFOA	0.0218	0.0140	0.0360	ND	ND	ND	NA	NA	0.07
10	1/1/2018 - 1/25/2018 & 10/10/2018-12/31/2018	Combined	0.0543	0.0380	0.0780	ND	ND	ND	NA	NA	0.07
17	1/1/2018 - 12/31/2018	PFOS	0.0883	0.0630	0.1120	ND	ND	ND	NA	NA	0.07
17	1/1/2018 - 12/31/2018	PFOA	0.0333	0.0260	0.0440	ND	ND	ND	NA	NA	0.07

Well #	Dates in Service in 2018	Contaminant	Raw Water Avg.	Raw Water Min.	Raw Water Max.	Treated Water Avg.	Treated Water Min.	Treated Water Max.	MCL	MCLG	HAL
17	1/1/2018 - 12/31/2018	Combined	0.1216	0.0960	0.1450	ND	ND	ND	NA	NA	0.07
21	1/1/2018 – 12/31/2018	PFOS	0.0104	0.0085	0.0130	ND	ND	0.0028	NA	NA	0.07
21	1/1/2018 – 12/31/2018	PFOA	0.0126	0.0090	0.0180	0.0020	ND	0.0052	NA	NA	0.07
21	1/1/2018 – 12/31/2018	Combined	0.0226	0.0120	0.0290	0.0026	ND	0.0073	NA	NA	0.07
26	1/1/2018 – 12/31/2018	PFOS	0.6727	0.4300	1.0380	ND	ND	ND	NA	NA	0.07
26	1/1/2018 – 12/31/2018	PFOA	0.3367	0.2600	0.4780	ND	ND	ND	NA	NA	0.07
26	1/1/2018 – 12/31/2018	Combined	1.0198	0.7300	1.4730	ND	ND	ND	NA	NA	0.07
40	1/1/2018 – 12/31/2018	PFOS	0.8210	0.4600	1.1880	ND	ND	0.0032	NA	NA	0.07
40	1/1/2018 – 12/31/2018	PFOA	0.0707	0.0500	0.0930	ND	ND	ND	NA	NA	0.07
40	1/1/2018 – 12/31/2018	Combined	0.8917	0.5100	1.2670	ND	ND	0.0032	NA	NA	0.07
1	NA	PFAS	NA	NA	NA	NA	NA	NA	NA	NA	NA
2	NA	PFAS	NA	NA	NA	NA	NA	NA	NA	NA	NA
3	NA	PFAS	NA	NA	NA	NA	NA	NA	NA	NA	NA
4	NA	PFAS	NA	NA	NA	NA	NA	NA	NA	NA	NA
6	NA	PFAS	NA	NA	NA	NA	NA	NA	NA	NA	NA
19	NA	PFAS	NA	NA	NA	NA	NA	NA	NA	NA	NA
20	NA	PFAS	NA	NA	NA	NA	NA	NA	NA	NA	NA
22	NA	PFAS	NA	NA	NA	NA	NA	NA	NA	NA	NA

Source: Horsham Water and Sewer Authority Consumer Confidence Report (2018), https://www.horshamwater-sewer.com/sites/default/files/ccr_reports/2018_ccr.pdf.

Warrington Township Water and Sewer Department (WTWSD) Sampling

Prior to the sale of the Warrington public water system in October 2019, the WTWSD public water system served approximately 8,000 accounts. WTWSD does not have an estimate representing the number of people served by these accounts; for a rough approximation, ATSDR applied a factor of three (or three people per account, which parallels information from the HWSA system) to this number of accounts for a population estimate of around 24,000. Of these, only a portion of the active accounts were impacted by the PFAS contamination, prior to the contaminated wells being taken off-line. WTWSD was comprised of two parts – an eastern part and a western part. Water in the eastern portion of the Township was supplied by eight groundwater supply wells. In the western portion of the township, water was purchased from North Wales Water Authority (NWWA). The NWWA primarily relies on surface water supplies from the Delaware River. Figure A-2 depicts the outline of the Warrington system components.

In the eastern portions of Warrington, 65% of the water was supplied by NWWA and 35% was supplied from groundwater wells. Historically the eastern portion of WTWSD relied on a greater

percentage of water from groundwater wells. There is an interconnection between the eastern and western sections of the system that is used when there is a need in the eastern section (WTWSD 2017).

Using the available water distribution system information, ATSDR determined that for recent conditions, the northern part of the eastern section of the WTWSD system generally received water from Well #11 that did not contain PFOA and PFOS. If any customers in the northern part of the system received water containing PFOA and PFOS, it was at levels below the EPA HA. The central part of the eastern section of the system generally received water from Wells # 3, 4, 5, 8, 9, and 10 that may have mixed with some water from Wells 1, 2, and 6. The southeastern part of the eastern section of the system may have received water containing PFOS+PFOA concentrations up to 10 times the EPA HA. Some areas in the central part of the eastern section may have received water containing PFOA and PFOS concentrations above the EPA HA, based on maximum detections in the groundwater supply wells providing water to that area (ATSDR 2017).

The maximum concentration of PFOS (0.67 µg/L) and PFOA (0.12 µg/L) in the WTWSD was detected in November 2014 in the blended wells 1, 2, and 6, as part of the UCMR3 sampling. However, the duration of this concentration in the water distribution system is not known. The western section of the Warrington system is supplied by water purchased from North Wales Water Authority and is not contaminated with PFAS. However, there is an interconnection between the eastern and western sections of the system which is used to supply the eastern section with water from the western section, when there is a need in the eastern section. Given the mixing within the eastern portion of the WTWSD, there is some uncertainty about past exposures levels at individual residential taps. It is likely that some public water users in the past could have had higher exposures to PFAS and some may have had minimal exposures. More detailed analyses of the water-distribution system would need to be conducted to estimate historical PFAS concentrations at specific areas in the past. These analyses would involve looking at the water-distribution system operating conditions, historical monthly well pumping records, and customer consumption information in more detail.

WTWSD Past Exposures

Sampling data for PFAS in the WTWSD is not available prior to 2014. This represents an important data gap and limitation in ATSDR's exposure assessment of past exposures. In October 2014, PFOS and PFOA were detected in multiple public wells in the WTWSD. PFAS sampling in Warrington included PFOA, PFOS, PFHxS, PFNA, PFBS, and PFHpA. Three of the eight wells in the southern portion of the WTWSD were above the EPA former PHAL for PFOS. The maximum PFOS levels were the following: well # 1 (0.21 µg/L), well # 2 (1.6 µg/L), and well # 6 (1.3 µg/L). Although the wells pump directly into the distribution system, wells # 1, 2, and 6 are blended together at a tank and enter the distribution system at one point. These wells constituted about 30% of the WTWSD supply. Well # 3, in the northeast area of the eastern section, and well # 9, which is centrally located in the eastern section, had very low levels of contamination (WTWSD 2017). ATSDR reviewed the PFAS sampling results from the WTWSD through December 2015 (Table A-5 and Figure A-3).

Based on the sampling data indicating levels above EPA's PHAL, wells # 1, 2, and 6 were taken out of service in October 2014. Following the EPA HA release on May 19, 2016, two additional WTWSD wells (wells # 3 and 9) with PFOA and PFOS levels above the EPA HA were also taken off-line. The results of the public water sampling for WTWSD showed PFOS and PFOA exceeding

the ATSDR child EMEG value in 1, 2, and 6 together and #3 and 9 supply wells. The combined maximum concentrations of PFOS and PFOA in the mixed distribution system was 0.79 µg/L (blended maximums of PFOS and PFOA were 0.666 µg/L and 0.12 µg/L, respectively) and a mean blended concentration of 0.106 µg/L. The maximum raw values of PFOS and PFOA (prior to well mixing and distribution) was 1.6 µg/L and 0.27 µg/L, respectively.

PFHxS was detected in the 2014 WTWSD sampling, ranging from non-detect to a maximum concentration of 0.24 µg/L. PFHxS was detected in wells # 1, 2, 6 together and #3 and 9 above the ATSDR child EMEG value at the groundwater entry point in the November 2014 sampling event (WTWSD 2014). UCMR-3 monitoring for PFAS is required at the entry point to the distribution system for each well and at any interconnection in operation. Subsequent sampling in WTWSD has shown non-detect to low levels of PFHxS in well #8 (WTWSD 2017).

Prior to approval of the sale of the WTWSD’s water system to NWWA in 2019, the *western district* of the Warrington Township was served entirely by water purchased from NWWA but three public wells (WTWSD well #s 1, 2, and 6) within the *eastern district* of Warrington Township supplied the portion of the township generally east of Elbow Lane. All public wells in the eastern district (excluding those that were previously shut down due to PFAS contamination levels) contributed to the water that was delivered to public water customers within the eastern district. To compensate for the loss of water from the 5 WTWSD public wells taken offline, the township also purchased additional water from NWWA as needed. While PFNA and PFBS were detected prior to mixing in WTWSD well #s 1, 2, and 6, the levels in the wells after mixing and distribution to eastern district customers were non-detect (Warrington Township 2019a).

Table A-5. Summary of Warrington past public water supply well sampling results for PFAS (based on UCMR3 sampling results November 2014 – December 2015).

Contaminant	Health-Based CV (µg/L)	Detection Limit (µg/L)	Minimum (µg/L)	Minimum Detected Value (µg/L)	Maximum (µg/L)	Mean (µg/L)†	Wells Above CV
PFOS	0.014 ATSDR intermediate child EMEG	0.04	ND	0.04	0.67	0.084	3*
PFOA	0.02 ATSDR intermediate child EMEG	0.02	ND	0.02	0.12	0.027	2*
PFHpA	No CV	0.01	ND	0.012	0.034	0.012	-
PFHxS	0.14 ATSDR intermediate child EMEG	0.03	ND	0.035	0.24	0.045	3±
PFNA	0.021 ATSDR intermediate child EMEG	0.02	ND	ND	ND	NA‡	0
PFBS	2	0.09	ND	ND	ND	NA‡	0

	MN Chronic Health-Based Value						
--	-------------------------------	--	--	--	--	--	--

Notes: Table presents sampling data, provided by WTWSD. The current CVs in drinking water are based on the best available scientific information. However, as new studies and information become available CVs can change.

* The maximum PFOS and PFOA concentrations in the blended samples were 0.67 µg/L and 0.12 µg/L, respectively. These data are based on the 8 WTWSD municipal wells and include wells that are currently off-line. Public wells 3 and 9 were taken off-line following the May 2016 release of the EPA HA for PFOS and PFOA. WTWSD Public wells 1, 2, and 6 were taken off-line in October 2014 and remain off-line.

±PFHxS was detected in wells # 1, 2, and 6 mixed together and in wells #3 and 9 above the ATSDR child intermediate EMEG at the groundwater entry point in the November 2014 sampling event. UCMR-3 monitoring for PFAS is required at the entry point to the distribution system for each well and at any interconnection in operation. It is important to note, these wells are mixed before entering the distribution system. The data in the above table represent entry point sampling.

† ATSDR used the detection limit to substitute for non-detect values.

‡ All the sampling results were below the detection limit and therefore a mean could not be calculated.

ND= Non-detect; NA = Not available; PFOS = Perfluorooctane sulfonate; PFOA = Perfluorooctanoic acid; PFHpA = Perfluoroheptanoic acid; PFHxA= Perfluorohexanoate; PFHxS = Perfluorohexylsulfonic acid; PFNA = Perfluoromonanoic acid; PFBS = Perfluorobutanesulfonic acid; EMEG= ATSDR Environmental Media Evaluation Guide

WTWSD Current Exposures

Tap water samples have not been collected for PFAS in Warrington Township. As of October 1, 2019, Warrington’s public water system was sold and operations fully transferred to NWWA (Warrington Township 2019b). Currently, 93% of NWWA water is from the Delaware River and 7% comes from groundwater sources. The 2016 to 2019 sampling data for NWWA has shown PFOS from non-detect to 0.0025 µg/L and PFOA from non-detect to 0.0045 µg/L (NWWA 2019). Maximum detected results for PFOS and PFOA at NWWA’s Forest Park water treatment plant are below ATSDR child and adult EMEGs (NWWA 2019).

Private Drinking Water Wells Sampling

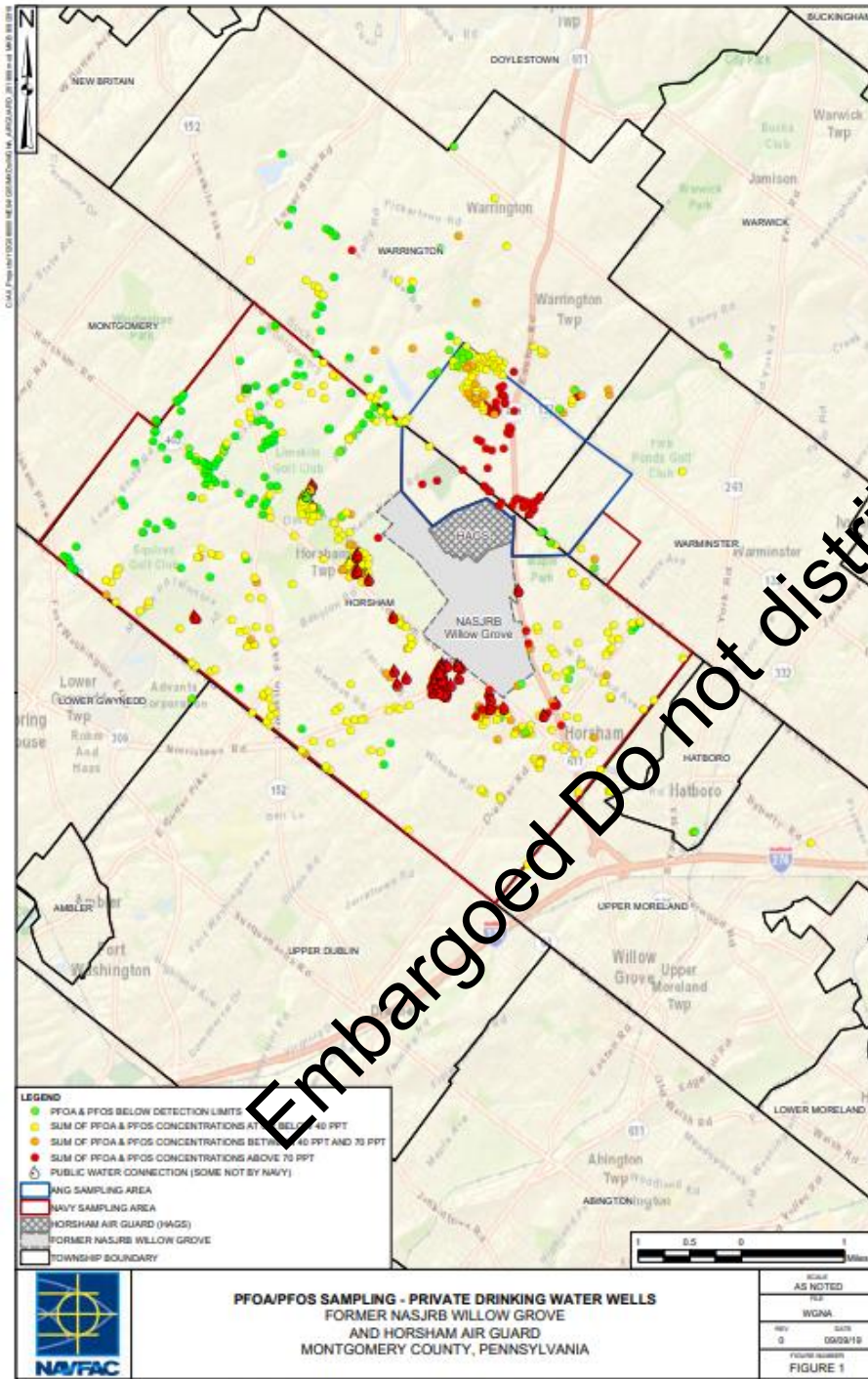
Private Drinking Water Wells Past Exposures

In 2014, due to the PFAS detections in the public water supplies, the Navy and Air National Guard requested EPA’s support in evaluating whether PFAS are present in private drinking water wells on properties near the site (Table A-6 and Figures A-6 and A-7). Properties for sampling were identified through database searches provided by local water authorities to the EPA, Navy, and Air Guard and included wells located in Horsham and Warrington Townships and a small portion of Warminster Township near the Air Guard portion of the site. Initial sampling of the private drinking water wells near the site was conducted by EPA on behalf of the Navy and Air National Guard. Private drinking water wells were sampled for PFOA, PFOS, PFHxS, PFNA, PFBS, and PFHpA.

As of April 2017, 640 residential private drinking water wells near the site were sampled. There are private drinking water wells near the site where the owners have not granted access for sampling. ATSDR has had discussions with EPA, Navy, and Air National Guard about addressing this data gap and continuing to encourage private drinking water well owners in the site area to participate in the sampling for PFAS.

Comprehensive PFAS groundwater plume maps are not currently available. The migration of PFOA and PFOS from on-base sources to drinking water sources is complex and is being investigated by the Navy and Air National Guard. However, based on the private drinking well sampling data reviewed by ATSDR, as shown in Figure A-6, the private drinking water wells that are located closer to the site appear to have higher levels of PFAS. The northeast and southwest sides of the sites showed the greatest number private drinking water wells with PFOA and PFOS exceeding the EPA HA.

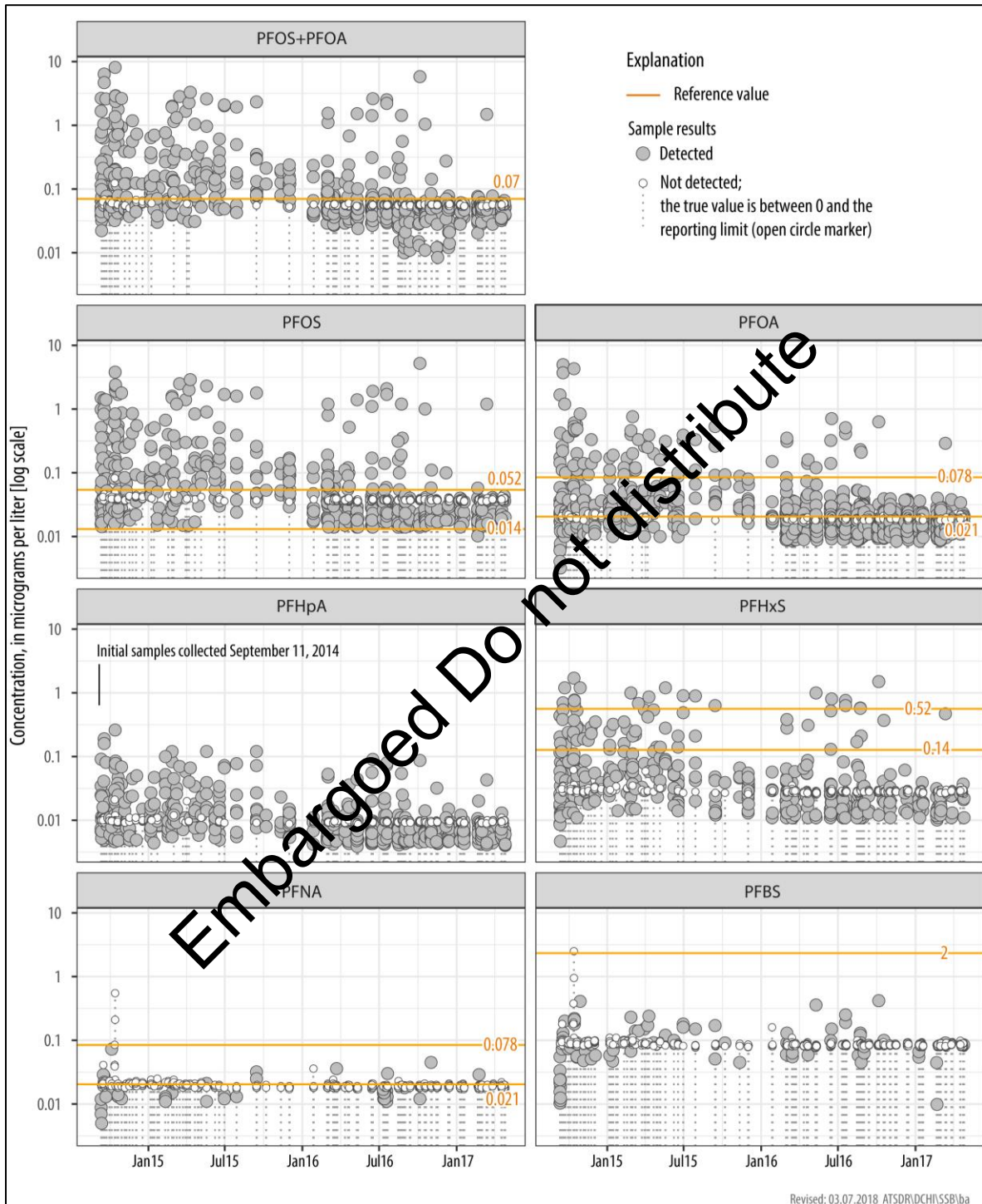
Figure A-6. Private drinking water well sampling results near the Willow Grove site for PFOA and PFOS (as of September 2019).



Source: DOD, US Navy.

https://www.bracpmo.navy.mil/brac_bases/northeast/reserve_base_willow_grove/documents.html

Figure A-7. Private drinking water well sampling results near the Willow Grove site by PFAS type (September 2014-April 2017).



Notes: PFOS= perfluorooctane sulfonate; PFOA= perfluorooctanoic acid; PFHpA = perfluoroheptanoic acid; PFHxS = perfluorohexane sulfonate; PFHxA = perfluorohexanoic acid; PFNA = perfluorononanoic acid; PFBS = perfluorobutane sulfonate;

Reference/comparison values (indicated with the reference value line in the charts above) = PFOS+PFOA – EPA HA of 0.07 µg/L; PFOA – ATSDR intermediate EMEG of 0.021 µg/L (children) and 0.078 µg/L (adults); PFOS ATSDR intermediate EMEG of 0.014 µg/L (children) and 0.052 µg/L (adults); PFHxS- ATSDR intermediate EMEG of 0.14 µg/L (children) and 0.52µg/L (adults); PFNA = ATSDR intermediate EMEG of 0.021 µg/L (children) and 0.078 µg/L (adults); PFBS = 2 µg/L (Minnesota Health-Based Comparison Value). The current CVs in drinking water are based on the best available scientific information. However, as new studies and information become available CVs can change.

Table A-6. Summary of Willow Grove private drinking water well sampling results for PFAS (as of April 2017).

Contaminant	Health-Based CV (µg/L)	Detection Limit (µg/L)	Minimum (µg/L)	Minimum Detected Value (µg/L)	Maximum (µg/L)	Mean (µg/L)†	Wells Above CV
PFOS	0.014 ATSDR intermediate child EMEG	0.003-0.43	ND	0.01	5.2*	0.054	309*
PFOA	0.021 ATSDR intermediate child EMEG	0.003-0.43	ND	0.003	5.0*	0.045	187*
PFHpA	No CV	0.002-1.3	ND	0.0039	0.26	0.008	No CV
PFHxS	0.14 ATSDR intermediate child EMEG	0.004-0.33	ND	0.0047	1.7	0.031	67
PFNA	0.021 ATSDR intermediate child EMEG	0.004-0.31	ND	0.005	0.073	NA‡	4
PFBS	2 MN Chronic Health-Based Value	0.001-1.3	ND	0.0099	0.42	NA‡	0

Notes: The current CVs in drinking water are based on the best available scientific information. However, as new studies and information become available CVs can change. If a private drinking water well was sampled more than once, the maximum value detected was used for the purposes of this table summary. Pre- and post-samples were considered duplicates.

* The maximum combined PFOA and PFOS in a single private drinking well was 8.1 µg/L. A total of 640 private drinking water wells tested, 309 and 187 private drinking water wells above the ATSDR intermediate child EMEG for PFOS and PFOA, respectively.

† The mean used the detection limit to substitute for non-detect values.

‡ The percent of non-detects in these datasets were high (>80% non-detects) and therefore a mean could not be calculated

ND = Non-detect; NA = Not available; PFOS = Perfluorooctane sulfonate; PFOA = Perfluorooctanoic acid; PFHpA = Perfluoroheptanoic acid; PFHxA = Perfluorohexanoate; PFHxS = Perfluorohexylsulfonic acid; PFNA = Perfluoromonanoic acid; PFBS = Perfluorobutanesulfonic acid; EMEG = ATSDR environmental media evaluation guide

Based on the private drinking water well sampling data collected from July 2014 through April 2017, the maximum combined concentrations (in a single well) of PFOS and PFOA was 8.1 µg/L. Approximately 31% of the private drinking water well samples were non-detect for PFOS or PFOA (198 of 640 wells sampled). The maximum PFOS and PFOA detected site-wide in a private drinking water well was 5.2 and 5.0 µg/L, respectively. The mean combined PFOS and PFOA level was 0.14 µg/L. As of April 2017, approximately 26% of private drinking water wells (164 of the 640 homes sampled) had combined levels of PFOA and PFOS above the EPA HA. Across this data set, 48% (309 of 640 homes sampled) had PFOS levels above the ATSDR intermediate child EMEG and 29% (187 of 640 homes sampled) had PFOA levels above the ATSDR intermediate child EMEG.

PFNA was detected above the ATSDR intermediate child EMEG (of 0.021 µg/L) in four private drinking water wells, with a maximum concentration of 0.073 µg/L. PFHxS was detected above the

ATSDR intermediate child EMEG of 0.14 µg/L in 67 private drinking water wells, with a maximum concentration of 1.7 µg/L.

PFHpA was detected in two private drinking water wells at a maximum concentration of 0.26 µg/L. However, ATSDR does not currently have a CV for PFHpA. PFBS was detected in 68 private drinking water wells, with a maximum was 0.42 µg/L. However, the results are below the Minnesota chronic drinking water value for PFBS of 2 µg/L.

Private Drinking Water Wells Current Exposures

Private drinking water well users with PFOS and PFOA above the EPA HA have since been provided bottled water or connected to the public water supply. The prior sections on HWSA and WTWSD current exposures are relevant for former private well users hooked up to those public water systems.

Private drinking water well users with PFOS and PFOA below the EPA HA but above 0.04 µg/L are part of the Navy or Air Guard resampling plan.

The Navy, Air Guard and local water utilities have worked diligently to complete the connections to public water for almost all of the private well water users with PFOS and/or PFOA contamination above the EPA HA. As of December 2019, on the Navy side there were only 7 properties that still need a connection to the public water supply, and two of these properties have their hookup scheduled. As of December 2019, on the Air Guard side there were 16 properties that were not connected yet (at one of these properties, the well is no longer in use). The remainder of these households not yet hooked up to public water are still being provided bottled water for drinking and cooking.

There are a few private drinking water wells in the area that have not been sampled due to access not being granted. Individuals within the site area that did not grant access for PFAS sampling may be at risk for health effects from ongoing exposures.

On-Base Drinking Water Sampling

Beginning in August 2014, on-base drinking water supply wells were sampled for PFAS (Table A-7 and Figure A-8). In addition to the portion of the base supplied by HSWA, these on-base drinking water supply wells served the full base, both the Navy and Air Guard sides. These sampling results represent concentrations at the supply well, not what was delivered to the tap. Based on the data reviewed, the maximum levels of PFOS and PFOA detected on-base were 13.7 µg/L and 8.1 µg/L, respectively. Since September 2014, the on-base drinking water wells have not been used for consumption, and bottled water is used on-base for drinking water. The past maximum levels detected in the on-base drinking water exceed the maximum levels observed in off-base public and private drinking water supplies.

Table A-7. On-base water supply well sampling results for PFOA and PFOS (µg/L), by building location.

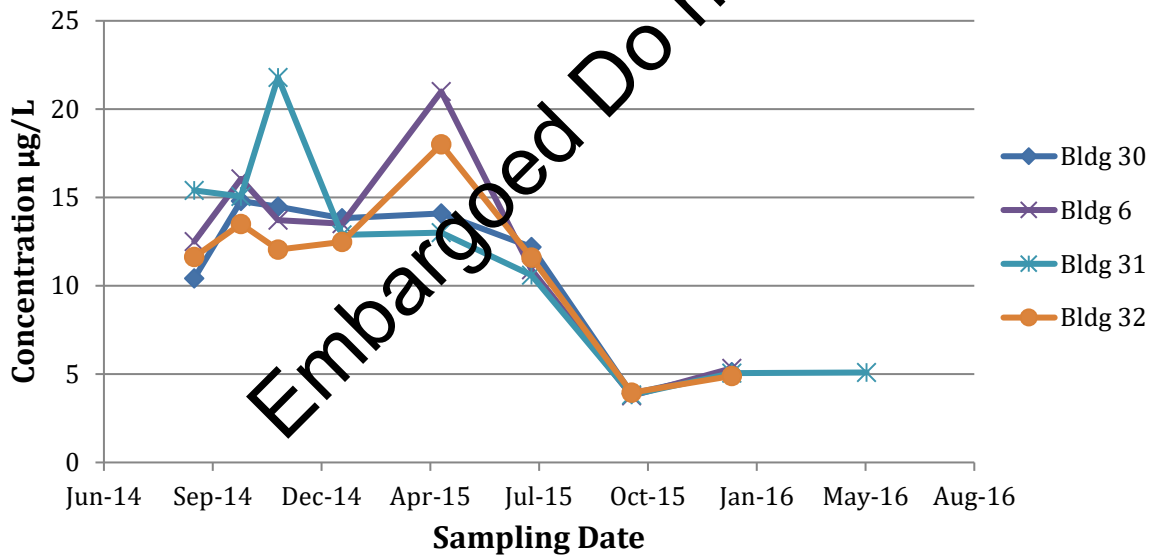
Sampling Date	Bldg. 31 Supply Well	Bldg. 31 Supply Well	Bldg. 32 Supply Well	Bldg. 32 Supply Well	Bldg. 30 Supply Well	Bldg. 30 Supply Well	Bldg. 6 Supply Well	Bldg. 6 Supply Well
	PFOS	PFOA	PFOS	PFOA	PFOS	PFOA	PFOS	PFOA
9/1/2014	13.1	2.3	8.1	3.6	8.2	2.2	9.8	2.7
10/14/2014	12.9	2.2	10.6	2.9	11.7	3.1	13	3.1
11/17/2014	13.7	8.1	8.1	4	11.4	3.1	10.7	3
1/15/2015	10.7	2.2	8.6	3.9	10.7	3.1	10.4	3.1
4/16/2015	13	0	15	3	12	2.1	18	3
7/8/2015	8.8	1.8	9.2	2.4	11	1.2	9.2	1.7
10/8/2015	3.4	0.4	3	1	3.2	0.7	0.1	0.7
1/8/2016	4.5	0.6	3.9	1	4.3	0.8	4.5	0.8
5/11/2016	4.4	0.7	NS	NS	NS	NS	NS	NS

NS= not sampled

µg/L = micrograms per liter

Source: Email communication from J Siciliano/Horsham Air Guard to L Wenger, ATSDR (2017).

Figure A-8. On-base water supply well sampling results for PFOA and PFOS (combined), by building location (June 2014-May 2016).



ATSDR acknowledges there was also a past exposure pathway for civilian employees, service members, and their families on the base in this community. Parallel to the situation for off-base public and private drinking water supplies, there is uncertainty on how long and at what concentrations former on-base service members and civilian employees may have been exposed to the contamination in the on-base drinking water supplies (e.g. a lack of historical sampling data before 2014). An additional layer of uncertainty for on-base exposures is that ATSDR does not have information on how the on-base water system functions, or any changes in the on-base well usage over time (e.g., additional supply wells, changes in the distribution system, filtration system, etc), or

specific information on the past exposed populations. ATSDR does not have enough information at this time to more fully evaluate past on-base exposures.

Surface Water and Biota Environmental Data

In 2015 through 2017, as part of the site Remedial Investigation (RI) at the Navy portion of the site and to further characterize the extent of PFAS surface water contamination, the Navy collected off-site surface water samples along the Little Neshaminy Creek, Pennypack Creek, Park Creek, and unnamed tributaries to these creeks located near the site. The Navy collected 33 surface water samples in February 2015 and 54 surface water samples and 57 sediment samples in April 2016. Locations that may have received runoff and/or AFFF drainage from the site were targeted for sampling. In August 2017, the Navy collected 27 surface water and 7 sediment samples from 11 sampling locations to evaluate PFAS in the storm water discharge along the base outfalls (Navy 2018).

The RI surface water sampling showed that PFOS concentrations were typically higher than PFOA. PFOS tended to be highest within first order streams or drainage swales on or immediately adjacent to the site. PFOS ranged from non-detect to 4.05 µg/L (detected at the western boundary of the Horsham Air Guard Station) and PFOA were non-detect to 0.251 µg/L. The highest PFOS levels in the surface water were found in an outfall located on the northwestern boundary of the site, the north ponding area, an unnamed tributary to Park Creek, and an intermittent tributary to Pennypack Creek. For the sediment sampling, PFOS was detected in 27 samples and PFOA in two samples. The maximum off-site sediment levels were 65 µg/kg and 1.9 µg/kg, for PFOS and PFOA, respectively, in an unnamed tributary near Norristown Road (Navy 2018).

Fish consumption by community members in the site area is a potential exposure pathway for possible further evaluation. Various residents have verbally reported to ATSDR at public meetings that they fish in waters near this site. The Pennsylvania Fish and Boat Commission (PFBC) stocks Pennypack Creek with trout. PFBC evaluated fish species in the tidal portions of further away and larger Neshaminy Creek and found that the tidal section of this creek supports a variety of game fish species for anglers to target, such as Channel Catfish, Common Carp, White Perch, Yellow Perch, and an assortment of sunfish species (Pennsylvania Fish & Boat Commission 2014). PADEP and PFBC conducted biological surveys at six locations each of the Little Neshaminy and Park Creeks in 1992 and found that fish quality in the Little Neshaminy ranged from poor to good, and in Park Creek ranged from not located and fair to good. Another biological assessment was conducted in 2002 to assess the biological health of the Little Neshaminy and Park Creeks at nine locations in these creeks and found overall that the quality of the macroinvertebrate community in the Little Neshaminy and Park Creeks suggests slight to moderate impairment (Heritage Conservancy 2007). Additional information on fish advisories (that does not currently include PFAS) issued by the PFBC can be found at this link: <http://www.dep.pa.gov/Business/Water/CleanWater/WaterQuality/FishConsumptionAdvisory/Pages/default.aspx>.

The consumption of fish is thought to be a potential additional pathway for PFAS exposure for both the general population and for people living near PFAS contaminated waters. Typically, elevated concentrations of PFAS in fish are found near areas involved in the production or manufacture of PFAS. We currently do not have fish tissue data for this site and cannot evaluate these exposures. In

a review article by Fromme et al. (2009), PFAS concentrations in fish from waters known to be contaminated with PFAS tended to be in the 10s to 100s of micrograms per kilogram ($\mu\text{g}/\text{kg}$), while concentrations in fish not near a known PFAS source ranged from non-detect to the low 10s of $\mu\text{g}/\text{kg}$. PFAS with carbon chain length lower than 8 tend to bioaccumulate less than PFOS, although overall the health effects of many short-chained PFAS have not been fully researched (EPA 2018b, ASTDR 2018). Both PFOS and PFOA are believed to bioaccumulate in fish, with the result that fish higher up the food chain can accumulate significant concentrations of the chemicals. In general, PFOS accumulates at a higher concentration than PFOA (Fromme et al 2009). For example, in the Great Lakes, concentrations of PFOS ranging from 59 to 297 ng/g were found for fish caught from 1999-2000. Concentrations of PFOA, however, did not meet the detection limit of 36 ng/g (Kannan et al 2005).

Various authors have found an association between elevated PFAS serum levels and consumption of fish from PFAS-contaminated waters. In Poland, Falandysz et al (2006) examined the blood serum levels of 45 participants, who indicated a high fish intake in their diet living near the Baltic Sea and found that the consumption of local fish resulted in statistically higher PFAS serum concentrations than those observed in comparison groups. Similarly, Noller et al (2008) examined the serum concentrations in people living near PFAS-contaminated waters in Arnsberg, Germany, and found that elevated PFOS blood serum concentrations were associated with the consumption of local fish. Given the potential for bioaccumulation in fish, the consumption of fish is a potential past and current completed exposure pathway.

Currently, ATSDR, EPA and the Commonwealth of Pennsylvania do not have screening values for PFAS in surface water or fish. The EPA HA only applies to exposure from drinking water and cannot be used to identify risk levels for ingestion of food, including fish (EPA 2016e). However, some states, such as Minnesota and Michigan, have developed guidance on fish sampling for PFOS and fish consumption advisories, based on PFOS levels in fish fillet samples (Minnesota 2018; Michigan 2014; Michigan 2018).

Exposure Assessment: Dose Calculations (Non Cancer)

ATSDR used the drinking water exposure information for HWSA, WTWSD, and the private well water described in the previous section along with assumptions about exposure duration and body weight/water consumption patterns by age group to calculate exposure doses, along with hazard quotients (HQs) for PFOS, PFOA, PFHxS, and PFNA. An HQ is the ratio of the exposure doses divided by the appropriate PFAS provisional MRL.

Consumers of the public and private drinking water experience an exposure to a mixture of PFAS compounds in the water. To evaluate the potential noncancerous health effects risk based on cumulative exposures to PFOA, PFOS, PFHxS, and PFNA (those PFAS with ATSDR-derived provisional MRLs in the ATSDR Toxicological Profile for PFAS released for public comment in 2018), ATSDR used the HI approach. The HI is the sum of the hazard quotients (HQs); an HQ is the estimated dose of a chemical divided by its MRL. If the hazard index is less than 1.0, it is unlikely that significant additive or toxic interactions would occur. If the HI is greater than 1.0, concern for the potential hazard of the mixture increases.

Only two studies (Carr et al. 2013; Wolf et al. 2014) have shown binary pairs of PFAS (i.e., comparing only two PFAS together) demonstrate concentration and response additivity at lower concentrations, but deviate from additivity at higher concentrations (Wolf et al. 2014). These possible interactions (or dose additivity) complicate the interpretation of the epidemiology data.

In the absence of data, chemical component-based approaches are used in risk assessment of chemical mixtures. Component chemicals, that are judged to be toxicologically similar, are evaluated by dose additive risk assessment methods that include the hazard index, relative potency factors, and toxicity equivalency factors. These methods are based on potency weighted dose addition and assume that there are no greater than or less than additive interactions among the chemicals in the dose region of interest. Because of these limited data, ATSDR cannot assume any mixture effect except additivity.

With the exception of the hazard index approach for PFOA, PFOS, PFHxS, and PFNA, there is not a broadly accepted scientific method to quantitatively evaluate the possible health effects of combined exposures to PFAS. In addition, as stated previously, not all PFAS share the same health outcomes. Therefore, ATSDR evaluated the scientific literature to determine what health effects from the chemicals in the PFAS mixture found in the drinking water at this site might have similar health endpoints.

In addition, to put these HQs into perspective, ATSDR calculated margins-of-exposure (MOEs). For this analysis, the MOE is the effect level from the animal study used to derive the provisional ATSDR MRL, divided by the dose derived from the maximum exposure concentration observed in the public and private drinking water systems. In contrast to the HQ, the lower the MOE, the closer the exposure was to effect levels which indicates more concern. ATSDR's MOEs for PFOS, PFOA, PFHxS, and PFNA are discussed in the Public Health Implications section later in this document.

ATSDR took a conservative and protective public health approach (including conservative assumptions about drinking water intake rates, exposure duration, maximum concentration, etc.) in evaluating the health risks for non-chronic exposures. ATSDR used default exposure assumptions defined by specific age groups with corresponding estimated exposure doses for each age group. Body weights were selected based on data from NHANES 1999 – 2006, as reported in the USEPA Exposure Factors Handbook (EPA 2011) and ATSDR's Exposure Dose Guidance (ATSDR 2016a). By calculating age-specific exposure doses, ATSDR can better assess the possible public health implications for site-specific conditions among different age populations under several exposure durations (ATSDR 2005, 2016a). ATSDR estimated central tendency exposure (CTE) and reasonable maximum exposure (RME) doses that may occur for each age group according to the following equation:

$$\text{Exposure Dose (mg/kg/day)} = \frac{\text{Drinking Water Intake (L/day)} \times \text{Drinking Water Concentration (mg/L)}}{\text{Body Weight (kg)}}$$

Because of the limitations in overall site exposure information, ATSDR conservatively estimated exposure doses using the maximum PFOS, PFOA, PFHxS, and PFNA water concentrations. CTE estimates assumed typical (or average) water intake levels among each age group. RME estimates

assumed reasonable maximum water intake levels (i.e. the 95th percentile, ATSDR 2016a) for each age group. Therefore, the RME is higher than average but still within a realistic exposure range. Described below are the results for the two public water supplies and private drinking water wells at this site.

Exposure Dose Calculations: HWSA Past Exposures

Estimated exposure doses were calculated for the RME (reasonable maximum exposure) and CTE (central tendency exposure) scenarios and compared to the ATSDR provisional MRLs described above. Tables A-8 through A-11 below provide a summary of the past exposure doses and HQs for the HWSA public supply wells. Table A-12 provides the Hazard Index for combined past exposures to PFOS, PFOA, PFHxS, and PFNA.

Table A-8. Horsham public water: Past PFOS environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Age Group	Exposure Assumptions		Exposure Assumptions Body Weight (kg)	Estimated Exposure Dose (mg/kg/day)		HQ PFOS	
	Drinking Water Intake (L/day)	Drinking Water Intake (L/day)		Maximum PFOS water concentration = 1.0 µg/L	Maximum PFOS water concentration = 1.0 µg/L	RME	CTE
	RME	CTE		RME	CTE	RME	CTE
Birth to < 1 yr	1.113	0.504	7.8	1.43E-04	6.46E-05	71.3	32.3
1 to < 2 yr	0.893	0.308	11.4	7.83E-05	2.70E-05	39.2	13.5
2 to < 6 yr	0.977	0.376	17.4	5.61E-05	2.16E-05	28.1	8.0
6 to < 11 yr	1.404	0.511	31.8	4.42E-05	1.61E-05	22.1	8.0
11 to <16 yr	1.976	0.637	56.8	3.48E-05	1.12E-05	17.4	5.6
16 to <21 yr	2.444	0.777	71.6	3.41E-05	1.08E-05	17.1	5.4
Adults ≥ 21 yr	3.092	1.222	80	3.87E-05	1.53E-05	19.3	7.7
Lactating Women	3.588	1.665	73	4.92E-05	2.28E-05	24.6	11.4
Pregnant Women	2.589	0.872	73	3.55E-05	1.19E-05	17.7	6.0

Notes: CTE = central tendency exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure, µg/L = micrograms per liter, yr = year; HQ= Hazard Quotient; bold= exceeds HQ of 1.0

Table A-9. Horsham public water: Past PFOA environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Age Group	Exposure Assumptions	Exposure Assumptions	Exposure Assumptions	Estimated Exposure Dose (mg/kg/day)	Estimated Exposure Dose (mg/kg/day)	HQ PFOA	HQ PFOA
	Drinking Water Intake (L/day)	Drinking Water Intake (L/day)	Body Weight (kg)	Maximum PFOA water concentration = 0.290 µg/L	Maximum PFOA water concentration = 0.290 µg/L	RME	CTE
	RME	CTE		RME	CTE	RME	CTE
Birth to < 1 yr	1.113	0.504	7.8	4.14E-05	1.87E-05	13.8	6.2
1 to < 2 yr	0.893	0.308	11.4	2.27E-05	7.84E-06	7.6	2.6
2 to < 6 yr	0.977	0.376	17.4	1.63E-05	6.07E-06	5.4	2.1
6 to < 11 yr	1.404	0.511	31.8	1.28E-05	4.66E-06	4.3	1.6
11 to <16 yr	1.976	0.637	56.8	1.01E-05	3.25E-06	3.4	1.1
16 to <21 yr	2.444	0.77	71.6	9.90E-06	3.12E-06	3.3	1.0
Adults ≥ 21 yr	3.092	1.227	80	1.12E-05	4.45E-06	3.7	1.5
Lactating Women	3.588	1.665	73	1.43E-05	6.61E-06	4.8	2.2
Pregnant Women	2.589	0.872	73	1.03E-05	3.46E-06	3.4	1.2

Notes: CTE = central tendency exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure, µg/L = micrograms per liter, yr = year; HQ= Hazard Quotient; bold= exceeds HQ of 1.0

Table A-10. Horsham public water: Past PFHxS environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Age Group	Exposure Assumptions	Exposure Assumptions	Exposure Assumptions	Estimated Exposure Dose (mg/kg/day)	Estimated Exposure Dose (mg/kg/day)	HQ PFHxS	HQ PFHxS
	Drinking Water Intake (L/day)	Drinking Water Intake (L/day)	Body Weight (kg)	Maximum PFHxS water concentration = 0.59 µg/L	Maximum PFHxS water concentration = 0.59 µg/L	RME	CTE
	RME	CTE		RME	CTE	RME	CTE
Birth to < 1 yr	1.113	0.504	7.8	8.42E-05	3.81E-05	4.2	1.9
1 to < 2 yr	0.893	0.308	11.4	4.62E-05	1.59E-05	2.3	0.8
2 to < 6 yr	0.977	0.376	17.4	3.31E-05	1.27E-05	1.7	0.5
6 to < 11 yr	1.404	0.511	31.8	2.60E-05	9.48E-06	1.3	0.5
11 to <16 yr	1.976	0.637	56.8	2.05E-05	6.62E-06	1.0	0.3
16 to <21 yr	2.444	0.77	71.6	2.01E-05	6.34E-06	1.0	0.3
Adults ≥ 21 yr	3.092	1.227	80	2.28E-05	9.05E-06	1.1	0.5

Lactating Women	3.588	1.665	73	2.90E-05	1.35E-05	1.4	0.7
Pregnant Women	2.589	0.872	73	2.09E-05	7.05E-06	1.0	0.4

Notes: CTE = central tendency exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure, µg/L = micrograms per liter, yr = year; HQ= Hazard Quotient; bold= exceeds HQ of 1.0

Table A-11. Horsham public water: Past PFNA environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Age Group	Exposure Assumptions	Exposure Assumptions	Exposure Assumptions	Estimated Exposure Dose (mg/kg/day)	Estimated Exposure Dose (mg/kg/day)	HQ PFNA	HQ PFNA
	Drinking Water Intake (L/day)	Drinking Water Intake (L/day)	Body Weight (kg)	Maximum PFNA water concentration = 0.0039 µg/L	Maximum PFNA water concentration = 0.0039 µg/L		
	RME	CTE		RME	CTE	RME	CTE
Birth to < 1 yr	1.113	0.504	7.8	5.57E-07	2.52E-07	0.19	0.08
1 to < 2 yr	0.893	0.308	11.4	3.96E-07	1.05E-07	0.10	0.04
2 to < 6 yr	0.977	0.376	17.4	2.19E-07	8.43E-08	0.07	0.02
6 to < 11 yr	1.404	0.511	31.8	1.72E-07	6.27E-08	0.06	0.02
11 to <16 yr	1.976	0.637	56.8	1.36E-07	4.37E-08	0.05	0.01
16 to <21 yr	2.444	0.77	71.6	1.33E-07	4.19E-08	0.04	0.01
Adults ≥ 21 yr	3.092	1.227	81.6	1.51E-07	5.98E-08	0.05	0.02
Lactating Women	3.588	1.665	73	1.92E-07	8.90E-08	0.06	0.03
Pregnant Women	2.589	0.872	73	1.38E-07	4.66E-08	0.05	0.02

Notes: CTE = central tendency exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure, µg/L = micrograms per liter, yr = year; HQ= Hazard Quotient.

Table A-12. Horsham public water: Hazard Index for combined past exposures to PFOS, PFOA, PFHxS, and PFNA.

Age Group	Exposure Assumptions	Exposure Assumptions	Exposure Assumptions	Hazard Index (HI)	Hazard Index (HI)
	Drinking Water Intake (L/day)	Drinking Water Intake (L/day)	Body Weight (kg)		
	RME	CTE		RME	CTE
Birth to < 1 yr	1.113	0.504	7.8	89.5	40.5
1 to < 2 yr	0.893	0.308	11.4	49.2	17.0
2 to < 6 yr	0.977	0.376	17.4	35.2	10.6
6 to < 11 yr	1.404	0.511	31.8	27.7	10.1

11 to <16 yr	1.976	0.637	56.8	21.8	7.0
16 to <21 yr	2.444	0.77	71.6	21.4	6.7
Adults ≥ 21 yr	3.092	1.227	80	24.3	9.6
Lactating Women	3.588	1.665	73	30.8	14.3
Pregnant Women	2.589	0.872	73	22.3	7.5

Notes: CTE = central tendency exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure, µg/L = micrograms per liter, yr = year; HI= Hazard Index, which sums the HQs for PFOS, PFOA, PFHxS, and PFNA; bold= exceeds HQ of 1.0.

For the Horsham public drinking water supply, using the RME and CTE assumptions, all age categories for PFOS and PFOA past exposures exceeded the HQ of 1.0. The youngest age categories having the highest HQs. For PFHxS, exposure calculations for all age categories exceeded the HQ of 1.0 using the RME exposure assumptions. For the CTE scenario, only the youngest age group (birth to 1 years of age) exceeded an HQ of 1.0. The HQ calculations for PFNA for all ages groups were below 1.0. The combined exposures (for PFOS, PFOA, PFHxS and PFNA) using the HI approach showed that all age categories exceeded an HI of 1.0 using the RME and CTE approaches. PFOS and PFOA were the largest overall contributors to HI. If the HI is greater than 1.0, concern for the potential hazard of the mixture increases. ATSDR used the maximum drinking water sampling results for these calculations as a conservative approach. ATSDR acknowledges that it is unlikely that an individual consumed the maximum level detected at a supply well, given the mixing in the system. However, we do not know if the maximum level observed in sampling represents the maximum level ever in the system, due to limitations in sampling information. As the PFAS levels increase, there is an increased potential for harmful non-cancer health effects in adults and particularly for young children and infants.

The exposure doses, based on past exposure in the Horsham water supply, are below the estimated PFOS NOAEL Human Equivalent Dose (HED) (6.1×10^{-4} mg/kg/day) for reduced pup body weight in the Luebker study (Luebker et al. 2005; EPA 2016b). In addition, the doses are below the estimated PFOA LOAEL_{HED} (5.3×10^{-3} mg/kg/day) for delayed bone ossification and accelerated puberty in males in the Lau et al. 2006 animal study (EPA 2016c). These estimated exposure doses do not take into account PFOA and PFOS exposures from non-drinking water sources. The discussion on public health evaluation is presented in the Public Health Implications Section later in this report.

Exposure Dose Calculations: WTWSD Past Exposures

Estimated past exposure doses were calculated for the RME (maximum) and CTE (average) scenarios and compared to the ATSDR provisional MRLs described above. Tables A-13 through A-15 below provide a summary of the past exposure doses and HQs for the WTWSD public supply wells. Table A-16 provides the Hazard Index for combined past exposures to PFOS, PFOA, and PFHxS (PFNA was not detected in this system).

Table A-13. Warrington public water: past PFOS environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Age Group	Exposure Assumptions	Exposure Assumptions	Exposure Assumptions	Estimated Exposure Dose (mg/kg/day)	Estimated Exposure Dose (mg/kg/day)	HQ PFOS	HQ PFOS
	Drinking Water Intake (L/day)	Drinking Water Intake (L/day)	Body Weight (kg)	Maximum PFOS water concentration = 0.67 µg/L	Maximum PFOS water concentration = 0.67 µg/L		
	RME	CTE		RME	CTE	RME	CTE
Birth to < 1 yr	1.113	0.504	7.8	9.52E-05	4.31E-05	47.6	21.5
1 to < 2 yr	0.893	0.308	11.4	5.22E-05	1.80E-05	26.1	9.0
2 to < 6 yr	0.977	0.376	17.4	3.75E-05	1.44E-05	18.7	5.4
6 to < 11 yr	1.404	0.511	31.8	2.94E-05	1.07E-05	14.7	5.4
11 to <16 yr	1.976	0.637	56.8	2.32E-05	7.48E-06	11.6	3.7
16 to <21 yr	2.444	0.77	71.6	2.28E-05	7.17E-06	11.4	3.6
Adults ≥ 21 yr	3.092	1.227	80	2.58E-05	1.02E-05	12.9	5.1
Lactating Women	3.588	1.665	73	3.28E-05	1.52E-05	16.4	7.6
Pregnant Women	2.589	0.872	73	2.37E-05	7.97E-06	11.8	4.0

Notes: CTE = central tendency exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure µg/L = micrograms per liter, yr = year; HQ= Hazard Quotient; bold= exceeds HQ of 1.0

Table A-14. Warrington public water: past PFOA environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Age Group	Exposure Assumptions	Exposure Assumptions	Exposure Assumptions	Estimated Exposure Dose (mg/kg/day)	Estimated Exposure Dose (mg/kg/day)	HQ PFOA	HQ PFOA
	Drinking Water Intake (L/day)	Drinking Water Intake (L/day)	Body Weight (kg)	Maximum PFOA water concentration = 0.12 µg/L	Maximum PFOA water concentration = 0.12 µg/L		
	RME	CTE		RME	CTE	RME	CTE
Birth to < 1 yr	1.113	0.504	7.8	1.71E-05	7.75E-06	5.7	2.6
1 to < 2 yr	0.893	0.308	11.4	9.40E-06	3.24E-06	3.1	1.1
2 to < 6 yr	0.977	0.376	17.4	6.74E-06	2.59E-06	2.2	0.9
6 to < 11 yr	1.404	0.511	31.8	5.30E-06	1.93E-06	1.8	0.6
11 to <16 yr	1.976	0.637	56.8	4.17E-06	1.35E-06	1.4	0.4
16 to <21 yr	2.444	0.77	71.6	4.10E-06	1.29E-06	1.4	0.4
Adults ≥ 21 yr	3.092	1.227	80	4.64E-06	1.84E-06	1.5	0.6
Lactating Women	3.588	1.665	73	5.90E-06	2.74E-06	2.0	0.9
Pregnant Women	2.589	0.872	73	4.26E-06	1.43E-06	1.4	0.5

Notes: CTE = central tendency exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure, µg/L = micrograms per liter, yr = year; HQ= Hazard Quotient; bold= exceeds HQ of 1.0

Table A-15. Warrington public water: past PFHxS environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Age Group	Exposure Assumptions	Exposure Assumptions	Exposure Assumptions	Estimated Exposure Dose (mg/kg/day)	Estimated Exposure Dose (mg/kg/day)	HQ PFHxS	HQ PFHxS
	Drinking Water Intake (L/day)	Drinking Water Intake (L/day)	Body Weight (kg)	Maximum PFHxS water concentration = 0.24 µg/L	Maximum PFHxS water concentration = 0.24 µg/L	RME	CTE
	RME	CTE		RME	CTE	RME	CTE
Birth to < 1 yr	1.113	0.504	7.8	3.42E-05	1.55E-05	1.7	0.8
1 to < 2 yr	0.893	0.308	11.4	1.88E-05	6.48E-06	0.9	0.3
2 to < 6 yr	0.977	0.376	17.4	1.35E-05	5.19E-06	0.7	0.2
6 to < 11 yr	1.404	0.511	31.8	1.06E-05	3.86E-06	0.5	0.2
11 to <16 yr	1.976	0.637	56.8	8.35E-06	2.69E-06	0.4	0.1
16 to <21 yr	2.444	0.77	71.6	8.19E-06	2.58E-06	0.4	0.1
Adults ≥ 21 yr	3.092	1.227	80	9.28E-06	3.68E-06	0.5	0.2
Lactating Women	3.588	1.665	73	1.18E-05	5.47E-06	0.6	0.3
Pregnant Women	2.589	0.872	73	8.35E-06	2.87E-06	0.4	0.1

Notes: CTE = central tendency exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure, µg/L = micrograms per liter, yr = year; HQ= Hazard Quotient; bold= exceeds HQ of 1.0

Table A-16. Warrington public water: Hazard Index for combined past exposures to PFOS, PFOA, and PFHxS (PFNA= non-detect).

Age Group	Exposure Assumptions	Exposure Assumptions	Exposure Assumptions	Hazard Index (HI)	Hazard Index (HI)
	Drinking Water Intake (L/day)	Drinking Water Intake (L/day)	Body Weight (kg)	RME	CTE
	RME	CTE		RME	CTE
Birth to < 1 yr	1.113	0.504	7.8	55.0	24.9
1 to < 2 yr	0.893	0.308	11.4	30.2	10.4
2 to < 6 yr	0.977	0.376	17.4	21.6	6.4
6 to < 11 yr	1.404	0.511	31.8	17.0	6.2
11 to <16 yr	1.976	0.637	56.8	13.4	4.3
16 to <21 yr	2.444	0.77	71.6	13.2	4.1
Adults ≥ 21 yr	3.092	1.227	80	14.9	5.9
Lactating Women	3.588	1.665	73	18.9	8.8
Pregnant Women	2.589	0.872	73	13.7	4.6

Notes: CTE = central tendency exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure, µg/L = micrograms per liter, yr = year; HI= Hazard Index, which sums the HQs for PFOS, PFOA and PFHxS (PFNA was non detect for this system) ; bold= exceeds HI of 1.0.

For past exposures in the WTWSD public drinking water supply wells, the HQ for PFOS exceeded 1.0 for all age categories using both RME and CTE. For PFOA, using RME method the HQ was greater than 1.0 for all age categories, and using the CTE approach was greater than 1.0 only for children less than 2 years of age. For PHxS, only the youngest age group (birth to <1 years) exceed

the HQ of 1.0 using RME assumptions. PFNA was non- detect in WTWSD, based on the data evaluated. The HI (combined PFOS, PFOA, and PFHxS) was greater than 1.0 for all age groups using the RME and CTE exposure assumptions.

The exposure doses, based on past exposures in the Warrington public water supply wells, are below the estimated PFOS NOAEL_{HED} (5.1×10^{-4} mg/kg/day) for reduced pup body weight in the Luebker study (Luebker et al. 2005; EPA 2016b). In addition, the doses are below the estimated PFOA LOAEL_{HED} (5.3×10^{-3} mg/kg/day) for delayed bone ossification and accelerated puberty in males in the Lau et al. 2006 animal study (EPA 2016c). These estimated exposure doses do not take into account PFOA and PFOS exposures from non-drinking water sources. The discussion on public health evaluation is presented in the Public Health Implications Section later in this document.

Exposure Dose Calculations: Private Drinking Water Wells Past Exposures

Estimated exposure doses were calculated for the RME (reasonable maximum exposure) and CTE (central tendency exposure) scenarios and compared to the ATSDR provisional MRLs described above. Tables A-17 through A-20 below provide a summary of the exposure doses and HQs for the private drinking water. Table A-21 provides the Hazard Index for combined exposures to PFOS, PFOA, PFHxS, and PFNA. While the ATSDR evaluation of private drinking water wells focused on the maximum private drinking water well concentrations as a worst-case-scenario, there was a range of PFAS concentrations in the individual private drinking water well sampled. As the PFAS levels increase in a private drinking water well, the potential increases for the possibility of harmful non-cancer health effects in adults and particularly for young children and infants. ATSDR is available, if requested, to evaluate and discuss individual private drinking water well sampling results and what the exposures mean for that family.

Table A-17. Private drinking water: past PFOS environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Age Group	Exposure Assumptions		Exposure Assumptions Body Weight (kg)	Estimated Exposure Dose (mg/kg/day)		HQ PFOS	
	Drinking Water Intake (L/day)	Drinking Water Intake (L/day)		Maximum PFOS water concentration = 5.2 µg/L	Maximum PFOS water concentration = 5.2 µg/L	RME	CTE
	RME	CTE		RME	CTE	RME	CTE
Birth to < 1 yr	1.113	0.504	7.8	7.42E-04	3.36E-04	371.0	168.0
1 to < 2 yr	0.893	0.308	11.4	4.07E-04	1.40E-04	203.7	70.2
2 to < 6 yr	0.977	0.376	17.4	2.92E-04	1.12E-04	146.0	41.8
6 to < 11 yr	1.404	0.511	31.8	2.30E-04	8.36E-05	114.8	41.8
11 to <16 yr	1.976	0.637	56.8	1.81E-04	5.83E-05	90.5	29.2
16 to <21 yr	2.444	0.77	71.6	1.77E-04	5.59E-05	88.7	28.0
Adults ≥ 21 yr	3.092	1.227	80	2.01E-04	7.98E-05	100.5	39.9
Lactating Women	3.588	1.665	73	2.56E-04	1.19E-04	127.8	59.3
Pregnant Women	2.589	0.872	73	1.84E-04	6.21E-05	92.2	31.1

Notes: CTE = central tendency exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure, µg/L = micrograms per liter, yr = year; HQ= Hazard Quotient; bold= exceeds HQ of 1.0

Table A-18. Private drinking water: past PFOA environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Age Group	Exposure Assumptions	Exposure Assumptions	Exposure Assumptions	Estimated Exposure Dose (mg/kg/day)	Estimated Exposure Dose (mg/kg/day)	HQ PFOA	HQ PFOA
	Drinking Water Intake (L/day)	Drinking Water Intake (L/day)	Body Weight (kg)	Maximum PFOA water concentration = 5.0 µg/L	Maximum PFOA water concentration = 5.0 µg/L		
	RME	CTE		RME	CTE	RME	CTE
Birth to <1 yr	1.113	0.504	7.8	7.13E-04	3.23E-04	237.8	107.7
1 to <2 yr	0.893	0.308	11.4	3.92E-04	1.35E-04	130.6	45.0
2 to <6 yr	0.977	0.376	17.4	2.81E-04	1.09E-04	93.6	36.0
6 to <11 yr	1.404	0.511	31.8	2.21E-04	8.03E-05	73.6	26.8
11 to <16 yr	1.976	0.637	56.8	1.74E-04	5.61E-05	58.0	18.7
16 to <21 yr	2.444	0.77	71.6	1.71E-04	5.38E-05	56.9	17.9
Adults ≥ 21 yr	3.092	1.227	80	1.93E-04	7.67E-05	64.4	25.6
Lactating Women	3.588	1.665	73	2.46E-04	1.14E-04	81.9	38.0
Pregnant Women	2.589	0.872	73	1.7E-04	5.97E-05	59.1	19.9

Notes: CTE = central tendency exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure, µg/L = micrograms per liter, yr = year; HQ= Hazard Quotient; bold= exceeds HQ of 1.0

Table A-19. Private drinking water: past PFHxS environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Age Group	Exposure Assumptions	Exposure Assumptions	Exposure Assumptions	Estimated Exposure Dose (mg/kg/day)	Estimated Exposure Dose (mg/kg/day)	HQ PFHxS	HQ PFHxS
	Drinking Water Intake (L/day)	Drinking Water Intake (L/day)	Body Weight (kg)	Maximum PFHxS water concentration = 5.0 µg/L	Maximum PFHxS water concentration = 5.0 µg/L		
	RME	CTE		RME	CTE	RME	CTE
Birth to < 1 yr	1.113	0.504	7.8	2.43E-04	1.10E-04	12.1	5.5
1 to < 2 yr	0.893	0.308	11.4	1.33E-04	4.59E-05	6.7	2.3
2 to < 6 yr	0.977	0.376	17.4	9.55E-05	3.67E-05	4.8	1.4
6 to < 11 yr	1.404	0.511	31.8	7.51E-05	2.73E-05	3.8	1.4
11 to <16 yr	1.976	0.637	56.8	5.91E-05	1.91E-05	3.0	1.0
16 to <21 yr	2.444	0.77	71.6	5.80E-05	1.83E-05	2.9	0.9
Adults ≥ 21 yr	3.092	1.227	80	6.57E-05	2.61E-05	3.3	1.3
Lactating Women	3.588	1.665	73	8.36E-05	3.88E-05	4.2	1.9
Pregnant Women	2.589	0.872	73	6.03E-05	2.03E-05	3.0	1.9

Notes: CTE = central tendency exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure, µg/L = micrograms per liter, yr = year; HQ= Hazard Quotient; bold= exceeds HQ of 1.0

Table A-20. Private drinking water: past PFNA environmental exposure assumptions, estimated exposure doses, and Hazard Quotients.

Age Group	Exposure Assumptions	Exposure Assumptions	Exposure Assumptions	Estimated Exposure Dose (mg/kg/day)	Estimated Exposure Dose (mg/kg/day)	HQ PFNA	HQ PFNA
	Drinking Water Intake (L/day)	Drinking Water Intake (L/day)	Body Weight (kg)	Maximum PFNA water concentration = 0.073 µg/L	Maximum PFNA water concentration = 0.073 µg/L		
	RME	CTE		RME	CTE	RME	CTE
Birth to < 1 yr	1.113	0.504	7.8	1.04E-05	4.72E-06	3.5	1.6
1 to < 2 yr	0.893	0.308	11.4	5.72E-06	1.97E-06	1.9	0.7
2 to < 6 yr	0.977	0.376	17.4	4.10E-06	1.58E-06	1.4	0.4
6 to < 11 yr	1.404	0.511	31.8	3.22E-06	1.17E-06	1.1	0.4
11 to <16 yr	1.976	0.637	56.8	2.54E-06	8.19E-07	0.8	0.3
16 to <21 yr	2.444	0.77	71.6	2.95E-06	7.85E-07	0.8	0.3
Adults ≥ 21 yr	3.092	1.227	80	3.82E-06	1.12E-06	0.9	0.4
Lactating Women	3.588	1.665	73	3.59E-06	1.67E-06	1.2	0.6
Pregnant Women	2.589	0.872	73	2.59E-06	8.72E-07	0.9	0.3

Notes: CTE = central tendency exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure, µg/L = micrograms per liter, yr = year; HQ= Hazard Quotient; bold= exceeds HQ of 1.0

Table A-21. Private drinking water: Hazard Index for combined past exposures to PFOS, PFOA, and PFHxS (PFNA=non-detect).

Age Group	Exposure Assumptions	Exposure Assumptions	Exposure Assumptions	Hazard Index (HI)	Hazard Index (HI)
	Drinking Water Intake (L/day)	Drinking Water Intake (L/day)	Body Weight (kg)		
	RME	CTE		RME	CTE
Birth to < 1 yr	1.113	0.504	7.8	487.8	220.9
1 to < 2 yr	0.893	0.308	11.4	267.8	92.4
2 to < 6 yr	0.977	0.376	17.4	191.9	62.9
6 to < 11 yr	1.404	0.511	31.8	150.9	54.9
11 to <16 yr	1.976	0.637	56.8	118.9	38.3
16 to <21 yr	2.444	0.77	71.6	116.7	36.8
Adults ≥ 21 yr	3.092	1.227	80	132.1	52.4
Lactating Women	3.588	1.665	73	168.0	78.0
Pregnant Women	2.589	0.872	73	121.2	40.8

Notes: This table uses the maximum values detected in a private water well. CTE = central tendency of exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure concentration, µg/L = micrograms per liter, yr = year; HI= Hazard Index, which sums the HQs for PFOS, PFOA, and PFHxS (PFNA was non detect in the private well with the maximum levels); bold= exceeds HI of 1.0; * The maximum total PFAS levels in a given well= 9.8 µg/L (PFOS = 3.8 µg/L, PFOA= 4.3 µg/L, PFHxS = 1.7 µg/L and PFNA= non-detect).

For the private drinking water wells, the HQ using the RME and CTE, for PFOA and PFOS exceeded 1.0 for all age groups. For PHxS, using RME and CTE assumptions, all the age categories exceeded 1.0 except ages 16 to 21 years (CTE assumption only). The HQ was exceeded for PFNA exposures using the RME (children < 11 years and lactating women) and CTE (children < than 1 years of age).

The maximum PFOS combined with PFOA in a given individual private drinking water well in the Willow Grove data set was 8.1 µg/L. However, ATSDR calculated the HI based on the maximum total PFAS level combined for PFOS, PFOA, PFHxS, and PFNA in an individual private drinking water well in the Willow Grove site data set. The maximum combined PFAS result for a private well at the Willow Grove site was 9.8 µg/L; this private well had PFOS at 3.8 µg/L, PFOA at 4.3 µg/L, PFHxS at 1.7 µg/L, and PFNA at non-detect. The HI ATSDR calculated for this private well was greater than 1.0 for all age groups using the RME and CTE exposure assumptions. If the HI is greater than 1.0, concern for the potential hazard of the mixture increases. As the PFAS levels increase, there is an increased potential for harmful non-cancer health effects in adults and particularly for young children and infants. ATSDR used the maximum drinking water sampling results as a conservative approach.

The estimated exposure doses, based on past exposure in the private drinking water wells, are below the estimated PFOS NOAEL_{HED} (5.1×10^{-4} mg/kg/day) for reduced pup body weight in the Luebker study (Luebker et al. 2005; EPA 2016b), with the exception of the RME for the birth to less than one year age category. In addition, they are below the estimated PFOA LOAEL_{HED} (5.3×10^{-3} mg/kg/day) for delayed bone ossification and accelerated puberty in males in the Lau et al. 2006 animal study (EPA 2016c). These estimated exposure doses do not take into account PFOA and PFOS exposures from non-drinking water sources.

Estimated Excess Cancer Risk Evaluation

Epidemiologic data suggest a link between PFOA exposure and elevated rates of kidney, prostate, and testicular cancer. However, additional studies are needed to confirm the link between PFOA and other PFAS exposures and cancer to say they are the cause. Currently, EPA considers the evidence suggestive that PFOA has the potential to be carcinogenic in humans and the Agency for Research on Cancer has determined that PFOA is possibly carcinogenic to humans (EPA 2016c). Animals given PFOA have shown higher rates of liver, testicular, and pancreatic tumors. We do not know if cancer at these three sites in animals results from a mode of action that is relevant to humans. Epidemiology studies of PFOS exposed workers observed an increased risk for some cancers; however, because of the small sample size, they were not statistically significant (Alexander et al. 2003; Alexander and Olsen 2007; Grice et al. 2006; Olsen et al. 2004). A causal link based on human studies between cancer and PFOS exposures remains uncertain. Animal studies have found limited, but suggestive evidence of PFOS exposure and increased incidence of liver, thyroid, and mammary tumors.

EPA calculated a PFOA oral slope factor⁵ as a comparison to the safety of their reference dose against carcinogenic effects, not as an official cancer oral slope factor for inclusion in their

⁵ EPA defines an oral slope factor as “An upper bound, approximating a 95% confidence limit, on the increased cancer risk from a lifetime oral exposure to an agent. This estimate, usually expressed in units of proportion (of a population) affected per mg/kg-day, is generally reserved for use in the low-dose region of the dose-response relationship, that is,

Integrated Risk Information System [EPA 2016b]⁶. Using the testicular cancer data from a 2012 rat study (Butenhoff et al. 2012) EPA calculated an oral slope factor of 0.07 mg/kg/day⁻¹ (EPA 2016c).

To estimate the lifetime excess cancer risk from exposure to PFOA in drinking water at this site, ATSDR used the following equation:

$$\text{Estimated excess cancer risk (PFOA)} = \frac{\text{Exposure dose (mg/kg/day)} \times \text{Oral slope factor (mg/kg/day)}^{-1} \times \text{Exposure time (years)}}{\text{Lifetime exposure (78 years)}}$$

ATSDR used the RME calculated for each water system to estimate cancer risk for PFOA. While it is not known when PFAS contaminated the groundwater and reached the public and private drinking water supply wells, for the numerator exposure term ATSDR used an exposure time of 40 years for adults based on the estimated historical site usage of PFAS, and an exposure time of 21 years for a child (representing an exposure from birth through age 21). Therefore, ATSDR assumed that adults were exposed to the maximum estimated PFOA concentration for a maximum of 40 years, which is likely to overestimate the estimated cancer risk.

The estimated cancer risk calculations, by age, for PFOA are presented in Table A-22. Based on these assumptions and assuming that the EPA oral slope factor on testicular cancer from a rat study approximates the actual cancer risk for PFOA, then the estimated adult cancer risk from exposure to the maximum detected PFOA concentration in the public water supply system is 4.02×10^{-7} (or 4 excess cases of cancer in 10,000,000 exposed individuals) in Horsham and is 1.7×10^{-7} (or 2 excess cases of cancer in 10,000,000 exposed individuals) in Warrington. This means that if 10 million people were similarly exposed, an additional 2-4 cases of cancer is estimated. For the private drinking water wells, the estimated cancer risk from exposure to the maximum PFOA concentration is 6.9×10^{-6} (or 7 excess cases of cancer in 1,000,000 exposed individuals). This means that if 1 million people were similarly exposed, an additional 7 cases of cancer is estimated. Please note, these are theoretical estimates that are a tool for making risk evaluations. These estimated cancer risk levels are considered a very low to low cancer risk. Exposures to lower PFOA concentrations would result in a lower estimated cancer risk. However, these theoretical cancer risk calculations must be viewed with caution because the EPA oral slope factor is not an official one for inclusion in IRIS, and other cancers that were elevated in epidemiological studies of PFOA exposure were not evaluated (i.e., kidney and prostate cancer).

Currently, EPA does not have oral slope factors for other PFAS because the animal data do not show a measurable or dose-response relationship. Therefore, ATSDR did not calculate the estimated cancer risk for PFAS exposures other than PFOA, and the actual cancer risk from combined PFAS exposures is uncertain.

for exposures corresponding to risks less than 1 in 100.” See also <https://www.epa.gov/fera/risk-assessment-carcinogenic-effects>.

⁶ The EPA IRIS assessment process is a rigorous seven-step process that includes development of a draft assessment, agency and interagency review, public and peer-review, and then agency and interagency review before finalization.

Table A-22. Estimated cancer risk for PFOA in public and private drinking water.

Age Group	Estimated Cancer Risk from Exposure to PFOA*	Estimated Cancer Risk from Exposure to PFOA*	Estimated Cancer Risk from Exposure to PFOA*	# of Exposure Yrs
	Horsham Public Wells (max PFOA of 0.29 µg/L)	Warrington Public Wells (max PFOA of 0.12 µg/L)	Private Drinking Water Wells (max PFOA of 5 µg/L)	
Child Birth to < 1 yr	3.72E-08	1.54E-08	6.40E-07	1
Child 1 to < 2 yr	2.04E-08	8.44E-09	3.52E-07	1
Child 2 to < 6 yr	5.85E-08	2.42E-08	1.01E-06	4
Child 6 to < 11 yr	5.74E-08	2.38E-08	9.92E-07	5
Child 11 to <16 yr	4.53E-08	1.87E-08	7.81E-07	5
Child 16 to <21 yr	4.44E-08	1.84E-08	7.67E-07	5
Children exposed for 21 years	2.63E-07	1.1E-07	4.54E-06	21
Adults (40 years)	4.02E-07	1.7E-07	6.31E-06	40

*Estimated cancer risk = RME exposure dose (mg/kg/day) x PFOA oral slope factor (0.07 mg/kg/day) x exposure years. While it is not known when PFAS contaminated the groundwater and reached the public and private water supply wells, ATSDR used an exposure time of 40 years for adults based on the estimated historical site usage of PFAS.

Public Health Implications

In this section, ATSDR summarizes some of the general public health implications of exposures to PFAS at this site, based on our evaluation of individual exposures and a limited mixtures analysis.

ATSDR used a conservative approach to evaluate the possibility for harmful health effects for noncancerous exposures until better methods are developed. ATSDR used a weight-of-evidence approach considering multiple exposures and factors to form our conclusions about PFAS exposures. ATSDR concluded drinking public water or private well water in the *past* could have increased the risk of harmful health effects, particularly for young children and infants.

Some (but not all) PFAS build up in the human body. The levels of some PFAS in humans go down slowly over time once exposure is reduced or stopped. Scientists across multiple federal agencies are studying how different amounts of PFAS in the body over time might affect human health. In addition, investigators are actively studying whether being exposed to multiple PFAS at the same time increases the risk of health effects.

Most existing research has focused on long-chain PFAS. These persist in the environment; bioaccumulate in wildlife and humans; and are toxic to laboratory animals, producing reproductive, developmental, and systemic effects in laboratory tests.

Long-chain PFAS comprise two sub-categories:

- perfluoroalkyl carboxylic acids (PFCAs) with eight or more carbons, including PFOA, and
- perfluoroalkane sulfonic acids (PFSAs) with six or more carbons, including
 - perfluorohexane sulfonic acid (PFHxS) and
 - perfluorooctane sulfonic acid (PFOS).

While persistent in the environment, PFCAs with fewer than eight carbons, such as perfluorohexanoic acid (PFHxA), and PFASs with fewer than six carbons, such as perfluorobutane sulfonic acid (PFBS), are generally less bioaccumulative in wildlife and humans (EPA 2018b). However, the health effects of many short-chained PFAS and new PFAS alternatives have not been fully researched.

It is important to remember that the likelihood of adverse health effects depends on several factors, such as the concentration of PFAS, as well as the frequency and duration of exposure. More frequent exposure can increase risk. Higher concentration and length of time exposed can lead to increased risk. The provisional MRLs developed for the four PFAS are protective for the entire population and are below levels where health endpoints such as toxicity to the kidney and liver, and high cholesterol may occur. In addition, ATSDR considered immune effects in the development of our MRLs as these effects may be more sensitive than developmental effects (ATSDR 2018). ATSDR considered several pieces of information to evaluate if health effects are likely from past exposures, including the following:

- Potential effects of exposures to PFOA, PFOS, PFNA and PFHxS (all PFAS with provisional ATSDR MRLs);
- Potential effects of exposures to a mixture of PFAS; and
- Potential effects on pre-existing conditions.

In the past, residents may have experienced higher or lower exposures that we cannot verify because we have no historical data. Background exposures from food, air, and consumer products would add to the cumulative exposures. Ultimately, all exposures contribute to the body burden and increase the risk of possible health effects. Those sources might include PFAS-contaminated food, hand-to-mouth transfer from surfaces previously treated with PFAS-containing stain protectants (e.g., carpet, most significant for infants and toddlers), or eating food packaged in material containing PFAS. We do not have enough information to identify the individual exposure sources to estimate the background exposure level in the public or private drinking water users.

A large number of studies have evaluated the toxicity of PFAS in humans by examining the possible association between serum PFAS levels and adverse health effects. The epidemiology studies fall into three broad categories: occupational exposure; exposure to PFOA-contaminated drinking water by residents living near a PFOA production facility; and general population exposure to background levels of perfluoroalkyls. Most of these studies have focused on PFOA and PFOS. As of today, based on studies in humans, scientists believe that some of the non-cancer health effects from PFAS exposure include:

- increased cholesterol levels
- changes in liver enzymes
- decreased vaccine response in children
- increased risk of high blood pressure or pre-eclampsia in pregnant women
- small decreases in infant birth weights

Although a number of scientific studies on PFAS health effects have been completed, outcomes of these studies have not been consistent and additional factors still need to be considered. More

research is needed to fully understand the possible negative health effects related to PFAS exposure. To date, scientists have learned that not all PFAS have the same health effects.

There are sufficient epidemiology data to identify possible sensitive targets for many of the PFAS. However, there are two major limitations to establishing dose-response relationships for these effects and using the epidemiology studies to derive the provisional intermediate MRLs: accurate identification of environmental exposure levels producing increased risk for adverse effects (exposure estimates and routes of exposure) and likely co-exposure to mixtures of PFAS (ATSDR 2018).

Exposures to PFOA and PFOS (individually)

Exposure doses from consuming public and private drinking water containing PFOA and PFOS were compared to the ATSDR provisional MRLs for PFOA and PFOS. Scientific information suggests an association between PFOA and PFOS exposure and various health effects. Scientific information suggests an association between PFOA and PFOS exposure and various health endpoints, including increased cholesterol levels, changes in liver enzymes, decreased vaccine response in children, increased risk of high blood pressure or pre-eclampsia in pregnant women, small decreases in infant birth weights, increased risk of kidney or testicular cancer. ATSDR based the PFOS and PFOA provisional intermediate MRLs on developmental endpoints, which we identified as the most sensitive health effect in the available animal studies (ATSDR 2018):

- **PFOA.** A provisional intermediate-duration (defined by ATSDR as exposure from 15-364 days) oral MRL of 3×10^{-6} mg/kg/day was derived for PFOA based on neurodevelopmental effects (i.e., altered activity at 5–8 weeks of age and skeletal alterations at 13 and 17 months of age) in the offspring of mice fed a diet containing PFOA (Koskela et al. 2016). The provisional intermediate MRL is based on a human equivalent dose (HED), lowest observed effect level (LOAEL) of 0.00087 mg/kg/day and a total uncertainty factor of 300 (10 for use of a LOAEL, 3 for extrapolation from animals to humans, and 10 for human variability).
- **PFOS.** For PFOS, a provisional intermediate-duration oral MRL of 2×10^{-6} mg/kg/day was derived based on developmental effects (i.e., delayed eye opening and transient decrease in body weight during lactation) in the offspring of rats administered PFOS (Luebker et al. 2005). The provisional intermediate MRL is based on a HED no observed adverse effect level (NOAEL) of 0.000515 mg/kg/day and a total uncertainty factor of 30 (3 for extrapolation from animals to humans with dosimetric adjustments and 10 for human variability) and a modifying factor of 10 for concern that immunotoxicity may be a more sensitive endpoint than developmental toxicity (ATSDR, 2018).

In addition to the developmental endpoints, the available epidemiology data identify several other potential health concerns related to exposures to PFOA and PFOS (ATSDR 2018):

PFOA

- Pregnancy-induced hypertension/pre-eclampsia
- Liver damage, as evidenced by increases in serum enzymes and decreases in serum bilirubin levels
- Increases in serum lipids, particularly total cholesterol and LDL cholesterol
- Increased risk of thyroid disease
- Decreased antibody response to vaccines

- Increased risk of asthma diagnosis
- Increased risk of decreased fertility
- Small (<20 g or 0.7 ounces per 1 ng/mL increase in blood perfluoroalkyl level) decreases in birth weight

PFOS

- Pregnancy-induced hypertension/pre-eclampsia
- Liver damage, as evidenced by increases in serum enzymes and decreases in serum bilirubin levels
- Increases in serum lipids, particularly total cholesterol and LDL cholesterol
- Increased risk of thyroid disease
- Decreased antibody response to vaccines
- Increased risk of decreased fertility
- Small (<20 g or 0.7 ounces per 1 ng/mL increase in blood perfluoroalkyl level) decreases in birth weight

As noted earlier, ATSDR calculated MOEs for exposures to PFOA and PFOS for the HSWA, Warrington, and private well users to put the estimated exposure doses into perspective:

1. For a HSWA public water user, assuming 100% of the PFAS exposure is from drinking water, the maximum PFOS exposure dose to a child younger than 1 year is 1.43×10^{-4} mg/kg/day. The MOE between that PFOS exposure dose and the LOAEL_{HED} (2.1×10^{-3} mg/kg/day) for developmental effects is about 15 for the RME scenario. The MOE between the exposure dose to PFOA and the LOAEL_{HED} (8.2×10^{-4} mg/kg/day) is about 20 for the RME scenario.
2. For a Warrington public water user, the maximum PFOS exposure dose for a child younger than 1 year is 9.52×10^{-5} mg/kg/day. The MOE between that PFOS exposure dose and the LOAEL_{HED} (2.1×10^{-3} mg/kg/day) for developmental effects is about 22 for the RME scenario. The MOE between the exposure dose of PFOA (1.71×10^{-5} mg/kg/day) and the estimated PFOA LOAEL_{HED} (8.2×10^{-4} mg/kg/day) is about 48 for the RME scenario.
3. For private well users, the maximum PFOS exposure dose for a child younger than 1 year is 7.24×10^{-4} mg/kg/day. The MOE between that PFOS exposure dose and the LOAEL_{HED} (2.1×10^{-3} mg/kg/day) for developmental effects is about 3 for the RME scenario. The MOE between the exposure dose of PFOA (1.71×10^{-5} mg/kg/day) and the estimated PFOA LOAEL_{HED} (8.2×10^{-4} mg/kg/day) is about 1.2 for the RME scenario.

The above MOE estimates support that past exposures to PFOS and PFOA from HWSA, Warrington, and private well users are of concern. These estimated drinking water exposures do not include additional PFOA and PFOS exposures from non-drinking water sources. ATSDR's margin of exposure calculations indicate that the most exposed population (children younger than 1 year) using the maximum levels of PFOS and PFOA detected in Horsham public water, Warrington public water, or a private well and drinking more than average amounts of water on a daily basis may have an increased risk of harmful effects.

ATSDR also evaluated the potential increased cancer risk for exposure to PFOA in drinking water. Some scientific information suggests a link between PFOA exposures and kidney and testicular cancer. Based on a lifetime exposure, the estimated cancer risk from past exposure to the maximum PFOA levels in public and private water wells is considered low. Scientific studies on PFOS or other PFAS compounds has not shown a quantitative or dose-response relationship for cancerous endpoints. Additional information on cancerous health effects are presented in the Cancer Effects section below.

Exposures to Perfluorononanoic acid (PFNA)

There have been human epidemiology studies of exposure to PFNA, but none of them were conducted in communities with drinking water contaminated with PFNA. Co-exposure to other PFAS make it difficult to draw conclusions about PFNA effects alone. However, the available epidemiology data identify several potential health hazards associated with exposure to PFNA including increases in serum lipids, particularly total cholesterol and LDL cholesterol, and decreased antibody response to vaccines (ATSDR 2018). Carcinogenic potential in animals exposed to PFNA has not been studied.

A provisional intermediate-duration oral MRL of 3×10^{-6} mg/kg/day was derived for PFNA based on decreased body weight gain and developmental delays in the offspring of mice administered PFNA via gavage on gestational days (GDs) 1–17 (Das et al. 2015). The provisional MRL is based on a HED NOAEL of 1.35×10^{-3} mg/kg/day and a total uncertainty factor of 30 (3 for extrapolation from animals to humans with dosimetric adjustments and 10 for human variability), and a modifying factor of 10 for database limitations. The LOAEL in this study is 3.0 mg/kg/day; adjusting this to a HED LOAEL using the same factor used for the NOAEL calculation (1,000) results in a HED LOAEL for this study of 0.003 mg/kg/day.

The intermediate-duration database consists of three developmental toxicity studies in rats and mice. The lowest LOAEL for developmental toxicity was 1.1 mg/kg/day in mice administered PFNA on GDs 1–18; at this dose, decreases in litter size and pup survival were observed (Wolf et al. 2010). At higher doses (2–5 mg/kg/day), decreases in pup body weight, delays in postnatal development (Das et al. 2015; Rogers et al. 2014; Wolf et al. 2010), increases in pup systolic blood pressure (Rogers et al. 2014), and reduced nephron endowment (Rogers et al. 2014) were observed. A study of PPAR α knockout mice did not find alterations in pup body weight or postnatal development at 2 mg/kg/day (Wolf et al. 2010).

PFNA was detected in both the Horsham water supply and the private drinking water wells, but only exceeded the ATSDR intermediate EMEG value for children (0.021 μ g/L) in the private drinking water wells. PFNA was not detected in the Warrington public drinking water wells. (As noted earlier, the private well with the highest total PFAS level combined was non-detect for PFNA.) To provide a MOE point of comparison for PFNA, ATSDR used the maximum single concentration detected for PFNA in the private well data set.

The highest dose for past exposures to PFNA alone in a private drinking water well was for a child (birth to less than one-year old, RME scenario). Based on this scenario, the maximum estimated PFNA exposure dose was 1.04×10^{-5} mg/kg/day. The MOE between the exposure dose and the HED LOAEL (0.003 mg/kg/day) described above for decreases in litter size and pup survival was about 288 for the RME scenario. Based on this calculation, it does not seem likely that exposure to

the highest level of PFNA detected in a private well to the most exposed population drinking more than the usual amounts of water would have an increased risk of health effects.

Exposures to Perfluorohexanesulfonic acid (PFHxS)

An ATSDR provisional intermediate-duration oral MRL of 2×10^{-5} mg/kg/day was derived for PFHxS based on thyroid follicular cell damage (considered the most sensitive health outcome) in adult male rats administered PFHxS for a minimum of 42 days (Butenhoff et al. 2009a; Hoberman and York 2003). The intermediate MRL is based on a HED NOAEL of 0.0047 mg/kg/day and a total uncertainty factor of 30 (3 for extrapolation from animals to humans and 10 for human variability) and a modifying factor of 10 for database limitations. The modifying factor for database limitations was added to account for the small number of studies examining the toxicity of PFHxS following intermediate-duration exposure and the limited scope of these studies examining immune effects, a sensitive endpoint for other PFAS, and general toxicity (ATSDR 2018). Using this weight-of-evidence approach, the available epidemiology data identify several potential health hazards of PFHxS in humans includes liver damage, as evidenced by increases in serum enzymes and decreases in serum bilirubin levels, and decreased antibody response to vaccines (ATSDR 2018).

Although more research is needed, current scientific information suggests liver (Gleason, et al. 2015), antibody response (immune) (Granjean et al. 2012; Mogenssen et al. 2015; Stein et al. 2016), thyroid (Butenhoff, et al. 2009; Hoberman and York 2003), and developmental effects (Viberg et al. 2013) are possible from PFHxS exposures. Two of these studies by Butenhoff, et al. (2009) and Hoberman and York (2003) studied the effect of PFHxS exposures in animals. They observed thyroid effects as the most sensitive outcome. Butenhoff, et al. (2009) identified a NOAEL of 1 mg/kg/day and a LOAEL of 3 mg/kg/day. In an NHANES analysis, serum PFHxS levels (mean of 2.5 ng/mL) were negatively associated with a change in non-HDL cholesterol, and increased odds of having high cholesterol were found with increasing serum levels (Odds ratio of 1.27, 95% Confidence Interval 1.11-1.45) (Nelson et al 2010).

PFHxS was detected in both the public and the private drinking water wells above the ATSDR intermediate child EMEG value. The tables above show potential exposure doses from PFHxS, based on the maximum PFHxS concentrations.

- Private Wells: The highest dose for past exposures to PFHxS alone was for a child (birth to less than one-year old, RME scenario), in private drinking water wells. Based on this scenario and the maximum estimated PFHxS concentration, the daily dose is 2.43×10^{-4} mg/kg/day. The MOE between the exposure dose and the LOAEL_{HED} (0.0047 mg/kg/day) for thyroid effects was about 19 for the RME scenario.
- Horsham Public Water: The PFHxS exposure dose for a child younger than 1 year is 8.42×10^{-5} mg/kg/day, based on for the maximum Horsham public well concentration. The MOE between the exposure dose and the LOAEL_{HED} (0.0047 mg/kg/day) for thyroid effects was about 56 for the RME scenario.
- Warrington Public Water: For Warrington, the PFHxS exposure dose for a child younger than 1 year is 3.42×10^{-5} mg/kg/day, based on for the maximum Warrington public well concentration. The MOE between the exposure dose and the LOAEL_{HED} (0.0047

mg/kg/day) for thyroid effects was about 137 for the RME scenario.

ATSDR's margin of exposure calculations indicate that the most exposed population (children younger than 1 year) using the maximum levels of PFHxS detected in Horsham public water or a private well and drinking more than average amounts of water on a daily basis may have an increased risk of harmful effects.

Exposures to Perfluoroheptanoic acid (PFHpA)

PFHpA was detected in both the public and the private drinking water wells. However, currently, ATSDR does not have a health-based CV for PFHpA which limits our ability to evaluate potential past exposures. ATSDR identified several human studies for cardiovascular disease, serum lipids, immune response, and other effects that found either a limited association or no association. No studies for PFHpA were identified to allow ATSDR to compare the exposure doses from the public and private drinking water to effect levels (i.e., NOAELs or LOAELs) (ATSDR 2018). Therefore, ATSDR cannot make any health conclusions for PFHpA at this time.

Exposures to Perfluorohexanoate (PFHxA)

Very limited information is available relating to the health effects of PFHxA exposure. PFHxA was only sampled for in the HWSA public water supply wells. It was detected in the HWSA public water supply wells at a maximum of 0.046 µg/L. However, ATSDR currently does not have a CV for PFHxA. One study evaluated the chronic oral (ingestion) toxicity of PFHxA in laboratory animals (Klaunig et al. 2015). Exposure to female rats to 200 mg/kg/day resulted in hematological alterations (decreases in red blood cells, hemoglobin levels, and increases in reticulocyte counts), renal effects (tubular degeneration, necrosis, increased urine volume, and reduced specific gravity), and liver effects (necrosis). No adverse alterations (NOAELs) were observed at 30 mg/kg/day (females) or at 100 mg/kg/day (males). One major uncertainty related to this study is that serum PFHxA levels were not measured. Based on this study alone, harmful effects are unlikely. However, this PFAS has not been studied as extensively as the PFAS with ATSDR provisional MRLs (especially for the most sensitive health endpoints such as developmental and immune effects), and the only identified chronic study has limitations.

Exposures to Other PFAS

Very little scientific information is available from either human or animal studies about the health effects of exposure to other PFAS. In 2015, HWSA also analyzed water samples for the PFHxA, PFDA, PFDoA, PFTA, PFTDA, and PFUA. The levels of these PFAS were non-detect. These additional PFAS were not sampled for in WTWSD public water supply wells or private drinking water wells. The health effects of many short-chained PFAS and new PFAS alternatives have not been fully researched.

Exposure to a Mixture of PFAS

To evaluate the potential risk for cumulative exposures to PFOA, PFOS, PFHxS, and PFNA (those PFAS with ATSDR-derived provisional MRLs), ATSDR calculated a hazard index. The hazard index approach assumes dose additivity to assess the non-cancer health effects of a mixture. The hazard index is the sum of the HQs for each of the four PFAS with ATSDR provisional MRLs. If the hazard index is less than 1.0, it is unlikely that significant additive or toxic interactions would occur; so no further evaluation is necessary. If the hazard index is greater than 1.0, concern for the

potential hazard of the mixture increases. Only two studies (Carr et al. 2013; Wolf et al. 2014) have shown binary pairs of PFAS (i.e., comparing only two PFAS together) demonstrate concentration and response additivity at lower concentrations, but deviate from additivity at higher concentrations (Wolf et al. 2014). These possible interactions (or dose additivity) complicate the interpretation of the epidemiology data. Because of these limited data, ATSDR cannot assume any mixture effect except additivity.

With the exception of the hazard index approach for PFOA, PFOS, PFHxS, and PFNA, there is not a broadly accepted scientific method to quantitatively evaluate the possible health effects of combined exposures to PFAS. In addition, as stated previously, not all PFAS share the same health outcomes. Therefore, ATSDR evaluated the scientific literature to determine what health effects from the chemicals in the PFAS mixture found in the Willow Grove site area might have similar health endpoints.

ATSDR calculated exposure doses and compared these exposure doses to the provisional ATSDR intermediate MRLs for PFOS, PFOA, PFHxS and PFNA to determine a hazard quotient (HQ). The sum of the HQs was used to determine a hazard index (HI).

The PFAS compounds PFOA, PFOS, PFHxS, and PFNA have the most scientific information and studies available on potential health effects. However, both human epidemiological or animal studies are available for some of the other PFAS detected in the public and private drinking water. As shown in Table A-23, each of the target organ systems have at least two PFAS compounds that have been shown to be associated with that effect either in animal or human epidemiological studies. Moreover, developmental, liver, and endocrine effects have at least four of the PFAS compounds associated with that health outcome. Therefore, the combined exposures to PFAS compounds in the drinking water of this community may have increased the risk for some of these non-cancer health outcomes. Specifically, the combined exposures to a mixture of PFOS, PFOA, PFHxS, and PFNA could have increased the risk for developmental and immune effects above what might be expected from exposure to any of these PFAS alone. For other PFAS associations and endpoints, however, the scientific information is far less certain.

Table A-23. PFAS and possible effects on organ systems.

PFAS	Cardiovascular	Developmental	Endocrine	Liver	Immune	Reproductive	Serum Lipid
PFBS	○	●	●	○	○	●	○
PFHpA	○	○	○	○	○	○	○
PFHxA	○	●	○	●	○	○	○
PFHxS	○	●	●	●	●	○	○
PFNA	○	●	○	○	○	○	●
PFOA	●	●	●	●	●	●	●
PFOS	●	●	●	●	●	●	●

Notes: ● = Indicates possible impacts on this target organ system; ○ = Indicates no impacts or insufficient information.

Abbreviation	Definition	Citation(s) for effects (if applicable)
PFBS	perfluorobutane sulfonic acid	Minnesota Department of Health 2017
PFHpA	perfluoroheptanoic acid	No effects or insufficient information on target organ systems
PFHxA	perfluorohexanoic acid	Iwai and Hoberman 2014; Klaunig et al. 2015

PFHxS	Perfluorohexane sulfonic acid	Butenhoff et al. 2009; Gleason et al. 2015; Grandjean et al. 2012; Morgensen et al. 2015; Viberg et al. 2013
PFNA	perfluorononanoic acid	Das et al. 2015; Starling et al. 2014; Zeng, et al. 2015
PFOA	perfluorooctanoic acid	ATSDR 2018a
PFOS	perfluorooctane sulfonic acid	ATSDR 2018a

Susceptible Populations: Pre-existing Conditions and Developmental Effects

The available epidemiology data identify several potential targets of toxicity of PFOA and PFOS, and people with pre-existing conditions may be unusually susceptible. For example, it appears that exposure to PFOA or PFOS may increase serum lipid levels, particularly cholesterol levels. Thus, an increase in serum cholesterol may result in a greater health impact in persons with high levels of cholesterol or with other existing cardiovascular risk factors. Similarly, increases in uric acid levels have been observed in persons with higher PFAS levels; increased uric acid may be associated with an increased risk of high blood pressure. Thus, people with hypertension may be at greater risk. The liver is a sensitive target in many animal species and might be a target in humans. Therefore, people with compromised liver function could be a susceptible population [ATSDR 2015, 2018a]. Finally, human studies have indicated that some PFAS may affect immune function [ATSDR 2018a]. Therefore, persons who are immunocompromised may also be a susceptible population to PFAS exposures. The relationship between PFOA and PFOS exposure and increased risk for cardiovascular disease is currently inconclusive. Additional research is needed to understand how exposure to these chemicals might affect people with pre-existing risk factors (such as elevated cholesterol) for cardiovascular disease.

ATSDR recognizes that the unique vulnerabilities of the unborn, infants, and children demand special consideration in communities affected by environmental contamination. A child's developing body systems can sustain damage if toxic exposures occur during critical growth stages. Children ingest a larger amount of water relative to body weight than adults, resulting in a higher intake of pollutants in proportion to body size. In addition, children exhibit hand-to-mouth behavior and could be exposed to PFAS from previously treated carpet materials.

In humans, exposure to PFAS before birth or in early childhood may result in decreased birth weight (Verner, M.A. 2015; Darrow, L.A., Stein, C.R., & Steenland, K. 2013; Olsen, G.W., Butenhoff, J.L, & Zobel, J. 2009; Fei, C. 2008), decreased immune responses (Locker, C., et al 2014; Dong, G.H. 2013), and hormonal effects later in life. More research is needed to understand the role of PFAS and effects in human development. A recent study found that PFAS exposures in children at levels similar to the NHANES average were associated with lower antibody responses to childhood immunizations and an increased risk of antibody concentrations below the level needed to provide long-term protection (Grandjean et al 2012). Reducing exposures to PFAS in infants and young children is extremely important because of their unique vulnerabilities. As evidence for this concern:

- Formula-fed infants consuming formula mixed with contaminated water would have a higher exposure compared to adults as a result of formula being their sole or primary food source and their smaller body weight.
- Evidence suggests that high serum (human blood) PFOA or PFOS levels are associated with lower birth weights. Studies of populations with lower serum PFOA or PFOS levels have not found significant associations with birth weight. Although significant associations were

found for the high serum group, decreases in birth weight were small and may not be biologically relevant (ATSDR 2018a).

Cancer Effects

Under the EPA 2005 cancer guidelines, the evidence for the carcinogenicity of PFOS is considered “suggestive of carcinogenicity,” but not sufficient to assess human carcinogenicity potential. Animal studies have found limited, but suggestive evidence of PFOS exposure and increased incidence of liver, thyroid, and mammary tumors. Epidemiologic data suggest a link between PFOA exposure and elevated rates of kidney and testicular cancer. However, additional studies are needed to confirm the link between PFOA and other PFAS exposures and cancer to establish causality. Currently, EPA considers the evidence suggestive that PFOA has the potential to be carcinogenic in humans and the International Agency for Research on Cancer (IARC) has determined that PFOA is possibly carcinogenic to humans (EPA 2016d; IARC 2017). The workgroup found that an increased risk of kidney cancer with a statistically significant exposure–response trend was reported in workers in a fluoropolymer production plant in West Virginia, and in an exposed community near the plant. Increases of about threefold in the risk of testicular cancer were reported in the most highly exposed residents of communities near the same plant (Benbrahim-Tallaa et al 2014). Carcinogenicity of other PFAS has not yet been fully evaluated. To date, human studies of PFAS have not demonstrated conclusive causal associations between PFAS exposure and cancer outcomes. In general, epidemiological associations for most cancer endpoints for both PFOA and PFOS are mixed (EPA 2016d).

In occupationally exposed workers, cancers associated with exposure to PFOS or PFOA include male reproductive, kidney and bladder cancers, though the associations are generally weak and are not consistent across studies. In addition, the sample sizes for many of these studies are small, such that caution is needed in interpreting the results. For example, in occupationally exposed populations:

- In a series of studies, Olsen, Alexander and colleagues examined the health effects of occupationally exposed workers at the 3M plant in Decatur, Alabama. In one study, the health claims of employees at the Decatur plant were used as an indicator of the workers’ morbidity. The authors found that an increased risk of medical care for reproductive male cancers was associated with long-term, high exposure workers, and that the risk ratio for medical care related to cancers was greatest for the employees with the highest and longest exposure to PFAS. A later study examining 2,083 workers at the plant showed that workers with a high exposure to PFOS had a risk 13 times greater than the general population for developing bladder cancer (standard mortality ratio (SMR) = 12.77, 95% confidence interval (CI) = 2.63-37.35) (Alexander et al 2003). A follow-up study, however, failed to demonstrate a statistically significant link between bladder cancer and PFOS exposure (Alexander B.H. & Olsen, G.W. 2007).
- A study of exposed workers at the 3M Cottage Grove plant in Minnesota showed a weak association between exposure to PFOA and prostate cancer (SMR = 2.03, 95% CI = 0.55-4.59) (Gilliland and Mandel 1993). However, follow-up studies of workers from the same plant were unable to demonstrate this link (ATSDR 2013, EFSA 2008).
- In a study of exposed workers at the DuPont Washington Works manufacturing facility in West Virginia, Leonard et al. (2008) found a statistically non-significant elevation of mortality risk for kidney cancers (SMR=1.56, 95% CI = 0.80-2.72). No statistically

significant excesses were reported for the other cancers studied, though the number of specific cancers reported in the exposed population was small.

Various studies have also examined cancer outcomes in non-occupationally exposed members of the general population. Cancers potentially linked with PFOS or PFOA exposure in the general population include testicular, kidney, and breast cancer, though results remain inconclusive. Additionally, no association has been observed between PFOS or PFOA exposure and a variety of other cancers. For example:

- In the C8 Science Panel's (2015) examination of 69,030 people exposed to PFOA-contaminated drinking water in Ohio and West Virginia, a "probable link analysis" on 21 types of cancer identified in their population concluded that there was a probable link between PFOA and both testicular cancer and kidney cancer, but no probable link between PFOA and the other types of cancer studied. The Panel did find some positive evidence linking PFOA exposure to thyroid cancer and melanoma, but did not judge that the link was statistically significant or supported by other studies. A median PFOA serum concentration of 28.2 µg/L was previously reported for the full population of 69,030 individuals (Frisbee et al 2009), but only 32,254 of these people were evaluated for cancer effects.
- A study of 57,053 Danish people with low PFOS and PFOA plasma concentrations relative to occupationally exposed workers found no significant link between PFOS and PFOA plasma concentrations in the general population and the risk of prostate, bladder, pancreatic, or liver cancer (Ericksen et al 2009). Plasma PFAS concentrations in the population ranged from 1-76.4 µg/L for PFOA and from 1-130.5 µg/L for PFOS. In the subgroup of cancer patients, median plasma concentrations of PFOA were 6.8 µg/L for men and 6.0 µg/L for women, while median plasma concentrations of PFOS were 35.1 µg/L for men and 32.1 µg/L for women.
- In a small study of 31 Greenlandic Inuit women with breast cancer, Bonefeld-Jorgensen et al. (2011) found a statistically significant increased risk of breast cancer associated with serum levels of PFOS and the total concentration of PFASs, though not with serum levels of PFOA. The median serum PFOS concentration was 45.6 µg/L with a range of 11.6-124 µg/L, while the median total concentration of PFASs was 48.2 µg/L with a range of 13.2-133 µg/L. The median PFOA concentration was 2.5 µg/L with a range of 0.2-7.2 µg/L.

Studies of animals given large amounts of PFOA found liver, testicular, and pancreatic cancers but more studies are needed to determine the risk of cancer for people (Butenhoff, J.L. et al 2012; Perkins R.G., et al 2004). From 2005 to 2013, the C8 Science Panel conducted an epidemiologic study of approximately 70,000 people in the Ohio River Valley examining a possible link between PFOA and testicular and kidney cancer (Vaugh B. Winquist A. & Steenland K. 2013). Additional studies are needed to evaluate this possible link. Occupational studies examining whether PFAS are linked to prostate, bladder and liver cancer in PFAS manufacturing workers have not found a link. Additional studies are underway (C8 Science Panel 2015; ATSDR 2018).

At this time, EPA does not have an oral slope factor for PFOS because the data do not show a quantitative or dose-response relationship. Therefore, ATSDR cannot quantitatively evaluate the estimated cancer risk from PFOS exposures. Based on the cancer risk calculations and exposure assumptions described previously, the estimated lifetime cancer risk (40 years of exposure) from exposure to the maximum detected PFOA concentration in the public water supply system is 4.02×10^{-06} (or 4 excess cases of cancer in 1,000,000 exposed individuals) in Horsham and is 1.7×10^{-07}

(or 2 excess cases of cancer in 1,000,000 exposed individuals) in Warrington. For the private wells, the estimated cancer risk from exposure to the maximum PFOA concentration is 6.9×10^{-6} (or 7 excess cases of cancer in 1,000,000 exposed individuals). These estimated cancer risk levels are considered very low cancer risk.

Cancer Incidence Data Reviews for this Community

In 2017, PADOH and ATSDR collaborated to produce an initial review of cancer data incidence rates for community members living in selected zip codes surrounding the site (Warminster 18974, Warrington 18976, and Horsham 19044) over the time period of 1985-2011. In 2018, PADOH produced a first addendum to update this review with the addition of pancreatic and pediatric cancers for these same zip codes and the same time period. For these analyses, PADOH calculated incidence rates using information reported to the Pennsylvania state cancer registry and compared this information at the zip code level to state wide or county wide rates by calculating standardized incidence ratios.

In May 2018, PADOH released a second cancer review addendum, which included additional years of cancer registry data, and used a more refined geographical analysis going down to the census block level (including geocoding of cancer cases) that more closely matches the water distribution system in the area of concern in southeast Pennsylvania. The second addendum supersedes the prior cancer data reviews.⁷ The key findings of PADOH's cancer incidence analysis for the area, based on the May 2018 second addendum refined analysis, include:

- The incidence rates for most of the cancers of concern (myeloma, non-Hodgkin lymphoma, cancers of bladder, kidney and childhood cancers) in all of the water service areas during 1995-2004 were similar to the incidence rates in the rest of Montgomery and Bucks counties and in the rest of Pennsylvania.
- During the period 2005-2014, male bladder cancer rates in the combined water service area and the HWSA area and the female bladder cancer rate in the WTWSD area were higher than the rates in the rest of Montgomery and Bucks counties. However, only the female bladder cancer rate in the WTWSD area remained higher during the same period when compared to the rate in the rest of Pennsylvania.
- The male kidney cancer rate in the Warminster Municipal Authority (WMA) area and the female kidney cancer rate in the combined water service area were higher than the rates in the rest of Montgomery and Bucks counties during 2005-2014, whereas only the male kidney cancer rate in the WMA area remained higher when compared to the rate in the rest of the state.
- The incidence rate for non-Hodgkin lymphoma among males in the WTWSD area during 2005-2014 was higher than the rate in the rest of Montgomery and Bucks counties; however, it was similar to the state rate during the same period.
- Female pancreatic cancer rates in both the WTWSD area and in the combined water service area were higher than rates in the rest of Montgomery and Bucks counties during 2005-

⁷ Addendum 2 to the August 2016 Cancer Data Review: Selected Zip codes of Warminster, Warrington, and Horsham, Pennsylvania is available at <https://www.health.pa.gov/topics/Documents/Environmental%20Health/Addendum%202%20to%20Cancer%20Data%20Review%201985-2013.pdf>.

2014, but only the rate in the WTWSD area was higher in comparison to the rate in the rest of the state.

- The male childhood cancer incidence rate in the WTWSD area was higher than the rates in the rest of Montgomery and Bucks counties and in the rest of Pennsylvania during 2005-2014.
- The incidence rates for cancers of the liver and of the testis in all water service areas were similar to rates in the rest of Montgomery and Bucks counties and in the rest of Pennsylvania during both periods.
- Bladder cancer incidence rates among women in HWSA were significantly lower during 2005-2014 compared to the rest of Montgomery and Bucks counties, as well as to the rest of Pennsylvania.
- The incidence rate for myeloma among men in WMA was significantly lower compared to the rest of Pennsylvania during 2005-2014.
- During 1995-2004, prostate cancer incidence rate in the combined water service area was significantly lower compared to the rest of Montgomery and Bucks counties. Compared to the rest of Pennsylvania, the rates were significantly lower in all water service areas (including the combined area) except WTWSD/NWWA during the same period.

This refined 2018 analysis by PADOH presents elevated incidence rates for some types of cancers in comparison to the rest of Montgomery and Bucks counties and to the rest of the state of Pennsylvania. However, these results do not show a consistent pattern. While it is possible to have different levels of contaminants of concern in the water supply in different water service areas depending on the source of water, the current results do not indicate consistently higher incidence rates in all service areas for any cancer type. The results also do not indicate consistently higher incidence rates for a given cancer in both sexes. Sex is not a known risk factor for any of the cancer types included in this analysis (except cancers of the prostate and testis).

People with certain risk factors may be more likely than others to develop cancers. For instance, the risk factors for pancreatic cancer include smoking, diabetes, pancreatitis and obesity, among others. Tobacco use is a major risk factor for bladder cancer as well. However, the current analysis was not able to consider the prevalence of these risk factors in the populations studied, as these types of data are not available in the cancer registry. Other pertinent information, such as the length of residence in the area and whether the individual was employed at the Warminster or Willow Grove bases, was also not available in the cancer registry for analysis. Cancer has a long latency period. For many cancer types, it may take decades for a cancer to develop and be diagnosed. People also migrate from one location to another, from one state to another, or even from one country to another, and, therefore, it becomes difficult to find the source of exposure that may have caused a particular cancer. Cancers diagnosed in PA residents are only reported to the PA cancer registry.

Even when a statistically significant increase in cancer incidence is detected, determining the validity of an association between an environmental agent and the development of cancer is difficult, as behavioral (e.g., nutrition, physical activity and substance use), genetic (e.g., inherited mutations, hormones and immune conditions) and environmental (e.g., chemicals, radiation, pathogens and other contaminants) factors interact and affect cancer growth. These factors may act together or in sequence to initiate or promote cancer. Some of the highest PFAS in drinking water in this area occurred among residents who used private drinking water wells but were within the

geographical area of public water service. However, in this analysis, no distinction was made between public water users and private well water users (PADOH 2018).

PADOH PFAS Exposures Assessment Technical Tools Pilot Biomonitoring Project

In 2018, the PADOH was awarded a grant from the Association of State and Territorial Health Officials (ASTHO), with support from CDC/ATSDR, to implement a pilot biomonitoring study using the CDC/ATSDR PFAS Exposures Assessment Technical Tools (PEATT). ATSDR developed the PFAS PEATT to help State, local, tribal, and territorial health departments conduct PFAS biomonitoring activities. The PEATT was designed to characterize exposure in an affected population using biomonitoring, and includes a protocol for statistically based representative sampling, risk communication materials, questionnaires, and water sampling protocol. The work that has taken place by PADOH will ultimately contribute to the overall body of knowledge we have on the topic of PFAS and refine what is needed to describe exposure in a community. The following provides a summary of the pilot study and results. Additional information and details on PADOH's biomonitoring in this community can be found in the final PEATT pilot project report, released in April 2019 (PADOH 2019).

PADOH implemented the PEATT Pilot Project in Montgomery and Bucks counties near the Willow Grove and Warminster sites. The selection area included the water service areas under the HWSA, WMA, WTWSD, NWWA, and Warminster Water Authority. This included the towns of: Ambler, Horsham, Hatboro, Chalfont, Warminster, Jamison, Warrington and North Wales. The area has 32,595 households with a population of 84,184 based on the 2010 census. PADOH used a one-stage cluster sampling of households for biomonitoring as indicated in the PEATT. Individuals who were currently living and had lived in the above-mentioned water service areas prior to June 1, 2016 (this date refers to when all public water wells in the area having PFOS/PFOA at or above EPA's HA and residents with private wells having levels above EPA's HA started receiving bottled water), were considered eligible for the study. The study goal was participation by 500 individuals from 350 households (estimated 2.6 individuals per household). Households were selected randomly from the list of all households within the service areas of the above-mentioned public water systems and all household members, including children (3 to 17 years), were recruited for biomonitoring.

Initial letters of interest along with eligibility forms were sent to 350 households in the affected region. The letter also included a questionnaire. The questionnaires asked about demographic factors, drinking water habits, years of residence in current and prior area homes, health conditions, pregnancy status if female, workplace locations, and water sources. Child questionnaires included questions about school/daycare water sources, as well as breastfeeding and formula consumption.

PADOH collaborated with the local health departments of Montgomery and Bucks counties, the Pennsylvania State Bureau of Laboratories (BOL) and New York State Department of Health (NYSDOH) in blood sample collection and analysis. This laboratory provided testing and analysis of an 11-compound panel of PFAS (PFBS, PFHpA, PFHxS, PFNA, PFOA, PFOS, PFDeA, PFUA, PFDoA, PFOSA, and MeFOSAA). A total of 235 individuals submitted blood samples for testing from May to September 2018. Of those tested, 12 (5.1%) were children aged 3-11 years, 19 (8.1%) were aged 12-19 years and 204 (86.8%) were aged 20 years or older. The majority of the individuals tested were females (n=131, 55.7%).

The pilot study showed residents in the study area had elevated levels of PFAS compared to the U.S. general population. The average levels of PFOA, PFOS, PFHxS and PFNA among participants of the study were higher than the average levels reported at the national level based on the 2013-2014 NHANES survey. However, elevated levels of PFAS observed among the community members in the current study are comparable to levels reported in other communities with PFAS contaminated drinking water. New Hampshire residents exposed to drinking water contaminated with PFAS from a nearby military base showed an average community level of 3.09 µg/L for PFOA and 8.59 µg/L for PFOS. In Minnesota, residents exposed to drinking water contaminated with PFAS from industrial sources had an average community level of 17 µg/L for PFOS.

Among the 11 PFAS tested for, only four compounds (PFOS, PFOA, PFHxS and PFNA) were detected consistently. PFOS was detected in all 235 participants. Two hundred and thirty-two, 233 and 185 participants had PFOA, PFHxS and PFNA in their serum samples, respectively. Overall, 75, 81, 94, and 59 percent of the study participants had levels exceeding the national average for PFOA (1.94 µg/L), PFOS (4.99 µg/L), PFHxS (1.35 µg/L) and PFNA (0.68 µg/L), respectively.

The levels of PFOA, PFOS, PFHxS and PFNA among different age groups within the community differed significantly. This is consistent with other studies that examined PFAS levels compared to age category, particularly with PFOS. Those reported using private wells as their drinking water source in the study had higher levels of PFOA, PFOS and PFNA in comparison to those using public water as the drinking water source. However, the levels were not significantly different ($P > 0.05$ for all). Males had higher PFAS levels than females except for PFNA, though the differences were not statistically significant. The lower levels found in females is often attributed to female elimination routes such as breast feeding and menstruation. The results also showed a strong association between participants' length of residence and PFAS serum levels with longer residence time corresponding to higher PFAS concentrations in participants' blood in general. Exceptions were the groups with less than five years of residential history and those with 10-19 years of residential history having higher levels for PFOS and PFNA than groups with 5-9 and 20-29 years of residential history respectively—an inconsistency that disappeared when residential length was regrouped to represent the groups with shorter versus longer residential histories. The study also indicated higher, though not statistically significant, PFAS levels, except for PFNA, among those who reported being ever employed on the military base.

The study participants were asked to report up to 10 health conditions they experienced and/or diagnosed with. Eighty-six participants (36.6%) did not report any health condition, 128 participants (54.5%) reported one to four conditions and 21 participants (9 %) reported five or more health conditions. Ninety-four participants reported at least one health condition, with 23 of them reporting two or more health conditions. The most frequently reported health condition was elevated cholesterol level, followed by endocrine disruptions and cancer.

Limitations and Uncertainties of Human Health Risks from PFAS Exposures

Several limitations and uncertainties, such as the following, affect efforts to evaluate human health risks from PFAS exposures in drinking water:

- 1) multiple exposure sources
- 2) lack of historical exposure data
- 3) incomplete information on AFFF used at Willow Grove and specific PFAS formulations

4) limited information and methods to assess public health implications

Multiple Exposure Sources

In addition to drinking water exposures, community members likely have additional exposures to PFAS from other sources. These could include food, dust, air, and consumer products (e.g. non-stick cookware, stain resistant carpets, water repellent clothing, cleaning products, personal care products, paints, etc.). Exposures might also occur by touching surfaces treated with a stain protector and then touching your mouth or food that you eat. All sources add to the amount of chemicals in your body and potential health effects.

Even after exposure to drinking water contaminated with PFAS ends, it takes a long time for levels of PFAS in a person's blood to decline. A half-life represents the time it takes for the level of PFAS in the serum to reduce by half. Most PFAS half-life studies have been conducted in adults or animals; there is a need to understand if these are applicable to children. There are no studies that have examined whether young animals are more or less susceptible than adults to perfluoroalkyls toxicity (ATSDR 2018). PFOA and PFOS have a very slow rate of elimination from serum; elimination half-lives are estimated to be several years in humans. Several studies have estimated the PFOA and PFOS half-lives in workers and residents. Studies of the half-life of PFOA have shown a range of potential half-life values from 2.3 years for environmentally exposed populations to 5.1 years for occupationally exposed populations. Olsen et al (2007) estimated the half-life of PFOA in humans to be 3.8 years and half-life of PFOS to be 5.4 years (ATSDR 2018). For PFHxS, the estimated half-life of 8.5 years is based on longitudinal measurements of serum concentrations of PFHxS in a group of retired fluorochemical production workers observed for a 5-year period (Olsen et al 2007). For PFNA, data on the half-life in the body is more limited, but one study showed a mean half-life for PFNA of 2.5 (young females) and 4.3 years (males and older females).

Lack of Historical Exposure Data

We do not know exactly how long and at what concentrations residents were exposed to PFAS in public and private drinking water sources. Historical sampling data are unavailable. Exposures might have occurred for years through PFAS movement in groundwater. PFAS compounds accumulate and remain in the body for years before elimination. Past and current exposures contribute to the overall health risks from PFAS.

Incomplete Information on AFFF used at Willow Grove and Specific PFAS Formulations

One of the challenges to evaluating exposures from an AFFF source is that we do not know all of the PFAS constituents and that these constituents have changed over time. Data on AFFF-impacted groundwater indicate that about 25% of the PFAS species remain unidentified (Houtz et al. 2013). A study by Barzen-Hanson et al (2017) resulted in the discovery of 40 novel classes of PFAS and the detection of 17 classes of previously reported PFAS, adding over 240 individual PFAS to the previous list that can now be associated with AFFF. Little is known about the newly discovered PFAS regarding the subsurface remediation strategies, transport, and toxicity (Barzen-Hanson et al. 2017).

Limited Information and Methods to Assess Public Health Implications

Methods are available to evaluate the public health implications of exposure to PFOA, PFOS, PFHxS, and PFNA (all PFAS with ATSDR-derived provisional MRLs). People are potentially

exposed to a mixture of PFAS compounds in their drinking water. Methods used to assess exposure to other environmental mixtures have not been developed for PFAS or might be appropriate only for PFOA, PFOS, PFHxS, and PFNA. ATSDR added hazard quotients to get a hazard index which is often used to assess risk to multiple chemicals. However, this approach may not provide an appropriate solution for all PFAS. ATSDR has not formally reviewed the Minnesota values for PFBS and PFBA that are referenced in this document, therefore these PFAS are not included in the mixture analyses in this document.

Most of the available information on the health effects of laboratory animals exposed to PFAS is derived from oral exposure studies. Humans and experimental animals differ in how their bodies absorb and react to PFAS. That leaves questions about how relevant effects in animals are to humans.

There remains significant uncertainty about the lowest concentration at which toxic effects might occur in people exposed to PFAS for many years. Therefore, people exposed for many years could be at increased health risk.

The current CVs for PFOS, PFOA, PFHxS, and PFNA in drinking water were calculated by ATSDR using the best available scientific information. They allow us to assess the potential risk from drinking water exposures. ATSDR CVs and MRLs are based on the most current PFAS science; however, the overall scientific knowledge on PFAS is still evolving. The toxicity information for other PFAS compounds is limited. Most of the PFAS detected with no ATSDR MRLs were short-chain and non-sulfonated. Short-chain and non-sulfonated PFAS are generally thought to be less bioaccumulative than the longer chain and sulfonated PFAS (EPA 2018b). However, the health effects of many short-chain PFAS and new PFAS alternatives have not been fully researched.

Because of these limitations, ATSDR used a conservative approach to evaluate health risks for noncancerous exposures until better methods are developed. ATSDR evidence on exposures and other factors for the evaluations. For noncancerous health effects, we calculated hazard quotients for PFOS, PFOA, PFHxS, and PFNA, the most thoroughly investigated PFAS compounds. If the hazard quotient exceeded one, we considered a potential exposure to be of concern. In evaluating health risks, we also considered other source contributions, other PFAS compounds in the mixture, and past exposures. We reviewed the available literature for likely health consequences from these exposures.

Community Concerns

Community members continue to express concerns regarding their exposures to PFAS and other chemicals in this site area. The concerns listed here are not exhaustive. In addition to concerns about health effects for themselves and their family members, a number of concerns have been raised about whether it is safe to breastfeed babies, requests for ways to further reduce exposures through water filtration, and biomonitoring. Below is a summary of the current scientific information on these topics.

Breastfeeding

Community members have expressed concerns over the health implications of PFAS exposure to infants who breastfeed. Developmental effects are the most sensitive adverse health effect resulting

from exposure through the mother to the fetus during gestation or to the infant during lactation. Transfer of maternal stores of PFAS to breast milk appears to be a significant potential route of by which the mother eliminates PFAS from her body (Morgensen 2015).

Comparisons of serum concentrations of women who did or did not breastfeed their infants showed that breastfeeding significantly decreases maternal serum concentrations of PFAS. The estimated decrease in maternal PFAS serum levels was estimated to be 2–3% decrease per month of breastfeeding. Concentrations of PFAS in breast milk also decrease with breastfeeding duration. PFAS have been detected in human breast milk; the reported maximum concentration of PFOA and PFOS are 0.21-0.49 and 0.36-0.64 ng/mL, respectively. Maximum concentrations of other perfluoroalkyl compounds are <0.18 ng/mL (ATSDR 2018). The PFAS concentrations in breast milk are approximately 1% of the corresponding maternal serum level indicating PFAS elimination through lactation is correlated with serum concentrations but is compound-dependent (Karrman, A., et al 2007).

Even though it is possible for PFAS to migrate from the bloodstream of mothers into their breast milk, this migration may be limited due to the ability of many PFAS to bind strongly to the proteins in blood. In a review of PFAS exposure pathways, Fromme et al (2009) reported concentrations of PFOS in human breast milk ranging from non-detect to 0.47 µg/L and concentrations of PFOA in human breast milk ranging from non-detect to 0.61 µg/L. For comparison, reported maternal blood concentrations of PFOS ranged from 4.9 µg/L to 107 µg/L and maternal blood concentrations of PFOA ranged from <0.5 µg/L to 41.5 µg/L. The maternal blood concentrations of PFOS and PFOA were from one to two orders of magnitude higher than those observed in breast milk.

To evaluate the potential exposure risk to infants, Fromme et al. (2009) examined the blood PFAS concentrations in a group of German infants and compared them with the concentrations observed in their mothers' breast milk. They found a significant association between the levels of PFOS in breast milk and the levels of PFOS in the infants' blood. PFOA was measurable in only a few of the breast milk samples analyzed, which limited the authors' ability to analyze potential associations between PFOA concentrations in breast milk and infant serum. For PFOS, the authors found that the body burden for infants aged six months was similar to that of their mothers, while for PFOA, the body burden was higher. As most of the infants in their study were exclusively breast-fed, they suggest that the exposure from breast milk is a likely cause of the observed PFAS body burdens in these infants (Fromme et al 2009). Another study assessed the association between PFAS concentrations in mother-child pairs in a population exposed to PFOA via drinking water. The concentration in breast milk as a proportion of the concentration in maternal serum ranges from 3.4% - 11% for PFOA, 1%-2% for PFOS, 0.7% - 5% for PFNA and 2%-3% for PFHxS (Mondal 2012).

There is uncertainty regarding the potential health risks associated with the PFAS found in breast milk. A woman's decision to breastfeed is an individual choice, made after consideration of many different factors (many unrelated to PFAS exposure) and in consultation with her healthcare providers. In general, breastfeeding is still recommended despite the presence of chemicals in breast milk (CDC 2015). There are many clear health and nutritional benefits of breastfeeding. For example, breastfeeding protects babies from infections and illnesses that include diarrhea, ear infections and pneumonia; breast-fed babies are less likely to develop asthma; children breast-fed

for six months are less likely to become obese; and breastfeeding reduces the risk of sudden infant death syndrome (SIDS) (US HHS 2011).⁸

Water Filtration

Community members with PFAS detections in their public or private drinking water have expressed interest in options to further reduce the level of PFAS in their drinking water. In November 2016, National Sanitation Foundation (NSF) announced a test method and protocol to verify a water treatment device's ability to reduce PFOA and PFOS to below the current EPA HA (NSF 2016). While ATSDR does not endorse a particular water filter, as of August 2018, NSF has certified 71 drinking water treatment units from seven manufacturers for PFOA and PFOS. The types of filters certified by NSF for PFOS and PFOA removal include counter top ("manual" fill/pitcher type), faucet installed, and "plumbed in"/separate from the tap type (NSF 2018).

EPA researchers have been studying a variety of technologies for PFAS removal in drinking water. Certain technologies, such as activated carbon adsorption, ion exchange resins, and high-pressure membranes, have been found to remove PFAS from drinking water, especially PFOA and PFOS. These technologies can be used in drinking water treatment facilities or even in homes (at the point-of-entry, where water enters the home, or the point-of-use, such as in a kitchen tap). Granulated activated carbon (GAC) treatment, is commonly used to adsorb natural organic compounds, taste and odor compounds, and synthetic organic chemicals in drinking water treatment systems, and is the most studied treatment for PFAS removal. GAC works well on longer-chain PFAS like PFOA and PFOS, but shorter chain PFAS like PFBS do not adsorb as well (EPA 2018a).

Even though these filtration options may work, it is important to ensure the following steps are taken:

- 1) That a certified filtration method is used by the consumer.
- 2) That the system is maintained.
- 3) That the water is tested to ensure that PFAS levels are reduced.

A number of studies have been conducted to determine the efficiency of water filtration devices for PFAS. A study by the Minnesota Department of Health demonstrates that water filtration devices (point-of-use devices at a single tap, faucet, or outlet) can remove some PFAS from water. PFAS concentrations in Phase I of the study were 3 µg/L for PFOA and PFOS each and Phase II was 0.6 µg/L for PFOA and 0.9 µg/L for PFOS. This study evaluated devices in two categories: those using granulated active carbon and those using multiple methods of removal in combination. All eleven devices evaluated removed PFAS in the field test to below the quantifiable detection limits used in this study. The researchers concluded that these results suggest that when applied, installed, operated, and maintained according to the manufacturers' specifications, the eleven devices tested will effectively remove PFAS at the concentrations tested for up to 500 gallons before service and/or replacement is required (Olsen and Paulson 2008).

Anumol et al (2015) evaluated point-of-use devices for removal of trace organics (including concentrations of PFOS and PFOA between 0.140 µg/L and 1.3 µg/L) and found significant removal

⁸ For additional guidance and information on talking to your doctor, please visit: https://www.atsdr.cdc.gov/pfas/docs/Talking_to_Doctor.pdf.

of PFOA and PFOS. The results in this study varied by type of filter (refrigerator or pitcher point-of-use), and efficiency of removal declined based on the manufactured expected lifetime and other water quality factors and contaminants present. Based on these studies, there currently are three general types of filtration systems that can reduce PFAS levels in water, if properly maintained: granulated activated carbon – either in refrigerator, faucet, or pitcher filters and some filtration systems installed on your water line; reverse osmosis; or granulated activated carbon used with reverse osmosis.

It is important to note that the Olsen and Paulson (2008) and the Anumol (2015) papers were published prior to when the NSF certification for PFAS was available.

Biomonitoring

CDC/ATSDR understand and acknowledge that individuals may want to know the level of PFAS in their blood. Currently, there are no health-based screening levels for specific PFAS that clinicians can compare to the concentrations measured in blood samples. No current guidelines tell us what levels of PFAS in blood are “safe” or “unsafe.” As a result, interpreting PFAS concentrations in an individual is limited in its use.

Blood tests for PFAS are most useful when they are part of a scientific investigation or a health study. A scientific investigation can show the range of blood PFAS levels in community members and may provide information on how the levels vary among different populations. The data from these studies can also help community members who were not tested to estimate their likely blood PFAS level.

However, PFAS blood tests are commercially available. An individual’s blood concentration of PFAS can be compared to PFAS concentrations measured in the general US population as part of NHANES, or to PFAS levels identified through population studies in other PFAS-impacted communities.

Acknowledgements

HSWA, WTWSD, PADOH, EPA, and DOD provided significant data in support of this evaluation. PADOH provided cancer incidence information and the contributed greatly to our understanding of blood levels in this community via the PEATT biomonitoring effort. LT Christine Lloyd, formerly of the ATSDR Region 3 office, contributed substantially to this document, and her efforts are gratefully acknowledged. This document was extensively reviewed by ATSDR scientists and PFAS subject matter experts.

References

Alexander, B.H., et al. (2003). Mortality of Employees of a Perfluorooctanesulphonyl Fluoride Manufacturing Facility. *Occup Environ Med*, 2003. 60(10): p. 722-9.

Alexander, B.H. & Olsen, G.W. (2007). Bladder Cancer in Perfluorooctanesulfonyl fluoride manufacturing workers. *Ann. Epidemiology*, 2007. 17(6): 471-8/

Anumol, T, Clarke, B., Merel, S. Snyder, S. (2015). Point-of-Use Devices for Attenuation of Trace Organic Compounds in Water. *Journal of American Water Works Association*, 2015. 107(9).

ATSDR (2002). Public Health Assessment: Willow Grove Naval Air Station/Air Reserve Station, Montgomery County, Pennsylvania. Available at:

<http://www.atsdr.cdc.gov/HAC/pha/WillowGrove051602-PA/WillowGrove051602-PA.pdf>

Accessed on December 30, 2019.

ATSDR (2005). Public Health Assessment Guidance Manual. Available at:

http://www.atsdr.cdc.gov/hac/PHAManual/PDFs/PHAGM_final_12-27-05.pdf Accessed on

December 30, 2019.

ATSDR (2016a). Exposure Dose Guidance for Water Ingestion, Version 2. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, October 26.

ATSDR (2016b). Letter Health Consultation: Perfluorochemicals (PFCs) Contamination in Public and Private Drinking Water Sources near the Former Naval Air Warfare Centers, Warminster, Bucks County, Pennsylvania. January 20, 2016.

https://www.atsdr.cdc.gov/HAC/pha/NavalAirWarfareCenter/Naval_Air_Warfare_Center_LHC_01-20-2016_508.pdf Accessed December 30, 2019.

ATSDR (2017). Feasibility Assessment for Epidemiological Studies at Pease International Tradeport, Portsmouth, New Hampshire. November 2017.

https://www.atsdr.cdc.gov/sites/pease/documents/Pease_Feasibility_Assessment_November-2017_508.pdf Accessed on December 30, 2019.

ATSDR (2018). Toxicological Profile for Perfluoroalkyls. Draft for Public Comment. June 2018.

Available at: <http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=1117&tid=237> Accessed on December 30, 2019.

Barzen-Hanson, K.A; Roberts, S.C.; Choyke, S.; Oetjen, K.; McAlees, A; Riddell, N; McCrindle, R; Ferguson, P.L.; Higgins, C.P.; and, Field, J.A. Discovery of 40 Classes of Per- and Polyfluoroalkyl Substances in Historical Aqueous Film-Forming Foams (AFFFs) and AFFF-Impacted Groundwater. *Environ. Sci. Technol.* 2017, 51(4), 2047–2057.

Benbrahim-Tallaa L., B. Lauby-Secretan, D. Loomis, KZ Guyton, Y. Grosse, FE Ghissassi, V Bouvard, Guha, H Mattack, and K Straif (2014). Carcinogenicity of Perfluorooctanoic Acid,

Tetrafluoroethylene, Dichloromethane, 1, 2-Dichloropropane, and 1, 3-Propane Sultone. *Lancet*, 2014. Vol. 15 pp 924-925.

Bonefeld-Jorgensen et al. (2011). Perfluorinated Compounds are Related to Breast Cancer Risk in Greenlandic Inuit: A Case Control Study. *Environ Health*, 2011 Oct 6;10:88.

Butenhoff, J.L., et al. (2012). Chronic Dietary Toxicity and Carcinogenicity Study with Potassium Perfluorooctanesulfonate in Sprague Dawley rats. *Toxicology*, 2012. 293(1-3): p. 1-15.

Butenhoff JL, Chang S, Ehresman DJ, et al. (2009). Evaluation of Potential Reproductive and Developmental Toxicity of Potassium Perfluorohexanesulfonate in Sprague Dawley Rats. *Reprod Toxicol*, 2009. 27:331-341.

C8 Science Panel (2015). C8 Probable Link Reports. Available at: http://www.c8sciencepanel.org/prob_link.html Accessed on December 30, 2019.

Calafat, A.M., et al. (2007). Polyfluoroalkyl Chemicals in the U.S. population: Data from the National Health and Nutrition Examination Survey (NHANES) 2001-2004 and Comparisons with NHANES 1999-2000. *Environ Health Perspect*, 2007. 115(5): p. 1596-602.

Carr CK, Watkins AM, Wolf CJ, et al. 2013. Testing for departures from additivity in mixtures of perfluoroalkyl acids (PFAAs). *Toxicology* 306:169-175. doi:10.1016/j.tox.2013.02.016.

CDC (2015). Exposure to Environmental Toxins. Available at: http://www.cdc.gov/breastfeeding/disease/environmental_toxins.htm. Accessed on December 30, 2019.

CDC (2018). National Health and Nutrition Examination Survey. <https://www.cdc.gov/nchs/nhanes/prev/nhanes.htm> Accessed on December 30, 2019.

Darrow, L.A., C.R. Stein, and W. Steenland (2013). Serum Perfluorooctanoic Acid and Perfluorooctane Sulfonate Concentrations in Relation to Birth Outcomes in the Mid-Ohio Valley, 2005-2010. *Environ Health Perspect*, 2013. 121(10): p. 1207-13.

Das KP, Grey BE, Rosen MR, et al (2015). Developmental Toxicity of Perfluorononanoic Acid in Mice. *Reprod Toxicol*. 2015 Jan; 51:133-44.

Dong, G.H., et al. (2013)., Serum polyfluoroalkyl concentrations, asthma outcomes, and immunological markers in a case-control study of Taiwanese children. *Environ Health Perspect*, 2013. 121(4): p. 507-13, 513e1-8.

EFSA 2008. European Food Safety Authority Annual Report 2008. Available at: <https://www.efsa.europa.eu/en/corporate/pub/ar08> Accessed on December 30, 2019.

EPA, USNAVY & PADEP (2015). Open House Meeting: Actions to Address Impacts to Drinking Water from Former Naval Air Station Joint Reserve Base Willow Grove and Former Naval Air Warfare Center Warminster. February 24-25, 2015. Available at: <https://www.horshamwater->

sewer.com/sites/default/files/public_information_session_open_house_handout_february_24th_25th_2015.pdf Accessed on December 30, 2019.

EPA (2011). U.S. Environmental Protection Agency, Exposure Factors Handbook 2011 Edition (Final). USEPA, 2011. Available at: <https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=236252> Accessed on December 30, 2019.

EPA (2015). Methods and Contaminants for the Unregulated Contaminant Monitoring Rule 3 (UCMR 3). Available at: <https://www.epa.gov/dwucmr/third-unregulated-contaminant-monitoring-rule> Accessed on December 30, 2019.

EPA (2016a). EPA Superfund Program: Willow Grove Naval Air and Air Reserve Stations, Horsham, PA. Available at: <https://cumulis.epa.gov/supercpad/cursites/csinfo.cfm?id=0303820> Accessed on December 30, 2019.

EPA (2016b). Drinking Water Health Advisory for Perfluorooctane Sulfonate (PFOS). EPA Office of Water. May 2016. https://www.epa.gov/sites/production/files/2016-05/documents/pfos_health_advisory_final-plain.pdf Accessed on December 30, 2019.

EPA (2016c). Drinking Water Advisory for Perfluorooctanoic Acid (PFOA). EPA Office of Water. May 2016. Available at: https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_health_advisory_final_508.pdf Accessed on September 22, 2017.

EPA (2016d). Drinking Water Health Advisories for PFOA and PFOS. Available at: <https://www.epa.gov/ground-water-and-drinking-water/drinking-water-health-advisories-pfoa-and-pfos> Accessed on December 30, 2019.

EPA (2016e). Factsheet PFOA and PFOS Drinking Water Health Advisories. EPA Office of Water. November 2016. Available at: https://www.epa.gov/sites/production/files/2016-06/documents/drinkingwaterhealthadvisories_pfoa_pfos_updated_5.31.16.pdf Accessed on December 30, 2019.

EPA (2018a). Reducing PFAS in Drinking Water with Treatment Technologies. August 23, 2018. <https://www.epa.gov/sciencematters/reducing-pfas-drinking-water-treatment-technologies> Accessed on December 30, 2019.

EPA (2018b) Risk Management for Per- and Polyfluoroalkyl Substances under TSCA. [updated 2018 July 20]. Available from: <https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/risk-management-and-polyfluoroalkyl-substances-pfass>. Accessed on December 30, 2019.

Falandysz et al (2006). Is Fish a Major Source of Fluorinated Surfactants and Repellents in Humans Living on the Baltic Coast? *Environ Sci Technol.*, 2006 Feb 1;40(3):748-51.

Fei, C., et al. (2008). Fetal Growth Indicators and Perfluorinated Chemicals: A Study in the Danish National Birth Cohort. *Am J Epidemiol*, 2008. 168(1): p. 66-72.

Foster Wheeler (2000). Draft Remedial Action Completion Report for F14 Crash Site, Site Remediation, Naval Air Station Joint Reserve Base, Willow Grove, Pennsylvania. September 2000.

Fromme, H., et al., (2009). Perfluorinated Compounds—Exposure Assessment for the General Population in Western countries. *Int J Hyg Environ Health*, 2009. 212(3): p. 239-70.

Gilliland, F.D. and J.S. Mandel (1993). Mortality Among Employees of a Perfluorooctanoic Acid Production Plant. *J Occup Med*, 1993. 35(9): p. 950-4.

Gleason, J.A., G.B. Post, and J.A. Fagliano (2015). Associations of Perfluorinated Chemical Serum Concentrations and Biomarkers of Liver Function and Uric Acid in the US population (NHANES), 2007-2010. *Environ Res*, 2015. 136: p. 8-14.

Grandjean, P, Andersen EW, Budtz-Jørgensen E, Flemming N, Mølbak K, Weihe P, Heilmann C. (2012). Serum Vaccine Antibody Concentrations in Children Exposed to Perfluorinated Compounds. *JAMA*, 2012. 307(4):391-397.

Heritage Conservancy (2007). Little Neshaminy Creek River Conservation Plan. September 2007. Available at http://www.heritageconservancy.org/wp-content/uploads/2012/03/085_l-nesh-plan.pdf. Accessed on December 30, 2019.

Hoberman AM, York RG (2003). Oral (gavage) combined repeated dose toxicity study of T-7706 with the reproduction/developmental toxicity screening test. *Argus Research.*, 2003.

Holzer et al. (2008). Assessment and Management of the First German case of a Contamination with Perfluorinated Compounds (PFC) in the Region Sauerland, North Rhine-Westphalia. *J Toxicol Environ Health.*, 2008;71(11-12):725-33

Horsham Air Guard (2017). Email communication from J. Siciliano/Horsham Air Guard to L. Werner /ATSDR Region 3, May 11, 2017.

Horsham Water and Sewer (2014). 2014 Consumer Confidence Report. https://www.horshamwater-sewer.com/sites/default/files/ccr_reports/2014%20CCR.pdf Accessed on December 30, 2019.

Horsham Land Redevelopment Authority (2016). NASJRB Willow Grove History. Available at: <http://www.hlra.org/nas-jrb-willow-grove/history.aspx> Accessed on December 30, 2019.

Horsham Township (2016). Horsham Township Point Use Water Testing Program. Available at: <http://www.horsham.org/docview.aspx?doctype=fd&docid=24469> Accessed on December 30, 2019.

Horsham Water and Sewer (2017). HWSA PFOS/PFOA Sampling Results Active Sources as of 2/17/17. Available at: https://www.horshamwater-sewer.com/sites/default/files/hwsa_pfes_pfoa_sampling_results_for_inactive_sources.pdf Accessed on December 30, 2019.

Horsham Township (2018). Horsham Water and Sewer Authority Updates.

<https://www.horsham.org/docview.aspx?docid=28036> Accessed on December 30, 2019.

Houtz, E. F.; Higgins, C. P.; Field, J. A.; Sedlak, D. L. Persistence of Perfluoroalkyl Acid Precursors in AFFF-Impacted Groundwater and Soil. *Environ. Sci. Technol.* 2013, 47 (15), 8187–8195.

IARC (2017). International Agency for Research on Cancer, Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 110. Some Chemicals Used as Solvents in Polymer Manufacture. Available at: <https://monographs.iarc.fr/ENG/Monographs/vol110/mono110-07.pdf>. Accessed on December 30, 2019.

Iwai H, Hoberman AM. 2014. Oral (gavage) combined developmental and perinatal/postnatal reproduction toxicity study of ammonium salt of perfluorinated hexanoic acid in mice. *Int J Toxicol.*, 2014: 33(3):219-237.

Kannan et al. (2005). Perfluorinated Compounds in Aquatic Organisms at Various Trophic Levels in a Great Lakes Food Chain. *Arch Environ Contam Toxicol.* 2005 May; 48(4):559-66

Karrman, A, et al (2007). Exposure of Perfluorinated Chemicals through Lactation: Levels of Matched Human Milk and Serum and a Temporal Trend, 1996–2004, in Sweden. *Environ Health Perspect*, 2007 Feb; 115 (2): 226-230.

Kato, K., et al. (2011). Trends in Exposure to Polyfluoroalkyl Chemicals in the U.S. Population: 1999 -2008. *Environ Sci Technol*, 2011. 45(19): p. 8037-45.

Klaunig JE, Shinohara M, Iwai H, et al. 2015. Evaluation of the chronic toxicity and carcinogenicity of perfluorohexanoic acid (PFHxA) in Sprague-Dawley rats. *Toxicol Pathol*, 2015: 43(2):209-220.

Lau, C., et al. (2006). Effects of Perfluorooctanoic Acid Exposure During Pregnancy in the Mouse. *Toxicol Sci*, 2006. 90(2):6. 570-8.

Leonard et al. (2008). Retrospective Cohort Mortality Study of Workers in a Polymer Production Plant including a Reference Population of Regional Workers. *Ann Epidemiol.* 2008 Jan;18(1):15-22.

Luebker, et al. (2005). Two-generation reproduction and cross-foster studies of perfluorooctanesulfonate (PFOS) in rats. *Toxicology*, 2005: 215: 129–48.

Michigan (2014). Technical Support Document for Assessment of Perfluorinated Chemicals and Selection of Perfluorooctane Sulfonate (PFOS) Reference Dose as the basis for Michigan Fish Consumption Screening Values. September 30, 2014. Available at: https://www.michigan.gov/documents/mdch/MDCH_PFOS_Health_Consultation_2014_468766_7.pdf Accessed on December 30, 2019.

Michigan (2018). PFAS in Michigan Fish. https://www.michigan.gov/som/0,4669,7-192-45414_45929_83470_83473-463860--,00.html Accessed on December 30, 2019.

Minnesota Department of Health (2017). Toxicological Summary for Perfluorobutane sulfonate. <http://www.health.state.mn.us/divs/eh/risk/guidance/gw/pfbsummary.pdf> Accessed on December 30, 2019.

Minnesota Department of Health (2018). State Issues Updated Fish Consumption Guidelines for 2018. <https://www.health.state.mn.us/news/pressrel/2018/fish050318.html> Accessed on December 30, 2019.

Minnesota Department of Health (2019). Minnesota Department of Health, Human health-based water guidance table. Available from: <https://www.health.state.mn.us/communities/environment/risk/guidance/gw/table.html> Accessed on December 30, 2019.

Mondal, et al. (2012). Relationships of Perfluorooctanoate and Perfluorooctane Sulfonate Serum Concentrations Between Mother-Child Pairs in a Population with Perfluorooctanoate Exposure from Drinking Water. *Environ Health Perspect*, 2012 May;120(5):752-7.

Naval Research Laboratory (NRL) (2015). Aqueous Film-Forming Foam. Available at: <http://www.nrl.navy.mil/accomplishments/materials/aqueous-film-foam/> Accessed on December 30, 2019.

Navy (2018). Remedial Investigation Data Report, Per and Polyfluorinated Substances Investigation Activities: NASJRB Willow Grove, PA. December

Nelson, JW, Hatch, EE, Webster, TF (2010). Exposure to Polyfluoroalkyl Chemicals and Cholesterol, Body Weight, and Insulin Resistance in the General U.S. population. *Environ Health Perspect* 118 (2): 197-202.

NSF (2016). New Protocol Certifies Products Proven Effective for Reducing PFOA and PFOS to Below EPA Health Advisory Levels. <http://www.nsf.org/newsroom/nsf-international-certifies-first-water-filters-pfoa> Accessed on December 30, 2019.

NSF (2018). NSF Product and Service Listing: Drinking Water Treatment Units for PFOS and PFOA. <http://info.nsf.org/Certified/DWTU/Listings.asp?ProductFunction=P473%7CPFOA+Reduction&ProductFunction=P473%7CPFOS+Reduction&ProductType=&submit2=Search> Accessed on December 30, 2019.

NWWA (2019). PFOA and PFOS Sampling at Forest Park Water Treatment Plant. Available at <http://www.nwwater.com/images/PDFs/4-19pfcs.pdf>. Accessed on December 30, 2019.

Olsen, G.W., J.L. Butenhoff, and L.R. Zobel (2009). Perfluoroalkyl Chemicals and Human Fetal Development: An Epidemiologic Review with Clinical and Toxicological Perspectives. *Reprod Toxicol*, 2009. 27(3-4): p. 212-30.

Olsen, PC and Paulson DJ, Water Science & Marketing, LLC (2008). Performance Evaluation – Removal of Perfluorochemicals (PFCs) with Point-of-Use (POU) Water Treatment Devices - Final Report. Prepared for State of Minnesota, Minnesota Department of Health. May 2008. Available at:

<http://www.health.state.mn.us/divs/eh/wells/waterquality/poudevicefinal.pdf> Accessed on December 30, 2019.

Olsen, G.W., et al. (2007). Half-life of serum Elimination of Perfluorooctanesulfonate, Perfluorohexanesulfonate, and Perfluorooctanoate in Retired Fluorochemical Production Workers. *Environ Health Perspect*, 2007. 115(9): p. 1298-305.

PADOH (2018). Addendum 2 to the August 2016 Cancer Data Review: Selected Zip Codes of Warminster, Warrington, and Horsham, Pennsylvania. Division of Environmental Health Epidemiology, May 2018. <https://www.health.pa.gov/topics/envirohealth/Pages/PFAS.aspx> Accessed on December 30, 2019.

PADOH (2019). PFAS Exposure Assessment Technical Toolkit (PEATT) Pilot Project. April 2019. <https://www.health.pa.gov/topics/Documents/Environmental%20Health/PEATT%20Pilot%20Project%20Final%20Report%20April%2029%202019.pdf> Accessed on December 30, 2019.

Pennsylvania Fish & Boat Commission (2014). Biologist Report- Darby and Neshaminy Creeks, Delaware and Bucks Counties, 2014 Tidal Largemouth Bass Survey. Available at: http://pfbc.pa.gov/images/reports/2015bio/6x08_28darbynesh.pdf Accessed on December 30, 2019.

Perkins, R.G., et al. (2004)., 13-week Dietary Toxicity Study of Ammonium Perfluorooctanoate (APFO) in Male Rats. *Drug Chem Toxicol*, 2004. 27(4) p. 561-78.

Prevedouros K, Cousins I T, Buck RC, et al. (2006). Sources, Fate and Transport of Perfluorocarboxylates. *Environ Sci Technol*, 2006. 40(1):32-44.

Rogers, J.M. et al (2014). Elevated blood pressure in offspring of rats exposed to diverse chemicals during pregnancy. *Toxicol Sci.*, 2014; 142(2):436- 46.

Starling AP, Engel SM, Whitworth RW, et al. (2014). Perfluoroalkyl substances and lipid concentrations in plasma during pregnancy among women in the Norwegian Mother and Child Cohort Study. *Environ Int* 62: 104–12.

Stein, et. al (2016). Perfluoroalkyl substance serum concentrations and immune response to FluMist vaccination among healthy adults. *Environ Res*, 2016. 149 (171-178).

Tetra Tech (2013). Five Year Review for the Former NAS JAB Willow Grove Horsham, PA. Prepared for Naval Facilities Engineering Command, BRAC Program Management Office East. September 2013. Available at: https://www.navfac.navy.mil/niris/MID_ATLANTIC/WILLOW_GROVE_NAS/N00158_000450.pdf Accessed on December 30, 2019.

U.S. Department of Health and Human Services (US HHS) (2011). The Surgeon General's Call to Action to Support Breastfeeding. Washington, DC: U.S. Department of Health and Human

Services, Office of the Surgeon General. Available at:
<https://www.cdc.gov/breastfeeding/resources/calltoaction.htm> Accessed on December 30, 2019.

Verner, M.A., et al. (2015). Associations of Perfluoroalkyl Substances (PFASs) with Lower birth Weight: An Evaluation of Potential Confounding by Glomerular Filtration Rate Using a Physiologically Based Pharmacokinetic Model (PBPK). *Environ Health Perspect*, 2015.

Viberg H, Lee I, Eriksson P. (2013). Adult dose-dependent behavioral and cognitive disturbances after a single neonatal PFHxS dose. *Toxicology*, 2013. 304:185–191.

Warrington Township (2019a). Water Contamination Information. Available at:
<http://www.warringtontownship.org/departments/water-sewer/water-contamination-info/> Accessed on December 30, 2019.

Warrington Township (2019b). Water and Sewer. Available at:
<http://www.warringtontownship.org/departments/water-sewer/>. Accessed on December 30, 2019.

WTWSD (2014). Warrington Township 2014 Consumer Confidence Report.
<http://www.warringtontownship.org/download/Water%20and%20Sewer%20department/2014CCR.pdf> Accessed on December 30, 2019.

Wolf, C.J., et al. (2010). Developmental effects of perfluorooctanoic acid in the mouse are dependent on peroxisome proliferator-activated receptor- α . *PPAR Res*. 2010

Wolf CJ, Rider CV, Lau C, et al. (2014). Evaluating the additivity of perfluoroalkyl acids in binary combinations on peroxisome proliferator-activated receptor- α activation. *Toxicology* 316:43-54. 10.1016/j.tox.2013.12.002.

Wu, X.M., et al. (2015). Serum Concentrations of Perfluorinated Compounds (PFC) Among Selected Populations of Children and Adults in California. *Environ Res*, 2015. 136: p. 264-73.

Zeng XW, Qian Z, Emo B. (2015). The association between perfluoroalkyl chemicals and serum lipid levels in children. *Sci Total Environ*, 2015.