Study of the Occurrence of Per- and Polyfluoroalkyl Substances (PFAS) in the Commonwealth's Public Drinking Water

To

Governor Ralph S. Northam,

And

The Chairmen/Chairwomen of the
House Committees on:
Agriculture, Chesapeake and Natural Resources and
Health, Welfare and Institutions,

and

Senate Committees on:
Agriculture, Conservation and Natural Resources and
Education and Health

Submitted By

Virginia PFAS Workgroup

Virginia Department of Health

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Staff from the Virginia Department of Health (VDH) Office of Drinking Water (ODW) prepared this report with input and feedback from the PFAS Workgroup and Subgroups, which include volunteer members representing the Virginia Department of Environmental Quality (DEQ), waterworks, waterworks advocacy groups, environmental organizations, the chemical industry, and consumers of public drinking water. Funding to study the occurrence of PFAS in public drinking water in Virginia, as required by 2020 Acts of Assembly Chapter 611, came from the U.S. Environmental Protection Agency (EPA), through a fiscal year 2021 grant to VDH to study emerging contaminants. The General Assembly appropriated $60,000 in fiscal year 2022 for VDH to do additional monitoring and testing. VDH staff are grateful for EPA's financial support and the engagement, insight, and contributions made by members of the PFAS Workgroup, Subgroups, and others noted below to undertake and complete this study.

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Once published, this report will be available in alternative formats upon request, and online at www.vdh.virginia.gov/drinking-water/pfas

Table of Contents

FIGURES

TABLES

APPENDICES

GLOSSARY AND ACRONYMS

"AFFF" means aqueous film-forming foam. AFFF is a fire suppressant used to extinguish flammable liquid fires such as fuel fires. Perfluorooctane sulfonic acid (PFOS) and other PFAS used in AFFF may be present as a result of the manufacturing process or as breakdown products.

"Community waterworks" (CWS) means a waterworks that serves at least 15 service connections used by year-round residents or regularly serves at least 25 year-round residents. Examples include municipal water systems, authorities, and residential subdivisions with their own water supplies.

"DEQ" means the Virginia Department of Environmental Quality.

"Entry point" means the place where water from the source after application of any treatment is delivered to the distribution system.

"EPA" means the U.S. Environmental Protection Agency.

"Finished water" means water that is introduced into the distribution system of a waterworks and is intended for distribution and consumption without further treatment, except as treatment is necessary to maintain water quality in the distribution system (e.g., booster disinfection).

"GAC" means granular activated carbon, a water treatment technology that can be used for PFAS removal. Powdered activated carbon (PAC) is a variant of GAC.

"GenX" refers to HFPO-DA or hexafluoropropylene oxide-dimer acid.

"HA" means a health advisory. "Under the Safe Drinking Water Act, EPA may publish HAs for contaminants that are not subject to any national primary drinking water regulation. SDWA section 1412(b)(1)(F) [42 U.S.C. § 300g-1(b)(1)(F)]. EPA develops HAs to provide information on … exposure [and] health effects … for drinking water contaminants. HAs describe concentrations of drinking water contaminants at which adverse health effects are not anticipated to occur over specific exposure durations (e.g., one-day, ten-days, and a lifetime). HAs serve as informal technical guidance to assist federal, state and local officials, as well as managers of public or community water systems in protecting public health. They are not regulations and should not be construed as legally enforceable federal standards. HAs may change as new information becomes available." https://www.regulations.gov/document/EPA-HQ-OW-2014-0138-0037

"HFPO-DA" means hexafluoropropylene oxide-dimer acid, a replacement chemical for PFOA that is associated with GenX.

"IX" means ion exchange treatment, a water treatment technology which is capable of removing PFAS from drinking water.

"LHA" means lifetime health advisory. The LHA is the concentration of a chemical in drinking water that is not expected to cause any adverse non carcinogenic effects for a lifetime of exposure, incorporating a drinking water relative source contribution factor of contaminant-specific data or a default of 20% of total exposure from all sources. The LHA is based on exposure of a 70-kg adult consuming 2 liters of water per day. EPA has established a LHA of 70 ppt for PFOA and PFOS individually or combined.

"MDL" means method detection limit. The MDL is defined as the minimum measured concentration of a substance that can be reported with 99% confidence that the measured concentration is distinguishable from method blank results. 40 C.F.R. Part 136, Appendix B.

"MCL" means maximum contaminant level. The MCL is the maximum permissible level of a contaminant in potable water that is delivered to any consumer of a waterworks. A MCL is an enforceable standard.

"MCLG" means maximum contaminant level goal. The MCLG is the maximum level of a contaminant in drinking water at which no known or anticipated adverse effect on the health of persons would occur, allowing an adequate margin of safety. 42 U.S. Code § 300 g-1(b)(4)(A). A MCLG is not an enforceable standard.

"ng/L" means nanograms per liter. 1 ng/L is equivalent to 1 part per trillion (ppt).

"Nontransient noncommunity waterworks" (NTNC) means a waterworks that is not a CWS, that regularly serves at least 25 of the same people at least six months of the year. Examples include schools, factories, and hospitals that have their own water supplies.

"ODW" means the Office of Drinking Water, a functional unit within the Virginia Department of Health with responsibility for regulating waterworks in Virginia.

"One-day health advisory" means the concentration of a chemical in drinking water that is not expected to cause any adverse non carcinogenic effects for up to one day of exposure. The one-day health advisory is intended to protect a 10-kg child consuming 1 liter of water per day.

"PFAS" means per- and polyfluoroalkyl substances and refers to a broad class of chemicals that includes PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, HFPO-DA, and thousands of others.

"PFBA" means perfluorobutyrate.

"PFBS" means perfluorobutane sulfonic acid, a replacement chemical for PFOS.

"PFHpA" means perfluoroheptanoic acid.

"PFHxA" means perfluorohexanoic acid.

"PFHxS" means perfluorohexane sulfonate.

"PFNA" means perfluorononanoic acid.

"PFOA" means perfluorooctanoic acid.

"PFOS" means perfluorooctane sulfonic acid.

"POTW" means publicly owned treatment works. This includes any devices and systems used in the storage, treatment, recycling and reclamation of municipal sewage or industrial wastes of a liquid nature.

"ppb" means parts per billion. 1 ppb is equivalent to 1 microgram per liter (µg/L).

"ppm" means parts per million. 1 ppm is equivalent to 1 milligram per liter (mg/L).

“ppt” means parts per trillion. 1 ppt is equivalent to 1 nanogram per liter (ng/L).

“PQL” means practical quantitation level. The PQL is the lowest level that can be reliably measured within specified limits of precision and accuracy during routine laboratory conditions. In general, the MDL < PQL.

“SDWA” means the Safe Drinking Water Act, codified at 42 U.S. Code §§ 300f through 300j-26. (https://www.epa.gov/sdwa).

“SIC” means Standard Industrial Classification code. A SIC code describes the primary business activity of a company.

“Ten-day health advisory” means the concentration of a chemical in drinking water that is not expected to cause any adverse non carcinogenic effects for up to ten days of exposure. The ten day health advisory is also intended to protect a 10-kg child consuming 1 liter of water per day.

“Transient noncommunity waterworks” (TNC) means a waterworks that is not a CWS, but serves transient customers in non-residential settings, such as campgrounds, motels, and restaurants that have their own water supplies. A TNC serves at least 25 persons daily for at least 60 days out of the year.

“µg/L” means micrograms per liter. 1 µg/L is equivalent to 1 part per billion (ppb).

“UCMR ” means EPA’s Unregulated Contaminant Monitoring Rule. EPA’s third rule (UCMR3, monitoring years 2013-2015) required waterworks to monitor for 6 PFAS: PFOS, PFOA, PFNA, PFHxS, PFHpA, and PFBS. The proposed fifth rule (UCMR5, monitoring years 2023-2025) includes 29 PFAS to “provide new data that is critically needed to improve EPA’s understanding of the frequency they [they] are found in the nation’s drinking water systems and at what levels.” https://www.epa.gov/dwucmr/fifth-unregulated-contaminant-monitoring-rule

“VAPA” means the Virginia Administrative Process Act, Chapter 40 (§ 2.2-4000 et seq.) of Title 2.2 of the Code of Virginia. The VAPA is the basic law conferring authority on agencies either to make regulations or case decisions as well as standardizing court review thereof.

“VDH” means the Virginia Department of Health.

“VPDES” means Virginia Pollutant Discharge Elimination System.

“Waterworks” means a system that serves piped water for human consumption to at least 15 service connections or 25 or more individuals for at least 60 days out of the year. EPA and some other states refer to a waterworks as a “public water system.”

1. EXECUTIVE SUMMARY

2020 Acts of Assembly Chapter 611 (HB586) required the Commissioner of Health to convene a work group to study the occurrence of perfluorooctanoic acid (PFOA), perfluorooctane sulfonate (PFOS), perfluorobutyrate (PFBA), perfluoroheptanoic acid (PFHpA), perfluorohexane sulfonate (PFHxS), perfluorononanoic acid (PFNA), and other perfluoroalkyl and polyfluoroalkyl substances (PFAS), as deemed necessary, in the Commonwealth's public drinking water. The work group may develop recommendations for specific maximum contaminant levels (MCLs) for PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS, as deemed necessary, for inclusion in regulations of the Board of Health applicable to waterworks.

The work group shall report its findings and recommendations to the Governor and the Chairmen/Chairwomen of the House Committees on Agriculture, Chesapeake and Natural Resources and Health, Welfare and Institutions and the Senate Committees on Agriculture, Conservation and Natural Resources and Education and Health by December 1, 2021.

This report contains the Virginia PFAS Workgroup's (PFAS Workgroup) findings and recommendations related to the occurrence of PFAS in the Commonwealth's public drinking water.

The Virginia Department of Health's (VDH) Office of Drinking Water (ODW) provided administrative support and technical guidance to the PFAS Workgroup, which planned, designed, and conducted the PFAS Sample Study (Sample Study) required by HB586 (*see* Appendix 1 for a list of PFAS Workgroup members). Information about PFAS contamination in drinking water in Virginia, which comes from the Sample Study, will inform the development and implementation of MCLs under Code of Virginia § 32.1-169 B. (effective January 1, 2022) and the Virginia Administrative Process Act, Code of Virginia §§ 2.2-4000 et seq.

PFAS Sample Study

HB586 limited the Sample Study to no more than 50 waterworks or major sources of supply. With this limitation, the Department of Planning and Budget (DPB) concluded VDH could absorb the costs to form the PFAS Workgroup, perform a literature survey, discuss results and data needed to drive regulatory decisions, and perform environmental sampling.[1] The Sample Study focused on the largest waterworks in Virginia and waterworks and major sources of supply near potential sources of PFAS contamination, within the legislative and financial limitations. VDH collaborated with the Virginia Department of Environmental Quality (DEQ) to identify potential sources of PFAS contamination and select waterworks for the Sample Study, which was voluntary for waterworks. The PFAS Workgroup reviewed the methodology and selection process and offered guidance on improving the Sample Study. A few waterworks that VDH

[1] *See* Department of Planning and Budget 2020 Fiscal Impact Statement for HB586ER, item #8 at: https://lis.virginia.gov/cgi-bin/legp604.exe?201+oth+HB586FER122+PDF.

identified for inclusion in the Sample Study declined to participate, citing ongoing construction or other maintenance projects as reasons to not participate.

Forty-five (45) waterworks participated in the Sample Study. They collected a total of 63 water samples from one or more locations because, in certain cases, there were multiple water sources or entry points. Results from the Sample Study found PFAS in quantities above the practical quantitation level (PQL) at 15 of 63 sample locations. Samples from 48 sample locations did not contain any PFAS or, if PFAS were present, they were below the PQL. In most cases, the PQL was 3.5 parts per trillion (ppt).

Findings

The results indicate that PFAS are present above the PQL (~3.5ppt) in drinking water produced from the Potomac River and Occoquan Reservoir, two major sources of water for waterworks in Northern Virginia. The amount and specific types of PFAS that may be in both sources are unknown because the Sample Study only tested finished water. Ten (10) samples from waterworks in the Northern Virginia region had at least one (1) PFAS present in a quantity above the PQL, but none were above the U.S. Environmental Protection Agency's (EPA) lifetime health advisory level of 70 ppt for PFOA and PFOS (individually or combined) and none exceeded any maximum contaminant level (MCL) established by other states.

The highest detected concentration of a compound was 57 ppt of HFPO-DA (hexafluoropropylene oxide-dimer acid) at Western Virginia Water Authority's Spring Hollow water treatment plant. HFPO-DA is commonly known as GenX, a type of PFAS used in place of PFOA (perfluorooctanoic acid). This was one of only two GenX detections in all of the samples tested in the Sample Study (the other was 4.0 ppt). No other PFAS were detected above the PQL at the two locations with GenX detections.

All other PFAS detections were 14 ppt or less. Information about PFAS contamination of drinking water in Virginia, which came from the Sample Study conducted pursuant to HB586, will help to inform the development and implementation of MCLs required by Code of Virginia § 32.1-169 B. However, with more than 1,050 community waterworks in Virginia, the majority of which are "small" (i.e., serving fewer than 3,300 consumers), the extent and level of PFAS contamination in drinking water from waterworks is still largely unknown.

ODW will conduct additional PFAS sampling in 2022 using $60,000 that the 2021 General Assembly appropriated and funding from EPA in the 2022 Public Water System Supervision Grant to study emerging contaminants.

Recommendations

1) The PFAS Workgroup recommends VDH and other agencies collect additional PFAS occurrence data in Virginia drinking water and major sources of supply. *See* page 43 for more detail.

2) The study design for the next round of sampling should consider factors such as: temporal data (i.e., collect a series of samples at one location to monitor seasonal variations in water quality, possibly due to variations in industrial cycles, river flows, temperatures, traffic patterns, agricultural runoffs, etc.); a focus on community waterworks; using a hybrid approach (as opposed to random sampling); prioritizing surface water sources; restricting samples to finished water (as opposed to raw or untreated water); excluding consecutive waterworks; and resampling waterworks that had PFAS > PQL during the Sample Study. *See* page 44.

3) The amount of funding and time for VDH and other state agencies such as DEQ to study the occurrence of PFAS in drinking water, drinking water sources, and potential sources of contamination will dictate the scope of additional sampling. The PFAS Workgroup recommends that the General Assembly consider funding additional resources at VDH and DEQ for enhanced sampling and more robust sample studies of drinking water, drinking water sources, and potential sources of contamination. *See* page 45.

4) When VDH and the Board of Health initiate the rulemaking process to establish MCLs for PFOA and PFOS, the Commonwealth needs to provide resources (time, money, and staff) to the agency so the process can be effective. To comply with the Administrative Process Act, an MCL should be based on toxicology and take into consideration such things as treatment costs, impacts from moving PFAS from one media to another, incremental costs, and downstream effects. The rulemaking also needs to consider impacts on small waterworks, including treatment options, costs, and how to pay when treatment is or would be required. *See* page 45.

5) VDH should include an analysis of environmental justice impacts that may flow from the promulgation of an MCL for any PFAS. The Commonwealth/VDH should also carefully assess whether and to what extent an MCL would improve protection of public health in communities already burdened by water, air and industrial pollution. *See* page 45.

6) The regulatory landscape for PFAS in solid waste and other media continues to evolve. The PFAS Workgroup recommends that this be factored in when the treatment technologies available do not destroy the contaminant but rather move it from one media to another. *See* page 46.

2. INTRODUCTION

2.1 Enabling Legislation

HB586 (2020 Acts of Assembly Chapter 611), sponsored by Delegate Elizabeth R. Guzman during the 2020 General Assembly session, reads as follows:

> *An Act to require the Commissioner of Health to convene a work group to study the occurrence of perfluorooctanoic acid (PFOA), perfluorooctane sulfonate (PFOS), perfluorobutyrate (PFBA), perfluoroheptanoic acid (PFHpA), perfluorohexane sulfonate (PFHxS), perfluorononanoic acid (PFNA), and other perfluoroalkyl and polyfluoroalkyl substances (PFAS) in the Commonwealth's public drinking water; report.*
>
> Approved April 2, 2020
>
> Be it enacted by the General Assembly of Virginia:
>
> **1.** *§ 1. That the Commissioner of Health shall convene a work group to study the occurrence of perfluorooctanoic acid (PFOA), perfluorooctane sulfonate (PFOS), perfluorobutyrate (PFBA), perfluoroheptanoic acid (PFHpA), perfluorohexane sulfonate (PFHxS), perfluorononanoic acid (PFNA), and other perfluoroalkyl and polyfluoroalkyl substances (PFAS), as deemed necessary, in the Commonwealth's public drinking water and may develop recommendations for specific maximum contaminant levels for PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS, as deemed necessary, for inclusion in regulations of the Board of Health applicable to waterworks. Such work group shall include representatives of waterworks owners and operators, including owners and operators of community waterworks, private companies that operate waterworks, advocacy groups representing owners and operators of waterworks, consumers of public drinking water, a manufacturer with chemistry experience, and such other stakeholders as the Commissioner of Health shall deem appropriate. The Office of Drinking Water of the Department of Health shall provide administrative and technical support for the work group. In completing its work, the work group (i) shall (a) determine current levels of PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS, as deemed necessary, contamination in the Commonwealth's public drinking water, provided that in making such determination of current levels, the Department of Health shall sample no more than 50 representative waterworks and major sources of water; (b) identify possible sources of such contamination, where identified; and (c) evaluate existing approaches to regulating PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS, as deemed necessary, in drinking water, including regulatory approaches adopted by other states and the federal government, and (ii) may develop recommendations for specific maximum contaminant levels for PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS, as deemed necessary, to be included in regulations of the Board of Health applicable to waterworks. The work group shall report its findings and recommendations to the Governor and the Chairmen of the House Committees on Agriculture, Chesapeake and Natural Resources and Health, Welfare and Institutions and the Senate Committees on Agriculture, Conservation and Natural Resources and Education and Health by December 1, 2021.*

2.2 Related Legislation

The Virginia Department of Health (VDH) submitted a report to the Chairmen of the Senate Committee on Education and Health and the House Committee on Health, Welfare and Institutions on October 1, 2021, detailing the agency's efforts towards establishing MCL regulations for PFAS and other emerging contaminants, as required by 2020 Acts of Assembly Chapter 1097 (HB1257). The report can be found on the Legislative Information System website, under "Reports to the General Assembly."

3. PER- AND POLYFLUOROALKYL SUBSTANCES (PFAS)

3.1 History, Development, Use of PFAS, Presence in the Environment, and Health Effects

PFAS, a class of synthetic organic chemicals, entered the national spotlight because of the potential risk that they pose to human health and the environment. While public attention to PFAS is relatively new, the chemicals themselves have been manufactured and used worldwide since the 1940s. The chemical structures of PFAS vary widely but all contain at least one fully fluorinated carbon atom. Strong Carbon-Fluorine (C-F) bonds and other physical and chemical characteristics make PFAS highly stable, heat-resistant, and oil- and water-repellent. PFAS have been widely used in consumer products such as nonstick cookware, waterproof apparel, stain-resistant textiles and carpets, personal care products, cleaners, waxes, and food packaging materials. PFAS also have numerous industrial applications – for instance, PFAS are used in chrome plating operations, and as the primary ingredient in aqueous film-forming foam (AFFF), the class of firefighting foam used to extinguish high-hazard flammable liquid fires.

While public attention to PFAS is relatively new, the chemicals themselves have been manufactured and used worldwide since the 1940s.

The unique properties that made PFAS desired chemicals in manufacturing also make them pervasive and persistent once released into the environment. PFAS easily migrate in the environment and cause contamination of soil, sediment, groundwater, and surface water. PFAS are known as "forever chemicals" because they are non-biodegradable and persistent in nature. As such, humans and animals can be exposed to PFAS through drinking water and eating contaminated fish and plants. There are various environmental exposure routes for humans and animals from the use of PFAS-containing consumer products and consumption of food packaged in PFAS-containing materials.

The existing body of scientific literature on PFAS in drinking water has focused on a limited number of PFAS, primarily PFOA and PFOS, but also includes information on GenX, PFBS, and other PFAS named in HB586 (section 2.1). Such literature has suggested an association between the exposure of PFOA, PFOS, and GenX at certain levels to human health effects ranging from developmental effects in fetuses and infants to certain forms of cancer.

Environmental concentrations of concern currently reach as low as the parts per trillion (ppt) range. Limited toxicity data is available for all of the more than 4,000 PFAS, so further study is necessary to understand potential health effects from PFAS.

3.2 PFAS in Virginia (prior to HB586)

In Virginia, PFAS has been detected at military facilities in the Tidewater area and at NASA's Wallops Island facility

In the past few years, the U.S. Environmental Protection Agency (EPA) began assessing PFAS, primarily in drinking water. Between 2013 and 2015, large public water systems (i.e., waterworks) were required to test their finished drinking water for six (6) specific PFAS, among other pollutants, under the EPA's Third Unregulated Contaminant Monitoring Rule (UCMR3) carried out according to the Safe Drinking Water Act (SDWA). In Virginia, 72 large community waterworks and 15 small waterworks[2] were tested for PFAS. Of 509 tests, only two (2) reported any PFAS detections above EPA's reporting limit. Upon retesting, confirmation samples did not show the detection of PFAS.

Nationwide, between 2013 and 2015, under the UCMR3, 1.3% of large public water systems nationwide reported detections of at least one PFAS that exceeded the reference concentration of 70 ppt. These systems were estimated to provide drinking water to approximately 5.5 million people. However, the reporting limits for PFOA and PFOS used in UCMR3 were high (20 ppt and 40 ppt) compared to current laboratory detection limits, which depend on the laboratory's capability and can be less than 2 ppt. The practical quantitation limit (PQL) for finished water samples analyzed in the HB586 Sample Study was generally 3.5 ppt.

In Virginia, PFAS has been detected at military facilities in the Tidewater area (*see* https://vcij.org/stories/virginias-toxic-military-legacy) and at NASA's Wallops Island facility (*see* https://www.nasa.gov/feature/background-latest-information-on-pfas-at-nasa-wallops/). In each of these cases, PFAS contamination is believed to be associated with the use of AFFF.

Another known site of PFAS contamination in Virginia is the DuPont Spruance Plant south of downtown Richmond. The DuPont Plant is a Resource Conservation and Recovery Act Corrective Action Site. Operations at the facility have resulted in soil and groundwater contamination necessitating remedial measures to protect human health and the environment. The groundwater constituents of potential concern identified include PFOA. EPA states the contaminated groundwater is not used for drinking water, and no down gradient users of off-site groundwater exist between the site boundary and the James River (*see*

[2] "Small" in the context of UCMR3 means a waterworks serving ≤ 10,000 people. Large waterworks serve more than 10,000 people. *See* 77 FR 26072, May 12, 2012. https://www.govinfo.gov/content/pkg/FR-2012-05-02/pdf/2012-9978.pdf

https://www.epa.gov/hwcorrectiveaction/hazardous-waste-cleanup-dupont-spruance-facility-richmond-va).

3.3 Existing Approaches to Regulating PFAS

To date, EPA has not established any enforceable limits on PFAS in drinking water. However, the agency has taken a number of steps including:

a. EPA Council on PFAS (ECP).

As described in a memorandum dated April 27, 2021, Michael S. Regan, EPA Administrator said the " ...EPA Council on PFAS will collaborate on cross-cutting strategies; advance new science; develop coordinated policies, regulations and communications; and engage with affected states, tribes, communities and stakeholders. The work of the ECP is not a substitute for the ongoing efforts by our national programs and regions to meaningfully address PFAS pollution and its impacts on public health and communities. The ECP will strive to build on and significantly enhance our capabilities through comprehensive, coordinated and results-driven multi-media actions."

The ECP developed the PFAS Strategic Roadmap (see below) to lay out EPA's whole-of-agency approach to tackling PFAS and set timelines by which the Agency plans to take concrete actions during the first term of the Biden-Harris Administration to deliver results for the American people.

b. EPA Lifetime Health Advisory Level

To provide Americans, including the most sensitive populations, with a margin of protection from a lifetime of exposure to PFOA and PFOS from drinking water, EPA established a lifetime health advisory (LHA) level of 70 ppt in 2016 for the chemicals individually or combined (PFOA+PFOS). LHA are not enforceable standards. The health advisory level offers a margin of protection for all Americans throughout their life from adverse health effects resulting from exposure to PFOA and PFOS in drinking water.

EPA established lifetime health advisory (LHA) levels of 70 ppt in 2016 for PFOA and PFOS individually or combined (PFOA+PFOS). LHA are not enforceable standards.

c. EPA Safe Drinking Water Act (SDWA) Processes.

i. EPA issued final regulatory determinations for PFOA and PFOS, which were contaminants on the fourth Contaminant Candidate List (CCL 4, *see* https://www.epa.gov/ccl/contaminant-candidate-list-4-ccl-4-0).

ii. The Fifth Unregulated Contaminant Monitoring Rule (UCMR5, *see* https://www.epa.gov/sites/default/files/2021-01/documents/ucmr5-proposal-factsheet-draft.pdf) will require waterworks to monitor for 29 PFAS that may be in drinking water.

iii. On July 19, 2021, EPA published Contaminant Candidate List 5 (CCL 5, *see* https://www.epa.gov/ccl/contaminant-candidate-list-5-ccl-5), which includes PFAS as a chemical group, not individual compounds.

d. Other Federal Actions: Legislative Activities

i. *The PFAS Action Act of 2021* (H.R. 2467) (passed House of Representatives July 21, 2021)

e. PFAS Strategic Roadmap

On October 18, 2021, EPA Administrator Michael S. Regan announced the agency's PFAS Strategic Roadmap: EPA's Commitments to Action 2021-2024 (Appendix 2), laying out a whole-of-agency approach to addressing PFAS. The roadmap sets timelines by which EPA plans to take specific actions and commits it to new policies to safeguard public health, protect the environment, and hold polluters accountable. The actions described in the roadmap each represent steps EPA plans to take to safeguard communities from PFAS contamination. Cumulatively, EPA believes these actions will build upon one another and lead to more enduring and protective solutions.

With respect to drinking water, the PFAS Strategic Roadmap calls for EPA to do the following (Appendix 2, pp. 12-13, 15):

i. Undertake nationwide monitoring for PFAS in drinking water under UCMR5, significantly expanding the number of waterworks participating in the program, pending sufficient appropriations by Congress (to pay for testing). (expected fall 2021)
ii. Establish a national primary drinking water regulation for PFOA and PFOS that would set enforceable limits and require monitoring of public water supplies, while evaluating additional PFAS and groups of PFAS. (proposed rule fall 2022, final rule fall 2023)
iii. Publish the final toxicity assessment for GenX and five additional PFAS – PFBA, PFHxA, PFHxS, PFNA, and PFDA – to better understand their human health and environmental effects. (expected fall 2021 and ongoing – EPA published the toxicity assessment for GenX on October, 25 2021, *see* https://www.epa.gov/pfas/genx-toxicity-assessments-documents)
iv. Publish health advisories for GenX and PFBS based on final toxicity assessments to enable tribes, states, and local governments to inform the public and take appropriate action. (expected spring 2022)
v. Publish improved analytical methods to enable 40 PFAS to be monitored in eight different environmental matrices, and to update methods for drinking-water monitoring. (expected fall 2022 and fall 2024)

States have taken different approaches to address public health concerns of PFAS in drinking water. The majority of states (31 at the time of this report)[3] have not taken action and utilize EPA's Lifetime Health Advisory (LHA) level of 70 ppt for PFOA and PFOS, individually or combined, as guidance.

Seven (7) states[4] have established special state guidance (but not regulations) for screening and investigatory purposes. These may be similar to or lower than EPA's LHA for PFOA and PFOS, or apply to other PFAS.

Eight (8) states have regulations substantially more stringent than EPA's LHA and three (3) are in the process of promulgating regulations.[5] Virginia is following this approach, but has not formally begun the rulemaking process. VDH expects to begin the process of establishing MCLs since the amendments to Code of Virginia § 32.1-169 become effective on January 1, 2022.

3.4 Virginia PFAS Workgroup

The State Health Commissioner formed the Virginia PFAS Workgroup in October 2020 to study the level of PFAS contamination in drinking water in Virginia.

HB586 includes four (4) specific responsibilities for the PFAS Workgroup to fulfill:

- Determine current levels of PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS, as deemed necessary, contamination in the Commonwealth's public drinking water, provided that in making such determination of current levels, the Department of Health shall sample no more than 50 representative waterworks and major sources of water;
- Identify possible sources of such contamination, where identified;
- Evaluate existing approaches to regulating PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS, as deemed necessary, in drinking water, including regulatory approaches adopted by other states and the federal government; and
- Report findings and recommendations to the Governor and Chairmen of the Senate Committees on Agriculture, Conservation and Natural Resources and Education and Health and the House Committees on Agriculture, Chesapeake and Natural Resources and Health, Welfare and Institutions by December 1, 2021.

HB586 includes one optional provision for the PFAS Workgroup – it may develop recommendations for specific MCLs for PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS, as deemed necessary, to be included in regulations of the Board of Health applicable to waterworks.

[3] AL, AR, AZ, CO, DE, FL, GA, HI, IA, ID, IN, KS, KY, LA, MD, MO, MS, MT, NE, ND, NM, NV, OK, OR, SC, SD, TN, TX, WV, WY, UT.
[4] AK, CT, OH, RI, IL, MN, NC.
[5] CA, MA, ME, MI, NH, NJ, NY, VT (PA, WA, and WI are in the process of promulgating regulations).

VDH, through its Office of Drinking Water, is responsible for providing administrative and technical support for the PFAS Workgroup.

Members of the PFAS Workgroup represent the following stakeholders (see Appendix 1 for a list of members and the groups they represent):

1. Community waterworks that serve more than 50,000 persons.
2. Community waterworks that serve less than 50,000 persons.
3. Community waterworks that serve less than 1,000 persons.
4. An advocacy group that represents waterworks in Virginia.
5. A chemical manufacturer with chemistry experience.
6. A consumer of public drinking water.
7. Non-governmental environmental organizations.
8. The Virginia Department of Environmental Quality (DEQ).
9. A local health district.

The State Toxicologist and an ODW staff member also serve on the PFAS Workgroup.

The ODW Deputy Director is the PFAS Workgroup leader.

The Virginia PFAS Workgroup kickoff meeting took place on October 20, 2020

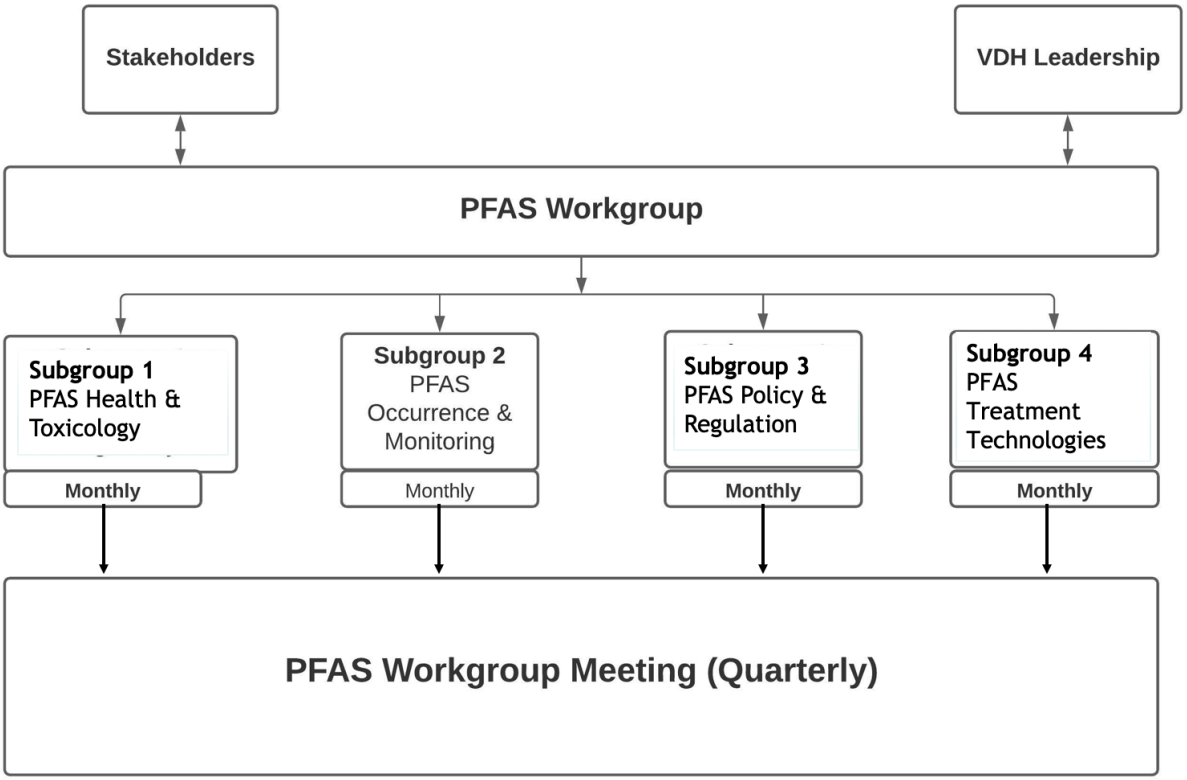


Figure 1. Virginia PFAS Workgroup and Subgroup functional structure.

3.4.1 Subgroups

PFAS Workgroup members, and members of the public, served in one or more of four (4) subgroups that focused on specific requirements of HB586 (Figure 1). The subgroups included, Health and Toxicology, Occurrence and Monitoring, Policy and Regulations, and Treatment Technologies. The State Toxicologist led the Health and Toxicology subgroup; ODW staff led and coordinated the other subgroups. Appendix 1 contains a list with members of each subgroup.

The Health and Toxicology Subgroup assessed the public health risk of the six (6) PFAS specified in HB586, using a toxicological database that is evolving as more studies are done on PFAS – individually and as a suite of compounds. EPA and a number of states have assessed the risk of PFAS in drinking water to varying degrees. The toxicology subgroup considered the health and toxicological methodologies and models adopted by EPA, the states, and current peer reviewed studies. As the toxicology subgroup completed its evaluations for the PFAS Workgroup, it considered the need for additional experts in the field of toxicology and epidemiology to assist with these efforts. The toxicology subgroup did not propose a safe level for the PFAS found in Virginia public waterworks to the PFAS Workgroup. Subgroup members concluded that more time, information, and expertise would be necessary to determine a safe level of PFAS in drinking water, or make recommendations for choosing to add or remove any specific PFAS ("as deemed necessary") from those studied.

The Monitoring and Occurrence Subgroup evaluated how to best determine the occurrence of PFAS in drinking water, including reviewing approaches adopted by other states and the federal government. The subgroup guided the PFAS Sample Study Design and implementation of the sampling program. After ODW completed quality assurance/quality control review of the sample results collected pursuant to HB586, the monitoring and occurrence subgroup tabulated the PFAS data from the Sample Study. This subgroup identified levels of PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS based on this data and made recommendations to the PFAS Workgroup regarding additional sampling.

The Policy and Regulations Subgroup evaluated approaches to regulating PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS as deemed necessary, in drinking water, including regulatory approaches adopted by other states and the federal government. As data and information about PFAS occurrence in Virginia became available from the Sample Study, the policy subgroup, based on input from other subgroups, did not develop recommendations for specific MCLs for PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, or any other PFAS, to present to the PFAS Workgroup for consideration. The subgroup considered methodologies other states developed to regulate PFAS in drinking water and, based on the information and resources available in Virginia, did not find that one framework could be best suited to establishing MCLs in Virginia. The subgroup discussed multiple paths for establishing MCLs within the limits of the enabling legislation, budget, timeframe to act, and extent of data that is available.

The Treatment Technologies Subgroup reviewed best available treatment technologies (BATT) for PFAS removal, including whether certain technologies are better for controlling PFOA, PFOS, PFBA, PFHpA, PFHxS, and/or PFNA. Subgroup members reviewed design criteria and practical treatment goals and/or limits of treatment technology for each PFAS species specified in HB586, including capital and operating costs for each technology. The treatment technologies subgroup considered whether pilot testing would be necessary and identified special considerations related to disposal of waste streams from the treatment process. Treatment technologies reviewed by the subgroup (GAC, IX and RO) generate a concentrated PFAS waste stream/product that requires special considerations for handling and disposal.

The seventeen (17) largest waterworks, eleven (11) groundwater waterworks, and twenty-two (22) source water intakes from waterworks were identified in Virginia PFAS sample study

3.4.2 PFAS Workgroup Meetings

Since its inception in October 2020, the PFAS Workgroup met seven (7) times: October 20, 2020 and January 19, March 4, April 29, July 27, September 10, and October 8, 2021.[6] Subgroups generally met on a monthly basis between December 2020 and October 2021.

In March 2021, the PFAS Workgroup approved a PFAS Sample Study Design (Appendix 3). The PFAS Workgroup recommended a hybrid approach to the Sample Study, which involved collecting water samples for testing from:

- The seventeen (17) largest waterworks in Virginia by population served;
- Eleven (11) waterworks that use groundwater as a primary source and are located within one mile of a potential source of PFAS, including unlined landfills and commercial and military airports where AFFF may have been used; and
- Twenty-two (22) waterworks (excluding the 17 largest waterworks) that have an intake in a major water supply downstream of potential high risk sources based on industrial use code (i.e., factories and other facilities that, based on the type of manufacturing or products produced, may have used compounds containing PFAS).

[6] Due to the coronavirus pandemic, all of the meetings prior to the July 27, 2021 PFAS Workgroup meeting were conducted by electronic communication means pursuant to the General Assembly's allowance for electronic meetings during the Governor's declared State of Emergency. The PFAS Workgroup and subgroups have followed the requirements in the budget bills to conduct meetings by electronic communication means. For the 2020-2022 biennium, *see* Item 4-0.01g of Chapter 552 of the 2021 Special Session I, https://budget.lis.virginia.gov/item/2020/1/HB30/Chapter/4/4-0.01/.

Meeting minutes are available on the Virginia Regulatory Town Hall website and ODW PFAS webpage at https://www.vdh.virginia.gov/drinking-water/pfas/.

Of the 50 waterworks identified in the PFAS Sample Study Design, 38 waterworks agreed to participate in the Sample Study. ODW reached out to additional waterworks and ultimately 45 waterworks agreed to participate. More details on the Sample Study are in Section 3.5; the Sample Study Design is included in Appendix 3 of the report.

In April 2021, the PFAS Workgroup helped VDH staff develop a "PFAS Communications Toolkit" for participating waterworks and the health districts where each of the waterworks is located. The toolkit contains information about the legislative requirements, PFAS, the Sample Study, fact sheets, and communication templates. The PFAS Communication Toolkit is included in Appendix 4. Sample collection and analysis occurred between May and August 2021.

The PFAS Workgroup received an update on preliminary results from the Sample Study at its July 2021 meeting. In September 2021, the PFAS Workgroup considered the structure and content of this report, and in October 2021, made its final recommendations for the report and its conclusions. VDH staff completed the report following the October 2021 meeting.

3.4.3 Literature Review

The PFAS Workgroup and VDH partnered with Old Dominion University to complete a literature review. VDH paid for the literature review using a grant from EPA to study emerging contaminants, which include PFAS. The literature review is included as Appendix 5 to this report.

3.5 PFAS Monitoring and Occurrence Study, a.k.a. Sample Study

VDH, in conjunction with the PFAS Workgroup, designed the Sample Study to prioritize sites for PFAS sampling and generate statewide occurrence data, subject to the limitations in HB586. VDH and the PFAS Workgroup considered several factors in developing the Sample Study, including:

- Waterworks size and population served;
- The location of potential sources of PFAS contamination (developed in conjunction with DEQ);
- The relative risk to consumers who receive water from waterworks that utilize source water that comes from areas that are near known or potential sources of PFAS contamination; and
- $40,000 in funding from a fiscal year 2021 EPA grant to study emerging contaminants that could be used to pay for sample analysis.

For purposes of the Sample Study, the term "potential sources of PFAS contamination" refers to facilities or locations that may be a source of PFAS based on historical use, existing literature, other available information (Standard Industrial Classification codes, Virginia Pollutant Discharge Elimination System permits, etc.), and/or the nature of the facility (airports, unlined landfills, etc.). This term is not meant to imply that these locations do in fact produce, use, or discharge PFAS; only that previous published work indicates the type of facility or activity may be associated with the production, use, disposal, or discharge of PFAS.

Further, the Sample Study did not determine the cause and effect relationship between potential sources of PFAS and PFAS found in drinking water or drinking water sources. The Sample Study and the sampling performed provided additional data regarding the occurrence of PFAS at a limited number of waterworks in Virginia. The Board of Health can use this information as part of the VAPA process to establish regulatory limits for PFAS pursuant to HB1257.

3.5.1 Sample Locations

The process of selecting sample locations involved a combination of geospatial analysis and programmatic review. The geospatial analysis included the creation of a Geographic Information System (GIS) project using ArcMap 10.4.1 that combined waterworks locations and information about potential sources of PFAS contamination. There are currently 2,811 waterworks in Virginia. They are classified based on the population they serve:

- Community Waterworks (CWS): Waterworks that serve at least 15 service connections used by year-round residents or regularly serve at least 25 year-round residents. Examples include municipal water systems, authorities, and residential subdivisions with their own water supplies.
- Nontransient Noncommunity Waterworks (NTNC): Waterworks that are not CWS and that regularly serve at least 25 of the same people at least six (6) months of the year. Examples include schools, factories, and hospitals that have their own water supplies.
- Transient Noncommunity Waterworks (TNC): Waterworks that are not CWS, but serve transient customers in non-residential settings, such as campgrounds, motels, and restaurants that have their own water supplies. A TNC serves at least 25 persons daily for at least 60 days out of the year.

ODW staff identified the largest waterworks in the state (based on population served) and plotted the locations of surface water intakes and groundwater wells used by CWS and NTNCs, potential discharge locations, including unlined landfills and airports, and major rivers in the state. ODW and the PFAS Workgroup identified three (3) strategies for selecting sites to be part of the Sample Study:

- The potential high and/or medium risk groundwater systems based on the potential sources of PFAS contamination;
- The large CWS (“large” means the waterworks provides water to more than 10,000 persons); and
- The water sources/intakes with potential to receive water from upstream sources of PFAS contamination.

Consumers served by CWS and NTNCs have a higher risk of exposure from drinking, cooking, bathing and showering, and other water uses because of the regular consumption of water over long periods of time, as compared to TNC consumers. For this reason, ODW limited the Sample Study to CWS and NTNCs. There are 1,093 CWS and 510 NTNCs, for a total initial sampling

pool of 1,603 waterworks, which collectively provide drinking water from 2,626 sources (e.g. wells, springs, and surface water sources).

VDH prioritized the list of CWS and NTNCs based on relative risk, considering the waterworks' proximity to the potential sources of PFAS contamination. Using a Geographic Information System, VDH established several data layers containing locational and other information specific to the potential sources of PFAS contamination. These layers include the following industries and land uses:

- Military or commercial airports (from U.S. Geological Survey data);
- Unlined landfills (data from DEQ);
- VPDES discharge locations (data from DEQ);
- Discharge points for publicly owned treatment works (POTWs) (data from DEQ);
- Major river networks in Virginia; and
- Waterworks size and population served.

A significant portion of the peer-reviewed, published literature on PFAS contamination focuses on contamination resulting from the use of AFFF, a product approved for use by the Federal Aviation Administration (FAA) and the U.S. Department of Defense (DoD). AFFF that meets DoD specifications for use at military facilities is a common source of PFAS and is frequently found at both military and large civilian airports, as well as many firefighting facilities. Other sources of PFAS associated with airports and the aeronautical industry include certain mechanical fluids. Given the number of products that can be found at airports and that potentially contain PFAS, airports are considered a likely source of PFAS contamination. For the purpose of the geospatial analysis, ODW staff considered large airports (meaning the airport is large enough to be classified as a public-use airport). ODW did not attempt to identify whether the airports had either an intentional or accidental release of AFFF, or if they conducted training with AFFF on site.

Peer-reviewed, published research also indicates that landfills and landfill leachate may be a potential source of PFAS contamination. Landfill leachate likely obtains PFAS from the myriad of consumer products that include PFAS and are commonly placed in landfills. Consumer products, food contact packaging, cosmetics, and electronics are examples of PFAS-containing products commonly found in garbage. There are landfills in Virginia that were constructed before they had to meet the requirements in Subtitle D of the Resource Conservation and Recovery Act (RCRA), meaning they are unlined and more likely to have leachate that reaches groundwater sources. The Subtitle D criteria do not apply to landfill units if they do not receive waste after October 9, 1991. *See* 40 C.F.R. § 258.1(c). DEQ recommended focusing on landfills that did not have linings, leachate collection systems, or other waste disposal facilities.

VDH designated any waterworks using a groundwater well located within 0.5 miles of an unlined landfill or airport as a potential high risk for PFAS influence. VDH designated other

waterworks using a groundwater well within between 0.5 mile and 1 mile of a known unlined landfill or airport as a potential medium risk water source.

VDH does not possess, and therefore did not consider, the following in evaluating potential high or medium risk groundwater waterworks/water sources.

- Data on PFAS levels in groundwater;
- Information on groundwater flow direction; or
- Information on water supply well recharge areas.

Based on the compilation of potential sources of PFAS contamination, VDH and the PFAS Workgroup selected 11 waterworks that use groundwater wells within one (1) mile of potential sources of PFAS contamination. These waterworks have a total of six (6) groundwater wells considered high risk and 13 groundwater wells that constitute a medium risk based on proximity to a potential source of PFAS contamination.

VDH also identified major surface water supplies based on potential sources of PFAS contamination that DEQ identified from SIC codes and information in VPDES permits. These included POTWs with significant industrial users and direct dischargers. The identified facilities potentially use and/or discharge PFAS; however, DEQ does not have effluent monitoring data for PFAS. DEQ noted that both current and historic discharges of PFAS could impact waterworks' surface water intakes. DEQ provided the Global Positioning System (GPS) coordinates for the discharge points to ODW. Using ArcGIS, ODW connected the discharge points to surface water bodies and identified them as potentially impacted by PFAS discharges. ODW traced the surface water bodies downstream to identify waterworks with surface water intakes potentially impacted by the discharges. This procedure identified 45 drinking water intakes potentially impacted by the discharges. ODW prioritized these 45 intakes as follows:

- ODW excluded intakes associated with the 17 largest waterworks because the entry point sampling addressed these intakes;
- ODW sorted remaining waterworks from the largest to the smallest population served;
- The Occurrence and Monitoring subgroup recommended including at least one sample location from the New River, Clinch River, and Dan River to ensure a wider distribution of sample sites;
- ODW selected impacted intakes starting with the largest population served, selecting two intakes on the river systems noted above; and
- ODW selected no more than one intake per waterworks.

Based on the limitation in HB586 of no more than 50 waterworks and major sources of water, and the number of waterworks selected via the hybrid approach, VDH selected 22 major sources of water for sampling. Maps 2 and 4 in the PFAS Sample Study Design (Appendix 3) show the locations of potential sources of PFAS contamination, surface water sources that are potentially

impacted by PFAS, and associated surface water intake locations selected for monitoring as part of the Sample Study.

VDH selected the 17 largest waterworks in the state, which serve approximately 4.5 million consumers. This group represents 23 raw water sources, 21 water treatment plants, and 12 consecutive connections. VDH selected to monitor drinking water at the entry points to the distribution system, at the water treatment plants, and at consecutive connections. All of these samples represent "finished water," which means the drinking water has gone through the waterworks treatment process before going into the distribution system.

3.5.2 Laboratory Analytical Services

VDH contracted with a laboratory through a competitive bidding process to prepare sample kits, ship them to the participating waterworks, provide return shipping, analyze the samples, and return results to VDH and the waterworks using EPA Method 533 for finished water samples and a comparable method for source (untreated) water samples. The laboratory had to meet accreditation and other requirements in VDH's Quality Assurance Project Plan, which EPA approved as a requirement for VDH to use the federal grant to pay for testing. The laboratory analyzed drinking water samples by EPA Method 533 because this method reports the analytes specified in HB586, whereas EPA Method 537.1 does not (it does not include PFBA). Other related requirements included:

The laboratory used US EPA method 533 to analyze finished water samples. Method 533 reports 25 specific PFAS.

- The laboratory will report the complete list of 25 analytes for Method 533.
- The laboratory will establish method reporting limits (MRLs) for each analyte based on the lowest concentration of standards used by the laboratory.
- The laboratory will meet National Environmental Laboratory Certification (NELAC) accreditation requirements.

The laboratory analyzed source water samples using a method employing solid phase extraction, liquid chromatograph/mass spectrometer/mass spectrometer (LC/MS/MS), and isotope dilution that met the requirements of Table B-15 of the DoD ELAP QSM (Environmental Laboratory Accreditation Program Quality Systems Manual). The laboratory had to analyze source water samples by another method since EPA Methods 537.1 and 533 are applicable only to drinking water. Other related requirements for source water analysis included:

- The laboratory will report the same analytes as EPA Method 533.
- The laboratory will use the same MRLs as EPA Method 533 or as agreed by VDH.
- The laboratory will hold accreditation for the DoD PFAS method by LC/MS/MS compliant with QSM 5.3 Table B-15.

3.6 PFAS Sample Study Results

Of the 50 waterworks identified, 38 agreed to participate in the study. ODW reached out to the next set of additional prioritized waterworks and ultimately 45 waterworks agreed to participate in the Sample Study (40 with surface water sources; 5 with groundwater sources). There are a total of 63 sample locations among the 45 waterworks because some waterworks have more than one treatment facility or water source. Examples include:

- Western Virginia Water Authority uses water from Carvins Cove and Spring Hollow Reservoir;
- Chesterfield County Water System uses water from the City of Richmond water treatment plant (source – James River), Lake Chesdin (from the Appomattox River Water Authority), and its own water treatment plant at the Swift Creek Reservoir;
- Fairfax Water operates the James J. Corbalis and Frank P. Griffith water treatment plants (treating water from the Potomac River and Occoquan Reservoir respectively); and
- Town of Bowling Green uses three separate wells.

Tables 1 and 2 provide a summary of the sample results. Figure 2 shows the sample locations.

PFAS in quantities above the practical quantitation level (PQL) was detected at 15 of 63 sample locations.

Waterworks received sample kits from the laboratory in May through August 2021. ODW and the laboratory provided training and specific instructions on sample collection procedures so that staff at each of the participating waterworks could collect the samples. Waterworks staff collected samples and returned them to the laboratory for analysis.

Quality assurance/quality control (QA/QC) review of the results revealed data inconsistencies with four (4) samples, so ODW requested the waterworks resample from each of the four (4) locations. Data inconsistencies means the water sample did not have any detected PFAS, but the field reagent blank (FRB), used for QA/QC purposes, had PFAS, which suggested the two were either switched or there was cross-contamination. Another data irregularity occurred when both the sample and FRB had PFAS, which suggested a sample collection error, or another data qualifier was out of the specified range for the FRB.

Results from the Sample Study, Tables 1 and 2, found PFAS in quantities above the practical quantitation level (PQL) at 15 of 63 sample locations. The highest detected concentration of a compound was 57 ppt of HFPO-DA, which is commonly known as GenX, a type of PFAS developed to replace use of PFOA. All other detections were 20 ppt or less.[7] Samples from 48 sample locations did not contain any PFAS above the PQL. This means PFAS were either not

[7] 20 ppt is significant since Massachusetts and Vermont established a maximum contaminant level (MCL) of 20 ppt for total PFAS, which differs from the approach of other states that established MCLs for individual PFAS.

present in the samples, or that the concentration was so low, in most cases less than 3.5 ppt, that it could not be reliably measured. Resamples resolved QA/QC questions with the data inconsistencies.

- PFOA was measured above the 3.5 ppt practical quantitation limit (PQL) at four sample locations. Measured concentrations were between 4.2 and 5.5 ppt.
- PFOS was measured above the 3.5 ppt PQL at seven sample locations. Measured concentrations were between 3.9 and 7.1 ppt.
- PFBA was measured above the 3.5 ppt PQL at 10 sample locations. Measured concentrations were between 3.7 and 12.0 ppt.
- PFHpA was measured above the 3.5 ppt PQL at three sample locations. Measured concentrations were between 4.1 and 5.5 ppt.

No water samples collected during the study exceeded the EPA LHA of 70 ppt for PFOA and PFOS, individually or combined.

- PFHxS was measured above the 3.5 ppt PQL at one sample location. The concentration was 4.9 ppt.
- PFNA was not detected in any samples at a concentration above the PQL.
- Four (4) additional PFAS that are not listed in HB586 were measured in samples. They include HFPO-DA, PFHxA (perfluorohexanoic acid), PFPeA (perfluoropentanoic acid), and PFBS (perfluorobutanesulfonic acid).

All of the samples that had PFAS present above the PQL, except one, were entry point samples. Neither VDH nor DEQ have collected additional samples to identify potential sources of PFAS contamination.

At this time, the results from the PFAS Sample Study suggest that PFAS may be present in water from the Potomac River and Occoquan Reservoir in an undetermined quantity. The sample study found levels of PFAS between the PQL and 10 to 12 ppt for certain PFAS in water from waterworks served by these sources. Other patterns or conclusions are not evident from the data at this time. For example, the results from the finished water sample from Fairfax Water's Potomac River source (Corbalis WTP) were all below the PQL, but the finished water sample Loudoun County submitted from its connection to Fairfax Water, which is from the same source, has some PFAS detections. The reason and sources of these detections is unknown at this time.

Table 1. Virginia Per- & Polyfluoroalkyl Substance sampling study results summary

Samples with analytes above the Practical Quantification Limit (PQL)

All samples were collected between May and September 2021

All results are parts per trillion (ppt)

Waterworks Name	**Virginia American Water Co. - Alexandria District**		**Arlington County**	**Fairfax County Water Authority**		**Loudoun Water - Central System**		**Stafford County Utilities**		**Prince William County Service Authority - East**	**City of Newport News**		**Town of Altavista**	**Western Virginia Water Authority**	**Washington County Service Authority**
City/County	City of Alexandria		Arlington County	Fairfax County		Loudoun County		Stafford County		Prince William County	City of Newport News		Campbell County	Roanoke County	Washington County
Sample Location	From Fairfax County Water Authority		From Washington Aqueduct	Griffith WTP	From Washington Aqueduct	Trap Rock WTP	From Fairfax County Water Authority	Smith Lake WTP	Lake Mooney WTP	From Fairfax County Water Authority	Harwoods Mill WTP	Lee Hall WTP	Staunton River + Reed Creek	Spring Hollow WTP	Middle Fork Water Treatment Plant
Water Type	Finished	Finished	Finished	Finished	Finished	Finished	Finished	Finished	Finished	Finished	Finished	Finished	Raw Intake	Finished	Finished
PFOA (ppt)	*	4.2	*	5.5	*	*	4.5	*	*	5.5	*	*	*	*	*
PFOS (ppt)	*	3.9	*	5.1	*	*	*	6.4	*	4.1	7.1	4.4	*	*	5.2
PFBA (ppt)	7.7	9.2	*	7.7	4.3	4.0	4.6	*	5.9	12	4.3	4.3	*	*	*
PFHpA (ppt)	*	*	*	4.4	*	*	5.5	*	*	4.1	*	*	*	*	*
PFHxS (ppt)	*	*	*	*	*	*	*	*	*	*	4.9	*	*	*	*
PFNA (ppt)	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
HFPO-DA (GenX)(ppt)	*	*	*	*	*	*	*	*	*	*	*	*	4.0	54[A]	*
PFHxA (ppt)	6.8	9.3	3.7	12	4.4	*	*	*	4.2	11	*	6.1	*	*	*
PFPeA (ppt)	7.4	10	4.1	14	4.2	*	*	*	5.5	12	*	4.5	*	*	*
PFBS (ppt)	*	4.2	*	5.6	*	*	*	*	*	4.8	*	*	*	*	*

* Results were below the Practical Quantitation Limit (PQL); PQL is the lowest level that can be reliably measured within specified limits of precision and accuracy during routine laboratory conditions.

[A] Average of two results, 51 ppt and 57 ppt
“Finished” means treated drinking water entering the distribution system. “Raw Intake” means untreated water, before treatment.
“WTP” means water treatment plant.

Table 2. Samples that did not contain PFAS above the Practical Quantification Limit *

Waterworks Name	City/County	Water Type	Sampling Point
Earlysville Forest	Albemarle County	Finished	Combined Wells
Peacock Hill Subdivision	Albemarle County	Finished	Combined Wells
Pungoteague Elementary School	Accomack County	Finished	Well
Town of Bowling Green	Caroline County	Finished	Combined Wells
Mountain View Elementary School	Rockbridge County	Finished	Well
Frederick Water	Frederick County	Finished	James Diehl WTP
Frederick Water	Frederick County	Finished	James T. Anderson WTP
Western Virginia Water Authority	Roanoke County	Finished	Carvins Cove WTP
City of Chesapeake - Northwest River System	City of Chesapeake	Finished	Northwest River WTP
City of Chesapeake - Northwest River System	City of Chesapeake	Finished	Lake Gaston WTP
City of Norfolk	City of Norfolk	Finished	Moores Bridges WTP
City of Norfolk	City of Norfolk	Finished	Kristen M Lentz WTP
City of Portsmouth	City of Portsmouth	Finished	Lake Kilby WTP
City of Virginia Beach	City of Virginia Beach	Finished	From City of Norfolk
Chesterfield County Central Water System	Chesterfield County	Finished	Addison Evans WTP
Chesterfield County Central Water System	Chesterfield County	Finished	From City of Richmond
Chesterfield County Central Water System	Chesterfield County	Finished	From Appomattox River Water Authority
Henrico County Water System	Henrico County	Finished	Henrico WTP
Henrico County Water System	Henrico County	Finished	From City of Richmond
City of Richmond	City of Richmond	Finished	Richmond WTP
City of Lynchburg	City of Lynchburg	Finished	Abert Water Treatment Plan
City of Lynchburg	City of Lynchburg	Finished	College Hill WTP
Fairfax County Water Authority	Fairfax County	Finished	Corbalis WTP
Prince William County Service Authority - West	Prince William County	Finished	City of Manassas WTP
Prince William County Service Authority - West	Prince William County	Finished	Fairfax County Water Authority
Spotsylvania County Utilities	Spotsylvania County	Finished	Ni River WTP
Spotsylvania County Utilities	Spotsylvania County	Finished	Motts Run WTP
NRV Regional Water Authority	Montgomery County	Raw Intake	New River
Radford Army Ammunition Plant	Montgomery County	Raw Intake	New River
Pulaski County Public Service Authority	Pulaski County	Raw Intake	Claytor Lake
Town of Richlands	Tazewell County	Raw Intake	Clinch River
Town of Wytheville	Wythe County	Raw Intake	Reed Creek
City of Radford	City of Radford	Raw Intake	New River
Town of Berryville	Clarke County	Raw Intake	Shenandoah River

Waterworks Name	City/County	Water Type	Sampling Point
Lake Monticello	Fluvanna County	Raw Intake	Rivanna River
Town of Front Royal	Warren County	Raw Intake	South Fork Shenandoah River
City of Salem	City of Salem	Raw Intake	Roanoke River
VA American Water Co., Hopewell District	City of Hopewell	Raw Intake	Appomattox River
James River Correctional Center	Goochland County	Raw Intake	James River
Hanover Suburban Water System	Hanover County	Raw Intake	North Anna River
Roanoke River Service Authority	Mecklenburg County	Raw Intake	Lake Gaston
Town of Farmville	Prince Edward County	Raw Intake	Appomattox River
City of Danville	City of Danville	Raw Intake	Dan River
Halifax County Service Authority - Leigh St Plant	Halifax County	Raw Intake	Dan River
Town of Leesburg	Loudoun County	Raw Intake	Potomac River

* Samples from 48 of the 63 sample locations did not contain any PFAS above the practical quantification limit (PQL). This means PFAS were either not present in the samples, or that the concentration was so low, in most cases less than 3.5 parts per trillion (ppt), that it could not be reliably measured.

The PQL is the lowest level that can be reliably measured within specified limits of precision and accuracy during routine laboratory conditions.

“Finished” means treated drinking water entering the distribution system.

“Raw Intake” means untreated source water, sampled at a water treatment plant.

“WTP”" means water treatment plant.

“Well” means water from one well, after treatment, if provided.

“Combined Wells” means water from two or more wells, after treatment, if provided.

“From” indicates finished water purchased from a waterworks.

All samples were collected between May and September 2021.

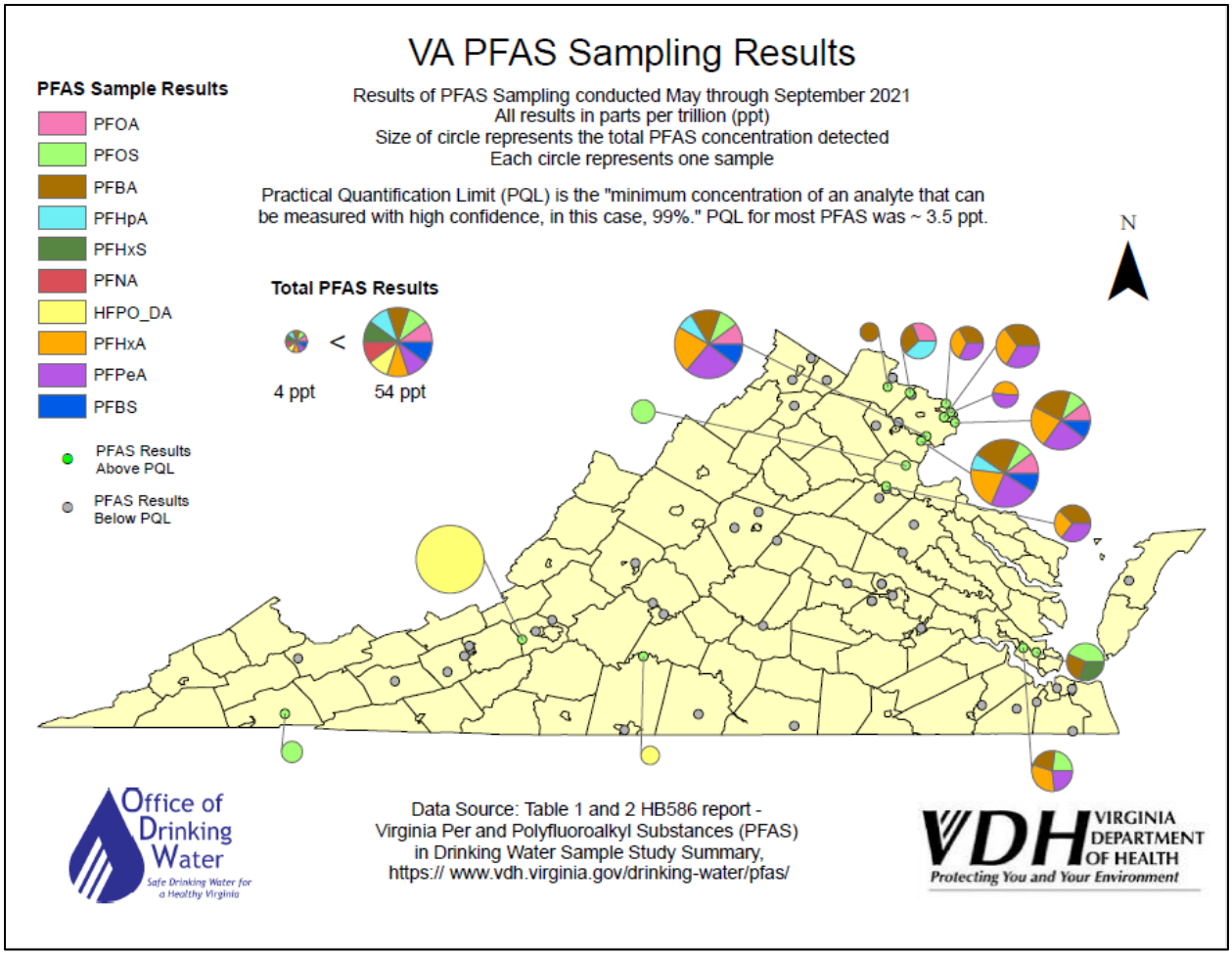


Figure 2. PFAS Sampling Results.

3.7 Treatment Technologies

Treatment processes that are most commonly employed by waterworks are not effective at removing PFAS, nor are some of the more "advanced technologies" that are sometimes used. These processes include coagulation-settling-filtration, chemical oxidation, and advanced oxidation processes, such as ultraviolet light oxidation. There are three treatment systems known to be effective for removal of persistent, water-soluble surfactants within the family of PFAS in drinking water down to part-per-trillion (ppt, ng/L) levels: Granular Activated Carbon, Ion Exchange Resins, and Reverse Osmosis Membranes (reverse osmosis). These technologies are more expensive than conventional treatment processes. Descriptions of each technology, along with disadvantages and unintended consequences, are included below. However, some general comments apply to all of the treatment technologies:

- Each of these three types of systems has been thoroughly validated for PFAS removal and installed in commercial operations. Therefore, each is considered fully demonstrated and ready for design engineering work for new installations.

- All have some inherent advantages and disadvantages so selection of a treatment system should be made based on local considerations that include:
    - volume of water to be treated,
    - composition of water to be treated, including co-contaminants, organic matter, and pH,
    - physical properties of specific PFAS being targeted for removal (ionic state, functional groups present, lipo- and hydrophobicity, chain length and branching, phase behavior and partitioning coefficients, volatility, and solubility),
    - expectations for performance in terms of trade-offs in levels of cleanup, cost, and environmental footprint, including remediation.
- In all cases, confirmation testing of the method's applicability for the local water must be conducted to validate performance and to estimate ongoing costs of operation (i.e., pilot testing must occur before design).
- Each type of system depends on selective binding or exclusion and does not destroy PFAS. Therefore, all technologies are likely to require further treatment of concentrated streams or saturated absorptive media. Actual PFAS destruction (or long-term storage) will bring additional operating costs to the PFAS treatment systems.
- Both capital and operating costs for each type of system will depend on what PFAS the Commonwealth elects to manage and what MCLs are established for those species. In general, both capital and operating costs for all systems increase as acceptable "breakthrough" concentrations of contaminants are reduced. However, some of these cost increases become nonlinear, particularly for so-called 'short-chain' PFAS, which may eliminate some treatment technologies from consideration if very low MCLs are established for those specific PFAS.
- Because MCLs for PFAS have not yet been established in Virginia, capital and operating costs can't reliably be estimated at this time.
- Similarly, all treatment systems also have an environmental footprint that also increases as acceptable "breakthrough" concentrations are reduced.
- Therefore, careful consideration of tradeoffs is needed to best serve the public.

It is important to stress that these commercial water treatment technologies are not PFAS destruction technologies. Rather, they act by sequestering PFAS onto substrates or into concentrated aqueous streams which must be further managed, with additional cost and environmental impacts.

The Interstate Technology and Regulatory Council (ITRC) is a state-led consortium of environmental professionals with public and private sector members from all 50 states and the District of Columbia, dedicated to reducing barriers to the use of innovative environmental technologies and processes. They have prepared PFAS fact sheets, including Fact Sheet 12 on Treatment Technologies, which goes into more detail on both treatment and destruction of PFAS in various media. *See* https://pfas-1.itrcweb.org/12-treatment-technologies/

3.7.1 Granular Activated Carbon

Granular activated carbon (GAC) has been used historically in water treatment processes to reduce or remove various contaminants and is the most studied treatment for PFAS removal (EPA 2018). Activated carbon is typically used for its highly porous structure as well as its large surface area for contaminants to attach (ITRC 2020). Activated carbon is made from organic materials with high carbon contents typically in a granular form: wood, lignite, coal. Removing PFAS from the water via GAC uses a physical mass transfer process from the aqueous phase onto solid media and does not use or involve chemical degradation or transformation (ITRC 2020). In this treatment process, water is taken from the source and directed through the treatment system where adsorption occurs. Figure 3 shows a standard GAC treatment process.

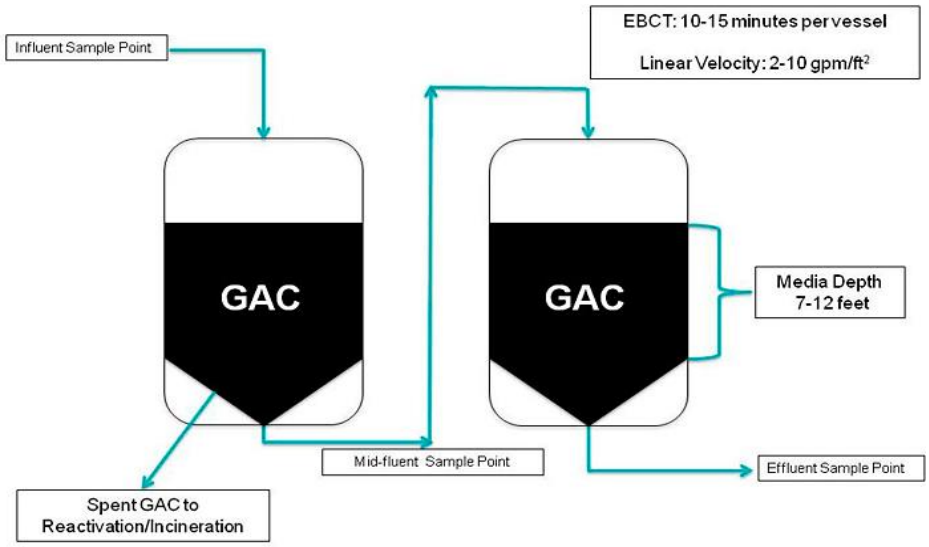


Figure 3. Typical GAC treatment system process flow diagram.

Source: Calgon Carbon Corporation (within ITRC 2020, PFAS – Per- and Polyfluoroalkyl Substances Treatment Technologies)

GAC has been analyzed and examined in several pilot and field studies. Peer reviewed literature studies confirm that perfluorinated sulfonates are more readily adsorbed than perfluoroalkyl acids. Long-chain PFAS are more readily absorbed than shorted chain PFAS, and the presence of competing co-contaminants can harm performance.

The EPA Drinking Water Treatability Database (https://www.epa.gov/water-research/drinking-water-treatability-database-tdb) reported that GAC is effective in removing:

- Up to greater than 99 percent of PFBA, PFBS, PFHpA, PFNA, PFHxS, and PFHpS (perfluoroheptanesulfonic acid) and
- 96 percent of PFNS from water.

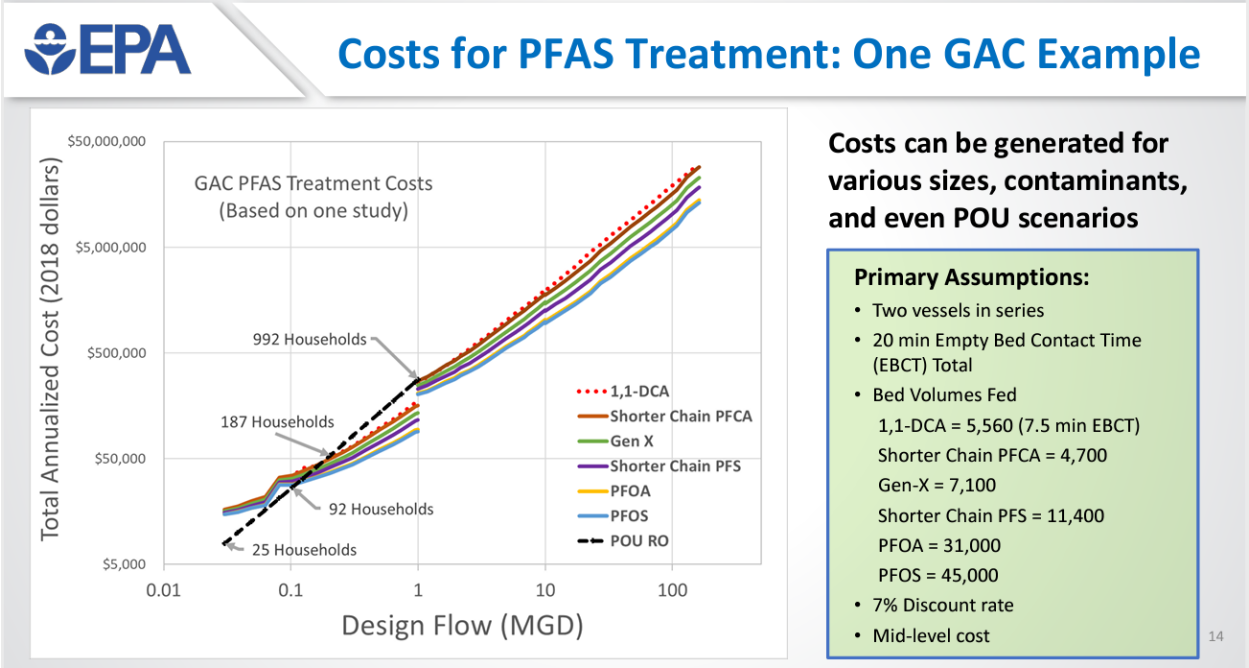
Some disadvantages and unintended environmental consequences of GAC include:

- Not being as effective for short-chain PFAS surfactants (as long-chain homologues), particularly at low ppt target concentrations;
- Volatile organic compounds (VOCs), inorganic minerals (particularly sulfonates and other functional groups that are similar to those on the targeted PFAS), and organic carbon contaminants are known to reduce removal efficiency and bed lifetimes for PFAS removal.
- The adsorption mechanism is not selective for PFAS surfactants and GAC can become loaded with other contaminants, reducing effectiveness for PFAS removal;
- There is modest overall capacity and slow kinetics for physical impurity adsorption compared with other technologies, which means larger physical site footprint and increased handling of contaminated GAC; and
- Production of GAC requires considerable energy input resulting in a large greenhouse gas (GHG) footprint for the material. Regeneration requires significant energy input and results in the loss of about 15% of the GAC to CO_2 emissions.

GAC is scalable for waterworks serving small populations. GAC systems have been successfully installed and operated at facilities ranging from large drinking water treatment facilities to household water treatment systems.

Cost Considerations and Factors

- Factors that affect Life Cycle costs include:
    - The energy intensity and energy source for operating the GAC facilities (e.g. pump systems and maintenance operations).
    - Greenhouse gas generation and heavy metal scrubbing operations needed to produce GAC from the source carbon (bitumen-based GACs are generally most efficient for PFAS removal).
    - The greenhouse gases generated to achieve GAC regeneration temperatures in kilns as well as the ~15% of GAC that is converted to CO2 during the regeneration operations.
- Factors impacting replacement and regeneration include
    - Input concentrations of PFAS to be removed and acceptable effluent levels to be achieved.
    - Co-contaminants and pre-treatment options installed ahead of the GAC system.
- Affordability. EPA provides estimated mid-level annual treatment costs in webinars on PFAS removal:



Powdered activated carbon (PAC) is a variant of GAC, in which the carbon granules are crushed to very fine particles, and fed to assist in a coagulation-settling-filtration process. The PAC absorbs the PFAS in the same way as in a GAC application, and can be very effective in removing some PFAS (with the same performance characteristics and limitations as GAC). The major drawbacks of PAC applications include:

- The settled sludge may contain significant levels of PFAS, which limits the potential disposal methods for those solids;
- PAC can't be regenerated, so it is a single-use option;
- PAC is generally applied for PFAS removal as a short-term bridge, while a longer-term permanent solution is developed and implemented; and
- The same Green House Gas and energy disadvantages as GAC.

3.7.2 Ion Exchange

GAC, IX and RO are three major treatment technologies for PFAS removal.

Ion exchange treatment (IX) is another treatment technology capable of removing PFAS from water. The resin beads used in IX consist of highly porous, polymeric materials which are acid, base, and water-insoluble, and are made from hydrocarbons. (EPA 2018). The IX resins are grouped into two groups, cationic and anionic, each serving a different purpose: cationic exchange resins (CER) remove positively charged contaminants, while anion exchange resins (AER) more effectively remove negatively charged contaminants, including PFAS (EPA 2018; ITRC 2020). The Ion

Exchange resins physically absorb charged contaminants through a combination of electrostatics and van der Waals forces. IX only works on charged PFAS.

There are two resin options for the treatment process, single-use or regenerable resins.

- *Single-use resins* are used until breakthrough, then removed and disposed of by high-temperature incineration or landfilling.
- *Regenerable resins* are used until breakthrough, then regenerated on-site with a specific solution to return resin to full exchange capacity.

Removing PFAS by ion exchange is a physical mass transfer process, similar to GAC, and does not involve chemical degradation or transformation (ITRC 2020). AER resins remove PFAS by forming ionic bonds with the sulfonic and carboxylic acid heads of PFOS and PFOA, while simultaneously the hydrophobic end of the PFAS structures adsorb onto the hydrophobic surfaces of the IX resins (ITRC 2020). Figure 4 shows a standard single-use resin ion exchange process.

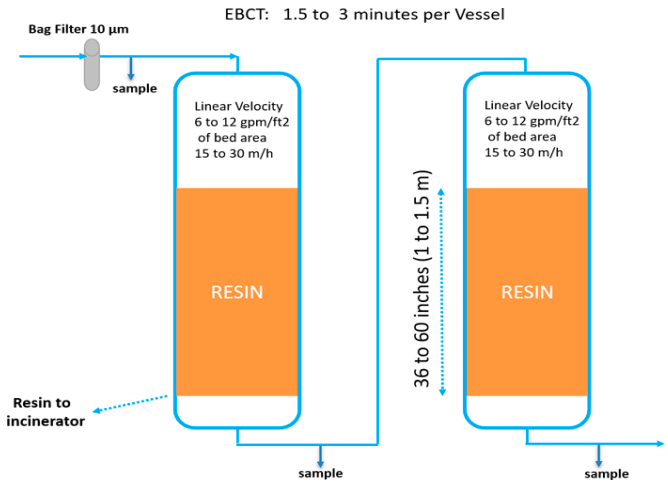


Figure 4. Single-use IX process flow diagram.

Source: Purolite Corp (from ITRC 2020 Per- and Polyfluoroalkyl Substances Treatment Technologies)

While not as commonly used as GAC, ion exchange technology for PFAS removal is well established. Pilot and field studies have shown that single-use resin has a higher removal capacity than regenerable resin, and is more fully exhausted in a lead-lag vessel. In addition, IX treatment systems typically have a smaller physical footprint than GAC treatment systems. However, literature shows that the relative efficiency of single-use and regenerable resin depends upon PFAS and co-contaminant influent concentrations and treatment goals.

The EPA Drinking Water Treatability Database for IX reports similar findings to GAC, but includes additional PFAS:

- Up to 90 percent removal of PFPeA;

- Up to 90 percent removal of PFPeS (perfluoropentanesulfonic acid);
- Up to greater than 99 percent removal of PFHxA; and
- Up to 97 percent removal of PFDA (perfluorodecanoic acid).

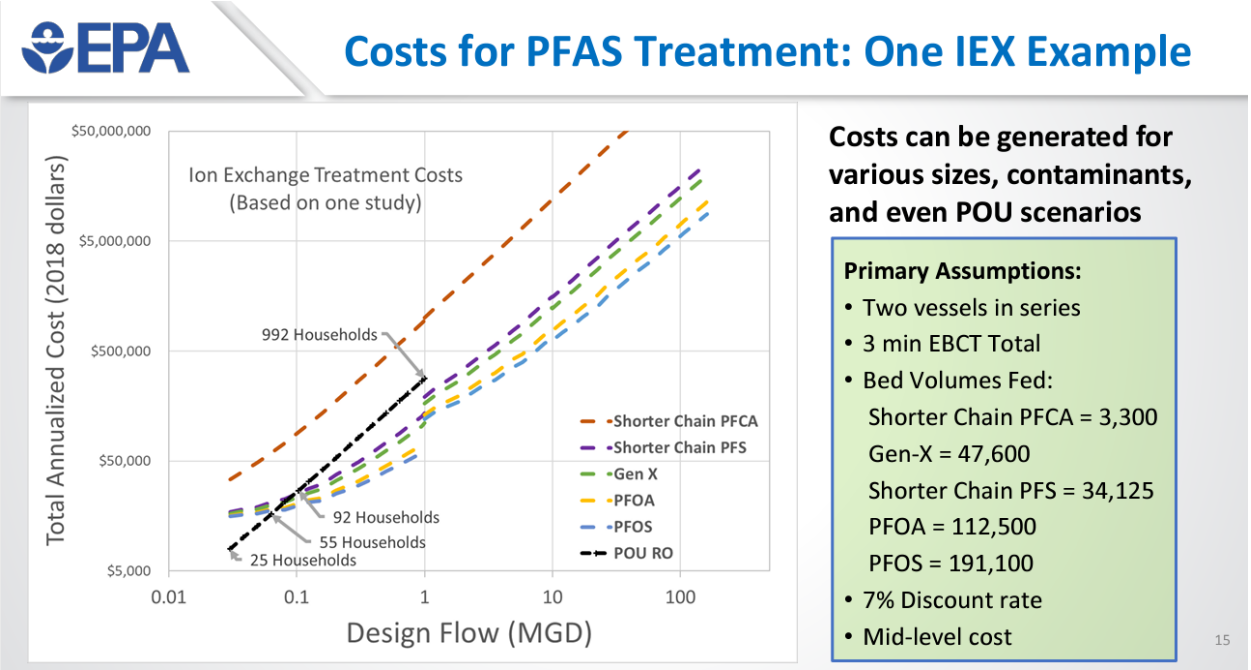
Some disadvantages of IX include generally being more expensive than GAC for long-chain PFAS removal if not regenerated; short-chain PFAS removal has been reported to be decreased by elevated chloride concentrations; the technology is selective for PFAS and generally does not remove uncharged co-contaminants; and, depending on the displace counter ion (e.g., chloride), it may require attention to corrosion control. Unintended environmental consequences, including the regenerative process, generally involve lower costs but will lead to concentrated PFAS waste solutions in need of disposal; demonstrated recycle technologies require energy (greenhouse gas emissions) for distillation processes; and non-regenerative processes will require high temperature incineration of resin-bound PFAS resulting in greenhouse gas emissions (about 5 – 10% that of GAC regeneration).

Ion exchange is scalable for waterworks serving small populations. Ion exchange systems have been successfully installed and operated at facilities ranging from large drinking water treatment facilities to household water treatment systems. In fact, the use of ion exchange tanks packed with cation exchange resins is commonly used at the household level for removal of 'hardness minerals' (generally calcium and magnesium) as well as other common cations (e.g. iron and lead salts).

Cost Considerations and Factors

- Factors that affect Life Cycle costs include:
    - The energy intensity and energy source for operating the IX facilities (e.g. pump systems and maintenance operations) which are generally lower than for GAC systems.
    - The technology employed to manufacture IX resins for use in IX systems. Many resins are based on functionalized polystyrene beads which have characteristic life cycle costs in water, solid waste and air emissions, including greenhouse gases.
    - The largest factor governing life cycle costs of IX systems surrounds the decision around single use vs regenerative resin systems. Single use systems will require incineration and resulting greenhouse gas footprints of ~15% that for GAC regeneration. Regeneration and recycle greenhouse gas penalties will depend on how the concentrated aqueous PFAS stream is handled (e.g. further concentration and incineration vs absorption and landfilling).
- Factors impacting replacement and regeneration include:
    - Input concentrations of PFAS surfactants to be removed and acceptable effluent levels to be achieved.
    - Co-contaminants and pre-treatment options installed ahead of the IX system. For example, hybrid GAC pre-treatment followed by IX 'finishing' a drinking water stream might be most cost, energy, and greenhouse gas effective for removing multiple PFAS surfactant anions from a drinking water stream.

Affordability. EPA provides estimated mid-level annual treatment costs in webinars on PFAS removal:



3.7.3 Membrane Separation (Reverse Osmosis)

Reverse Osmosis (RO) membranes have been used for both wastewater and drinking water purification for decades in both industrial/municipal and household systems. PFAS removal using membranes in processes such as reverse osmosis is effective. Reverse osmosis removes PFAS by pushing highly pressurized water through a semipermeable membrane (ITRC 2020). These membranes remove most organic and inorganic compounds, and new technology has increased efficiency while lowering operating pressures and costs. However, waste discharge (the concentrate or reject stream) from the reverse osmosis process will have concentrated levels of the various PFAS removed from the feed water, making disposal difficult (Appleman, 2014).

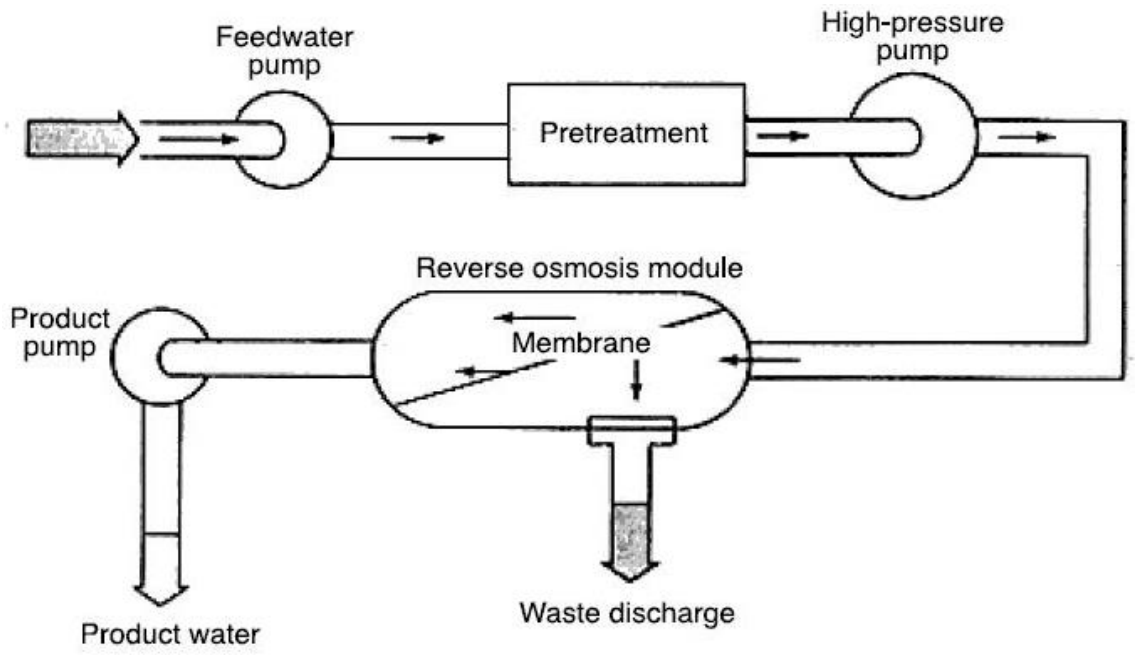


Figure 5. Reverse osmosis plant simple process diagram.

Source: RO Water Treatment Plant. Accessed from https://www.thewatertreatments.com/water-treatment-filtration/reverse-osmosis-plant-ro-desalination/

Treated water passes through the membrane, and then the rejected water is collected for disposal or discharge. See Figure 5.

Reverse osmosis has been combined with nanofiltration to increase PFAS removal. (ITRC 2020). Nanofiltration provides high water flux at lower operating pressures, and combining it with reverse osmosis utilizes properties of both. Nanofiltration alone will not achieve PFAS removal equivalent to reverse osmosis.

Removal of PFAS using RO membrane separation is extremely effective. Pilot and field studies support that RO membranes achieved PFAS removals up to greater than 99 percent.

The EPA Drinking Water Treatability Database (TDB) reports similar findings to GAC and IX. *See* https://www.epa.gov/water-research/drinking-water-treatability-database-tdb.

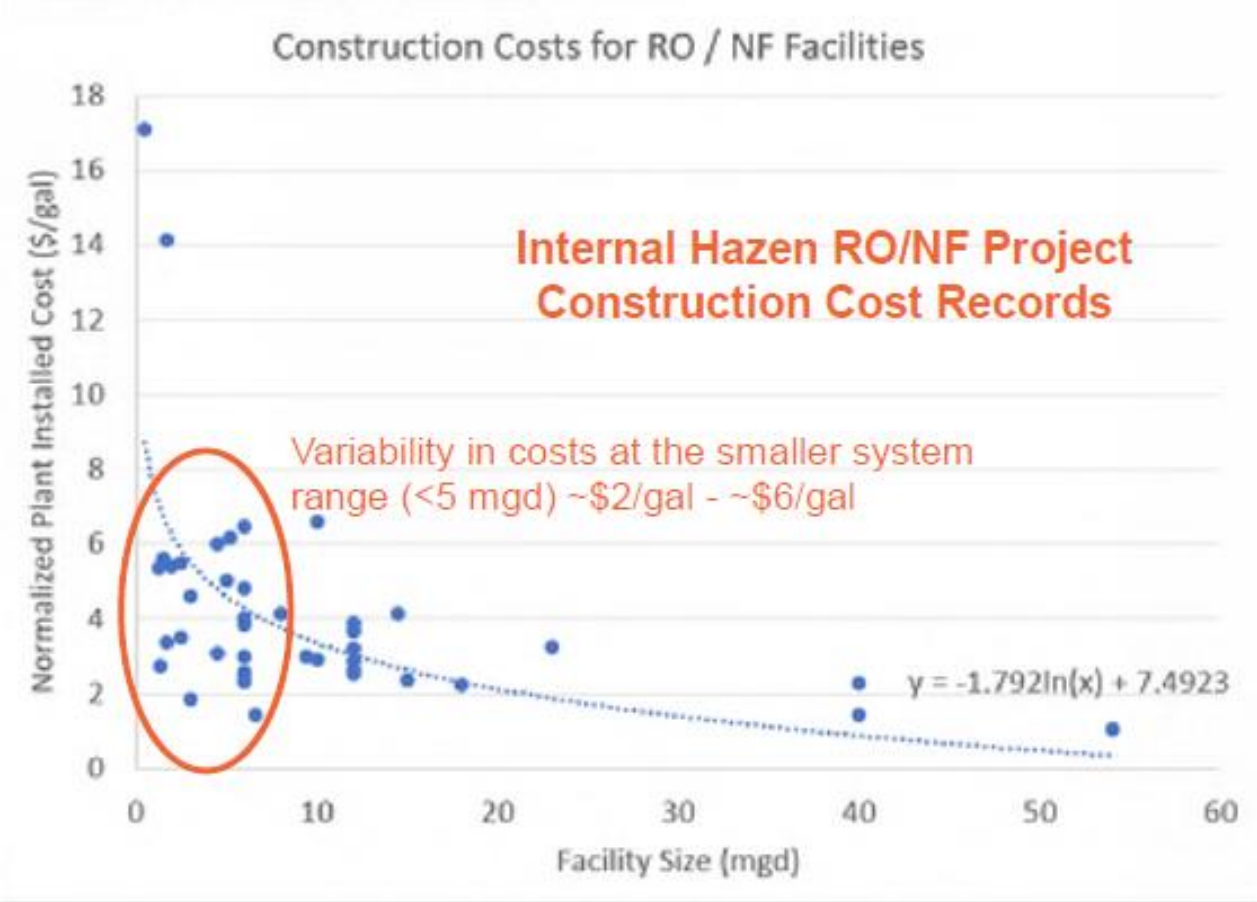
RO systems have been successfully installed and operated at facilities ranging from large sea water desalination facilities to household water treatment systems. In fact, among the most rapidly growing applications of RO technology have been house- or apartment-complex-size units for personal water consumption in China and India.

Cost Considerations and Factors

- Factors that affect Life Cycle costs include:

    - The technology employed to manufacture RO membranes, combined with their long useful life lead to life cycle costs in water, solid waste and air emissions, including greenhouse gases, well below those for IX resin and GAC production.
    - The energy intensity and energy source for operating the RO facilities (e.g. pump systems and maintenance operations) which are generally 2 – 4X those of IX and GAC systems.
    - The largest factor governing life cycle costs of RO systems surrounds the decision around how to manage the large effluent process (reject) stream, which is 10 – 20% of the size of the input water stream and only modestly more concentrated (5 – 10X) in water-soluble PFAS surfactants. As this is a very large stream, the life cycle costs in either greenhouse gases or wastewater are very large, dwarfing those of GAC and IX systems.
- Factors impacting replacement and regeneration include:
    - Input concentrations of PFAS to be removed and acceptable effluent levels to be achieved.
    - Co-contaminants and pre-treatment options installed ahead of the RO system which will dictate the timeline for chemical cleaning and backwashing membranes to reduce fouling. To minimize downtime and permanent fouling ultrafiltration membranes are often used to pre-treat input streams to RO membranes.

Affordability. EPA methodologies can (and should) be used to estimate costs of RO systems for comparisons with other alternatives. However, the PFAS Workgroup only found published EPA cost comparisons for point-of-use 'household' systems. In these cases, the effluent streams are expected to flow to wastewater treatment facilities or septic systems. A consulting firm (Hazen) did provide the PFAS Workgroup a description of the cost in removing water-soluble PFAS from a treatment system they designed. Their construction-cost estimates are shown below with operating costs only estimated for a single, 10 million gallon per day water treatment plant in Alabama (where annual operating and maintenance costs were estimated at $2.7 million, compared with $0.4 Million for IX and $0.65 Million for GAC treatment of that specific stream.



3.7.4 Treatment Costs

EPA has compiled work breakdown structure-based models on the cost of adding granular activated carbon treatment, anion exchange treatment, and reverse osmosis treatment to drinking water facilities. While these models and documents are free to the public, they are not specific to PFAS or any other pollutant. The EPA also supplies Excel templates in which treatment facility specifications can be entered to determine the cost of adding said technology to the specific plant. All of the above information can be viewed and retrieved from the Drinking Water Treatment Technology Unit Cost Models at https://www.epa.gov/sdwa/drinking-water-treatment-technology-unit-cost-models.

3.8 Health & Toxicology

The widespread use of PFAS in consumer products and its stability in the environment has resulted in PFAS being identified in the U.S. general population biomonitoring studies as early as 1999. (CDC 2021). Epidemiological studies, where PFAS were found in the environment and drinking water at relevant exposure levels, have examined possible relationships between levels of PFAS in blood and harmful health effects in people. Research involving humans suggests that high levels of certain PFAS may lead to the following: decrease in birth weight, decreased

vaccine response in children, increased risk of high blood pressure or pre-eclampsia in pregnant women, increased cholesterol levels, changes in liver enzymes, and increased risk of kidney or testicular cancer. (ATSDR, 2021).

PFOA and PFOS were voluntarily phased out by its primary manufacturer and eight other major companies from global production starting in 2006. (EPA 2006a; EPA 2021b). These efforts and an increase in public awareness have resulted in a steady decline of some PFAS in the U. S. population according to recent biomonitoring studies. (CDC 2021). After the phase out of PFOA, PFOS, and PFOA-related chemicals, other perfluoroalkyl substances have been developed or brought in as replacements for PFAS. Replacements include using non fluorinated chemicals, alternate technologies, and shorter chain PFAS. (ITRC 2020). However, several studies published show that replacement PFAS may not be less hazardous than the traditionally used long-chain PFAS. One of these replacement compounds is HFPO-DA, which is commonly known as GenX. GenX is used as a replacement for PFOA, and since its usage, the EPA has completed a Toxicity Assessment that can be found at https://www.epa.gov/pfas/genx-toxicity-assessments-documents.

The persistent, bio-accumulative, and toxic nature of PFAS is unique among organic drinking water contaminants, causing concern about potential toxicological effects in humans. Possible exposure routes include ingestion, inhalation, and dermal absorption. The consumption of PFAS from drinking water is of increasing concern in the United States, as well as worldwide, because of their widespread detection in public water systems and private domestic wells. (U.S. EPA, 2021b). Infants are a sensitive subpopulation for the adverse effects of PFAS. Their exposures from contaminated water, either from prepared formula or via maternal transfer to breast milk, are much higher than in older individuals. (Post et al. 2017; Goeden et al. 2019).

The Health and Toxicology subgroup researched and evaluated animal and epidemiological studies and risk assessments for PFAS in drinking water, including PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS. Old Dominion University's literature review, which can be found in Appendix 5, presents a table that summarizes current evidence on cancer, immunotoxicity, reproductive toxicity, developmental toxicity, and liver toxicity associated with exposures to PFAS.

3.8.1 Reference Doses

Nine (9) states – California, New Jersey, New Hampshire, New York, Michigan, Washington, Minnesota, Vermont, and Massachusetts – have developed reference doses for PFOA and PFOS based on findings from animal studies. State reference doses for PFOA range from 1.5 to 18 nanograms per kilogram per day (ng/kg/d), while the EPA reference dose is 20 ng/kg/d. State reference doses for PFOS range from 1.8 to 5 ng/kg/d. Toxicological information about other PFAS is more limited. Details on how the states developed reference doses are found in the literature review from Old Dominion University (Appendix 5).

Nine states – California, New Jersey, New Hampshire, New York, Michigan, Washington, Minnesota, Vermont, and Massachusetts – have developed reference doses for PFOA and PFOS based on findings from animal studies.

3.8.2 Relative Source Contribution and Exposure Factors

The EPA default value for relative source contribution of PFAS in drinking water, as a portion of the overall daily dose, is 20%. In the absence of data to the contrary, this is the value that the Board of Health should consider in establishing MCLs as directed by HB1257.

With respect to exposure factors, in the past, EPA has used 70 kilograms (kg) for the body weight, and 2 liters (L) as the amount of drinking water consumed daily. Several states with PFAS MCLs still use these values. Current exposure factors used in assessing drinking water risk are 80 kg body weight and 2.4 L of water ingested daily. Mathematically, the difference between the new and old values is 0.03 L/kg-day and 0.028 L/kg-day, respectively, which is negligible. Many states with PFAS MCLs use the model for lactating women, which was developed by Minnesota and relies on the breastfed infant as the sensitive receptor.

3.8.3 Toxicological Information Underpinning the Development of MCLs for PFAS

The minimum risk levels (MRLs) in the Agency for Toxic Substances Disease Registry's (ATSDR) final Toxicological Profile for Perfluoroalkyls (ATSDR, 2021), which includes PFOS, PFOA, PFHxS, and PFNA, are meant as screening values and are not the same thing as MCLs. While other states have MCLs for PFBA, PFHpA, PFHxS, and PFNA, no other state has an MCL for PFBA.

Risk assessment metrics (critical study, uncertainty factors, point of departure, human equivalent dose, and relative source contribution) used by each state can be found in tables included in Old Dominion University's literature review (Appendix 5).

States with MCLs for PFOA and PFOS include Massachusetts, Michigan, New Hampshire, New Jersey, New York, and Vermont. Michigan has the lowest MCL for PFOA, 8 ppt, and New York has the lowest MCL for PFOS, 10 ppt. Massachusetts and Vermont have the highest MCLs, 20 ppt, because their MCL is the sum of six (6) and five (5) PFAS, respectively, so technically

PFOA or PFOS in drinking water in those states can be as high as 20 ppt if one is present but no other PFAS are detected.

All states used an animal study to develop their reference doses for PFOA and PFOS. For PFOA, Massachusetts and Vermont used a 2006 developmental study by Lau et. al (Lau, 2006); Michigan used a 2011 developmental study by Onishchenko et. al (Onishchenko, 2011) and a 2016 developmental study by Koskela et al. (Koskela, 2016); New York used a 2011 study by Macon et al. that reported increased liver weight (Macon, 2011); and New Jersey and New Hampshire used a 2006 study by Loveless et. al., that also evaluated increased liver weight (Loveless (2006). EPA used the 2006 study by Lau et al. and ATSDR used the 2016 study by Koskela et al. to develop a minimal risk level for PFOA. ATSDR is also cited by states as using the 2011 study by Onishchenko in conjunction with the Koskela study.

For PFOS, states either used a 2009 immune response study by Dong et al. (Dong, 2009), or a 2005 two-generation reproduction study by Luebker et al. New Hampshire also used a 2011 immune response study by Dong et al. Additionally, EPA and ATSDR used the Luebker et al. 2005 study to develop a reference dose and a minimal risk level, respectively.

PFHpA was included in MCLs developed by Massachusetts (sum of 6 PFAS not to exceed 20 ppt) and Vermont (sum of 5 PFAS not to exceed 20 ppt). No states have developed an MCL for PFHpA based on toxicity. Rather, states concluded that it would be equipotent to PFOA based on structural similarity.

Four (4) states have MCLs for PFHxS: Massachusetts (sum of 6 PFAS not to exceed 20 ppt), Vermont (sum of 5 PFAS not to exceed 20 ppt), Michigan (51 ppt) and New Hampshire (18 ppt). Massachusetts concluded that structural similarity between PFHxS, PFOS, and PFOA justified assigning a toxicity value to PFHxS similar to PFOS and PFOA. Michigan used a 2018 National Toxicology Program report that evaluated PFHxS effect on the thyroxin (T4) levels. Vermont also considered thyroid toxicity and behavioral outcomes in developing their MCL. New Hampshire used a study by Chang that evaluated effects on reproduction. (Chang, 2018).

Five (5) states had an MCL for PFNA. Michigan used a 2015 developmental study by Das. (Das, 2015). Massachusetts also considered the Das study and concluded that PFNA toxicity would be similar to PFOS and PFOA and could be "additive" resulting in an MCL that is 20 ppt for the sum of six (6) PFAS. New Jersey and New Hampshire used 2015 liver weight data from the Das study to develop their MCL. Vermont used changes in liver weight and developmental toxicity to develop their MCL for PFNA, which is the sum of five PFAS not to exceed 20 ppt.

3.9 Establishing Regulatory Limits on PFAS – EPA

In May 2016, soon after the conclusion of the UCMR3 sampling, EPA issued a Lifetime Health Advisory (LHA) for levels of two specific PFAS in drinking water; PFOA and PFOS at 70 ppt, either individually or combined. Per EPA guidelines, Virginia uses 70 ppt as the LHA for PFOA and PFOS in drinking water. The announcement of EPA's LHA, along with high-profile news reporting on PFAS contamination sites such as those in Parkersburg, West Virginia,

Minneapolis-St. Paul, Minnesota, Portsmouth, New Hampshire, and Hoosick Falls, New York, caused many states to evaluate the PFAS levels detected in their public water systems and consider how best to address the possibility of contamination of public and private drinking water supplies. Hence, many state drinking water programs or environmental protection agencies began to address PFAS. In May 2018, EPA hosted a National Leadership Summit on PFAS. As a follow-up to the many concerns raised by states and stakeholder groups, EPA held Regional Community Engagement events in communities impacted by PFAS in drinking water and committed to prepare an action plan to address PFAS nationwide.

The March 3, 2021 Federal Register (86 FR 12272) included notice that EPA is making final determinations to regulate PFOS and PFOA, in drinking water and to not regulate six (6) contaminants (1,1-dichloroethane, acetochlor, methyl bromide (bromomethane), metolachlor, nitrobenzene, and RDX). With the final Regulatory Determinations for PFOA and PFOS, EPA will move forward to implement the national primary drinking water regulation development process for these two PFAS. In the PFAS Strategic Roadmap (Appendix 2), EPA stated the agency expects to issue a proposed regulation in Fall 2022 (before the statutory deadline of March 2023) and a final regulation in Fall 2023. In addition, EPA has re-proposed the Fifth Unregulated Contaminant Monitoring Rule (UCMR 5) (86 FR 13846, March 11, 2021) to collect new data on PFAS in drinking water. As proposed, UCMR 5 would provide new data to improve EPA's understanding of the frequency that 29 PFAS are found in the nation's drinking water systems and at what levels. All public water systems serving more than 3,300 customers and an additional 800 representative small systems will participate in the sampling program if it goes forward as proposed.

With its decision to regulate PFOA and PFOS in drinking water, EPA will continue to follow the rulemaking process established in the SDWA. Information about the federal requirements for establishing an MCL or treatment technique is in Section 6.1 of the report required for HB1257.

3.10 Regulatory Limits on PFAS – States

As noted in the toxicology section above, several states have developed regulatory limits for PFOA, PFOS, and other PFAS that are lower than EPA's Lifetime Health Advisory of 70 ppt. These are summarized in Table 3.

Table 3. Limits on PFAS established by other states and EPA.

All specified quantities are ppt.

	CA	CT	Mass.	MI	MN	NH	NJ	NY	VT	Virginia	EPA
	Notification Level*	Action Level	MCL	MCL	Health Advisory	MCL	MCL	MCL	MCL	MCL	Lifetime Health Advisory
PFOA	5.1	✓	✓	8	35	12	14	10	✓	Study /estab.	✓
PFOS	6.5	✓	✓	16	15	15	13	10	✓	Study /estab.	✓
PFNA		✓	✓	6		11	13		✓	Study	
PFHxS		✓	✓	51	47	18			✓	Study	
PFHpA		✓	✓						✓	Study	
PFDA			✓								
PFBS				420	2,000						
PFHxA				400000							
Gen X				370							
PFBA					7,000					Study	
SUM		70	20						20		70

*California requires waterworks to take a source out of service if a chemical is present in drinking water at a concentration greater than the notification level – this is referred to as the "response level." For PFOA and PFOS, California has lowered the response levels from 70 ppt combined to 10 ppt for PFOA and 40 ppt for PFOS based on a running four-quarter average.

Check marks indicate which PFAS are included in a limit that is a sum of chemicals.

"Study" indicates the specific PFAS is included among those in HB586. "Study/estab." means that the Board of Health will be required to establish an MCL for PFOA and PFOS when the amendments to Code of Virginia § 32.1-169 become effective on January 1, 2022. The Board of Health may also consider establishing MCLs for other PFAS.

4. CONCLUSIONS

1) In October 2020, the State Health Commissioner convened a work group, the Virginia PFAS Workgroup (PFAS Workgroup), to study the occurrence of perfluorooctanoic acid (PFOA), perfluorooctane sulfonate (PFOS), perfluorobutyrate (PFBA), perfluoroheptanoic acid (PFHpA), perfluorohexane sulfonate (PFHxS), perfluorononanoic acid (PFNA), and other perfluoroalkyl and polyfluoroalkyl substances (PFAS), as deemed necessary, in the Commonwealth's public drinking water. Members of the PFAS Workgroup represent waterworks owners and operators, including owners and operators of community waterworks, private companies that operate waterworks, advocacy groups representing owners and operators of waterworks, consumers of public drinking water, a manufacturer with chemistry experience, and other stakeholders.

2) The Office of Drinking Water (ODW), a functional unit within the Virginia Department of Health (VDH), provided administrative and technical support for the PFAS Workgroup from its formation through the development and submission of this report.

3) ODW, in conjunction with the PFAS Workgroup, designed and implemented the Sample Study to prioritize sites for measuring PFAS concentrations in drinking water and major sources of water and generate statewide occurrence data, subject to the limitations in 2020 Acts of Assembly Chapter 611 (HB586) and the state budget. Because of the limitations, the PFAS Sampling and Monitoring Study in Virginia (Sample Study) was biased to focus on the following:

a. Waterworks serving the largest number of consumers (the 17 largest waterworks in the state, which provide water to approximately 4.5 million consumers);

b. Waterworks that may be impacted by potential sources of PFAS contamination (11 waterworks that use groundwater as their water source and have a well or wells within 1 mile of an unlined landfill or major airport, which are potential sources of PFAS contamination); and

c. Waterworks with a surface water source that are downstream of a potential source of PFAS contamination, excluding the 17 largest waterworks in the state (23 waterworks).

d. 45 waterworks agreed to participate in the Sample Study. Only five (5) of the waterworks use groundwater as their water source.

4) Personnel at the 45 waterworks that participated in the Sample Study collected a total of 63 water samples between May and August 2021 and submitted them to a laboratory under contract with ODW to perform analysis for 25 specific PFAS. The six (6) PFAS specified in HB586 were among the 25.

5) Following quality assurance/quality control (QA/QC) review of the sample reports by ODW staff, ODW requested resampling at 4 locations because of QA/QC issues.

6) Samples from 48 of the 63 sample locations did not contain any PFAS above the practical quantitation level (PQL). The PQL is the lowest level that can be reliably measured within specified limits of precision and accuracy during routine laboratory conditions. This means PFAS were either not present in the samples, or that the concentration was so low, in most cases less than 3.5 parts per trillion (ppt), that it could not be reliably measured.

7) Samples from 15 of the 63 sample locations had at least one PFAS in a concentration above the PQL. Ten (10) samples came from waterworks in the Northern Virginia region. The major sources of water for these waterworks include the Potomac River and Occoquan Reservoir. All of the samples were finished water, meaning that it had gone through treatment and was ready for distribution to consumers.

8) Only one waterworks outside of the Northern Virginia area had results indicating more than one PFAS was present in its finished water in quantities above the PQL. Three other waterworks in Southwest Virginia had results indicating one PFAS was present. Results from two of these waterworks identified the presence of hexafluoropropylene oxide-dimer acid (HFPO-DA), which is commonly known as GenX. The highest detected concentration of a compound found during the Sample Study was 54 ppt of GenX.

9) Testing did not determine the source of PFAS detected at any of the waterworks.

10) None of the PFAS were above the U.S. Environmental Protection Agency's (EPA) health advisory level of 70 ppt for PFOA and PFOS (individually or combined) and none exceeded any of the maximum contaminant levels (MCLs) established by other states.

11) The occurrence data indicates PFAS are present in drinking water in some localities in Virginia. However, there are more than 1,050 community waterworks in Virginia. The Sample Study was a one-time sampling event and was limited to 45 community waterworks. It was not a comprehensive evaluation of the extent or nature of PFAS contamination in public drinking water across the state.

12) Eight (8) states have established an MCL or other regulatory requirement for one or more PFAS in drinking water. All of the MCLs that states have established are more stringent (lower) than EPA's Health Advisory Level of 70 ppt for PFOA and PFOS. Another seven (7) states have established special state guidance (but not regulations) for PFAS in drinking water for screening and investigatory purposes. The guidance levels are similar to or lower than EPA's Health Advisory Level for PFOA and PFOS, or apply to other specific PFAS. Other states have conducted testing to determine if PFAS is present in drinking water, and, in some cases, to what extent. However, most states have not taken action to establish either regulatory limits or state guidance levels for PFAS, individually or as a class of compounds.

13) PFAS Workgroup members examined the approach and scientific research/methods other states relied on to establish MCLs for PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS. Based on the scientific research that is currently available, VDH and the Board of Health

could move forward to develop MCLs for two PFAS, PFOS and PFOA, given what other states have done to develop their own MCLs.

14) However, the PFAS Workgroup is not going to recommend any specific MCLs for PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS, to be included in the regulations of the Board of Health applicable to waterworks. The PFAS Workgroup cited a number of reasons for not recommending any MCLs:

a. The Health & Toxicology subgroup noted that there is sufficient toxicological data to support states' MCLs for PFOA and PFOS, at a minimum. However, there is insufficient existing toxicological data to support maximum contaminant level goals (MCLGs) and MCLs for other PFAS (PFBA, PFHpA, PFHxS, and PFNA) in Virginia drinking water.

b. The majority of community waterworks in Virginia are classified as "small," meaning they serve fewer than 3,300 persons. The Sample Study was not a comprehensive evaluation of the extent or nature of PFAS contamination in public drinking water across the state and did not, by design, consider how many small waterworks use water that contains PFAS, what level is present, if any, or what the implications would be for setting an MCL for one or more PFAS.

c. The Code of Virginia requires the Board of Health to consider protection of public health and the financial impact of regulations in the rulemaking process. *See* Code of Virginia §§ 2.2-4007.04 and 32.1-170. The SDWA also requires this evaluation. *See* 42 U.S.C. § 300g-1(b).

i. If the Board of Health establishes an MCL for any PFAS and subsequent monitoring at a waterworks showed that it needed to install treatment to comply with the MCL, the cost to install treatment, typically either granulated activated carbon, ion-exchange, or reverse osmosis, would likely exceed one million dollars. Due to the expense of treatment, small waterworks could most acutely experience the impact of establishing MCLs if PFAS were found above an established MCL because they have a smaller customer base amongst whom they can spread the cost of compliance.

ii. There are a number of questions about PFAS-contaminated residuals from treatment processes that will factor into the cost-benefit analysis, particularly when a source for the PFAS contamination is suspected or known and removal at the source can be accomplished and funded by the appropriate party (the polluter).

iii. VDH and the Board of Health need more occurrence data and time to make an informed, reasonable decision regarding the financial impact of MCLs for all of the specified contaminants.

iv. Additionally, more time, information, and expertise is needed to evaluate the toxicological data for PFAS, and determine appropriate MCL levels to protect public health.

d. EPA made a regulatory determination to establish MCLs for PFOA and PFOS. As a primacy state under the Safe Drinking Water Act, Virginia (i.e., the Board of Health) will have to adopt MCLs for these PFAS that are no less stringent than EPA's when they become effective. In the PFAS Strategic Roadmap (Appendix 2), EPA stated the agency expects to issue a proposed regulation in Fall 2022 (before the statutory deadline of March 2023) and a final regulation in Fall 2023.

e. Virginia has historically relied on EPA to establish standards for drinking water contaminants, then adopted those standards in the Board of Health's regulations for waterworks.

15) This report does not address possible PFAS impacts to private drinking water/private wells, which rely on groundwater sources.

16) The General Assembly appropriated $60,000 in fiscal year 2022 for VDH "to continue its study of the occurrence of perfluorooctanoic acid (PFOA), perfluorooctane sulfonate (PFOS), and other perfluoroalkyl and polyfluoroalkyl substances (PFAS) in the Commonwealth's public drinking water and to develop recommendations for specific maximum contaminant levels for PFOA, PFOS, and other PFAS for inclusion in regulations of the Board of Health applicable to waterworks." https://budget.lis.virginia.gov/amendment/2021/2/HB1800/Introduced/CR/307/2c/.

a. ODW's expenditures for the Sample Study that is the subject of this report totaled $90,000, which came from a fiscal year 2021 grant from EPA to study emerging contaminants. Project costs included contracting with a laboratory for sample kits, shipping, and analysis ($41,000); contracting with Old Dominion University to perform a literature review ($10,000), and salary for the State Toxicologist, who works for a different operation unit within VDH ($39,000).

b. ODW received another grant from EPA in fiscal year 2022 to study emerging contaminants and will be able to dedicate a portion of that grant (again, approximately $90,000) to study the occurrence of PFAS in public drinking water.

17) Appendix 6 contains a list of resources that provide additional information about PFAS generally, VDH contacts for technical support on PFAS in the public drinking water supply, resources from other states, and information about Virginia's Drinking Water State Revolving Fund (DWSRF) program. The DWSRF may be a funding source for waterworks interested in installing new treatment technologies.

5. RECOMMENDATIONS

Based on the results of the Sample Study, Literature Review, and efforts from the Subgroups, the PFAS Workgroup makes the following recommendations:

1) There is a significant need for additional PFAS occurrence data in Virginia drinking water and major sources of supply. The PFAS Workgroup recommends VDH and other agencies collect additional PFAS occurrence data.

a. The current monitoring study was limited to 45 waterworks out of approximately 2,811 waterworks in Virginia. While the study covered a majority of the population served by Virginia waterworks, and considered some sources with elevated risk of PFAS contamination, it left significant numbers of waterworks untested and the results were based on a single sampling event.

i. Waterworks that use groundwater were underrepresented in the Sample Study (five (5) waterworks out of approximately 2,400 groundwater waterworks in Virginia).

ii. Additional occurrence data will come from the U.S. Environmental Protection Agency's (EPA) Fifth Unregulated Contaminant Monitoring Rule (UCMR5). UCMR5 will provide data on the occurrence of 29 PFAS at all community waterworks serving 3,300 or more consumers and selected small waterworks (those serving 25 to 3,299 consumers) in Virginia. Nationwide, EPA will select a total of 800 small waterworks. However, the total number of "small" waterworks in Virginia that participate in UCMR5 testing (a statistical sampling) will be limited to approximately 20 to 25. (*See* Fifth Unregulated Contaminant Monitoring Rule, https://www.epa.gov/dwucmr/fifth-unregulated-contaminant-monitoring-rule). Sampling will occur from January 2023 through December 2025. Subject to the availability of appropriations and sufficient laboratory capacity, EPA will pay for testing (analytical costs) at all waterworks serving 10,000 or fewer consumers.

iii. HB586 named, and the PFAS Workgroup largely focused on, six (6)PFAS based on what compounds had been found or reported within Virginia through 2020. However, the Sample Study found hexafluoropropylene oxide-dimer acid (HFPO-DA), which is commonly known as GenX, at two locations, and it was the PFAS with the highest reported concentration level (57 ppt). The PFAS Workgroup and Literature Review did not compile information on GenX. Future testing, research, and regulatory discussions should consider GenX.

iv. Additional sampling and monitoring for PFAS is needed to broaden the scope of the sampling dataset, particularly for small waterworks, and ensure that a broader cross section of drinking water sources in Virginia, including

groundwater sources, will be monitored to determine if PFAS are present and, if so, in what quantities.

2) The next round of sampling should consider the following in the sample study design:

a. Develop a temporal data set of PFAS occurrence in drinking water. Due to limited resources, the current monitoring study was a single snapshot in time and did not address seasonal variations in water quality, possibly due to variations in industrial cycles, river flows, temperatures, traffic patterns, agricultural runoffs etc. Surface water sources are known to vary in water quality, depending on the season and weather. Additional samples over time, covering different seasons will provide a measure of how PFAS levels vary over time. EPA's UCMR5 will provide some additional temporal data about PFAS in Virginia drinking water as it calls for waterworks to collect four quarterly samples at surface water waterworks and two samples at groundwater waterworks. The sampling interval is intended to confirm that detections of PFAS are real, reliable and consistent.

b. Focus on community waterworks, then representative nontransient noncommunity waterworks (NTNC). Raise the priority for certain NTNC where there is a significant opportunity to protect public health because of the population served or, if PFAS are present, the extent of contamination.

c. Consider a hybrid approach for the sample study design, as opposed to random sampling. A hybrid approach could include potential risk or likelihood of finding PFAS in drinking water, location of waterworks/source water to potential sources of PFAS contamination, or other factors.

d. Based on the presence of PFAS in more samples from surface water sources than groundwater sources in the Sample Study, consider surface water and GUDI (groundwater under the direct influence of surface water) sources as higher risk of PFAS contamination than groundwater sources.

e. VDH should focus on entry point (finished water) sampling. The Virginia Department of Environmental Quality (DEQ), as the agency that implements EPA's Clean Water Act requirements, will focus on surface water and groundwater sources and potential sources of contamination.

f. Exclude consecutive waterworks from future sampling because the wholesale waterworks (i.e., those that supply finished drinking water) will have samples collected following treatment and there should not be a change in water quality at the consecutive waterworks.

g. Collect additional samples at all of the 15 waterworks that had PFAS > PQL during the Sample Study.

h. With a budget of $150,000 for fiscal year 2022 (consisting of a $60,000 appropriation from the General Assembly and $90,000 from an EPA grant to study emerging contaminants), VDH should continue its study of the occurrence of PFAS in public drinking water in Virginia. If analysis of drinking water samples is by EPA Method 533, based on a sample analysis cost of $175 to $300 per sample, the budget could allow for analysis of 500 to 857 samples. However, ODW estimates 25% of the samples will be used for field reagent blank samples and repeat samples. Therefore, ODW estimates 400 to 685 drinking water samples will be available under the sampling budget. This range falls far short of the total number of waterworks using surface water or groundwater under the direct influence of surface water (173 waterworks) plus the number of community waterworks using groundwater as their source water (715 waterworks).

3) The amount of funding and time for VDH and other state agencies such as DEQ to study the occurrence of PFAS in drinking water, drinking water sources, and potential sources of contamination will dictate the scope of additional sampling. As a result, the PFAS Workgroup recommends that the General Assembly consider funding additional resources at VDH and DEQ for enhanced sampling and more robust sample studies of drinking water, drinking water sources, and potential sources of contamination.

4) When VDH and the Board of Health initiate the rulemaking process to establish MCLs for PFOA and PFOS, the Commonwealth needs to provide resources (time, money, and staff) to the agency so the process can be effective. To comply with the Administrative Process Act, an MCL should be based on toxicology and take into consideration such things as treatment costs, impacts from moving PFAS from one media to another, incremental costs, and downstream effects. The rulemaking also needs to consider impacts on small waterworks, including treatment options, costs, and how to pay when treatment is or would be required.

a. VDH should develop a needs assessment to establish MCLs for PFOA and PFOS. This could include hiring toxicologists (possibly to form a panel), performing additional sampling, and conducting sufficient research to ensure there is a worthwhile rulemaking process.

b. Convene a panel of toxicologists to determine whether or not to regulate other PFAS ("as deemed necessary"). The Sample Study detected other PFAS outside of the 6 named in HB586.

5) VDH should include an analysis of environmental justice impacts that may flow from the promulgation of an MCL for any PFAS. The Commonwealth/VDH should also carefully assess whether and to what extent an MCL would improve protection of public health in communities already burdened by water, air and industrial pollution.

6) The regulatory landscape for PFAS in solid waste and other media continues to evolve. The PFAS Workgroup recommends that this be factored in when the treatment technologies available do not destroy the contaminant but rather move it from one media to another.

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APPENDIX 1 - PFAS Workgroup Members.

1. Community waterworks that serve more than 50,000 persons.	Chris Harbin/Jillian Terhune	Norfolk Department of Public Utilities
	David Jurgens	City of Chesapeake Department of Public Utilities
	Jamie Bain Hedges	Fairfax Water
	Mike Hotaling	Newport News
	Michael McEvoy	Western Virginia Water Authority
	Jessica Edwards-Brandt	Loudoun Water
	Christian Volk, Ph.D.	Virginia American Water
	Russ Navratil	Henrico County
2. Community waterworks that serve less than 50,000 persons.	John J. Aulbach, P.E. / Dan Hingley, P.E.	Aqua Virginia
	Mark Estes	Halifax County PSA
3. Community waterworks that serve less than 1,000 persons.	Wendy Eikenberry	Augusta County Service Authority
4. Advocacy group that represents waterworks in Virginia.	Geneva Hudgins / Russ Navratil	Virginia Section American Water Works Association
	Andrea W. Wortzel	Mission H20
	Steve Herzog	Virginia Water Environment Association
5. Chemical manufacturer with chemistry experience.	Stephen P. Risotto	American Chemistry Council
	Henry Bryndza, Ph.D.	DuPont (retired)
6. A consumer of public drinking water.	Dr. William Mann	
7. Non-governmental environmental organizations.	Anna Killius	James River Association
	Phillip Musegaas	Potomac Riverkeeper Network
	Michael Town / Christopher Leyen	Virginia League of Conservation Voters
8. The Virginia Department of Environmental Quality (DEQ).	Jeff Steers / Benjamin Holland	
9. A local health district.	Dr. Noelle Bissell	Director, New River Health District
The State Toxicologist	Dwight Flammia, Ph.D.	
ODW staff member	Robert Edelman, P.E.	Director, ODW Division of Technical Services
PFAS Workgroup leader	Tony Singh, Ph.D.	Deputy Director, ODW

APPENDIX 1 – Subgroup Members

Health and Toxicology Subgroup
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Mark Estes (Halifax County Service Authority)
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Benjamin Holland (Virginia Department of Environmental Quality)
David Jurgens (City of Chesapeake)
Chris Leyen (Virginia League of Conservation Voters)
Paul Nyffeler (Chem Law)
Erin Reilly (James River Association)
Steve Risotto (American Chemistry Council)
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Jillian Terhune (City of Norfolk)
Andrea Wortzel (Mission H2O)
William Mann, MD (Physician/Public)

Monitoring and Occurrence Subgroup
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Mark Estes (Halifax County Service Authority)
Dwight Flammia (State Toxicologist)
Jamie Bain Hedges (Fairfax Water)
Jack Hinshelwood (VDH ODW)
David Jurgens (City of Chesapeake)
Anna Killius (James River Association)
Mike McEvoy (Western Virginia Water Authority)
Tony Singh (VDH ODW)
Jeff Steers (Virginia Department of Environmental Quality)

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Wendy Eikenberry (Augusta County Service Authority)
Jamie Bain Hedges (Fairfax Water)
Mike McEvoy (Western Virginia Water Authority)
Phillip Musegaas (Potomac Riverkeeper Network)
Russ Navratil (Virginia Section, American Water Works Association)
Paul Nyffeler (Chem Law)
Steve Risotto (American Chemistry Council)

Jillian Terhune (City of Norfolk)
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Wendy Eikenberry (Augusta County Service Authority)
Mark Estes (Halifax County Service Authority)
Chris Harbin (City of Norfolk)
Jamie Bain Hedges (Fairfax Water)
Jack Hinshelwood (VDH ODW)
Mike Hotaling (Newport News Water Works)
Mike McEvoy (Western Virginia Water Authority)
Russ Navratil (Virginia Section American Water Works Association)
Kelly Ryan (Virginia American Water)

APPENDIX 2 – U.S. Environmental Protection Agency PFAS Strategic Roadmap: EPA's Commitments to Action 2021-2024

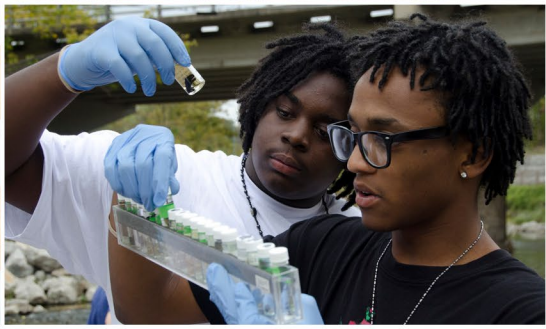
United States
Environmental Protection
Agency

PFAS Strategic Roadmap: EPA's Commitments to Action 2021–2024









A Note from EPA Administrator Michael S. Regan

For far too long, communities across the United States have been suffering from exposure to PFAS pollution. As the science has continued to develop, we know more now than ever about how PFAS build up in our bodies over long periods of time, and how they can cause adverse health effects that can devastate families. As Secretary of the North Carolina Department of Environmental Quality, I saw this devastation firsthand. For years, the Cape Fear River had been contaminated by these persistent "forever" chemicals. As I spoke with families and concerned citizens, I could feel their suffering and frustration with inaction. I knew my job was going to be trying and complex. But we were able to begin to address this pervasive problem by following the science, following the law, and bringing all stakeholders to the table.

As one of my earliest actions as EPA Administrator, I established the EPA Council on PFAS and charged it with developing an ambitious plan of action to further the science and research, to restrict these dangerous chemicals from getting into the environment, and to immediately move to remediate the problem in communities across the country. EPA's PFAS strategic roadmap is our plan to deliver tangible public health benefits to all people who are impacted by these chemicals—regardless of their zip code or the color of their skin.

Since I've been EPA Administrator, I have become acutely aware of the invaluable and central role EPA has in protecting public health in America. For more than 50 years, EPA has implemented and enforced laws that protect people from dangerous pollution in the air they breathe, the water they drink, and the land that forms the foundation of their communities. At the same time, my experience in North Carolina reinforced that EPA cannot solve these challenges alone. We can only make progress if we work in close collaboration with Tribes, states, localities, and stakeholders to enact solutions that follow the science and stand the test of time. To affect meaningful change, engagement, transparency, and accountability will be critical as we move forward.

This roadmap will not solve our PFAS challenges overnight. But it will turn the tide by harnessing the collective resources and authority across federal, Tribal, state, and local governments to empower meaningful action now.

I want to thank the co-chairs of the EPA Council on PFAS—Radhika Fox, Assistant Administrator for Water, and Deb Szaro, Acting Regional Administrator in Region 1—for their leadership in guiding the development of this strategy.

Let's get to work.

Administrator Michael S. Regan

PFAS Council Members

The following policy and technical leaders serve as members of the EPA Council on PFAS. They have been instrumental in working with their respective offices to develop the Agency's strategy. The Council will continue to coordinate across all EPA offices and Regions to accelerate progress on PFAS.

Co-Chairs

Radhika Fox, Assistant Administrator for Water

Deb Szaro, Acting Regional Administrator, Region 1

Office of the Administrator

John Lucey, Special Assistant to the Administrator

Andrea Drinkard, Senior Advisor to the Deputy Administrator

Office of Air and Radiation

John Shoaff, Director, Air Policy and Program Support

Office of Chemical Safety and Pollution Prevention

Jeffrey Dawson, Science Advisor

Tala Henry, Deputy Director, Pollution Prevention and Toxics

Office of Enforcement and Compliance Assurance

Cyndy Mackey, Director, Site Remediation Enforcement

Karin Leff, Director, Federal Facilities Enforcement

Office of General Counsel

Dawn Messier, Deputy Associate General Counsel, Water

Jen Lewis, Deputy Associate General Counsel, Solid Waste and Emergency Response

Office of Land and Emergency Management

Dana Stalcup, Deputy Director, Superfund Remediation and Technology Innovation

Dawn Banks, Director, Policy Analysis and Regulatory Management

Office of Research and Development

Tim Watkins, Acting Director, Center for Public Health and Environmental Assessment

Susan Burden, PFAS Executive Lead

Office of Water

Jennifer McLain, Director, Ground Water and Drinking Water

Deborah Nagle, Director, Science and Technology

Zachary Schafer, Senior Advisor to the Assistant Administrator

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Contents

Introduction

Harmful per- and poly-fluoroalkyl substances (PFAS) are an urgent public health and environmental issue facing communities across the United States. PFAS have been manufactured and used in a variety of industries in the United States and around the globe since the 1940s, and they are still being used today. Because of the duration and breadth of use, PFAS can be found in surface water, groundwater, soil, and air—from remote rural areas to densely-populated urban centers. A growing body of scientific evidence shows that exposure at certain levels to specific PFAS can adversely impact human health and other living things. Despite these concerns, PFAS are still used in a wide range of consumer products and industrial applications.

Every level of government—federal, Tribal, state, and local—needs to exercise increased and sustained leadership to accelerate progress to clean up PFAS contamination, prevent new contamination, and make game-changing breakthroughs in the scientific understanding of PFAS. The EPA Council on PFAS developed this strategic roadmap to lay out EPA's whole-of-agency approach to addressing PFAS. To deliver needed protections for the American people, the roadmap sets timelines by which the Agency plans to take specific actions during the first term of the Biden-Harris Administration. The strategic roadmap builds on and accelerates implementation of policy actions identified in the Agency's 2019 action plan and commits to bolder new policies to safeguard public health, protect the environment, and hold polluters accountable.

The risks posed by PFAS demand that the Agency attack the problem on multiple fronts at the same time. EPA must leverage the full range of statutory authorities to confront the human health and ecological risks of PFAS. The actions described in this document each represent important and meaningful steps to safeguard communities from PFAS contamination. Cumulatively, these actions will build upon one another and lead to more enduring and protective solutions.

EPA's integrated approach to PFAS is focused on three central directives:

- **Research.** Invest in research, development, and innovation to increase understanding of PFAS exposures and toxicities, human health and ecological effects, and effective interventions that incorporate the best available science.
- **Restrict.** Pursue a comprehensive approach to proactively prevent PFAS from entering air, land, and water at levels that can adversely impact human health and the environment.
- **Remediate.** Broaden and accelerate the cleanup of PFAS contamination to protect human health and ecological systems.

The Agency's Approach

EPA's approach is shaped by the unique challenges to addressing PFAS contamination. EPA cannot solve the problem of "forever chemicals" by tackling one route of exposure or one use at a time. Rather, EPA needs to take a lifecycle approach to PFAS in order to make meaningful progress. PFAS pollution is not a legacy issue—these chemicals remain in use in U.S. commerce. As such, EPA cannot focus solely on cleaning up the downstream impacts of PFAS pollution. The Agency needs to also look upstream to prevent new PFAS contamination from entering air, land, and water and exposing communities. As the Agency takes tangible actions both upstream and downstream, EPA will continue to pursue a rigorous scientific agenda to better characterize toxicities, understand exposure pathways, and identify new methods to avert and remediate PFAS pollution. As EPA learns more about the family of PFAS chemicals, the Agency can do more to protect public health and the environment. In all this work, EPA will seek to hold polluters accountable for the contamination they cause and ensure disadvantaged communities equitably benefit from solutions.

Consider the Lifecycle of PFAS

EPA will account for the full lifecycle of PFAS, their unique properties, the ubiquity of their uses, and the multiple pathways for exposure.

PFAS are a group of synthetic chemicals that continue to be released into the environment throughout the lifecycle of manufacturing, processing, distribution in commerce, use, and disposal. Each action in this cycle creates environmental contamination and human and ecological exposure. Exacerbating this challenge is that some PFAS persist in the environment. PFAS are synthesized for many different uses, ranging from firefighting foams, to coatings for clothes and furniture, to food contact substances. Many PFAS are also used in industrial processes and applications, such as in the manufacturing of other chemicals and products. PFAS can be released into the environment during manufacturing and processing as well as during industrial and commercial use. Products known to contain PFAS are regularly disposed of in landfills and by incineration, which can also lead to the release of PFAS. Many PFAS have unique properties that prevent their complete breakdown in the environment, which means that even removing PFAS from contaminated areas can create PFAS-contaminated waste. This is currently unregulated in most cases.

Get Upstream of the Problem

EPA will bring deeper focus to preventing PFAS from entering the environment in the first place—a foundational step to reducing the exposure and potential risks of future PFAS contamination.

Intervening at the beginning of the PFAS lifecycle—before they have entered the environment—is a foundational element of EPA's whole-of-agency approach. While hundreds of individual PFAS compounds are in production and use,[i] a relatively

modest number of industrial facilities produce PFAS feedstock,[ii] and a relatively narrow set of industries directly discharge PFAS into water or soil or generate air emissions in large quantities.[iii] This context helps to pinpoint clear opportunities to restrict releases into the environment. EPA will use its authorities to impose appropriate limitations on the introduction of new unsafe PFAS into commerce and will, as appropriate, use all available regulatory and permitting authorities to limit emissions and discharges from industrial facilities. This approach does not eliminate the need for remediation where releases and exposures have already occurred, but it is a critical step to preventing ongoing concentrated contamination of soil and surface and groundwaters.

Hold Polluters Accountable

EPA will seek to hold polluters and other responsible parties accountable for their actions and for PFAS remediation efforts.

Many communities and ecosystems are continuously exposed to PFAS in soil, surface water, groundwater, and air. Areas can be exposed due to their proximity to industrial sites, airports, military bases, land where biosolids containing PFAS have been applied, and other sites where PFAS have been produced or used and disposed of for specific and repeated purposes. When EPA becomes aware of a situation that poses a serious threat to human health or the environment, the Agency will take appropriate action. For other sites where contamination may have occurred, the presence of certain PFAS in these environments necessitates coordinated action to understand what specific PFAS have been released, locations where they are found, where they may be transported through air, soil, and water in the future, and what remediation is necessary. EPA will seek to hold polluters and other responsible parties accountable for their actions, ensuring that they assume responsibility for remediation efforts and prevent any future releases.

Ensure Science-Based Decision-Making

EPA will invest in scientific research to fill gaps in understanding of PFAS, to identify which additional PFAS may pose human health and ecological risks at which exposure levels, and to develop methods to test, measure, remove, and destroy them.

EPA's decisions regarding PFAS will be grounded in scientific evidence and analysis. The current body of scientific evidence clearly indicates that there are real, present, and significant hazards associated with specific PFAS, but significant gaps remain related to the impacts of other PFAS on human health and in the environment. Regulatory development, either at the state or federal level, would greatly benefit from a deeper scientific understanding of the exposure pathways, toxicities, and potential health impacts of less-studied PFAS. The federal government, states, industry, academia, and nonprofit organizations—with appropriate coordination and resources—have the capability to conduct this necessary research.

EPA is conducting new research to better understand the similar and different characteristics of specific PFAS and whether and how to address groups and categories of PFAS. The Agency is focused on improving its ability to address multiple chemicals at once, thereby accelerating the effectiveness of regulations, enforcement actions, and the tools and technologies needed to remove PFAS from air, land, and water.

To break the cycle of contamination and exposure from PFAS, additional research is needed to identify and/or develop techniques to permanently dispose of or destroy these durable compounds. Government agencies, industry, and private laboratories need tools and validated methods to measure PFAS in air, land, and water to identify pollution sources, demonstrate facility compliance, hold polluters accountable, and support communities during and after cleanups.

Prioritize Protection of Disadvantaged Communities

When taking action on PFAS, EPA will ensure that disadvantaged communities have equitable access to solutions.

Many known and potential sources of PFAS contamination (including military bases, airports, industrial facilities, and waste management and disposal sites) are near low-income communities and communities of color. EPA needs to ensure these affected populations have an opportunity to participate in and influence the Agency's decision-making. This may call for the Agency to seek out and facilitate the communities' engagement by providing culturally appropriate information and accommodations for people with Limited English Proficiency, facilitating community access to public meetings and comment periods, and offering technical assistance to build community-based capacity for participation. EPA's actions need to consider the unique on-the-ground conditions in these communities, such as outdated infrastructure, to help ensure they benefit equitably from policy solutions.

EPA will also collect more data and develop new methodologies to understand PFAS exposure pathways in disadvantaged communities; to what extent PFAS pollution contributes to the cumulative burden of exposures from multiple sources in these communities; and how non-environmental stressors, such as systemic socioeconomic disparities, can exacerbate the impacts of pollution exposure and vice versa.

Goals and Objectives

EPA's comprehensive approach to addressing PFAS is guided by the following goals and objectives.

RESEARCH

Invest in research, development, and innovation to increase understanding of PFAS exposures and toxicities, human health and ecological effects, and effective interventions that incorporate the best available science.

Objectives

- Build the evidence base on individual PFAS and define categories of PFAS to establish toxicity values and methods.
- Increase scientific understanding on the universe of PFAS, sources of environmental contamination, exposure pathways, and human health and ecological effects.
- Expand research on current and emerging PFAS treatment, remediation, destruction, disposal, and control technologies.
- Conduct research to understand how PFAS contribute to the cumulative burden of pollution in communities with environmental justice concerns.

RESTRICT

Pursue a comprehensive approach to proactively prevent PFAS from entering air, land, and water at levels that can adversely impact human health and the environment.

Objectives

- Use and harmonize actions under all available statutory authorities to control and prevent PFAS contamination and minimize exposure to PFAS during consumer and industrial uses.
- Place responsibility for limiting exposures and addressing hazards of PFAS on manufacturers, processors, distributors, importers, industrial and other significant users, dischargers, and treatment and disposal facilities.
- Establish voluntary programs to reduce PFAS use and release.
- Prevent or minimize PFAS discharges and emissions in all communities, regardless of income, race, or language barriers.

REMEDIATE

Broaden and accelerate the cleanup of PFAS contamination to protect human health and ecological systems.

Objectives

- Harmonize actions under all available statutory authorities to address PFAS contamination to protect people, communities, and the environment.
- Maximize responsible party performance and funding for investigations and cleanup of PFAS contamination.
- Help ensure that communities impacted by PFAS receive resources and assistance to address contamination, regardless of income, race, or language barriers.
- Accelerate the deployment of treatment, remediation, destruction, disposal, and mitigation technologies for PFAS, and ensure that disposal and destruction activities do not create new pollution problems in communities with environmental justice concerns.

Key Actions

This section summarizes the bold actions that EPA plans to take from 2021 through 2024 on PFAS, as well as some ongoing efforts thereafter. The actions described in this roadmap are subject to the availability of appropriations and other resources. Each of these actions—led by EPA's program offices—are significant building blocks in the Agency's comprehensive strategy to protect public health and ecosystems by researching, restricting, and remediating PFAS contamination. As EPA takes each of these actions, it also commits to transparent, equitable, and inclusive engagement with all stakeholders to inform the Agency's work.

These are not the only actions underway at EPA, nor will they be the last. As the Agency does more, it will learn more. And as EPA learns more, it will do more. As EPA continues to build the evidence base, as regulatory work matures, and as EPA learns more from its partnerships across the country, the Agency will deliver additional actions commensurate with the urgency and scale of response that the PFAS problem demands.

Office of Chemical Safety and Pollution Prevention

Publish national PFAS testing strategy

Expected Fall 2021

EPA needs to evaluate a large number of PFAS for potential human health and ecological effects. Most PFAS have limited or no toxicity data. To address this data gap, EPA is developing a national PFAS testing strategy to deepen understanding of the impacts of categories of PFAS, including potential hazards to human health and the environment. This will help EPA identify and select PFAS for which the Agency will require testing using Toxic Substances Control Act (TSCA) authorities. In the 2020 National Defense Authorization Act (NDAA), Congress directed EPA to develop a process for prioritizing which PFAS or classes of PFAS should be subject to additional research efforts based on potential for human exposure to, toxicity of, and other available information. EPA will also identify existing test data for PFAS (both publicly available and submitted to EPA under TSCA) that will be considered prior to requiring further testing to ensure adherence to the TSCA goal of reducing animal testing. EPA will use the testing strategy to identify important gaps in existing data and to select representative chemical(s) within identified categories as priorities for additional studies. EPA expects to exercise its TSCA Section 4 order authority to require PFAS manufacturers to conduct and fund the studies. EPA plans to issue the first round of test orders on the selected PFAS by the end of 2021.

Ensure a robust review process for new PFAS

Efforts Ongoing

EPA's TSCA New Chemicals program plays an important gatekeeper role in ensuring the safety of new chemicals, including new PFAS, prior to their entry in U.S. commerce. Where unreasonable

risks are identified as part of the review process, EPA must mitigate those risks before any manufacturing activity can commence. The 2016 TSCA amendments require EPA to review and make a determination regarding the potential risks for each new chemical submission. Since early 2021, EPA has taken steps to ensure that new PFAS are subject to rigorous reviews and appropriate safeguards, including making changes to the policies and processes underpinning reviews and determinations on new chemicals to better align with the 2016 amendments. In addition, EPA has previously allowed some new PFAS to enter the market through low-volume exemptions (LVEs), following an expedited, 30-day review process. In April 2021, the Agency announced that it would generally expect to deny pending and future LVE submissions for PFAS based on the complexity of PFAS chemistry, potential health effects, and their longevity and persistence in the environment. Moving forward, EPA will apply a rigorous premanufacture notice review process for new PFAS to ensure these substances are safe before they enter commerce.

Review previous decisions on PFAS

Efforts Ongoing

EPA is also looking at PFAS that it has previously reviewed through the TSCA New Chemicals program, including those that it reviewed prior to the 2016 TSCA amendments. For example, EPA recently launched a stewardship program to encourage companies to voluntarily withdraw previously granted PFAS LVEs. EPA also plans to revisit past PFAS regulatory decisions and address those that are insufficiently protective. As part of this effort, the Agency could impose additional notice requirements to ensure it can review PFAS before they are used in new ways that might present concerns.

In addition, EPA plans to issue TSCA Section 5(e) orders for existing PFAS for which significant new use notices (e.g., a new manufacturing process for an existing PFAS, or a new use or user) have recently been filed with EPA. The orders would impose rigorous safety requirements as a condition of allowing the significant new use to commence.

More broadly, EPA is planning to improve approaches for overall tracking and enforcement of requirements in new chemical consent orders and significant new use rules (SNURs) to ensure that companies are complying with the terms of those agreements and regulatory notice requirements.

Close the door on abandoned PFAS and uses

Expected Summer 2022

Many existing chemicals (i.e., those that are already in commerce and listed on the TSCA Inventory of chemicals), including PFAS, are currently not subject to any type of restriction under TSCA. In some instances, the chemicals themselves have not been actively manufactured for many years. In others, chemicals may have certain past uses that have been abandoned. Absent restriction, manufacturers are free to begin using those abandoned chemicals or resume those abandoned uses at any time. Under TSCA, by rule, EPA can designate uses of a chemical that are not currently ongoing—and potentially *all* uses associated with an inactive chemical—as "significant new uses." Doing so ensures that an entity must first submit a notice and certain information to EPA before it can resume use of that chemical or use. TSCA then requires EPA to review and make an affirmative determination on the potential risks to health and the environment and to require safety measures to address unreasonable risks before allowing the PFAS use to resume. EPA is considering how it can apply this authority to help address abandoned uses of PFAS as well as future uses of PFAS on the inactive portion of the TSCA Inventory.

Enhance PFAS reporting under the Toxics Release Inventory

Expected Spring 2022

The Toxics Release Inventory (TRI) helps EPA compile data and information on releases of certain chemicals and supports informed decision-making by companies, government agencies, non-governmental organizations, and the public. Pursuant to the 2020 NDAA, certain industry sectors must report certain PFAS releases to TRI. However, certain

exemptions and exclusions remain for those PFAS reporters, which significantly limited the amount of data that EPA received for these chemicals in the first year of reporting.[iv] To enhance the quality and quantity of PFAS information collected through TRI, EPA intends to propose a rulemaking in 2022 to categorize the PFAS on the TRI list as "Chemicals of Special Concern" and to remove the de minimis eligibility from supplier notification requirements for all "Chemicals of Special Concern." EPA will also continue to update the list of PFAS subject to TRI and expects to announce an additional rulemaking to add more PFAS to TRI in 2022, as required by the 2020 NDAA.

Finalize new PFAS reporting under TSCA Section 8

Expected Winter 2022

TSCA Section 8(a)(7) provides authority for EPA to collect existing information on PFAS. In June 2021, EPA published a proposed data-gathering rule that would collect certain information on any PFAS manufactured since 2011, including information on uses, production volumes, disposal, exposures, and hazards. EPA will consider public comments on the proposal and finalize it before January 1, 2023. Ultimately, information received under this rule will enable EPA to better characterize the sources and quantities of manufactured PFAS in the United States and will assist the Agency in its future research, monitoring, and regulatory efforts.

Office of Water

Undertake nationwide monitoring for PFAS in drinking water

Final Rule Expected Fall 2021

The Safe Drinking Water Act (SDWA) establishes a data-driven and risk-based process to assess drinking water contaminants of emerging concern. Under SDWA, EPA requires water systems to conduct sampling for unregulated contaminants every five years. EPA published the proposed Fifth Unregulated Contaminant Monitoring Rule (UCMR 5) in March 2021. As proposed, UCMR 5 would provide new data that is critically needed to improve EPA's understanding of the frequency that 29 PFAS are found in the nation's drinking water systems and at what levels. The proposed UCMR 5 would significantly expand the number of drinking water systems participating in the program, pending sufficient appropriations by Congress. The data gathered from an expanded set of drinking water systems would improve EPA's ability to conduct state and local assessments of contamination, including analyses of potential environmental justice impacts. As proposed, and if funds are appropriated by Congress, all public water systems serving 3,300 or more people and 800 representative public water systems serving fewer than 3,300 would collect samples during a 12-month period from January 2023 through December 2025. EPA is considering comments on the proposed UCMR 5 and preparing a final rule. Going forward, EPA will continue to prioritize additional PFAS for inclusion in UCMR 6 and beyond, as techniques to measure these additional substances in drinking water are developed and validated.

Establish a national primary drinking water regulation for PFOA and PFOS

Proposed Rule Expected Fall 2022, Final Rule Expected Fall 2023

Under the SDWA, EPA has the authority to set enforceable National Primary Drinking Water Regulations (NPDWRs) for drinking water contaminants and require monitoring of public water

supplies. To date, EPA has regulated more than 90 drinking water contaminants but has not established national drinking water regulations for any PFAS. In March 2021, EPA published the Fourth Regulatory Determinations, including a final determination to regulate Perfluorooctanoic acid (PFOA) and Perfluorooctane sulfonic acid (PFOS) in drinking water. The Agency is now developing a proposed NPDWR for these chemicals. As EPA undertakes this action, the Agency is also evaluating additional PFAS and considering regulatory actions to address groups of PFAS. EPA expects to issue a proposed regulation in Fall 2022 (before the Agency's statutory deadline of March 2023). The Agency anticipates issuing a final regulation in Fall 2023 after considering public comments on the proposal. Going forward, EPA will continue to analyze whether NPDWR revisions can improve public health protection as additional PFAS are found in drinking water.

Publish the final toxicity assessment for GenX and five additional PFAS

Expected Fall 2021 and Ongoing

EPA plans to publish the toxicity assessments for two PFAS, hexafluoropropylene oxide dimer acid and its ammonium salt. These two chemicals are known as "GenX chemicals." GenX chemicals have been found in surface water, groundwater, drinking water, rainwater, and air emissions. GenX chemicals are known to impact human health and ecosystems. Scientists have observed liver and kidney toxicity, immune effects, hematological effects, reproductive and developmental effects, and cancer in animals exposed to GenX chemicals. Completing a toxicity assessment for GenX is essential to better understanding its effects on people and the environment. EPA can use this information to develop health advisories that will help communities make informed decisions to better protect human health and ecological wellness. The Office of Research and Development is also currently developing toxicity assessments for five other PFAS—PFBA, PFHxA, PFHxS, PFNA, and PFDA.

Publish health advisories for GenX and PFBS

Expected Spring 2022

PFAS contamination has impacted drinking water quality across the country, including in underserved rural areas and communities of color. SDWA authorizes EPA to develop non-enforceable and non-regulatory drinking water health advisories to help Tribes, states, and local governments inform the public and determine whether local actions are needed to address public health impacts in these communities. Health advisories offer a margin of protection by defining a level of drinking water concentration at or below which lifetime exposure is not anticipated to lead to adverse health effects. They include information on health effects, analytical methodologies, and treatment technologies and are designed to protect all lifestages. EPA will publish health advisories for Perfluorobutane sulfonic acid (PFBS) and GenX chemicals based on final toxicity assessments. The Agency will develop accompanying fact sheets in different languages to facilitate access to information on GenX and other PFAS. Going forward, EPA will develop health advisories as the Agency completes toxicity assessments for additional PFAS.

Restrict PFAS discharges from industrial sources through a multi-faceted Effluent Limitations Guidelines program

Expected 2022 and Ongoing

Effluent Limitations Guidelines (ELGs) are a powerful tool to limit pollutants from entering the nation's waters. ELGs establish national technology-based regulatory limits on the level of specified pollutants in wastewater discharged into surface waters and into municipal sewage treatment facilities. EPA has been conducting a PFAS multi-industry study to inform the extent and nature of PFAS discharges. Based on this study, EPA is taking a proactive approach to restrict PFAS discharges from multiple industrial categories. EPA plans to make significant progress in its ELG regulatory work by the end of 2024. EPA has established timelines for action—whether it is data collection

or rulemaking—on the nine industrial categories in the proposed PFAS Action Act of 2021, as well as other industrial categories such as landfills. EPA's multi-faceted approach entails:

- Undertake rulemaking to restrict PFAS discharges from industrial categories where EPA has the data to do so—including the guidelines for organic chemicals, plastics and synthetic fibers (OCPSF), metal finishing, and electroplating. Proposed rule is expected in Summer 2023 for OCPSF and Summer 2024 for metal finishing and electroplating.
- Launch detailed studies on facilities where EPA has preliminary data on PFAS discharges, but the data are currently insufficient to support a potential rulemaking. These include electrical and electronic components, textile mills, and landfills. EPA expects these studies to be complete by Fall 2022 to inform decision making about a future rulemaking by the end of 2022.
- Initiate data reviews for industrial categories for which there is little known information on PFAS discharges, including leather tanning and finishing, plastics molding and forming, and paint formulating. EPA expects to complete these data reviews by Winter 2023 to inform whether there are sufficient data to initiate a potential rulemaking.
- Monitor industrial categories where the phaseout of PFAS is projected by 2024, including pulp, paper, paperboard, and airports. The results of this monitoring, and whether future regulatory action is needed, will be addressed in the Final ELG Plan 15 in Fall 2022.

Leverage NPDES permitting to reduce PFAS discharges to waterways

Expected Winter 2022

The National Pollutant Discharge Elimination System (NPDES) program interfaces with many pathways by which PFAS travel and are released into the environment and ultimately impact people and water quality. EPA will seek to proactively use existing NPDES authorities to reduce discharges of PFAS at the source and obtain more comprehensive information through monitoring on the sources of PFAS and quantity of PFAS discharged by these sources. EPA will use the effluent monitoring data to inform which industrial categories the Agency should study for future ELGs actions to restrict PFAS in wastewater discharges.

- **Leverage federally-issued NPDES permits to reduce PFAS discharges.**[v] EPA will propose monitoring requirements at facilities where PFAS are expected or suspected to be present in wastewater and stormwater discharges, using EPA's recently published analytical method 1633, which covers 40 unique PFAS. In addition, EPA will propose, as appropriate, that NPDES permits: 1) contain conditions based on product elimination and substitution when a reasonable alternative to using PFAS is available in the industrial process; 2) require best management practices to address PFAS-containing firefighting foams for stormwater permits; 3) require enhanced public notification and engagement with downstream communities and public water systems; and 4) require pretreatment programs to include source control and best management practices to protect wastewater treatment plant discharges and biosolid applications.
- **Issue new guidance to state permitting authorities to address PFAS in NPDES permits.** EPA will issue new guidance recommending that state-issued permits that do not already include monitoring requirements for PFAS use EPA's recently published analytical method 1633, which covers 40 unique PFAS, at facilities where PFAS is expected or suspected to be present in wastewater and stormwater discharges. In addition, the new guidance will recommend the full suite of permitting approaches that EPA will use in federally-issued permits. The guidance will enable communities to work closely with their state permitting authorities to suggest monitoring at facilities suspected of containing PFAS.

Publish multi-laboratory validated analytical method for 40 PFAS

Expected Fall 2022

In September 2021, EPA (in collaboration with the Department of Defense) published a single-laboratory validated method to detect PFAS. The method can measure up to 40 specific PFAS compounds in eight environmental matrices (including wastewater, surface water and biosolids) and has numerous applications, including NPDES compliance monitoring. EPA and DOD are continuing this collaboration to complete a multi-laboratory validation of the method. EPA expects to publish the multi-lab validated method online by Fall 2022. Following the publication of the method, EPA will initiate a rulemaking to propose the promulgation of this method under the Clean Water Act (CWA).

Publish updates to PFAS analytical methods to monitor drinking water

Expected Fall 2024

SDWA requires EPA to use scientifically robust and validated analytical methods to assess the occurrence of contaminants of emerging concern, such as an unidentified or newly detected PFAS chemical. EPA will update and validate analytical methods to monitor additional PFAS. First, EPA will review reports of PFAS of concern and seek to procure certified reference standards that are essential for accurate and selective quantitation of emerging PFAS of concern in drinking water samples. EPA will evaluate analytical methods previously published for monitoring PFAS in drinking water (EPA Methods 533 and 537.1) to determine the efficacy of expanding the established target PFAS analyte list to include any emerging PFAS. Upon conclusion of this evaluation, EPA will complete multi-laboratory validation studies and peer review and publish updated EPA PFAS analytical methods for drinking water, making them available to support future drinking water monitoring programs.

Publish final recommended ambient water quality criteria for PFAS

Expected Winter 2022 and Fall 2024

EPA will develop national recommended ambient water quality criteria for PFAS to protect aquatic life and human health. Tribes and states use EPA-recommended water quality criteria to develop water quality standards to protect and restore waters, issue permits to control PFAS discharges, and assess the cumulative impact of PFAS pollution on local communities. EPA will publish recommended aquatic life criteria for PFOA and PFOS and benchmarks for other PFAS that do not have sufficient data to define a recommended aquatic life criteria value. EPA will first develop human health criteria for PFOA and PFOS, taking into account drinking water and fish consumption. This initiative will consider the latest scientific information and will develop human health criteria for additional PFAS when final toxicity assessments are available. Additionally, EPA will support Tribes in developing water quality standards that will protect waters under Tribal jurisdiction under the same framework as waters in adjacent states. Aquatic life criteria are expected in Winter 2022, and human health criteria are expected Fall 2024.

Monitor fish tissue for PFAS from the nation's lakes and evaluate human biomarkers for PFAS

Expected Summer 2022

States and Tribes have highlighted fish tissue data in lakes as a critical information need. Food and water consumption are important pathways of PFAS exposure, and PFAS can accumulate in fish tissue. In fact, EPA monitoring to date shows the presence of PFAS, at varying levels, in approximately 100 percent of fish tested in the Great Lakes and large rivers. In Summer 2022, EPA will collect fish tissue in the National Lakes Assessment for the first national study of PFAS in fish tissue in U.S. lakes. This will provide a better understanding of where PFAS fish tissue contamination is occurring, which

PFAS are involved, and the severity of the problem. The new data will complement EPA's analyses of PFAS in fish tissue and allow EPA to better understand unique impacts on subsistence fishers, who may eat fish from contaminated waterbodies in higher quantities. EPA's preliminary analysis on whether concentrations of certain PFAS compounds in human blood could be associated with eating fish using the Centers for Disease Control and Prevention's National Health and Nutrition Examination Survey (NHANES) data found a positive correlation. Completing this analysis will help make clear the importance of the fish consumption pathway for protecting communities. EPA will continue to pursue collaboration with Tribal and federal partners to investigate this issue of mutual interest.

Finalize list of PFAS for use in fish advisory programs

Expected Spring 2023

EPA will publish a list of PFAS for state and Tribal fish advisory programs that are either known or thought to be in samples of edible freshwater fish in high occurrence nationwide. This list will serve as guidance to state and Tribal fish tissue monitoring and advisory programs so that they know which PFAS to monitor and how to set fish advisories for PFAS that have human health impacts via fish consumption. This information will encourage more robust data collection from fish advisory programs and promote consistency of fish tissue PFAS monitoring results in EPA's publicly accessible Water Quality Portal. By issuing advisories for PFAS, state and Tribal programs can provide high-risk populations, including communities and individuals who depend on subsistence fishing, with more information about how to protect their health.

Finalize risk assessment for PFOA and PFOS in biosolids

Expected Winter 2024

Biosolids, or sewage sludge, from wastewater treatment facilities can sometimes contain PFAS. When spread on agricultural fields, the PFAS can contaminate crops and livestock. The CWA authorizes EPA to set pollutant limits and monitoring and reporting requirements for contaminants in biosolids if sufficient scientific evidence shows that there is potential harm to human health or the environment. A risk assessment is key to determining the potential harm associated with human exposure to chemicals. EPA will complete the risk assessment for PFOA and PFOS in biosolids by Winter 2024. The risk assessment will serve as the basis for determining whether regulation of PFOA and PFOS in biosolids is appropriate. If EPA determines that a regulation is appropriate, biosolids standards would improve the protection of public health and wildlife health from health effects resulting from exposure to biosolids containing PFOA and PFOS.

Office of Land and Emergency Management

Propose to designate certain PFAS as CERCLA hazardous substances

Proposed rule expected Spring 2022; Final rule expected Summer 2023

EPA is developing a Notice of Proposed Rulemaking to designate PFOA and PFOS as Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) hazardous substances. Such designations would require facilities across the country to report on PFOA and PFOS releases that meet or exceed the reportable quantity assigned to these substances. The hazardous substance designations would also enhance the ability of federal, Tribal, state, and local authorities to obtain information regarding the location and extent of releases. EPA or other agencies could also seek cost recovery or contributions for costs incurred for the cleanup. The proposed rulemaking will be available for public comment in Spring 2022. The Agency commits to conducting robust stakeholder engagement with communities near PFAS-contaminated sites.

Issue advance notice of proposed rulemaking on various PFAS under CERCLA

Expected Spring 2022

In addition to developing a Notice of Proposed Rulemaking designating PFOA and PFOS as hazardous substances under CERCLA, EPA is developing an Advance Notice of Proposed Rulemaking to seek public input on whether to similarly designate other PFAS. The Agency may request input regarding the potential hazardous substance designation for precursors to PFAS, additional PFAS, and groups or subgroups of PFAS. The Agency will engage robustly with communities near PFAS-contaminated sites to seek their input and learn about their lived experiences. Going forward, EPA will consider designating additional PFAS as hazardous substances under CERCLA as more specific information related to the health effects of those PFAS and methods to measure them in groundwater are developed.

Issue updated guidance on destroying and disposing of certain PFAS and PFAS-containing materials

Expected by Fall 2023

The 2020 NDAA requires that EPA publish interim guidance on destroying and disposing of PFAS and certain identified non-consumer PFAS-containing materials. It also requires that EPA revise that guidance at least every three years, as appropriate. EPA published the first interim guidance in December 2020 for public comment. It identifies three technologies that are commercially available to either destroy or dispose of PFAS and PFAS-containing materials and outlines the significant uncertainties and information gaps that exist concerning the technologies' ability to destroy or dispose of PFAS while minimizing the migration of PFAS to the environment. The guidance also highlights research that is underway and planned to address some of these information gaps. Furthermore, the interim guidance identifies existing EPA tools, methods, and approaches to characterize and assess the risks to disproportionately impacted people of color and low-income communities living near likely PFAS destruction or disposal sites. EPA's updated guidance will address the public comments and reflect newly published research results. Since the publication of the interim guidance, EPA and other agencies have been conducting relevant research on destruction and disposal technologies. EPA anticipates that additional research data will become available starting in 2022. EPA will update the guidance when sufficient useful information is available and no later than the statutory deadline of December 2023.

Office of Air and Radiation

Build the technical foundation to address PFAS air emissions

Expected Fall 2022 and Ongoing

The Clean Air Act requires EPA to regulate emissions of hazardous air pollutants (HAPs), which are pollutants that are known or suspected to cause cancer or other serious health effects. At present, EPA actively works with Tribal, state, and local governments to reduce air emissions of 187 HAPs to the environment. While PFAS are not currently listed as HAPs under the Clean Air Act, EPA is building the technical foundation on PFAS air emissions to inform future decisions. EPA is conducting ongoing work to:

- Identify sources of PFAS air emissions;
- Develop and finalize monitoring approaches for measuring stack emissions and ambient concentrations of PFAS;
- Develop information on cost-effective mitigation technologies; and
- Increase understanding of the fate and transport of PFAS air emissions to assess their potential for impacting human health via contaminated groundwater and other media pathways.

EPA will use a range of tools, such as EJSCREEN, to determine if PFAS air pollution disproportionately affects communities with environmental justice concerns. Data from other ongoing EPA activities, such as field tests, TRI submissions, and new TSCA reporting and recordkeeping requirements, will help EPA collect additional information on sources and releases. By Fall 2022, EPA will evaluate mitigation options, including listing certain PFAS as hazardous air pollutants and/or pursuing other regulatory and non-regulatory approaches. The Agency will continue to collect necessary supporting technical information on an ongoing basis.

Office of Research and Development

Develop and validate methods to detect and measure PFAS in the environment

Ongoing Actions

Robust, accurate methods for detecting and measuring PFAS in air, land, and water are essential for understanding which PFAS are in the environment and how much are present. These methods are also essential for evaluating the effectiveness of different technologies for removing PFAS from air, land, and water and for implementing future regulations. To date, EPA has developed validated methods to measure 29 PFAS in drinking water and 24 PFAS in groundwater, surface water, and wastewater. EPA has also developed a method for measuring selected PFAS in air emissions. EPA will build on this work by developing additional targeted methods for detecting and measuring specific PFAS and non-targeted methods for identifying unknown PFAS in the environment. EPA also recognizes the need for "total PFAS" methods that can measure the amount of PFAS in environmental samples without identifying specific PFAS. EPA will increase its efforts to develop and, if appropriate, validate "total PFAS" methods, focusing on air emissions, wastewater, and drinking water. Near-term deliverables include:

- Draft total adsorbable fluorine method for wastewater for potential laboratory validation (Fall 2021);
- Draft method for measuring additional PFAS in air emissions (Fall 2022); and
- Draft methods and approaches for evaluating PFAS leaching from solid materials (Fall 2022).

Advance the science to assess human health and environmental risks from PFAS

Ongoing Actions

EPA will expand understanding of the toxicity of PFAS through several ongoing research activities. First, EPA will continue to develop human health toxicity assessments for individual PFAS under EPA's Integrated Risk Information System (IRIS) Program,

and if needed, other fit-for-purpose toxicity values. When combined with exposure information and other important considerations, EPA can use these toxicity assessments to assess potential human health risks to determine if, and when, it is appropriate to address these chemicals. Most PFAS, however, have limited or no toxicity data to inform human health or ecological toxicity assessments. To better understand human health and ecological toxicity across a wider variety of PFAS, EPA will continue to compile and summarize available and relevant scientific information on PFAS and conduct toxicity testing on individual PFAS and PFAS mixtures. This will inform the development and refinement of PFAS categories for hazard assessment. EPA will also conduct research to identify PFAS sources in the outdoor and indoor environment, to characterize PFAS movement through the environment, and to identify the relative importance of different human exposure pathways to PFAS (e.g., ingestion of contaminated food or water, interaction with household articles or consumer products, and inhalation of indoor or outdoor air containing PFAS). EPA also will work to characterize how exposure to PFAS may contribute to cumulative impacts on communities, particularly communities with environmental justice concerns. Near-term deliverables include:

- Identify initial PFAS categories to inform TSCA test orders as part of the PFAS National Testing Strategy (Fall 2021)
- Consolidate and update data on chemical/physical properties, human health toxicity and toxicokinetics, and ecotoxicity (Spring 2022 – Fall 2024)
- Complete draft PFHxS, PFHxA, PFNA, and PFDA IRIS assessments for public comment and peer review (Spring – Fall 2022)
- Complete and publish the final PFBA IRIS assessment (Fall 2022)

Evaluate and develop technologies for reducing PFAS in the environment

Ongoing Actions

EPA needs new data and information on the effectiveness of different technologies and approaches for removing PFAS from the environment and managing PFAS and PFAS-containing materials to inform decisions on drinking water and wastewater treatment, contaminated site cleanup and remediation, air emission controls, and end-of-life materials management. This information is also needed to better ensure that particular treatment and waste management technologies and approaches do not themselves lead to additional PFAS exposures, particularly in overburdened communities where treatment and waste management facilities are often located. Toward that end, EPA will continue efforts to develop approaches for characterizing PFAS in source waters, at contaminated sites, and near PFAS production and treatment/disposal facilities. EPA will also continue to evaluate and develop technologies for drinking water and wastewater treatment, contaminated site remediation, air emission controls, and destruction and disposal of PFAS-containing materials and waste streams. These efforts include conducting laboratory- and pilot-scale studies, which will inform the design of full-scale field studies done in partnership with facilities and states to evaluate real-world applications of different PFAS removal technologies and management approaches.

EPA will prioritize efforts to evaluate conventional thermal treatment of PFAS-containing wastes and air emissions and assess the effectiveness of conventional drinking water and wastewater treatment processes. EPA will also continue to evaluate and advance the application of innovative, non-thermal technologies to treat PFAS waste and PFAS-contaminated materials. Building upon these evaluations, EPA will document the performance of PFAS removal technologies and establish technology-based PFAS categories that identify the list of PFAS that are effectively removed through the application of the associated technology. Near-term deliverables include:

- Collect data to inform the 2023 guidance on destroying and disposing of certain PFAS and PFAS-containing materials (Spring 2022 – Fall 2023);
- Identify initial PFAS categories for removal technologies (Summer 2022); and
- Develop effective PFAS treatment technologies for drinking water systems (Fall 2022).

Cross-Program

Engage directly with affected communities in every EPA Region

Expected Fall 2021 and Ongoing

EPA must fully understand the challenges facing individuals and communities grappling with PFAS contamination to understand their lived experiences and determine the most effective interventions. As recommended by the National Environmental Justice Advisory Council (NEJAC), EPA will meet with affected communities in each EPA Region to hear how PFAS contamination impacts their lives and livelihoods. EPA will use the knowledge from these engagements to inform the implementation of the actions described in this roadmap. EPA will also use the input to develop and share information to reduce potential health risks in the near term and help communities on the path to remediation and recovery from PFAS contamination.

Use enforcement tools to better identify and address PFAS releases at facilities

Ongoing Actions

EPA is initiating actions under multiple environmental authorities—RCRA, TSCA, CWA, SDWA and CERCLA—to identify past and ongoing releases of PFAS into the environment at facilities where PFAS has been used, manufactured, discharged, disposed of, released, and/or spilled. EPA is conducting inspections, issuing information requests, and collecting data to understand the level of contamination and current risks posed by PFAS to surrounding communities and will seek to address threats to human health with all its available tools. For example, EPA's enforcement authorities allow the Agency, under certain circumstances, to require parties responsible for PFAS contamination to characterize the nature and extent of PFAS contamination, to put controls in place to expeditiously limit future releases, and to address contaminated drinking water, soils, and other contaminated media. When EPA becomes aware of a potential imminent and substantial endangerment situation where PFAS poses a threat to human health, the Agency will swiftly employ its expertise to assess the situation and take appropriate action, including using statutorily authorized powers.

Accelerate public health protections by identifying PFAS categories

Expected Winter 2021 and Ongoing

To accelerate EPA's ability to address PFAS and deliver public health protections sooner, EPA is working to break the large, diverse class of PFAS into smaller categories based on similarities across defined parameters (such as chemical structure, physical and chemical properties, and toxicological properties). EPA plans to initially categorize PFAS using two approaches. In the first approach, EPA plans to use toxicity and toxicokinetic data to develop PFAS categories for further hazard assessment and to inform hazard- or risk-based decisions. In the second approach, EPA plans to develop PFAS categories based on removal technologies using existing understanding of treatment, remediation, destruction, disposal, control, and mitigation principles.

EPA plans to use the PFAS categories developed from these two approaches to identify gaps in coverage from either a hazard assessment or removal technology perspective, which will help EPA prioritize future actions to research, restrict, and remediate PFAS. For example, EPA may choose to prioritize research to characterize the toxicity of PFAS that are not being addressed by regulations that require the implementation of removal technologies. Conversely, EPA may prioritize research to evaluate the efficacy of technologies designed to remove PFAS that are included in a hazard-based category with relatively higher toxicities. To support coordination and integration of information across PFAS categories, EPA plans to develop a PFAS categorization database that will capture key characteristics of individual PFAS, including category assignments.

Establish a PFAS Voluntary Stewardship Program

Expected Spring 2022

Reduction of PFAS exposure through regulatory means can take time to develop, finalize, and implement. Moreover, current PFAS regulatory efforts do not extend to all of the approximately 600 PFAS currently in commerce. As a companion to other efforts described in this roadmap, EPA will establish a voluntary stewardship program challenging industry to reduce overall releases of PFAS into the environment. The program, which will not supplant industry's regulatory or compliance requirements, will call on industry to go beyond those requirements by reporting all PFAS releases in order to establish a baseline and then continuing to report to measure progress in reducing releases over time. EPA will validate industry efforts to meet reduction targets and timelines.

Educate the public about the risks of PFAS

Expected Fall 2021 and Ongoing

Addressing PFAS contamination is a critical part of EPA's mission to protect human health and the environment. This important mission cannot be achieved without effectively communicating with communities, individuals, businesses, the media, and Tribal, state, and local partners about the known and potential health risks associated with these chemicals. When EPA communicates risk, it is the Agency's goal to provide meaningful, understandable, and actionable information to many audiences. To accomplish this goal, EPA will make available key explainers that help the public understand what PFAS are, how they are used, and how PFAS can impact their health and their lives. These explainers and other educational materials will be published in multiple languages, and the Agency will work to ensure information reaches targeted communities (including those with limited access to technology and resources).

Issue an annual public report on progress towards PFAS commitments

Winter 2022 and Ongoing

EPA is committed to acting on PFAS with transparency and accountability. On an annual basis, EPA will report to the public on the status of the actions outlined in this roadmap, as well as future actions the Agency may take. EPA will also engage regularly with communities experiencing PFAS contamination, co-regulators, industry, environmental groups, community leaders, and other stakeholders to clearly communicate its actions and to stay abreast of evolving needs.

Conclusion

Every level of government—federal, Tribal, state, and local—needs to exercise increased and sustained leadership to accelerate progress to clean up PFAS contamination, prevent new contamination, and make game-changing breakthroughs in the scientific understanding of PFAS. This strategic roadmap represents the Agency's commitment to the American people on what EPA seeks to deliver from 2021 to 2024.

The risks posed by PFAS demand that the Agency take a whole-of-agency approach to attack the problem from multiple directions. Focusing only on remediating legacy contamination, for example, does nothing to prevent new contamination from occurring. Focusing only on preventing future contamination fails to minimize risks to human health that exist today. To build more enduring, comprehensive, and protective solutions, EPA seeks to leverage its full range of statutory authorities and work with its partners—including other federal agencies, state and Tribal regulators, scientists, industry, public health officials, and communities living with PFAS contamination—to implement this multi-media approach and achieve tangible benefits for human health and the environment.[vi]

Endnotes

[i] Approximately 650 PFAS are currently in commerce under TSCA, roughly half of which were grandfathered into the TSCA inventory.

[ii] EPA has identified 6-8 facilities that produce PFAS feedstock.

[iii] Key industries with significant documented discharges include PFAS production and processing, metal finishing, airports, pulp and paper, landfills, and textile and carpet manufacturing.

[iv] Examples include de minimis exemption, supplier notification requirements, and applicability of those requirements to wastes.

[v] Federally-issued permits are those that EPA issues in MA, NH, NM, DC, territories, federal waters, and Indian Country (and federal facilities in DE, CO, VT, WA).

[vi] This document provides information to the public on how EPA intends to exercise its discretion in implementing statutory and regulatory provisions that apply to PFAS. Those provisions contain legally binding requirements, and this document does not substitute for those statutory and regulatory provisions or regulations, nor is it a regulation itself.

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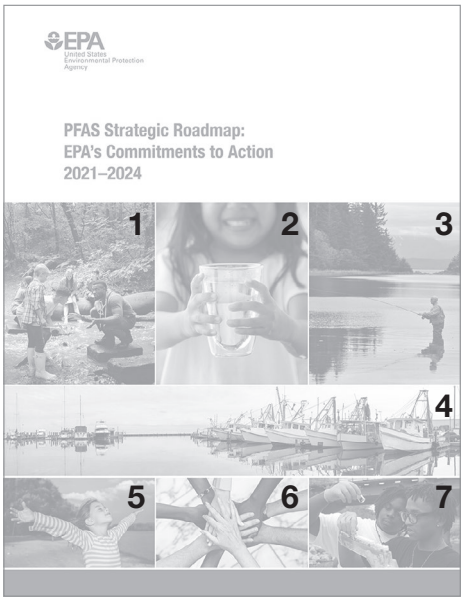

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United States
Environmental Protection
Agency

EPA-100-K-21-002
October 2021

APPENDIX 3 – Virginia PFAS Sample Study Design

PFAS Sampling & Monitoring Study in Virginia Drinking Water

Virginia Department of Health - Office of Drinking Water

Developed in conjunction with the VA PFAS Workgroup

PFAS Sampling & Monitoring Study in Virginia Drinking Water

Virginia Department of Health
Office of Drinking Water

In conjunction with the
VA PFAS Workgroup

March 2021

TABLE OF CONTENTS

1. Introduction

Per- and poly fluoroalkyl substances (PFAS) are man-made, industrially produced compounds. Production of these chemicals began in the 1940s and there are now more than 5,000 different chemicals in the PFAS family. A wide variety of products, including stain resistant fabric coatings, non-stick coatings (Teflon), food packaging, and firefighting foam contain PFAS. Two of the most extensively produced and studied chemicals in the PFAS family are perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS).

PFOA/PFOS, and many other chemicals in the PFAS family, do not easily break down in the environment and are easily transportable in the atmosphere, surface water, soil, and groundwater. Exposure to humans can occur by eating, inhaling, or even touching the product. The U.S. Environmental Protection Agency (EPA) reports that scientists have found traces of one or more PFAS in the blood of nearly all the people they tested in the USA. Possible health effects associated with exposure to chemicals in the PFAS family include developmental effects to fetuses during pregnancy or to breastfed infants (e.g., low birth weight, accelerated puberty, skeletal variations), cancer (e.g., testicular, kidney), liver effects (e.g., tissue damage), immune effects (e.g., antibody production and immunity), thyroid effects, and other effects (e.g., cholesterol changes).

From 2013 to 2015, EPA evaluated the occurrence of PFOA, PFOS, and four other PFAS compounds at 4,920 public water systems (PWS; also referred to as "waterworks" in Virginia) across the U.S. as part of its "Third Unregulated Contaminant Monitoring Rule" (UCMR 3) evaluation. The data did not reveal significant occurrences of PFOA, PFOS, or other PFAS in Virginia; however, UCMR 3 had reporting limits of 20 parts per trillion (ppt) and 40 ppt for PFOA and PFOS, respectively. Two Virginia waterworks detected PFAS compounds, but follow-up sampling did not identify the source or an impact to drinking water supplies. EPA found that 1.3% of the public water systems monitored under the UCMR 3 had measured concentrations of PFOA and PFOS that were greater than the EPA's lifetime Health Advisory (70 parts per trillion (ppt)).

On May 16, 2016, EPA issued a Lifetime Health Advisory of 70 ppt for combined PFOA and PFOS and, on February 22, 2021, EPA announced it will move forward with final regulatory determinations (i.e., establish regulatory standards) for PFOA and PFOS under the Safe Drinking Water Act. However, since PFAS as a whole, or as individual compounds, are not subject to federally established regulatory limits at this time, there is scant monitoring data of PFAS occurrence in Virginia's waterworks and major sources of water.

House Bill (HB) 586 (2020 Acts of Assembly Chapter 0611) seeks to prevent adverse health effects and protect public health by studying the occurrence of PFAS in drinking water. The legislation requires the State Health Commissioner, who acts through the Virginia Department of Health (VDH), to convene a workgroup to study the occurrence of six specific PFAS (PFOA, PFOS, perfluorobutyrate (PFBA),

perfluoroheptanoic acid (PFHpA), perfluorohexane sulfonate (PFHxS), perfluorononanoic acid (PFNA)) and other PFAS, as deemed necessary, that may be present in drinking water from waterworks, identify possible sources of such contamination, and evaluate approaches to regulating PFAS. The workgroup may recommend maximum contaminant levels (MCLs) for inclusion in regulations of the Board of Health applicable to waterworks. The workgroup will report its findings to the Governor and legislative committees by December 1, 2021. To determine the occurrence of PFAS contamination, the bill requires VDH to sample no more than 50 representative waterworks and major sources of water.

HB1257 (2020 Acts of Assembly Chapter 1097) directs the Board of Health to adopt regulations establishing MCLs for PFOA, PFOS, and other PFAS as it deems necessary. The effective date for HB1257 is January 1, 2022, so that the Board can consider the findings and recommendations that come from the work performed to satisfy the requirements in HB586.

To implement HB586, VDH, through its Office of Drinking Water (ODW), formed the Virginia (VA) PFAS Workgroup. In the October 20, 2020, kickoff meeting for the VA PFAS Workgroup, members accepted and formed four subgroups to focus on (1) PFAS Health and Toxicology, (2) PFAS Occurrence and Monitoring, (3) PFAS Policy and Regulations and (4) PFAS Treatment Technologies. These subgroups meet on a monthly basis and bring summaries of their findings and recommendations to the quarterly main VA PFAS Workgroup meetings.

2. Purpose

This document, the PFAS Sampling & Monitoring Study in Virginia Drinking Water (Sampling Plan), summarizes the approach VDH will follow to determine the occurrence of PFAS in drinking water and in major sources of water in Virginia. VDH developed the Sampling Plan in conjunction with the VA PFAS Workgroup.

As specified in the legislation, this study is limited to drinking water produced by waterworks and major water sources used by waterworks. It does not include water from private wells or other sources. "Waterworks" means a system that serves piped water for human consumption to at least 15 service connections or 25 or more individuals for at least 60 days out of the year. Code of Virginia § 32.1-167.

3. Objectives

The PFAS Occurrence and Monitoring Subgroup evaluated existing approaches to sample and monitor for PFAS chemicals in drinking water, including approaches adopted by other states and the federal government. Based on available data, funding, time limitations, and other states' experience, the Subgroup recommend an approach (study design) for a limited sampling program to determine the occurrence of PFAS in Virginia by sampling no more than 50 representative waterworks and major sources of water. The Occurrence and Monitoring Subgroup met on January

13 and February 4, 2021 to develop and review its recommended approach and presented it to the VA PFAS Workgroup at a meeting on March 4, 2021. Members of the VA PFAS Workgroup could review a draft of the Sampling Plan prior to the meeting. At the meeting, they voted to support the recommended approach that is described in this Sampling Plan.

Upon implementing the Sampling Plan, VDH, through ODW, will coordinate sample collection from the representative waterworks and major sources of water. As results of analysis come in from the laboratory, ODW and the PFAS Occurrence and Monitoring Subgroup will tabulate PFAS data from the sampling program and other existing PFAS monitoring data that waterworks make available to VDH. The Subgroup will also evaluate the data to determine current levels of PFAS (PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS as deemed necessary) contamination in the Commonwealth's public drinking water.

4. Methodology

The Sampling Plan is intended to prioritize sites for PFAS sampling and generate statewide occurrence data. VDH and the VA PFAS Workgroup considered several factors in developing the Sampling Plan, including:

- The location of potential sources of PFAS contamination;
- Known locations of PFAS contamination;
- The relative risk to consumers who receive water from waterworks that utilize source water that comes from areas that are near known or potential sources of PFAS contamination;
- Population served; and
- Available funding: $40,000.

> *It should be noted that, for the purpose of the Sampling Plan, the term "**potential sources of PFAS contamination**" refers to facilities or locations that may be a source of PFAS based on historical use, existing literature, other available information (SIC codes, VPDES permits etc.), and/or the nature of the facility (airports, unlined landfills etc.). This term is not meant to imply that these locations do in fact produce, use, or discharge PFAS chemicals specifically, only that previous published work indicates the type of facility or activity may be associated with the production, use, or discharge of PFAS.*
>
> *It should be noted that implementation of the Sampling Plan is not intended to determine the causality between potential sources of PFAS and PFAS found in drinking water sources. The purpose of this plan and the sampling to be performed as a result of this plan is intended to provide additional data regarding the occurrence of PFAS in Virginia public drinking water.*

4.1 Sample collection costs/supplies

The sample collection costs and supplies are described in the Quality Assurance Program Plan (QAPP) VDH developed and submitted to EPA for approval and the responses to VDH's bid process for laboratories. The most recent QAPP is dated 10/30/20. VDH is in the process of revising the QAPP to reflect the selection of a lab to perform sample analyses.

4.2 Selection of Sample Locations

The process of selecting sample locations involved a combination of geospatial analysis and programmatic review. The geospatial analysis included the creation of a Geographic Information System (GIS) project using ArcMap 10.4.1 that combined waterworks locations and information about potential sources of PFAS contamination. There are currently 2,811 waterworks (also referred to as "public water systems" (PWSs)) in Virginia. Waterworks are classified based on the characteristics of the population they serve:

- **Community Water Systems (CWS):** A waterworks that provides water to the same population year-round. Examples are municipal water systems, authorities, and residential subdivisions with their own water supplies.
- **Nontransient Noncommunity (NTNC) Water Systems:** A waterworks that is not a CWS, but that regularly serves at least 25 of the same people at least six months of the year. Examples include schools, factories, and hospitals that have their own water supplies.
- **Transient Noncommunity (TNC) Water Systems:** A waterworks that serves transient customers in non-residential settings, such as campgrounds, motels, and restaurants that have their own water supplies.

The PFAS sampling sites selection is primarily based on the following considerations:

- Available funding ($40,000 for sample collection and PFAS analysis);
- HB586 limits sampling to no more than 50 waterworks and major sources of water;
- Relative potential for PFAS contamination in water that is used by waterworks as a source (either groundwater or surface water); and
- Maximum public health risk reduction (i.e., if there is PFAS, how many people are potentially consuming water that is contaminated – "large" waterworks serve more consumers, therefore if their source water is contaminated, they are potentially putting more people at risk). Large waterworks are defined as serving more than 50,000 persons. See 40 CFR § 141.2.

ODW staff identified the largest waterworks in the state (based on population served) and plotted the locations of surface water intakes and groundwater wells used by community and nontransient noncommunity water systems, potential discharge locations, including unlined landfills and airports, and major rivers in the state. Using three different strategies, described below, VDH and the VA PFAS Workgroup identified (1) potential high and/or medium risk groundwater systems based on the potential sources of PFAS contamination, (2) large community water

systems, and (3) water sources/intakes with higher possibility of potential PFAS contamination.

Consumers served by CWSs and NTNCs have a higher risk of exposure from drinking, cooking, bathing and showering, and other water uses. For this reason, the Sampling Plan was limited to CWSs and NTNCs. There are 1,093 CWSs and 510 NTNCs, for a total initial sampling pool of 1,603 waterworks which collectively provide drinking water from 2,626 sources (e.g. wells, springs, and surface water sources).

VDH prioritized the list of waterworks based on relative risk, considering the waterworks proximity to the potential sources of PFAS contamination. Using the GIS system, VDH established several data layers containing locational and other information specific to the potential sources of PFAS contamination. These layers include the following industries and land uses:

- Military or commercial airports (from U.S. Geological Survey data)
- Unlined landfills (data from the Virginia Department of Environmental Quality (DEQ))
- Virginia Pollutant Discharge Elimination System (VPDES) discharge data
- Discharge points for Publicly Owned Treatment Works (POTWs)
- Waterworks size and population served data

4.2 Identification of Potential at-Risk Groundwater Waterworks

A significant portion of the peer-reviewed, published literature on PFAS contamination focuses on contamination resulting from the use of Aqueous Fire Fighting Foam (AFFF), a product mandated for use by the Federal Aviation Administration (FAA). AFFF that meets U.S. Department of Defense specifics for use at military facilities is a common source of PFAS and is frequently found at both military and civilian airports. Other sources of PFAS associated with airports and the aeronautical industry include wire insulation and certain mechanical fluids. Given the number of products that can be found at airports and that potentially contain PFAS, airports are considered a likely source of PFAS contamination. For the purpose of the geospatial analysis, ODW staff only considered large airports (meaning the airport is large enough to be classified as a public-use airport). ODW did not attempt to identify whether the airports had either on-purpose or accidental releases of AFFF or if they conducted training with AFFF on site.

Peer-reviewed, published research also indicates that landfills, specifically landfill leachate, are a source of PFAS contamination. Landfill leachate likely obtains PFAS from the myriad of consumer products that include PFAS and are commonly placed in the garbage. Without going into the full list of likely consumer products, food contact packaging, cosmetics, and electronics are all examples of PFAS-containing products that can commonly be found in the garbage. There are landfills in Virginia that were constructed before they had to meet the requirements in Subtitle D of the Resource Conservation and Recovery Act (RCRA), meaning they are unlined and

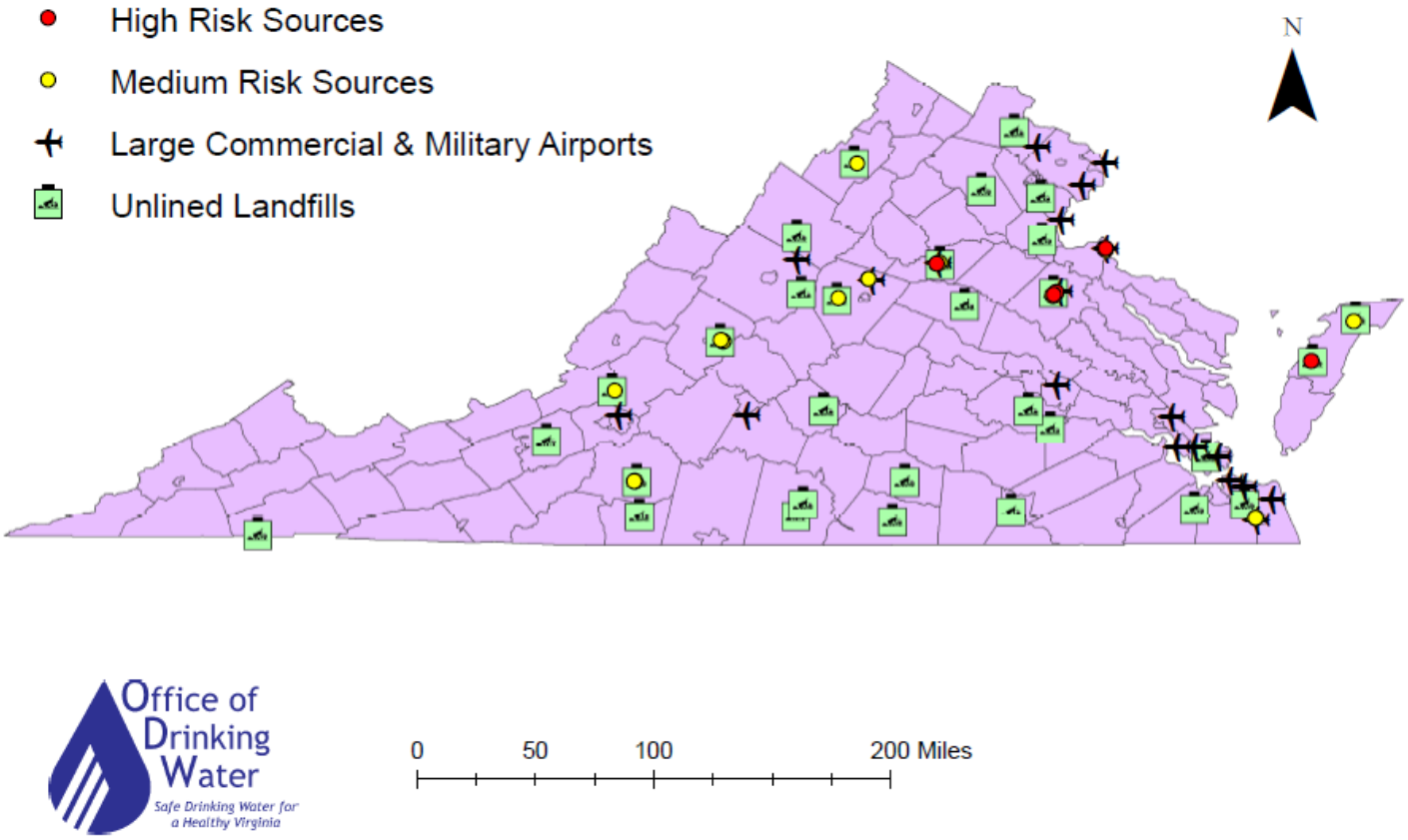
more likely to have leachate that reaches groundwater sources. DEQ recommended focusing on these landfills over Subtitle D landfills (which are lined and have leachate collection systems) and other waste disposal facilities.

For purposes of the Sampling Plan and in order to minimize duplication of effort, VDH designated any waterworks using a groundwater well located within ½ mile of an unlined landfill or airport (potential sources of PFAS contamination) (as potential high risk water source. VDH designated waterworks using a groundwater well located within ½ mile to 1 mile of a known unlined landfill or airport as potential medium risk water source.

VDH did not consider the following in evaluating potential high or medium risk groundwater waterworks/water sources.

- Data on PFAS levels in groundwater
- Information on groundwater flow direction
- Information on water supply well recharge areas

Based on the compilation of potential sources of PFAS contamination, VDH and the VA PFAS Workgroup selected 11 waterworks that use groundwater wells that are located within 1 mile of potential sources of PFAS contamination. These waterworks utilize a total of 6 groundwater wells that constitute a high risk and 13 groundwater wells that constitute a medium risk based on the proximity of the well to the potential source of PFAS contamination. See Figure 1 and Table 1.



Map 1. Groundwater Waterworks downstream of potential PFAS discharges

Table 1 . Potential high and medium risk Groundwater systems

System Name	PWSID	Facility Name	ID	System Type	Population Served	High or Medium
NAVAL SUPPORT FACILITY_ DAHLGREN	6099340	WELL 3 - BLDG 274A (RESERVOIR WELL)	WL003	C	11000	H
NAVAL SUPPORT FACILITY_ DAHLGREN	6099340	WELL 1 - BLDG 1288 (BRONSON WELL)	WL001	C	11000	H
BOWLING GREEN_ TOWN OF	6033550	WELL 4	WL004	C	1152	H
PUNGOTEAGUE ELEMENTARY SCHOOL	3001790	WELL	WL001	NTNC	610	H
RSA ROUTE 20	6137120	WELL #2 (MAY LANE)	WL002	C	387	H
FT A P HILL - HEADQUARTERS	6033251	WELL HQ #2 (PWAT 28)	WL028	C	180	H
NAVAL SUPPORT FACILITY_ DAHLGREN	6099340	WELL 2 - BLDG 1190 (CASKEY WELL)	WL002	C	11000	M
BOWLING GREEN_ TOWN OF	6033550	WELL 5	WL005	C	1152	M
BOWLING GREEN_ TOWN OF	6033550	WELL 1A	WL01A	C	1152	M
LONG HOLLOW	2163400	LHWDC WELL 1	WL001	C	578	M
LONG HOLLOW	2163400	LHWDC WELL 2	WL002	C	578	M
EARLYSVILLE FOREST	2003255	WELL 6	WL006	C	488	M
EARLYSVILLE FOREST	2003255	WELL 5	WL005	C	488	M
PEACOCK HILL SUBDIVISION	2003650	WELL 8	WL008	C	475	M
RSA ROUTE 20	6137120	WELL #1 (PORTER RD)	WL001	C	387	M
MOUNTAIN VIEW ELEM SCHOOL	2163560	MTN VIEW WELL	WL001	NTNC	250	M
ROANOKE CEMENT COMPANY	2023180	WELL - ROANOKE CEMENT COMPANY	WL001	NTNC	190	M
FT A P HILL - HEADQUARTERS	6033251	WELL HQ #1 (PWAT 29)	WL029	C	180	M
FRANKLIN COUNTY COMMERCE CENTER	5067137	WELL NO. 5	WL005	NTNC	103	M

**Note:* This establishes relative risk and is not exact. This approach identified wells and waterworks for monitoring in this study. Map 1 shows the distribution of large airports and unlined landfills across Virginia. *The list of waterworks sources identified within ½ mile of a potential source of PFAS contamination is subject to change as new information becomes available. Additional waterworks may be added or removed from the list.*

5. Identification of Potential at-Risk Surface Water Sources

ODW identified major surface water supplies based on potential sources of PFAS contamination that DEQ identified from industrial classification codes and information in discharge permits. These included::

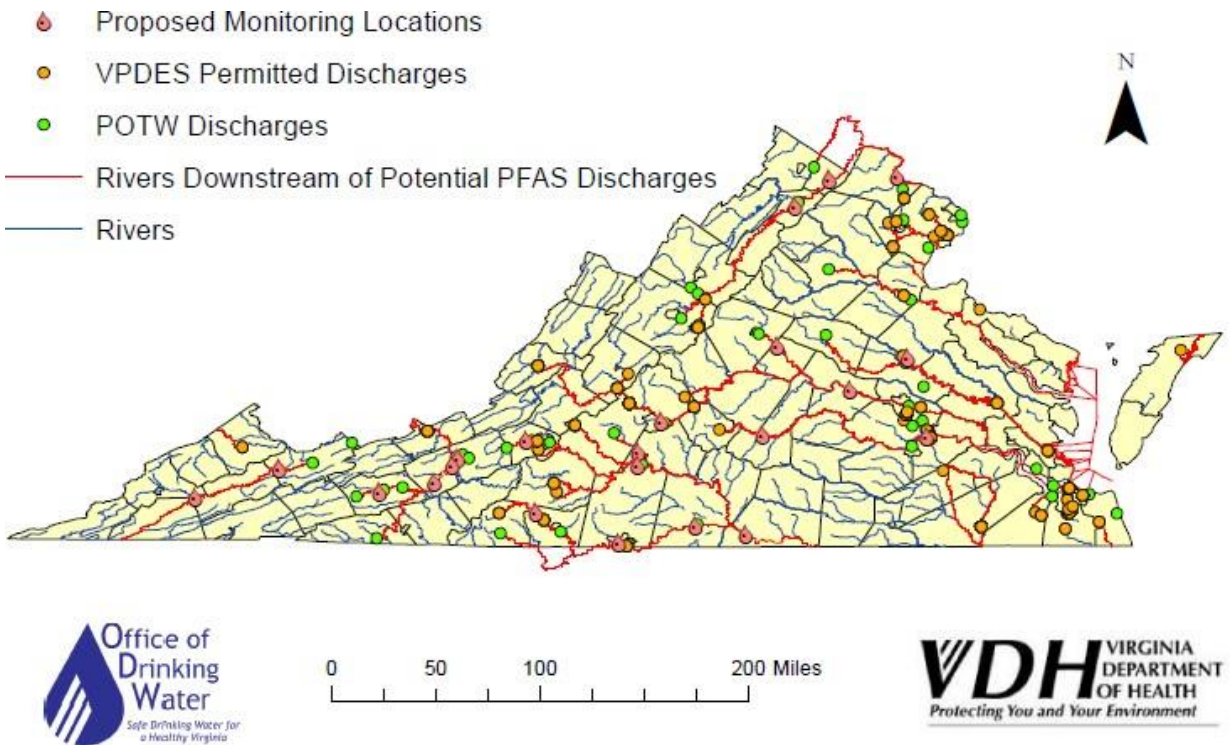
- Publicly Owned Treatment Works (POTWs) with Significant Industrial Users

- Virginia Pollution Discharge Elimination System (VPDES) discharge permits (direct dischargers)

DEQ identified the POTWs and VPDES discharges based on Standard Industrial Classification (SIC) codes for Significant Industrial Users and direct dischargers and activities with potential to involve PFAS. The identified facilities potentially use and/or discharge PFAS; however, DEQ does not have effluent monitoring data for PFAS. DEQ noted that both current and past/historic discharges of PFAS could impact waterworks' surface water intakes. DEQ provided the GPS coordinates for the discharge points to ODW. Using GIS, , ODW connected the discharge points to surface water bodies and identified them as potentially impacted by PFAS discharges. ODW traced the surface water bodies downstream to identify waterworks' with surface water intakes potentially impacted by the discharges. This procedure identified 45 waterworks' drinking water intakes. ODW prioritized these 45 water intakes as follows:

- Excluded intakes associated with the 17 large waterworks because the entry point sampling addresses these intakes.
- Sorted remaining waterworks from largest to smallest population served.
- The Occurrence and Monitoring Subgroup recommended including at least one sample location that from each of the New River, Clinch River, and Dan River.
- Select impacted intakes starting with largest population served, selecting two intakes on the river systems noted above.
- Selected no more than one intake per waterworks.

Based on the limitation in the enabling legislation of no more than 50 waterworks and major sources of water and the number of waterworks selected in part 1 and 2 of the hybrid approach, VDH selected 22 major sources of water for this phase. Map 2 shows the locations of potential sources of PFAS contamination, surface water sources that are potentially impacted by PFAS and associated surface water intake locations selected for monitoring as part of the Sampling Plan. Table 2 lists the associated waterworks.



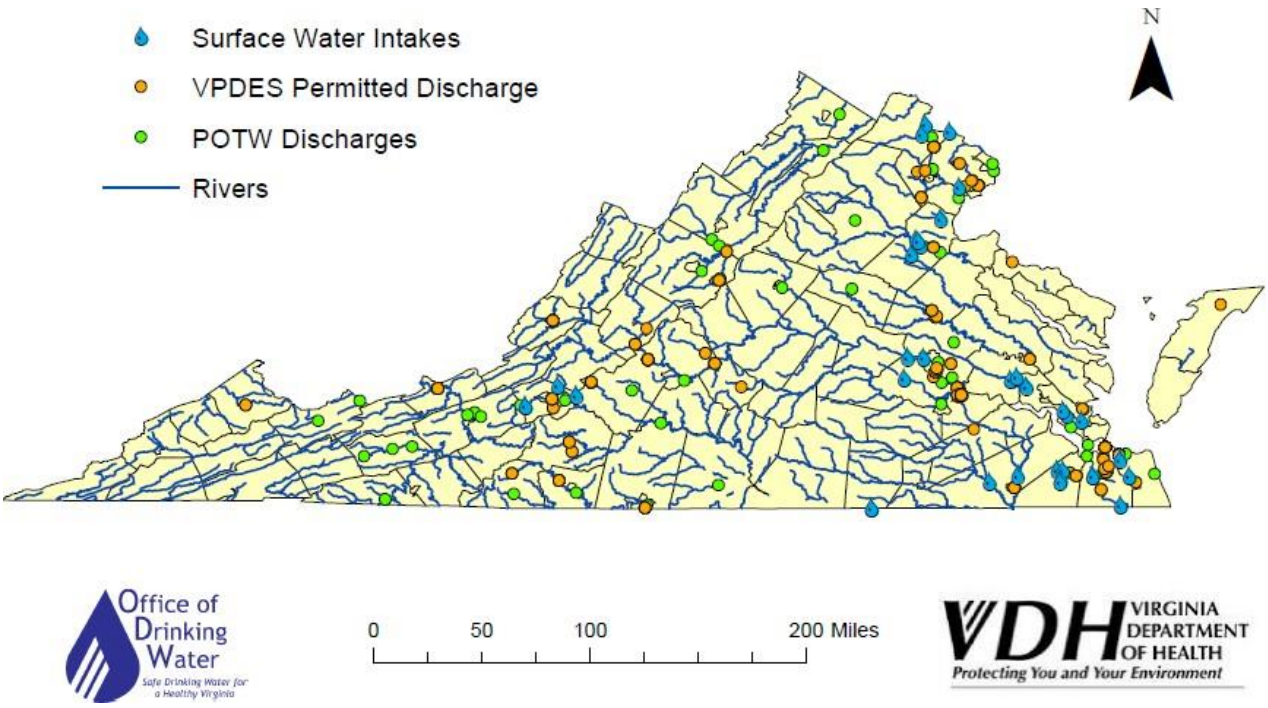
Map 2. Major Water Sources, consisting of surface water intakes.

Table 2. Major Water Sources - Waterworks Surface Water Intakes Identified for Sampling

PWSID	System	Facility
5680200	LYNCHBURG, CITY OF	JAMES RIVER-COLLEGE HILL
4085398	HANOVER SUBURBAN WATER SYSTEM	NORTH ANNA RWI
1121057	NRV REGIONAL WATER AUTHORITY	NEW RIVER (RAW WATER) PUMP STATION
6107300	LEESBURG_ TOWN OF	POTOMAC INTAKE
5590100	DANVILLE, CITY OF	DAN RIVER INTAKE
5089852	UPPER SMITH RIVER WATER SUPPLY	SMITH RIVER INTAKE
3670800	VIRGINIA-AMERICAN WATER CO.	APPOMATTOX RIVER
2775300	CITY OF SALEM WTP	ROANOKE RIVER
5031150	CAMPBELL COUNTY CENTRAL SYSTEM	BIG OTTER RIVER
1750100	RADFORD_ CITY OF	INTAKE ON NEW RIVER
2187406	FRONT ROYAL_ TOWN OF	SOUTH FORK SHENANDOAH RIVER
2065480	LAKE MONTICELLO	RIVANNA RIVER
1195900	WISE COUNTY REGIONAL WATER SYSTEM	CLINCH RIVER INTAKE
1155641	PULASKI COUNTY PSA	CLAYTOR LAKE
5780600	HCSA- LEIGH STREET PLANT	DAN RIVER INTAKE
5147170	FARMVILLE_ TOWN OF	APPOMATTOX RIVER
1197810	WYTHEVILLE_ TOWN OF	REED CREEK
4075735	JAMES RIVER CORRECTIONAL CTR	JAMES RIVER INTAKE
1185695	RICHLANDS_ TOWN OF	IN001 - CLINCH RIVER INTAKE
2043125	BERRYVILLE_ TOWN OF	SHENANDOAH RIVER
5031050	ALTAVISTA, TOWN OF	STAUNTON RIVER
5117310	CLARKSVILLE_ TOWN OF	KERR RESERVOIR INTAKE

6. Higher Public Health reduction - Largest waterworks in Virginia

This involves sampling at entry points and consecutive connections representative of the water entering the 17 largest waterworks in Virginia. The 17 largest waterworks provide water to more than half of Virginia residents. Maps 3 and 4 show the distribution of the intakes represented by this group of 17 waterworks. Several of the waterworks in this group have more than one surface water intake.



Map 3 Proposed 17 large waterworks in Virginia.

Table 3. Seventeen (17) large community waterworks in the Commonwealth of Virginia

PWSID	PWS name	City / County	Population	# EPs	# CCs
6059501	FAIRFAX COUNTY WATER AUTHORITY	FAIRFAX COUNTY	1074422	2	1
3810900	VIRGINIA BEACH, CITY OF	VIRGINIA BEACH	446067	0	1
3700500	NEWPORT NEWS, CITY OF	NEWPORT NEWS	407300	2	0
4041845	CHESTERFIELD CO CENTRAL WATER SYSTEM	CHESTERFIELD	320658	1	2

PWSID	PWS name	City / County	Population	# EPs	# CCs
4087125	HENRICO COUNTY WATER SYSTEM	HENRICO	292000	1	1
6107350	LOUDOUN WATER - CENTRAL SYSTEM	LOUDOUN	286202	1	1
3710100	NORFOLK, CITY OF	NORFOLK	234220	2	0
6013010	ARLINGTON COUNTY	ARLINGTON	215000	0	1
4760100	RICHMOND, CITY OF	RICHMOND CITY	197000	1	0
3550051	CITY OF CHESAPEAKE - NORTHWEST RIVER SYS	CHESAPEAKE	166704	2	0
2770900	WESTERN VIRGINIA WATER AUTHORITY	ROANOKE CITY	155000	2	0
6153600	PWCSA - EAST	PRINCE WILLIAM	153000	0	1
6510010	ALEXANDRIA, CITY OF	ALEXANDRIA	146970	0	2
6153251	PWCSA - WEST	PRINCE WILLIAM	130001	0	2
3740600	PORTSMOUTH, CITY OF	PORTSMOUTH	120400	1	0
6179100	STAFFORD COUNTY UTILITIES	STAFFORD	112285	2	0
6177300	SPOTSYLVANIA COUNTY UTILITIES	SPOTSYLVANIA	84390	2	0
Totals				19	12
Total Samples (EP + CC)				31	

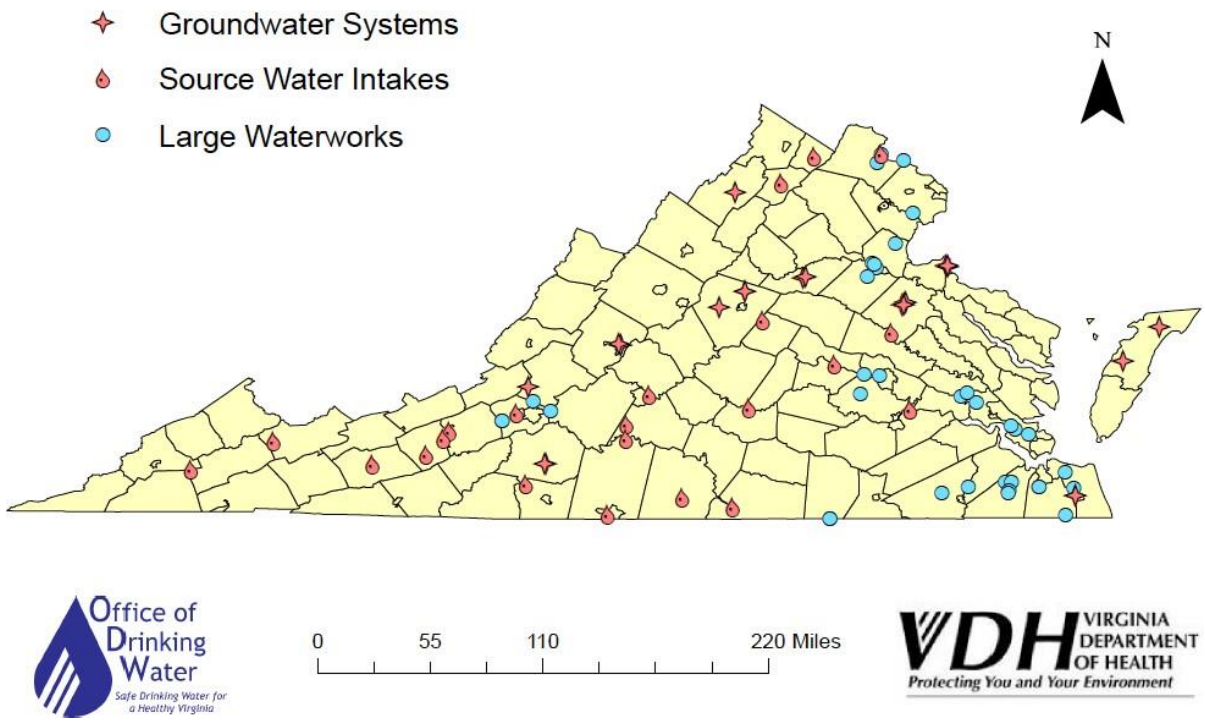
Population = Retail population, not including consecutive customers
EP = Entry point sample point, at a surface water treatment plant
CC = Consecutive Connection sample points
SWTP = Surface water treatment plant
Raw = untreated source water

7. Hybrid Approach Summary

The following table summarizes the numbers of waterworks, sample points, and population served for the hybrid PFAS sampling plan (Table 4 and map 4).

Table 4. Summary of the waterworks and water sources

	Sample Points	Waterworks	Population Served
17 Large Waterworks	31	17	4,541,619
Groundwater - High Risk	6	5	13,329
Groundwater - Medium Risk	13	6	2,084
Major Water Sources	22	22	536,322
Total	72	50	5,093,354



Map 4. The locations of proposed sampling sites for the PFAS Sampling study in Virginia Drinking Water

8. Sampling Approach

VDH intends to request waterworks identified in the Sampling Plan to collect samples for the PFAS study at specific locations. These locations include:

- Entry points (EP) – Locations where finished drinking water enters the distribution system (post treatment).
- Consecutive Connections (CC) – Locations where finished drinking water is transferred from one waterworks to another.
- Intakes – Locations where source water is withdrawn from the water source, such as a river, stream or reservoir, before any treatment.

9. Number of samples per location

The sampling program will take the following approach:

- One sample per location
- To be consistent with the EPA's sampling requirements for Method 533 (see Section 11 below), field reagent blanks (FRBs) will be submitted with each PFAS sample collected as part of the sampling study.
- Confirmation samples
    - A detection > Method Reporting Level (MRL) for a specific PFAS analyte may trigger the collection of a confirmatory sample
    - VDH has the goal of taking confirmation samples upon detection of PFAS; however, VDH may limit confirmation samples due to budget constraints.
    - VDH will prioritize confirmation samples based on:
        - Detection of specific PFAS analytes, such as the analytes in HB586, or detection of PFOS or PFOA, which have an EPA health advisory level.
        - Concentration of the analyte detected.
        - If the level of PFOS plus PFOA exceeds 70 ppt, which is the EPA lifetime Health Advisory Level.
        - Other published toxicity or health effects levels or information.

10. Sample Analysis and Logistics

VDH will utilize a contract laboratory for the PFAS analytical services. The laboratory will ship sample kits, along with sampling instructions, directly to the identified waterworks (sampling sites). VDH, in conjunction with the laboratory, will provide a training video on how to collect the samples. The waterworks staff will collect the samples and return the samples to the laboratory via prepaid shipping labels. The waterworks will not be required to pay for sample analysis or shipping as part of the Sampling Plan.

11. Analytical Method Selection

The laboratory will analyze drinking water samples by EPA Method 533. This

method reports the analytes specified in HB586, whereas EPA Method 537.1 does not report all the analytes specified in HB586 because it does not include PFBA. Other related considerations include:

- The laboratory will report the complete list of 25 analytes for Method 533.
- The laboratory will establish method reporting limits (MRLs) for each analyte based on the lowest concentration of standards used by the laboratory.
- The laboratory will meet NELAC Accreditation requirements.

The laboratory will analyze source water samples using a method employing solid phase extraction, liquid chromatography/mass spectrometry/mass spectrometry (LC/MS/MS), and isotope dilution that will meet the requirements of Table B-15 of the DoD ELAP QSM. The laboratory must analyze source water samples by another method since EPA Method 537.1 and 533 are applicable only to drinking water. Other related considerations include:

- The laboratory will report the same analytes as EPA Method 533.
- The laboratory will use the same MRLs as EPA Method 533 or as agreed by VDH
- The laboratory will hold accreditation for the DoD PFAS method by LC/MS/MS compliant with QSM 5.3 Table B-15.

12. Collecting Existing PFAS Monitoring Data in Virginia Drinking Water

As part of the Sampling Plan, VDH will request waterworks to optionally share existing PFAS monitoring data. Criteria include:

- Sampled and analyzed in 2018 to date
- EPA Methods 533, 537.1, 537.1, a DoD method, proprietary commercial, proprietary commercial with DOD compliance, etc. Also submit the name of the lab and the reporting levels used.
- Samples at entry points, consecutive connection, or raw water
- Analytical work passes QA/QC

13. Modification of the Sampling Plan

VDH will retain flexibility to make minor modifications and amendments to the Sampling Plan as the agency implements it. Minor modifications could include specifying field reagent blanks for all samples, adding EPA's guidelines for responding to situations where PFAS levels (perfluorooctanoic acid (PFOA) + perfluorooctanesulfonic acid (PFOS)) exceed 70 ppt), and replacing one sampling site with another if a waterworks would decline the request to collect a sample or not be using a source or entry point that is currently identified in the plan. VDH will not make substantive changes to the Sampling Plan without informing the VA PFAS Workgroup.

Because there is very limited data on PFAS occurrence in Virginia, VDH may make adjustments as needed to carry out the Sampling Plan described herein. Adjustment could

include changes to the sample locations, waterworks, intakes, sampling method and/or QA/QC samples (if needed). If VDH anticipates the need to make substantive changes to the Sampling Plan, due to factors such as budget, PFAS levels above EPA's Health Advisory Level in one or more locations (indicating a public health risk), or other unforeseen events, VDH will meet with the VA PFAS Workgroup before implementing substantive changes.

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Acronyms

AFFF – Aqueous Fire Fighting Foam
CWS – Community Water System
DEQ – Virginia Department of Environmental Quality
DoD ELAP QSM – Department of Defense Environmental Laboratory Accreditation Program Quality System Manual
VDH – Virginia Department of Health
EP – Entry Point
EP – Entry Point
EPA – U.S. Environmental Protection Agency GIS - Geographic Information System
MRL – Method Reporting Level
NTNC – Nontransient Noncommunity Water System
NELAC – National Environmental Laboratories Accreditation Conference
PFAS – Per- and polyfluoroalkyl substances
PFOA – Perfluorooctanoic acid
PFOS – Perfluorooctane sulfonate
PWS – Public Water System, aka, "waterworks"
SIC – Standard Industrial Classification system
TNC – Transient Noncommunity Water System
UCMR 3 – Unregulated Contaminant Monitoring Rule (3rd Round)

APPENDIX 4 – PFAS Communications Toolkit

Communication Toolkit for VA PFAS Sampling Study

Virginia Department of Health - Office of Drinking Water

Communication Toolkit for VA PFAS Sampling Study

Virginia Department of Health
Office of Drinking Water

In conjunction with the
Virginia PFAS Workgroup

May 2021

Introduction

House Bill (HB) 586 (2020 Acts of Assembly Chapter 0611) seeks to prevent potential adverse health effects and protect public health by studying the occurrence of per- and polyfluoroalkyl substances (PFAS) in drinking water. The legislation requires the State Health Commissioner, who acts through the Virginia Department of Health (VDH), to convene a workgroup to study the occurrence of six specific PFAS (perfluorooctanoic acid (PFOA), perfluorooctane sulfonate (PFOS), perfluorobutyrate (PFBA), perfluoroheptanoic acid (PFHpA), perfluorohexane sulfonate (PFHxS), perfluorononanoic acid (PFNA)[1]) and other PFAS, as deemed necessary, that may be present in drinking water from waterworks, identify possible sources of such contamination and evaluate approaches to regulating PFAS. The workgroup may recommend maximum contaminant levels (MCLs) for inclusion in regulations of the Board of Health applicable to waterworks. The workgroup will report its findings to the Governor and legislative committees by December 1, 2021. To determine the occurrence of PFAS contamination, the legislation requires VDH to sample no more than 50 representative waterworks and major sources of water.

HB1257 (2020 Acts of Assembly Chapter 1097) directs the Board of Health to adopt regulations establishing MCLs for PFOA and PFOS, as well as other PFAS as it deems necessary. The effective date for HB1257 is January 1, 2022, so that the Board can consider the findings and recommendations that come from the work performed to satisfy the requirements in HB586.

To implement HB586, VDH, through its Office of Drinking Water (ODW), formed the Virginia (VA) PFAS Workgroup. In the October 20, 2020, kickoff meeting for the VA PFAS Workgroup, members accepted and formed four subgroups to focus on (1) PFAS Health and Toxicology, (2) PFAS Occurrence and Monitoring, (3) PFAS Policy and Regulations and (4) PFAS Treatment Technologies. These subgroups meet on a monthly basis and provide summaries of their findings and recommendations to the VA PFAS Workgroup during its quarterly meetings.

The Communication Toolkit for VA PFAS Sampling Study consists of the following sections:

1. Purpose
2. PFAS Sampling Study: Data Review, Verification and Validation
3. PFAS Sampling Results: Guidelines for Publication
4. Drinking Water Assessment, Prevention and Response Toolbox for Waterworks
5. Expectations for Waterworks that Receive Results of PFOA + PFOS >70 ppt
6. Fact Sheets and Letter Templates
7. Additional Resources

[1] Many PFAS can exist in various ionic states (for example, acids, anions, cations), which has important implications for their chemical and physical properties. House Bill 586 listed some PFAS in their acid form (PFOA - perfluorooctanoic acid) and others in their anionic form (PFOS – perfluorooctane sulfonate). This Toolkit uses the anionic form of a given PFAS name (e.g., PFOA – perfluorooctanoate; PFOS – perfluorooctane sulfonate), as this is the state in which most PFAS exist in the environment. See the List of Common PFAS in Appendix B and https://pfas-1.itrcweb.org/fact_sheets_page/PFAS_Fact_Sheet_Naming_Conventions_April2020.pdf.

1. Purpose

VDH, in collaboration with VA PFAS Workgroup, developed a VA PFAS Sampling Study (Sampling Study) to sample 50 select waterworks and major water sources in Virginia. VDH will conduct the sampling study between April and the end of June, 2021. In planning the sampling study, VA PFAS Workgroup members also felt the need to develop public awareness material on the presence of PFAS in drinking water. This document, the Communication Toolkit for VA PFAS Sampling Study, summarizes the approach VDH will follow to monitor, evaluate and release results and information from the Sampling Study. It also contains fact sheets, guidelines and other resources for waterworks and local health departments to use to interpret results and respond to inquiries about PFAS, testing and health concerns related to PFAS.

As specified in the legislation, the Sampling Study is limited to drinking water produced by waterworks and major water sources used by waterworks. It does not include water from private wells or other sources. "Waterworks" is defined in state law and means a system that serves piped water for human consumption to at least 15 service connections or 25 or more individuals for at least 60 days out of the year. Code of Virginia § 32.1-167.

2. PFAS Sampling Study: Data Review, Verification and Validation

Upon implementing the Sampling Study, VDH, through ODW, will coordinate sample collection from the representative waterworks and major sources of water. As results of analysis arrive from the contract laboratory, ODW will tabulate PFAS data from the Sampling Study and from other existing PFAS monitoring data that waterworks choose to share with VDH. The Monitoring and Occurrence subgroup will also evaluate the data to assess current levels of PFAS (PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA and other PFAS as deemed necessary) in the Commonwealth's public drinking water.

ODW staff will review laboratory reports in accordance with the Quality Assurance Project Plan (QAPP) that the U.S. Environmental Protection Agency approved for the Sampling Study. The QAPP guides evaluation of the data to determine if the results are valid and usable. It also discusses strategies to evaluate data not meeting the quality control criteria in a way that does not result in unintended biases that may occur if combined with fully compliant data. The QAPP specifies that the Study Director is responsible for determining whether any data is usable as received, is usable after adding appropriate data qualifiers, or is incapable of meeting the applicable quality control criteria even if data qualifiers were employed.

Data review will begin with comparison of the laboratory reports (received as .pdf files) and Electronic Data Deliverable (EDD) files transmitted to ODW to confirm that both documents reflect equivalent data. In addition, ODW will review:

- Each sample report for data qualifiers indicating a data quality problem;
- The field reagent blanks associated with each water sample to confirm the field reagent blank is clean;
- The recovery of analytes near or at the Method Recovery Limit (MRL) to confirm results are within method limits;
- Chain of custody information in the data and compare it with the contents of the laboratory report to confirm sample location, sample collection time and date, and

evaluate sample hold times for compliance with the method requirements; and
- The case narrative for data qualifiers.

If any review identifies a data quality problem, ODW will initiate an in-depth review of the data for the affected water sample and related samples. In addition, ODW will conduct in-depth review of at least 5% of the water samples for quality assurance purposes.

The in-depth review will confirm that each analysis complied with the method requirements, including sample preservation and holding times, instrument performance checks, initial calibration, quality control samples, continuing calibration checks, field duplicates, blank samples, surrogate analyte recovery, internal standards, target analyte identification and quantification, and performance evaluation samples. Based on the in-depth review, ODW will determine if the reported results meet the method requirements and if the data is usable.

A checklist on the lab quality assurance/quality control (QA/QC) procedures is provided in Appendix A.

All water sample results will undergo data review before becoming public facing or sharing with the VA PFAS Workgroup and/or related subgroups.

3. PFAS Sampling Results: Guidelines for Publication

VDH plans to collect, perform QA/QC review and compile all of the data from the Sampling Study, develop a web-based method for sharing results and, in conjunction with the VA PFAS Workgroup, draft the required report for the legislature and Governor before posting the data on its website. VDH does not intend to post results from individual waterworks on its website upon receipt or immediately following review, verification and validation (as described in Section 2) so that the agency and VA PFAS Workgroup have an opportunity to review the dataset as a whole, assess the extent of PFAS in drinking water, complete the requirements in the legislation and, most importantly, provide appropriate context and resources for parties that are interested in the results and their implications for the Commonwealth.

VDH will provide a technical contact to assist waterworks that participate in the sampling with media inquiries. If VDH receives a request for records (i.e., sampling results) before making the data available to the public, under Virginia's Freedom of Information Act (FOIA), Code of Virginia § 2.2-3700 et seq., VDH is required to provide the records unless they are subject to an exemption. Because VDH does not anticipate that the sampling results will qualify for a recognized exemption, ODW will notify the associated waterworks as soon as practicable (typically within 24 hours) when a FOIA request is received so the waterworks can prepare, if necessary, a specific public comment.

4. Drinking Water Assessment, Prevention and Response Toolbox for Waterworks

This toolbox helps public water systems (waterworks) (a) assess per- and polyfluoroalkyl substances (PFAS) contamination, (b) guide responses to test results when compared to the U.S. Environmental Protection Agency's (EPA) drinking water lifetime health advisory level of 70 parts per trillion (ppt) for the concentration of perfluorooctanoate (PFOA) and perfluorooctane

sulfonate (PFOS), individually or combined and (c) evaluate means that, depending on the source of contamination, water source(s) and waterworks capabilities, may prevent or reduce PFAS contamination.

4.1 Proactive tools for assessing and preventing PFAS contamination

- Use the Virginia Department of Health (VDH), Office of Drinking Water (ODW) PFAS website to understand basics and health risks of PFAS
- Assess risk to source water:
    - Proximity to potential sources of PFAS releases to the environment:
        - Industrial facilities that produce, process, or use PFAS chemicals or products in manufacturing or other activities (current or past)
        - Areas where PFAS-containing Class B firefighting foams are stored, used, or released such as airports, military bases and fire stations (current or past)
        - Waste management facilities, such as landfills
        - Wastewater treatment residuals and areas of biosolids production and application (elevated PFAS levels are more likely to be found in residuals and biosolids from wastewater treatment facilities that received wastewater from industrial sources)
    - Source water vulnerability to contamination based on proximity to known, suspected, or potential sources of PFAS contamination such as those listed above
    - If you need source water protection assistance, contact ODW's Division of Technical Services at (804) 864-7500
- Implement measures to reduce risk by:
    - Evaluating potential approaches with stakeholders
    - Raising awareness of PFAS contamination
    - Working with the Virginia Department of Environmental Quality to gain information about facilities with potential PFAS releases and PFAS use/storage/disposal for better understanding and ways to reduce risk
- Sample treated water and at risk sources for PFAS
    - Lab primer to help you find a lab, select a test method and collect a sample

4.2 Recommended response tools – treated water sample results with PFOA+PFOS ≤ 70 ppt

- Notify customers of test results (e.g., monthly bill, mailing, utility website, social media) and include results in the waterworks Consumer Confidence Report (i.e., when reporting 2021 water quality data)
    - Use the VDH letter template (Section 6.1) to help your customers understand that PFOA and PFOS concentrations were at or below the lifetime health advisory, PFAS that are not PFOA and PFOS (e.g., PFBA, PFHpA, PFHxS, PFNA) may have similar health effects and the concentrations associated with those risks are not well known at present, health info is still being developed, including risks for children and pregnant women (more information can be found here), and any next steps you have planned.

- Based on detectable concentrations, evaluate:
    - Risk to source water and implement best management practices (BMPs) (above)
    - Strategies on how to minimize exposure
    - Taking additional source and/or entry point samples
    - Removing any source with levels above the health advisory
- Contact ODW for assistance with activities listed above

4.3 Recommended response tools – treated water sample results with PFOA+PFOS > 70 ppt

- Notify ODW as soon as practicable. If it is after business hours or a weekend, please contact the **Waterworks Emergency After-Hours Call Center at 1-866-531-3068** to establish coordination on public notification, if deemed necessary by ODW and the waterworks, and follow up actions
- Resample to verify levels are above the lifetime health advisory
- Reduce exposure risk by notifying potentially affected customers using the VDH letter template (Section 6.2)
- Identify strategies for decreasing levels in water (e.g., operational, alternate sources, blending)
- Consider additional risk communications and holding a community meeting for potentially impacted residents. Possible resources include:
    - PFAS removal using household water treatment systems at the point-of-use (POU) or point-of-entry (POE)
    - In-home water filtration options
- Identify solutions for waterworks to consistently and reliably reduce PFAS below the lifetime health advisory level (e.g., treatment, removal/remediation of PFAS source)
    - Share in-home treatment options with residents
- Determine options for long term mitigation and treatment
    - Gather data to identify PFAS sources
    - Assess risk to source water and implement BMPs (as listed above)

4.4 Additional information

- EPA list of certified labs
- EPA: information on PFCs
- Agency for Toxic Substances and Disease Registry (ATSDR)
- Under sink treatment systems:
    - The Minnesota Study for point of use systems
    - New York guidance on point of use
- What's in my water: information about PFAS from the American Water Works Association

5. Expectations for Waterworks that Receive Results of PFOA + PFOS >70 ppt

In the event that results come back from a waterworks that indicate the amount of PFOA, PFOS, or PFOA and PFOS combined exceeds 70 parts per trillion (ppt), VDH expects the waterworks to respond in a way that is protective of public health and consistent with the U.S. Environmental Protection Agency's guidance associated with its lifetime health advisory level.

Background

To provide Americans, including the most sensitive populations, with a margin of protection from a lifetime of exposure to perfluorooctanoate (PFOA) and perfluorooctane sulfonate (PFOS) from drinking water, in 2016 EPA established a lifetime health advisory level of 70 parts per trillion (ppt). When both PFOA and PFOS are found in drinking water, the combined concentrations of PFOA and PFOS should be compared with the 70 ppt health advisory level.

Health advisories provide information on contaminants that can cause human health effects and are known or anticipated to occur in drinking water. EPA's health advisories are non-enforceable and non-regulatory and provide technical information to state agencies and other public health officials on health effects, analytical methodologies and treatment technologies associated with drinking water contamination. EPA's lifetime health advisory level for PFOA and PFOS offers a margin of protection for all Americans throughout their life from adverse health effects resulting from exposure to PFOA and PFOS in drinking water.

Virginia does not have a regulatory standard at this time. VDH will follow EPA's recommended actions for waterworks that receive results of PFOA and PFOS that exceed the lifetime health advisory level.

Assess Contamination

Waterworks are expected to conduct confirmation sampling. If results from implementation of the Sampling Plan indicate that drinking water (samples collected at the entry point to the distribution system) contains PFOA and PFOS at individual or combined concentrations greater than 70 ppt, VDH expects the waterworks to undertake additional sampling within two weeks of learning of the result to assess the level, scope and localized source of contamination to inform next steps.

Waterworks should promptly notify and perform confirmation sampling in coordination with the Department of Health (monitoring drinking water quality) and Department of Environmental Quality (identifying the source of contamination).

Inform Consumers

If the average of the initial result and the confirmation sample is greater than EPA's lifetime health advisory level, confirming that drinking water contains PFOA and PFOS at individual or combined concentrations greater than 70 parts per trillion, waterworks should also promptly provide consumers with information about the levels of PFOA and PFOS in their drinking water. This notice should include specific information on the risks to fetuses during pregnancy and breastfed and formula-fed infants from exposure to drinking water with an individual or combined concentration of PFOA and PFOS above EPA's lifetime health advisory level of 70 ppt. In addition, it should identify options that consumers may consider to reduce risk such as seeking an alternative drinking water source, or in the case of parents of formula-fed infants, using formula that does not require adding water.

Conduct Enhanced Monitoring

Following confirmation that PFOA and/or PFOS levels exceed the 70 ppt lifetime health advisory, waterworks should begin a program of monthly monitoring that continues until results are reliably and consistently below 70 ppt. "Reliably and consistently" means that though a waterworks detects contaminants in its water supply, it has sufficient knowledge of the source or extent of the contamination to predict that the lifetime health advisory level would not be exceeded in the future (i.e., wide variations in analytical results or an analytical result which is close to the lifetime health advisory are examples of situations where waterworks would not be reliably and consistently below the lifetime health advisory).

Evaluate and Take Steps to Limit Exposure

Depending on the source of contamination, water source(s) and waterworks capabilities, several options may be available to waterworks to lower concentrations of PFOA and PFOS in the drinking water supply. In some cases, waterworks may be able to reduce concentrations of perfluoroalkyl substances, including PFOA and PFOS, for example, by closing contaminated wells or changing the rates of blending of water sources, where the available quantity of drinking water is not compromised. Alternatively, waterworks can treat source water with activated carbon or high pressure membrane systems (e.g., reverse osmosis) to remove PFOA and PFOS from drinking water. These treatment systems are used by some waterworks today, but should be carefully designed and maintained to ensure that they are effective for treating PFOA and PFOS. In some communities, entities have provided bottled water to consumers while steps to reduce or remove PFOA or PFOS from drinking water or to establish a new water supply are completed.

Many home drinking water treatment units are certified by independent accredited third party organizations against American National Standards Institute (ANSI) standards to verify their contaminant removal claims. NSF International (NSF®) has developed a protocol for NSF/ANSI Standards 53 and 58 that establishes minimum requirements for materials, design and construction, and performance of point-of-use (POU) activated carbon drinking water treatment systems and reverse osmosis systems that are designed to reduce PFOA and PFOS in public water supplies. The protocol has been established to certify systems (e.g., home treatment systems) that meet the minimum requirements. The systems are evaluated for contaminant reduction by challenging them with an influent of 1.5±30% µg/L (total of both PFOA and PFOS) and must reduce this concentration by more than 95% to 0.07 µg/L or less (total of both PFOA and PFOS) throughout the manufacturer's stated life of the treatment system. Product certification to this protocol for testing home treatment systems verifies that devices effectively reduces PFOA and PFOS to acceptable levels.

6. Fact Sheets and Letter Templates

Fact sheet 1 and 2 are presented in Appendix B and C respectively. These are intended for waterworks and local health departments to use to respond to inquiries from the media and/or consumers about PFAS, the Sampling Study and/or health risks associated with PFAS. Fact sheet 1 (Appendix B) contains more general information. Fact sheet 2 (Appendix C) contains more information about health effects. Select the appropriate fact sheet based on the nature of the inquiry and intended audience.

6.1 Letter Template for a Common Message (For PFAS concentration below 70 ppt):

The template is available for waterworks/localities to use to provide general information to consumers and/or the media in response to an inquiry (media or FOIA) about the sample result/results that came from the PFAS Sampling Study that are specific to the waterworks. If the sum of PFOA + PFOS exceeds the health advisory level, the waterworks should follow the guidelines in Section 5.

Thank you for inquiring about the results of the sampling for per- and polyfluoroalkyl substances (PFAS) in the drinking water at **LOCATION/WATERWORKS NAME**.

House Bill 586 (2020) initiated a study of PFAS from no more than 50 waterworks and/or water sources in Virginia. The Virginia Department of Health (VDH), through its Office of Drinking Water, and a work group with representatives from waterworks, advocacy groups and citizens, selected the waterworks and water sources to generate data that VDH could use to begin the process of establishing appropriate regulatory requirements for PFAS in drinking water. The goal of the study is to (1) protect public health and (2) begin to understand the extent and nature of PFAS contamination in drinking water to minimize risk. VDH is working closely with waterworks throughout the Commonwealth, including **WATERWORKS NAME,** to ensure water is safe, complies with all State and Federal drinking water standards and meets other recommended advisory levels for specified contaminants.

In [*month*], **WATERWORKS NAME** collected [*a sample / # samples*] of water and submitted [*it / them*] to a VDH-contracted laboratory. The laboratory used an analytical method approved by the U.S. Environmental Protection Agency (EPA) to analyze the sample[*s*] for the presence of 25 individual PFAS. Following analysis, the laboratory provided the results to VDH and **WATERWORKS NAME** for review. A copy of the results is attached.

EPA has not set a regulatory limit for any PFAS in drinking water. However, EPA has established a lifetime health advisory level of 70 parts per trillion (ppt) for two specific PFAS, perfluorooctanoate (PFOA) and perfluorooctane sulfonate (PFOS). The concentration of PFOA and PFOS found at **WATERWORKS NAME** during this study was **XXX** ppt, which is below the lifetime health advisory level. There is no immediate adverse health concern due to the presence of PFAS at the observed concentrations. [*The **XXXXX** City / County Government, along with*] **WATERWORKS NAME** and VDH continue to stress the importance of source water protection and our collaborative role in keeping drinking water supplies safe.

VDH is continuing to evaluate this issue to determine whether and how PFAS should be regulated in Virginia. Additionally, the Virginia Department of Environmental Quality (DEQ) is assessing potential environmental sources of PFAS in Virginia. Updates on the VDH and DEQ efforts can be found at the following websites:

https://www.vdh.virginia.gov/drinking-water/pfas/
https://www.deq.virginia.gov/get-involved/the-environment-you/per-and-polyfluoroalkyl-substances-pfas

Please feel free to contact me if you have any other questions.

Sincerely,

6.2 Letter Template for a Common Message (For PFAS concentration above 70 ppt):

The template is available for waterworks/localities to use to provide general information to consumers and/or the media in response to an inquiry (media or FOIA) about the sample result/results that came from the PFAS Sampling Study that are specific to the waterworks.

Thank you for inquiring about the results of the sampling for per- and polyfluoroalkyl substances (PFAS) in the drinking water at **LOCATION/WATERWORKS NAME**.

House Bill 586 (2020) initiated a study of PFAS from no more than 50 waterworks and/or water sources in Virginia. The Virginia Department of Health (VDH), through its Office of Drinking Water, and a work group with representatives from waterworks, advocacy groups and citizens, selected the waterworks and water sources to generate data that VDH could use to begin the process of establishing appropriate regulatory requirements for PFAS in drinking water. The goal of the study is to (1) protect public health and (2) begin to understand the extent and nature of PFAS contamination in drinking water to minimize risk. VDH is working closely with waterworks throughout the Commonwealth, including **WATERWORKS NAME,** to ensure water is safe, complies with all State and Federal drinking water standards and meets other recommended advisory levels for specified contaminants.

In [*month*], **WATERWORKS NAME** collected [*a sample / # samples*] of water and submitted [*it / them*] to a VDH-contracted laboratory. The laboratory used an analytical method approved by the U.S. Environmental Protection Agency (EPA) to analyze the sample[*s*] for the presence of 25 individual PFAS. Following analysis, the laboratory provided the results to VDH and **WATERWORKS NAME** for review. A copy of the results is attached.

EPA has not set a regulatory limit for any PFAS in drinking water. However, EPA has established a lifetime health advisory level of 70 parts per trillion (ppt) for two specific PFAS, perfluorooctanoate (PFOA) and perfluorooctane sulfonate (PFOS). The sum concentration of PFOA and PFOS found at **WATERWORKS NAME** during this study was **XXX** ppt, which is above EPA's 70 ppt lifetime health advisory level. Follow-up sampling confirmed the result.

Although EPA's health advisory level for PFOA and PFOS is not an enforceable regulatory standard, it offers a margin of protection for all Americans from adverse health effects resulting from a lifetime's exposure to PFOA and PFOS in drinking water. Out of an abundance of caution, **WATERWORKS NAME** has voluntarily decided to use EPA's lifetime health advisory as a basis for taking action to address the presence of PFOA and PFOS in the drinking water it provides. The initial actions **WATERWORKS NAME** will take, in coordination with [***XXXXX*** *City / County Government and*] VDH, include the following:

[*OPTIONS A WATERWORKS MAY TAKE IN RESPONSE TO PFOA/PFOS > 70 PPT - MODIFY LIST AS NEEDED TO REFECT ACTUAL CONDITIONS/SITUATION*]

- Collecting additional samples within two weeks of learning of the result to assess the level, scope and nature of contamination to inform next steps;
- Working with the Virginia Department of Environmental Quality to identify the source of contamination;
- Evaluating ways to reduce the level of PFOA and PFOS in drinking water we provide to consumers;
- Promptly providing consumers with information from VDH and EPA about the known health risks associated with PFOA and PFOS in their drinking water, including specific information on the risks to fetuses during pregnancy and breastfed and formula-fed infants from exposure to drinking water with an individual or combined concentration of PFOA and PFOS above EPA's lifetime health advisory level of 70 ppt (see https://www.epa.gov/pfas); and
- Conducting additional testing to monitor PFOA and PFOS levels in drinking water that will consider the current state of knowledge about risk and health effects.

Based on the results of VDH's PFAS study, EPA's lifetime health advisory and the development of any regulatory limits for PFOA, PFOS, or other PFAS, the **WATERWORKS NAME** response to this situation is likely to evolve over time.

[*Consumers / As a consumer, you*] may want to consider using an alternative water source that is free of PFAS for any activity in which [*they / you*] might ingest water. These activities include drinking, food preparation, brushing teeth and preparing infant formula. [*They / You*] might also consider an in-home filtration system. [*WATERWORKS MAY ADD A STATEMENT HERE ADDRESSING THE COST OF IN-HOME TREATMENT, IF IT WILL BE THE CONSUMER'S RESPONSIBILITY TO INSTALL/MAINTAIN OR IF THE WATERWORKS WILL PROVIDE FULL OR PARTIAL SUPPORT (AND FOR HOW LONG).*] Granular activated carbon filters or reverse osmosis water treatment devices are technologies that are capable of reducing the level of PFAS in drinking water. If a treatment is used, it is important to follow the manufacturer's guidelines for maintenance and operation. NSF International, an independent and accredited organization, certifies products proven effective for reducing PFOA and PFOS below the EPA lifetime health advisory level of 70 ppt, but they may not remove other types of PFAS. (http://info.nsf.org/Certified/DWTU/)

VDH is continuing to evaluate this issue to determine whether and how PFAS should be regulated in Virginia. Additionally, the Virginia Department of Environmental Quality (DEQ) is assessing potential environmental sources of PFAS in Virginia. Updates on the VDH and DEQ efforts can be found at the following websites:

https://www.vdh.virginia.gov/drinking-water/pfas/
https://www.deq.virginia.gov/get-involved/the-environment-you/per-and-polyfluoroalkyl-substances-pfas

Please feel free to contact me if you have any other questions.

Sincerely,

7. **Additional Resources:**

Technical Support

1. The VDH Local Health Districts can assist with local inquiries on PFAS and associated health risks. LHDs locations can be found at https://www.vdh.virginia.gov/health-department-locator/
2. The VDH Office of Drinking Water Regional Field Offices can assist with technical and engineering assistance. More information on the ODW's regional field offices is available at https://www.vdh.virginia.gov/content/uploads/sites/14/2020/04/ODW_Website_Map.pdf
3. PFAS resources for states are available at https://www.epa.gov/research-states/pfas-resources-states

Funding for Treating PFAS in Drinking Water

1. Waterworks interested in installing new treatment technologies can apply to use funding available through ODW's Drinking Water State Revolving Fund (DWSRF) program. More information on this can be found at https://www.vdh.virginia.gov/drinking-water/drinking-water-state-revolving-fund-program/

General Information on PFAS

1. EPA's PFAS webpage: https://www.epa.gov/pfas
2. Interstate Technology Regulatory Council (ITRC) PFAS webpage: https://pfas-1.itrcweb.org
3. ATSDR PFAS webpage: https://www.atsdr.cdc.gov/pfas/index.html

Other State Resources

1. The Association of State Drinking Water Administrators (ASDWA) provides a good overview of states' efforts on PFAs in drinking water: https://www.asdwa.org/pfas/
2. The Environment Council of States (ECOS) webpage with PFAS information: https://www.ecos.org/pfas/
3. Michigan: https://www.environmentalcouncil.org/pfas_in_michigan

Appendix - A

Table 1. Initial Demonstration of Capability (IDC) Quality Control Requirements*

Method Reference	Requirement	Specification and Frequency	Acceptance Criteria
Section 10.2.2	Establish retention times for branched isomers	Each time chromatographic conditions change	All isomers of each analyte must elute within the same MRM window.
Section 9.1.1	Demonstration of low system background	Analyze a Laboratory Reagent Blank (LRB) after the highest standard in the calibration range.	Demonstrate that the method analytes are less than one-third of the Minimum Reporting Level (MRL).
Section 9.1.2	Demonstration of precision	Extract and analyze 7 replicate Laboratory Fortified Blanks (LFBs) near the mid-range concentration.	Percent relative standard deviation must be ≤20%.
Section 9.1.3	Demonstration of accuracy	Calculate mean recovery for replicates used in Section 9.1.2.	Mean recovery within 70–130% of the true value.
Section 9.1.4	MRL confirmation	Fortify and analyze 7 replicate LFBs at the proposed MRL concentration. Confirm that the Upper Prediction Interval of Results (PIR) and Lower PIR meet the recovery criteria.	Upper PIR ≤150% Lower PIR ≥50%
Section 9.1.5	Calibration Verification	Analyze mid-level QCS.	Results must be within 70–130% of the true value.

Table 2. Ongoing Quality Control Requirements*

Method Reference	Requirement	Specification and Frequency	Acceptance Criteria
Section 10.3	Initial calibration	Use the isotope dilution calibration technique to generate a linear or quadratic calibration curve. Use at least 5 standard concentrations. Evaluate the calibration curve as described in Section 10.3.5.	When each calibration standard is calculated as an unknown using the calibration curve, analytes fortified at or below the MRL should be within 50–150% of the true value. Analytes fortified at all other levels should be within 70–130% of the true value.
Section 9.2.1	Laboratory Reagent Blank (LRB)	Include one LRB with each Extraction Batch. Analyze one LRB with each Analysis Batch.	Demonstrate that all method analytes are below one-third the Minimum Reporting Level (MRL) and that possible interference from reagents and glassware do not prevent identification and quantitation of method analytes.
Section 9.2.3	Laboratory Fortified Blank	Include one LFB with each Extraction Batch.	For analytes fortified at concentrations ≤2 x the MRL, the result must be within 50–150% of the true value; 70– 130% of the true value if fortified at concentrations greater than 2 x the MRL.
Section 10.4	Continuing Calibration Check (CCC)	Verify initial calibration by analyzing a low-level CCC (concentrations at or below the MRL for each analyte) at the beginning of each Analysis Batch. Subsequent CCCs are required after every tenth field sample and to complete the batch.	The lowest level CCC must be within 50–150% of the true value. All other levels must be within 70–130% of the true value.

Method Reference	Requirement	Specification and Frequency	Acceptance Criteria
Section 9.2.4	Isotope performance standards	Isotope performance standards are added to all standards and sample extracts.	Peak area counts for each isotope performance standard must be within 50–150% of the average peak area in the initial calibration.
Section 9.2.5	Isotope dilution analogues	Isotope dilution analogues are added to all samples prior to extraction.	50%–200% recovery for each analogue
Section 9.2.6	Laboratory Fortified Sample Matrix (LFSM)	Include one LFSM per Extraction Batch. Fortify the LFSM with method analytes at a concentration close to but greater than the native concentrations (if known).	For analytes fortified at concentrations ≤2 x the MRL, the result must be within 50–150% of the true value; 70– 130% of the true value if fortified at concentrations greater than 2 x the MRL.
Section 9.2.7	Laboratory Fortified Sample Matrix Duplicate (LFSMD) or Field Duplicate (FD)	Include at least one LFSMD or FD with each Extraction Batch.	For LFSMDs or FDs, relative percent differences must be ≤30% (≤50% if analyte concentration ≤2 x the MRL).
Section 9.2.8	Field Reagent Blank (FRB)	Analyze the FRB if any analyte is detected in the associated field samples.	If an analyte detected in the field sample is present in the associated FRB at greater than one-third the MRL, the results for that analyte are invalid.
Section 9.2.9	Calibration Verification using QCS	Perform a Calibration Verification at least quarterly.	Results must be within 70–130% of the true value.

*Source: USEPA Method 533 publication

APPENDIX B
FACT SHEET FOR CONSUMERS

Virginia Department of Health Office of Drinking Water
Per- and Polyfluoroalkyl Substances (PFAS) Fact Sheet

May 2021

What are Per- and Polyfluoroalkyl Substances (PFAS)?

Per- and polyfluoroalkyl substances (PFAS) are a large family of man-made chemicals that have been used worldwide, including the United States, in consumer products, industrial applications and in firefighting since the 1940s. There are between 6,000 and 10,000 different chemical compounds in the PFAS family and they are used to make products that resist heat, oil, stains, grease and water. Perfluorooctanoate (PFOA) and perfluorooctane sulfonate (PFOS) were the two most commonly produced PFAS historically and are currently the most studied chemicals in the PFAS family. PFOA and PFOS are no longer manufactured in the United States, but other types of PFAS have been developed to take their place. In general, chemicals in the PFAS family:

- are stable and many do not break down easily in the environment (they are persistent);
- do not occur naturally, yet are widespread in the environment because of their broad uses;
- may be found in people, wildlife and fish world-wide; and
- can build up in biological tissues over time (people, wildlife, fish) if exposure continues (they bioaccumulate).

Are PFAS harmful and how can PFAS affect people's health?

Human health effects from PFAS exposure are not completely understood because all of the individual chemicals in the PFAS family have not been examined to determine the health effects. Studies have shown that exposure to some PFAS may affect developmental stages (growth, learning, behavior) of infants and older children; lower a woman's chance of pregnancy; disrupt the body's hormones; increase cholesterol; and increase cancer risk. Some scientific studies suggest that certain PFAS may affect different systems in the body. However, although these same scientific studies have shown, for example, PFAS to be associated with increased cholesterol levels in humans, the studies have not, at this time, shown an association between the presence of these compounds and any increased risk of heart disease. Scientists are working to better understand how exposure to PFAS might affect people's health – especially how exposure to PFAS in water, food and other exposure pathways may be harmful. At this time, scientists are still learning about the health effects of exposures to mixtures of PFAS and need more time to study health effects of many distinct PFAS.

How can I be exposed to PFAS?

Because PFAS are man-made, they can be found near areas where they are (or were) manufactured; in some industrial applications, such as electroplating, textiles, pulp and paper; and/or in some manufactured products. Since PFOA and PFOS are no longer manufactured in the U.S., other types of PFAS have replaced PFOA and PFOS in some products. Many common consumer and industrial products still contain PFAS, including some:

- nonstick cookware
- food packaging (microwave popcorn bags, fast food wrappers, sliced cheese wrappers, pizza boxes)
- stain-resistant carpets, fabrics and water-resistant clothing
- paints, varnishes and sealants
- cosmetics, toothpaste and dental floss
- fire-fighting foams

Ingestion (swallowing) of food or water containing PFAS is the exposure route of primary concern. PFAS exposure by contact with PFAS-containing compounds through dermal absorption (touching and

passing through the skin) and inhalation during showering/bathing are lesser human health concerns at this time.

How do PFAS get into drinking water?

A drinking water source may be contaminated by PFAS from a specific source such as a PFAS manufacturer, industrial user of PFAS, air emissions containing PFAS, wastewater discharges containing PFAS, landfill leachate containing PFAS and/or airports and firefighter training facilities that used firefighting foam containing PFAS. It is also possible for a drinking water source to become contaminated with PFAS due to precipitation, because of the presence of PFAS in the environment.

What is a Lifetime Health Advisory and what Lifetime Health Advisories have been established for PFAS?

A Lifetime Health Advisory (LHA) is just that, an advisory. It is not a primary drinking water standard (also called a Maximum Contaminant Level, or MCL) which is an enforceable regulatory standard under the Safe Drinking Water Act. The U.S. Environmental Protection Agency (EPA) has established a LHA for two types of PFAS, PFOA and PFOS, at 70 parts per trillion, individually or combined. The LHA is not a level that guarantees there will be no risk of harm from PFAS if a person stays below that level for their entire lives, nor is it a level that guarantees an increased risk of harm from PFAS if that level is exceeded. Current scientific data indicates that an LHA will be protective of most typical water users, including pregnant and nursing women, young children and the elderly. The LHA is based on long-term exposure, on the order of 70 years.

For perspective, one part per trillion is the equivalent of four grains of sugar in an Olympic sized swimming pool, or the equivalent of one second in 32,000 years. EPA has not established a short-term health advisory or a MCL for PFOA, PFOS, or other PFAS.

What is EPA doing about PFAS?

Through EPA's PFAS Action Plan, the Agency has made a final determination to regulate PFOA and PFOS and is moving forward with developing and implementing enforceable drinking water standards (MCLs) for these (and possibly other) PFAS. EPA also proposed preliminary groundwater remediation goals for PFOA and PFOS at 70 ppt (individually or combined) in areas where groundwater may be used for drinking water. EPA is also implementing a national sampling program between 2023 and 2025, called the Unregulated Contaminant Monitoring Rule 5, that will have waterworks collect and analyze samples to determine if 29 specified PFAS are present in drinking water and, if so, in what concentrations.

What is the Virginia Department of Health (VDH) doing about PFAS?

From 2013 to 2015, as part of EPA's national sampling program, Unregulated Contaminant Monitoring Rule 3, Virginia waterworks conducted testing for six (6) PFAS, including PFOA and PFOS, at all waterworks that served more than 10,000 persons and at some smaller waterworks randomly selected by EPA.

- Of 498 samples collected and analyzed, two (2) samples from two (2) waterworks returned detections of PFAS: one with perfluoroheptanoic acid (PFHpA) at 12 ppt; the other with PFOA at 22 ppt. None of the samples exceeded EPA's LHA of 70 ppt for PFOA and/or PFOS.
- Both waterworks collected follow-up samples, but neither detected PFAS in those samples.
- Advances in analytical capabilities are now able to detect lower levels of PFAS and more specific compounds within the broader family of PFAS.

While awaiting national guidance from EPA regarding MCLs or other enforceable regulatory limits for PFAS, Virginia has developed a multi-faceted strategy to begin to assess and address PFAS in drinking

water. As part of this strategy, VDH has:

1. Convened a Virginia PFAS Workgroup with stakeholders from waterworks (small, medium and large), non-governmental environmental organizations, consumer advocacy groups, chemical manufacturers and other subject matter experts.
2. Planned a PFAS sampling study of 50 waterworks and major sources of drinking water that began in the second quarter of 2021 and includes:
   - The 17 largest community waterworks (by population served) which collectively serve over 50% of the State's residents;
   - Waterworks that use groundwater as their source and have well(s) less than a mile from a potential source of PFAS; and
   - Waterworks not among the 17 largest waterworks that have a surface water intake located in an area with higher vulnerability to potential PFAS sources.
3. With the drinking water study underway, VDH is actively working with stakeholder groups and the Virginia Department of Environmental Quality on studying and regulating PFAS, including recommendations for establishing enforceable regulatory limits for PFOA, PFOS and other chemicals and, if needed, what those limits should be.

How can I reduce my exposure to PFAS?

Because PFAS are present in so many consumer products and throughout our environment, one cannot reasonably expect to prevent PFAS exposure altogether. However, in addition to exercising consumer choices to minimize exposure, some steps can be taken to reduce exposure to PFAS in drinking water:

- Contact your drinking water provider to ask for information about PFAS levels in your drinking water.
- If your drinking water contains levels of PFOA or PFOS greater than the EPA LHA of 70 ppt, either individually or combined, consider using an alternative water source for any activity in which you might swallow water. These activities include drinking, food preparation, brushing teeth and preparing infant formula.
- Water with a PFOA and/or PFOS level greater than the LHA is understood to be safe for bathing, showering or washing clothes and cleaning.
- NSF approved activated carbon filtration or reverse osmosis membranes are effective in reducing PFOA and PFOS in water supplies (if the manufacturer's recommended usage instructions are followed).
- Read consumer product labels. If they include information about PFAS in the products, avoid using those with PFAS. Note that not all products have this information.

How can I learn more about PFAS?

U.S. Environmental Protection Agency	
- Basic Information about PFAS	https://www.epa.gov/pfas
- Drinking Water PFOA and PFOS Lifetime Health Advisory	https://www.epa.gov/ground-water-and-drinking-water/drinking-water-health-advisories-pfoa-and-pfos
- Technical Fact Sheet – PFOS and PFOA	https://www.epa.gov/sites/production/files/2017-12/documents/ffrrofactsheet_contaminants_pfos_pfoa_11-20-17_508_0.pdf
VDH Office of Drinking Water	https://www.vdh.virginia.gov/drinking-water/pfas
VA Dept. of Environmental Quality	https://www.deq.virginia.gov/get-involved/the-environment-you/per-and-polyfluoroalkyl-substances-pfas
U.S. Agency for Toxic Substances and Disease Registry	https://www.atsdr.cdc.gov/pfas/
CDC ATSDR PFAS page	https://www.cdc.gov/exposurereport/index.html

Food and Drug Administration	https://www.fda.gov/food/newsevents/constituentupdates/ucm479465.htm
National Toxicology Program	https://ntp.niehs.nih.gov/pubhealth/hat/noms/pfoa/index.html
Interstate Technology Regulatory Council (IRTC)	https://pfas-1.itrcweb.org/

List of Common PFAS and their Abbreviations

Abbreviation	Chemical Name (acid and anionic (salt) versions)
PFOS	Perfluorooctane sulfonic acid / Perfluorooctane sulfonate
PFOA (or C8)	Perfluorooctanoic acid / Perfluorooctanoate
PFNA	Perfluorononanoic acid / Perfluorononanoate
PFDA	Perfluorodecanoic acid / Perfluorononanoate
PFOSA (or FOSA)	Perfluorooctane sulfonamide
PFHxS	Perfluorohexane sulfonic acid / Perfluorohexane sulfonate
PFHpA	Perfluoroheptanoic acid / Perfluoroheptanoate
PFBA	Perfluorobutanoic acid

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APPENDIX C
ALTERNATIVE FACT SHEET FOR CONSUMERS

General Information re Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water

• Per- and polyfluoroalkyl substances (PFAS) are a family of chemicals with many commercial and industrial uses.
• Certain PFAS have been associated with a variety of adverse health effects in humans, but it has not been definitively established that PFAS cause these effects.
• Six states, including Michigan, Massachusetts and New Jersey, have established drinking water regulations for specific compounds within the PFAS family, including PFOA (perfluorooctanoate), PFOS (perfluorooctane sulfonate) and PFNA (perfluorononanoate). Virginia, through the State Board of Health and Virginia Department of Health (VDH), is conducting research through occurrence monitoring to determine levels of PFAS contamination in drinking water and major water sources. VDH formed a stakeholder workgroup to help assess the data and recommend whether and how to establish regulations for PFOA and PFOS in drinking water in Virginia, as directed by legislation that passed during the 2020 General Assembly Session.

What are PFAS?
Per- and polyfluoroalkyl substances (PFAS) are a complex family of manmade fluorinated organic chemicals which have been produced since the mid-20th century. It has been estimated that the PFAS family may include approximately 6,000 to 10,000 chemicals, with a recent inventory identifying more than 4,700 PFAS that could have been, or may be, on the global market. The unique physical and chemical properties of PFAS impart oil and water repellency, temperature resistance and friction reduction to a wide range of products used by consumers and industry. For example, PFAS, have been used in coatings for textiles, paper products and cookware, and to formulate some firefighting foams. They have a range of applications in the aerospace, photographic imaging, semiconductor, automotive, construction, electronics and aviation industries.

How can I be exposed to PFAS?
While consumer products and food (via packaging) are a common source of exposure to these chemicals for most people, drinking water can be an additional source of exposure in communities where these chemicals have contaminated water supplies. Such contamination is typically localized and associated with a specific facility, for example, an airfield at which these chemicals were used for firefighting or a facility where they were produced or used. PFAS can enter drinking water through industrial release to water, air, or soil; discharges from sewage treatment plants; land application of contaminated sludge; and use of fire-fighting foam. Recent scientific investigations have indicated that PFAS present in the atmosphere can also lead to PFAS contamination in precipitation.

Are PFAS harmful and how can PFAS affect people's health?
Research and information on the health effects of PFAS in humans and animals is continually becoming available. In animal testing, some PFAS have been found to cause developmental, immune, neurobehavioral, liver, endocrine and metabolic toxicity, generally at levels well above known human exposures. Some studies of the general population, communities with drinking water exposures to certain PFAS, and exposed workers suggest that PFAS increase the risk of a number of health effects. The most consistent human health effect findings for PFOA (perfluorooctanoate) – the most well-studied of the PFAS – include increases in serum cholesterol, some liver enzymes and uric acid levels. For PFOS (perfluorooctane sulfonate), another well-studied PFAS, the most consistently found human health effects include increased serum cholesterol and uric acid levels. PFOA and PFOS have also been associated with decreased antibody response following vaccination.

How can PFAS affect children?
In animal testing, some PFAS cause developmental effects. In humans, exposure to PFAS at elevated levels before birth or in early childhood may result in decreased birth weight, decreased immune responses and hormonal effects later in life. More research is needed to understand the role of PFAS in developmental effects.

Infants and children consume more water per body weight than older individuals, so their exposures may be higher than adults in communities with elevated levels of PFAS in drinking water. They may also be more sensitive to the effects of PFAS.

When PFAS are elevated in a drinking water supply, it is advisable to use alternative water sources to prepare infant formula for bottle-fed babies. Beverages for infants and children, such as juice made from concentrate, should also be prepared with water from alternate sources. PFAS have also been discovered in breast milk in some cases. Based on the scientific understanding at this time, since the benefits of breast-feeding are well-established, infants should continue to be breast-fed.

What is a Lifetime Health Advisory (LHA)? Are there LHAs for PFAS?
In 2016, the U.S. Environmental Protection Agency (EPA) issued Lifetime Health Advisories (LHA) for PFOA and PFOS in drinking water at 70 parts per trillion (ppt), individually or combined. A LHA is non-

enforceable guidance that identifies the concentration of a contaminant in drinking water at which EPA has concluded adverse health effects are not anticipated to occur during a person's lifetime. EPA began a process to establish enforceable regulatory limits for PFOA and PFOS in drinking water in 2021.

Some states have begun to establish their own limits for specific PFAS. For example, in 2018, New Jersey became the first state to establish a drinking water standard for a PFAS chemical when it set a Maximum Contaminant Level (MCL) for PFNA (perfluorononanoate) at 13 ppt. The New Jersey Department of Environmental Protection has also established enforceable MCLs for PFOA (14 ppt) and PFOS (13 ppt).

What is being done to address PFAS in drinking water in Virginia?
During the 2020 General Assembly Session, the Legislature passed and Governor Northam signed House Bill 586 (2020 Acts of Assembly Chapter 0611), making it effective on July 1, 2020. The legislation requires the State Health Commissioner to convene a workgroup to study the occurrence of PFAS in drinking water in Virginia. The workgroup is responsible for (1) determining current levels of PFAS in the Commonwealth's public drinking water based on samples from no more than 50 representative waterworks and major sources of water; (2) identifying possible sources of PFAS; (3) evaluating existing approaches to regulating PFAS in drinking water, including regulatory approaches adopted by other states and the federal government; and (4) at its discretion, developing recommendations for specific regulatory limits for PFAS, which the Board of Health may decide to incorporate in the Waterworks Regulations through the rulemaking process outlined in Virginia's Administrative Process Act.

In October 2020, the Virginia Department of Health (VDH) convened a Virginia PFAS Workgroup comprising of representatives from waterworks, advocacy groups, chemical manufacturers, non-governmental environmental organizations, subject matter experts and the general public. More details on this can be found at www.vdh.virginia.gov/drinking-water/pfas.

VDH, through the Office of Drinking Water (ODW) and the Virginia PFAS Workgroup, developed a plan to sample drinking water and major sources of water in Virginia. The PFAS Sampling & Monitoring Study in Virginia Drinking Water (Sampling Plan), identifies 50 waterworks and major sources of water for sampling based on factors including the population served, proximity to potential sources of PFAS contamination and geographic location. Implementation of the Sampling Plan enables VDH to begin to assess the scope of PFAS contamination in drinking water in the Commonwealth, subject to the limitations set by the General Assembly in the legislation.

VDH, in conjunction with the Virginia PFAS Workgroup, will compile and review the results from around the Commonwealth, ensuring they meet appropriate quality assurance/quality control guidelines. VDH and the Virginia PFAS Workgroup will use the results and other research required by the legislation to complete and submit a report to the Governor and General Assembly about the presence of PFAS in drinking water in Virginia by December 1, 2021.

A second bill, House Bill 1257 (2020 Acts of Assembly Chapter 1097) directs the Board of Health to adopt regulations establishing MCLs for PFOA and PFOS, as well as any other PFAS it deems necessary. The effective date for HB1257 is January 1, 2022, so that the Board can consider the findings and recommendations that come from the work performed to satisfy the requirements in House Bill 586.

How do I know if I have PFAS in my drinking water?
Large public waterworks in Virginia and the rest of the country, along with a subset of smaller waterworks, were required to test for some PFAS from 2013 to 2015 as part of the EPA Third Unregulated Contaminant Monitoring Rule implementation (UCMR3). In Virginia, two waterworks detected PFAS during UCMR3, but neither found concentrations above the reporting level and PFAS was not detected during follow-up sampling. In 2019, Congress passed a law requiring at least 29 PFAS to be included in the Fifth Unregulated

Contaminant Monitoring Rule implementation (UCMR5). UCMR5 sampling is scheduled to take place between 2023 and 2025.

VDH recently completed testing at 50 waterworks and major sources of water supply using an analysis method that detects 25 different types of PFAS in lower concentrations than UCMR3. These results will be available to the public on VDH's website. Additionally, this information can be obtained directly from the waterworks that serves your area if they participated in the study. If you use water from a private well, the only way to know whether it has PFAS is to have it tested. To find a laboratory certified to test, you can contact the Virginia Division of Consolidated Laboratory Services at (804) 648-4480 or at https://dgs.virginia.gov/division-of-consolidated-laboratory-services/certification-accreditation/find-a-lab/

What should I do if I am concerned about PFAS in my drinking water?
PFAS are not removed from water by boiling. If tap or well water is found to contain PFAS, people may choose to use home water filters or other alternate water sources for drinking and cooking to reduce exposure to PFAS. However, PFAS has been detected in some brands of bottled water and use of home filtering technologies does not guarantee that all PFAS will be removed from filtered water.

Granular activated carbon filters or reverse osmosis water treatment devices are technologies that are capable of reducing the level of PFAS in drinking water. If a treatment is used, it is important to follow the manufacturer's guidelines for maintenance and operation. NSF International, an independent and accredited organization, certifies products proven effective for reducing PFOA and PFOS below the EPA LHA of 70 ppt, but they may not remove other types of PFAS. (http://info.nsf.org/Certified/DWTU/).

What can blood testing for PFAS tell me?
Since 1999, the U.S. Centers for Disease Control (CDC) has measured several types of PFAS in the U.S. population as part of the National Health and Nutrition Examination Survey (NHANES). NHANES is a survey that measures the health and nutritional status of adults and children in the United States. With the decrease in production and use of some types of PFAS, the national levels of these types of PFAS have also dropped over time. From 1999 to 2014, blood PFOA and PFOS levels declined by more than 60% and 80%, respectively (www.cdc.gov/exposurereport). Nevertheless, the general U.S. population had average blood serum levels of 1.4-2.1 parts per billion (ppb) for PFOA and 4.3-6.3 ppb for PFOS between 2011–2018 (https://www.cdc.gov/exposurereport/pfas_early_release.html).

PFAS can be measured in your blood serum but this is not a routine test. While a blood test may indicate whether you have been exposed to PFAS, results cannot be used to predict your health effects nor can they be linked to specific health problems. Also, test results alone cannot be used to specifically identify sources of exposure, and there is no treatment to reduce levels of PFAS in blood. This information can be used to determine if the levels of PFAS in your blood are higher than national background levels. For example, if your concentration is higher than the 95th percentile, this means your blood serum concentration is higher than the concentration found in 95% of the U.S. population.

Additional Resources:

Basic Information about PFAS from EPA
https://www.epa.gov/pfas

EPA's Drinking Water PFOA and PFOS Lifetime Health Advisory
https://www.epa.gov/ground-water-and-drinking-water/drinking-water-health-advisories-pfoa-and-pfos

EPA's Technical Fact Sheet – PFOS and PFOA
https://www.epa.gov/sites/production/files/2017- 12/documents/ffrrofactsheet_contaminants_pfos_pfoa_11-

20-17_508_0.pdf

Virginia Department of Health PFAS website
https://www.vdh.virginia.gov/drinking-water/pfas

Centers for Disease Control and Prevention
https://www.atsdr.cdc.gov/pfas/

https://www.cdc.gov/exposurereport/index.html

Food and Drug Administration
https://www.fda.gov/food/newsevents/constituentupdates/ucm479465.htm

National Toxicology Program
https://ntp.niehs.nih.gov/pubhealth/hat/noms/pfoa/index.html

Interstate Technology Regulatory Council (IRTC)
https://pfas-1.itrcweb.org

APPENDIX 5 – PFAS LITERATURE REVIEW by OLD DOMINION UNIVERSITY

The Study of the Occurrence of Per- and Polyfluoroalkyl Substances (PFAS) in the Commonwealth's Public Drinking Water

Virginia Department of Health
Office of Drinking Water
September 2021

The Study of the Occurrence of Per- and Polyfluoroalkyl Substances (PFAS) in the Commonwealth's Public Drinking Water

Virginia Department of Health
Office of Drinking Water

In conjunction with the

Community & Environmental Health
Old Dominion University

&

Virginia PFAS Workgroup

September 2021

Table of Contents

List of Figures

List of Tables

Acronyms

EPA	Environmental Protection Agency (U.S.)
FHxSA	Fluorohexanesulphone amide
GAC	Granular activated carbon
GenX	Generation X
IEX	Ion exchange
ITRC	Interstate Technology and Regulatory Council
LOAEL	Lowest-observed-adverse effect level
MCL	Maximum contaminant limit
ng/L	Nanograms per liter, equivalent to parts per trillion
NHANES	National Health and Nutrition Examination Survey
NOAEL	No-observed-adverse-effect level
PFAA	Perfluorinated alkyl acids
PFAS	Per- and polyfluoroalkyl substances
PFBA	Perfluorobutyrate
PFBS	Perfluorobutanesulfonic acid
PFDA	Perfluorodecanoic acid
PFCA	Perfluorinated carboxylic acid
PFECA	Perfluoropolyether carboxylic acids
PFHpA	Perfluoroheptanoic acid
PFHxS	Perfluorohexanesulphonic acid
PFNA	Perfluorononanoic acid
PFOA	Perfluorooctanoic acid
PFOS	Perfluorooctanesulfonic acid
PFUA	Perfluoroundecanoic acid

RO	Reverse osmosis
UF	Uncertainty factor
PFCs	Polyfluoroalkyl compounds

Introduction

During the spring of 2021, researchers at Old Dominion University (ODU) performed a review of published scientific research and other literature about a class of chemical compounds known as per- and polyfluoroalkyl substances (PFAS). The purpose of the literature review was to locate information on the health effects, toxicity, treatment methods for drinking water, and other states regulation of PFAS to support efforts by the Virginia Department of Health to establish regulatory limits for PFAS in drinking water under the Waterworks Regulations, 12VAC5-590-10 et seq. This report summarizes the body of literature ODU collected.

PFAS are a wide assortment of anthropogenic chemicals, that have been manufactured since the late 1940s and early 1950s (Niu et al. 2016) using electrochemical fluorination and telomerization (Banks et al. 2013). Perfluoroalkyl carboxylates and perfluoroalkyl sulfonates, known collectively as perfluoroalkyl acids (PFAAs), are a subset of PFAS with totally fluorinated carbon chains of varying length and a negatively charged carboxylate or sulfonate group. Figure 1. This group of PFAS are the most commonly detected in the environment, and they are significant as precursors being able to transform into more persistent forms (ITRC, 2020).

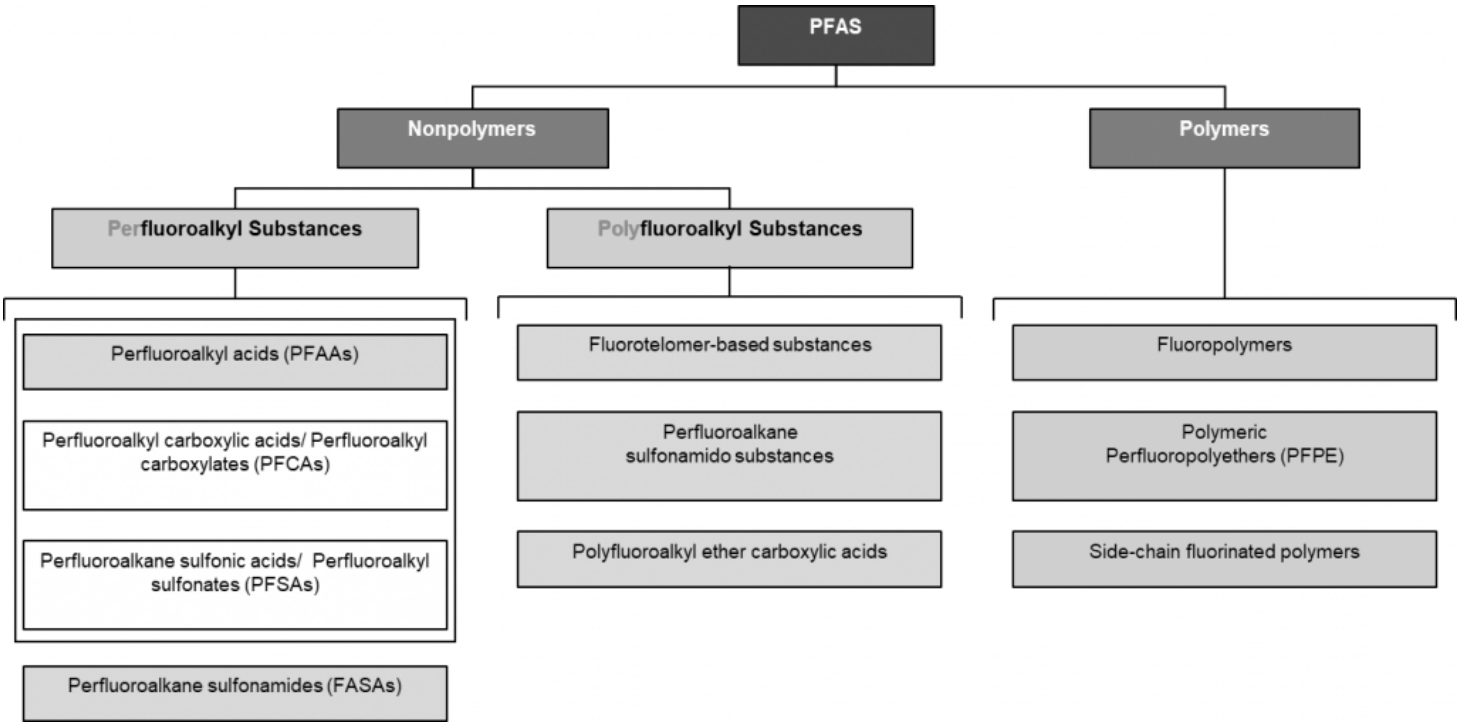

Figure 1. The PFAS Family Tree
Source: *PFAS Technical and Regulatory Guidance Document and Fact Sheets* by ITRC. Chapter 2.2 Chemistry, Terminology, and Acronyms

Figure 2 illustrates the PFAS lifecycle from PFAS synthesis to ecological exposure. Among PFAS, perfluorooctanoate (PFOA) and perfluorooctanesulfonate (PFOS), two of several perfluoroalkyl and polyfluoroalkyl substances (PFAS), were widely integrated into industrial processes and consumer products beginning in the 1950s. PFASs are mainly used as surfactants or surface protection agents due to their water- and oil-repelling properties and the chemical and thermal stability of their characteristic carbon-fluorine bonds. PFAS are found in an abundance of consumer products, ranging from cookware to stain protectors to food wrappers to cosmetics. Because of the great strength of their carbon–fluorine bonds, PFAS are highly persistent and are resistant to environmental degradation. They have been called “forever chemicals.” Long-chain

perfluorinated alkyl acids (PFAAs, ≥ 8 carbons for carboxylates, ≥6 carbons for sulfonates) and other long-chain PFASs are highly bio-accumulative in humans. Due to their widespread use and their ability to bioaccumulate into living organisms, they are now present globally in environmental media and biota, including humans (Wang et al. 2017; Buck et al. 2011). The pervasiveness of PFOA and PFOS and their long clearance half-lives in humans have provoked intense interests in understanding the potential human health impact of long-term exposure to the chemicals.

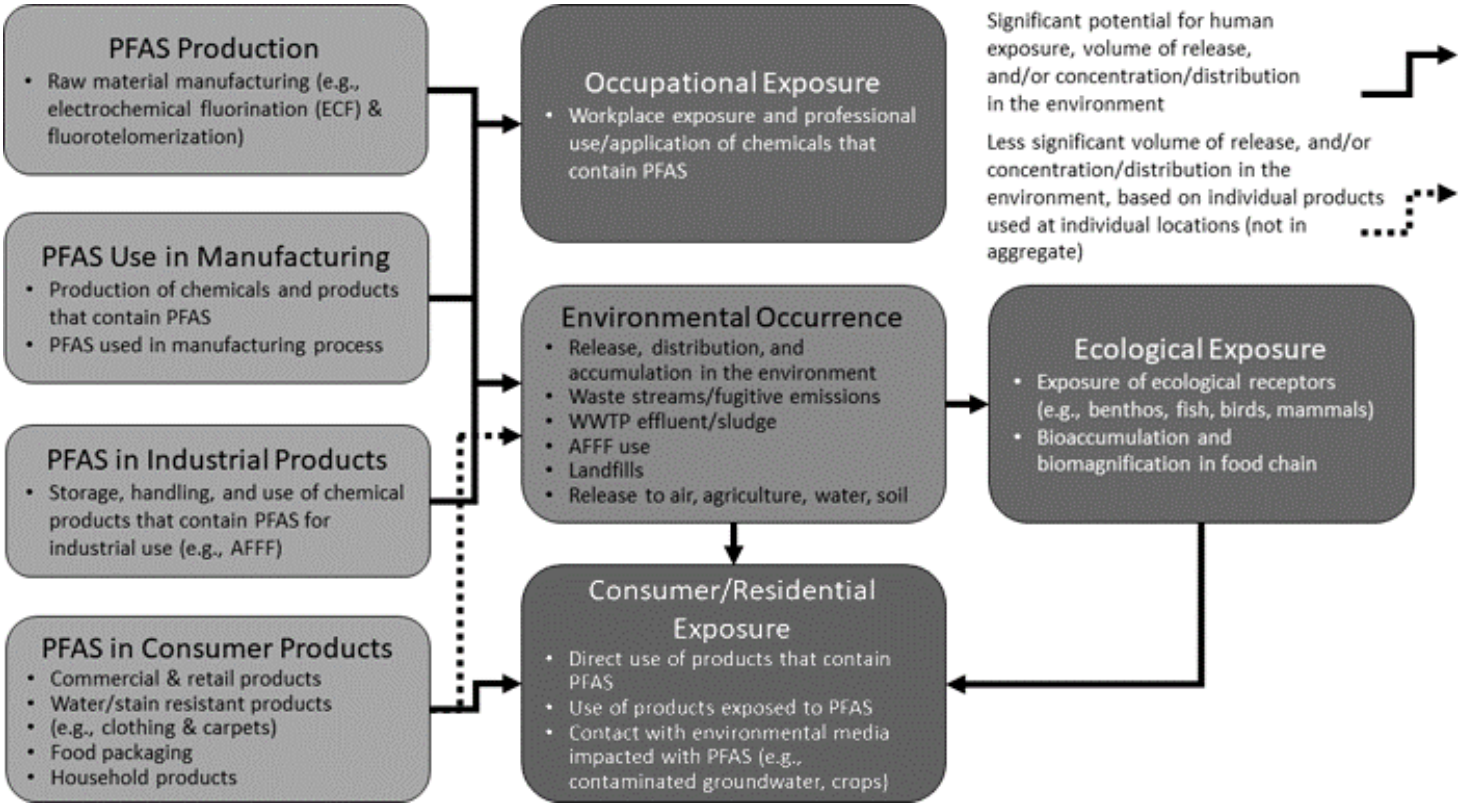

Figure 2. Generalized PFAS uses and relative exposure and environmental impact potential from PFAS life cycle
Source: *PFAS Technical and Regulatory Guidance Document and Fact Sheets* by ITRC. Chapter 2.1 Environmental Significance

Since the 1970s, occupational exposure studies have found PFAS in blood samples of exposed workers. Other studies detected PFAS in blood samples from the general population (ITRC, 2020). As shown in Figure 3, PFAS became a concern after the early 1990s when analytical methods to detect low levels of PFAS in the environment became widely available, and the levels detected were enough for potential human health effects (ITRC, 2020). In light of their associations with adverse health outcomes in humans, PFOS was voluntarily phased out by its primary manufacturer and eight major companies voluntarily agreed to phase out their global production of PFOA and PFOA-related chemicals in 2006 (U.S. EPA 2006; U.S. EPA, 2021b). Serum levels of both chemicals, especially PFOA, have correspondingly declined over time in these regions. However, body burdens of long-chain PFAAs remain elevated for many years after exposure ends, because of their long human half-lives (several years) (Post et al., 2017; ITRC, 2020). After the phase out of PFOS, PFOA, and PFOA-related chemicals, other perfluoroalkyl substances have been developed or brought in as replacements for PFAS compounds. Replacements include using nonfluorinated chemicals, alternate technologies, and shorter chain PFAS (ITRC, 2020). However, several studies published show that replacement compounds may not be less hazardous than the traditionally used long-chain PFAS. One of these replacement compounds is GenX, trade name for a polymerization processing aid formulation that contains ammonium 2,3,3,3-tetrafluoro-2 (heptafluoropropoxy) propanoate. GenX is used as a replacement for PFOA, and since its usage, the EPA has

completed a Toxicity Assessment that can be found at https://www.epa.gov/pfas/genx-toxicity-assessments-documents

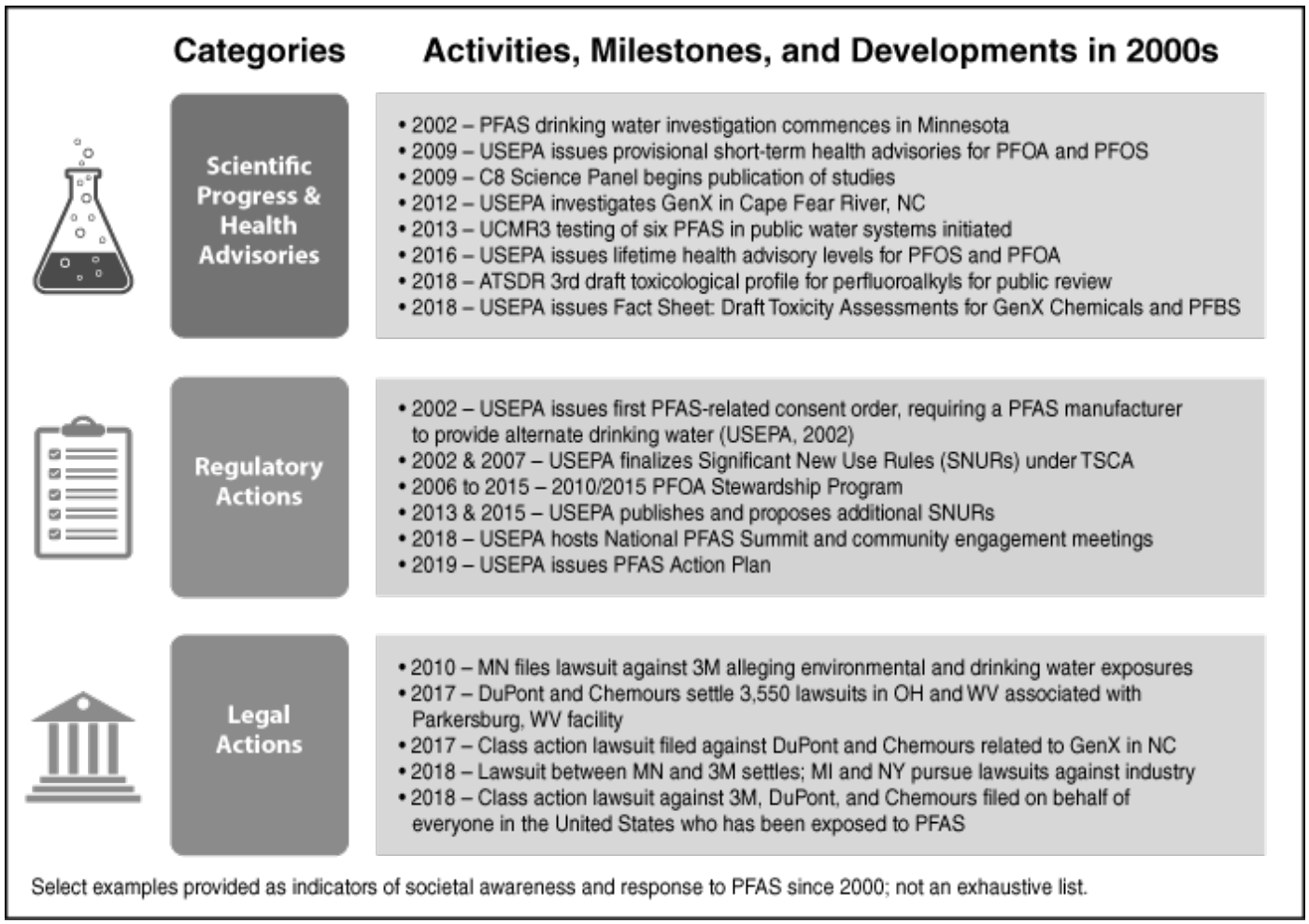

Figure 3. Growing awareness and concern since the early 2000s
Source: *PFAS Technical and Regulatory Guidance Document and Fact Sheets* by ITRC. Chapter 2.3 Emerging Health and Environmental Concerns

Human exposure routes include ingestion, inhalation, and dermal absorption. The consumption of PFASs from drinking water are of increasing concern in the United States, as well as worldwide, because of their widespread detection in public water systems and private domestic wells (U.S. EPA, 2021b). Because of their persistent, bio-accumulative, and toxic nature, PFAS are unique among organic drinking water contaminants. Infants are a sensitive subpopulation for the adverse effects of PFAS. Their exposures from contaminated water, either from prepared formula or via maternal transfer to breast milk, are much higher than in older individuals (Post et al. 2017; Goeden et al. 2019).

The U.S. Environmental Protection Agency (U.S. EPA) has not established a national drinking water standard (i.e. a maximum contaminant level (MCLs) or treatment technique) for PFAS, either as a group of compounds or for individual compounds, in the United States (Association of State Drinking Water Administrators 2019). In 2016, the U.S. EPA finalized nonregulatory lifetime drinking water Health Advisories of 70 ng/L for the individual and total concentrations of PFOA and PFOS, the 8-carbon PFAS that are the best-known and most thoroughly studied members of the PFAS (U.S. EPA 2021). As of May 2020, nine U.S. states have concluded that the U.S. EPA lifetime Health Advisories are not sufficiently protective and have developed more stringent drinking water standards or guidance values for PFOA and PFOS (Figure 4).

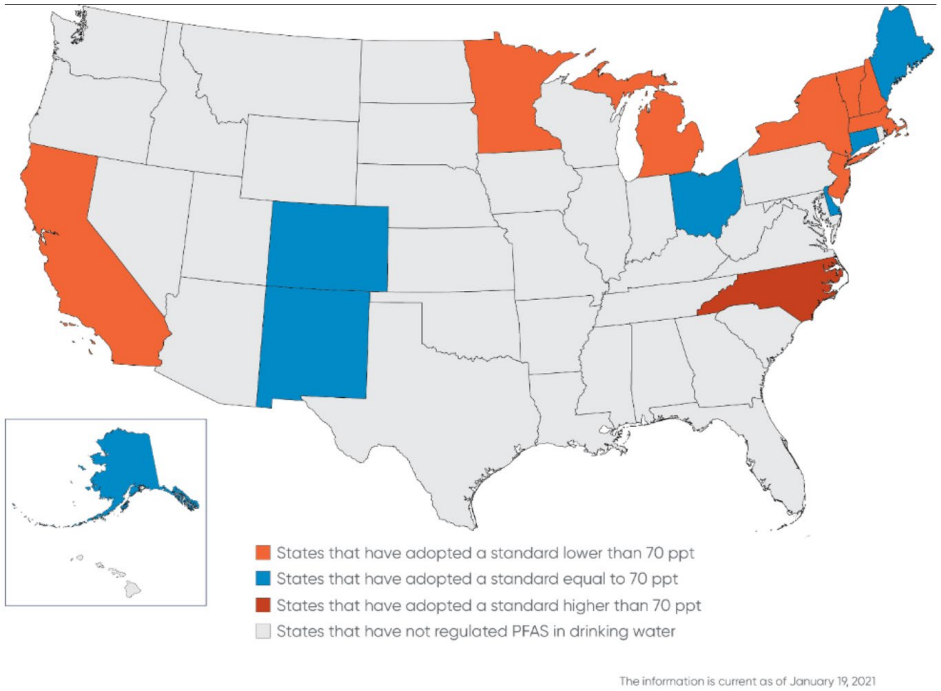

Figure 4 United States map showing PFAS regulations state by state
Source: State-by-State Regulation of PFAS Substances in Drinking Water received from https://www.bclplaw.com/en-US/insights/state-by-state-regulation-of-pfas-substances-in-drinking-water.html

Virginia Initiatives

The Commonwealth of Virginia is one of the many states taking a closer look at PFAS in the drinking water supply and drinking water sources. The Virginia General Assembly has directed the State Health Commissioner to establish a workgroup to study the occurrence of the following PFAS as directed in the Virginia Acts of Assembly Chapter 611 [H 586]: perfluorooctanoic acid (PFOA), perfluorooctane sulfonate (PFOS), perfluorobutyrate (PFBA), perfluoroheptanoic acid (PFHpA), perfluorohexane sulfonate (PFHxS), perfluorononanoic acid (PFNA). The workgroup was tasked with the following items for completion:

a. Determine current levels of PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS, as deemed necessary, contamination in the Commonwealth's public drinking water, provided that in making such determination of current levels, the Department of Health shall sample no more than 50 representative waterworks and major sources of water;
b. Identify possible sources of such contamination, where identified; and
c. Evaluate existing approaches to regulating PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS, as deemed necessary, in drinking water, including regulatory approaches adopted by other states and the federal government.

Additionally, the workgroup may develop recommendations for specific maximum contaminant levels for PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS, as deemed necessary, to be included in regulations of the Board of Health applicable to waterworks. Virginia Acts of Assembly Chapter 1097 [H 1257], effective January 1, 2022, requires the Department of Health to adopt regulations that establish MCLs for PFOA, PFOS, and other PFAS as deemed necessary. The Office of Drinking Water divided the duties of the workgroup into four subgroups – Health and Toxicology, Monitoring and Occurrence, Policy and Regulation, and Treatment Technology. In light of the General Assembly's direction, an extensive literature review was

conducted for the workgroups in an effort to compile relevant research and current standards to aid the creation of Virginia's PFAS plan.

Health and Toxicology

The Health and Toxicology subgroup was tasked with researching and evaluating animal and epidemiological studies that states and the U.S. EPA have used to develop regulatory limits for PFAS in drinking water. ODW researchers reviewed animal studies to evaluate the existing evidence on the toxicity of PFAS compounds, including PFOA, PFOS, PFBA, PFHpA, PFHxS, PFNA, and other PFAS. In general, animal studies focused on the relationship between sort- and long-term exposure to specific PFAS and their effect on reproduction, growth and development in juveniles. Most animal studies involved dosing rats and mice to extrapolate impacts on humans. Broad overviews of the research most commonly cited and/or followed by states and the U.S. EPA can be found below.

ANIMAL STUDIES

Reproductive toxicity

Luebker et al. (2005) looked into the reproductive and developmental toxicity of PFOS using female Crl:CD®(SD)IGS VAF/Plus® rats. The study period was two-generational reproductive periods, and doses were either 0.4, 0.8, 1.0, 1.2, 1.6, and 2.0 mg PFOS/kg/day. The study showed dose levels for the dose-response and etiological investigation 0.0, 0.4, 0.8, 1.0, 1.2, 1.6, and 2.0 mg/kg/day. There was a decrease in gestation length, viability through lactation at day 5 ($\geq$ 0.8 mg/kg), and a decrease in viability through lactation at day 5.

Yahia et al. (2010) used pregnant ICR mice to determine the reproductive toxicity of PFOA. The mice were dosed from gestational day 0 through 17 with either 1, 5, and 10 mg/kg PFOA daily by gavage, and at day 18 prenatal and postnatal evaluations occurred. Throughout the study, no maternal death occurred. There was an increase in liver weight, hepatocellular hypertrophy, necrosis, mitosis at 10 mg PFOA/kg. There was a decrease in fetal body weight at 5 and 10 mg PFOA/kg as well as a decrease in neonatal survival rate at 5 and 10 mg PFOA/kg.

Qin et al. (2013) found PFOS decreased or dislocated junction proteins (i.e., ZO-1, occludin, claudin-11, and connexin-43) and increased proteins related to the MAPK signaling pathway of Sertoli cells, whereas basal ectoplasmic specialization proteins did not change. Sertoli cells appear to be a new cellular target for PFOS. Together with disruption of BTB integrity and function, these cells play an important role in PFOS-induced male reproductive toxicity.

Eggert et al. (2019) assessed the effects of PFOA (0-100 μg/ML) on fetal and adult rat testis. This study shows that the levels of cAMP, progesterone, testosterone, and expression of StAR decreased significantly in PFOA concentrations of 50 and 100 μg/ml. PFOA affected cell populations significantly by decreasing the amount of diploid, proliferating, meiotic I, and G2/M-phase cells in adult rat testis. However, PFOA did not affect fetal, proliferating, or adult rat Sertoli cells, but an increased tendency of apoptosis in fetal Leydig cells was observed.

A recent meta-analysis on sixteen studies was performed to assess male reproductive toxicity of PFOA in rodent studies based on level of testosterone and estradiol in serum, development of reproductive organs, pathological changes of reproductive organs, and parameters of semen. The study reported that the lower serum testosterone levels, decreased absolute testicular and epididymal weights, higher serum estradiol levels, elevated relative testicular and seminal vesicle weights and increased incidence of Leydig cell adenoma and percentage of abnormal sperm were observed in the exposed group compared with the control group. PFOA exposure

heightens the reproductive system damage in male rodents. However, many studies included in the review did not identify mechanisms by which PFOA induces changes to the male reproductive system (Wang et al., 2021).

Developmental toxicity

Luebker et al. (2005) conducted a 6-week laboratory study to determine the reproductive toxicity of PFOS before mating, during mating, and, for females, through gestation and lactation, across two generations. Doses for the experiment were 0, 0.1, 0.4, 1.6, and 3.2 mg PFOS/kg/day via oral gavage. Results showed no adverse effects in F0 females or their fetuses upon cesarean sectioning at gestation day 10. PFOS did not affect reproductive performance. Neonatal toxicity in F1 pups occurred only at a maternal dose of 1.6 mg/(kg day) or higher. In utero exposure to PFOS contributed to post-natal pup mortality in an additive fashion.

Lau et al. (2006) used CD-1 mice to determine the developmental effects of PFOA. Mice were studied from gestational day (GD) 1 through 17 and were dosed with either 1, 3, 5, 10, 20, or 40 mg/kg PFOA by oral gavage daily. At the end of the study period, weight gain in dams that carried the pregnancy to term was significantly lower in the 20-mg PFOA/kg group. The incidence of live birth in group B mice was significantly lowered by PFOA. Postnatal survival was severely compromised at 10 or 20 mg/kg, and moderately so at 5 mg/kg.

Macon et al. (2011) used CD-1 mice to determine the developmental toxicity of PFOA. The experiment included two separate studies: a full-gestation study of a 17-day period (days 1-17) and a late-gestation study of a 7-day period (day 10-17) with doses ranging from 0, 0.3, 1.0, and 3.0 mg PFOA/kg body weight/day, and 0, 0.01, 0.1, and 1.0 mg PFOA/kg body weight/day, respectively. No observable adverse effect level for mammary developmental delays was observed. There was an increase in offspring relative liver weights in all treatment groups in the full-gestation study and the 1.0 mg PFOA/kg group in the late-gestation study. Results showed an increase in mammary epithelial growth in the offspring, and at postnatal day 21, mammary glands from the 1.0 mg/kg GD 10– 17 group had significantly less longitudinal epithelial growth and fewer terminal end buds compared with controls ($p < 0.05$).

Zeng et al. (2011) used Sprague-Dawley rats to determine the developmental neurotoxicity of PFOS. The experimental period lasted 21 days and dams received 0.1, 0.6, and 2.0 mg/kg body weight PFOS by gavage from gestational day 2 (GD2) to GD21. The results showed an increase in gene expression of inflammation biomarkers AP-1, NF-kB, cytokines interleukin and tumor necrosis factor (TNF)-a, and cAMP response element-binding protein. There was an increase in astrocyte activation markers, glial fibrillary acidic protein, S100 calcium-binding protein B in the hippocampus, and cortex upregulated on PND0 or PND21. Finally, there was a decrease in Synapsin 1and synaptophysin in the cortex and hippocampus.

Onishchenko et al. (2011) looked into the developmental toxicity of PFOS and PFOA using C57BL/6/Bkl mice. The study period was gestation plus 21 days post-birth. Mice were dosed with either 0.3 mg/kg of PFOS or PFOA throughout the pregnancy. The study showed a decrease in locomotion in a novel environment and reduced muscle strength (males only) by PFOS exposure. There was changed exploratory behavior in male and female offspring, and increased global activity in males in their home cage by PFOA exposure.

Das et al. (2015) looked into the developmental toxicity of PFNA using CD-1 mice. The experimental period was 17 days. Mice were dosed with either 1, 3, 5, or 10 mg PFNA/kg body weight per day. Failed pregnancy at 10 mg PFNA/kg occurred. There were no effects on pregnancy and pups' survival at 5 mg PFNA/kg or lower doses in the first 10 days of life. 80% of these neonates died in the first 10 days of life. Hepatomegaly occurred in the pregnant dams at 5 mg/kg or lower doses.

Koskela et al. (2016) used C57BL/6 mice to look into the developmental toxicity of PFOA over 17 months. The mice were dosed with 0.3 mg PFOA/kg/day. Increased femoral periosteal area, decreased mineral density of

tibias, and increased osteocalcin expression were observed. Additionally, there was a decrease in osteocalcin expression and calcium secretion in osteoblasts at 100 μM and above as well as an increase in osteoclasts numbers and resorption activity increased dose-dependently from 0.1–1.0 μM, but decreased at higher concentrations.

Blake et al. (2020) looked into PFOA and GenX, specifically their developmental toxicity on CD-1 mice. The study spanned either from embryonic day (0) to 1.5 days, 11.5 days, or 17.5 days. Mice dosed with PFOA received either 0, 1, or 5 mg/kg while mice dosed with GenX received either 0, 2, or 10 mg/kg. At the end of the study, there was an increase in gestational weight gain (10 mg/kg/d GenX exposure). Additionally, there was a decrease in embryo weight (5 mg/kg/d PFOA). The results showed an increase in the incidence of placental abnormalities, higher maternal liver weights, changes in liver histopathology, embryo–placenta weight ratios (1 - 10 mg/kg).

Animal studies showed that health effects from PFOA and PFOS impacted the development of the offspring. However, evidence for other FPAS compounds is still lacking

Immunotoxicity

DeWitt et al. (2008) used C56BL/6J mice and C57BL/6N mice to determine the immunological toxicity of PFOA. The C56BL/6J mice were dosed for 10 days at either 0 or 30 mg PFOA/kg/day, while the C57BL/6N mice were dosed for 15 days in a range of 0-30 mg/kg/day of PFOA through drinking water. The doses produced a decrease of IgM antibody synthesis and an increase in IgG titers were elevated at 3.75 and 7.5 mg PFOA/kg/day. The lowest observed adverse effect level was identified as 3.75 mg PFOA/kg body weight/day and a benchmark dose of 3 mg PFOA/kg body weight/day also was calculated.

Peden-Adams et al. (2008) used B6C3F1 mice to determine the immunological toxicity of PFOS. The mice underwent a 28-day period with dosing of 0, 0.005, 0.05, 0.1, 0.5, 1, or 5 mg/kg total administered dose. At the end of the study, there was no lymphocyte proliferation altered in either gender. There was an increase in natural killer cell activity in male mice only, and T-cell subpopulations modulated in males only at 0.1 mg/kg.

Dong et al. (2009) looked into the chronic immunotoxicity of PFOS using C57Bl/6 mice over six days. Mice were dosed with either 0, 0.5, 5, 25, 50, or 125 mg PFOS/kg. There was an increase in liver mass at ≥ 5 mg PFOS. Altered lymphocyte proliferation and natural killer cell activity were observed as well as plaque-forming cell response was suppressed (≥5 mg/kg). There was no observed and lowest observed adverse effect level at 0.5 and 5 mg/kg total administered dose, respectively.

Torres et al. (2020) studied the immunological toxicity of PFOS using adult male and female wild-type C57BL/6 mice. The mice were studied over either a 2- or 4-week period and were dosed with either 3 μg/kg/day of PFOS (2-week period) or 1.5 μg/kg/day of PFOS (4-week period). At the end of the study, there was no weight loss during exposure (100 ng/ml in serum). Additionally, there was no effect on T-cell development. Exposure to PFOS at 1.5 μg/kg/day for 4 weeks did not affect weight loss, survival, and viral clearance. Also, there was no suppression if immune cell development or antigen-specific immune response.

Zeng et al. (2021) looked into PFOA and its immunological toxicity using BALB/c mice. The mice were studied for seven days and were dosed with PFOA in either 10, 50, and 100 pg. Doses were administered intratracheally after each ovalbumin (OVA)-induced asthma, which is the same equivalent to breathing in PFOA from the atmosphere for 10, 30, and 60 weeks respectively. The study showed that in the OVA, OVA+10 pg PFOA and OVA+50 pg PFOA groups there was an increase in aggravated airway hyperresponsiveness and T helper type 2 (Th2) airway inflammation in asthmatic mice. Additionally, there was a decrease in the expression of the GR mRNA and protein.

Animal studies support that PFOA and PFOS are associated with toxicity in the immune system with respect to anti-gen-specific antibody suppression responses. However, evidence for other PFAS compounds are very limited or weak.

Liver toxicity

Butenhot et al. (2012) used Sprague Dawley rats to determine liver toxicity of Potassium PFOA. They dosed the rates at 0, 0.5, 2, 5, and 20 g/g (ppm) over a span of 104 weeks. At the end of the experiment, there was a 10% increase in hepatic tumors was 8 ppm for both sexes. There was also an increase in proliferation of endoplasmic reticulum, vacuolation, and an increase in eosinophilic granulation of the cytoplasm.

Salter et al. (2021) looked into liver toxicity of PFOS using adult male C57BL/6N mice over six weeks. The mice were fed *ad libitum* or a 25% reduced-calorie diet concomitant with either vehicle (water) or 100 μg PFOS/kg/day via oral gavage. A dose of 2.5 μM was used for glucose production testing. The study found no altered CR-induced weight loss, white adipose tissue mass, or variation (?) change in (?) liver weight gain (?) over 6 weeks. There was an increase in hepatic triglyceride accumulation in hepatocytes due to a decrease in phosphorylated AMPK expression in the liver. There was disrupted hepatic lipid and glucose homeostasis at 2.5 μM.

Frawley et al. (2018) studied PFDA and its effects on liver toxicity in Sprague-Dawley rats and B6C3F1/N mice. The rats were studied for 28 days and dosed with 0-2.0 mg PFDA/kg by oral gavage daily. The mice were studied for 4 weeks and received a dose once a week ranging from 0-5.0 mg PFDA/kg. There was observed hepatocyte necrosis and hepatomegaly (0.5 mg PFDA/kg/d) as well as observed hepatomegaly (≥0.625 mg PFDA/kg/week), while splenic atrophy was observed at 5.0 mg PFDA/kg/week. There was a decrease in total spleen cells, and Ig þ and NK þ cells (5.0 mg PFDA/kg/week). No changes occurred in rats' leukocytes, and the dosing of PFDA altered the balance of immune cell populations in lymphoid tissues in mice.

Cancer

A chronic feeding study was conducted using male Crl:CD ® BR (CD) rats and a dietary PFOA concentration of either 0 or 300 ppm (Biegel et al. 2001). PFOA increased the incidence of proliferative acinar cell lesions at the highest dietary concentration of 300 ppm. More and larger focal proliferative pancreatic acinar cell lesions and a greater tendency for progression to adenoma in lesions were observed.

A 2-year dietary study was conducted to assess the carcinogenicity potential of PFOA. Groups of 50 male and 50 female Sprague – Dawley (Crl:CD ® BR) rats were fed diets containing 0, 30, or 300 ppm ammonium perfluorooctanoate for up to 2 years (Butenhoff et al. 2012a). No mortality differences were observed between the treated and control groups. Increased frequency of various non-neoplastic lesions of the testis in males, the mammary gland in females, and the liver in both sexes were observed. Testicular Leydig cell adenoma in the high-dose males and mammary fibroadenoma in both treated groups of females were statistically significantly increased. The same group conducted another following up study. A 2-year feeding study of potassium PFOS at concentrations up to 20 ppm in the diet using both male and female Sprague– Dawley [Crl:CD ® (SD)IGS BR] rats. The study reported multiple non-neoplastic changes in the liver, including hepatocellular hypertrophy with proliferation of endoplasmic reticulum, vacuolation, and increased eosinophilic granulation of the cytoplasm in both males and females at the higher exposure concentrations. Statistically significant increases in hepatocellular adenoma incidence were observed in both male and female rats of the 20-ppm dose treatment groups. Hepatocellular carcinoma only observed in a 20-ppm dose group female (Butenhoff et al 2012b).

Filgo et al. (2015) conducted an 18-month (gestation days 1 to 17) feeding study at exposed daily doses from 0.01-5 mg/kg/BW in the diet using two strains of mice: wild type and PPARα-knockout (KO) 129/Sv. The female offspring were necropsied at 18 months. Hepatocellular adenomas formed in PFOA-exposed PPARα-KO 129/Sv and CD-1 mice. Hepatocellular hypertrophy was significantly increased by PFOA exposure in CD-1, and an increased severity was found in WT 129/Sv mice. PFOA significantly increased nonneoplastic liver lesions in PPARα-KO mice (hepatocyte hypertrophy, bile duct hyperplasia, and hematopoietic cell proliferation).

A scientific workshop was held in September 2010 to conduct a comprehensive, systematic review and assessment of the potential human relevance of evidence regarding the nongenotoxic modes of liver tumorigenesis that are mediated by nuclear receptors, including PPARα, CAR, and PXR. The workshop's panel reviewed a series of comprehensive review papers (Andersen et al. 2014; Budinsky et al. 2014; Corton et al. 2014; Elcombe et al. 2014) and suggested that for PPARα agonists, including PFOA and PFOS, the workshop panel identified the following sequence of key events in the mode of action for hepatic tumor induction in rodents: 1) PPAR α activation in the liver; 2) alteration of cell growth pathways in the liver; 3) perturbation of hepatic cell growth and survival, leading to the formation of new preneoplastic liver cells and the induction of new focal liver lesions; 4) selective clonal expansion of preneoplastic foci; 5) transformation and outgrowth of preneoplastic liver cells into adenomas (Corton et al. 2014). However, Filgo et al recently reported that PPARα is not required for PFOA-induced liver lesions in mice (Filgo et al., 2015). The mechanisms by which PFOA and PFOS induce liver tumors in rats appear not to be relevant to the potential carcinogenicity of these compounds in humans (Chang et al., 2014).

Laboratory studies have shown that PFOA exposure induces tumors in rats, e.g., hepatocellular adenoma Testicular Leydig cell adenoma. However, underlying mechanisms for the carcinogenic effects haven't been well defined (Chang et al. 2004).

Reference Doses

Nine states, California (CA), New Jersey (NJ), New Hampshire (NH), New York (NY), Michigan (MI), Washington (WA), Minnesota (MN), Vermont (VT), and Massachusetts (MA), have developed Reference Doses for PFOA and PFOS based on findings from animal studies. State Reference Doses for PFOA range from 1.5 to 18 ng/kg/d, while the U.S. EPA Reference Dose is 20 ng/kg/d. To develop References Doses for PFOA, NJ, NH, and NY used increased relative liver weight of rats for the critical endpoint for Reference Doses (Loveless et al. 2006; Macon et al. 2011), whereas MI and WA used developmental toxicity regarding changes in motor function and bone morphology/differences in mice. The increased relative liver weight is a well-established and sensitive effect of PFOA that follows a monotonic doses response, with the effect increasing with dose. Other states, including MI, WA, MN, VT, and MA, used developmental endpoints as the critical endpoint for Reference Doses (Onishchenko et al. 2011; Koskela et al. 2016). The developmental endpoints are nonmonotonic, with the greatest effects at the lowest dose and smaller effects as the dose increases. The dose-response relationship below the lowest-observed-adverse effect level (LOAEL) is undefined. The U.S. EPA uses an uncertainty factor to extrapolate from the LOAEL to the no-observed-adverse-effect level (NOAEL). The point of departure for increase liver weight is lower confidence limit on the benchmark dose (BMDL), derived from serum PFOA levels measured at the end of the dosing period in the animal studies, whereas those for developmental effects at LOAELs based on modeled average serum PFOA levels in the animal studies.

The total uncertainty factors used in the state and U.S. EPA PFOA Reference Doses range from 100 to 1000. The uncertainty factor was determined by adjustment on intraspecies, interspecies, less-than-chronic, LOAEL-to-NOAEL, and database. The default intraspecies uncertainty factor (for sensitive human subpopulations) of 10 was used. Also, an interspecies (animal-to-human) uncertainty factor of 3 was used rather than the default value

of 10. Since the animal studies based on the MCL lasted throughout gestation and early postnatal life, no adjustment is made for the less-than-chronic duration (i.e., uncertainly factor of 1). To account for interspecies toxicokinetic and toxicodynamic differences, the default animal-to-human uncertainty factor of 10 is composed of 2 factors of $10^{0.5}$ (rounded to 3) each. The animal studies based on the MCL lasted throughout gestation and early postnatal life.

State References Doses for PFOS range from 1.8 to 5 ng/kg/d . MN, NH, WA, MI, NJ, and NY developed the references doses based on critical effects of decreased antibody response to foreign antigen in mice exposed to PFOS, whereas MA, VT, and USEPA based on the developmental toxicity of decreased rat's offspring body weight. The decreased immune response in mice is a more sensitive toxicological effect of PFOA than the developmental endpoint. Also, the decrease immune response in mice is relevant to response in humans. That is supported by epidemiological associations of PFOA with decreased vaccine response and increased incidence of infectious disease, analogous effects in humans (New Jersey Drinking Water Quality Institute Health Effects Subcommittee 2018; Pachkowski et al., 2019). The point of departures for all state and USEPA PFOA Reference Doses are NOAELs. The NOAELs developed by the six states (MN, NH, WA, MI, NJ, and NY) are serum PFOS levels quantified at the end of dosing in the mouse studies, whereas the NOAELS for developmental effects used by MA, VT, and U.S. EPA are modeled average serum PFOA levels from the rate study. NOAELs used by MN/NH/WA, MI/NJ/NY, and MA/VT/USEPA were 2,620 ng/mL, 674 ng/mL, and 6,260 ng/mL, respectively. Due to differences in critical effects, human subjects, and exposure conditions, a human half-life for a clearance factor (5.4 yr.) adopted by the U.S. EPA/MA/VT/NJ/NY is longer than the one (3.4 yr.) used by MN/NH/WA/MI. The U.S. EPA/MA/VT/NJ/NY estimated the human half-life based on decline serum levels in retired fluorochemical workers, whereas MN/NH/WA/MI based on the decline in serum levels after community exposure to contaminated drinking water ceased (Li et al., 2018).

The total uncertainty factor used in the state and U.S. EPA PFOS Reference Doses ranges from 30 to 100 (Table 7). All of the state and U.S. EPA Reference Doses use the default intraspecies uncertainty factor (for sensitive human subpopulations) of 10. Also, an interspecies (animal-to-human) uncertainty factor of 3 was used rather than the default value of 10. No adjustment for LOAEL-to-NOAEL extrapolation (i.e., uncertainty factor of 1) was needed because the point of departures for all state and U.S. EPA Reference Doses were NOAELs. An uncertainty factor of 3 for more sensitive toxicological effects was used to account for decreased immune response at lower doses in the MA Reference Dose. In addition, an uncertainty factor of 3 to account for potential thyroid effects at doses below the NOAEL for a decreased immune response was used by three states.

EPIDEMIOLOGICAL STUDIES

Reproductive toxicity

Studies are conducted to examine associations between PFAS exposure and male/female reproductive outcomes. Male reproductive outcomes include semen characteristics, reproductive hormones/related outcomes. Female reproductive outcomes include fecundability, infertility/subfecundability, and reproductive hormones

Bach et al., (2015), Buck Louis et al., (2013), and Vele et al. (2015) found no associations between exposure to PFOS and fecundability. However, Jorgensen et al., (2014) found a tendency towards lower fecundability in women. Vestergaard et al. (2012), Buck Louis et al., (2013) found no association regarding PFOS. Bach et al (2015) found no associations regarding PFHpS, PFNA, and PFDA. Jorgensen et al (2014) and Bach et al., (2105) found no associations between PFHxS and fecundability.

Velez et al., (2015) and Bach et al., (2105b) found that PFOA tended to be associated with infertility. However, Jorgensen et al. (2014) and Bach et al. (2015a) found no association between PFOA exposure and fertility. Velez et al. (2015) found no association between PFOS exposure and infertility. Bach et al., (2015a) found no association for PFNA. Jorgensen et al. (2014) and Bach et al. (2015a) found no association between PFHxS. Bach et al., (2015) found no association between exposure to PFHpS, PFDA, and infertility.

Lewis et al. (2015) found that there were no consistent associations between 4 PFAS (PFOA, PFOS, PFHxS, PFNA) and testosterone levels. Barrett et al. (2015) found that PFOA, PFNA, PFDA, and PFHxS were not clearly associated with estradiol and progesterone. Tsai et al. (2015) found that higher PFOS was associated with lower testosterone and sex hormone-binding globulin (SHBG) in adolescents, but not adults. However, there were no clear associations between other PFASs, including PFOS, PFNA, PFUnA (perfluoroundecanoic acid, a breakdown product of PFOA), and SHBG. Toft et al. (2012) reported PFOA exposure was associated with a higher percentage of motile sperm. However, Joensen et al. (2013) and Buck Louis et al. (2015) found no consistent associations. For semen volume, total sperm count, and sperm concentration, none of the studies found consistent associations between exposure to any PFAS (PFOA, PFOS, PFHxS, PFHpS, PFNA, PFDA, PFOSA) (Barrett et al. 2015; Buck Louis et al. 2015; Den Hond et al. 2015). Additionally, no consistent associations were observed between PFAS (PFOS, PFNA, PFDA, and PFHxS) and motility (Joensen et al. 2013; Buck Louis et al. 2015). Joensen et al. (2013), Buck Louis et al. (2015), and Den Hond et al. (2015) found no relationship between a lower percentage of morphologically normal sperm and higher exposure to PFOS and PFHxS. Additionally, levels of PFNA, PFDA, PFHpS, and PFOA were not consistently associated with overall sperm morphology (Joensen et al. 2013; Buck Louis et al. 2015; Den Hond et al. 2015). Studies reported inconsistent associations between PFAS (PFOA, PFHxS, PFOS, and PFNA) and sperm DNA integrity or fragmentation (Buck Louis et al. 2015; Governini et al. 2015; Leter et al. 2014). For reproductive hormones, inconsistent results were found between PFAS (PFDA, PFOS, PFNA, PFHpS, and/or PFHxS) and testosterone, androgen, or estradiol (Joensen et al. 2013, Lewis et al. 2015; Tsai et al. 2015; Den Hond et al 2015).

Immunotoxicity

Grandjean et al. (2012) performed the first prospective study of PFAS and antibody suppression-related effects. This study reported that maternal PFOS serum concentrations (geometric mean of 27.3 ng/mL) collected during the last trimester of pregnancy were negatively associated with anti-diphtheria antibody concentrations in five-year-old children (N=532). In a separate prospective study in general population exposures in Norway, Granum et al. (2013) observed that concentrations of four PFAS, PFOA, PFOS, PFHxS, and PFNA, in maternal blood (median concentrations were 1.1, 0.3, 0.3, and 5.5 ng/mL respectively) collected at the time of delivery, were all inversely correlated with the level of anti-rubella antibodies measured in three-year-old children (N=56). Additionally, they reported that maternal levels of PFOA and PFNA were positively correlated with the number of episodes of common colds in the children (Granum et al. 2013).

In a large cross-sectional analysis of data (N=1,191) from the U.S. National Health and Nutrition Examination Survey (NHANES), Stein et al. (2015) reported that decreases in anti-mumps antibodies were associated with increases in serum concentrations of PFOA and PFOS (geometric means of 20.8 and 4.13 ng/mL respectively) and anti-rubella antibodies were associated with increases in serum concentrations of PFOA, PFOS, and PFHxS (geometric mean of 2.47 ng/mL). Kielsen et al. (2016) conducted a small cross-sectional study of 12 adult volunteers from the general human population in Denmark and reported a negative association between serum concentrations of most of the eight different PFASs they measured and anti-tetanus and diphtheria antibodies.

Prospective studies of birth cohorts from the general human population (N ranging from ~200 to 2,000+ subjects/study) did not find associations between PFOA/PFOS levels in maternal serum or cord blood and Type I hypersensitivity reactions in the children (Granum et al., 2013; Wang et al. 2011; Okada et al. 2012; Okada et

al. 2014; Smit et al. 2015) or IgE (Immunoglobulin E) levels were inconsistent among studies (Wang et al. 2011; Okada et al. 2012; Ashley-Martin et al. 2015).

In three separate analyses of NHANES data, Humblet et al. (2014) and Stein et al. (2015) reported positive associations of PFOA and PFOS with several respiratory hypersensitivity outcomes (N=1,877 and 638, respectively) in children. Buser and Scinicariello (2016) reported that serum PFOA and PFOS were associated with an increase in self-reported food allergies. Stein et al. (2016) examined the relationship between serum concentrations of eight different PFASs, including PFOA and PFOS, and vaccination to FluMist intranasal live attenuated influenza vaccine in a small group of healthy adults from the general U.S. population (N=78). Chen et al. (2018) reported that cord blood PFOA concentrations were positively associated with the development of atopic dermatitis (a type of dermatitis associated with asthma and allergic rhinitis) in female children during the first 24 months of life.

Developmental toxicity

Bach et al (2105), Negri et al. (2017), and Steenland et al. (2018) suggest that prenatal exposure to PFOS and PFOA may be associated with adverse birth outcomes, such as lower birth weight and smaller gestational age. Chu et al. (2020) reported that greater maternal serum levels of all PFAS alternatives were significantly associated with lower birth weight, adjusted for confounding variables. Neonates with low birth weight and preterm birth that reflect fetal growth restriction (Nardozza et al., 2017) are at greater risk of death (Saigal and Doyle, 2008; Crump et al., 2011), neurodevelopmental delays (Aylward, 2014), cardiovascular disorders (Pocobelli et al. 2016) and other adverse health effects throughout life (Blencowe et al. 2012). Li et al. (2017) indicated associations between greater cord blood PFOS and lower birth weight.

Thyroid disease

Lopez-Espinosa et al (2012) found that measured PFOA child serum levels were associated with a higher risk of thyroid disease (mostly hypothyroidism, n = 61). However, serum PFOA was not associated with subclinical hypo- or hyper-thyroidism based on cross-sectional analyses of individual hormone levels.

Winquist and Steenland (2014) reported a probable link between PFOS and thyroid disease based on the study of 32,000 participants in the mid-Ohio Valley. A significant trend of increasing risk hypo- and hyper-thyroid diseases was observed among adult females in relation to both cumulative and serum PFOA level at diagnosis. The clearest trend was for female hyperthyroidism in relation to serum PFOA at the time of diagnosis.

Steenland et al (2015) reported findings from a parallel study with Dupont Plant workers (n = 3713; 80% male). The study observed a trend of increasing thyroid disease risk across quartiles of modeled serum PFOA (with a 10 year lag) for males, but no evidence of trends with the log of cumulative exposure for either males or females.

Cancer

Barry et al (2013) and Viera et al (2013) reported the evidence for an association of PFOA with testicular cancer. Viera et al (2013) observed a relatively positive exposure-response for this cancer. However, evidence on testicular cancer mortality related to PFOA exposure is limited (Leonard et al. 2008).

Barry et al (2013) and Steenland and Woskie (2012) linked evidence for kidney cancer among adults living near a chemical plant and among workers, respectively. Also, Shearer et al. (2020) recently conducted a population-based case-control study with 324 renal cancer cases and 324 individually matched controls and reported a

positive exposure-response trend with renal cancer for several PFAS, including PFOA and PFOS. However, Raleigh et al (2014) reported that no significant increase in kidney cancer in a high-exposure occupational cohort of 3M workers based on mortality or incidence (16 exposed incident cases). Mastrantonio et al. (2017) conducted an ecologic study in the Veneto region of Italy and didn't find an excess of kidney cancer overall (95% CI 1.06–1.65) when comparing areas with PFOA-contaminated drinking water (as well as some other PFAS) with areas with non-contaminated water.

In summary, epidemiologic evidence supports that PFOA and PFOS are immunotoxic with respect to the suppression of anti-specific antibody responses, but uneven evidence for an association with infectious disease and other immune-related health outcomes (Steenland et al. 2020). Levels of confidence for members of the PFAS family of compounds on reproductive toxicity are very low for human data for all reproductive-related outcomes. The evidence for an association between PFOA and thyroid disease is suggestive but inconsistent. For cancer, the epidemiological studies provide supportive evidence, but not definitive for kidney and testicular cancers (Raleigh et al. 2014; Mastrantonio et al; 2017; Steenland et al. 2020). Currently, there have been no studies of cancer in children.

Monitoring and Occurrence

The U.S. EPA's third Unregulated Contaminant Monitoring Rule (UCMR3) 2017 report documented occurrence data for six PFAS (perfluorobutanesulfonic acid (PFBS), PFHxS, PFHpA, PFOA, PFOS, and PFNA (U.S. EPA 2017). The report includes nearly 37,000 PFAS sample results, originating from 4,920 U.S. drinking water utilities, collected between 2013 and 2015. PFOA and PFOS were detected more frequently across all treatment system sizes and sources at 1.03% and 0.79%, respectively, and PFOS also had the highest maximum concentration of 7,000 ng/L. The UCMR3 data also showed that 72% of all PFAS detections occurred in groundwater. Detections of one or more PFAS were 5.6 times more frequent in large than small public water system (PWS) in UCMR3 when considering both surface and groundwater sources together but small PWS had greater total PFAS concentrations when detected (300 vs. 170 ng/L) (Guelfo & Adamson 2018). Average total PFAS concentrations were higher in groundwater than in surface water across all system sizes (210 ng/L vs. 90 ng/L). Dilution by the receiving water body and potential complexation with the sediment and natural matter may attribute to lower surface water concentrations and the lower detection frequency in surface waters.

PFBS was found only in large systems with low detection frequency (0.05%). PFBS was detected more frequently in surface water than groundwater with notably high mean concentrations of 212ng/L in surface water and 136 ng/L in groundwater (Guelfo & Adamson 2018). PFBS also had the highest minimum reporting level of all monitored PFAS at 90 ng/L; therefore, reported PFBS detection may have been underreported relative to other PFAS. It is likely due to its weaker sorption than the longer-chain PFAS.

The mean PFHxS concentration was highest in small groundwater systems (409 ng/L), but a higher detection frequency (0.86%) and maximum concentration (1,600 ng/L) were found in large groundwater systems. PFHpA exhibited the lowest mean concentration in large surface (19 ng/L) and groundwater (28 ng/L) systems. Mean PFNA concentrations were highest in large surface (54 ng/L) and groundwater (35 ng/L) systems. Lower surface water concentrations have been attributed to dilution from the receiving water body and potential complexation with the sediment and natural matter, and this may explain the lower detection frequency in surface waters.

Since 1999, the National Health and Nutrition Examination Survey (NHANES) has measured blood PFAS in the U.S. population. Long-chain PFAAs including PFOA, PFOS, PFNA, and PFHxS are found in the low parts per billion (ng/ml) range in the blood serum of almost all residents of the US (CDC, 2017; Kato et al., 2015). From 1999 to 2014, serum levels of both PFOA and PFOS have declined by more than 60% and 80%,

respectively, in the general population based on the CDC's NHANES data. The reduction may be attributed to the action that beginning in the early 2000's the major manufacturers voluntarily started to phase out the two compounds in facility emissions and product content. However, serum levels of other PFAS which have not been phased out, including PFNA, have increased.

Biomonitoring studies have been conducted to measure PFAS levels in workers in PFAS manufacturing facilities, communities with contaminated drinking water, Red Cross blood donors, and the general U.S. population as well**.** Figure 15 shows PFOA and PFOS levels measured in different exposed populations, compared to levels CD measures in the general population in 2011-2012 and 2013-2014 (CDC, 2021; ATSDR 2016; Hew Hampshire Department of Health and Human Services, 2015). Workers in PFAS manufacturing facilities had the highest concentrations of PFOA and PFOS, following by communities in contaminated drinking water.

Despite relatively low levels of PFAS in drinking water and decreased PFAS detected in human serum, continuous exposure to drinking water contaminated with low PFAA levels was predicted to significantly increase serum PFAS levels (Post 2017), particularly for these long-chain PFAAs, which are not metabolized and are slowly excreted with human half-lives of several years. That allows PFAA serum levels to remain elevated and cumulated for many years after exposure ends (Chang et al., 2008; Olsen et al., 2009). Studies of exposed communities and predictions based on toxicokinetic factors show that low levels of PFAAs in drinking water (i.e., well below 100 ng/L [parts per trillion]) substantially increase blood serum levels. Empirical observations and toxicokinetic models demonstrate that serum PFOA levels in adults increase on average by more than 100 times the drinking water concentration (Bartell et al., 2017; Post et al. 2017), with greater predicted increases for PFOS and PFNA. Even with no additional exposure from contaminated drinking water and decreased PFAS serum in the general population, the Health Effects Subcommittee of the New Jersey Drinking Water Quality Institute expressed concerns about the impact of the predicted increases in serum level because of multiple human health effects (e.g., increased serum cholesterol, decreased response to vaccinations, and others) are associated with the serum PFAS levels prevalent in the general population (Post 2020; Hew Hampshire Department of Health and Human Services, 2015).

Policy and Regulation

Under the lengthy and complex process for national regulation of new drinking water contaminants established by legislation in 1996, the U.S. EPA have not established national drinking water standards (i.e., maximum contaminant levels [MCLs]) for PFAS (Association of State Drinking Water Administrators 2019). In 2016, the U.S. EPA (U.S. Environmental Protection Agency 2016a, 2016b) finalized nonregulatory lifetime drinking water Health Advisories of 70 ng/L for the individual and total concentrations of PFOA and PFOS, the most thoroughly studied members of the PFAS class. These lifetime Health Advisories updated the earlier U.S. EPA provisional short-term Health Advisories (applicable to exposure durations of weeks to months) of 400 ng/L for PFOA and 200 ng/L for PFOS that were established in 2009 (US Environmental Protection Agency 2009).

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Some states have developed more stringent drinking water standards or guidance values for PFOA and PFOS Seven of the nine states have developed Reference Doses for both PFOA and PFOS, whereas Vermont uses the U.S. EPA Referenced Doses without modification (Post 2020). Some states have developed guidelines for other PFAS (Environmental Council of the States 2020; Interstate Technology and Regulatory Council 2020b), and the US Agency for Toxic Substances and Disease Registry (2018) has developed toxicity factors for PFOA and PFOS that are approximately an order of magnitude lower than the U.S. EPA's.

Drinking water guidelines are developed based on noncarcinogenic and/or cancer effects. The primary consideration in the development of drinking water guidelines are the toxicity factor and the exposure

assumptions. When suitable data are available, human studies are preferred as the basis for risk assessment. Although evidence for multiple human health effects from PFOA, PFOS and other long-chain PFAAs has become available, the U.S. EPA and states have concluded that the human data have limitations that preclude their use as the primary basis for risk assessment. For example, the dose-response relationship for a health endpoint cannot be determined for individual PFAS due to frequent correlations among multiple PFAS (Post 2017). Therefore, the U.S. EPA and states have used animal toxicology data for developing current U.S. drinking water guidelines for PFAS.

The U.S. EPA stated that risk assessment for carcinogenic effects are generally based on the "nonthreshold assumption" that there is some risk from any dose (U.S. EPA 2005). Drinking water guidelines for carcinogenic effects are developed with a cancer potency factor (ng/kg body wt/day) that relates dose to cancer risk and a specific cancer risk. For noncarcinogenic effects, drinking water guidelines are developed with a Reference Dose (ng/kg body wt/day). Current state and U.S. EPA guidelines for these PFAS are based on noncarcinogenic effects (i.e., a Reference Dose), with the exception of California's recent PFOA guidelines which is based on cancer potency factors and a cancer risk levels of 1 in-10,000 (10^{-4}) for response levels.

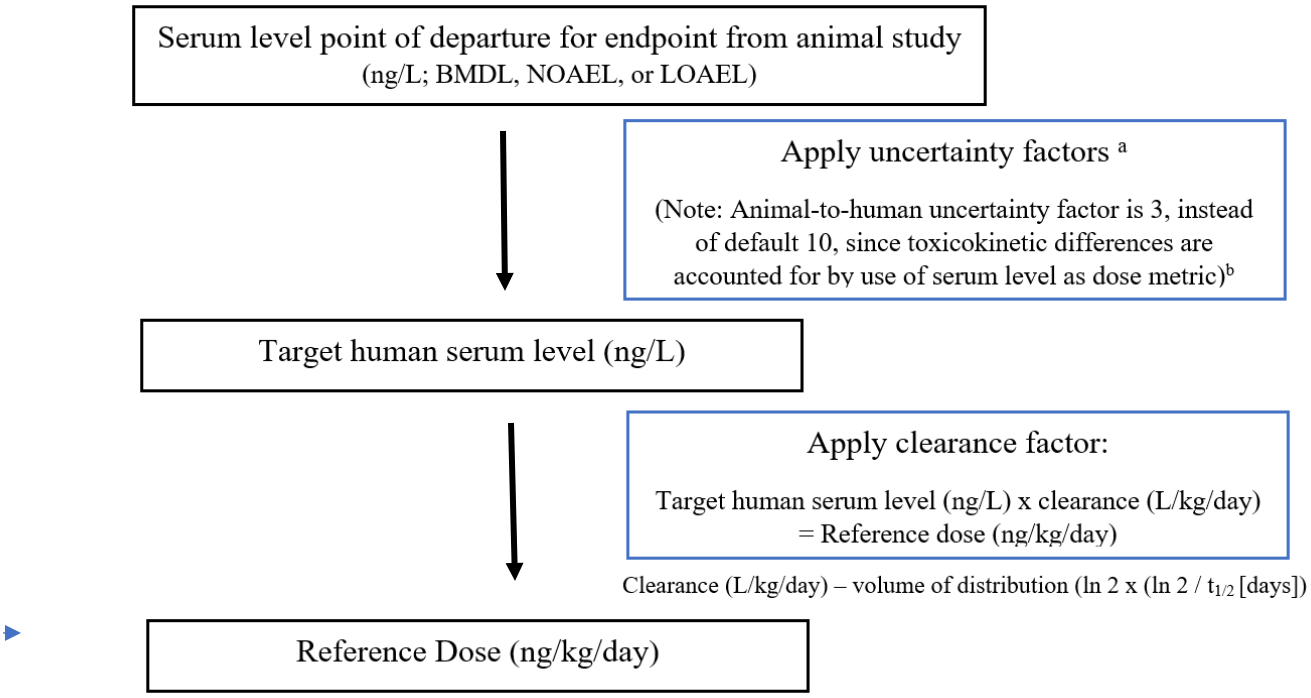


Figure 5 Process for development of per- and polyfluoroalkyl substance (PFAS) Reference Doses

[a] Uncertainty factors were applied after application of the clearance factor in some cases; this approach is mathematically equivalent to the approach shown and does not affect the resulting Reference Dose.
[b] All uncertainty factors used for state and U.S. EPA BMDL = lower confidence limit on the benchmark dose; LOAEL = lowest-observed–adverse effect level; NOAEL = no-observed– adverse effect level; RfD = Reference Dose.

Source: *Recent US State and Federal Drinking Water Guidelines for Per- and Polyfluoroalkyl Substances* by G. B. Post, 2020.

The guidelines developed based on noncarcinogenic effects are determined by three parameters: Reference Dose, relative source contribution, and ingestion rate. A Reference Dose is defined by the USEPA as an estimate of daily oral exposure to the human population that is likely to be without an appreciable risk of deleterious effects during a lifetime. A Reference Dose is based on a critical effect, which is the most sensitive toxicological endpoint that is well established, adverse, and considered relevant to humans (Figure 5).

New Jersey (NJ), New Hampshire (NH), and New York (NY) developed Reference Doses by considering the toxicological effects of increased liver weight in mice (Loveless et al., 2006; Macon et al., 2011). The NY Drinking Water Quality Institute Health Effects Subcommittee concluded that increased liver weight caused by PFOA progresses to more severe hepatic effects and is relevant to humans based on mode-of-action data (New Jersey Drinking Water Quality Institute Health Effects Subcommittee 2017a). Other states, Michigan (MI), Washington (WA), Minnesota (MN), Vermont (VT), and Massachusetts (MA), developed Reference Doses by considering development endpoints. MI and WA considered neurobehavioral and skeletal effects that persist to adulthood (Onishchenko et al. 2011; Koskela et al. 2016), whereas MN, VT, MA, and US EPA use less sensitive development effects; delayed ossification (males) and accelerated puberty (Lau et al. 2006).

Treatment Technologies

The Treatment Technology subgroup was tasked with researching and evaluating current methods for PFAS removal from drinking water supplies. There are currently three PFAS removal technologies widely used and available: granular activated carbon, ion exchange, and membrane separation (reverse osmosis). This section includes a description of each technology, along with pilot and field studies, and cost breakdown.

Granular Activated Carbon

Granular Activated Carbon (GAC) has been used historically in water treatment processes to reduce or remove organic contaminants and is the most studied treatment for PFAS removal (EPA 2018). Activated carbon is typically used for its highly porous structure as well as its large surface area for contaminants to attach (ITRC 2020). Activated carbon is made from organic materials with high carbon contents typically in a granular form: primarily wood, lignite, coal, or coconut shell. Removing PFAS from the water via GAC utilizes a physical mass transfer process from the aqueous phase onto solid media and does not use or involve chemical degradation or transformation (ITRC 2020). In this treatment process, water is taken from the source and directed through the treatment system where adsorption occurs. Figure 6 shows a standard GAC treatment process.

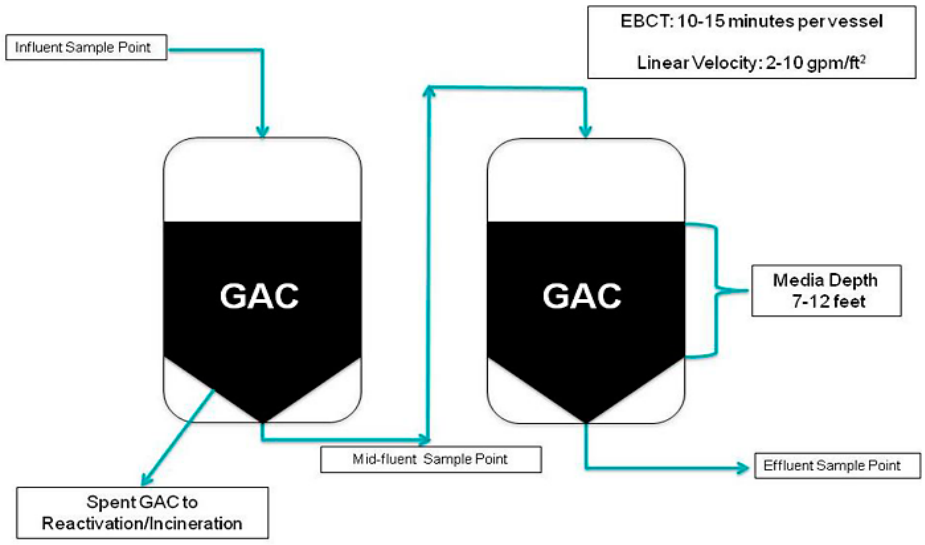
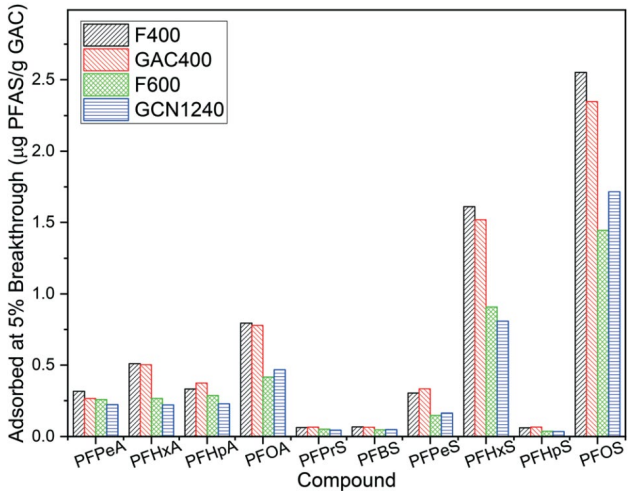


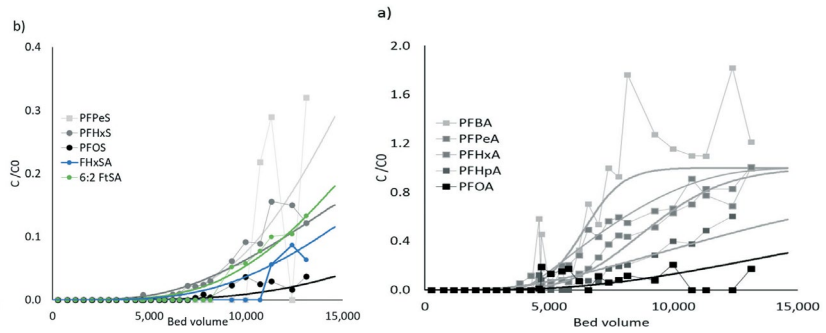
Figure 6 Typical GAC Treatment System Process Flow Diagram

Source: Calgon Carbon Corporation within ITRC PFAS – Per- and Polyfluoroalkyl Substances Treatment Technologies

GAC is the most commonly studied treatment technology, and has been analyzed and examined in several pilot and field studies. Literature confirms that perfluorinated sulfonates are more readily adsorbed than perfluoroalkyl acids, long-chain PFAS are more readily absorbed than shorted chain PFAS, and the presence of competing co-contaminants can harm performance (Appleman 2013; Appleman 2014; McCleaf 2017; Inyang 2017; Knappe 2018; Gagliano 2020; Belkouteb 2020; Kothawala 2017; Park 2020a; Zeng 2020; Wang 2020). Below is a breakdown and summary of relevant pilot and field studies involving PFAS removal using GAC technology.

The US EPA Drinking Water Treatability Database (TDB) reports the following selected findings from recent literature in support of GAC on the removal of PFAS from water:

	Title	Author/Year	Key Findings
PILOT STUDIES	Removal of per- and polyfluoroalkyl substances (PFASs) from contaminated groundwater using granular activated carbon: a pilot-scale study with breakthrough modeling	Liu, Werner, Bellona, 2019	Four commercially available granular activated carbons were compared to remove both long-chain and short-chain PFAS.  Figure 7 Adsorbed PFAS mass per gram of carbon at 5% breakthrough for all carbons tested Chain length dependent breakthrough was observed with exceptions for PFHpA and PFHpS. Carbon type mattered less for shorter chain compounds, but F400 and GAC400 performed 40–50% better than F600 and GCN1240 for longer chain compounds. This is most likely due to higher percentages of transport pores resulting in less intraparticle diffusion limitations.
	Recently Detected Drinking Water Contaminants: GenX and Other Per- and Polyfluoroalkyl Ether Acids	Hopkins, Sun, DeWitt, Knappe, 2018	PFEA removal was studied in a post-filter GAC adsorber. The empty bed contact time (EBCT) averaged 14 min, and the plant treated water for approximately 9 h/day. From the article, *"*Both the full-scale and pilot-scale results illustrate that GAC is only somewhat effective for controlling GenX in the context of treating coagulated Cape Fear River water. Recognizing that the absorbability of PFASs decreases with decreasing perfluorinated carbon chain length."
	Pilot scale removal of per- and	Rodowa, Knappe, Chiang,	The pilot-scale system was installed alongside a full-scale GAC treatment system to mimic the operational conditions of the full-scale GAC system and to study PFAS breakthrough.

	polyfluoroalkyl substances and precursors from AFFF-impacted groundwater by granular activated carbon	Pohlmann, Varley, Bodour, Field, 2020	 Figure 8 Breakthrough curves with model fits for a) PFSAs, FHxSA, and 6:2 FTS in lag vessel effluent

	Title	Author/Year	Key Findings
FIELD STUDIES	Treatment of poly- and perfluoroalkyl substances in U.S. full-scale water treatment systems	Appleman, Higgins, Quinones, Vanderford, Kolstad, Zeigler-Holady, Dickenson, 2014	Four full-scale GAC systems were examined. Longer chain PFAS were more effectively removed via GAC. Table 1 show that if the shorter chain PFBA is targeted for removal, an alternative treatment strategy would need to be used rather than GAC. Table 1 Percent removal for most effective PFAS treatment technologies - GAC
	Recommendation on Perfluorinated Compound Treatment Options for Drinking Water	Cummings, Matarazzo, Nelson, Sickels, Storms, 2015	From report, *"At the present time the members of the treatment subcommittee recommend that the use of granulated activated carbon (GAC) or an equally efficient technology should be considered for treatment of PFNA, PFOA and PFOS detected above the DWQI recommended MCL subject to the on-site pilot testing performance results."* The subcommittee consulted multiple case studies: Little Hocking, Ohio; Oakdale, Minnesota; New Jersey American Water, Penns Grove; New Jersey American Water, Logan System Birch Creek; and Amsterdam, Netherlands. The below table shows the pre-

Site	#7	#20
Treatment	GAC	GAC
Sample date	8/21/2012	4/25/2007–4/22/2008
PFBA	33%	−17%
PFPeA	74%	>22%
PFHxA	91%	>68%
PFHpA	>89%	N/A
PFOA	>48%	>92%
PFNA	>37%	N/A
PFDA	N/A	N/A
PFUnA	N/A	N/A
PFDoA	N/A	N/A
PFBS	>96%	N/A
PFHxS	>96%	>41%
PFOS	>89%	>95%
PFDS	N/A	N/A
FOSA	N/A	N/A
N-MeFOSAA	N/A	N/A
N-EtFOSAA	N/A	N/A

and post-treatment data from the Logan System Birch Creek case study

Date	Raw Water (ng/L)	GAC Treated Water (ng/L)
9/27/2011	60	N/A
2/14/2012	50	N/A
6/14/2012	72	<5
8/1/2012		<5
11/27/2012	46	<5
1/18/2013		<5
2/28/2013	54	<5
5/30/2013	50	<5
4/24/2014	70	<5
9/10/14	18	<5

Table 2 New Jersey American Water – Logan System Birch Creek PFNA Pre- and Post-Treatment Data

Table 2 shows that the Birch Creek study had detections of PFNA, PFOA, and three other PFCs, and all of which were removed below the reporting level of 5 ng/L with the installation of granulated activated carbon.

The Environmental Protection Agency (EPA) Drinking Water Treatability Database (TDB) reports the following findings from recent literature in support of GAC on the removal of PFAS from water:

- Up to greater than 99 percent removal of PFBA
- Up to greater than 99 percent removal of PFBS
- Up to greater than 99 percent removal of PFHpA
- Up to greater than 99 percent removal of PFNA
- Up to greater than 99 percent removal of PFHxS
- Up to greater than 99 percent removal of PFHpS
- Up to greater than 99 percent removal of PFNA
- 96 percent removal of PFNS

Ion Exchange

Ion exchange treatment, or resins, is another treatment technology capable of removing PFAS from water. The resins consist of highly porous, polymeric material which is acid, base, and water-insoluble, and are made from hydrocarbons**.** (EPA 2018). The ion exchange resins are grouped into two groups, cationic and anionic, each serving a different purpose: cationic exchange resins (CER) remove positively charged contaminants, while anion exchange resins (AER) more effectively remove negatively charged contaminants including PFAS (USEPA 2018; ITRC 2020). The resins act like magnets, attracting ionic contaminants as water passes through the system. There are two resin options for the treatment process, single-use or regenerable resins.

Single-use resins are used until breakthrough, then removed and disposed of by high-temperature incineration or landfilling.
Regenerable resins are used until breakthrough, then regenerated on-site with a specific solution to return resin to full exchange capacity.

Removing PFAS by ion exchange is a physical mass transfer process, similar to GAC, and does not involve chemical degradation or transformation (ITRC 2020). AER resins remove PFAS by forming ionic bonds with the sulfonic and carboxylic acid heads of PFOS and PFOA, while simultaneously the hydrophobic end of the PFAS structures adsorb onto the hydrophobic surfaces of the resins (ITRC 2020). Figure 9 shows a standard single-use resin ion exchange process.

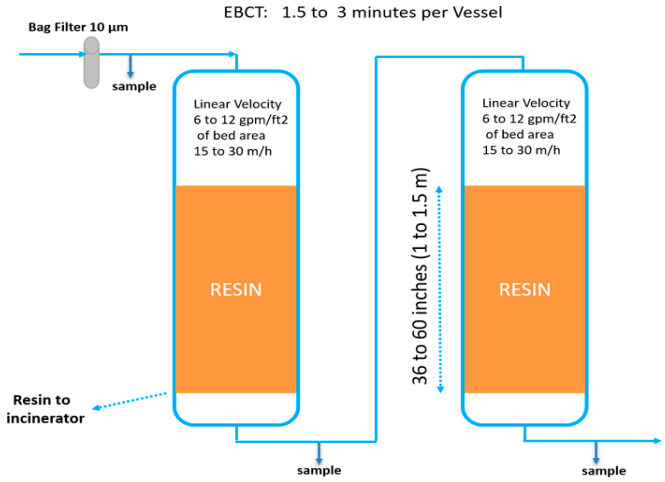
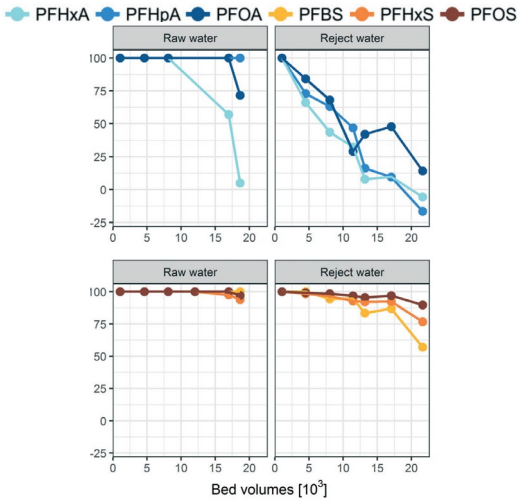


Figure 9 Single-use IEX process flow diagram

Source: Purolite Corporation within ITRC PFAS – Per- and Polyfluoroalkyl Substances Treatment Technologies

While not as commonly used as GAC, ion exchange technology for PFAS removal is well established. Pilot and field studies have shown that single-use resin has a higher removal capacity than regenerable resin, and is more fully exhausted in a lead-lag vessel comparatively (McCleaf 2017; Kothawala 2017; Zeng 2020; Wang 2020; Dudley 2014; Arevalo 2014; Boodoo 2018; Zaggia 2016; Lombardo 2018; Chuan 2017; Woodard 2017; Widefield Water and Sanitation District 2018; Casey 2018; Mohseni 2019; Mende 2019; Franke 2019; Schaefer 2019; Dixit 2020; Dixit 2021; Hopkins 2018; Kumarasamy 2020; Park 2020b; Yan 2020). However, literature shows that the relative efficiency of single-use and regenerable reins depends upon PFAS and co-contaminant influent concentrations and treatment goals (Appleman 2014; McCleaf 2017; Knappe 2018; Gagliano 2020; Zaggia 2016; Woodard 2017; Kumarasamy 2020; Park 2020b). Below is a breakdown and summary of selected relevant pilot and field studies involving PFAS removal using ion-exchange technology:

	Title	Author/Year	Key Findings
PILOT STUDIES	Removal Efficiency of Multiple Poly- and Perfluoroalkyl Substances (PFASs) in Drinking Water using Granular Activated Carbon (GAC) and Anion Exchange (AE) Column Tests	McCleaf, Englund, Ostlund, Lindegren, Wiberg, Ahrens, 2017	The removal of 14 different PFASs was monitored for a 217-day period. Saw a clear relationship between perfluorocarbon chain length and removal efficiency of PFASs while PFASs with sulfonate functional groups displayed greater removal efficiency than those with carboxylate groups. Time to column breakthrough increased with increasing perfluorocarbon chain length and was greater for the PFSAs than the PFCAs.
	Use of Strong Anion Exchange Resins for the Removal of Perfluoroalkylated Substances from	Zaggia, Conte, Falletti,	The strength of hydrophobic interactions between the functional group of the resin and PFAS molecules plays a key role in explaining differences in term of exchange capacity. Resins A600E and A520E showed a reduced sorption capacity compared to resin A532E. A600E and A520E can be

	Contaminated Drinking Water in Batch and Continuous Pilot Plants	Fant, Chiorboli, 2016	regenerated, but A532E requires concentrated solutions thus was regarded as non-regenerable. Transmission electron analysis on saturated resins showed that large molecular macro-aggregates of PFAS can form in the intraparticle pores of resin. This indicates that ion exchange is not the only mechanism involved in PFAS removal.
	Efficient removal of per- and polyfluoroalkyl substances (PFASs) in drinking water treatment: nanofiltration combined with active carbon or anion exchange	Franke, McCleaf, Lindegren, Ahrens 2019	This studied combined nanofiltration with granular activated carbon and anion exchange. Anion exchange had 3x greater half-time of saturation than GAC. However, anion exchange showed a higher rate of decreasing efficiency, while GAC removed approximately 20% of incoming PFAS concentrations consistently after treatment of 15000 bed volumes.  Figure 10 Removal efficiency [%] of frequently detected PFCAs (top) and PFSAs (bottom) for the evaluated granular activated carbon material (F400, left) and anion exchange resin (A600, right) depending on bed volumes treated
	Assessing Rapid Small-Scale Column Tests for Treatment of Perfluoroalkyl Acids by Anion Exchange Resin	Schaefer, Nguyen, Ho, Im, LeBlanc 2018	The study saw that short-chained PFAAs migrated through columns more rapidly than long-chained PFAAs. Additionally, for a given chain length, the perfluorinated carboxylates migrated through the anion exchange resins more rapidly than the corresponding perfluorinated sulfonates. RSSCT approach to expedite bench-scale testing can be applied for PFAA uptake onto AERs.

FIELD STUDIES	Treatment of poly- and perfluoroalkyl substances in U.S. full-scale water treatment systems	Appleman, Higgins, Quinones, Vanderford, Kolstad, Zeigler-Holady, Dickenson 2014	Two anion exchange treatments were examined at the full-scale in this study. The resin was successful in reducing some PFAS levels; the reduction rates were as followed: PFHpA 46%, PFOA 75%, PFBS 81%, PFNA >67%, PFHxS >9% and PFOS >92%. PFSAs were preferably removed over PFCAs as shown in Table 3 below.

Site	#14	#14
Treatment	AIX	AIX
Sample date	5/30/2012	9/19/2012
PFBA	−9%	0%
PFPeA	0%	0%
PFHxA	14%	−14%
PFHpA	54%	38%
PFOA	76%	73%
PFNA	N/A	>67%
PFDA	N/A	N/A
PFUnA	N/A	N/A
PFDoA	N/A	N/A
PFBS	83%	80%
PFHxS	>97%	>98%
PFOS	>90%	>94%
PFDS	N/A	N/A
FOSA	N/A	N/A
N-MeFOSAA	N/A	N/A
N-EtFOSAA	N/A	N/A

Table 3 Percent removal for most effective PFAS treatment technologies - AIX

Membrane Separation (Reverse Osmosis)

PFAS removal using membranes in processes such as reverse osmosis is proven to be extremely effective. Reverse osmosis works to remove PFAS by pushing highly pressurized water through a semipermeable membrane (ITRC 2020). The need to pressurize the water to push it through the membranes results in significant increases in energy costs. These membranes reject certain organic and inorganic compounds, depending on the sizes of the pores and compounds, and new technology has increased efficiency while lowering operating pressures and costs. However, it is important to note that the waste discharge from the Reverse Osmosis process will contain concentrated levels of the PFAS removed from the source water, making disposal difficult (Appleman, 2014). Treated water passes through the membrane, and the rejected water is collected for disposal or discharge. Reverse osmosis has been combined with nanofiltration to increase PFAS removal (ITRC 2020). Nanofiltration provides high water flux at low operating pressure, and combining it with reverse osmosis utilizes properties of both.

Nanofiltration membranes reject hardness to a high degree but will pass sodium chloride. The product water will retain some minerals.

Reverse osmosis membranes reject all salts to a high degree and are tighter. The product water will contain very few dissolved minerals and very few dissolved organic molecules.

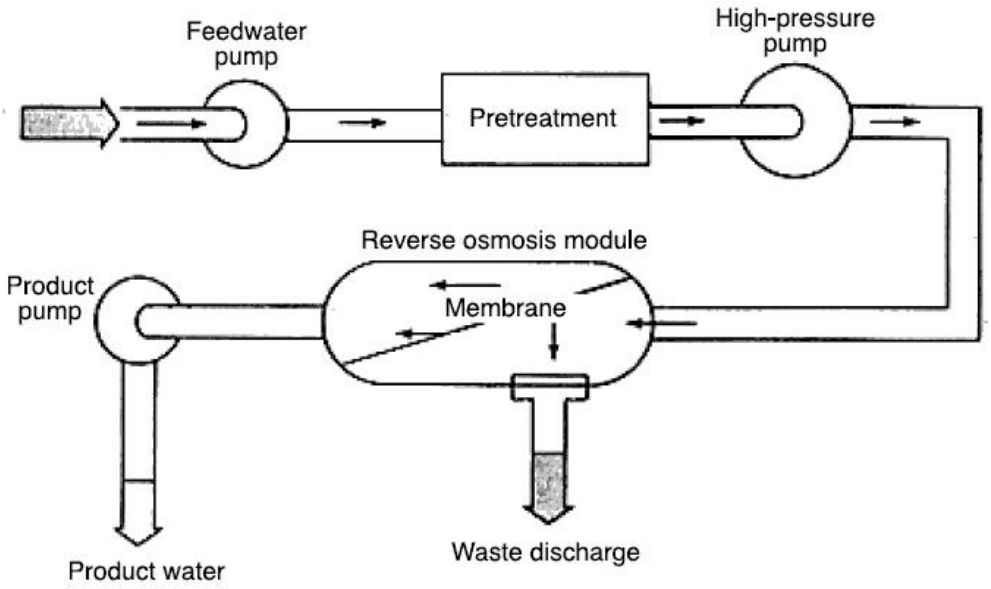


Figure 11 Reverse Osmosis Plant Simple Process
Source: RO Water Treatment Plant. Accessed from https://www.thewatertreatments.com/water-treatment-filtration/reverse-osmosis-plant-ro-desalination/

Reverse osmosis membrane separation is extremely effective in PFAS removal. Pilot and field studies have supported that reverse osmosis (RO) membranes achieved PFAS removals of up to greater than 99 percent (Appleman 2014; Smith 2018; Franke 2019; Horst 2018; Thompson 2011; Quiñones 2009; Dickenson 2016). Below is a breakdown and summary of relevant pilot and field studies involving PFAS removal using membrane separation and/or reverse osmosis technology.

	Title	Author/ Year	Key Findings
PILOT STUDIES	Efficient removal of per- and polyfluoroalkyl substances (PFASs) in drinking water treatment: nanofiltration combined with active carbon or anion exchange	Franke, McCleaf, Lindegren, Ahrens, 2019	This studied combined nanofiltration with granular activated carbon and anion exchange. Table 4 shows the average concentrations of frequently detected PFASs in raw water, membrane permeate and reject water throughout the full 35-week membrane experiment.
	Water Treatment Technologies for PFAS: The Next Generation	Horst, McDonough, Ross,	The article reports that reverse osmosis is highly effective for PFAS removal, greater than 99%. However, the technology has limitations including capital cost for larger systems and low throughput flow rates

	PFBA	PFPeA	PFHxA	PFHpA	PFOA	PFBS	PFHxS	PFOS
MW [g mol^{-1}]	213.03	263.04	313.04	363.05	413.06	299.09	399.10	499.12
Raw water	<6.0	<7.0	14	<8.0	<10	11	110	39
Permeate	<6.0	<5.0	<5.0	<5.0	<5.0	<5.0	<10	<7.0
Reject water	12	12	65	15	35	47	470	190
C_{REJ}/C_{RAW}	>2.0	>1.7	4.6	>1.9	>3.5	4.3	4.4	5.0

Table 4 Average concentrations [ng L−1] of frequently detected PFASs in raw water, membrane permeate and reject water throughout the full 35 week membrane experiment

		Dickson, Miles, Hurst, Storch, 2018	which constrain the range of useful applications in point of entry [to a service connection treatment] systems.
FIELD STUDIES	Treatment of poly- and perfluoroalkyl substances in U.S. full-scale water treatment systems	Appleman, Higgins, Quinones, Vanderford, Kolstad, Zeigler-Holady, Dickenson, 2014	With low pressure membrane filtration, that facility that utilized polypropylene membranes with 0.2 micron rated pore size reported only small reductions of contaminant levels (24% of PFOS, 44% of PFDoA and 42% of PFOSA). However, RO was effective at removing short-chained PFAS such as PFBA, which was unmanageable through all other treatment technologies.

The EPA Drinking Water Treatability Database (TDB) reports the following findings from recent literature in support of membrane separation technology on the removal of PFAS from drinking water (USEPA, 2017). There are issues with contaminated waste generated during the process.

- Up to 99.9 percent removal of PFBA
- Up to 99.8 percent removal of PFBS
- Up to greater than 99 percent removal of PFPeA
- Up to greater than 99 percent removal of PFHxA
- Up to greater than 99 percent removal of PFHxS
- Up to 99 percent removal of PFHpA
- Up to 99 percent removal of PFNA
- Up to greater than 99 percent removal of PFDA
- Up to 99 percent removal of PFDS
- Up to 99 percent removal of PFUnA

Treatment Costs

The Environmental Protection Agency has compiled work breakdown structure-based models on the cost of adding granular activated carbon treatment, anion exchange treatment, and reverse osmosis treatment to drinking water facilities. While these models and documents are free to the public, they are not specific to PFAS or any other pollutant. The EPA also supplies Excel templates in which treatment facility specs can be entered to determine the cost of adding said technology to the specific plant. All the above information can be viewed and retrieved from the Drinking Water Treatment Technology Unit Cost Models, accessed at https://www.epa.gov/sdwa/drinking-water-treatment-technology-unit-cost-models.

For GAC treatment, the EPA provides one example on costs for incorporating this particular treatment technology. The below graph charts the possible costs of GAC with the following assumptions in mind: Two vessels in series; 20-minute Empty Bed Contact Time total; Bed Volumes fed (1,1-DCA = 5,560 (7.5-minute EBCT); Shorter Chain PFCA = 4,700; Gen-X = 7,100; Shorter Chain PFS = 11,400; PFOA = 31,000; PFOS = 45,000); seven percent Discount rate; Mid-level cost.

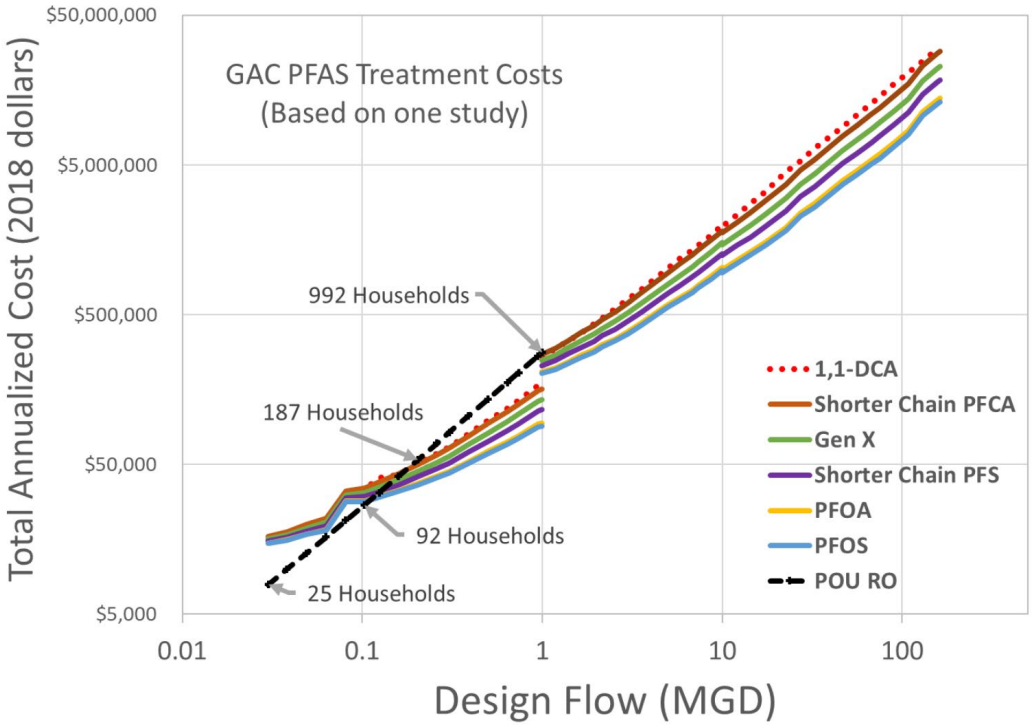


Figure 12 Costs for PFAS Treatment – One GAC Example
Source: EPA's "PFAS Treatment in Drinking Water and Wastewater – State of the Science" webinar

The EPA also provides one example for predicted costs of incorporating IEX treatment technology into a drinking water treatment plant, again with assumptions: Two vessels in series; 3-minute EBCT total; bed volume fed (Shorter Chain PFCA = 3,300; Gen-X = 47,600; Shorter Chain PFS = 34,125; PFOA = 112,500; PFOS = 191,100); seven percent Discount rate; Mid-Level cost. these costs are for removal of the one specific PFAS compound, not a group of compounds, and does not include disposal of contaminated media.

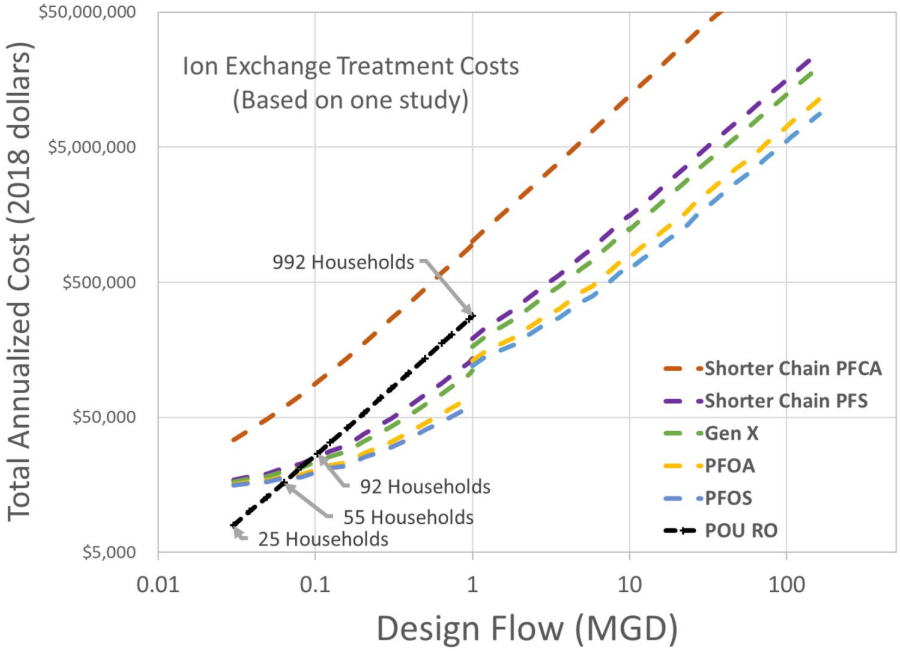


Figure 13 Costs for PFAS Treatment – One IX Example
Source: EPA's "PFAS Treatment in Drinking Water and Wastewater – State of the Science" webinar

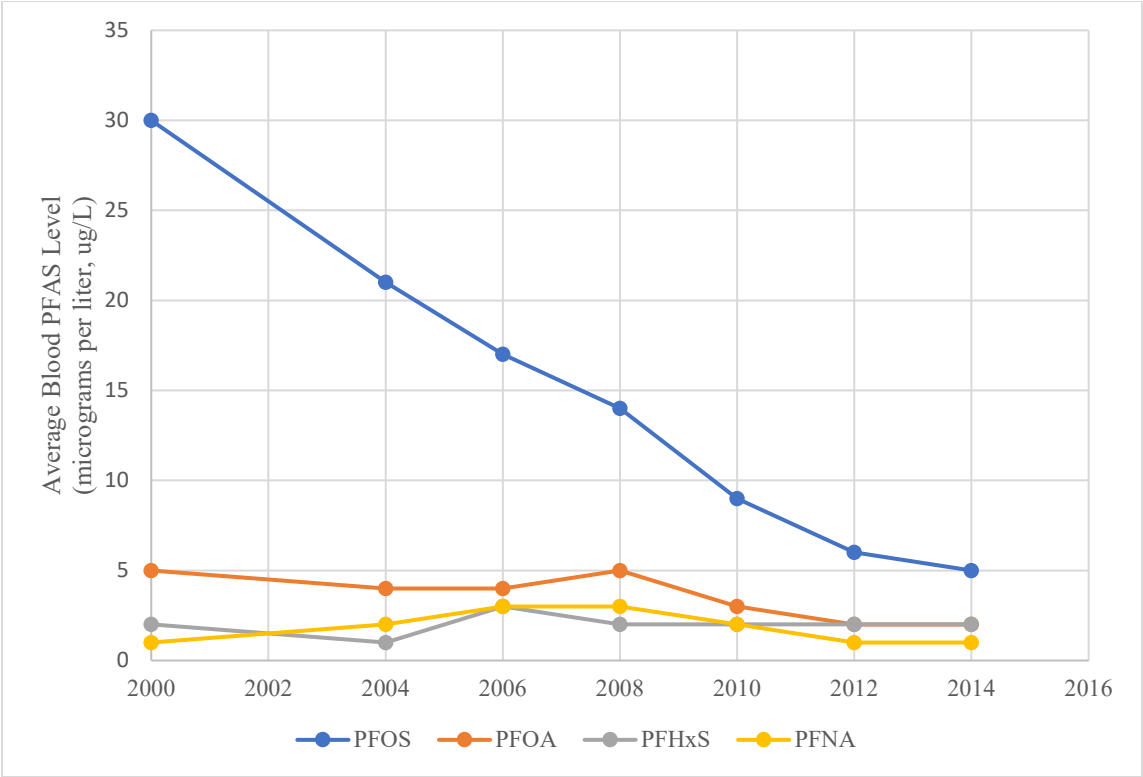


Figure 14 Blood Levels of the Most Common PFAS in People in the United States from 2000-2014

Source: Centers for Disease Control and Prevention. Fourth Report on Human Exposure to Environmental Chemicals, Updated Tables, (January 2017). Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

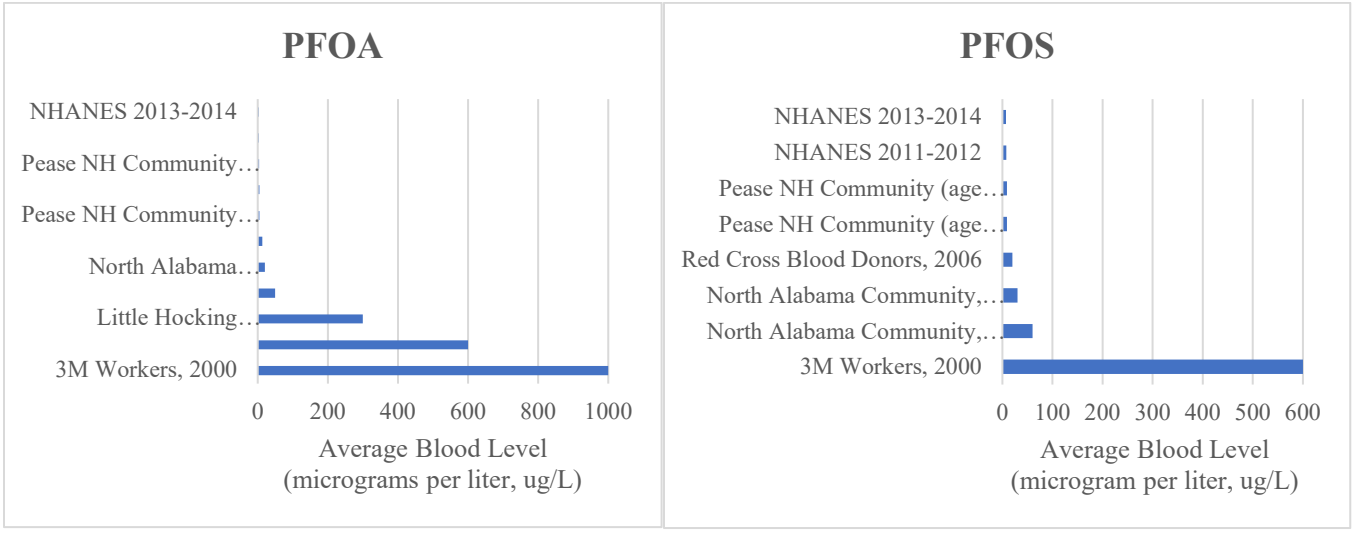


Figure 15 PFOA and PFOS Levels Measured in Different Exposed Populations, Compared to Levels CDC Measured in the General U.S. Population in 2011-2012 and 2013-2014

Source: Centers for Disease Control and Prevention. Fourth Report on Human Exposure to Environmental Chemicals, Updated Tables, (January 2017). Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

Levels of PFOA are above the proposed standard MCLs in 10 -11 percent of public water systems sampled in the state according to data reported by the NHDES (New Hampshire Dept of Environmental Services, 2019b). Levels of PFOS, PFNA, and PFHxS each exceed the proposed standards in less than 5 percent of public water

systems (New Hampshire Dept of Environmental Services, 2019b). Early estimates were that only about 7- 9 percent of water systems in the state would need to be upgraded (Freise, 2020). Those water systems with high levels of PFOS, PFNA, and PFHxS are the ones that also have levels of PFOA above standards. The estimated costs for public water systems apply only to those public water systems where concentrations of one of the four substances in drinking water exceeds the proposed standards. If the water systems that will need to be upgraded serve about 11 percent of the state's population, then the average total annual cost per capita of upgrading public health systems in those communities will be between $68 and $139. Total annual costs for public water systems cost range between 10 million and 20 million (Table17) (Miller, 2020).

Table 5 Results of Animal Studies of the Association Between Exposure to PFAS and Toxicity

PFAS	ANIMAL	EXPOSURE PERIOD	DOSE	BIOLOGICAL RESPONSE	BIOLOGOCAL SYSTEM/ TOXICITY	REFERENCE
Potassium PFOS	Sprague Dawley rats	104 weeks	0, 0.5, 2, 5, and 20 g/g (ppm)	A 10% increase in hepatic tumors was 8 ppm for both sexes. Hepatocellular hepatocellular in liver, with ↑ proliferation of endoplasmic reticulum, vacuolation, and ↑ eosinophilic granulation of the cytoplasm	Liver/cancer	Butenhot et al. 2012
PFOS	Sprague-Dawley rats	Birth (PND0) to PND21	Dams received 0.1, 0.6 and 2.0 mg/kg bw PFOS by gavage from gestational day 2 (GD2) to GD21.	↑ Gene expression of inflammation biomarkers AP-1, NF-kB, cytokines interleukin and tumor necrosis factor (TNF)-a, and cAMP response element-binding protein ↑Astrocyte activation markers, glial fibrillary acidic protein and S100 calcium binding protein B in hippocampus and cortex upregulated on PND0 or PND21. ↓Synapsin 1(Syn1) and synaptophysin (Syn) in cortex and hippocampus	Developmental neurotoxicity	Zeng et al. 2011.
PFOS	Rats	6 weeks prior to mating, during mating, and, for females, through gestation and lactation, across two generations.	0, 0.1, 0.4, 1.6, and 3.2 mg/kg/day via oral gavage	No adverse effects were observed in F0 females or their fetuses upon caesarean sectioning at gestation day 10. PFOS did not affect reproductive performance. Neonatal toxicity in F1 pups occurred **only** at a maternal dose of 1.6 mg/ (kg day) or higher. In utero exposure to PFOS contribute to post-natal pup mortality in an additive fashion.	Reproductive toxicity	Luebker et al. 2005.
PFOS	B6C3F1 mice	28 days	0, 0.005, 0.05, 0.1, 0.5, 1, or 5 mg/kg	No lymphocyte proliferation altered in either gender. ↑Natural killer cell activity in male **only**	Immunotoxicity	Peden-Adams et al. 2008.

			total administered dose	T-cell subpopulations modulated in males only at 0.1 mg/kg.		
PFOS	C57BL/6 mice	0, 0.5, 5, 25, 50, or 125 mg/kg	60 days	↑Liver mass at ≥5 mg PFOS Altered lymphocyte proliferation and natural killer cell activity Plaque forming cell response was suppressed (≥5 mg/kg). No observed and lowest observed adverse effect level at 0.5 and 5 mg/kg, respectively. Affected immunity function in mice at levels approximately 50-fold for highly exposed human populations.	Chronic immunotoxicity	Dong et al. 2009.
PFOS	Female Crl:CD®(SD)IGS VAF/Plus® rats	0.4, 0.8, 1.0, 1.2, 1.6 and 2.0 mg/kg/day	Two-generational reproduction periods	Dose levels for the dose–response and etiological investigation 0.0, 0.4, 0.8, 1.0, 1.2, 1.6, and 2.0 mg/kg/day. ↓Gestation length, viability through lactation day 5 (≥ 0.8 mg/kg) ↓Viability through lactation day 5 No relationship between decreased neonatal survival and reductions in lipids, glucose utilization, or thyroid hormones. Late-stage fetal development may be affected in pups exposed to PFOS in utero and may contribute to the observed mortality.	Reproductive/Developmental toxicity	Luebker et al. 2005.
PFOS	Adult male C57BL/6N mice	6 weeks	100 µg PFOS/kg/day via oral gavage 2.5 µM for glucose production testing	No alter CR-induced weight loss, white adipose tissue mass, or liver weight over 6 weeks. ↑ hepatic triglyceride accumulation in hepatocytes due to ↓ phosphorylated AMPK expression in liver. Disrupted hepatic lipid and glucose homeostasis at 2.5 µM	Liver toxicity	Salter et al. 2021.

PFOS	Adult male and female wild-type C57BL/6 mice	2 or 4 weeks	3 and 1.5 µg/kg/day	No weight loss during exposure (100 ng/ml in serum). No effect on T-cell development. Exposure to PFOS at 1.5 µg/kg/day of PFOS for 4 weeks does not affect weight loss, survival and viral clearance (1.5 µg/kg/day). No suppression on the immune cell development or antigen specific immune response.	Immunotoxicity	Torres et al. 2020.
PFOA	C56BL/6J mice, C57BL/6N mice	C56BL/6J mice: 10 days; C57BL/6N mice: 15 days	C56BL/6J mice: 0 or 30 mg PFOA/kg/day C57BL/6N mice: 0-30 mg/kg/day of PFOA in drinking water.	↓ IgM antibody synthesis ↑ IgG titers were elevated at 3.75 and 7.5 mg PFOA/kg/day.	Immunotoxicity	DeWitt et al. 2008
PFOA	CD-1 Mice	Full-gestation: 17-day period (days 1-17). Late-gestation: 7-day period (days 10-17)	Full-gestation study: administered 0, 0.3, 1.0, and 3.0 mg PFOA/kg body weight /day from gestation days (GD) 1–17. Late-gestation study: 0, 0.01, 0.1, and 1.0 mg PFOA/kg body weight/day from GD 10–17.	A no observable adverse effect level for mammary developmental delays ↑offspring relative liver weights in all treatment groups in the full-gestation study and in the 1.0 mg PFOA/kg group in the late-gestation study. ↑ mammary epithelial growth in the offspring At postnatal day 21, mammary glands from the 1.0 mg/kg GD 10– 17 group had significantly less longitudinal epithelial growth and fewer terminal end buds compared with controls ($p < 0.05$).	Developmental Effects	Macon et al. 2011
PFOA	Pregnant ICR mice	Gestational day 0 to 17 and 18 for prenatal and postnatal	1, 5 and 10 mg/kg/daily by gavage	No maternal death. ↑ Liver weight, hepatocellular hypertrophy, necrosis, mitosis at 10 mg/kg.	Reproductive toxicity	Yahia et al. 2010.

		evaluations, respectively		↓Fetal body weight at 5 and 10 mg/kg. ↓ Neonatal survival rate at 5 and 10 mg/kg.		
PFOA	CD-1 mice	Gestational day (GD) 1 to 17	1, 3, 5, 10, 20, or 40 mg/kg/daily by oral gavage	Weight gain in dams that carried pregnancy to term was significantly lower in the 20-mg/kg group. The incidence of live birth in group B mice was significantly lowered by PFOA. Postnatal survival was severely compromised at 10 or 20 mg/kg, and moderately so at 5 mg/kg.	Reproductive toxicity	Mouse by Lau et al. 2006.
PFOA	Male and female C57BL/6 and BALB/c mice	5 weeks	One diet was the control diet with no added PFOA. The other diet contained 3.5 mg PFOA (Sigma–Aldrich)/kg diet.	Marked hypercholesterolemia in male and female C57BL/6 mice. C57BL/6 female mice being most responsive to PFOA.	Dietary Toxicity	Rebholz et al. 2016.
PFOA	C57BL/6 mice	0.3 mg /kg/day	17 months	↑femoral periosteal area ↓ Mineral density of tibias. ↑Osteocalcin expression and calcium secretion in osteoblasts at low concentrations ↓ Osteocalcin expression and calcium secretion in osteoblasts at 100 μM and above ↑ Osteoclasts numbers and resorption activity increased dose-dependently from 0.1–1.0 μM, but decreased at higher concentrations.	Developmental toxicity	Koskela et al. 2016
PFOA	BALB/c mice	7 days	PFOA in either 10, 50, and 100 pg was administered intratracheally	↑aggravated airway hyperresponsiveness and T helper type 2 (Th2) airway inflammation in asthmatic mice	Immunological toxicity	Zeng et al. 2021.

			after each ovalbumin-induced asthma, which is the same equivalent to breathing in PFOA from the atmosphere for 10, 30, and 60 weeks respectively.	↓expression of the GR mRNA was markedly reduced in the OVA (0.17±0.02), OVA+10 pg PFOA (0.13±0.06), OVA+50 pg PFOA (0.09±0.04) and OVA+100 pg PFOA (0.04±0.01) groups		
PFNA	CD-1 mice	1, 3, 5, or 10 mg PFNA/kg body weight per day on GD 1–17	17 days	Failed pregnancy at 10 mg/kg No effects on pregnancy and pups' survival at 5 mg/kg or lower doses in the first 10 days of life. 80% of these neonates died in the first 10 days of life Hepatomegaly in the pregnant dams at 5 mg/kg or lower doses.	Developmental toxicity	Das et al. 2015.
APFO	Male Crl:CD®BR rats	13 weeks	0, 1, 10, 30, and 100 ppm (equivalent to 0, 0.06, 0.64, 1.94, and 6.5 mg/kg/day) via diet	Liver weights, PCoAO activity, and hepatocyte hypertrophy were increased in the 10ppm dose group and above and were reversible in recovery. Hormone levels appeared unchanged. Serum PFOA concentrations at the end of the treatment period were 7.1, 41, 70, and 138 mg/mL in the 1, 10, 30 and 100 ppm dose groups.	Dietary Toxicity	Perkins et al. 2004.
PFHxA	Sprague-Dawley Rats	104 weeks	0, 2.5, 15, and 100 mg/kg/day of PFHxA (males) and 5, 30, and 200 mg/kg/day of PFHxA (females)	No effects on body weights or motor activity, serum chemistry, hematology, histologic parameters in the kidneys. ↓A dose-dependent in survival in **only** female rats No tumorigenic in male or female SD rats	Chronic Toxicity Carcinogenicity	Klaunig et al. 2015.
PFDA	Sprague–Dawley rats B6C3F1/N mice	Sprague-Dawley: 28 days	Sprague–Dawley: 0–2.0 mg PFDA/kg by oral gavage daily	Observed hepatocyte necrosis and hepatomegaly (0.5 mg PFDA/kg/d). Observed hepatomegaly (≥0.625 mg) PFDA/kg/week, while splenic atrophy	Liver and immune toxicity	Frawley et al. 2018.

		B6C3F1/N: 4 weeks	B6C3F1/N: once/week to 0–5.0 mg PFDA/kg	was observed at 5.0 mg PFDA/kg/week. ↓Total spleen cells, and Ig þ and NK þ cells (5.0 mg PFDA/kg/week). No changes in rats' leukocyte Alter the balance of immune cell populations in lymphoid tissues in mice.		
PFOS PFOA	C57BL/6/Bkl mice	0.3 mg/kg of PFOS or PFOA throughout pregnancy	Gestation + 21 days post-birth	↓ Locomotion in a novel environment and reduced muscle strength (males only) by PFOS exposure. Changed exploratory behavior in male and female offspring, and increased global activity in males in their home cage by PFOA exposure	Developmental toxicity	Onishchenko et al. 2011.
PFOA, PFBA	Mice	5 weeks	PFOA, 0.1, 1 and 5 mg/kg body weight via intake of drinking water PFBA, 5 mg/kg body weight	↑Marked moderate liver hypertrophy with signs of cell injury (5 mg/kg/body weight). Milder liver toxicity No concurrent evidence of lipid peroxidation and oxidative stress. No evidence of genotoxicity in liver.	Liver toxicity	Crebelli et al. 2019.
PFOA, GenX	CD-1 mice	Embryonic day (0) to 1.5 to 11.5 or 17.5 days	PFOA: 0, 1, or 5 mg/kg GenX: 0, 2, or 10 mg/kg	↑Gestational weight gain (10 mg/kg/d GenX exposure). ↓Embryo weight (5 mg/kg/d PFOA). ↑Incidence of placental abnormalities, higher maternal liver weights, changes in liver histopathology, embryo–placenta weight ratios (1 - 10 mg/kg)	Developmental toxicity	Blake et al. 2020.
PFDA	Sprague–Dawley rats B6C3F1/N mice	Sprague-Dawley: 28 days B6C3F1/N: 4 weeks	Sprague–Dawley: 0–2.0 mg PFDA/kg by oral gavage daily B6C3F1/N: once/week to 0–5.0 mg PFDA/kg	Observed hepatocyte necrosis and hepatomegaly (0.5 mg PFDA/kg/d). Observed hepatomegaly (≥0.625 mg) PFDA/kg/week, while splenic atrophy was observed at 5.0 mg PFDA/kg/week. ↓Total spleen cells, and Ig þ and NK þ cells (5.0 mg PFDA/kg/week). No changes in rats' leukocyte Alter the balance of immune cell populations in lymphoid tissues in mice.	Immune toxicity/cancer	Frawley et al. 2018.

PFOA	Kunming mice	Gestation day 0 through day 17	1, 2.5 or 5 mg/kg/BW PFOA daily by gavage during gestation	↓Survival number of offspring mice at weaning. No changes in the testicular index of offspring mice. Maternal exposure reduced the level of testosterone in the male offspring mice on post-natal day 21 but increased in 1 mg/kg group and decreased in 2.5 and 5 mg/kg groups on PND 70. ↓Leydig cells' number in 2.5 and 5 mg/kg PFOA groups on PND 21 and PND 70.	Developmental toxicity	Song et al. 2018.
PFOA	Sprague-Dawley rats	9 days	0, 25 or 50 mg/kg/day	↓ Serum testosterone levels by day 21 and day 56 post-EDS treatment. The expression levels of Leydig cell specific genes downregulated. Development of Leydig cells inhibited	Reproductive toxicity	Lu et al. 2019.
PFOS	Sprague-Dawley rats	Gestation days 2 to 21 for dosing, liver samples were taken at 21 days post-natal	0.1, 0.6 and 2.0 mg PFOS/kg/d and 0.05% Tween 80 as control by gavage	↓Global DNA methylation and methylation of LINE-1 regulatory only in the 2.0 mg/kg/d group. Up to 30% of critical CpG sites in GSTP promoter region were methylated in the livers of exposed rats, while p16 promoter methylation was not affected. Early-induced changes in cytosines within the GSTP gene promoter region as a possible biomarker of hepatic PFOS burden.	Hepatic toxicity	Wan et al. 2010.

Table 6 Results of Epidemiologic Studies of the Association between Exposure to PFAS and Human Health Impact

PFAS	STUDY DESIGN	SUBJECT	SAMPLE SIZE	DOSE MEASURE EXPOSURE ASSESSMNT	SAMPLE MEASURE	HEALTH IMPACT	REFERENCE
PFOS	Retrospective cohort	Manufacturing workers	1985	Work history records for estimating PFOS exposure and weighted with biological monitoring data.	Postal questionnaire to subjects to determine record of bladder cancer	No evidence of an association between bladder cancer and PFOS exposure. The limited size of the population prohibits a conclusive exposure response analysis.	Alexander et al. 2007.
PFOS, PFHxS	Cohort	Women from the Ronneby cohort, with high exposure to PFOS and PFHxS, emanating from drinking water	53	Serum levels of PFAS were analyzed using LC/MS/MS. High coverage microRNA expression was analyzed qPCR using Ingenuity pathway analysis (IPA).	Serum sample	Associated with downregulation of specific microRNAs (DNA methyltransferase 3 alpha, epidermal growth factor receptor, 3-hydroxy-3-methylglutaryl-CoA reductase, nuclear receptor subfamily 1, group H, member 3, peroxisome proliferator-activated receptor alpha, prostaglandin-endoperoxide synthase 2, and tumor growth factor alpha) with cardiovascular function, Alzheimer's disease, growth of cancer cell lines and cancer.	Xu et al. 2020.
PFOS, PFOA, PFHxS, PFHpS, PFNA,	Case–control study nested within the Danish National	Compared pregnancies ending in miscarriage during weeks	220	Measured in maternal plasma collected in early gestation;	Plasma sample	Maternal exposures to higher levels of PFOA, PFHpS, and PFAS mixtures were associated with the risk of miscarriage	Liew et al. 2020.

PFDA, PFOSA	Birth Cohort (DNBC, 1996–2002).	12–22 of gestation, with 218 pregnancies resulting in live births.				↑Odds for miscarriage associated with increasing PFOA and PFHpS levels. ↑ORs for the second or third quartile of PFHxS or PFOS ↑64% odds for miscarriage with PFAS	
PFOS, PFHxS	Cohort	Women from the Ronneby cohort	3000	Serum levels of PFAS were analyzed using LC/MS/MS. High coverage microRNA expression was analyzed by next generation sequencing and verified by qPCR	Serum sample	Associated with downregulation of specific microRNAs with cardiovascular function and disease, Alzheimer's disease, growth of cancer cell lines and cancer.	Xu et al. 2020.
PFNA	Longitudinal analyses	All individuals employed at a polymer production facility between1 January 1989 and 1 July 2003	630	Liver enzyme function and blood lipids. Detailed work histories, available for determining exposure category.	Liver enzyme function and blood lipids	Differences by exposure group for all laboratory measures, adjusted for age and body mass index, were small and not clinically significant.	Mundt et al. 2007.
PFOA	Nested case-control studies with data from 1979-2004	on 67,294 male and 19,404 female workers in DuPont plants in the Appalachian region	5,791	Exposure-response analyses using Standard Mortality Rates were conducted with cumulative serum levels, in terms of ppm-years.	Serum sample	Positive exposure-response trends for malignant and nonmalignant renal disease. Kidney cancer contributed to multiple-cause mortality ↑Cause-specific mortality at 350 ng/mL PFOA for mesothelioma, diabetes mellitus, and chronic renal disease.	Steenland et al. 2012.

						Significant positive exposure-response trends occurred for both malignant and nonmalignant renal disease. No trend for diabetes or heart disease mortality.	
PFOA	Cohort study	This cohort included employees of a 3M Company plant where APFO production began in 1947	3993	Comprehensive biologic monitoring data were not available for this cohort. Estimates of exposure intensity were limited to a qualitative assessment in the form of relative exposure weights assigned to the job exposure matrix.	Serum sample PFOA estimated	Ammonium perfluorooctanoate exposure was not associated with liver, pancreatic, and testicular cancer or cirrhosis of the liver. Exposure was associated with prostate cancer, cerebrovascular disease, and diabetes.	Lundin et al. 2009.
PFOA, PFOS	Cross-sectional	Individuals exposed to background levels of PFOS and elevated concentrations of PFOA through drinking water	290	RNA was extracted from whole blood samples using the PAX gene Blood mRNA kit.	Serum sample	Positive associations between gene expression involved in cholesterol metabolism and exposure to PFOA or PFOS. PFOA and PFOS may promote a hyper-cholesterol emic environment, with wider implications for human disease.	Fletcher et al. 2013.
PFOA, PFNA, PFDA, PFHxS	Prospective birth cohort study from 2012-2015	Pregnant women	1056	Prenatal information was collected by an interview with the women and from medical records.	Blood plasma sample	Atopic dermatitis (AD) was diagnosed in 173 (25.2%) children during the first 24 months. A log-unit increase in PFOA was associated	Chen et al. 2018.

				Fetal umbilical cord blood was collected at birth, children followed at 6, 12 and 24 months		with a 2.1-fold increase in AD risk in female children after adjusting for potential confounders. The highest quartile of PFNA, PFDA and PFHxS were associated with AD No significant associations were found in male children.	
PFHxS, PFOS, PFOA, PFNA, PFDA	Cohort of Faroese children whose mothers were recruited during pregnancy	Children	559	Serum concentrations of five PFAS were measured at three time points and determined their association with immunoglobulin E (IgE)	Serum sample	Among 22 MMR-unvaccinated children, higher levels of the five PFAS at age 5 years were associated with increased odds of asthma at ages 5 and 13. Pre-natal PFAS exposure was not associated with childhood asthma or allergic diseases	Timmermann et al. 2017.
PFOA, PFNA, PFOS, PFHxS	Cross-sectional	healthy adults	78	Measured anti-A H1N1 antibody response and cytokine and chemokine concentrations in serum pre-vaccination, 3 days postvaccination, and 30 days post-vaccination	Serum sample	No observed readily discernable or consistent pattern between PFAS concentration and baseline cytokine, chemokine, or mucosal IgA concentration, or between PFAS concentration and change in these immune markers between baseline and FluMist response states.	Stein et al. 2016.
PFOA	Cohort	U.S. adults	32,254	Autoimmune disease via self-reported PFOA cumulative exposure	Serum sample	Increased incidence of ulcerative colitis was associated with PFOA exposure.	Steenland et al. 2013.

PFOA	Cross-sectional	White residents who were plaintiffs or potential plaintiffs in a lawsuit	566	Questionnaire	Self-reported results	Exposed subjects with statistically significant greater prevalence of angina, myocardial infarction, and stroke, chronic bronchitis, shortness of breath on stairs, and/or asthma	Anderson-Mahoney et al. 2008.
PFCs	Case–control	Asthmatic children and non-asthmatic controls from Taiwan	231 asthmatic children and 225 non-asthmatic controls	Serum concentrations of PFCs and levels of immunological markers were measured. Associations of PFC quartiles with concentrations of immunological markers and asthma outcomes were estimated using multivariable regression models.	Serum sample, immunological markers	PFOS, PFOA, and subsets of the other PFCs were positively associated with serum IgE concentrations, absolute eosinophil counts, eosinophilic cationic protein concentrations, and asthma severity scores among asthmatics.	Dong et al. 2013.
PFOA, PFNA, PFHxS, PFOS	Cross-sectional	Children aged 12 – 19 in the Food allergies in adolescent participants (ages 12–19 years)	1,338	Performed multivariate logistic regression to analyze the association between individual PFASs with food sensitization in NHANES 2005–2006 and food allergies in NHANES 2007–2010.	Serum sample	Serum PFOA, PFOS, and PFHxS were statistically significantly associated with higher odds to have self-reported food allergies in NHANES 2007– 2010. When using IgE levels as a marker of food sensitization, serum PFNA was inversely associated with food sensitization.	Buser et al. 2016.
PFOA, PFOS,	Cohort	Participants 12–19 years of age from the 1999–	1,877	Sera were analyzed for 12 PFCs among a subsample of	Serum sample	PFOA was associated with higher odds of ever having received a diagnosis of	Humblet et al. 2014.

PFNA, PFHxS		2000 and 2003–2008 NHANES		participants ≥ 12 years of age from NHANES 1999–2000 and 2003–2008.		asthma whereas for PFOS there were inverse relationships with both asthma and wheezing. No associations were seen between the other PFCs and any outcome.	
PFHxS, PFHpA, PFOS, PFOA, PFNA, PFDA, PFUnDA, PFDoDA	Cohort	Self-reported healthy volunteers.	12	Serum-PFAS concentrations were measured and their antibody responses prospectively followed for 30 days after a booster vaccination with diphtheria and tetanus	Serum sample	Serum-PFAS concentrations were positively correlated, and positively associated, with age and male sex. The specific antibody concentrations in serum were increased from Day 4 to Day 10 post-booster, after which a constant concentration was reached. Serum PFAS concentrations showed significant negative associations with the rate of increase in the antibody responses.	Kielsen et al. 2016.
PFOS, PFHxS, PFOA, PFNA	Cross-sectional	Children participants	1,831	Examined PFAS serum concentration in relation to measles, mumps, and rubella antibody concentrations	Serum sample	Observed no adverse association between exposure and current allergic conditions, including asthma.	Stein et al. 2016.
PFOA, PFNA, PFHxS, PFOS	Prospective birth-cohort up to 3 years of age	Birth-cohort BraMat, a sub-cohort of the Norwegian Mother and Child Cohort	99	Blood samples were collected from the mothers at the time of delivery and from the children at the age of 3 years.	Serum sample	There was an inverse association between the level of anti-rubella antibodies in the children's serum at age 3 years and the concentrations of the	Granum et al. 2013.

		Study (MoBa), pregnant women from Oslo and Akershus, Norway, were recruited during 2007–2008				four PFAS. There was a positive association between the maternal concentrations of PFOA and PFNA and the number of episodes of common cold for the children, and between PFOA and PFHxS and the number of episodes of gastroenteritis. No associations were found between maternal PFAS concentrations and the allergy- and asthma-related health outcomes investigated.	
PFOS, PFOA, PFHxS	Cohort	Children from births during 1997–2000	464	PFAS concentrations Antibodies against diphtheria and tetanus	Serum sample	Concentrations of all three 7-year PFAS concentrations were individually associated with a decrease in concentrations of antibodies. The three 7-year concentrations were combined and showed that a 2-fold increase in PFAS was associated with a decrease by 54.4 % in the antibody concentration.	Mogensen et al. 2015.
PFOA, PFHxS, PFNA, PFDA, PFOS	Prospective study of a birth cohort	Children aged 5 and 7 years.	656, 587 followed-up	Serum antibody concentrations against tetanus and diphtheria	Serum sample	PFOS and PFOA showed the strongest negative correlations with antibody concentrations at age 5 years. A 2-fold increase in PFOS and PFOA concentrations	Grandjean et al. 2012.

						at age 5 years was associated with odds ratios between 2.38 and 4.20.	
PFOA	Cross-sectional	Adults	7,869 OH cases and 17,238 WV cases	Higher PFOA serum levels may be associated with testicular, kidney, prostate, and ovarian cancers and non-Hodgkin lymphoma.	Serum sample and historical records	Positive association between kidney cancer and the very high and high serum exposure categories [AOR = 2.0 (95% CI: 1.0, 3.9) n = 9 and 2.0 (95% CI: 1.3, 3.2) n = 22, respectively]	Vieira et al. 2013.
PFOA	Cohort	Workers	5791	Serum PFOA level Renal disease	Serum sample	Significant positive exposure (PFOA at 350 ng/mL)-response trends occurred for both malignant and nonmalignant renal disease. No exposure-response trend was seen for diabetes or heart disease mortality.	Steenland and Woskie. 2012.
PFOA	Cohort	Adults	2,507 cancer cases/3225 4	Cumulative serum PFOS estimated\ Cancer cases	Serum	PFOA exposure was associated with kidney and testicular cancers. Positive trends with increasing exposures for both cancers.	Barry et al. 2013.
PFHxS PFOS PFDS PFPeA PFHxA PFHpA PFOA PFNA PFDA PFUnD	Cross-sectional	Patients who had undergone liver transplantation for a range of conditions	Serum (n = 79) and liver (n = 66) samples	PFOS, PFHxS, PFOA, and PFNA concentrations were lower than those previously reported from Australia for 2002–2003, and 2006–2007	Serum samples Liver samples	All samples showed detectable PFOS (serum:0.621–126 ng/mL; liver: 0.375–42.5 ng/g wet wt) and PFOA (serum: 0.437–45.5 ng/mL; liver: 0.101–2.25 ng/g wet wt) concentrations.	Yeung et al. 2013.

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PFHxS, PFOS, PFOA, PFNA, PFDA, PFUnDA	Case-control	Prostate cancer cases	201 cases/186 control	A higher risk for prostate cancer in cases with heredity as a risk factor.	Serum Estimated PFAS	The analyzed PFAAs yielded statistically significant higher ORs in cases with a first-degree relative reporting prostate cancer.	Hardell et al. 2014.
PFOA	Meta-analysis of 24 studies	Births in total across all studies	19,094	log-transformed PFOA.	Serum sample Urine Birthweight	A change of birthweight of −10.5g (−16.7, −4.4) for every ng/ml PFOA in maternal or cord blood. Also found little evidence of an association between PFOA and birthweight	Steenland et al. 2018.
PFOA	Cohort	Community participants ≥ 20 years of age. Worker recruited from an occupational cohort, formed for previous mortality studies.	Community = 28,541 Work = 3,713 Total = 32,254	Participants completed surveys during 2008–2011. Retrospective serum PFOA concentration estimates, controlling for sex, race, education, smoking, alcohol use, body mass index, and diabetes.	Self-reported results	Hypercholesterolemia incidence increased with increasing cumulative PFOA exposure, most notably among males 40–60 years of age. There was no apparent association between PFOA exposure and hypertension or coronary artery disease incidence.	Winquist and Steenland. 2014.
PFOA, PFOS, PFNA, PFHxS	Cross-sectional	Children participants living near the DuPont plant in West Virginia	9,660	Measured serum creatinine, PFOA, PFOS, PFNA, and PFHxS, and calculated eGFR.	Serum	An interquartile range of serum PFOA concentrations was associated with a decrease in eGFR of 0.75 mL/min/1.73 m^2. Measured serum levels of PFOS, PFNA, and PFHxS were also cross-sectionally	Watkins et al. 2013.

						associated with decreased eGFR. Predicted serum PFOA levels at birth and during the first ten years of life were not related to eGFR.	
PFOA	Cohort	Workers or next of kin belonging to an occupational-exposure cohort	3,713	Estimated lifetime PFOA serum levels via a job-exposure matrix based on over 2000 serum measurements. Non-occupational exposure from drinking water was also estimated.	Self-reported results	Ulcerative colitis and rheumatoid arthritis showed a significant trend with increasing PFOA dose. Positive non-significant trends were observed for prostate cancer, non-hepatitis liver disease and male hypothyroidism. A significant negative trend was found for bladder cancer and asthma with medication.	Steenland et al. 2015.
PFOA, PFOS	Cross-sectional	Community residents aged 18 years or above, who drank water contaminated with PFOA from a chemical plant in West Virginia	46,294	Serum samples were extracted from the participates and analyzed to determine lipid profile. The mean levels of serum PFOA and PFOS in 2005–2006 were 80 ng/mL and 22 ng/mL respectively.	Serum	All lipid outcomes except high density lipoprotein cholesterol showed significant increasing trends; high density lipoprotein cholesterol showed no association. The odds ratios for high cholesterol by increasing quartile of PFOA, were 1.00, 1.21, 1.33, and 1.40 and were similar for PFOS quartiles.	Steenland et al. 2009.
PFOA	Cohort	Pregnancies of participants in	11,737	Serum PFOA was measured and reproductive and	Serum	No association between PFOA and miscarriage, preterm birth, term low	Savitz et al. 2012.

		the C8 Health Project		residential histories were obtained.		birthweight, and birth defects.	
PFOS, PFOA, PFHxS, PFNA, PFHpS, PFDA	Cohort	Pregnancies from the Danish National Birth Cohort (DNBC)	3,535	Maternal plasma concentrations were measured for six types of PFASs in early pregnancy.	Plasma	Several PFAS were associated with a reduction in birth weight and gestational age. A nearly 2-fold increase in risks of preterm birth for the higher quartiles of PFOA and PFOS exposure. Risk of preterm birth was increased for PFNA, PFHpS, and PFDA in higher exposure ranges.	Meng et al. 2018.
PFOS, PFOA, PFHxS, PFHpS, PFNA, PFDA, PFOSA	Cohort	Pregnancies from the Danish National Birth Cohort (DNBC) during 1996 - 2002	1,592	Child IQ was assessed at age 5 years using the Wechsler Primary and Preschool Scales of Intelligence–Revised.	IQ level Serum	PFOS and PFOA were detected in all samples, and five additional PFAS were quantified in >80% of the samples. Found no strong associations between a natural-log unit increase in each of the seven PFASs we evaluated and child IQ scores. A few positive and negative associations were found in the sex-stratified PFAS quartile analyses, but the patterns were inconsistent.	Liew et al. 2018.
PFOA, PFOS, PFNA, PFHxS	Cross-sectional	Participants aged 12–19 years of the 2003–2010 National Health and Nutrition	1,960	Study outcomes were estimated glomerular filtration rate (eGFR) and serum uric acid	Serum	Adolescents in the highest PFOA and PFOS quartile had a lower eGFR, 6.84 mL/min/1.73 m^2 and 9.69 mL/min/1.73 m^2 respectively, compared to the lowest	Kataria et al. 2015.

		Examination Surveys				quartile. Highest PFOA and PFOS quartiles were also associated with 0.21 mg/dL and 0.19 mg/dL increases in uric acid, respectively.	
PFOA, PFOS	Case-control	Newly pregnant women residing in the Municipality of Odense, Denmark	2,874	Serum samples were taken at time of recruitment and stored.	Serum	Women with the highest fertile of exposure to PFNA and PFDA in pregnancy had odds ratios for miscarriage of 16.5 and 2.67, respectively, as compared to the lowest fertile. The association with PFHxS was in the same direction, but not statistically significant, while no association was found with PFOA and PFOS.	Jensen et al. 2015.
PFOS, PFOA, PFNA, PFHxS, PFBS, PFDA, PFUA	Cross-sectional	Umbilical cord collected between 2011 and 2012 in Shanghai, China	687	Plasma samples were collected from the umbilical cords. Chronic hypertension, gestational hypertension, and preeclampsia was obtained from medical records.	Plasma Medical records	PFBS, PFHxS, and PFUA were associated with gestational hypertension and preeclampsia.Women with a higher level of standardized ln-transformed PFBS had an increased odds of preeclampsia and overall HDP.	Huang et al. 2019.
PFOS, PFOA, PFNA, PFHxS	Cross-sectional	Children ages 12-15 that were part of the National Health and Nutrition Examination Survey in	571	Parental report of a previous diagnosis by a doctor or health care professional of ADHD in the child.	Serum sample; self-reported results	48 of 571 children included in the analysis had been diagnosed with ADHD. The adjusted odds ratio for parentally reported ADHD in association with a	Hoffman et al. 2010.

		1999–2000 and 2003–2004.		PFAS levels in serum samples from each child.		1-µg/L increase in serum PFOS was 1.03. Observed a nonsignificant positive association with PFNA.	
PFOS, PFOA, PFHxS	Cohort	Mother-child pairs from Project Viva, a longitudinal Boston-area birth cohort enrolled during 1999–2002	1,668	Plasma collected from women during pregnancy (median 9.7 weeks gestation) and from children at a visit in mid-childhood (median age 7.7 years)	Plasma sample	Prenatal PFAS concentrations were associated with both better and worse cognitive Performance. Children with top quartile prenatal concentrations of some PFASs had better visual motor abilities in early childhood and non-verbal IQ and visual memory in mid-childhood, while children with upper quartile prenatal PFOA and PFOS had lower mid-childhood visual-motor scores. Visual-motor scores on the Wide Range Assessment of Visual Motor Abilities were lower among children with higher PFHxS. Upper quartiles of childhood PFOA and PFOS were also associated with somewhat lower childhood scores.	Harris et al. 2018.
PFOA, PFOS	Cross-sectional	Children from the C8 Health Project	12,476	Serum lipids were collected to determine the following: total, HDL, and LDL	Serum sample	Mean serum PFOA and PFOS concentrations were 69.2±111.9 ng/mL and 22.7±12.6 ng/mL. PFOA was significantly associated	Frisbee et al. 2010.

				cholesterol, and fasting Triglycerides.		with increased total and LDL cholesterol, and PFOS was significantly associated with increased total, HDL, and LDL cholesterol. Observed effects were non-linear, with larger increases in total and LDL cholesterol occurring the lowest range of particularly PFOA.	
PFOA, PFOS	Cohort	Birth outcomes from 2005 through 2010 in a Mid-Ohio Valley community	1,630	Women provided serum for PFOA and PFOS measurement in 2005–2006 and reported reproductive histories in subsequent follow-up interviews.	Serum sample; self-reported results	Little or no evidence of association between maternal serum PFOA or PFOS and preterm birth or low birth weight. Serum PFOA and PFOS were both positively associated with pregnancy-induced hypertension, with adjusted odds ratios per log unit increase in PFOA and PFOS of 1.27 and 1.47, respectively.	Darrow et al. 2013.
PFOA, PFOS	Cohort	Women in 2005–2006 who reported pregnancy outcome in a population of the mid-Ohio valley	1,129	Serum PFOA and PFOS concentrations were collected from women; Preconception serum levels were analyzed in relation to miscarriage using logistic regression and generalized estimating equations.	Serum sample	Little evidence of association between PFOA and miscarriage. For PFOS, the odds ratio of miscarriage per log ng/ml increase was 1.21 in sub analyses restricted to each woman's first pregnancy conceived, and after the serum measurement, the odds ratio was 1.34. Elevated odds ratios for the	Darrow et al. 2014.

						top 4 quintiles relative to the first quintile, without a monotonic trend. Positive associations between PFOS and miscarriage were strongest among nulligravid pregnancies.	
PFOA	Cohort	Participants were recruited from the C8 Health Project who were 20 years or older.	30,723	Participants completed surveys reporting demographic, medical, and residential history information. Alanine aminotransferase (ALT), γ-glutamyltransferase (GGT) and direct bilirubin, markers of liver toxicity, were obtained from blood samples.	Serum sample; self-reported results	Modeled cumulative serum PFOA was positively associated with ALT levels indicating possible liver toxicity. An increase from the first to the fifth quintile of cumulative PFOA exposure was associated with a 6% increase in ALT levels and 16% increased odds of having above-normal ALT. PFOA was associated with decreased direct bilirubin. Observed no evidence of an effect of cumulative exposure on all liver disease, nor on enlarged liver, fatty liver, and cirrhosis only .	Darrow et al. 2016.

Table 7 Occurrence of Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water Treated by Drinking Water Utility System

Small Systems – Surface Water (n = 1,199)								
			Concentration (ng/L)					
PFAS	***# Detects***	***Det.Freq.(%)***	***Min. Reporting Level***	***Mean***	***SD***	***Median***	***P90***	***Maximum***
PFBS	0	0	90	--	--	--	--	--
PFHxS	0	0	30	--	--	--	--	--
PFHpA	0	0	10	--	--	--	--	--
PFOA	0	0	20	--	--	--	--	--
PFOS	2	0.17	40	54	6	54	58	59
PFNA	0	0	20	--	--	--	--	--
Large Systems – Surface Water (n = 13,279)								
			Concentration (ng/L)					
PFAS	***# Detects***	***Det.Freq.(%)***	***Min. Reporting Level***	***Mean***	***SD***	***Median***	***P90***	***Maximum***
PFBS	12	0.09	90	212	94	185	357	370
PFHxS	28	0.21	30	69	35	62	79	190
PFHpA	92	0.69	10	19	11	15	39	60
PFOA	101	0.76	20	31	13	29	41	100
PFOS	66	0.50	40	77	56	57	140	400
PFNA	1	0.01	20	54	--	54	54	54

Small Systems – Groundwater (n = 2,075)								
			Concentration (ng/L)					
PFAS	***# Detects***	***Det.Freq.(%)***	***Min. Reporting Level***	***Mean***	***SD***	***Median***	***P90***	***Maximum***
PFBS	0	0	90	--	--	--	--	
PFHxS	4	0.19	30	409	348	404	730	
PFHpA	4	0.19	10	41	35	33	87	
PFOA	4	0.19	20	100	85	81	206	
PFOS	4	0.19	40	158	127	142	300	
PFNA	1	0.05	20	26	--	26	26	
Large Systems – Groundwater (n = 20,419)								
			Concentration (ng/L)					
PFAS	***# Detects***	***Det.Freq.(%)***	***Min. Reporting Level***	***Mean***	***SD***	***Median***	***P90***	***Maximum***
PFBS	7	0.03	90	136	49	110	196	220
PFHxS	175	0.86	30	144	174	79	330	1600
PFHpA	140	0.69	10	28	43	20	53	410
PFOA	274	1.34	20	45	46	30	74	349
PFOS	220	1.08	40	200	603	64	383	7000
PFNA	17	0.08	20	35	11	32	52	56

Source: *Occurrence of Per- and Polyfluoroalkyl Substances (PFAS) in Source Water and Their Treatment in*

Drinking Water by Crone et al, 2020. Table is titled "UCMR3 PFAS Concentrations by Drinking Water Utility System Size and Source Water Type."

Table 8 PFAS Concentrations in Human Serum

PFAS	HUMAN	AGE	GENDER	SAMPLE	CONCENTRATION	REFERENCE
PFOS	NHANES 2003–2004	12 – 19 years	Male/female	Serum	Geometric mean (µg/L): 20.7 (19.2 – 22.3)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 2003–2004	20 – 39 years	Male/female	Serum	Geometric mean (µg/L): 19.3 (17.5 – 21.4)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 2003–2004	40 – 59 years	Male/female	Serum	Geometric mean (µg/L): 18.7 (17.3 – 20.1)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 2003–2004	≥ 60 years	Male/female	Serum	Geometric mean (µg/L): 23.2 (20.8 – 25.9)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 2003–2004	Mexican American	Male/female	Serum	Geometric mean (µg/L): 14.7 (13.0 – 16.6)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 2003–2004	Non-Hispanic black	Male/female	Serum	Geometric mean (µg/L): 21.6 (19.1 – 24.4)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 2003–2004	Non-Hispanic white	Male/female	Serum	Geometric mean (µg/L): 21.4 (19.9 – 23.1)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 2003–2004	Female	Female	Serum	Geometric mean (µg/L): 18.4 (17.0 – 20.0)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 2003–2004	Male	Male	Serum	Geometric mean (µg/L): 23.3 (21.1 – 25.6)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 2003–2004	12 – 19 years	Male/female	Serum	Geometric mean (µg/L): 3.9 (3.5 – 4.4)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 2003–2004	20 – 39 years	Male/female	Serum	Geometric mean (µg/L): 3.9 (3.6 – 4.2)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 2003–2004	40 – 59 years	Male/female	Serum	Geometric mean (µg/L): 4.2 (3.8 – 4.8)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 2003–2004	≥ 60 years	Male/female	Serum	Geometric mean (µg/L): 3.7 (3.3 – 4.1)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 2003–2004	Mexican American	Male/female	Serum	Geometric mean (µg/L): 3.1 (2.8 – 3.4)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 2003–2004	Non-Hispanic black	Male/female	Serum	Geometric mean (µg/L): 3.4 (3.0 – 3.8)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 2003–2004	Non-Hispanic white	Male/female	Serum	Geometric mean (µg/L): 4.2 (3.9 – 4.5)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 2003–2004	Adults	Female	Serum	Geometric mean (µg/L): 3.5 (3.2 – 3.8)	Calafat et al. (2007a).; Calafat et al. (2007b).

PFOA	NHANES 2003–2004	Adults	Male	Serum	Geometric mean (µg/L): 4.5 (4.1 – 4.9)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 2003–2004	12 – 19 years	Male/female	Serum	Geometric mean (µg/L): 2.4 (2.1 – 2.9)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 2003–2004	20 – 39 years	Male/female	Serum	Geometric mean (µg/L): 1.8 (1.6 – 2.0)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 2003–2004	40 – 59 years	Male/female	Serum	Geometric mean (µg/L): 1.9 (1.6 – 2.2)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 2003–2004	≥ 60 years	Male/female	Serum	Geometric mean (µg/L): 2.0 (1.7 – 2.4)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 2003–2004	Mexican American	Male/female	Serum	Geometric mean (µg/L): 1.4 (1.2 – 1.7)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 2003–2004	Non-Hispanic black	Male and female	Serum	Geometric mean (µg/L): 1.9 (1.6 – 2.3)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 2003–2004	Non-Hispanic white	Male/female	Serum	Geometric mean (µg/L): 2.0 (1.8 – 2.3)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 2003–2004	Adults	Female	Serum	Geometric mean (µg/L): 1.7 (1.6 – 1.9)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 2003–2004	Adults	Male	Serum	Geometric mean (µg/L): 2.2 (1.9 – 2.5)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 2003–2004	12 – 19 years	Male/female	Serum	Geometric mean (µg/L): 0.9 (0.7 – 1.0)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 2003–2004	20 – 39 years	Male/female	Serum	Geometric mean (µg/L): 1.0 (0.8 – 1.1)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 2003–2004	40 – 59 years	Male/female	Serum	Geometric mean (µg/L): 1.1 (0.9 – 1.4)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 2003–2004	≥ 60 years	Male/female	Serum	Geometric mean (µg/L): 0.8 (0.7 – 1.0)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 2003–2004	Mexican American	Male/female	Serum	Geometric mean (µg/L): 0.7 (0.6 – 0.8)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 2003–2004	Non-Hispanic black	Male/female	Serum	Geometric mean (µg/L): 1.1 (0.8 – 1.5)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 2003–2004	Non-Hispanic white	Male/female	Serum	Geometric mean (µg/L): 1.0 (0.8 – 1.1)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 2003–2004	Adults	Female	Serum	Geometric mean (µg/L): 0.9 (0.7 – 1.0)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 2003–2004	Adults	Male	Serum	Geometric mean (µg/L): 1.1 (0.9 – 1.3)	Calafat et al. (2007a).; Calafat et al. (2007b).

PFOS	NHANES 1999 - 2000	12 – 19 years	Male/female	Serum	Geometric mean (µg/L): 29.1 (26.2 – 32.4)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 1999 - 2000	20 – 39 years	Male/female	Serum	Geometric mean (µg/L): 27.5 (24.9 – 30.2)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 1999 - 2000	40 – 59 years	Male/female	Serum	Geometric mean (µg/L): 33.0 (28.0 – 38.8)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 1999 - 2000	≥ 60 years	Male/female	Serum	Geometric mean (µg/L): 33.3 (28.5 – 38.8)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 1999 - 2000	Mexican American	Male/female	Serum	Geometric mean (µg/L): 22.7 (19.8 – 25.9)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 1999 - 2000	Non-Hispanic black	Male/female	Serum	Geometric mean (µg/L): 33.0 (26.2 – 41.6)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 1999 - 2000	Non-Hispanic white	Male/female	Serum	Geometric mean (µg/L): 32.0 (29.1 – 35.2)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 1999 - 2000	Adults	Female	Serum	Geometric mean (µg/L): 28.0 (24.6 – 31.8)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOS	NHANES 1999 - 2000	Adults	Male	Serum	Geometric mean (µg/L): 33.4 (29.6 – 37.6)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 1999 - 2000	12 – 19 years	Male/female	Serum	Geometric mean (µg/L): 5.5 (5.0 – 6.0)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 1999 - 2000	20 – 39 years	Male/female	Serum	Geometric mean (µg/L): 5.2 (4.7 – 5.7)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 1999 - 2000	40 – 59 years	Male/female	Serum	Geometric mean (µg/L): 5.4 (4.7 – 6.2)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 1999 - 2000	≥ 60 years	Male/female	Serum	Geometric mean (µg/L): 4.8 (4.3 – 5.5)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 1999 - 2000	Mexican American	Male/female	Serum	Geometric mean (µg/L): 3.9 (3.6 – 4.2)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 1999 - 2000	Non-Hispanic black	Male/female	Serum	Geometric mean (µg/L): 4.8 (4.1 – 5.6)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 1999 - 2000	Non-Hispanic white	Male/female	Serum	Geometric mean (µg/L): 5.6 (5.0 – 6.2)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 1999 - 2000	Adults	Female	Serum	Geometric mean (µg/L): 4.8 (4.3 – 5.3)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOA	NHANES 1999 - 2000	Adults	Male	Serum	Geometric mean (µg/L): 5.7 (5.2 – 6.3)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 1999 - 2000	12 – 19 years	Male/female	Serum	Geometric mean (µg/L): 2.7 (2.1 – 3.4)	Calafat et al. (2007a).; Calafat et al. (2007b).

PFHxS	NHANES 1999 - 2000	20 – 39 years	Male/female	Serum	Geometric mean (µg/L): 2.0 (1.7 – 2.3)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 1999 - 2000	40 – 59 years	Male/female	Serum	Geometric mean (µg/L): 2.1 (1.8 – 2.3)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 1999 - 2000	≥ 60 years	Male/female	Serum	Geometric mean (µg/L): 2.2 (1.9 – 2.5)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 1999 - 2000	Mexican American	Male/female	Serum	Geometric mean (µg/L): 1.5 (1.1 – 1.9)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 1999 - 2000	Non-Hispanic black	Male/female	Serum	Geometric mean (µg/L): 2.2 (1.6 – 2.9)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 1999 - 2000	Non-Hispanic white	Male/female	Serum	Geometric mean (µg/L): 2.3 (2.0 – 2.5)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 1999 - 2000	Adults	Female	Serum	Geometric mean (µg/L): 1.8 (1.6 – 2.1)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFHxS	NHANES 1999 - 2000	Adults	Male	Serum	Geometric mean (µg/L): 2.6 (2.3 – 3.0)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 1999 - 2000	12 – 19 years	Male/female	Serum	Geometric mean (µg/L): 0.5 (0.4 – 0.5)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 1999 - 2000	20 – 39 years	Male/female	Serum	Geometric mean (µg/L): 0.5 (0.4 – 0.6)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 1999 - 2000	40 – 59 years	Male/female	Serum	Geometric mean (µg/L): 0.6 (0.4 – 0.7)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 1999 - 2000	≥ 60 years	Male/female	Serum	Geometric mean (µg/L): 0.6 (0.5 – 0.8)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 1999 - 2000	Mexican American	Male/female	Serum	Geometric mean (µg/L): 0.3 (0.3 – 0.4)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 1999 - 2000	Non-Hispanic black	Male/female	Serum	Geometric mean (µg/L): 0.8 (0.6 – 1.0)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 1999 - 2000	Non-Hispanic white	Male/female	Serum	Geometric mean (µg/L): 0.6 (0.5 – 0.7)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 1999 - 2000	Adults	Female	Serum	Geometric mean (µg/L): 0.5 (0.4 – 0.6)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFNA	NHANES 1999 - 2000	Adults	Male	Serum	Geometric mean (µg/L): 0.6 (0.5 – 0.7)	Calafat et al. (2007a).; Calafat et al. (2007b).
PFOSA	NHANES 1999 - 2000	12 – 19 years	Male/female	Serum	Geometric mean (µg/L): 0.4 (0.4 – 0.5)	Calafat et al. (2007b).
PFOSA	NHANES 1999 - 2000	20 – 39 years	Male/female	Serum	Geometric mean (µg/L): 0.3 (0.3 – 0.4)	Calafat et al. (2007b).

PFOSA	NHANES 1999 - 2000	40 – 59 years	Male/female	Serum	Geometric mean (µg/L): 0.4 (0.3 – 0.5)	Calafat et al. (2007b).
PFOSA	NHANES 1999 - 2000	≥ 60 years	Male/female	Serum	Geometric mean (µg/L): 0.3 (0.3 – 0.4)	Calafat et al. (2007b).
PFOSA	NHANES 1999 - 2000	Mexican American	Male/female	Serum	Geometric mean (µg/L): 0.3 (0.3 – 0.3)	Calafat et al. (2007b).
PFOSA	NHANES 1999 - 2000	Non-Hispanic black	Male/female	Serum	Geometric mean (µg/L): 0.4 (0.3 – 0.4)	Calafat et al. (2007b).
PFOSA	NHANES 1999 - 2000	Non-Hispanic white	Male/female	Serum	Geometric mean (µg/L): 0.4 (0.3 – 0.5)	Calafat et al. (2007b).
PFOSA	NHANES 1999 - 2000	Adults	Female	Serum	Geometric mean (µg/L): 0.4 (0.3 – 0.4)	Calafat et al. (2007b).
PFOSA	NHANES 1999 - 2000	Adults	Male	Serum	Geometric mean (µg/L): 0.4 (0.3 – 0.4)	Calafat et al. (2007b).
PFHxS	American Red Cross Blood Donors 2000-01	Adults	Male/female	Serum	Geometric mean (ng/mL): 2.25 (2.13 – 2.38)	Olsen et al. (2012).
PFHxS	American Red Cross Blood Donors 2000-01	Adults	Male	Serum	Geometric mean (ng/mL): 2.52 (2.33 – 2.72)	Olsen et al. (2012).
PFHxS	American Red Cross Blood Donors 2000-01	Adults	Female	Serum	Geometric mean (ng/mL): 2.01 (1.86 – 2.13)	Olsen et al. (2012).
PFHxS	American Red Cross Blood Donors 2006	Adults	Male/female	Serum	Geometric mean (ng/mL): 1.52 (1.43 – 1.62)	Olsen et al. (2012).
PFHxS	American Red Cross Blood Donors 2006	Adults	Male	Serum	Geometric mean (ng/mL): 1.94 (1.78 – 2.11)	Olsen et al. (2012).
PFHxS	American Red Cross Blood Donors 2006	Adults	Female	Serum	Geometric mean (ng/mL): 1.19 (1.10 – 1.29)	Olsen et al. (2012).
PFHxS	American Red Cross Blood Donors 2010	Adults	Male/female	Serum	Geometric mean (ng/mL): 1.34 (1.25 – 1.42)	Olsen et al. (2012).
PFHxS	American Red Cross Blood Donors 2010	Adults	Male	Serum	Geometric mean (ng/mL): 1.65 (1.51 – 1.80)	Olsen et al. (2012).
PFHxS	American Red Cross Blood Donors 2010	Adults	Female	Serum	Geometric mean (ng/mL): 1.08 (0.99 – 1.18)	Olsen et al. (2012).
PFOS	American Red Cross Blood Donors 2000-01	Adults	Male/female	Serum	Geometric mean (ng/mL): 34.9 (33.3 – 36.5)	Olsen et al. (2012).
PFOS	American Red Cross Blood Donors 2000-01	Adults	Male	Serum	Geometric mean (ng/mL): 37.8 (35.5 – 40.3)	Olsen et al. (2012).
PFOS	American Red Cross Blood Donors 2000-01	Adults	Female	Serum	Geometric mean (ng/mL): 32.1 (30.0 – 34.3)	Olsen et al. (2012).
PFOS	American Red Cross Blood Donors 2006	Adults	Male/female	Serum	Geometric mean (ng/mL): 14.5 (13.9 – 15.2)	Olsen et al. (2012).

PFOS	American Red Cross Blood Donors 2006	Adults	Male	Serum	Geometric mean (ng/mL): 17.1 (16.2 – 18.1)	Olsen et al. (2012).
PFOS	American Red Cross Blood Donors 2006	Adults	Female	Serum	Geometric mean (ng/mL): 12.3 (11.5 – 13.1)	Olsen et al. (2012).
PFOS	American Red Cross Blood Donors 2010	Adults	Male/female	Serum	Geometric mean (ng/mL): 8.3 (7.9 – 8.8)	Olsen et al. (2012).
PFOS	American Red Cross Blood Donors 2010	Adults	Male	Serum	Geometric mean (ng/mL): 9.7 (9.0 – 10.4)	Olsen et al. (2012).
PFOS	American Red Cross Blood Donors 2010	Adults	Female	Serum	Geometric mean (ng/mL): 7.2 (6.7 – 7.7)	Olsen et al. (2012).
PFOA	American Red Cross Blood Donors 2000-01	Adults	Male/female	Serum	Geometric mean (ng/mL): 4.70 (4.50 – 4.92)	Olsen et al. (2012).
PFOA	American Red Cross Blood Donors 2000-01	Adults	Male	Serum	Geometric mean (ng/mL): 5.02 (4.43 – 5.73)	Olsen et al. (2012).
PFOA	American Red Cross Blood Donors 2000-01	Adults	Female	Serum	Geometric mean (ng/mL): 4.39 (4.12 – 4.69)	Olsen et al. (2012).
PFOA	American Red Cross Blood Donors 2006	Adults	Male/female	Serum	Geometric mean (ng/mL): 3.44 (3.30 – 3.58)	Olsen et al. (2012).
PFOA	American Red Cross Blood Donors 2006	Adults	Male	Serum	Geometric mean (ng/mL): 3.95 (3.74 – 4.17)	Olsen et al. (2012).
PFOA	American Red Cross Blood Donors 2006	Adults	Female	Serum	Geometric mean (ng/mL): 3.00 (2.83 – 3.18)	Olsen et al. (2012).
PFOA	American Red Cross Blood Donors 2010	Adults	Male/female	Serum	Geometric mean (ng/mL): 2.44 (2.33 – 2.56)	Olsen et al. (2012).
PFOA	American Red Cross Blood Donors 2010	Adults	Male	Serum	Geometric mean (ng/mL): 2.69 (2.53 – 2.87)	Olsen et al. (2012).
PFOA	American Red Cross Blood Donors 2010	Adults	Female	Serum	Geometric mean (ng/mL): 2.22 (2.06 – 2.38)	Olsen et al. (2012).
PFNA	American Red Cross Blood Donors 2000-01	Adults	Male/female	Serum	Geometric mean (ng/mL): 0.57 (0.55 – 0.60)	Olsen et al. (2012).
PFNA	American Red Cross Blood Donors 2000-01	Adults	Male	Serum	Geometric mean (ng/mL): 0.62 (0.59 – 0.66)	Olsen et al. (2012).
PFNA	American Red Cross Blood Donors 2000-01	Adults	Female	Serum	Geometric mean (ng/mL): 0.53 (0.50 – 0.56)	Olsen et al. (2012).
PFNA	American Red Cross Blood Donors 2006	Adults	Male/female	Serum	Geometric mean (ng/mL): 0.97 (0.93 – 1.00)	Olsen et al. (2012).
PFNA	American Red Cross Blood Donors 2006	Adults	Male	Serum	Geometric mean (ng/mL): 1.04 (0.99 – 1.10)	Olsen et al. (2012).

PFNA	American Red Cross Blood Donors 2006	Adults	Female	Serum	Geometric mean (ng/mL): 0.90 (0.85 – 0.95)	Olsen et al. (2012).
PFNA	American Red Cross Blood Donors 2010	Adults	Male/female	Serum	Geometric mean (ng/mL): 0.83 (0.79 – 0.87)	Olsen et al. (2012).
PFNA	American Red Cross Blood Donors 2010	Adults	Male	Serum	Geometric mean (ng/mL): 0.86 (0.81 – 0.92)	Olsen et al. (2012).
PFNA	American Red Cross Blood Donors 2010	Adults	Female	Serum	Geometric mean (ng/mL): 0.79 (0.74 – 0.85)	Olsen et al. (2012).
PFOS	Carpet treated with Scotchgard	Adults/children	Male/female	Serum	15.2-108 ng/mL	Beesoon et al. (2012)
PFOA	Carpet treated with Scotchgard	Adults/children	Male/female	Serum	2.40-9.23 ng/mL	Beesoon et al. (2012)
PFOA	Municipal well community reported by CDC	Adults/children	Male/female	Serum	15.4 ng/mL	Landsteiner et al., 2014
PFOS	Municipal well community reported by CDC	Adults/children	Male/female	Serum	35.9 ng/mL	Landsteiner et al., 2014

Table 9 State and US Environmental Protection Agency guidelines for PFAS

	PFAS (ng/L)						
	PFOA	**PFOS**	**PFNA**	**PFHxS**	**PFHpA**	**PFDA**	**TOTAL**
No. of carbons	8	8	9	6	7	10	Yes (2)
USEPA	70	70	--	--	--	--	No
CA	10	40	--	--	--	--	Yes (5)
CT	70	70	70	70	70	--	Yes (6)
MA	20	20	20	20	20	20	No
MI	8	16	6	51	--	--	No
MN	35	15	--	47	--	--	No
NH	12	15	11	18	--	--	No
NJ	14	13	13	--	--	--	No
NY	10	10	--	--	--	--	No
NC	--	--	--	--	--	--	--
OH	70	70	21	140	--	--	Yes (2)
VT	20	20	20	20	20	--	Yes (5)
WA	10	15	14	70	--	--	--

Source: *Recent US State and Federal Drinking Water Guidelines for Per- and Polyfluoroalkyl Substances* by Gloria B. Post.

Table 10 Toxicological Basis of state and US Environmental Protection Agency Reference Doses for perfluorooctanoic acid

	NJ	NH	NY	MI	WA	MN	VT/USEPA	MA
Critical Effect	Increased liver weight			Developmental				
				Neurobehavioral; skeletal		Accelerated puberty (males); delayed ossification		
Species	Mouse							
Study	Loveless et al. (2006)		Macon et al. (2011)	Onishchenko et al. (2011) Koskela et al. (2016)		Lau et al. (2006)		
Serum PFOA metric	Measured			Modeled average				
Point of departure (nh/mL)	4350 (BMDL)		1060 (BMDL)	8290 (LOAEL)		38,000 (LOAEL)		
Uncertainty factor	10							
Intraspecies	10							
Interspecies	3							
Shorter-than-chronic	1							
LOAEL-to-NOAEL	1			3	10	3	10	
Database	10	3	3	3	1	3	1	3
Total	300	100	100	300	300	300	300	1000
Clearance factor	0.00014 L/kg/d (Lorber and Egeghy 2011)							
Reference Dose (ng/kg/d)	2	6.1	1.5	3.9	3	18	20	5

Source: *Recent US State and Federal Drinking Water Guidelines for Per- and Polyfluoroalkyl* Substances by Gloria B. Post.

Table 11 Toxicological Basis of State and US Environmental Protection Agency Reference Doses for Perfluorooctane Sulfonate

	MN/NH/WA	MI	NJ/NY	MA	VT/USEPA
Critical effect	Decreased antibody response to foreign antigen			Developmental-decreased offspring body weight	
Species	Mouse			Rat	
Study	Dong et al. (2011).	Dong et al. (2009).		Luebker et al. (2005).	
Serum PFOS metric	Measured			Modeled average	
Point of departure (ng/mL)	2620 (NOAEL)	674 (NOAEL)		6260 (NOAEL)	
Uncertainty factor	10				
Intraspecies	10				
Interspecies	3				
Shorter-than-chronic	1				
LOAEL-to-NOAEL	1				
Database	3	1		3	1
Total	100	30		100	30
Clearance factor	0.00013 L/kg/d (Human $t_{1/2}$ 3.4 yr.; Li et al. 2018)		0.000081 L/kg/d (Human $t_{1/2}$ 5.4 yr.; US Environmental Protection Agency 2016b)		
Reference Dose (ng/kg/d)	3	2.9	1.8 or 2	5	20

Source: *Recent US State and Federal Drinking Water Guidelines for Per- and Polyfluoroalkyl Substances* by Gloria B. Post.

Table 12 Massachusetts Guidelines for PFAS in Drinking Water

	MASSACHUSETTS	
	PFAS6 (PFOS, PFOA, PFHxS, PFNA, PFHpA, PFDA)	
	DECISION POINT	RATIONAL
CRITICAL STUDY	**PFOS** Luebker et al. (2005a) Sprague-Dawley rat Decreased F2 pup body weight.	[no information could be found]
	PFOA Lau et al. (2006) CD-1 mice Decreased pup ossification and accelerated male puberty.	[no information could be found]
	PFHxS [no information could be found]	[no information could be found]
	PFNA [no information could be found]	[no information could be found]
	PFHpA [no information could be found]	[no information could be found]
	PFDA [no information could be found]	[no information could be found]
POINT OF DEPARTURE (POD)	**PFOS** NOAEL range rats = 6–20 LOAEL range 25-38	Bayesian benchmark dose modeling (BBMD) was used to provide a consistent point of departure for comparison across the PFAS.
	PFOA NOAEL range 0.829–13; LOAEL range 8.29-39	
	PFHxS 27 (NOAEL)19 (no serum level reported at the LOAEL), 49 days mice (insufficient and limited database)	
	PFNA 9 (TWA NOAEL)17; 12 (LOAEL), 17 days, mice (insufficient and limited database)	
	PFHpA [no information could be found]	
	PFDA No in vivo animal data	
	PFOS	

HUMAN EQUIVALENT DOSE (HED)	Clearance factor = 0.000081 L/kg/d	Cl = Vd x (ln2/t1/2) Where: Cl = clearance (L/kg bw/day) Vd = volume of distribution in the human body (L/kg bw) ln2 = 0.693 t1/2 = half-life in humans (days) Human equivalent doses were estimated by adjusting the animal PFAS serum concentration10 by the human clearance rate estimated for each PFAS, applying the approach used by USEPA and ATSDR.
	PFOA Clearance factor = 0.00014 L/kg/d	
	PFHxS [no information could be found]	[no information could be found]
	PFNA [no information could be found]	[no information could be found]
	PFHpA [no information could be found]	[no information could be found]
	PFDA [no information could be found]	[no information could be found]
UNCERTAINTY FACTORS BROKEN DOWN	**PFOS** Total uncertainty factor of 100 10 for Intraspecies 3 for Interspecies 3 for database	This reflects substantial data indicating these compounds cause effects at lower doses than relied upon in the USEPA RfD derivations and provide a greater degree of health protection to sensitive groups. The conclusion is based on the following: RfDs for PFOA and PFOS lower than the USEPA (2016a,b) values are warranted to account for data from multiple studies (summarized in the Tables in this section and further assessed in Appendix 2, reporting effects in laboratory animals at dose levels below those used as POD in the USEPA RfD derivations. The weight of the evidence is compelling regarding potential effects at lower exposure levels. However,
	PFOA Total uncertainty factor of 1000 10 for Intraspecies 3 for Interspecies 10 for LOAEL-to-NOAEL 3 for database	
	PHHxS [no information could be found]	
	PFNA [no information could be found]	
	PFHpA [no information could be found]	
	PFDA	

	[no information could be found]	various issues relating to study design, execution and data interpretation have raised questions regarding the appropriateness of alternative PODs based on the lower dose effect data from the individual studies. These are discussed in Appendix 2. Thus, although lower PODs can be supported for PFOA and PFOS, MassDEP ORS has taken an alternative approach, which we conclude is preferable, to account for the lower dose effect data. This approach relies on the application of a database uncertainty factor (UFD) of 101/2 in the PFOA and PFOS RfD derivations. Application of an UFD is an approach that is consistent with well-established protocols used by federal and state agencies and has been used by several states, as well as ATSDR (2018a), for deriving PFAS toxicity values. Although MassDEP is adopting RfDs lower than those issued by USEPA for PFOA and PFOS in 2016, the data underlying the USEPA RfD values provide appropriate starting points for the MassDEP RfD because they were well documented and considered multiple studies and PODs.
TOXICITY VALUE	**PFOS** Reference dose = 5 ng/kg/d	MassDEP ORS concluded that the toxicity value (RfD) for compounds in this subgroup of longer-chain PFAS should be adjusted downward from that used in the 2018 ORSG derivation.
	PFOA Reference dose = 5 ng/kg/d	
	PFHxS [no information could be found]	
	PFNA [no information could be found]	
	PFHpA [no information could be found]	
	PFDA	

	[no information could be found]	
EXPOSURE PARAMETERS & DRINKING WATER HBVS	**PFOS, PFOA, PFHxS, PFNA, PFHpA, PFDA** The PFAS6 Maximum Contaminant Level (MCL) shall be 0.000020 milligrams per liter (mg/l) or 20 nanograms per liter (ng/l).	Consistent with the proposed groundwater (GW-1) clean up standard in the Massachusetts Contingency Plan, 310 CMR 40.0000 and technical information from MassDEP's Office of Research and Standards.
	PFOS, PFOA, PFHxS, PFNA, PFHpA, PFDA Effective June 12, 2020.	[no information could be found]
RELAVANT STUDIES RELEASED SINCE MCL THAT AUGMENT THE CRITICAL STUDY SELECTED BY THE STATE AT THE TIME	**PFOS, PFOA, PFHxS, PFNA, PFHpA, PFDA** [no information could be found]	Not later than December 31, 2023, and once every three years thereafter, the Department shall perform a review of relevant developments in the science, assessment, and regulation of PFAS in drinking water for the purpose of evaluating whether to amend 310 CMR 22.07G(3) in light of any advancements in analytical or treatment technology, toxicology and/or any other relevant information.
REFERENCE SOURCES CONSIDERED	**PFOS, PFOA, PFHxS, PFNA, PFHpA, PFDA** Lau et al. (2006); Luebket et l. (2005a); Luebker et al. (2005b); Mass DEP (2019); US EPA (2005); US EPA (2009); United States Environmental Protection Agency. Method 537; US EPA. (2016a); US EPA. (2016b).	

Table 13 Michigan Guidelines for PFAS in Drinking Water

	MICHIGAN	
	PFOA, PFNA, PFHxA, PFOS, PFHxS	
	DECISION POINT	**RATIONAL**
CRITICAL STUDY	**PFOA** Onishchenko N, Fischer C, Wan Ibrahim WN, Negri S, Spulber S, Cottica D, Ceccatelli S. 2011. Prenatal exposure to PFOS or PFOA alters motor function in mice in a sex-related manner. Neurotox. Res. 19(3):452-61. Koskela A, Finnilä MA, Korkalainen M, Spulber S, Koponen J, Håkanss on H, Tuukkanen J, Viluksela M. 2016. Effects of developmental exposure to perfluorooctanoic acid (PFOA) on long bone morphology and bone cell differentiation. Toxicol. Appl. Pharmacol. 301:14-21.	The Workgroup reviewed the available evaluation and selected the ATSDR (2018) critical studies. The Workgroup concluded that the ATSDR position was defensible with respect to range and sensitivity of health endpoints identified and considered in ATSDR (2018).
	PFNA Das KP, Grey BE, Rosen MB, et al. 2015. Developmental toxicity of perfluorononanoic acid in mice. Reproductive Toxicology 51:133- 144.	The Workgroup reviewed the available evaluations and focused on the assessments by ATSDR and New Jersey. Das et al. (2015) was selected by both ATSDR (2018) and NJDEP (2015).
	PFHxA Klaunig, J.E., Shinohara, M., Iwai, H., Chengelis, C.P., Kirkpatrick, J.B., Wang, Z., Bruner, R.H., 2015. Evaluation of the chronic toxicity and carcinogenicity of perfluorohexanoic acid (PFHxA) in Sprague-Dawley rats. Toxicol. Pathol. 43 (2), 209–220.	The Workgroup reviewed the Luz et al. (2019) compiled information and development of a toxicity value. The Workgroup was in agreement with Luz et al. (2019) on selection of the chronic study (Klaunig et al. 2015) for toxicity value development.
	PFOS Dong GH, Zhang YH, Zheng L, Liu W, Jin YH, He QC. (2009). Chronic effects of perfluorooctanesulfonate exposure on immunotoxicity in adult male C57BL/6 mice. Arch Toxicol. 83(9):805-815.	The Workgroup discussed the available evaluations, particularly MDH (2019) and New Jersey Department of Environmental Protection (NJDEP) (2018), and selected a critical study with an immune system functional assay rather than observational data.
	PFHxS	

	NTP 2018 TOX-96: Toxicity Report Tables and Curves for Short-term Studies: Perfluorinated Compounds: Sulfonates and personal communication between MDH and NTP project manager Dr. Chad Blystone (as cited in the HRA Toxicology Review Worksheet for PFHxS, last revised 3/8/2019)	The Workgroup reviewed available evaluations and focused on the ones from Minnesota Department of Health (2019) and ATSDR (2018). In both evaluations, thyroid endpoints were selected. The Workgroup discussed Chang et al. (2018) and concluded that the health outcome (reduction in litter size) was a marginal effect.
POINT OF DEPARTURE (POD)	**PFOA** The average serum concentration was estimated in the mice (8.29 mg/L) using a three-compartment pharmacokinetic model using animal species-, strain-, sex-specific parameters.	The Workgroup decided that serum-based points of departure were appropriate for PFAS.
	PFNA A NOAEL of 1 mg/kg/day was identified for developmental effects. The average serum concentration for NOAEL (1 mg/kg/day) was estimated (6.8 mg/L) in dams using an empirical clearance model. The estimated time-weighted average serum concentration corresponding to the NOAEL was 6.8 mg/L.	The Workgroup decided that serum-based points of departure were appropriate for PFAS.
	PFHxA Critical effect renal tubular degeneration and renal papillary necrosis in female rats – BMDL10 90.4 mg/kg/day	The Workgroup noted that the Benchmark Dose approach is preferred over the use of a NOAEL/LOAEL.
	PFOS The NOAEL for suppression of plaque forming cell response and increase in liver mass was 0.5 mg/kg total administered dose which corresponded to a serum concentration of 0.674 mg/L.	The Workgroup decided that serum-based points of departure were appropriate for PFAS.
	PFHxS POD of 32.4 mg/L serum concentration for male rats based on BMDL20. A BMR of 20% was used in the BMD modeling based on clinical and toxicological knowledge regarding adverse outcomes associated with decreases in circulating thyroid hormones. MDH stated that 20% provided a more statistically reliable and biologically significant BMR. (MDH conducted	The Workgroup decided that serum-based points of departure were appropriate for PFAS. Although the Workgroup concluded that the Chang et al. (2018) health outcome was marginal, they did note that the serum concentration at the NOAEL for Chang et al. (2018) was

	Benchmark Dose modeling and provided modeling run data in the HRA Toxicology Review Worksheet for PFHxS, last revised 3/8/2019.	equivalent to the serum concentration at the selected POD.
HUMAN EQUIVALENT DOSE (HED)	**PFOA** The time-weighted average serum concentration of 8.29 mg/L was converted to the HED using the below equation. LOAELHED = (TWA serum x ke x Vd) = 0.001163 mg/kg/day Ke = 0.000825175 (8.2 x 10-4) based on a human serum half-life of 840 days (Bartell et al. 2010) Vd = 0.17 L/kg	The Workgroup selected the PFOA serum half-life of 840 days (2.3 years) as more relevant for exposure to the general population as this half-life corresponds to data from Bartell et al. (2010) in which 200 individuals (100 men, 100 women) were exposed by drinking PFOA contaminated water. The Workgroup selected the volume of distribution based on human data, when available.
	PFNA The time-weighted average serum concentration of 6.8 mg/L was converted to the HED using the below equation. NOAELHED = (TWA serum x ke x Vd) = 0.000665 mg/kg/day Ke = 0.000489165 (4.8 x 10-4) based on a human serum half-life of 1417 days Vd = 0.2 L/kg	The Workgroup discussed the human serum half-lives available from Zhang et al. (2013), which were an arithmetic mean of 2.5 years (913 days) for 50-year-old or younger females and 4.3 years (1570 days) for females older than 50 years old and all males. An average of 3.9 years (1417 days) was calculated based on those averages. The Workgroup selected the calculated average as it would better represent the entire population.
	PFHxA Therefore, the BMD was adjusted by (80kg/0.45 kg)¼ = 3.65. The resulting PODHED (90.4 mg/kg/day divided by 3.65) = 24.8 mg/kg/day.	The Workgroup discussed the description of the Benchmark Dose modeling conducted by Luz et al. (2019) and concluded the modeling was adequate for use. The Workgroup did not conduct their own Benchmark Dose modeling. The Workgroup took into consideration the available serum half-life data presented in Russell et al. (2013) and concluded that, unlike most PFAS, allometric scaling could be supported.

	PFOS The serum concentration of 0.674 mg/L was converted to the HED using the below equation. NOAELHED = (TWA serum x ke x Vd) = 0.0000866 mg/kg/day Ke = 0.000558539 (5.5 x 10-4) based on a human serum half-life of 1241 days Vd = 0.23 L/kg	The Workgroup selected the serum half-life from a nonoccupationally exposed population as it is closer to the general population's exposure. The Workgroup selected volume of distributions based on human data, when available.
	PFHxS The POD (32.4 mg/L) was multiplied by a toxicokinetic adjustment based on the chemical's specific clearance rate of 0.000090 L/kg-d (Vd = 0.25 L/kg, half-life = 1935 days) for a human equivalent dose of 0.00292 mg/kg/day.	The Workgroup selected the human serum half-life from Li et al. (2018) as it was a non-occupational population drinking water with elevated PFAS.
UNCERTAINTY FACTORS BROKEN DOWN	**PFOA** A total uncertainty factor of 300: 3 (100.5) for LOAEL to NOAEL 10 for human variability 3 (100.5) for animal to human variability 1 for subchronic to chronic 3 (100.5) for database deficiencies (endocrine effects)	The Workgroup discussed the use of an uncertainty factor of 3 for use of a LOAEL. They noted that a NOAEL for immune effects was similar to the LOAEL selected and that the selected LOAEL represented less severe effects. The Workgroup concluded that use of the 3 (100.5) would be sufficiently protective. The Workgroup added a database uncertainty factor of 3 (100.5) for deficiencies the database regarding endocrine effects. The Workgroup noted that the mammary gland effects may signal a concern for other low dose endocrine effects.
	PFNA A total uncertainty factor of 300: 1 for LOAEL to NOAEL 10 for human variability 3 (100.5) for animal to human variability 1 for subchronic to chronic 10 for database deficiencies was used	The Workgroup discussed the uncertainty factors selected by ATSDR (2018) and agreed that those selected were appropriate.
	PFHxA Total uncertainty factor of 300: 1 for LOAEL to NOAEL 10 for human variability 3 (100.5) for animal to human variability 1 for subchronic to chronic	The Workgroup discussed the uncertainty factors and selected an uncertainty factor of 10 for database deficiencies. Several items noted were that the available studies were

	10 for database deficiencies – lack of additional chronic toxicity studies and no additional developmental data in a second species, and immune and thyroid endpoints	largely in one species, with no mouse or non-human primate data, and that there was insufficient information addressing immune or thyroid endpoints.
	PFOS A total uncertainty factor of 30: 1 for LOAEL to NOAEL 10 for human variability 3 (100.5) for animal to human difference (toxicodynamic) 1 for subchronic to chronic 1 for database deficiencies	The Workgroup reviewed the uncertainty factors selected by MDH (2019) and adjusted the database uncertainty factor to 1 based on the critical study selection. With consideration of the selected immunotoxicity endpoint, the database uncertainty factor of 1 was supported by the assessments by USEPA (2016), NJDEP (2018), ATSDR (2018) and New Hampshire (2019).
	PFHxS A total uncertainty factor of 300: 1 for LOAEL to NOAEL 10 for human variability 3 (100.5) for animal to human variability (toxicodynamic differences) 1 for subchronic to chronic 10 for database deficiencies - to address concerns for early life sensitivity and lack of 2-generation or immunotoxicity studies	The Workgroup reviewed the uncertainty factors used by MDH (2019) and concluded that the database uncertainty factor of 10 was very defensible in this situation, especially for the lack of information on early-life sensitivity.
TOXICITY VALUE	**PFOA** 3.9 ng/kg/day (3.9 x 10-6 mg/kg/day) which corresponds to a serum concentration of 0.028 mg/L	Serum levels used in development of these toxicity levels are not meant to indicate a level where health effects are likely. These serum levels are calculated to be at a point where no or minimal risk exists for people drinking water with a certain PFAS.
	PFNA 2.2 ng/kg/day (2.2 x 10-6 mg/kg/day) which corresponds to a serum concentration of 0.023 mg/L Serum levels used in development of these toxicity levels are not meant to indicate a level where health effects are likely. These serum levels are calculated to be at a point where no or minimal risk exists for people drinking water with a certain PFAS.	Human equivalent dose or serum level divided by the total uncertainty factors = toxicity value
	PFHxA 83,000 ng/kg/day (8.3 mg/kg/day)	

		Human equivalent dose divided by the total uncertainty factor = toxicity value
	PFOS 2.89 ng/kg/day (2.89 x 10-6 mg/kg/day) which corresponds to a serum concentration of 0.022 µg/ml Serum levels used in development of these toxicity levels are not meant to indicate a level where health effects are likely. These serum levels are calculated to be at a point where no or minimal risk exists for people drinking water with a certain PFAS.	Human equivalent dose or serum level divided by the total uncertainty and modifying factors = toxicity value
	PFHxS 9.7 ng/kg/day (9.7 x 10-6 mg/kg/day) which corresponds to a serum concentration of 0.11 µg/ml Serum levels used in development of these toxicity levels are not meant to indicate a level where health effects are likely. These serum levels are calculated to be at a point where no or minimal risk exists for people drinking water with a certain PFAS.	Human equivalent dose or serum level divided by the total uncertainty factors = toxicity value
EXPOSURE PARAMETERS & DRINKING WATER HBVS	**PFOA** Breast-fed infant, which is also protective of a formula-fed infant. Placental transfer of 87% Breastmilk transfer of 5.2% Human Serum half-life of 840 days Volume of distribution of 0.17 L/kg 95th percentile drinking water intake, consumers only, from birth to more than 21 years old. Upper percentile (mean plus two standard deviations) breast milk intake rate. Time-weighted average water ingestion rate from birth to 30-35 years of age (to calculate maternal serum concentration at delivery). Relative Source Contribution of 50% (0.5) Based on NHANES 95th percentiles for 3-11 (2013-2014) and over 12 years old (2015-2016) participants (CDC 2019). Drinking water HBV: 8 ng/L (ppt)	The Workgroup discussed the Goeden et al. (2019) model which considered full life stage exposure, from fetal exposure, to infant exposure through breastfeeding, and into adulthood. While the model was also developed for a formula-fed infant, the breastfed infant scenario is protective of a formula-fed infant. The Workgroup selected this model for developing drinking water HBVs when the needed inputs were available.

	PFNA Breast-fed infant, which is also protective of a formula-fed infant Placental transfer of 69% Breastmilk transfer of 3.2% Half-life = 1417 days (3.9 years) Volume of distribution = 0.2 L/kg 95th percentile drinking water intake, consumers only, from birth to more than 21 years old Upper percentile (mean plus two standard deviations) breast milk intake rate Time-weighted average water ingestion rate from birth to 30-35 years of age (to calculate maternal serum concentration at delivery) Relative Source Contribution of 50% (0.5) Based on NHANES 95th percentiles for 3-11 (2013-2014) and over 12 years old (2015-2016) Drinking water HBV: 6 ng/L (ppt)	The Workgroup discussed the Goeden et al. (2019) model which considered full life stage exposure, from fetal exposure, to infant exposure through breastfeeding, and into adulthood. While the model was also developed for a formula-fed infant, the breastfed infant scenario is protective of a formula-fed infant. The Workgroup selected this model for developing drinking water HBVs when the needed inputs were available.
	PFHxA 95th percentile of water intake for consumers only (direct and indirect consumption) for adults (>21 years old) of 3.353 L/day, per Table 3-1, USEPA Exposure Factors Handbook, 2019. An adult body weight of 80 kilograms was used. A default Relative Source Contribution of 20% was included. Drinking water HBV: 400,000 ng/L (ppt) (400 micrograms per Liter or parts per billion)	The Workgroup discussed the use of an upper percentile water intake. The 95th percentile for consumers only was selected as it would protect those drinking larger amounts of water. As no human serum data were available to assess the population's exposure to PFHxA from sources other than drinking water, a default Relative Source Contribution of 20% was selected consistent with USEPA (2000) guidance. The Workgroup evaluated the protectiveness of the renal tubular degeneration and renal papillary necrosis in relation to the reduced pup weights observed in Loveless et al. (2009). Available data did not support Benchmark Dose Modeling for further evaluation of Loveless et al. (2009) data.

		Numeric HBV derived and justified using the above information in the following equation: HBV = (RSC x Toxicity value x Body weight) / Water in take
	PFOS Breast-fed infant, which is also protective of a formula-fed infant Placental transfer of 43% Breastmilk transfer of 1.3% Human serum half-life of 1241 days (3.2 years) Volume of distribution of 0.23 L/kg 95th percentile drinking water intake, consumers only, from birth to more than 21 years old. Upper percentile (mean plus two standard deviations) breast milk intake rate. Time-weighted average water ingestion rate from birth to 30-35 years of age (to calculate maternal serum concentration at delivery). Relative Source Contribution of 50% Based on NHANES 95th percentiles for 3-11 (2013-2014) and over 12 years old (2015-2016) participants. Drinking water HBV: 16 ng/L (ppt)	The Workgroup discussed the Goeden et al. (2019) model which considered full life stage exposure, from fetal exposure, to infant exposure through breastfeeding, and into adulthood. While the model was also developed for a formula-fed infant, the breastfed infant scenario is protective of a formula-fed infant. The Workgroup selected this model for developing drinking water HBVs when the needed inputs were available.
	PFHxS Breast-fed infant, which is also protective of a formula-fed infant Placental transfer of 80% Breastmilk transfer of 1.2% Human serum half-life of 1935 days Volume of distribution of 0.25 L/kg 95th percentile drinking water intake, consumers only, from birth to more than 21 years. Upper percentile (mean plus two standard deviations) breast milk intake rate. Time-weighted average water ingestion rate from birth to 30-35 years of age (to calculate maternal serum concentration at delivery). Relative Source Contribution of 50% (0.5) Based on NHANES 95th percentiles for 3-11 (2013-2014) and over 12 years old (2015-	The Workgroup discussed the Goeden et al. (2019) model which considered full life stage exposure, from fetal exposure, to infant exposure through breastfeeding, and into adulthood. While the model was also developed for a formula-fed infant, the breastfed infant scenario is protective of a formula-fed infant. The Workgroup selected this model for developing drinking water HBVs when the needed inputs were available.

	2016) participants (CDC 2019) Drinking water HBV: 51 ng/L (ppt)	
	PFOA [no information could be found]	[no information could be found]
	PFNA [no information could be found]	[no information could be found]
	PFHxA [no information could be found]	[no information could be found]
	PFOS [no information could be found]	[no information could be found]
	PFHxS [no information could be found]	[no information could be found]
RELAVANT STUDIES RELEASED SINCE MCL THAT AUGMENT THE CRITICAL STUDY SELECTED BY THE STATE AT THE TIME	**PFOA** [no information could be found]	[no information could be found]
	PFNA [no information could be found]	[no information could be found]
	PFHxA [no information could be found]	[no information could be found]
	PFOS [no information could be found]	[no information could be found]
	PFHxS [no information could be found]	[no information could be found]
REFERENCE SOURCES CONSIDERED	**PFOA** ATSDR. (2018). Agency for Toxic Substances and Disease Registry. Toxicological Profile for Perfluoroalkyls. Draft for Public Comment. June 2018. Bartell SM, Calafat AM, Lyu C, et al. 2010. Rate of decline in serum PFOA concentrations after granular activated carbon filtration at two public water systems in Ohio and West Virginia. Environ Health Perspect 118(2):222-228. CDC. (2019). (Center for Disease Control) Fourth National Report on Human Exposure to Environmental Chemicals, Updated Tables, January 2019, Volume One. Koskela A, Finnilä MA, Korkalainen M, Spulber S, Koponen J, Håkanss on H, Tuukkanen J, Viluksela M. 2016. Effects of developmental exposure to perfluorooctanoic acid (PFOA) on long bone morphology and bone cell	

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Table 14 New Jersey Guidelines for PFAS in Drinking Water

	NEW JERSEY	
	PFOA, PFOS, PFNA	
	DECISION POINT	RATIONAL
CRITICAL STUDY	**PFOA** Loveless et al. 2006. Comparative responses of rats and mice to linear/branched, linear, or branched ammonium perfluorooctanoate (APFO).	[no information could be found]
	PFOS Dong et al. 2009. Chronic effects of perfluorooctanesulfonate exposure on immunotoxicity in adult male C57BL/6 mice.	[no information could be found]
	PFNA [no information could be found]	[no information could be found]
POINT OF DEPARTURE (POD)	**PFOA** 4350 (BMDL)	[no information could be found]
	PFOS 674 (NOAEL)	[no information could be found]
	PFNA [no information could be found]	[no information could be found]
HUMAN EQUIVALENT DOSE (HED)	**PFOA** Clearance factor = 0.000081 L/kg/d	A clearance factor (1.4 x 10-4 L/kg/day) which relates serum PFOA concentrations to human PFOA doses was applied to the Target Human Serum Levels to develop Reference Doses. For delayed mammary gland development, the Target Human Serum Level is 0.8 ng/ml, which is below the median serum PFOA level in the U.S. general population.
	PFOS Clearance factor = 0.000081 L/kg/d	A clearance factor (1.4 x 10-4 L/kg/day) which relates serum PFOA concentrations to human PFOA doses was applied to the Target Human Serum Levels to develop Reference Doses. For delayed mammary gland

		development, the Target Human Serum Level is 0.8 ng/ml, which is below the median serum PFOA level in the U.S. general population.
	PFNA [no information could be found]	[no information could be found]
UNCERTAINTY FACTORS BROKEN DOWN	**PFOA** A total uncertainty factor of 300 10 for Intraspecies 3 for Interspecies 10 for database	This Target Human Serum Level and Reference Dose incorporate uncertainty factors to protect sensitive human subpopulations, to account for toxicodynamic differences between human and experimental animals, and to protect for more sensitive endpoints that occur from developmental exposures (delayed mammary gland development, persistent hepatic toxicity, and others).
	PFOS A total uncertainty factor of 30 10 for Intraspecies 3 for Interspecies	This Target Human Serum Level and Reference Dose incorporate uncertainty factors to protect sensitive human subpopulations, to account for toxicodynamic differences between human and experimental animals, and to protect for more sensitive endpoints that occur from developmental exposures (delayed mammary gland development, persistent hepatic toxicity, and others).
	PFNA A total uncertainty factor of 1000: 10 for intra-individual human variability 3 for toxicodynamic differences between human and experimental animals 10 for less than chronic exposure duration 3 to account for the incomplete toxicology database	Based on available toxicokinetic data from animal and humans, a ratio of 200:1 was used to estimate the increase in PFNA in human blood serum from ongoing exposure to a given concentration of PFNA in drinking water. To account for sources of exposure to PFNA other than drinking water, a chemical specific Relative Source Contribution factor of 50% was developed based on the most recent (2011-12) NHANES data

		for the 95th percentile PFNA serum level in the U.S. general population.
TOXICITY VALUE	**PFOA** Reference dose = 2 ng/kg/d	For each of these endpoints, benchmark dose modeling of serum PFOA levels from mouse studies was performed and appropriate uncertainty factors were applied to develop a Target Human Serum Level (analogous to a Reference Dose but on a serum level basis).
	PFOS Reference dose = 1/8 ng/kg/dd	For each of these endpoints, benchmark dose modeling of serum PFOA levels from mouse studies was performed and appropriate uncertainty factors were applied to develop a Target Human Serum Level (analogous to a Reference Dose but on a serum level basis).
	PFNA Target human serum level, 4.9 ng/ml, is analogous to a Reference Dose but based on serum level rather than administered dose.	Because the same administered dose results in a much higher internal dose in humans than in experimental animals, interspecies comparison for PFNA are made based on internal dose (serum level) rather than administered dose.
EXPOSURE PARAMETERS & DRINKING WATER HBVS	**PFOA, PFOS** The Health Effects Subcommittee used a risk assessment approach intended to protect for chronic drinking water exposure to develop a Health-based MCL of 14 ng/L (0.014 µg/L), and the Testing Subcommittee developed an analytical PQL of 6 ng/L (0.006 µg/L). Health-based MCL of 14 ng/L, and the Testing Subcommittee determined a PQL of 6 ng/L.	A Health-based Maximum Contaminant Level (Health-based MCL) for PFOA was developed using a risk assessment approach intended to protect for chronic (lifetime) drinking water exposure.
	PFNA The Health Effects Subcommittee concluded that a Health-based MCL of 13 ng/L is scientifically defensible and is protective for	The Health Effects Subcommittee conducted an extensive literature search to identify scientific studies relevant to development of a Health-based MCL protective for

	chronic (lifetime) exposure, and the Testing Subcommittee determined a PQL of 5 ng/L.	chronic (lifetime) drinking water exposure to PFNA. Human epidemiology studies which found associations with health effects at levels of exposure prevalent in the general population provide support for the health-based MCL but were not used as the basis for quantitative risk assessment.
	PFOA, PFOS, PFNA [no information could be found]	
RELAVANT STUDIES RELEASED SINCE MCL THAT AUGMENT THE CRITICAL STUDY SELECTED BY THE STATE AT THE TIME	**PFOA** [no information could be found]	[no information could be found]
	PFOS [no information could be found]	[no information could be found]
	PFHxS [no information could be found]	[no information could be found]
	PFNA [no information could be found]	[no information could be found]
REFERENCE SOURCES CONSIDERED	**PFOA, PFOS, PFNA** Vermont Department of Health, Drinking Water Guidance. http://www.healthvermont.gov/sites/default/files/documents/pdf/ENV_ECP_GeneralScreeningValues_Water.pdf USEPA, 2016. Health Effects Support Document for Perfluorooctanoic Acid (PFOA). 822-R-16-003. U.S. Environmental Protection Agency Office of Water (4304T) Health and Ecological Criteria Division, Washington, D.C. USEPA, 2016. Health Effects Support Document for Perfluorooctanesulfonic Acid (PFOS). 822-R-16-00. U.S. Environmental Protection Agency Office of Water (4304T) Health and Ecological Criteria Division, Washington, D.C. USEPA, 1990. Seminar Publication. Risk Assessment, Management and Communication of Drinking Water Contamination. EPA/625/4-89/024. Washington, D.C. U.S. EP, Office of Drinking Water, Office of Water. USEPA (U.S. Environmental Protection Agency). 2000. Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health. EPA-822-B-00-004. U.S. Environmental Protection Agency, Office of Water, Office of Science and Technology, Washington, DC. Retrieved May 2016. http://www.nj.gov/drbc/library/documents/EPA_human-healthcriteria2000.pdf.	

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Table 15 New Hampshire Guidelines for PFAS in Drinking Water

	NEW HAMPSHIRE	
	PFOA, PFOS, PFHxS, PFNA	
	DECISION POINT	RATIONAL
CRITICAL STUDY	**PFOA** Loveless et al., 2006, NJ DWQI 2017, increased relative liver weight in mice.	[no information could be found]
	PFOS Dong et al. 2011, sub-chronic effect of perfluorooctanesulfonate (PFOS) on the balance of type 1 and type 2 cytokine in adult C57BL6 mice	[no information could be found]
	PFHxS Chang et al., 2018, reduced litter size in mice.	[no information could be found]
	PFNA Das et al., 2015, NJ DWQI 2018, increased relative liver weight in mice.	[no information could be found]
POINT OF DEPARTURE (POD)	**PFOA** Animal Serum Dose (ng/mL) = 4,351	Differences in the final MCL are due to NH's use of the transgenerational exposure model for breastfeeding.
	PFOS Animal Serum Dose (ng/mL) = 6,260	[no information could be found]
	PFHxS Animal Serum Dose (ng/mL) = 27,200	[no information could be found]
	PFNA Animal Serum Dose (ng/mL) = 4,900	[no information could be found]
HUMAN EQUIVALENT DOSE (HED)	**PFOA** Target serum level = 43.5 ng/mL Dosimetric Adjustment Factor (L/kg/d) = 1.20E-04	The POD represents an internal animal serum level associated with the adverse health outcome of concern. Dividing the POD by the total uncertainty factor yields a protective target serum level equivalent for the human population. This is not a clinical or diagnostic value, nor should it be interpreted as such.
	PFOS Target serum level = 62.6 ng/mL Dosimetric Adjustment Factor (L/kg/d) = 1.28E-04	[no information could be found]
	PFHxS	[no information could be found]

	Target serum dose = 90.7 ng/mL Dosimetric Adjustment Factor (L/kg/d) = 1.03E-04	
	PFNA Target serum level = 16.3 ng/mL Dosimetric Adjustment Factor (L/kg/d) = 1.52E-04	[no information could be found]
UNCERTAINTY FACTORS BROKEN DOWN	**PFOA** A total uncertainty factor of 100: 10 Intraspecies variability 3 Interspecies variability 3 Database limitations	The POD is reduced by uncertainty factors to take into account incomplete knowledge regarding critical factors such as when there is incomplete knowledge of human variability and sensitivity; in cases where short-term studies are used to protect against effects from long-term exposure, and when the usual required studies to set a standard are missing.
	PFOS A total uncertainty factor of 100: 10 Intraspecies variability 3 Interspecies variability 3 Database limitations	The POD is reduced by uncertainty factors to take into account incomplete knowledge regarding critical factors such as when there is incomplete knowledge of human variability and sensitivity; in cases where short-term studies are used to protect against effects from long-term exposure, and when the usual required studies to set a standard are missing.
	PFHxS A total uncertainty factor of 300: 10 Intraspecies variability 3 Interspecies variability 10 Database limitations	The POD is reduced by uncertainty factors to take into account incomplete knowledge regarding critical factors such as when there is incomplete knowledge of human variability and sensitivity; in cases where short-term studies are used to protect against effects from long-term exposure, and when the usual required studies to set a standard are missing.
	PFNA A total uncertainty factor of 300: 10 Intraspecies variability 3 Interspecies variability 10 Database limitations	The POD is reduced by uncertainty factors to take into account incomplete knowledge regarding critical factors such as when there is incomplete knowledge of human

		variability and sensitivity; in cases where short-term studies are used to protect against effects from long-term exposure, and when the usual required studies to set a standard are missing.
TOXICITY VALUE	**PFOA** Reference dose = 5.1 ng/kg-d	[no information could be found]
	PFOS Reference dose = 8.0 ng/kg/d	[no information could be found]
	PFHxS Reference dose (ng/kg/d) = 9.3	[no information could be found]
	PFNA Reference dose (ng/kg/d) = 2.5	[no information could be found]
EXPOSURE PARAMETERS & DRINKING WATER HBVS	**PFOA** Initial Proposed MCL/AGQS: 38 ppt Final Proposed MCL/AGQS: 12 ppt	[no information could be found]
	PFOS Initial Proposed MCL/AGQS: 70 ppt Final Proposed MCL/AGQS: 15 ppt	[no information could be found]
	PFHxS Initial Proposed MCL/AGQS: 85 ppt Final Proposed MCL/AGQS: 18 ppt	[no information could be found]
	PFNA Initial Proposed MCL/AGQS: 23 ppt Final Proposed MCL/AGQS: 11 ppt	[no information could be found]
YEAR	**PFOA, PFOS, PFHxS, PFNA** The effective date was September 30, 2019. However, effective December 31, 2019, the Merrimack County Superior Court issued a preliminary injunction barring enforcement of these rules due to the alleged failure of NHDES to appropriately consider the costs and benefits of the rules. The former AGQS rule of 70 ppt for PFOA, PFOS, or combined concentrations of the two chemicals, as adopted by NHDES in 2016, remains in effect, while the 2019 rules are under an injunction.	
RELAVANT STUDIES RELEASED SINCE MCL THAT	**PFOA** [no information could be found]	[no information could be found]
	PFOS [no information could be found]	[no information could be found]

AUGMENT THE CRITICAL STUDY SELECTED BY THE STATE AT THE TIME		
	PFHxS [no information could be found]	[no information could be found]
	PFNA [no information could be found]	[no information could be found]
REFERENCE SOURCES CONSIDERED	**PFOA** Li et al., 2017, serum-derived half-life estimates from men and women exposed to PFAS via drinking water. Loveless et al., 2006, NJ DWQI 2017, increased relative liver weight in mice.	
	PFOS Li et al., 2017, serum-derived half-life estimates from men and women exposed to PFAS via drinking water. Luebker et al., 2005a, EPA 2016b, reduced pup weight and developmental delays in rats.	
	PFHxS Chang et al., 2018, reduced litter size in mice. Li et al., 2017, serum-derived half-life estimates from men and women exposed to PFAS via drinking water.	
	PFNA Das et al., 2015, NJ DWQI 2018, increased relative liver weight in mice. Zhang et al., 2013, ATSDR 2018, urine-derived half-life from community exposure to PFNA.	

Table 16 Vermont Guidelines for PFAS in Drinking Water

	VERMONT	
	PFOA, PFOS, PFHpA, PFHxS, PFNA	
	DECISION POINT	RATIONAL
CRITICAL STUDY	**PFOA** Lau et al. 2006. Effects of perfluorooctanoic acid exposure during pregnancy in the mouse.	[no information could be found]
	PFOS Luebker et al. 2005. Two-generation reproduction and cross-foster studies of perfluorooctanesulfonate (PFOS) in rats.	[no information could be found]
	PFHpA [no information could be found]	[no information could be found]
	PFHxS [no information could be found]	[no information could be found]
	PFNA [no information could be found]	[no information could be found]
POINT OF DEPARTURE (POD)	**PFOA** 38000 (LOAEL)	[no information could be found]
	PFOS 6260 (NOAEL)	[no information could be found]
	PFHpA [no information could be found]	[no information could be found]
	PFHxS [no information could be found]	[no information could be found]
	PFNA [no information could be found]	[no information could be found]
HUMAN EQUIVALENT DOSE (HED)	**PFOA** Clearance factor = 0.00014 (L/kg/d)	[no information could be found]
	PFOS Clearance factor = 0.000081 (L/kg/d)	[no information could be found]
	PFHpA [no information could be found]	[no information could be found]

	PFHxS [no information could be found]	[no information could be found]
	PFNA [no information could be found]	[no information could be found]
UNCERTAINTY FACTORS BROKEN DOWN	**PFOA** Total uncertainty factor of 300 10 for Intraspecies 3 for Interspecies 10 for LOAEL to NOAEL	[no information could be found]
	PFOS Total uncertainty factor of 30 10 for Intraspecies 3 for Interspecies	[no information could be found]
	PFHpA [no information could be found]	[no information could be found]
	PFHxS [no information could be found]	[no information could be found]
	PFNA [no information could be found]	[no information could be found]
TOXICITY VALUE	**PFOA** RfDo = 2x10-5 mg/kgBW-d Reference dose = 20 (ng/kg/d)	The Department applied the oral reference dose for PFOA and PFOS to the sum of PFOA, PFOS, PFHxS, PFHpA, and PFNA.
	PFOS RfDo = 2x10-5 mg/kgBW-d Reference dose = 20 (ng/kg/d)	The Department applied the oral reference dose for PFOA and PFOS to the sum of PFOA, PFOS, PFHxS, PFHpA, and PFNA.
	PFHpA [no information could be found]	[no information could be found]
	PFHxS [no information could be found]	[no information could be found]
	PFNA [no information could be found]	[no information could be found]

EXPOSURE PARAMETERS & DRINKING WATER HBVS	**PFOA, PFOS, PFHpA, PFHxS, PFNA** DWHA= (HQ)(RfDo)(1/BWAIR)(CF)(RSC) = (1)(2 x 10-5 mg/kg BW-day)(1/0.175 L/kg BW-day)(1000 µg/mg)(0.2) = 0.02285 µg/L (ppb) = 0.02285 µg/L (ppb) x 1000 ng/ µg = 22.9 ng/L (ppt) ≈ 20 ppt DWHA = Drinking Water Health Advisory HQ= Hazard Quotient RfDo= chronic oral reference dose BWAIR= Body Weight adjusted Water Intake Rate CF= Units Conversion Factor RSC= Relative Source Contribution The 95th percentile Body Weight Adjusted Water Intake Rate for the first year of life based on combined direct and indirect water intake from community water supplies for consumers only is 0.175 L/kgBW-d MCL: 20 nanograms per liter (ng/L). The sum of these five PFAS cannot exceed 20 ng/L.	Calculated a candidate drinking water advisory for consideration based on the cancer endpoint using the information provided in EPA's 2016 Health Effects Support Documents for PFOA11 and PFOS12 and determined that derivation of the Health Advisory based on the noncancer endpoint is more protective.
YEAR	**PFOA** [no information could be found]	[no information could be found]
	PFOS [no information could be found]	[no information could be found]
	PFHpA [no information could be found]	[no information could be found]
	PFHxS [no information could be found]	[no information could be found]
	PFNA [no information could be found]	[no information could be found]
RELAVANT STUDIES RELEASED SINCE MCL THAT AUGMENT	**PFOA** [no information could be found]	[no information could be found]
	PFOS [no information could be found]	[no information could be found]

THE CRITICAL STUDY SELECTED BY THE STATE AT THE TIME	**PFHpA** [no information could be found]	[no information could be found]
	PFHxS [no information could be found]	[no information could be found]
	PFNA [no information could be found]	[no information could be found]

Table 17 Change in Costs In order to Meet Proposed Standards

Source: *Cost Benefit Analysis of Proposed New Hampshire Maximum Contaminant Levels (MCLs) and Ambient Ground Water Quality Standards (AGQS) for PFAS Substances* by T. Miller.

Category	Low Estimate	High Estimate
Additional Capital Costs – Public Water systems	$ 63,195,633	$ 137,651,262
Annual payment for capital costs amortized for 30 years at 3 percent – public water systems	$ 3,224,194	$ 7,022,865
Annual Sampling Costs – Public Water Systems	$ 101,202	$ 259,584
Additional Treatment Costs – Public Water Systems	$ 6,799,640	$ 13,221,524
Total Annual Cost – Public Water Systems	**$ 10,125,036**	**$ 20,503,973**
Total Cost per capita – Public Water Systems	**$ 68**	**$ 139**

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APPENDIX 6 – Resources

Study of the Occurrence of Per- and Polyfluoroalkyl Substances (PFAS) in the Commonwealth's Public Drinking Water

General Information on PFAS

PFAS resources for states are available at:
https://www.epa.gov/research-states/pfas- resources-states
Basic Information about PFAS from EPA
https://www.epa.gov/pfas
EPA's Drinking Water PFOA and PFOS Lifetime Health Advisory
https://www.epa.gov/ground-water-and-drinking-water/drinking-water-health-advisories-pfoa-and-pfos
EPA's Technical Fact Sheet – PFOS and PFOA
https://www.epa.gov/sites/default/files/2017-12/documents/ffrrofactsheet_contaminants_pfos_pfoa_11-20-17_508_0.pdf
Centers for Disease Control and Prevention
https://www.atsdr.cdc.gov/pfas/
https://www.cdc.gov/exposurereport/index.html
Food and Drug Administration
https://www.fda.gov/food/newsevents/constituentupdates/ucm479465.htm
National Toxicology Program
https://ntp.niehs.nih.gov/pubhealth/hat/noms/pfoa/index.html
Interstate Technology Regulatory Council (IRTC)
https://pfas-1.itrcweb.org

Virginia - Technical Support

Office of Drinking Water, Virginia Department of Health
https://www.vdh.virginia.gov/drinking-water/pfas/
The VDH Local Health Districts can assist with inquiries on PFAS and associated health risks.
https://www.vdh.virginia.gov/health- department-locator/
Tony S. Singh, Deputy Director and PFAS Program Lead, VDH -ODW
Email: Tony.Singh@vdh.virginia.gov
Dwayne Roadcap, Director, VDH Office of Drinking Water
Email: Dwayne.Roadcap@vdh.virginia.gov
Dwight Flammia, State Toxicologist, VDH Office of Environmental Health Services
Email: Dwight.Flammia@vdh.virginia.gov
Lorrie Andrew-Spear, Risk Communications Manager, VDH Office of Communications
Email: Lorrie.Andrew-Spear@vdh.virginia.gov

Other State Resources

The Association of State Drinking Water Administrators (ASDWA) provides a good overview of states' efforts on PFAs in drinking water:
https://www.asdwa.org/pfas/

The Environment Council of States (ECOS) webpage with PFAS information:
https://www.ecos.org/pfas/
Michigan PFAS in Water:
https://www.environmentalcouncil.org/pfas_in_michigan

Funding for Treating PFAS in Drinking Water

Waterworks interested in installing new treatment technologies can apply to use funding available through ODW's Drinking Water State Revolving Fund (DWSRF) program. More information on this can be found at https://www.vdh.virginia.gov/drinking- water/drinking-water-state-revolving-fund-program/