May 16th, 2021

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Transmitted via email to RA-EPEQB@pa.gov

Re: Rulemaking Petition to Set a Drinking Water MCL for PFOA (25 Pa. Code Chapter 109)

Delaware Riverkeeper Network's Response to the Pennsylvania Department of Environmental Protection Evaluation Report on the DRN Petition for Rulemaking to Set an MCL for PFOA

Dear Ms. Griffin:

We submit this letter on behalf of the Delaware Riverkeeper Network as a response to the Pennsylvania Department of Environmental Protection's Evaluation Report on the DRN Petition for Rulemaking to Set an MCL for PFOA.

Delaware Riverkeeper Network ("DRN") supports the greatest protection that can be attained for the public from exposure to PFAS compounds. This was the foundation for DRN's 2017 rulemaking petition submitted pursuant to 25 Pa. Code § 23 to set a drinking water maximum contaminant level for Perfluorooctanoic Acid ("PFOA") at 1 part per trillion ("ppt") or not to exceed 6 ppt ("Rulemaking Petition"). The Evaluation Report dated April 16, 2021 from the Pennsylvania Department of Environmental Protection ("DEP" or the "Department") concluded, "[a]s a result, it is recommended that the

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number advocated for in the Petition for Rulemaking not be the basis for a proposed rulemaking to establish an MCL for PFOA."[1]

DEP states in the Evaluation Report that they do, however, plan to move ahead with establishing an MCL for PFOA: "While the Department agrees that it should move forward with a proposed rulemaking to set an MCL for PFOA, it does not believe that DRN's proposed MCL was developed appropriately. The Department's proposed rulemaking should be based on available data, studies, and science, and should consider all factors such as health effects, technical limitations, and cost as required under the Federal SDWA and RRA. As a result, the Department recommends that the EQB move forward with a proposed rulemaking to establish an MCL for PFOA. The Department anticipates that it will have a proposed rulemaking developed by the fourth quarter of 2021."[2]

The Evaluation Report goes on to state that other PFAS compounds will also be addressed. DRN fully supports that DEP issue a rulemaking for PFAS compounds proposing statewide MCLs that are protective of human health and the environment. As DRN stated in the Rulemaking Petition, "Once it is known how widespread these contaminants are in the state, it will become clear that an MCL is immediately necessary for PFCs statewide, starting with PFOA."[3] DRN considers the anticipated action by DEP to be a priority for the Commonwealth and one that should have occurred years ago.

In fact, action to establish an MCL for PFOA should have been taken by DEP at least four years ago based on the evidence that DRN submitted to the Environmental Quality Board in May 2017. We felt the need for a safe drinking water standard was urgent at that time but, unfortunately, DEP delayed such regulatory action until its recent announcement that it plans to propose rulemaking.

Emergency action could have been taken to address the PFAS water contamination crisis in Pennsylvania to avoid the exposure to dangerous levels of PFOA in drinking water that people have now had to endure in the interim. DRN welcomes the state's intention to propose an MCL for PFOA but nothing

[1] Exhibit "A" at 22.
[2] *Id.*
[3] Exhibit "B" at 15.

will erase the damage caused by increased levels of risk to which PADEP has contributed. Pennsylvania residents, workers, and visitors have been exposed to this highly toxic compound for additional periods of time due to DEP's regulatory inaction and delays, increasing their risk of developing adverse health effects linked to PFOA. The fact that PFAS build up in human blood means that even very small doses can result in high concentrations in a person's body over time; higher blood levels increase risk of harm. Pennsylvania's lack of a regulatory requirement for its removal from drinking water has had direct and unacceptable consequences for the public. This harm is what DRN was trying to prevent with the Rulemaking Petition.

DRN agrees that DEP's proposed rulemaking "…should be based on available data, studies, and science…" DRN also agrees, as DEP stated, that "[t]he setting of an MCL is not as simple as just picking a number."[4] This is why DRN submitted extensive technical information gathered from reliable sources in support of the need for an MCL for PFOA, including the New Jersey Drinking Water Quality Institute ("NJDWQI"), the body that analyzes and recommends maximum contaminant levels for contaminants under the New Jersey Safe Drinking Water Act.[5]

DRN also submitted several attachments to the Rulemaking Petition produced by the NJDWQI in support of their recommendation for NJDEP to set an MCL for PFOA, providing ample technical support for the availability of effective treatment systems to remove PFOA from drinking water (Attachment 4); the availability of sampling and testing methods readily available for use by laboratories (Attachment 5); the findings of the Health Effects subcommittee, including a widely recognized risk assessment analysis (Attachment 1); and a comprehensive Basis and Background document supporting the recommended MCL (Attachment 3). These attachments were discussed in the text of the Rulemaking Petition, providing technical information that could have been used by DEP in a proposal for rulemaking.[6]

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[4] Exhibit "A" at 21.
[5] *See* https://www.state.nj.us/dep/watersupply/g_boards_dwqi.html ("The NJDWQI is responsible for developing Maximum Contaminant Levels (MCL) or standards for hazardous contaminants in drinking water and for recommending those standards as well as recommendations for the implementation of the drinking water quality program to the Commissioner of the N.J. Department of Environmental Protection (NJDEP).").
[6] Exhibit "B" at 21.

Unfortunately for all involved, once DEP finally decided to respond to the Rulemaking Petition, it appears to be more interested in providing unfounded criticisms, outright ignoring the supporting technical material attached thereto, and shirking its responsibilities and obligations under Pennsylvania law than doing what is best for the citizens of the Commonwealth. It will be shown that DRN's Rulemaking Petition was, in fact, legally sufficient even after the hurdles that DEP established. Further, DEP's proposed MCLG for PFOA of 8 ppt is legally inadequate and fails to rise to the level of a standard based exclusively on the protection of public health with an adequate margin of safety. Finally, in the event that DEP and EQB refuse to act in a manner that is best for the majority of the people they are supposed to serve, DEP and EQB have a constitutional obligation, one that U.S. EPA is not itself subject to, to set the PFOA MCL no higher than the ultimately proposed MCLG, currently at 8 ppt.

I. DRN's Rulemaking Petition was Legally Adequate and, as a Result, DEP and EQB Should Establish an MCL for PFOA of 1 ppt but not to Exceed 6 ppt.

a. Brief Overview of the EQB Petition Process.

It is worthy of a brief review of how the regulatory process works. The procedure for establishing an MCL is governed by regulations for the Environmental Quality Board's "policy" for rulemaking. See 71 P.S. § 510-20, 25 Pa. Code §§ 23.1–23.8. Under the EQB's Petition process, after the DEP determines that a citizen's Petition is complete, the Petition is sent to the EQB. 25 Pa. Code § 23.2. At the next EQB meeting, the Petitioner and DEP make presentations. 25 Pa. Code § 23.4. If the EQB accepts the Petition, as was the case here, then the next step is for the Department to prepare a report and to make a recommendation on rulemaking to the EQB. 25 Pa. Code § 23.6. The Petitioner has the right to receive and to respond to DEP's report. 25 Pa. Code § 23.7, 25 Pa. Code § 23.8. DEP will then make a recommendation to the EQB based on the report and the comments received by the petitioner, with EQB voting on whether or not to adopt the proposed regulation. If the EQB votes to approve the proposed regulations, public notice and comment and rulemaking procedures follow with the Independent Regulatory Review Commission. 71 P.S. §745.5, 71 P.S. §745.5.

Accordingly, DEP also asserts that any Rulemaking Petition under the Pennsylvania Safe Drinking Water Act must also address the factors required by the Federal Safe Drinking Water Act[7] as well as those in Pennsylvania's Regulatory Review Act.[8] "Among other things, the Department must consider technical limitations such as available analytical methods and detection and reporting limits, treatability of the contaminant and available treatment technologies, and costs."[9] Under the Federal Safe Drinking Water Act the process for establishing an MCL begins first with establishing an MCLG, which is the "maximum level of a contaminant in drinking water at which no known or anticipated adverse effect on the health of persons served would occur, and which allows an adequate margin of safety," and is a non-enforceable health goal.[10] The key difference between an MCLG and an MCL is that MCLGs consider only public health and not the limits of detection and treatment technology effectiveness, whereas MCLs permit practical considerations such as costs versus benefits, best available technology or treatment approaches, and other relevant factors like data quality and nature of the risks.[11]

b. DRN's Rulemaking Petition met the requirements of the Federal Safe Drinking Water Act and the Regulatory Review Act and was legally-sufficient.

According to DEP's Evaluation Report on the DRN Rulemaking Petition to set an MCL for PFAS, "DRN did not consider all of the relevant factors when recommending the MCL for PFOA not to exceed 6 ppt."[12]

> For example, which water systems must comply with the MCL, what are the approved analytical methods, which treatment technologies are approved, how will systems monitor for the contaminant, and how will compliance be determined? All of these details are missing from the Petition for Rulemaking, so it is unclear of the recommended MCL would apply or be implemented.[13]

DEP also goes on to criticize the failure of the Rulemaking Petition to address the costs and benefits associated with the promulgation of a new MCL.

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[7] *See* 42 U.S.C. §§ 300f—300j-9; *see also* 40 CFR Parts 141, 142, and 143.
[8] 71 P.S. §§ 745.1—745.15.
[9] Exhibit "A" at 19 (citing 71 P.S. § 745.5b).
[10] 40 CFR § 141.2.
[11] Exhibit "A" at 19-20.
[12] *Id*. at 20.
[13] *Id*.

DRN finds this assertion to be a gross mischaracterization of the facts at hand. Taking these accusations one at a time it will be shown that either DRN did address the factors raised by DEP in the Rulemaking Petition, or, at most, DRN excluded considerations that it believed represented programmatic knowledge that DEP already had at its disposal.

i. Which Water Systems Must Comply with the MCL?

As an initial matter, the Rulemaking Petition addresses this issue directly:

> Regarding the lack of data evaluating whether PFOA contamination is a statewide problem, as discussed at II.B.3. above, there is already ample data showing that PFOA presents a significant health risk in Bucks and Montgomery Counties' drinking water sources, effecting, at a minimum, over 70,000 residents. The Department need only engage in a targeted review of other similarly situated facilities statewide that are likely sources of PFOA and PFOS. These sites include: military facilities, firefighting and aviation testing sites; fire departments where foam was stored, used and/or tested; aqueous firefighting foam manufacturers, testers, and suppliers; airports; wastewater treatment facilities and their discharge points; sewage sludge and dredge spoils application sites; and manufacturing sites that manufactured or used PFCs in their process.[14]

Based on the information provided in the Rulemaking Petition, sampling could have been performed within the months following its submission and expeditiously completed to provide data to inform the agency as to the occurrence of PFOA and PFAS in locations statewide. Instead, a sampling plan was not implemented by DEP until more than two years after the Rulemaking Petition was submitted—Phase 1 was begun in June 2019 and carried out through 2020. There is no reasonable excuse for delaying sampling for two years after DRN's plea for action in the Rulemaking Petition.

Beyond this, this consideration is a programmatic one that is embedded in the regulations that govern the administration of the Pennsylvania Safe Drinking Water Act, something that the Department shouldn't need to be referred to. Turning to the Pennsylvania Code, § 109.3 provides that "[t]his chapter applies to each public water system, unless the public water system meets all of the following conditions: (1) Consists only of distribution and storage facilities, and does not have collection and treatment facilities. (2) Obtains all of its water from, but is not owned or operated by, a public water system to which this

[14] Exhibit "B" at 20-21.

chapter applies. (3) Does not sell water to any person. (4) Does not provide water for potable purposes to carriers which convey passengers in interstate commerce." Further, § 109.202(a)(1) provides that "[a] public water system shall supply drinking water that complies with the primary MCLs, MRDLs and treatment technique requirements adopted by the EQB under the act."

Thus, in the event that DRN's Rulemaking Petition was adopted by the EQB, it naturally follows that all public water systems would need to comply with the MCL unless they met all of the conditions list in Section 109.3. As Pennsylvania is one of two states that does not regulate private water systems, such systems would not be required to comply with the MCL proposed by DRN. DRN did not feel as though it was necessary to include this in the Rulemaking Petition as DEP and the EQB are the agencies responsible for administering the Commonwealth's Safe Drinking Water Act and the enforcement of MCLs.

ii. What are the Approved Analytical Methods?

The next "shortcoming" identified by DEP was that the Rulemaking Petition failed to identify the approved analytical methods for PFAS. Once again, this is an area that DRN felt DEP would have the institutional expertise and experience necessary to know this information without DRN spelling it out even more explicitly than it already did. Contrary to DEP's characterization, DRN's Rulemaking Petition actually does include as an Attachment the *Report on the Development of a Practical Quantitation Level for Perfluorooctanoic Acid (PFOA) in Drinking Water* prepared by the New Jersey Drinking Water Quality Institute Testing Subcommittee, which deals with approved analytical methods extensively.[15] Thus,

[15] NJDWQI Testing Subcommittee Draft Report on the Development of a Practical Quantitation Level for Perfluorooctanoic Acid (PFOA) in Drinking Water (August 29, 2016) at 5-6 ("The MWH Laboratories proprietary method, MWH SOP-HPLC 12 (also referred to as MWH PFC Extra), was the analytical method used in the 2009 NJDEP PFC Study. This method was approved by [the Office of Quality Assurance] and offered lower reporting limits for the PFCs of concern. . .. The MWH-PFC Extra reporting limit for PFOA is 5 ng/L."); *Id.* at 6 ("Table 2 provides a summary of PFOA laboratory information obtained from the NJDEP PFC database for samples collected between June 2006 and April 2016. It includes analytical methods, RLs, MDLs, and the number of analyses performed with those RLs/MDLs by three laboratories."); *Id.* fn. 6 (acknowledging that Table 2 "does not include all laboratories capable of performing PFC analysis, only those *that analyzed New Jersey public water systems samples during June 2006-April 2016*.") (emphasis added); *Id.* at 7 ("There are currently three drinking water analytical methods that have been approved by NJDEP OQA as [Department Sanctioned Analytical Methods] for the analysis of PFOA. These consist of EPA 537 and two proprietary methods: EV-LC-0012 Rev 12 developed by Test America-Denver and MWH-SOP-HPLC12 Rev 4.0 developed by Eurofins Eaton Analytical (California). These three DSAMs are similar in that they utilize solid phase extraction, isotope dilution and electrospray ionization with LC/MS/MS."), *available at* http://www.nj.gov/dep/watersupply/pdf/testing-subcompql-pfoa-8.29.16KA.pdf. *See* Exhibit "B" fn. 113.

approved analytical methods were indeed addressed in the Rulemaking Petition. Further, DEP was also aware of approved analytical methods as far back as 2009, when U.S. "EPA published Method 537, a solid-phase extraction and liquid chromatography/tandem mass spectrometry (LC/MS/MS) method for perfluorinated alkyl acids (which includes PFOA)."[16] "The EPA established the specific analytical methods to be used for analyzing the UCMR3 contaminants. The PFOA analysis, which also included the analysis of the other five PFCs mentioned above, was performed exclusively with EPA Method 537 version 1.1 for the UCMR3."[17] An interesting note is that, in Table 3, NJDEP identified Eurofins Lancaster Laboratories Environmental as one of the "Laboratories Certified by NJDEP Office of Quality Assurance for Analysis of PFOA in Drinking Water," which happens to be a Pennsylvania-based laboratory.[18]

U.S. EPA's performance of the UCMR3 in 2013-2015 included the sampling and analysis of both public and private wells throughout Pennsylvania to determine the presence of PFOAs in drinking water. This is something that DEP was involved in, thus evidencing the Department's awareness of the U.S. EPA-approved analytical method in regards to PFAS. Further demonstrating the fact that DEP was aware of the U.S. EPA-approved analytical method, is DEP itself. In the Evaluation Report, DEP acknowledges that they began sampling "in June of 2019 and included analysis of six (6) PFAS . . . to be consistent with EPA's UCMR 3. However, the Department had the opportunity in 2020 to expand the sampling to 18 PFAS by using EPA Method 537.1."[19] The Department's "[Bureau of Laboratories] was able to achieve proficiency for EPA Method 537.1 and received accreditation from New Jersey in December of 2019."[20]

Thus, not only did DRN include approved analytical methods in its Rulemaking Petition, but DEP was also fully aware of approved analytical methods as far back as 2009. In fact, NJDEP identified that it was analyzing PFAS samples as far back as June 2006. At best, DEP could potentially say that approved

[16] NJDWQI Testing Subcommittee Draft Report on the Development of a Practical Quantitation Level for Perfluorooctanoic Acid (PFOA) in Drinking Water (August 29, 2016) at 2, *available at* http://www.nj.gov/dep/watersupply/pdf/testing-subcompql-pfoa-8.29.16KA.pdf.
[17] *Id*. at 8.
[18] *Id*.
[19] Exhibit "A" at 16.
[20] *Id*. at 17.

analytical methods were not a keystone feature of the Rulemaking Petition. However, given that it was actually included in the Rulemaking Petition and the fact that DEP itself was aware of approved analytical methods at least as early as 2009, its assertion that DRN's Rulemaking Petition failed entirely to address that factor is unfounded.

iii. Which Treatment Technologies are Approved?

Next, DEP purportedly accuses DRN of failing to identify which treatment technologies are approved for PFAS in the Rulemaking Petition. DRN would direct DEP to page 11 of the Rulemaking Petition, where it is clearly identified that Horsham Township has undertaken the installation of "granulated activated carbon (GAC) treatment to remove PFC's."[21] As above, the use of GAC is also addressed in the reports compiled by NJDWQI included as Exhibits to the Rulemaking Petition, and the use of GAC as a treatment technology for PFAS has also been known to DEP for some time now.

It was identified by the NJDWQI that:

> PFOA can be removed to levels below the recommended Health-based MCL of 14 ng/L and the recommended PQL of 6 ng/L with treatment technologies, such as granulated activated carbon (GAC) and reverse osmosis. GAC has been successfully installed at New Jersey public water systems to treat PFCs including PFOA. An additional benefit of the treatment technologies used to remove PFOA is that they also remove many other contaminants that may also be present.[22]

Further:

> The Treatment Subcommittee recommended that granular activated carbon or an equally efficient treatment removal technology can be used when PFOA is detected above the recommended MCL, subject to on-site pilot testing performance results, and concluded that the availability of treatment options is not anticipated to be a limiting factor in the development of a recommended MCL for PFOA . . .[23]

In addition to the feature of the GAC treatment technology in the body of the Rulemaking Petition as well as in the Attachments thereto, the use of this technology was also highlighted by DEP itself in a 2018

[21] Exhibit "B" at 11.
[22] Maximum Contaminant Level Recommendation for Perfluorooctanoic Acid in Drinking Water, Basis and Background, New Jersey Drinking Water Quality Institute at page 2 of the Letter submitted to Commissioner Bob Martin by Keith R. Cooper, Ph.D. (June 27, 2016), *available at* http://www.nj.gov/dep/watersupply/pdf/pfoa-recommend.pdf. *See* Exhibit "B" fn. 64.
[23] *Id*. at Executive Summary, page 1.

PFAS Community Engagement Event State Panel Discussion held in Horsham, Pennsylvania. In the DEP section of the presentation, the "Environmental Cleanup Program Actions" portion notes that "[f]or state-led sites, the Program has/will provide private well owners with bottled water, GAC filters, and/or connection to public water."[24] In that same Panel Discussion, Aqua America's Chief Environmental Officer goes on to tout its instrument to detect PFAS and the use of GAC filters as a treatment for PFAS.[25] Finally, in the portion of the Panel Discussion where Warrington Township presents its PFAS Response, it highlights the fact that after public wells were taken offline due to exceedance of EPA Provisional Health Advisory Level, "Granular Activated Carbon treatment of Wells 1, 2, & 6, now installed."[26]

Not only have GAC filtration systems been approved as treatment techniques for PFAS, something DEP was aware of prior to questioning DRN as to "what treatment technologies are approved," but GAC filters are so common in practice that both GAC10 and GAC20 are defined in the Pennsylvania Code under Section 109.1. DRN's Rulemaking Petition goes even further, however, and includes as an attachment the Cambridge Environmental Report, which provides additional information regarding testing and treatment.

> Further research by DRN's experts Cambridge Environmental Consulting conclude that while granulated activated carbon has been highly effective in removing PFCs, the best available and economically achievable technology to remove PFOA from dilute aqueous streams at public water supplies is reverse osmosis. Additionally, the NJDWQI Testing Subcommittee has recommended a practical quantification limit (PQL) of 6 ng/L for PFOA. DRN's experts Cambridge Environmental Consulting (CEC) have reviewed the PQL recommendation from the NJDWQI Testing Subcommittee and conclude that by using the method detection limit (MDL) approach a PQL of 3.0 ppt is achievable and by using the minimum reporting level (MRL) approach to determine a PQL for PFOA, a MRL of 2.0 ppt is achievable.[27]

Treatment and its benefits were further discussed in Section C of the Rulemaking Petition.[28]

Thus, it is clear that DRN's Rulemaking Petition addressed this factor at length, offering not one but two effective treatment methodologies for removing PFAS from drinking water supplies. For DEP to act as

[24] PFAS Community Engagement Event State Panel Discussion, Pennsylvania Department of Environmental Protection (July 25, 2018) at slide 34, *available at* master_combined_horshampresentationsjuly26_0.pdf (epa.gov).
[25] *Id*. at slide 56-57.
[26] *Id*. at slide 82.
[27] Exhibit "B" at 21.
[28] *Id*. at 22.

though it has no idea what treatment techniques are approved for PFAS and to further assert that the Rulemaking Petition failed to address the topic is, at best, disingenuous.

iv. How will Systems Monitor for the Contaminant?

In response to the question of how systems will monitor for PFAS, this is again a question that DEP has both experience and expertise in. First, it was addressed in the body of the Rulemaking Petition by referencing the regulations that govern the Pennsylvania Safe Drinking Water Act. On page four, it's noted that "if the Department has reason to believe a contaminant is present in the public water system and creates a health risk to the users of the public water system, the Department 'may require a public water supplier to conduct special monitoring for an unregulated contaminant.'"[29] The regulations go on to provide that the "Department will provide a schedule for sampling, instructions for sampling methods and handling samples, and analytical procedures to be followed by public water systems to perform special monitoring."[30]

The regulations were written in this way specifically because the legislature entrusted DEP to serve its intended purpose of being the voice of authority and expertise when dealing with a harmful new contaminant. For DEP to turn around and say that DRN should be the one guiding them on how to require public water suppliers to monitor for contaminants is a dereliction of the Department's duty, one which puts thousands of citizens of the Commonwealth at risk.

Further, monitoring for PFAS in public water supply systems was already undertaken in the state of Pennsylvania back in 2013 to 2015. This, of course, was done via the U.S. EPA's Unregulated Contaminant Monitoring Rule 3 water supply sampling. Again, this is a process that DEP was involved in[31], so the Department is clearly aware of the ability to monitor for PFAS and the regulations governing the Pennsylvania Safe Drinking Water Act entrust the Department to establish monitoring schedules and requirements as it sees fit.

[29] Exhibit "B" at 4 (citing 25 Pa. Code § 109.302(c)).
[30] 25 Pa. Code § 109.302(d).
[31] *See* 40 C.F.R. § 141.40(a)(5)(vi).

v. How will Compliance be Determined?

To answer the question of how compliance with a new MCL for PFAS will be achieved, all that needs to be done, yet again, is a quick consultation with the regulations governing the Pennsylvania Safe Drinking Water Act. Generally speaking, public water suppliers are subject to compliance cycles nine years in length "during which public water suppliers shall monitor for contaminants."[32] These compliance cycles are broken down into three, three-year calendar compliance periods each, with the initial compliance period being the first full three-year compliance period during which a public water supply is required to monitor for a contaminant.[33]

Under general requirements, public water suppliers shall "[p]rovide treatment adequate to assure that the public health is protected."[34] Similarly, "[a] public water system shall supply drinking water that complies with the primary MCLs, MRDLs and treatment technique requirements adopted by the EQB under the act.[35] Thus, compliance is achieved when public water suppliers are providing water that assures public health is protected, which is achieved by complying with the MCL established by DEP and adopted by EQB.

Further, in the event that DEP is aware of difficulties in achieving compliance with the MCL, DEP is granted the authority to issue variances upon finding that a "public water system has installed and is using the best treatment technology, treatment methods or other means that the Department in concurrence with the Administrator finds are generally available to reduce the level of the contaminant, and has determined that alternative sources of water are not reasonably available."[36] In this event, the regulations establish that Department will issue a schedule for compliance with the MCL requirement covered by the variance, providing accommodation for up to two years.[37]

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[32] 25 Pa. Code § 109.1.
[33] *Id*.
[34] 25 Pa. Code § 109.4(2).
[35] 25 Pa. Code § 109.202(a)(1).
[36] 25 Pa. Code § 109.901(a)(1)
[37] 25 Pa. Code § 109.908(a)(1); 25 Pa. Code § 109.908(c).

Again, these are all safeguards built into the regulations because of the uncertainties that are inherent in a program as far-reaching and complex as the Pennsylvania Safe Drinking Water Act. The legislators exclusively entrusted DEP with the authorities detailed above because it is the Department that administers the program and is expected to have the expertise necessary to make these determinations. It is completely contrary to the legislative intent for DEP to turn around and require third parties who were not granted these powers by the state, such as DRN, to wield the authority and make decisions that affect the entire Commonwealth.

vi. What are the Costs and Benefits Associated with the MCL?

The Evaluation Report also criticizes the lack of a cost analysis in the Petition.[38] DRN does provide comment on this issue in the Rulemaking Petition:

> The additional step requiring the Department to conduct a cost/benefit study of the proposed MCL for PFOA can be accomplished by the Department recognizing the imminent health consequences of PFOA in the public drinking water supply and prioritizing this work. The Department need only remember that the General Assembly has entrusted it to protect the drinking water supply of the citizens of Pennsylvania and that the Pennsylvania Constitution provides that each citizen has a right to clean and safe drinking water.[39]

Additional discussion of the benefits are in the Rulemaking Petition, including but not limited to: "Benefits include greater protection from disease that is correlated with exposure to PFOA and the multiple benefits of the removal of other potentially dangerous contaminants that are filtered out by the employed treatment technology, specifically through the use of recommended activated carbon filtration."[40] The Rulemaking Petition also points out that people who have been exposed and have PFOA in their body will benefit from removal of the contaminant from drinking water because it will allow PFOA to be slowly excreted, reducing the concentration in their blood, the only way to rid the body of PFOA.[41]

> The Rulemaking Petition goes on to identify ancillary benefits to the environment as well:
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> Delaware River Estuary surface water and fish flesh in the Delaware River Estuary contain concentrations of PFOA and other PFCs. The treatment and removal of PFOA from drinking

[38] Exhibit "A" at 19-20.
[39] Exhibit "B" at 21.
[40] *Id.* at 22
[41] *Id.*

> water will reduce the concentrations and distribution of PFOA, reducing the exposure to wildlife and to humans who consume fish, reducing the population's intake of PFOA-contaminated food.[42]

The Petition also provides evidence in a referenced document about the economic value of clean water. "Improved water quality can also increase the property values of nearby communities"[43] by 6 to 25%, citing EPA and other authorities.[44] This oft-cited reference document was provided with the Rulemaking Petition as Attachment 6.[45] It contains other relevant economic values for water and other natural resources.

Once again, the critique offered by DEP proves to be false and misleading. DRN's Rulemaking Petition clearly provided considerations of costs and benefits both in the body of the Petition as well as in the ample Attachments it included in support of the recommendations contained therein.

c. As DRN has Shown DEP's Reasoning for Dismissing the Rulemaking Petition are Inaccurate, it Should be Reconsidered and the MCL for PFOA Should be 1 ppt, But Should Not Exceed 6 ppt.

DEP dismissed the Rulemaking Petition for its failure to consider additional factors that were listed out in its Evaluation Report. As has been shown in this response, however, the factors listed by DEP have been addressed by DRN extensively, both in the body of the Rulemaking Petition itself as well as in the various Attachments that were included as part of its recommendation. The fact that DEP missed this information, information which was crucial to the thoroughly researched and support recommendation, leads DRN to question the quality and accuracy of the evaluation DEP provided. Thus, DRN asserts that a full consideration of the original Rulemaking Petition is in order. DRN's Rulemaking Petition, which recommends the adoption of an MCL for PFOA of 1 ppt but not to exceed 6 ppt, speaks for itself. The Rulemaking Petition should be read in its entirety, including all of the supporting documents that were

[42] *Id*. (citing Contaminants of Emerging Concern In the Tidal Delaware River, Delaware River Basin Commission (July 2012), *available at* http://www.nj.gov/drbc/library/documents/contaminants-of-emerging-concernAug2013rev.pdf).
[43] Exhibit "B" at 21.
[44] Gerald J. Kauffman, *Socioeconomic Value of the Delaware River Basin in Delaware, New Jersey, New York, and Pennsylvania*, Oct. 2011, at 35, *available at* https://nj.gov/drbc/library/documents/SocioeconomicValueDRB-UDEL-FinalRpt.pdf.
[45] *See* Exhibit "B" fn. 117.

attached, and DEP's critical consideration of the Petition should be reflected in its forthcoming recommendation to the EQB.

As stated previously, DRN agrees that "[t]he setting of an MCL is not as simple as just picking a number."[46] This is one of the reasons DRN submitted the report by Cambridge Environmental Consulting which supports the Rulemaking Petition's MCL for PFOA of 1 ppt, or alternatively, no higher than 6 ppt.[47] The range was provided to allow for the use of DEP's judgment in deciding which studies to rely upon to set the health-based MCL based on lifetime exposure:

> CEC's recommendation of a MCL of 1 ppt is consistent with the values found pursuant to the immunotoxic epidemiologic study and/or animal studies showing adverse developmental effects. However, if these values are excluded, the CEC has identified that the PFOA MCL should be no greater than 6 ppt to assure protection of children.[48]

The CEC report ("the Cambridge Report") is explained in the Rulemaking Petition and the toxicological analysis is transparently disclosed within the report, which was provided as Attachment 2 in the Rulemaking Petition.[49] The evidence to support the most vulnerable of population, children, is also provided. In summary, CEC cites to a different study than the two studies used by NJDWQI as the toxicological basis. Instead, the Cambridge Report cites to a report by Grandjean and Budtz-Jørgensen that represents the greatest sensitivity to PFOA so far studied, un-confounded by exposure to other chemical contaminants.[50] The Cambridge Report stated adequate toxicity data already existed for the more sensitive delayed mammary gland development endpoint, so this endpoint must be used when calculating an MCL, which yielded a proposed MCL for PFOA of 1 ppt.[51]

The Cambridge Report went on to disagree with NJDWQI's use of adult default exposure values because it omits protection for the population's most vulnerable exposure group, children, who have a

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[46] Exhibit "A" at 21.
[47] Fardin Z. Oliaei & Don L. Kriens, T*echnical Analyses of New Jersey Drinking Water Quality Institute—Proposed Health-Based Maximum Contaminant Level (MCL) for Perfluorooctanoic Acid (PFOA) in Drinking Water*, Cambridge Environmental Consulting (Nov. 18, 2016), *available at* https://www.delawareriverkeeper.org/sites/default/files/cvr ltr PFOA mcl cmnt11.19.combinedpdf.pdf.
[48] Exhibit "B" at 19 (citing Oliaei, *supra* note 47, at 3).
[49] *See* Exhibit "B" fn. 63.
[50] Oliaei, *supra* note 47, at 5-6.
[51] *Id.* at 7.

greater rate of food and drinking water consumption based on body weight than adults do.[52] The Cambridge Report asserted that MCL calculations using increased liver weight as an endpoint should, at a minimum, be based on children exposure values for drinking water intakes and body weight. For children aged 1-6, the consultants recommend an MCL of 5.65 ppt, which is where the rounded value of 6 ppt in the Rulemaking Petition was derived.[53]

DEP rejected DRN's Rulemaking Petition because it failed to address a number of factors that DEP listed out. Because it has now been shown that, as a factual matter, the Rulemaking Petition did address these factors, DRN's Rulemaking Petition should be reconsidered as the basis for DEP's recommendation. An MCL for PFOA in Pennsylvania should be set at 1 ppt or, in the alternative, should not exceed 6 ppt.

II. DEP's Proposed MCLG of 8 ppt Does not Rise to the Level Necessary for a Standard Based Exclusively on Public Health Considerations.

As defined under the Federal Safe Drinking Water Act, a maximum contaminant level goal ("MCLG") "means the maximum level of a contaminant in drinking water at which no known or anticipated adverse effect on the health effect of persons served would occur, and which allows an adequate margin of safety. Maximum contaminant level goals are nonenforceable health goals."[54] DEP and U.S. EPA further clarify that "MCLGs consider only public health and not the limits of detection and treatment technology effectiveness. Therefore, MCLGs sometimes are set at levels which water systems cannot meet because of technological limitations."[55] As a result, because the MCLG is purely a goal and aims to be as protective of human health as possible, the calculation of MCLGs should utilize the most conservative and scientifically accurate figures available. The Drexel PFAS Advisory Group ("DPAG") and, by extension, DEP utilized an approach that resulted in a proposed MCLG that is not as protective of public health as it should be.

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[52] *Id*. at 8.
[53] *Id*. at 9-10.
[54] 40 C.F.R. § 141.2.
[55] Exhibit "A" at 19; U.S. EPA, *How EPA Regulates Drinking Water Contaminants*, epa.gov, https://19january2017snapshot.epa.gov/dwregdev/how-epa-regulates-drinking-water-contaminants_.html.

In developing a recommended MCLG for PFOA, the DPAG utilized a similar approach as CEC did to develop the Cambridge Report, the report submitted by DRN in support of its original Rulemaking Petition. That approach involved extrapolating data from existing studies and using that information to calculate an acceptable contaminant level for PFOA. Because both of the approaches involved selecting some studies to use while disregarding others, there is undeniably some level of subjectivity that is inherent in the process. The problem is that the efforts by DPAG, which again were supposed to yield a nonenforceable health goal at which no known or anticipated adverse effect on the health of persons served would occur, resulted in a proposed MCLG that is between 7 ppt and 2 ppt higher than the level proposed in the Cambridge Report. This is especially concerning considering that the development of the enforceable level, the MCL, is based on the MCLG. The ultimate MCL is supposed to be as close as feasible to the MCLG, meaning that it can be equal to or greater than the MCLG, but cannot be lower than the MCLG. To discern how this could happen, it is helpful to look at the way DPAG arrived at its recommendation.

DRN believes the most up-to-date research and analysis being conducted by states and agencies should be considered by DEP in its proposed rulemaking and by DPAG in its research. The final Toxicological Profile for Perfluoroalkyls ("Final Tox Profile") has been published by ATSDR in May of 2021.[56] In reviewing the documents, it appears that there are at least 163 new references to reports and studies utilized by ATSDR between the 2018 draft and the final report. In comparing the reference list of the Final Tox Profile with the reference list provided DPAG, it appears only five of these newly referenced reports were used by DPAG. These reports should be reviewed by DPAG and DEP to ensure that the most up-to-date and comprehensive science is being considered—DEP itself included this charge in its Evaluation Report: "The Department's proposed rulemaking should be based on available data, studies, and science . . .."[57] Thus, by DEP's own standards, the DPAG recommendation falls far short.

[56] Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Perfluoroalkyls*, cdc.gov, https://wwwn.cdc.gov/TSP/ToxProfiles/ToxProfiles.aspx?id=1117&tid=237.
[57] Exhibit "A" at 22.

Further, a critical difference between the variables used in the Cambridge Report to develop the recommended MCL for PFOA and the variables employed by DPAG to develop its recommended MCLG is the use of different studies to identify the critical effect. The Cambridge Report recommended a range between 0.75 ppt (rounded to 1ppt) and 5.65 ppt (rounded to 6ppt) based on using different studies with different critical effects. As stated previously, the proposed MCL of 1 ppt identified delayed mammary gland development in mice as a sensitive endpoint and noted that nine different studies showed this toxicity effect.[58] These studies are discussed in the Cambridge Report. The Cambridge Report also advised that if liver weight was used as an endpoint, as done by the NJDWQI, then the MCL calculation should be based on children exposure values for body weight and drinking water intakes and CEC explained why (ages 1 to 6).[59] This calculation by CEC yielded the proposed MCL of 6 ppt for PFOA.[60]

The DPAG Report, on the other hand, chose studies by Koskela (2016) and Onishchenko (2011) as the basis for their conclusions. These studies identified development effects in mice as the critical effects: "The ATSDR selected identical LOAELs from Onishchenko (2011) and Koskela (2016). Both studies had the same populations of laboratory animals and evaluated a single dosing group. These studies identified developmental effects (neurobehavioral and skeletal) as critical. The DPAG selected Koskela (2016) and Onishchenko (2011) as the critical studies."[61] It is worth noting that only one dose level is provided: .03mg/kg/day.[62]

The DPAG Report also adopted the use of the model used by ATSDR in their 2018 draft report to arrive at the Point of Departure (POD): "From Onishchenko and Koskela, the ATSDR estimated the POD average serum concentration in the mice (8.29 mg/L) using a three-compartment pharmacokinetic model (Wambaugh 2013) using animal species-, strain-, sex-specific parameters. This was adopted by the DPAG

[58] Oliaei, *supra* note 47, at 6.
[59] *Id.* at 8
[60] *Id.*
[61] Exhibit "A", Appendix 1 at 22.
[62] *Id.*

as the POD for PFOA."[63] These reports are discussed in the DPAG Report. It is worth noting that the final ATSDR Tox Profile arrived at a POD average serum concentration of 0.000821, rather than the 0.000829 mg/kg/day concentration that was the value in the ATSDR 2018 draft report.[64] The POD value is used in the calculation of the MCLG and, obviously, it is imperative that this effort reflect the most accurate and up-to-date scientific information available. As such, DEP and DPAG need to update the MCLG calculation in order to reflect the lower POD average serum concentration arrived at by ATSDR.

The sources used by DPAG varied for other variable factors employed to develop their recommended MCLG of 8 ppt. Most important was the use of the Goeden Model: "This resulted in a THSV of 0.028 mg/L for the Goeden Model. Setting the target for the breast fed infant as 0.014 (50%RSC), the MCLG for drinking water is recommended to be 8 ng/L (8PPT) to protect breastfed infants and throughout life."[65] The DPAG Report concludes that young children, including breastfed infants, are protected from adverse health effects at 8ppt, based on the use of the Goeden Model.[66]

The Cambridge Report, however, employing the studies that identify delayed mammary gland development as the endpoint, illustrates that this critical effect yields an MCL of 0.75 ppt, rounded to 1 ppt.[67] DRN supports a standard that is fully protective of the most vulnerable population, breastfed infants and young children, and, based on the Cambridge report, asserts that 1 ppt is required to achieve this. If the endpoint is considered to be liver weight, then the MCL must be no greater than 6 ppt, as concluded in the Cambridge Report. With a maximum difference of 7 ppt and a minimum difference of 2 ppt by two separate entities based on surveying existing peer-reviewed studies, DEP cannot be said to have met the legal standard for setting an MCLG. In other words, the DEP-recommended MCLG of 8 ppt is not the "maximum

[63] *Id.* at 23.
[64] Toxicological Profile for Perfluoroalkyls, Agency for Toxic Substances and Disease Registry, at A-19 (May 2021), *available at* https://wwwn.cdc.gov/TSP/ToxProfiles/ToxProfiles.aspx?id=1117&tid=237.
[65] Exhibit "A", Appendix 1 at 28.
[66] *Id.* at 31.
[67] Oliaei, *supra* note 47, at 7.

level of a contaminant in drinking water at which no known or anticipated adverse effect on the health effect of persons served would occur" and should not be accepted by the EQB.

III. In the Alternative, DEP Must Improve Upon the DPAG Report and Promulgate an MCLG and MCL for PFOA that Adheres to its Constitutional Obligations Under the Environmental Rights Amendment.

a. Although DPAG's Work can Still be Improved, it is Critical for DEP and the EQB to Act to Regulate PFAS in the Face of U.S. EPA Inaction.

DPAG's review of the recommendations and the supporting documents of other agencies was critical to inform DPAG's analysis and ultimately its conclusions and recommendations. DRN agrees with the DPAG's decision to not employ a summative approach and to instead develop an individual MCLG for each PFAS that were charged with studying, including PFOA. There is, however, room for improvement in terms of the work that was done by DPAG.

DRN recommends that DEP include in the review a paper that is not listed in the references for the DPAG Report which provides an overview of state regulation and background materials regarding PFAS: Gloria B. Post, "Recent US State and Federal Drinking Water Guidelines for Per- And Polyfluoroalkyl Substances (PFAS)" ("the Post Paper").[68] DRN also recommends that the Fact Sheets containing the Water and Soil Value Tables produced by the Interstate Technology and Regulatory Council updated in August 2020[69], likewise be consulted. Since, pursuant DEP's standards, the Department's proposed rulemaking should be based on available data, studies, and science, these two resources, which were not available at the time the Workbook was completed, should be consulted by the Department as it prepares its recommendation for the EQB.

In that recommendation it is also critical that DEP explain clearly, for the benefit of the public and all stakeholders, the pressing need for an MCL for PFOA (and other PFAS compounds) to be adopted on a

[68] Gloria B. Post, *Recent US State and Federal Drinking Water Guidelines for Per- and Polyfluoroalkyl Substances*, 40 Environmental Toxicology and Chemistry 550, 553 (2020), *available at* https://setac.onlinelibrary.wiley.com/doi/epdf/10.1002/etc.4863.

[69] Interstate Technology and Regulatory Council, *PFAS Fact Sheets, Tables for Water and Soil Values, available at* https://pfas-1.itrcweb.org/fact-sheets/.

statewide level. It is important that the justification for statewide action be robustly advocated by DEP to raise public understanding of the imperative for action and the benefits of the adoption of an MCL for PFOA and other PFAS. U.S. EPA has not moved forward expeditiously and has employed flawed analysis to arrive at their Health Advisory Level ("HAL") for PFOA and PFOS of 70 ppt, which is not protective of human health. The application of the HAL by Pennsylvania as threshold for action to address PFAS contamination of drinking water sources has left many water users exposed to dangerous concentrations of the contaminants in the water they drink and use every day. This situation has developed into a water crisis in many Pennsylvania communities that must be remedied. However, the adoption of a federal MCL, regardless of whether it is protective or not, is not on the near-term horizon, despite the urgent need.

Action at the federal level has been exceedingly slow and, looking at past performance, may not yield a federally-mandated safe drinking water standard for years to come. EPA's longtime performance regarding setting new MCLs offers a dim view of the likelihood of speedy adoption of MCLs for PFAS anytime in the near future. "There are no national drinking water standards (i.e. Maximum Contaminant Levels; MCLs) for PFAS in the U.S., and no MCLs for new contaminants have been established by the U.S. Environmental Protection Agency (EPA) under the lengthy and complex process for national regulation of new drinking water contaminants established by legislation in 1996."[70] This is why nine other states, as of May 2020, have turned to state drinking water laws and related environmental and public health regulations in order to protect their citizens. All nine states to consider the EPA Lifetime HAL have found it fails to be sufficiently protective and have developed more stringent drinking water standards or guidance values for PFOA and PFOS.[71]

There are several reasons for stricter standards and guidelines in current state PFAS regulation. One is the increase in studies available to scientists to develop risk assessments.[72] Another reason is the

[70] Post, *supra* note 68, at 551.
[71] *Id*. at 553.
[72] *Id*. at 552 (". . . drinking water guidelines for PFOA and PFOS have decreased by orders of magnitude since the early 2000s. These decreases in guideline values over time are due to both the emergence of new health effects information and newer interpretations of the information that was available when the older guidelines were developed.").

recognition that people are exposed to many sources of PFAS such as consumer products, in addition to drinking water, that result in most people having PFAS in their blood despite not being exposed via their drinking water. This calls for a standard that reflects the fact that exposure through drinking water is only one of many cumulative sources adding to the background levels of PFAS in an individual's body, which can make even very small amounts of PFAS in drinking water that much more dangerous.

Another reason for stricter, i.e., lower, standards is that the reference dose used by U.S. EPA in developing the HAL is higher than what most states have used. The different reference doses used result in different outcomes in terms of MCLs or MCLGs. While state reference doses for PFOA have ranged from 1.5 to 18 ng/kg/day, U.S. EPA's reference dose is 20 ng/kg/day.[73] "All of the state [r]eference [d]oses consider toxicological effects that are more sensitive and/or judged to be more appropriate than the developmental effects (delated ossification, accelerated puberty) used by the USEPA, either as the critical effect or through the application of an uncertainty factor for database limitations."[74] Further, "several states concluded that an uncertainty factor is needed to account for delayed mammary gland development and other low-dose developmental effects (persistent liver toxicity, neurobehavioral effects, persistent skeletal changes) of PFOA."[75] This is in contrast to U.S. EPA, who "dismissed delayed mammary gland development from consideration in risk assessment for reasons [ ] *that do not appear to have a valid scientific basis and/or apply equally to the endpoints that are the basis for the USEPA Reference Dose*."[76]

Thus, it is imperative that DEP and the EQB establish regulations for PFAS, including PFOA, in our drinking water. It is clear that U.S. EPA has not taken the action necessary to protect the public from ingesting harmful contaminants, and what little action it has taken has been woefully uninformed and utilizes dated methods of analysis. The responsibility thus falls upon the state to step in to fill that role. In order to do so efficiently and effectively, however, DEP must continue to consider the most up-to-date

[73] *Id*. at 555.
[74] *Id*.
[75] *Id*. at 557.
[76] *Id*.

scientific information available including the sources recommended by DRN in this subsection. Not only will that ensure the most protective standards possible for the citizens of the Commonwealth, but the work of DEP may also help guide U.S. EPA when it finally acts to address PFAS.

b. DEP is Constitutionally Constrained in its Ability to Lessen the MCLG to Establish an MCL in a way that U.S. EPA is not.

While DEP listed out a variety of considerations to which they were beholden in the process of establishing an MCL/MCLG for PFOA, including the Federal Safe Drinking Water Act, Pennsylvania State Drinking Water Act, and the Regulatory Review Act, the Department failed to include the most controlling, and limiting, consideration at play: the Environmental Rights Amendment of the Pennsylvania Constitution.

Pennsylvania is one of the few states that had the forethought to enshrine an Environmental Rights Amendment in its Constitution. The environmental rights reserved in that provision are on par with other fundamental and dearly held civil rights.[77] Specifically, Article I Section 27 of the Pennsylvania Constitution states:

> The people have a right to clean air, pure water, and to the preservation of the natural, scenic, historic and esthetic values of the environment. Pennsylvania's public natural resources are the common property of all the people, including generations yet to come. As trustee of these resources, the Commonwealth shall conserve and maintain them for the benefit of all the people.[78]

"The first right is contained in the first clause, which is a prohibitory clause declaring the right of citizens to clean air and pure water, and to the preservation of natural, scenic, historic and esthetic values of the environment."[79] This clause "places a limitation on the state's power to act contrary to this right."[80] According to the Pennsylvania Supreme Court, the Commonwealth, as trustee, is a fiduciary with the obligation to comply with the terms and standards established in the trust:

> The explicit terms of the trust require the government to "conserve and maintain" the corpus of the trust. The plain meaning of the term conserve and maintain implicates a duty to

[77] *Robinson Twp., Washington County v. Comm.*, 83 A.3d 901, 947-48 (Pa. 2013) (citing Pa. Const. Art. I, Preamble; Pa. Const. Art. I, § 25; *see also* Pa. Const. art. I, § 2 ["Article I is the Commonwealth's Declaration of Rights, which delineates the terms of the social contract between the government and the people that are of such 'general, great and essential' quality as to be ensconced as 'inviolate.'"]).
[78] Pa. Const. Art. I, § 27 (the "Environmental Rights Amendment" or the "ERA").
[79] *Pennsylvania Envtl. Def. Found. V. Comm.*, 161 A.3d 911, 931 (Pa. 2017) (citing *Robinson Twp.*, 83 A.3d at 951).
[80] *PEDF*, 161 A.3d at 931.

> prevent and remedy the degradation, diminution, or depletion of our public natural resources. As a fiduciary, the Commonwealth has a duty to act towards the corpus of the trust—the public natural resources—with prudence, loyalty, and impartiality.[81]

Under the Supreme Court's holding, there are two basic duties on the Commonwealth as a trustee. "First, the Commonwealth has a duty to prohibit the degradation, diminution, and depletion of our public natural resources, whether these harms might result from direct state action or from the actions of private parties. Second, the Commonwealth must act affirmatively via legislative action to protect the environment."[82] There exists no similar constitutional provision, law, or regulation at the federal level. This means that while DEP is correct in that it must make the same considerations as U.S. EPA in setting an MCL under the Federal Safe Drinking Water Act, it must also adhere to constitutional obligations that U.S. EPA does not have.

These requirements must be in the forefront of DEP's mind while it makes its MCLG recommendation to the EQB. Once the MCLG has been established, DEP will need to establish an enforceable standard, the MCL. At the federal level, EPA then takes cost into consideration through preparing a health risk reduction and cost analysis in support of any standard.[83] "Where the benefits of a new MCL do not justify the costs, EPA may adjust the MCL for a particular class or group of systems to a level that maximizes health risk reduction benefits at a cost that is justified by the benefits."[84] For DEP, however, although cost is a consideration that may be taken in setting an MCL, it has a constitutional obligation to take affirmative action to protect the Commonwealth's right to pure water. Thus, the affirmative duty to protect the environment and prohibit the diminution of our public natural resources is a factor that must be given greater weight in setting an MCL than the weight afforded to the cost of the regulation. As a result, DEP has an even greater obligation to protect the environment than U.S. EPA does, and the deliberative process in setting an MCL for PFOA must reflect that.

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[81] *Id*. at 932 (citations omitted).
[82] *Id*. at 933 (citations omitted).
[83] Exhibit "A" at 20.
[84] *Id*.

Respectfully submitted,

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Date: May 16, 2021

EXHIBIT A

PENNSYLVANIA DEPARTMENT OF ENVIRONMENTAL PROTECTION

EVALUATION REPORT

ON THE

DELAWARE RIVERKEEPER NETWORK PETITION FOR RULEMAKING

TO SET AN MCL FOR PFOA

April 16, 2021

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A. DESCRIPTION OF THE PETITION FOR RULEMAKING PROCEDURE

Any person may petition the Environmental Quality Board ("EQB") to initiate a rulemaking proceeding for the issuance, amendment, or repeal of a regulation administered and enforced by the Pennsylvania Department of Environmental Protection ("Department"). 71 P.S. § 510-20(h). The EQB has developed a policy for processing petitions for rulemaking. *See* 25 Pa. Code Chapter 23 (relating to Policy for Processing Petitions – Statement of Policy). Among other things, a petition for rulemaking must contain the following information: (1) the petitioner's name, address, and telephone number; (2) a description of the action requested including suggested regulatory language if the petition requests the EQB to adopt or amend regulations; (3) the reason the petitioner is requesting the action from the EQB; and (4) the types of persons, businesses, and organizations likely to be impacted by the proposal. 25 Pa. Code § 23.1 (relating to Petitions). When a petition for rulemaking is submitted, the Department examines the petition before it is submitted to the EQB to determine if it meets the following conditions: (1) the petition is complete as required by § 23.1; (2) the petition requests an action that can be taken by the EQB; and (3) the requested action does not conflict with Federal law. 25 Pa. Code § 23.2 (relating to Departmental review).

The Department then notifies the EQB and the petitioner of its determination. If the Department determines that the petition is not appropriate, the notification will state why and give the petitioner 30 days to modify the request. 25 Pa. Code § 23.3 (relating to Notification).

Where the Department determines that a petition is appropriate, the petitioner may make a five-minute presentation to the EQB and the Department will also make a recommendation as to whether to accept the petition. 25 Pa. Code § 23.4 (relating to Oral presentation).

The EQB may refuse to accept a petition if: (1) the EQB has within the past two years considered the issue addressed in the petition; (2) the action requested by the petitioner is currently under litigation; (3) the requested action is inappropriate for policy or regulatory considerations; or (4) the petition involves an issue previously considered by the EQB, and it does not contain information that is new or sufficiently different to warrant reconsideration of that issue. 25 Pa. Code § 23.5 (relating to Board determination).

If the EQB accepts the petition, a notice of acceptance will be published in the *Pennsylvania Bulletin* and a report will be prepared. 25 Pa. Code § 23.6 (relating to Notice of acceptance and Department report).

Once the report is completed, the Department will send a copy of it to the petitioner who may then submit to the Department a written response to the report within 30 days of the mailing of the report. 25 Pa. Code § 23.7 (relating to Response to report).

The Department will prepare a recommendation to the EQB based on the report and comments received from the petitioner. If regulatory amendments are recommended, the Department will develop a proposed rulemaking for EQB consideration within 6 months after the Department mailed its report to the petitioner. If regulatory amendments are not recommended, the Department will present its recommendation and basis to the EQB at the first meeting occurring at least 45 days after the Department mailed its report to the petitioner. 25 Pa. Code § 23.8 (relating to Board consideration).

B. DESCRIPTION OF THE DELAWARE RIVERKEEPER NETWORK PETITION

1. Procedural Description

On May 8, 2017, the EQB received a petition to promulgate a rule to set a drinking water maximum contaminant level (MCL) for perfluorooctanoic acid (PFOA) not to exceed 6 parts per trillion (ppt or nanograms per liter (ng/L)).

The petition was submitted by Tracy Carluccio, Deputy Director on behalf of the Delaware Riverkeeper Network (DRN), 925 Canal Street, Suite 3701, Bristol, PA 19007.

On June 22, 2017, the Department sent a letter to Ms. Carluccio that notified DRN that the petition met the established criteria in Section 23.2 of the EQB's petition policy. The letter also set August 15, 2017 as the date the EQB would consider the petition.

At the August 15, 2017 EQB meeting, Ms. Carluccio, on behalf of DRN, made a brief presentation as to why the EQB should accept the petition for further study. The Department recommended that the EQB accept the petition for further study. The EQB voted unanimously to accept the petition for further study.

On August 26, 2017, the Department published a notice of acceptance of the petition in the *Pennsylvania Bulletin*. *See* 47 Pa.B. 4986 (August 26, 2017).

2. Petition Description

The petition asserts that the EQB should promulgate a rule "to set an MCL for PFOA not to exceed 6 ppt." In support of this petition, Ms. Carluccio, on behalf of DRN, cites PFOA monitoring data from the U.S. Environmental Protection Agency's (EPA) Unregulated Contaminant Monitoring Rule 3 (UCMR 3), 77 FR 26072 (May 2, 2012), information and data from several contamination sites in Bucks and Montgomery counties and other sites across the

state, and scientific studies and reports to support the conclusions that PFOA is in many public water systems in Pennsylvania, that the EPA's Health Advisory Level (HAL) of 70 ppt is ineffective at protecting public health, and that a more protective standard not to exceed 6 ppt should be set for PFOA to protect Pennsylvania citizens. *See* Petition, p. 15. *Please Note: No suggested regulatory language was provided by DRN.*

C. DEPARTMENT RESPONSE TO THE PETITION

1. PFOA

PFOA is a man-made chemical in a large family of chemicals called per- and poly-fluoroalkyl substances (PFAS), which are used to make products more resistant to stains, grease, and water. Major U.S. manufacturers voluntarily agreed to phase out production of PFOA by the end of 2015. However, exposure remains possible due to its widespread use and legacy in the environment from former manufacturing sites and sites where PFOA was used. PFOA has been found in both groundwater and surface water in Pennsylvania and across the country. PFOA is a concern because it readily dissolves in water, bioaccumulates, and is persistent in the environment.

The Department became aware of PFOA detections in public water systems as a result of EPA's UCMR 3 rule. The Federal Safe Drinking Water Act (Federal SDWA) requires EPA to establish criteria for a program to monitor not more than 30 unregulated contaminants every 5 years. The purpose of the rule is to gather occurrence data and refine analytical methods in order to inform a regulatory determination. Monitoring for 28 chemicals and two viruses was conducted by select public water systems (those serving greater than 10,000 people and a random selection of smaller systems) from January 2013 through December 2015. This included 175 public water systems in Pennsylvania. The UCMR rules are direct implementation rules with EPA as the lead agency and states providing assistance. Six (6) out of 175 public water systems had detections for PFOA:

- Warminster Municipal Authority
- Warrington Township Water & Sewer Department
- Horsham Water & Sewer Authority
- United Water -- Harrisburg (now Suez)

- Doylestown Township Municipal Authority
- Aqua PA – Bristol

2. Status of an MCL for PFOA

The Department is authorized to administer and enforce environmental regulations under the Pennsylvania Safe Drinking Water Act (Pennsylvania SDWA), 35 P.S. § 721.5. The EQB is authorized to adopt such rules and regulations, governing the provision of drinking water to the public, as it deems necessary for the implementation of the Pennsylvania SDWA, 35 P.S. § 721.4. Under the SDWA, an MCL is defined as the maximum permissible level of a contaminant in water which is delivered to any user of a public water system.

The Federal SDWA authorizes EPA to set national health-based standards to protect against contaminants that may be found in drinking water, 42 U.S.C. § 300g-1. Under the Federal SDWA, EPA promulgates primary MCLs, which are enforceable standards. EPA may also publish health advisories, which are non-enforceable and non-regulatory, for contaminants not subject to any national primary drinking water regulation. The Federal SDWA grants States primary enforcement responsibility (primacy) for public water systems when EPA determines that a State meets certain requirements, including adopting drinking water regulations that are no less stringent than the national primary drinking water regulations promulgated by EPA, 42 U.S.C. § 300g-2.

The Pennsylvania SDWA was enacted in 1984. The Pennsylvania SDWA imposed a mandatory duty upon the Department to adopt a public water supply program that includes certain program elements necessary to assume primacy under the Federal SDWA, including MCLs. The Department established a public water supply program that met the criteria and was granted primacy by EPA on November 30, 1984. 50 FR 342 (January 3, 1985).

The Pennsylvania SDWA provides direction regarding how MCLs are to be developed, 35 P.S. § 721.4(a). Under the Pennsylvania SDWA, the EQB *shall* adopt MCLs no less stringent than those promulgated under the Federal SDWA for all contaminants regulated under the national primary drinking water regulations. In addition, the EQB *may* adopt MCLs for any contaminant that an MCL has not been promulgated. EPA has not promulgated an MCL for PFOA under the national primary drinking water regulations. EPA has published a health advisory for PFOA, which established a combined lifetime HAL of 70 ppt for PFOA and perfluorooctanesulfonic acid (PFOS). 81 FR 33250 (May 25, 2016).

As referenced above, the Petition for Rulemaking was presented at the August 15, 2017 EQB meeting, at which the Department recommended that the EQB accept the petition for further evaluation to help inform whether additional measures are needed to protect public health. During the meeting, the Department stated that it had never in its history set an MCL and would require toxicology expertise to evaluate the rulemaking petition and prepare the report. It was expected that this would require independent work, research, and review. The Department provided updates to the EQB on June 19, 2018 and June 18, 2019, where the Department expressed the need for more time and provided a summary of the challenges and actions taken to secure the necessary expertise to evaluate the rulemaking petition and prepare this report. These and other actions taken by the Department to address PFOA are described below in Section 3.

3. Department actions to address PFOA

a. Actions to implement EPA's HAL as an interim measure

Following EPA's publication in May 2016 of the final HAL of 70 ppt for the combined concentration of PFOA and PFOS, the Department developed its strategy in July 2016 for

addressing PFOA and PFOS levels in public water systems that exceed the HAL. The Department's strategy is based on existing authority and long-standing policies and procedures for implementing HALs. The Department's authority to address unregulated contaminants includes the following:

- Pennsylvania SDWA, Section 10. Emergencies and imminent hazards.

(b) Department may order temporary emergency actions.—The department, upon receipt of information that a contaminant which is present in or is likely to enter a public water system may present an imminent and substantial risk to the health of persons, may take or order a public water system to take such temporary emergency actions as it deems necessary in order to protect the health of such persons. The department may assess the responsible water supplier with costs of temporary actions taken by the department, except where such action is in the normal course of its duties.

(c) Department may implement emergency measures.—The department shall be authorized to implement whatever measures may be necessary and appropriate to notify the public of an emergency or imminent hazard and to assess costs of notification on the responsible water supplier.

- Title 25 Pa. Code § 109.4. General requirements.

Public water suppliers shall:

(1) Protect the water sources under the supplier's control.

(2) Provide treatment adequate to assure that the public health is protected.

(3) Provide and effectively operate and maintain public water system facilities.

(4) Take whatever investigative or corrective action is necessary to assure that safe and potable water is continuously supplied to the users.

- Title 25 Pa. Code § 109.302. Special monitoring requirements.

(b) The Department may require a public water supplier to conduct additional monitoring to provide information on contamination of the water supply where a potential health hazard may exist in the water supply and monitoring required under § 109.301 may not be adequate to protect the public health.

(c) The Department may require a public water supplier to conduct special monitoring for an unregulated contaminant if the Department has reason to believe the contaminant is present in the public water system and creates a health risk to the users of the public water system.

The Department's long-standing risk management strategy for unregulated contaminants can be found in the following guidance: *Health Effects and Risk Management Guidance* (383-0400-104).

As per the guidance and long-standing protocols, when levels exceed a lifetime HAL, a Tier 2 situation has occurred. Water supplier follow-up actions may include:

- One-hour reporting of sample results to the Department (25 Pa. Code § 109.701(a)(3)) to ensure the Department is immediately alerted to the situation and can provide the necessary oversight regarding investigative and corrective actions
- Collection of confirmation samples (25 Pa. Code § 109.302(c))
- Issuance of Tier 2 Public Notification (PN) within 30 days of receipt of sample results exceeding the HAL (25 Pa. Code § 109.409)
- Quarterly monitoring at each entry point (EP) to the distribution system that exceeded the HAL (25 Pa. Code § 109.302(d)) to continue to track contaminant levels

- If levels continue to exceed the HAL, additional actions may be needed to reduce levels to below the HAL (taking contaminated sources off-line, blending, installing treatment, etc.) (25 Pa. Code § 109.4)

Taken together, these actions implemented EPA's HAL prior to submission of the petition, and served as an interim measure while the Department evaluated whether the HAL is sufficiently protective.

b. Toxicology services contract

At the time of submission of the petition, neither the Department nor the Pennsylvania Department of Health (DOH) employed a full-time toxicologist. The DOH had access to a retired toxicologist on a very limited basis (90 days per year) as an annuitant. The DOH recognized the need to hire one or more full time toxicologists and initiated the hiring process in late 2017. The DOH began interviewing candidates in January of 2018, but had difficulty filling the position for various reasons. The DOH was finally able to fill the toxicologist position in July of 2019.

While the DOH was working to fill the toxicologist position, the Department moved forward in early 2019 with plans to secure additional toxicology resources to assist in evaluating the petition. The Department developed a scope of work and began soliciting interest in a toxicology services contract in May of 2019. The Department reviewed the submitted quotes for services in July of 2019 and awarded the contract to Drexel University. The contract was finalized and executed in December of 2019. The contract was for a one-year period and included: (1) a review and analysis of work by other states and federal agencies that had developed PFAS action levels and MCLs; and (2) an independent review of the data, science, and studies, and development of recommended maximum contaminant level goals (MCLG) for select PFAS. MCLGs are non-

enforceable as they are developed solely based on health effects and do not take into consideration other factors, such as limitations with analytical methods and available treatment technologies and cost. MCLGs are the starting point for determining MCLs. Please refer to Section D.2. for more information about MCLGs and the process to set MCLs.

The scope of work included the review of several PFAS in addition to PFOA to provide the Department with more complete health effects information for additional PFAS of concern, to better position the Department to address co-occurring PFAS, to align with state sampling efforts, and to create efficiencies in evaluating multiple PFAS simultaneously. The additional PFAS include PFOS, perfluorobutane sulfonic acid (PFBS), perfluorononanoic acid (PFNA), perfluorohexanesulfonic acid (PFHxS), and perfluoroheptanoic acid (PFHpA). The contract continued throughout 2020, with Drexel providing updates to Department and DOH staff every few months. The project experienced some delays due to the COVID-19 pandemic. The project deliverables were completed and submitted to the Department at the end of January 2021. The deliverables include the "Drexel PFAS Workbook", which contains the review and analysis of work by other states and federal agencies, and the "MCLG Drinking Water Recommendations for PFAS in the Commonwealth of Pennsylvania" report. These documents are included in the Appendix to this report. Here is a brief summary of Drexel's report.

Drexel's MCLG Drinking Water Recommendations for PFAS Report: The report was developed by the Drexel PFAS Advisory Group (DPAG), which is a unique multidisciplinary team consisting of experts in the fields of medical toxicology, epidemiology, environmental toxicology, drinking water standards, and risk assessment. The DPAG evaluated existing and proposed standards from across the country. The DPAG was also charged with developing recommended

MCLGs. In order to do this, the DPAG reviewed the pertinent literature and work done across the country, and independently developed recommended MCLGs.

As mentioned previously and as further discussed in the report, MCLGs are non-enforceable as they are developed solely based on health effects and do not take into consideration other factors, such as limitations with analytical methods and available treatment technologies and cost. MCLGs are the starting point for determining MCLs. The DPAG's recommended MCLG for PFOA is 8 ppt. The DPAG conducted a literature search and review of the available evidence and recommendations from various agencies and developed an MCLG recommendation based on Non-Cancer endpoints. The report includes a discussion of the relevant inputs. The DPAG selected Koskela (2016) and Onishchenko (2011) as the critical studies. Table 1 below represents DPAG's development of the Non-Cancer MCLG for PFOA.

Table 1. The Drexel PFAS Advisory Group's development of the Non-Cancer MCLG for PFOA

PFOA	
Dose Response Modeling Method	**LOAEL**
POD	The average serum concentration was estimated in the mice (8.29 mg/L) using a three-compartment pharmacokinetic model (Wambaugh et al. 2013) using animal species, strain, sex-specific parameters. (ATSDR 2018)
HED = POD x DAF (mg/kg/d)	DAF = Ke x Vd Ke = 0.000825175 (8.2×10^{-4}) based on a human serum half-life of 840 days (Bartell et al. 2010) Vd = 0.17 L/kg (Thompson et al. 2010) $HED_{LOAEL} = POD_{LOAEL} \times DAF$ $HED_{LOAEL} = POD_{LOAEL} \times Ke \times Vd$ $HED_{LOAEL} = 8.29$ mg/L x 0.0000825175 x 0.17 L/kg $HED_{LOAEL} = 0.001163$ mg/kg/d or 1.163×10^{-3} mg/kg/d
Uncertainty Extrapolation	
Human Variability (UFH)	10 (standard)
Animal to Human (UFA)	3 (DAF applied)
Subchronic to Chronic (UFS)	1 (Chronic effect studied)
LOAEL to NOAEL (UFL)	10 (standard)
Database (UFD)	1
Total Composite (UFT)	300
RfD = HED/UFT (mg/kg/d)	RfD = 0.001163 mg/kg/d/300 RfD = 3.9 ng/kg/day (3.9×10^{-6} mg/kg/d)
THSV = POD / UFT	THSV= 8.29 mg/L/ 300 THSV= 0.028 mg/L
Receptor	Infant exposure via breastmilk for 1 year, from mother chronically exposed via water, followed by lifetime of exposure via drinking water. Protective for short-term, subchronic and chronic. (also protective of formula fed infant). Goeden Model Parameters: Placental transfer of 87% and breastmilk transfer of 5.2% (MDH (2020 PFOA)). The Human Serum half-life is set at 840 days (Bartell et al. 2010). The Volume of distribution of 0.17 L/kg (Thompson et al. [2010]) Other factors include, 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 is used to calculate maternal serum concentration at delivery. (Goeden et al. [2019]) A Relative Source Contribution of 50% (0.5) is applied and based on studies which showed that infants RSC is similar to NHANES 95th percentiles for 3-11 (2013-2014) and over 12 years old (2015-2016) participants. (CDC 2019)
Chronic Non-Cancer MCLG	The model produces a Chronic Non-Cancer MCLG of 8 ng/L (ppt). This protects health during the growth and development of a breast fed infant. Figure 2

c. PFAS sampling plan

During this same time period, the Department announced it would begin sampling for PFAS at public water systems across the state. The PFAS Sampling Plan was developed in early 2019 and the final plan was posted to the Department's PFAS webpage in April of 2019.

The PFAS Sampling Plan is intended to prioritize sites for PFAS sampling and generate statewide occurrence data. Several factors were considered in developing the plan including:

- Location of potential sources of PFAS contamination (PSOC)
- Known locations of PFAS contamination
- Relative risk to users of nearby public water system sources of drinking water
- Selection of public water system sources to serve as a control group
- Available funds - $500,000

The selection process involved a combination of spatial analysis and programmatic review. The spatial analysis included the creation of a Geographic Information System (GIS) project using ArcMap 10.4.1 that focused on public water system source locations and information about PSOCs. The sampling pool was prioritized based on relative risk and included community water systems and nontransient noncommunity water systems.

In order to prioritize sampling, the selection process included an assessment of the potential risk from nearby PSOCs. Several layers containing locational and other information specific to PSOCs were created or otherwise included in the GIS. These layers include the following industries and land uses:

- Military bases
- Fire training schools/sites
- Airports
- Landfills
- HSCA sites
- Superfund sites

- Manufacturing facilities:
  - Apparel and other products made from fabrics
  - Chemicals
  - Electronic and electrical equipment
  - Fabricated metal products
  - Paper products
  - Plastic products
  - Textile and leather products
  - Upholstered furniture

Based on the compilation of PSOCs, the information was used to select public water system sources that are located within ½ mile of a PSOC. The targeted sample pool included approximately 493 public water system sources. A second query was performed to identify baseline sources to serve as a control group. Baseline sources are located in a HUC-12 watershed (a watershed assigned a 12-digit hydrologic unit code, or HUC, by the U.S. Geological Survey) with at least 75% forested land and at least five miles from a PSOC. Figure 1 is a map of the pool of public water system sources for sampling.

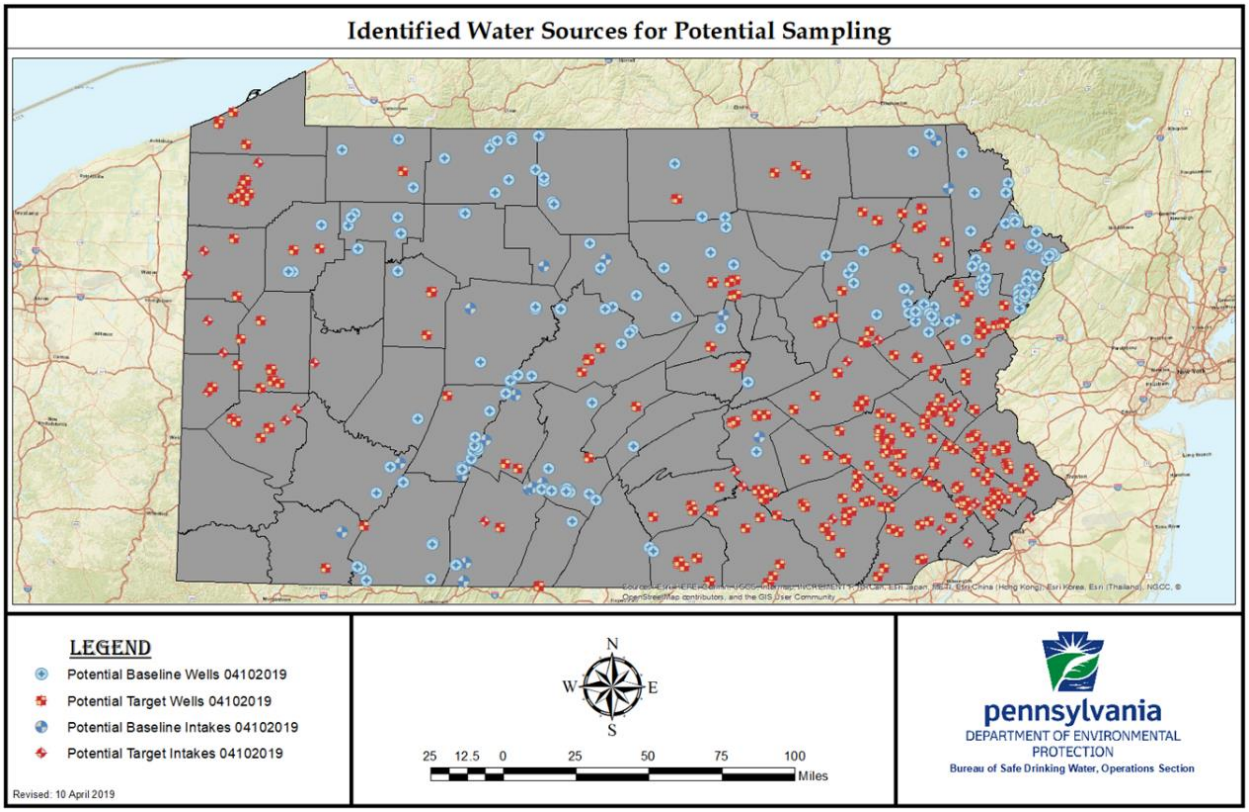

Figure 1. Public water system sources identified for sampling.

The Sampling Plan also includes maps of the various GIS data layers of PSOCs. Figure 2 is an example of the map of industrial sites.

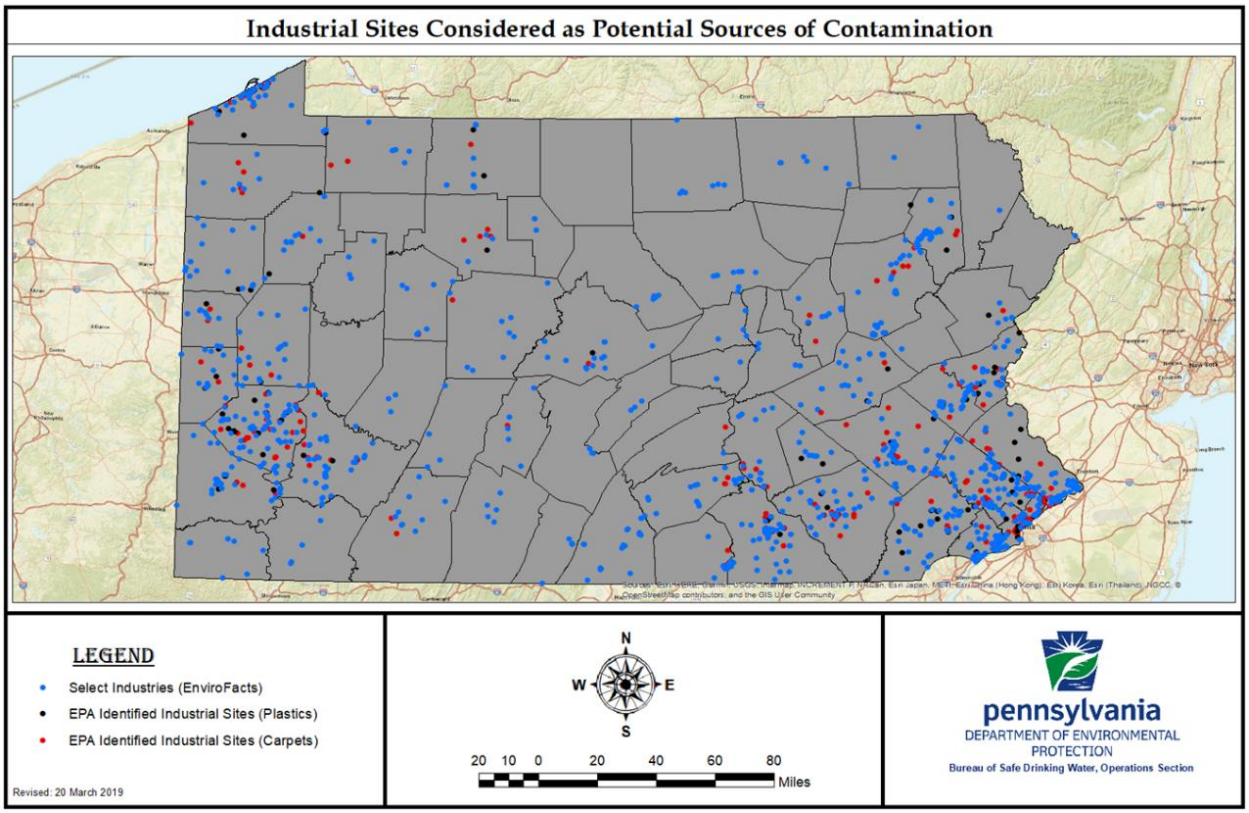

Figure 2. Potential sources of PFAS contamination (PSOC).

The final plan included the collection of samples from 360 targeted public water system sources and 40 baseline sources for a total of 400 samples. Sampling began in June of 2019 and included analysis of six (6) PFAS (PFOS, PFOA, PFNA, PFHxS, PFHpA, and PFBS) to be consistent with EPA's UCMR 3. However, the Department had the opportunity in 2020 to expand the sampling to 18 PFAS by using EPA Method 537.1. Sampling was repeated for the public water systems that were sampled in 2019, and sampling continued for the remainder of the water systems throughout 2020. Note that sampling was halted in March of 2020 due to the pandemic and stay-at-home orders. Sampling resumed in August of 2020 under an approved return to work plan with

appropriate health and safety measures. The first release of 2020 sample results was posted to the Department's PFAS webpage on March 12, 2021 and included 114 samples collected from February through September 2020. Here is the link: Statewide_Sampling_Plan_2020_Results.

Sampling was completed by the end of March 2021. However, results for approximately 20 samples are still pending, and the review of quality assurance data for other recently reported results is ongoing. Table 2 presents a brief summary of the PFOA sample results to date (Note: The Department anticipates that all results will be received and confirmed in time to include a complete summary of PFOA samples in the final report presented to the EQB):

Table 2. Summary of PFOA sample results to date

	PFOA	Units
Average	3.2	ng/l
Median	ND	ng/l
Minimum	ND	ng/l
Maximum	59.6	ng/l
# Detects	40	
Average Detect Value	9.0	ng/l
Median Detect Value	6.5	ng/l
Min Detect Value	4.0	ng/l
Max Detect Value	59.6	ng/l

d. BOL PFAS analytical capabilities

The Department's Bureau of Laboratories (BOL) also worked to purchase and install lab equipment to conduct PFAS testing. BOL was able to achieve proficiency for EPA Method 537.1 and received accreditation from New Jersey in December of 2019. BOL was instrumental in assisting with completing the work under the PFAS Sampling Plan.

D. DEPARTMENT ANALYSIS OF THE PETITION FOR RULEMAKING

1. The Petition Contends that an MCL should be set for PFOA not to exceed 6 ppt

DRN contends that EPA's HAL of 70 ppt has been shown to be ineffective at protecting the public health. Petition p. 2. DRN references two studies and reports to support this: the New Jersey Drinking Water Quality Institute (NJDWQI) report and the Cambridge Environmental Consulting (CEC) study. Petition p. 15.

According to DRN, the NJDWQI transmitted to the New Jersey Department of Environmental Protection its recommendation of an MCL for PFOA of 14 ppt. And while DRN referenced the NJDWQI work as supportive of its conclusion, it also stated that NJDWQI's recommendation may not be protective enough.

DRN also referenced a report prepared by CEC of an evaluation of the NJDWQI work. The CEC study disagreed with several of NJDWQI's findings and concluded that the proposed drinking water MCL for PFOA of 14 ppt is not adequately protective of all population segments. Instead, the CEC study recommended that the proposed MCL for PFOA should be lowered to 1 ppt, or alternatively, should be no higher than 6 ppt. Petition p. 19.

2. Recommendation

The Petition for Rulemaking recommends that the EQB should promulgate a rule to set an MCL for PFOA not to exceed 6 ppt. Petition p. 18. However, DRN fails to recognize the process that the Department must follow when setting an MCL. Specifically, the Department must consider other factors in addition to health effects when proposing an MCL as required by the Federal SDWA and Pennsylvania's Regulatory Review Act (RRA), 71 P.S. §§ 745.1—745.15.

Among other things, the Department must consider technical limitations such as available analytical methods and detection and reporting limits, treatability of the contaminant and available treatment technologies, and costs. 71 P.S. § 745.5b.

In addition to state requirements, the Department needs to consult the Federal SDWA and its implementing regulations. *See* 42 U.S.C. §§ 300f—300j-9; *see also* 40 CFR Parts 141, 142, and 143. For example, within the definitions in the Federal SDWA:

- "MCLG" means the maximum level of a contaminant in drinking water at which no known or anticipated adverse effect on the health of persons served would occur, and which allows an adequate margin of safety. MCLGs are non-enforceable health goals.
- "MCL" means the maximum permissible level of a contaminant in water which is delivered to any user of a public water system.

EPA further explains the difference between MCLGs and MCLs and how the agency sets standards at the following link: www.epa.gov/sdwa/how-epa-regulates-drinking-water-contaminants. In establishing an MCL, the Department would also be informed by EPA's procedure to establish an MCL as detailed below. It is important to understand the process of setting an MCL because similar criteria are required of the Department under the RRA. In addition, in order to retain primacy, the Department's standard setting process would need to be as stringent as the federal process.

After reviewing health effects data, EPA sets an MCLG. MCLGs are non-enforceable public health goals. MCLGs consider only public health and not the limits of detection and treatment technology effectiveness. Therefore, MCLGs sometimes are set at levels which water systems cannot meet because of technological limitations.

Once the MCLG is determined, EPA sets an enforceable standard. In most cases, the standard is an MCL. The MCL is set as close to the MCLG as feasible. Taking cost into consideration, EPA must determine the feasible MCL. This is defined by the Federal SDWA as the level that may be achieved with:

- use of the best available technology or treatment approaches
- other means which EPA finds are available (after examination for efficiency under field conditions, not solely under laboratory conditions)

As a part of the rule analysis, the Federal SDWA also requires EPA to prepare a health risk reduction and cost analysis in support of any standard. EPA must analyze the quantifiable and non-quantifiable benefits that are likely to occur as the result of compliance with the proposed standard. EPA must also analyze certain increased costs that will result from the proposed drinking water standard. In addition, EPA must consider:

- Incremental costs and benefits associated with the proposed and alternative MCL values
- The contaminant's adverse health effects on the general population and sensitive subpopulations
- Any increased health risk to the general population that may occur as a result of the new MCL
- Other relevant factors such as data quality and the nature of the risks

Where the benefits of a new MCL do not justify the costs, EPA may adjust the MCL for a particular class or group of systems to a level that maximizes health risk reduction benefits at a cost that is justified by the benefits.

The setting of an MCL is not as simple as just picking a number. MCL rules must include the necessary provisions to define applicability, the means to comply, and how compliance will be determined. For example, which water systems must comply with the MCL, what are the approved analytical methods, which treatment technologies are approved, how will systems monitor for the contaminant, and how will compliance be determined? All of these details are missing from the Petition for Rulemaking, so it is unclear how the recommended MCL would apply or be implemented.

In analyzing the Petition for Rulemaking, the Department has determined that DRN did not consider all of the relevant factors when recommending the MCL for PFOA not to exceed 6 ppt. As a result, it is recommended that the number advocated for in the Petition for Rulemaking not be the basis for a proposed rulemaking to establish an MCL for PFOA.

E. CONCLUSION

The Department has implemented a number of actions to address PFOA and protect public health. As a result of the work done by Drexel University on behalf of the Department and the occurrence data generated from the PFAS Sampling Plan, the Department believes that additional measures are needed to further protect the public. However, DRN did not include all of the relevant factors that the Department must consider when proposing an MCL. As a result, it is recommended that the number advocated for in the Petition for Rulemaking not be the basis for a proposed rulemaking to establish an MCL for PFOA. While the Department agrees that it should move forward with a proposed rulemaking to set an MCL for PFOA, it does not believe that DRN's proposed MCL was developed appropriately. The Department's proposed rulemaking should be based on available data, studies, and science, and should consider all factors such as health effects, technical limitations, and cost as required under the Federal SDWA and RRA. As a result, the Department recommends that the EQB move forward with a proposed rulemaking to establish an MCL for PFOA. The Department anticipates that it will have a proposed rulemaking developed by the fourth quarter of 2021.

F. APPENDIX

1. Maximum Contaminant Level Goal Drinking Water Recommendations for Per- and Polyfluoroalkyl Substances (PFAS) in the Commonwealth of Pennsylvania, The Drexel PFAS Advisory Board, January 2021.

2. Drexel PFAS Workbook, June 2020.

Maximum Contaminant Level Goal Drinking Water Recommendations for Per- and Polyfluoroalkyl Substances (PFAS) in the Commonwealth of Pennsylvania

By
The Drexel PFAS Advisory Group

January 2021

Table of Contents

1. Executive Summary

The Drexel PFS Advisory Group (DPAG) is a unique multidisciplinary team engaged by the Commonwealth of PA to provide recommendations for Maximum Allowable Contaminant Level Goals MCLGs to the Commonwealth of Pennsylvania for Per- and polyfluoroalkyl substances (PFAS) in drinking water. Observational epidemiology supports the need for drinking water values below the current recommendations of the United States Environmental Protection Agency (US EPA) lifetime health advisory LHA level of 70 ppt for PFOS and PFOA individually or in combination. Furthermore, the identification of other PFAS in drinking water requires a broader consensus consideration of all these substances. As of this report, the US EPA has not initiated its process for establishing MCLs or MCLGs under the Safe Drinking Water Act. Therefore, specific guidelines for the Commonwealth of Pennsylvania were deemed necessary to protect the safety and well-being of Pennsylvanians.

The DAPG consist of experts in the fields of medical toxicology, epidemiology, environmental toxicology, water drinking standards, and risk assessment. The biographies of the members of the DPAG are included as Appendix A.

The Pennsylvania Department of Environmental Protection (PADEP) tasked the DPAG to review the existing and proposed PFA standards from across the country and independently develop MCLGs to inform the initial phase of the rulemaking process for establishing state drinking water standards. (Appendix B and C) The effort commenced in January 2020 and continued to the delivery of this report. Because of restrictions on

face-to-face interactions due to the Covid19 pandemic, much of the advisory groups work was done through virtual conferences between DPAG and PA DEP during 2020.

The DPAG methodically evaluated existing and proposed standards from across the country for PFAs considered under US EPA method 537.1. PADEP asked DPAG to provide specific recommendations on perfluorononanoic acid (PFNA), perfluorooctanesulfonic acid (PFOS), perfluorooctanoic acid (PFOA), perfluorohexanesulfonic acid (PFHxS), perfluoroheptanoic acid (PFHpA), and perfluorobutanesulfonic acid (PFBS). DPAG added the ammonium salt of hexafluoropropylene oxide dimer (GenX) to the list of reviewed PFAS. This latter addition was approved by the PA DEP.

PA DEP charged the advisory group with producing MCLGs within a year. Hence, the initial effort was to review the existing national and state derive PFA assessments, review the pertinent literature in a focused manner, and generally benefit from prior efforts to develop PFAS health-based values. Once complete, the DPAG independently reconsidered all of the PFAS in question and formed draft recommendations for the PA DEP in the summer of 2020.

The PA DEP placed no expectations on the DPAG other than a scientifically defensible approach in developing these values.

Furthermore, by charging a group with developing MCLGs, the commonwealth asked that we focus on developing values that were not as much influenced by technical difficulties necessary to achieve them – e.g. measurement, remediation, or other mitigation. DPAG purposely sought to maintain an independent mindset with developing these MCLGs and to focus on identifying concentrations that would protect

human health. Each consideration and the evidence behind the evaluation as well as methodical calculation are included in the individual summaries. The Reference Dose and recommended Chronic Non-Cancer MCLGs for the seven PFAS considered are Table 1.

PFAS	Reference Dose	MCLG proposed
perfluorooctanoic acid (PFOA)	3.9 ng/kg/day	8 PPT
perfluorooctanesulfonic acid (PFOS)	3.1 ng/kg/day	14 PPT
perfluorononanoic acid (PFNA)	2.2 ng/kg/day	6 PPT
perfluorohexanesulfonic acid (PFHxS)	4.0 ng/kg/day	20 PPT
perfluoroheptanoic acid (PFHpA)	None derived	8 PPT
perfluorobutanesulfonic acid (PFBS)	39 ng/kg/day	55 PPT
ammonium salt of hexafluoropropylene oxide dimer (GenX)	75 ng/kg/day	108 PPT

Table 1: Summary of Reference Dose and proposed Chronic Non-Cancer MCLG for perfluorononanoic acid (PFNA), perfluorooctanesulfonic acid (PFOS), perfluorooctanoic acid (PFOA), perfluorohexanesulfonic acid (PFHxS), perfluoroheptanoic acid (PFHpA), perfluorobutanesulfonic acid (PFBS), and the ammonium salt of hexafluoropropylene oxide dimer (GenX)

2. Background

Per- and polyfluoroalkyl substances (PFAS), and the polymers and surfactants made from them, are a large family of greater than 4000 man-made chemicals that contain carbon, fluorine, and other elements and have been used widely in many industrial and consumer applications since the 1950's. Perfluoroalkyl substances are aliphatic substances where all of the carbons are attached to fluorine with the exception of the last one. Polyfluoroalkyl substances are aliphatic substances where at least one, but not all of the carbons are attached to fluorine and contain the perfluoroalkyl moiety (C_nF_{2n+1}).

The carbon-fluorine bond is stable and strong. The perfluoroalkyl moiety's chemical and thermal stability as well as its lipophobic and hydrophobic properties allow it to be very useful in a variety of industries world-wide. They are used to help make products more resistant to oils, grease, stains, and water, and they are used in many industries because they help reduce friction, through their surfactant applications by lowering their surface tension properties i.e. automotive, construction, aerospace. These properties also contribute to their bioaccumulation and environmental persistence. The length of the fluorinated carbon chain distinguishes the short from the long chain PFAS. Long chain PFAS are perfluoroalkyl carboxylic acids with 8 or more carbon chains and perfloroalkane sulfonic acids with 6 carbon chains and greater. While not specifically stated, perfluoroalkyl chains with 7 or greater carbon atoms are generally considered long chain. The fluorinated carbon chain length determines properties that influence the substance behavior in the environment, organisms, and bioaccumulation. Long chain

compounds include PFNA (9 carbon carboxylic acid), PFOA (8 carbon carboxylic acid), PFHpA (7 carbon carboxylic acid), PFOS (8 carbon sulfonic acid), and PFHxS (7 carbon sulfonic acid). Short chain PFAS include GenX chemicals (6 carbon oxide dimer acid), and PFBS (4 carbon sulfonic acid).

PFASs are present in the environment as a result of their use in a wide array of industrial, commercial, and residential products and applications, including newspaper printing, textile and paper production, metal plating, surfactants in fluoropolymer production, and aqueous film-forming foams (AFFFs), and include consumer products such as outdoor apparel, dental floss, and car wax (Prevedouros 2006, Paul 2008, Konwick 2008). PFASs are emitted to the environment both directly throughout their product and use cycle and indirectly from transformations of their precursors. The majority of emissions are released directly into aquatic environments (Prevedouros 2006, Paul 2008); however, accurate quantification of emissions and resulting environmental exposure are largely lacking (Guo 2009).

2.a. PFAS in Wastewater

PFAS have been found in wastewater treatment plant influents from both municipal and industrial sources, with treated wastewater effluents and sewage sludges (including biosolids) now being viewed as major sources of PFAS to the aquatic environment (Ahrens 2011), which may substantially impact rural water sources. A range of poly- and perfluoroalkyl acids (PFAA) have been routinely detected in wastewater effluents in various countries, including the United States (US) (see review by Hamid 2016). In addition to treated wastewater, various PFAS compounds have been detected in sewage sludges (Venkatesan 2013). In fact, a review by Clarke (2011) ranked PFAS as

the highest priority group of emerging contaminants in biosolids. Taken together, due to the unmitigated use of PFAS in consumer products and the long-term persistence of these compounds, reuse of treated wastewater or land application of biosolids may present a source of PFAS that impact rural communities and agricultural operations.

2.b. PFAS from Landfill Leachate

Due to the widespread use of PFAS in commercial products, various congeners and concentrations of PFAS are likely to be present in all landfills. Landfills receiving waste from industrial facilities (e.g., paints, textiles used in furniture, carpet, upholstery) are expected to have higher concertation of PFAS (Guerra 2014, ITRC 2020). However, low concentrations of PFAS have been detected in the range of ppt to ppb levels at municipal landfills likely due to the use of PFAS on some paper products (Arvaniti 2012, Renou 2008, ITRC 2020). It is important to note that some landfills transferred their leachate to WWTPs for treatment. Perfluoroalkyl sulfonic acids (PFSAs) and Perfluoroalkyl carboxylic acids (PFCAs) are the most common PFASs in landfills, which are known as PFAAs. PFCAs and PFSAs have the carbon chain length C4-C18 as well as C4-C10, respectively. Additionally, PFAAs precursors (e.g., FTOH, n:2 FTCA, and n:2 FTUCAs) existing in the consumer products (Ye 2015; Kotthoff 2015) can degrade to PFAAs throughout disposal in the landfill and product use (Lang 2016, Allred 2015).

2.c. PFAS from the use of AFFF

The U.S. Department of Defense (DoD) has used aqueous film forming foam (AFFF) to suppress fires since the 1970s. PFASs are known to contaminate over 500 DoD sites (Thompson 2012), and repeated historic use at firefighter training areas has

resulted in groundwater and porous media contamination, with groundwater concentrations of select PFASs reaching low mg/L levels (Moody 1999, 2000, 2003, Anderson 2016, Murray 2010, Backe 2013, McGuire 2014, Filipovic 2015, Schultz 2004). While PFAAs are often not the dominant PFASs in AFFF formulations at impacted sites, PFAAs and 6:2 FtS are often the dominant PFASs found in contaminated groundwater (Backe 2013, Houtz 2013, McGuire 2014, Schultz 2004). The predominance of PFAAs in groundwaters is hypothesized to be a result of abiotic and biotic reactions in the subsurface that transform the parent PFAS compounds in AFFF formulation (*e.g.*, fluorotelomer thioamido sulfonates, FtTAoS) into FtSs and PFAAs (Harding-Marjanovic 2015).

2.d. PFAS Fate and Transport in the Environment

While there are many aspects that make PFASs chemistry unique, of particular note are their biological and chemical stability, promoting their persistence in the environment), and the comparatively high solubility limits and adsorptive nature of some PFASs, especially of shorter chain length, making them relatively mobile in aqueous systems (Zareitalabad 2013). Perfluoroalkyl acids (PFAAs), which have a negatively charged head group, low volatility, and high water solubility, are considered to be highly mobile in aqueous phases (Ahrens 2011, Ahrens and Bundschuh 2014), and PFAA transport has often been observed or inferred in the environment (Moody 1999, Lindstrom 2011, McGuire 2014, Baduel 2015, Filipovic 2015). As a consequence of such mobility and concerns of their human health effects, drinking water wells at several downstream localities of DoD sites have been temporarily abandoned. The sorption behavior of PFASs is influenced by their physicochemical properties which vary

depending on their functional head group and chain length (Ahrens 2009, 2011, Ahrens and Ebinghaus 2010). PFAA sorption generally increases with increasing chain length. Longer chain length PFAAs have been demonstrated to bioaccumulate and possibly biomagnify. (Prevedouros 2006, Conder 2008) In addition to the ecological effects, bioaccumulation within a food web may lead to human exposure through dietary consumption (e.g., fish). As a consequence, sediments and biota are considered to act as a sink for longer chains PFAAs in aquatic ecosystems.

3. Approach

The DPAG reviewed a number of recommendations made by EPA and State agencies that chose to create a summative approach to PFAS, combining multiple minimal risk levels or advisory levels into one cumulative drinking water value. No clear consensus exists on this approach and the use of a summative approach was clearly designed to be a shortcut based on a presumption that the agents all have similar health effects and endpoints. While this approach may work for other toxins such as dioxins, furans, and coplanar polychlorinated biphenyls, it does not appear to be based on evidence available for PFAS. The DPAG therefore committed early in the process to developing an individual MCLG for each of the requested PFAS. DPAG further recommends that all PFAS be reviewed individually as they arise for analysis, even if the individual MCLG ultimately needs to be based on chemical similarities to other PFAS only (e.g. see PFHpA in our recommendations).

For each of the PFAS studied, the DPAG identified points of departure and rationale for selection from risk assessments published by other states, the EPA, and a TSTR. DPAG then assessed the underlying critical studies driving the selection of the POD. Every effort was made to use the experience and published findings from other agencies and build and refine on these as much as possible into a best practice approach. USEPA (2000), Beck (2016)

3.a. Maximum Contaminant Level Goals

Maximum Contaminant Level Goals (MCLGs) are maximum drinking water concentrations designed to protect human health. MCLGs are non-enforceable as they are chosen solely based on protection of human health and do not take into account whether analytical testing is available to detect the contaminant at the MCLG level or whether adequate technology exists to remediate or remove the contaminant at the MCLG level. Conversely, Maximum Contaminant Levels (MCLs), are derived from MCLGs but also take into account the availability of analytical testing, adequate technology for contaminant remediation, efficacy under field conditions, and cost. MCLGs include a margin of safety incorporated into the level via the use of uncertainty factors that ensures no adverse human health effects would result from lifetime exposure to the contaminant in drinking water at the MCLG level. MCLGs are derived separately for and non-cancer endpoints and cancer endpoints.

3.b. Non-Cancer Endpoints

The derivation of an MCLG is based on the assumption that for non-cancer endpoints, a dose threshold exists. Doses above that threshold potentially place a

person at risk for an adverse human health effect, whereas below that threshold the person is not at risk. To ensure that exposure at the MCLG and below does not place any person, including vulnerable populations, at risk, an adequate margin of safety is built into the derivation.

Available animal model studies are reviewed to determine the point of departure (POD), which is the first step in the MCLG derivation. The point of departure (POD) may be an administered dose, a modeled dose, or a serum level. If the POD is a serum level, a dose adjustment factor may be applied to derive a dose. In considering animal model studies as candidates for the POD, a number of factors should be considered, study duration (acute, subacute, chronic), route of exposure, intensity of exposure, study quality, relevance of the animal model adverse health effect to human health, and interspecies differences in absorption, distribution, metabolism and excretion of the substance. Animal model studies may be considered irrelevant for the derivation of an MCLG based on the above considerations and therefore not be used for the POD.

If an animal model study meets the criteria discussed above and is considered relevant to human health, then it serves as a candidate along with other such studies for the POD. Several PODs are available. The most commonly used POD is the no-observed-adverse-effect level (NOAEL), the highest dose administered in the animal model study that did not result in toxicity where toxicity is defined by alteration of biomarkers, change in body weight or body weight gain, lesions, or anatomical abnormalities at necropsy. In some circumstances, such as the absence of a NOAEL in an animal model study, the lowest-observed adverse-effect level (LOAEL) may be used as the POD. (USEPA 2002)

An alternative POD that may be used with robust datasets is the lower confidence limit of the benchmark dose (BMDL). Calculating the BMDL requires sufficient datapoints from the animal model study/studies that a dose-response curve can be modeled. The benchmark response (BMR) is the acceptable level of change in the animal model adverse health effect. A BMR of 10% is typically considered the acceptable level of change as it is at or near the limit of sensitivity of many bioassays. For continuous variables (e.g. body weight), a BMR of 10% corresponds to a 10% deviation in the outcome of interest, whereas for quantal data (e.g. organ toxicity) a BMR of 10% corresponds to a 10% increase in the incidence of the adverse effect. Statistical modelling of the dose response curve is used to calculate the dose that corresponds to the chosen BMR, known as the benchmark dose (BMD), and the lower 95% one-sided (or two-sided) confidence limit of the BMD is the BMDL. The DPAG, in discussion with the PA DEP, determined that the BMDL that corresponded to a BMD with a BMR of 10% (referred to as the $BMDL_{10}$) would be the default POD when the BMD method was employed. (USEPA 2012)

The EPA recommends a number of approaches to derive human equivalent oral exposures (HED) from a laboratory animal species derived POD. (USEPA 2002) The preferred approach is physiologically-based toxicokinetic modeling applying a dose adjustment factor. The DAF is multiplied by the animal exposure (in mg/kg/d) to achieve the human equivalent exposure (in mg/kg/d). In lieu of data to support either of these types of approaches, body weight scaling to the 3⁄4 power (i.e., BW3/4) is endorsed as a general default procedure to extrapolate toxicologically equivalent doses of orally administered agents from all laboratory animals to humans for the purposes of deriving

an oral Reference Dose (RfD). Use of these methods is generally combined with a default interspecies uncertainty factor, UFA, reduced from 10 to $10^{0.5}$.

Once the HED is identified, the reference dose (RfD) is calculated by dividing the HED by uncertainty factors (UF) to create an adequate margin of safety. UFs have a value between 10^0 (i.e. 1), $10^{0.5}$ (i.e. 3), or 10^1 (i.e. 10). A default UFH of 10 is applied for the potential variability in sensitivity to the exposure in the human population. An UFA of 10 each is applied for the uncertainty of extrapolation from an animal model to humans unless some dose adjustment factor can be accurately applied. A default UFL of 10 is applied when the LOAEL is used rather than the NOAEL or BMD. A UFS is applied when extrapolating from sub-chronic animal model studies to chronic human exposure. An additional UFD, referred to as a modifying factor, may be applied to account for uncertainty about the quality of the study or data set. All the UFS are multiplied to develop a UFT, or total uncertainty factor. Figure 1 provides an illustration but does not represent an actual PFA or the order of endpoints.

Point of Departure Determination

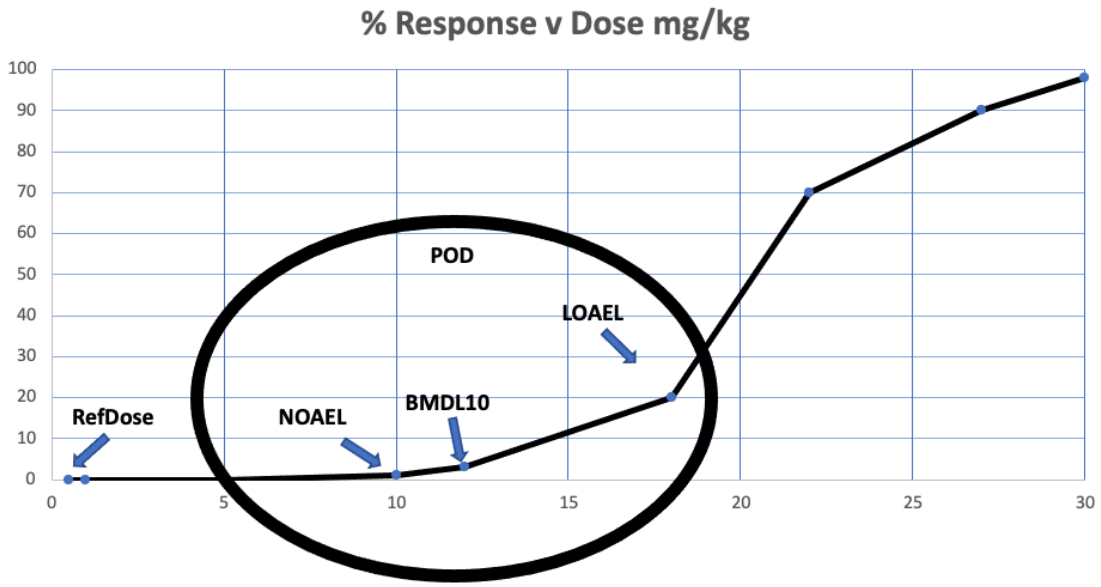

Figure 1: POD sought amongst various endpoints (LOAEL, NOAEL, $BMDL_{10}$) and then a Reference Dose derived.

The RfD is typically expressed in mg/kg/d and is the daily ingested dose of a substance that is considered to be without an increased risk of an adverse human health effect. The RfD can be converted into a Drinking Water Equivalent Level (DWEL), the concentration of the substance in water that would yield the RfD for the target population based on established drinking water rates. If the POD suggests that the target population is adults, then standard assumptions about weight (e.g. 70-kg adult) and consumption (2-L of water per day) are used. Different weight and consumption standards are applied if the POD suggest the target population is, for example, infants.

The MCLG is subsequently derived from the DWEL by accounting for the relative source contribution (RSC) of drinking water to total daily dose of the substance so that the total daily dose does not exceed the RfD. For substances where the relative source contribution is unknown, a default RSC of 0.2 is used. When the relative contribution of various sources to daily dose has been determined, the RSC of drinking water may be used instead of the default RSC but may be no greater than 0.8 to account for potential unknown exposure sources. (USEPA 2000)

3.c. Goeden Model discussion

An alternative method to convert RfD to MCLG is the transgenerational toxicokinetic model. This approach considers water consumption from conception to adulthood and adjusts for the fact that relative source contribution of water is higher early in life. It assumes that a child will have a certain level of exposure in-utero because of the PFA in the mother's body and further exposure during breastfeeding or bottle feeding. This model requires specific toxicokinetic information about the substance in question and cannot be applied to every substance. The model for this report was provided to the DPAG by Minnesota Department of Health (MDH) as an excel spreadsheet. Parameters for this model are listed in Appendix C. Although RfD was always calculated, the POD serum level was divided by UFT to determine a corresponding internal target human serum level (THSV). Working backward from the target human serum level, reduced by 50% to account for the RSC of an infant, an MCLG was derived from the model so that the highest serum level ever achieved from birth to adulthood never exceeded the reference dose. The model had sufficient data for application to MCLG recommendations for PFOA, PFOS, PFNA, and PFHxS. Table 2 lists some of the key

model parameters and the preferred tendency (central or upper) of the parameter. Please note: The THSV is useful for informing public health policy and interpreting population-based exposure potential. This value is based on population-based parameters and should not be used for clinical assessment or for interpreting serum levels in individuals.

Model Parameter	Tendency of Parameter	PFOA	PFOS	PFHxS	PFNA
Half-Life, days	Central	840 [a]	1241 [b]	1935	1417 [c]
Placental Transfer Ratio	Central	0.87 [d]	40 [d]	0.70 [d]	0.69 [d]
Breastmilk Transfer Ratio	Central	0.052 [d]	0.017 [d]	0.014 [d]	0.032 [d]
Volume of Distribution (V_d), L/kg	Central	0.170 [e]	0.230 [e]	0.25 [f]	0.200 [d,g]
Relative Source Contribution (RSC), %	Central	50	50	50	50
Duration of Exclusive Breastfeeding, months	Upper	12	12	12	12
a) Bartell 2010; b) Li 2018; c) Zhang 2013; d) MDH 2020, 2019; e) Thompson 2010; f) Sundstrom 2012; Ali 2019 g) ATSDR 2018					

Table 2: Exposure Model Parameters used in transgenerational model (Goeden 2019) for derivation of proposed MCLG.

3.d. Cancer Endpoints

MCLGs for cancer endpoints are historically set at zero although there may be scenarios under which a non-zero MCLG is appropriate for a cancer endpoint. The rationale behind a zero MCLG for cancer endpoints is that historically extrapolation of cancer risk from high dose animal studies to low dose human exposures was performed using the linear no-threshold model. The absence of a threshold in this extrapolation

model results in some cancer risk being associated with any dose. Therefore, the only level goal that can be considered protective of human health is zero. (USEPA 2005)

Current carcinogen risk assessment allows for the consideration of threshold effects in extrapolation of cancer risk. A threshold effect may be present if cancer is only observed when an exposure meets a certain intensity or duration. However, the absence of cancer at low level exposures should not be assumed to constitute a threshold as low level exposures may be associated with cancer risk that is undetected due to studies that are underpowered to detect cancer at that exposure intensity. The mechanism by which the carcinogen increases cancer risk may inform whether a threshold effect is present. If the carcinogen induces cancer secondary to a toxic effect then the threshold is the dose at which the toxic effect occurs and doses below that threshold, after applying uncertainty factors, should be considered non-carcinogenic. MCLGs for carcinogens that act by a mutagenic mode of action are still set at zero as the linear-no threshold model is most appropriate for that mechanism.

Substances that are only carcinogenic above a certain exposure intensity or duration may have non-zero MCLGs utilizing the same derivation process as for non-cancer endpoints, discussed above. For such substances, the MCLG for the cancer endpoint and the MCLG for the non-cancer endpoint are both derived and the lower value of the two serves as the overall MCLG for the substance.

Numerous epidemiological studies of PFAS, especially PFOA and PFOS, have examined occupational and environmental exposures but have failed to detect consistent findings across studies. (Bonefeld-Jorgensen 2011, Chang ET 2014, Eriksen 2009, Hardell 2014, Innes 2014, Klaunig 2015, Yeung 2013). The International Agency

for Research on Cancer (IARC) has classified PFOA as “possibly carcinogenic to humans” (Group 2B), based on limited evidence in humans that it can cause testicular and kidney cancer, and limited evidence in lab animals. The EPA has not officially classified PFOA as to its carcinogenicity. EPA’s Scientific Advisory Board, based mainly from studies in lab animals, stated that PFOA shows “suggestive evidence of carcinogenicity, but not sufficient to assess human carcinogenic potential.”

PFOA and PFOS show positive associations with cancers of the prostate, kidney, testis, and thyroid but with a) only small elevations in relative risk intervals (0.5 and 2.0 (with 95% confidence intervals including 1.0), b) evidence of negative associations as well, and c) inconsistencies across the studies. Furthermore, exposure response relationships do not follow the monotonic pattern of increasing dose causing increasing response. The strongest example is that associations found at lower environmental community studies are not supported by those found in the workplace where exposures are higher by one or two orders of magnitude. Furthermore, although animal studies support target organ as the liver, testis (Leydig cells), and pancreas (acinar cells), these are not the types of cancers identified by human studies. Some drinking water recommendations rely on an effect produced by expression of peroxisome proliferator-activated receptor-alpha (PPARalpha) which is specific to rodents. For example, CEPA (2019) and NJDEP (2017, 2018) have cancer minimal risk levels for PFOA and PFOS derived heavily from animal studies. After careful review, the DPAG concluded that cancer endpoints for PFAS that rely heavily on animal studies are not supported by the totality of human and animal evidence. Furthermore, there is insufficient evidence to argue that Non-Cancer MCLGs would not be protective of cancer risk.

4. PFOA

After a literature search and a review of the available evidence and recommendations from various agencies, the DPAG developed an MCLG recommendation for PFOA based on Non-Cancer endpoints. The agencies with the most relevant inputs were the US EPA, the ATSDR (ATSDR 2018), the MDH (MDH 2020 PFOA), NJDEP (NJDEP 2017), and MDHHS (MDHHS 2019). The US EPA selected Lau (2006) because it met their criteria for chronic exposure, multiple dose groups, use of a concurrent control, and with serum data amenable for modeling. (US EPA 2016) MDH used Lau (2006) as well and used the serum level estimated by US EPA. The ATSDR selected identical LOAELs from Onishchenko (2011) and Koskela (2016). Both studies had the same populations of laboratory animals and evaluated a single dosing group. These studies identified developmental effects (neurobehavioral and skeletal) as critical. The DPAG selected Koskela (2016) and Onishchenko (2011) as the critical studies. (ATSDR 2018, Appendix A, Table A8)

The serum concentration at the LOAEL of 0.3 mg/kg/d from Onishchenko (2011) and Koskela (2016) was below the modeled serum concentrations from two immunotoxicity studies evaluated by ATSDR (a sensitive effect seen in other PFAS). (Lau 2006) MDHHS also selected the critical studies by ATSDR as also being protective for immunotoxicity. (MDDHS 2019) The DPAG rejected the BMDL from Loveless (2006) used by NJDEP. Loveless (2006) was a 14-day exposure study in rats and mice, with liver weight changes being the critical effect identified. NJDEP (2017) Liver weight changes, in and of themselves, translate questionably as an adverse effect in humans

and the POD identified was higher than those when considering immunotoxicity. From Onishchenko and Koskela, the ATSDR estimated the POD average serum concentration in the mice (8.29 mg/L) using a three-compartment pharmacokinetic model (Wambaugh 2013) using animal species-, strain-, sex-specific parameters. This was adopted by the DPAG as the POD for PFOA.

4.a. Review of Critical Studies

Koskela (2016) investigated the administration of PFOA at a dose of 0.3 mg/kg/d administered orally mixed with food to pregnant C57BL/6/Bkl mice starting on GD1 to investigate developmental outcomes on long bone morphology and bone cell differentiation. Female offspring were sacrificed at the age of 13 or 17 months for examination.

Body weights of PFOA exposed offspring were higher than controls throughout the lifetime of the animals, reaching statistical significance at 13 and 17 months. Significant increases in the femur and tibial periosteal area and medullary area were seen at 17 months but not at 13 months in PFOA exposed offspring. Tibial mineral density was decreased in PFOA exposed offspring at both 13 and 17 months. Femur and tibial cortical area, trabecular parameters, and femur mineral density were unaffected by PFOA exposure. There was no significant effect of PFOA exposure on biomechanical properties of the femur or tibia. Concentration of PFOA in pooled tibias and femurs was significantly greater in exposed offspring at both 13 and 17 months.

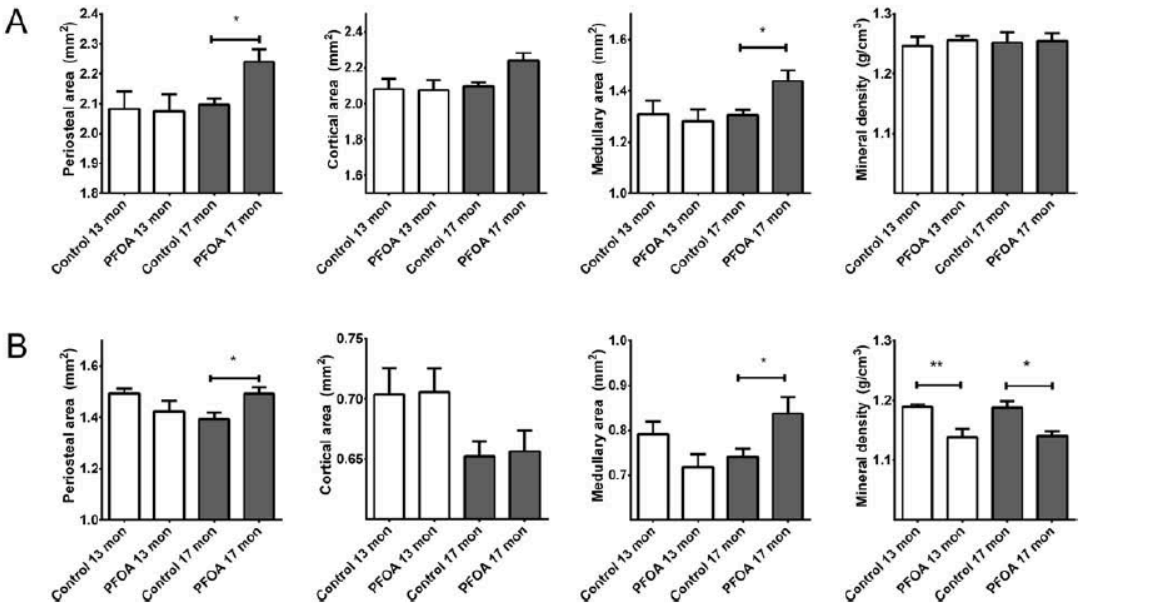

Fig. 2. Effects of PFOA on morphometrical parameters of femurs (A) and tibias (B) as analyzed by microCT. The cortical VOI reference point was set to the point where complete fusion of the growth plate was observed, offset being 250 and height 500 cross-sections proximally. Group mean ± SE, $n = 5$. $^{*}p < 0.05$, $^{**}p < 0.01$.

Figure 2: Effects of PFOA reproduced from (Koskela 2016). This represents the selected PFOA critical effect of morphometric parameters of femurs and tibias at 13 and 17 months - dosing is 0.3 mg/kg/d (LOAEL). The average serum concentration was estimated in the mice (8.29 mg/L) using a three-compartment pharmacokinetic model (Wambaugh 2013) using animal species, strain, sex-specific parameters. (ATSDR 2018)

In an in vitro study, the effect of PFOA on the viability of MC3T3 osteoblast precursor cells were assessed using an MTT-test on days 1, 7, and 10. A significant decrease in cell viability was seen on days 7 and 10 at a PFOA concentration of 100 mcM and above but not at a concentration of 10 mcM. A significant decrease in the alkaline phosphatase activity of osteoblasts was seen at day 7 at a PFOA concentration of 100 mcM and above but not at a concentration of 10 mcM. An increase in calcium and in OCN mRNA was seen at PFOA concentrations of 1 and 10 mcM but not at higher concentrations.

In a second in vitro study investigating the effect of PFOA on osteoclasts, the number of TRACP+ cells containing three or more nuclei was increased at PFOA concentration of 10 mcM and above with evidence for a dose response relationship. Osteoclasts were not significantly affected at 1 mcM. Resorption pit area was significantly increased at a PFOA concentration of 1 mcM, but with no evidence of a dose response relationship and a decrease in pit area with increasing PFOA concentration.

Onishchenko (2011) investigated the administration of PFOA or PFOS at a dose of 0.3 mg/kg/d administered orally via food to pregnant C57BL/6/Bkl mice starting on GD1 to investigate Motor function, circadian activity, and emotion-related behavior in exposed offspring. One pump per litter was sacrificed at birth for brain and liver tissue samples of PFOS and PFOA levels. Offspring were weaned on postnatal day 21 and injected subcutaneously with microtransponders. Test for locomotor and circadian activity were performed at age of 5 to 8 weeks. Animals were tested for emotion-related behavior in elevated plus maze and forced swim test. Test for motor strength and motor coordination were performed in animals at 3 to 4 months old.

Administration of PFOS or PFOA did not affect damn weight gain, litter size, or sex ratio. There were no differences in offspring body or brain weight between groups at birth. Absolute liver weight was increased in PFOA-exposed offspring as compared to controls, but not in PFOS-exposed offspring. Among exposed pups, PFOS concentrations at birth or greater than PFOA concentrations in the brain, but lower in the liver.

PFOS-exposed males walked significantly less than male controls when exploring a new environment, while PFOS-exposed females do not differ from controls. PFOA exposure did not have a significant effect on locomotor activity in either sex.

Circadian activity was measured using the TraffiCage system. During adaptation to the new cage, PFOS-exposed males displayed decreased activity during the first two hours of the test, while PFOS-exposed females displayed decreased activity during the first hour only. PFOA-exposed males were more active during the first hour of the test, while PFOA-exposed females demonstrated decreased activity as compared to controls. After habituation to the cage, PFOS exposure After habituation to the cage, PFOS exposure did not significantly affect activity counts over light or dark periods, either in males or females. PFOA exposed males demonstrated greater activity as compared to controls, especially during the dark phase, while PFOA exposure in females had no effect on activity level. PFOS exposure was associated with a greater number of inactive periods during both light and dark phase in both males and females, although only the difference in females reached statistical significance. PFOA demonstrated an opposite effect, decreasing the number of inactive periods in both light and dark phase which met significance in both phases for males but only in the light phase for females. (see Figure 3)

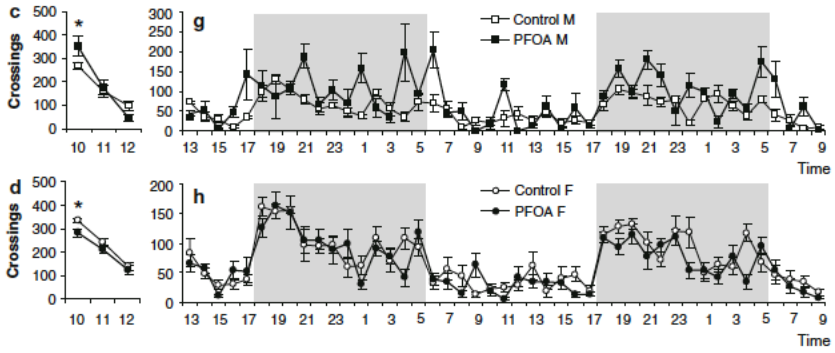

Fig. 2 Novelty-induced (**a–d**) and circadian activity (**e–h**) over 48 h in the home cage and social group in male and female mice prenatally exposed to PFOS or PFOA. Activity counts presented as number of antenna crossings in the TraffiCage (see "Materials and Methods" section for details). Gray areas correspond to a dark phase of the light–dark cycle. * $P < 0.05$, $n = 6$–10)

Figure 3: Figure reproduced from Onishchenko (2011). This was selected as a PFOA critical effect for change in inactive periods seen at 0.3 mg/kg/d (LOAEL). (Onishchenko 2011) The average serum concentration was estimated in the mice (8.29 mg/L) using a three-compartment pharmacokinetic model (Wambaugh 2013) using animal species, strain, sex-specific parameters. (ATSDR 2018). Note: because the POD dose and pharmacokinetic model are the same as Koskela (2016), the derived POD serum concentrations are the same.

Evaluation for anxiety-related behavior in the elevated plus maze demonstrated that PFOS-exposed male mice walked less total distance than did controls, which was consistent with previous findings of decreased locomotor activity in this group, but which based on time spent in open and closed arms did not seem to reflect changes in anxiety-related behavior. No significant differences in anxiety-related behavior were noted in PFOS-exposed females or in PFO- exposed males or females.

No effect of PFOA or PFOS was demonstrated in either sex in depression-like behavior in the forced swimming test.

Muscle strength in the hanging wire test was less in PFOS-exposed males who had significantly shorter fall latency than controls. No effect was seen in PFOS-exposed female mice or in PFOA exposure in either sex.

Inconsistent findings were demonstrated between PFOS and PFOA exposure and motor coordination in the accelerating rotarod test. PFOA-exposed females had shorter

fall latency in every trial, but it only met statistical significance in 1 of 4 trials, while PFOA exposed males had similar fall latencies as compared to controls. PFOS-exposed females had shorter fall latency in 2 of 4 trials while PFOS-exposed males had shorter fall latency that was significant in only one of four trials.

4.b. Development of MCLG

Following the approach used by MDHHS and MDH to identify a species-specific DAF, DPAG selected the PFOA serum half-life of 840 days (2.3 years). (Bartell 2010) This was considered more relevant for exposure to the general population than occupational exposure studies used by ATSDR. (ATSDR 2018, Bartell 2010). studied 200 individuals (100 men, 100 women) exposed by drinking PFOA-contaminated water. DAPG used the volume of distribution (Vd = 0.17 L/kg) selected by MDHHS and MDH that was based on human data. (Thompson 2010). These were the references used by EPA in 2016 when they derived a PFOA clearance of 1.4 x 10-4 l/k/d and developed their health advisory level.

DPAG accepted the UFs selected by ATSDR for a UFT of 300. (ATSDR 2018) This resulted in a THSV of 0.028 mg/L for the Goeden Model. Setting the target for the breast fed infant as 0.014 (50%RSC), the MCLG for drinking water is recommended to be 8 ng/L (8PPT) to protect breastfed infants and throughout life. (Figure 4, Table 3)

PFOA	
Dose Response Modeling Method	**LOAEL**
POD	The average serum concentration was estimated in the mice (8.29 mg/L) using a three-compartment pharmacokinetic model (Wambaugh 2013) using animal species, strain, sex-specific parameters. (ATSDR 2018)
HED = POD x DAF (mg/kg/d)	DAF = Ke x Vd Ke = 0.000825175 (8.2×10^{-4}) based on a human serum half-life of 840 days (Bartell 2010) Vd = 0.17 L/kg (Thompson 2010) $HED_{LOAEL} = POD_{LOAEL} \times DAF$ $HED_{LOAEL} = POD_{LOAEL} \times Ke \times Vd$ $HED_{LOAEL} = 8.29$ mg/L x 0.0000825175 x 0.17 L/kg $HED_{LOAEL} = 0.001163$ mg/kg/d or 1.163×10^{-3} mg/kg/d
Uncertainty Extrapolation	
Human Variability (UFH)	10 (standard)
Animal to Human (UFA)	3 (DAF applied)
Subchronic to Chronic (UFS)	1 (Chronic effect studied)
LOAEL to NOAEL (UFL)	10 (standard)
Database (UFD)	1
Total Composite (UFT)	300
RfD = HED/UFT (mg/kg/d)	RfD = 0.001163 mg/kg/d/300 RfD = 3.9 ng/kg/day (3.9×10^{-6} mg/kg/d)
THSV = POD / UFT	THSV= 8.29 mg/L/ 300 THSV= 0.028 mg/L
Receptor	Infant exposure via breastmilk for 1 year, from mother chronically exposed via water, followed by lifetime of exposure via drinking water. Protective for short-term, subchronic and chronic. (also protective of formula fed infant). Goeden Model Parameters: Placental transfer of 87% and breastmilk transfer of 5.2% (MDH (2020 PFOA)). The Human Serum half-life is set at 840 days (Bartell 2010).

	The Volume of distribution of 0.17 L/kg (Thompson 2010) Other factors include, 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 is used to calculate maternal serum concentration at delivery. (Goeden 2019) A Relative Source Contribution of 50% (0.5) is applied and based on studies which showed that infants RSC is similar to NHANES 95th percentiles for 3-11 (2013-2014) and over 12 years old (2015-2016) participants. (CDC 2019)
Chronic Non-Cancer MCLG	The model produces a Chronic Non-Cancer MCLG of 8 ng/L (ppt). This protects health during the growth and development of a breast fed infant. (Figure 4)

Table 3: Development of Non-Cancer MCLG for PFOA

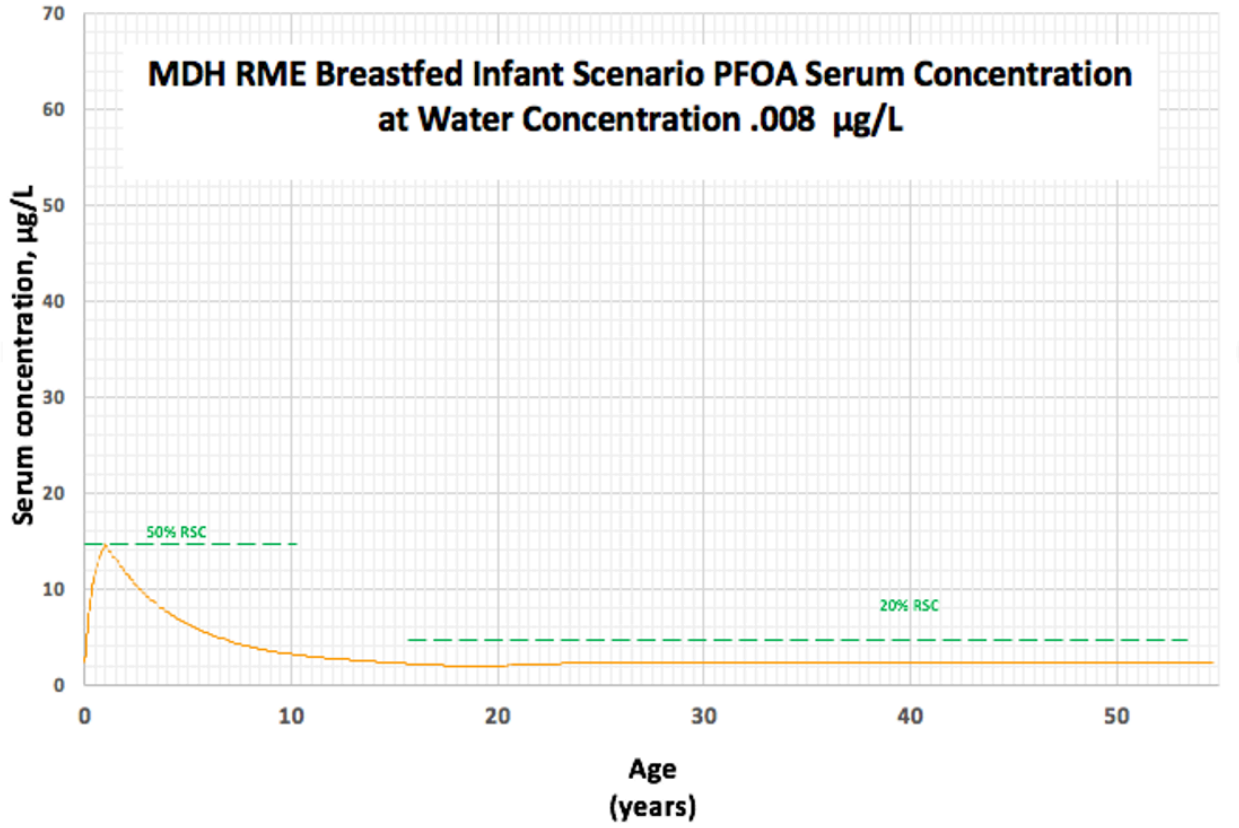

Figure 4. Using the Goeden Model, the POD and its parameters for PFOA were converted to an THSV of 0.028 mg/L. An RSC set at 50% means that half of this (0.014 mg/L) will be from ingested drinking water. The MCLG of PFOA in drinking water should then be set at 0.008 ug/L or 8 PPT to protect from adverse health events.

5. PFOS

After a literature search and a review of the available evidence and recommendations from various agencies, the DPAG developed an MCLG recommendation for PFOS based on Non-Cancer endpoints. DPAG reviewed a number of candidate MRL levels developed by US EPA and ATSDR. (ATSDR 2018, Dong l 2011, Pachkowski 2019, Peden-Adams 2008, Vassiliadou 2010, Butenhoff 2009) Although immune function has not been examined following chronic-duration oral exposure in laboratory animal studies, the lowest LOAEL doses were for immunological effects in intermediate-duration animal studies. These were seen at doses lower than hepatotoxicity or developmental effects. ATSDR did not select an immunotoxicity study as a critical study but did develop a “candidate MRL” using the immunotoxicity study by Dong (2011). The NOAEL endpoint was suppression of natural killer cell activity and anti-Sheep Red Blood Cell Antibody response in mice. Laboratory animal studies, particularly studies in mice, provide supporting evidence of the immunotoxicity of PFOS. Human epidemiological studies are consistent with this evidence as well. After the calculation of HEDs and application of UFs to all of these studies, the resultant MRLs were nearly identical to those using other studies by agencies such as MDHHS. Thus, DPAG concluded the study by Dong l (2011) and the POD of 2.36 mg/L were appropriate. This study was selected over the other immunotoxicity studies because it identified the highest NOAEL for immunotoxicity and the longest exposure duration.

5.a. Review of Critical Study

Dong l (2011) administered PFOS to adult male C57DL6 mice to investigate immunotoxicity outcomes. PFOS with 2% Tween 80 was administered by oral garage daily for 60 days to a targeted total administer dose over that period of 0, 0.5, 1, 5, 25, and 50 mg/kg body weight with controls being administered deionized water with solubilizer only. 12 mice were included in each group. Mice were immunized on the 54th day of PFOS dosing by intravenous injection of sheep red blood cells (SRBC). Six of the 12 mice from each treatment group or sacrificed seven days later and blood was obtained by cardiac puncture. The remaining six mice were administered a booster immunization of SRBC to the right rear foot pad on the final day of PFOS dosing to investigate delayed type hypersensitivity response (DTH) and other immunoglobulin assays.

Mice exposed at the highest dose of 50 mg/kg had significantly lower body weight as compared to controls; however, body weight change was insignificant at other dose levels. Similarly, food intake on the final day of dosing was significantly less at the highest 50 mg/kg dosing group as compared to controls but was there was no significant difference at other dose levels. Relative spleen and thymus weights were decreased at the highest 50 mg/kg dose, but not significantly different than other dose levels. Relative liver weight was increased at both the 25 mg/kg dose and 50 mg/kg dose as compared to controls.

Serum PFOS concentration increased in a dose response fashion with increasing absolute dose administered. There was no significant effect of treatment dose on serum corticosterone level.

IFNgamma level was significantly decreased at the 50 mg/kg dose, without significant changes at other dose levels. IL-4 levels were significantly increased at the 5 mg/kg dose and above. For both IFNgamma and IL-4, changes in levels were largely dose-dependent except at the lowest 0.5 mg/kg dose. The number of cells secreting IL-2 and IL-10 were decreased and increased, respectively, in the 50 mg/kg dose group, but no significant differences were seen at lower dose regimens. As with other cytokines, changes in levels were largely does dependent at the higher dose regimens only.

With respect to immunoglobulin synthesis, IgM levels declined with a dose-response relationship at the 5 mg/kg dose and above. IgG, IgG1, and IgE production were all increased only at the 50 mg/kg dose with other lower dose regimens not affecting serum levels. IgG2a levels and delayed-type hypersensitivity response were unaffected by PFOS administration.

5.b. Development of MCLG

Dong (2011) identified immune suppression, specifically increased IL-4 and decreased Sheep RBC specific IgM levels in the mouse model. Doses administered over 60 days were converted to mg/kg/d by dividing by 60 days. Thus, doses were 0, 0.00833, 0.0167, 0.0833, 0.4167, and 0.8333 mg/kg/d. The NOAEL of 0.0167 mg/kg/day (total dose over 60 days of 1 mg/kg) was selected because it was the highest dose without a statistically significant effect. (Figure 5 is reproduced from Dong (2011; Figure 1)

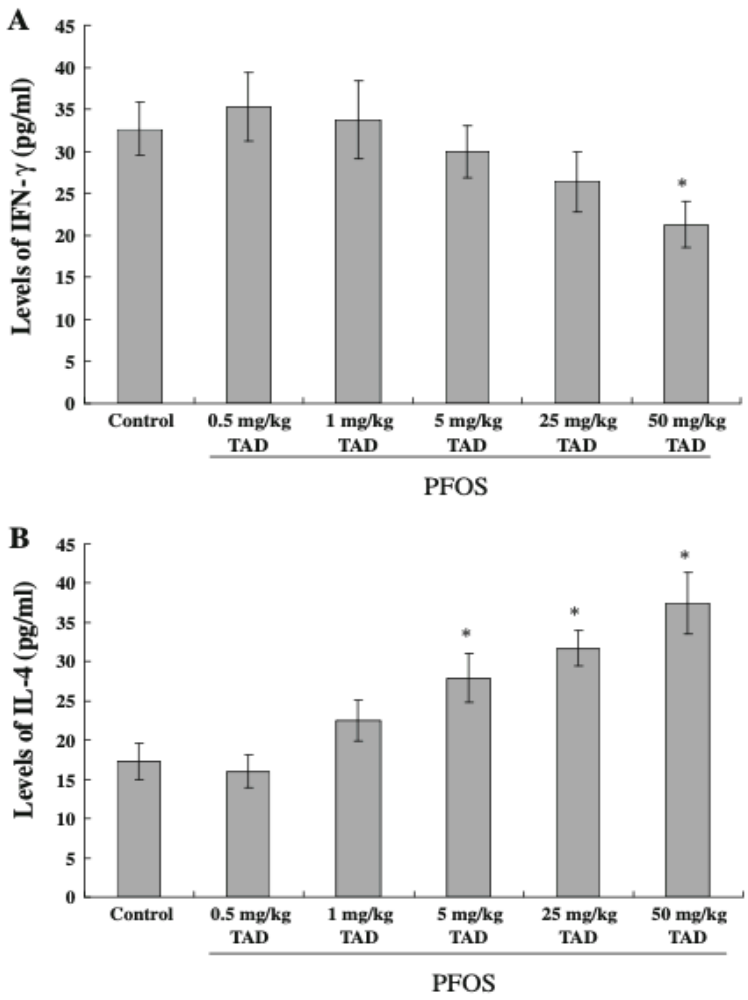

Fig. 1 IFN-γ and IL-4 levels in the splenocyte culture supernatant of splenocytes harvested from mice 24 h after the last of their 60 days of treatment, i.e., daily oral exposures to PFOS. Data are presented as mean ($\pm$SE) of results obtained using ELISA kits. *Significantly different from respective control ($P \leq 0.05$). The data were log transformed as required for statistical analysis. *TAD* Total Administered Dose over the course of 60 days. $n = 6$ in each group

Figure 5: NOAEL critical effect of increased IL-4 levels determined by Dong 2011. The dose administered is over 60 days and is thus converted to the daily dose of 0.0167 mg/kg/day (total dose of 1 mg/kg over 60 days).

Dong provided the serum PFOS level at each dose and thus the 1 mg/kg dose results in a serum PFOS level of 2.36 mg/L (+/- 0.47). This is found in Figure 6.

Table 1 PFOS concentrations in serum (mg/L), body weight, and organ indices in adult male C57BL/6 mice treated with PFOS orally for 60 days

PFOS (mg/kg TAD)	*n*	Serum PFOS (mg/L)	Serum corticosterone (ng/L)	Body weight change[a]	Food intake from day 60 to day 61 (g)	Spleen index[b]	Thymus index[b]	Liver index[b]	Kidney index[b]
Control	6	0.05 ± 0.01	443.28 ± 31.69	4.57 ± 0.42	5.06 ± 0.35	0.45 ± 0.03	0.30 ± 0.03	5.23 ± 0.16	1.48 ± 0.05
0.5	6	1.07 ± 0.11	434.62 ± 28.93	4.81 ± 0.36	5.42 ± 0.42	0.47 ± 0.02	0.27 ± 0.02	5.16 ± 0.14	1.53 ± 0.05
1	6	2.36 ± 0.47	387.14 ± 35.08	5.14 ± 0.45	5.11 ± 0.28	0.46 ± 0.02	0.33 ± 0.02	5.29 ± 0.21	1.57 ± 0.06
5	6	10.75 ± 0.82*	369.87 ± 27.51	4.83 ± 0.34	4.87 ± 0.33	0.46 ± 0.03	0.31 ± 0.02	5.75 ± 0.17	1.54 ± 0.03
25	6	22.64 ± 2.29*	453.76 ± 42.12	3.92 ± 0.47	4.42 ± 0.27	0.42 ± 0.03	0.24 ± 0.02	6.33 ± 0.16*	1.44 ± 0.06
50	6	51.71 ± 3.81*	528.39 ± 33.94	2.16 ± 0.29*	3.39 ± 0.35*	0.35 ± 0.02*	0.21 ± 0.01*	8.04 ± 0.20*	1.41 ± 0.04

PFOS concentrations, body weight (change), and organ weight data did not require transformation for statistical analysis

TAD Total Administered Dose over the course of 60 days

* Indicates that value is significantly different from respective control ($P \leq 0.05$). Data are reported as mean ± SE

[a] Body weight (BW) change denotes change in weight from regimen start to finish: [Postexposure BW (g) − Pre-exposure BW (g)]

[b] Calculated as: [organ weight (g)/body weight (g)] × 100

Figure 6: Serum PFOS level reported by Dong (2011) Table 1.

DPAG followed the approach adopted by MDH and MDHHS and applied the PFOS specific clearance rate of 1241 days (Li 2018) and the EPA reported Vd of 0.23 L/kg to develop the DAF. DPAG agreed with MDHHS application of a UFT of 100. This produced a THSV of 0.024 mg/mL. Setting the target to protect the breast fed infant as 0.012 mg/mL (50%RSC), the MCLG for drinking water is recommended to be 8 ng/L (8PPT) to protect breast fed infants and throughout life. (Figure 7, Table 4)

PFOS	
Dose Response Modeling Method	**NOAEL**
POD	2.36 µg/mL(or 2.36 mg/L)
HED = POD x DAF (mg/kg/d)	Toxicokinetic Adjustment based on Chemical- Specific Clearance Rate (Li 2018, MDH 2020 PFOS) DAF = Vd (L/kg) x (Ln2/Half-life, days) DAF = 0.23 L/kg x (0.693/1241 days) = DAF = 0.00013 L/kg/d HED = POD x DAF (mg/kg/d) HED = 2.36 mg/L x 0.00013 L/kg/d HED = 0.000307 mg/kg/d

Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3 (DAF applied)
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	3
Total Composite (UFT)	100
RfD = HED/UFT (mg/kg/d)	RfD = HED/UFT (mg/kg/d) RfD = 0.000307 mg/kg-d/100 RfD = 3.1 ng/kg/d or 3.1x 10^{-6} mg/kg-d
THSV = POD/UFT	TSHV = 2.36 mg/L/100 TSHV = 0.024 mg/mL
Receptor	Infant exposure via breastmilk for 1 year, from mother chronically exposed via water, followed by lifetime of exposure via drinking water. Protective for short-term, subchronic and chronic. The 95th percentile water intake rates (Table 3-1 and 3-3, USEPA 2019) or upper percentile breastmilk intake rates (Table 15-1, USEPA 2019) were used. Breast-fed infant, which is also protective of a formula-fed infant using Minnesota Department of Health Model based on Goeden (2019). Placental transfer of 40% (MDH 2020 PFOS). Breastmilk transfer of 1.7% (MDH 2020 PFOS). Human Serum half-life of 1241 days (Li 2018) Volume of distribution of 0.23 L/kg (USA EPA 2016c) 95th percentile drinking water intake, consumers only, from birth to more than 21 years old (Goeden [2019]) Upper percentile (mean plus two

	standard deviations) breast milk intake rate (Goeden 2019) Time-weighted average water ingestion rate from birth to 30-35 years of age (to calculate maternal serum concentration at delivery) (Goeden 2019)
Chronic Non-Cancer MCLG	The model produces a Chronic Non-Cancer MCLG of 14 ng/L (ppt). This protects health during the growth and development of a breast fed infant. Figure 7

Table 4: Development of Non-Cancer MCLG for PFOS

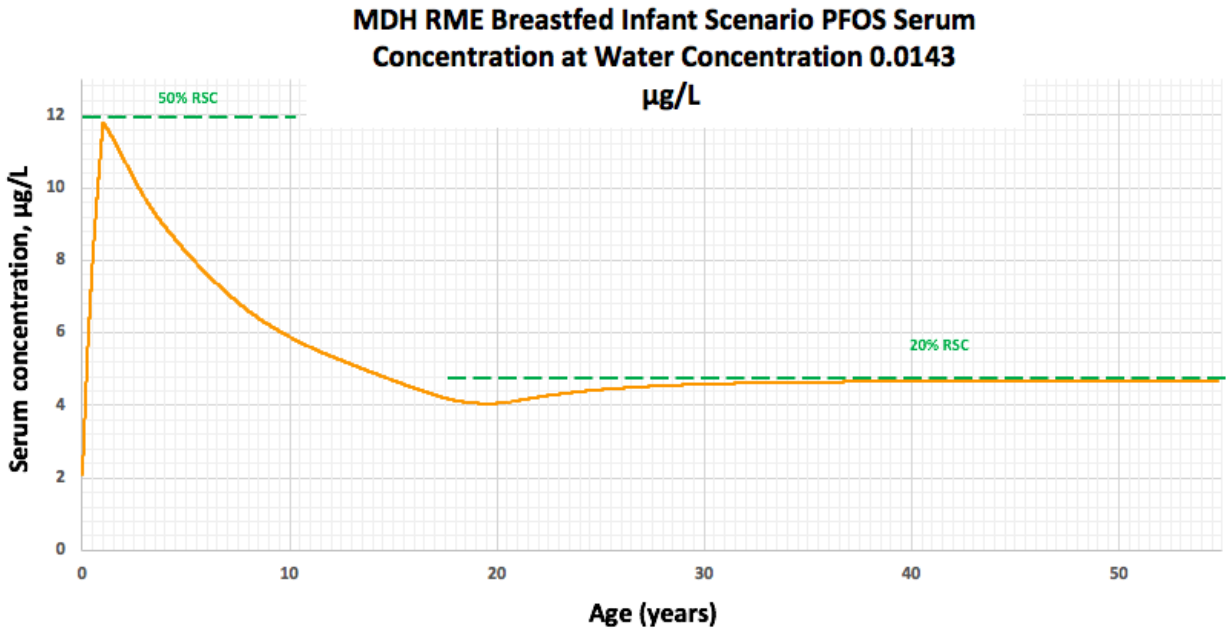

Figure 7. Using the Goeden Model, the reference dose and its parameters for PFOS were converted to an THSV of 0.024 mg/L. An RSC set at 50% means that half of this (0.012 mg/L) will be from ingested drinking water. The MCLG of PFOS in drinking water should then be set at 0.014 ug/L or 14 PPT to protect the breast fed infant from adverse health events.

6. PFNA

After a literature search and a review of the available evidence and recommendations from various agencies, the DPAG developed an MCLG recommendation for PFNA based on Non-Cancer endpoints. The critical study identified was Das (2015). ATSDR released a provisional minimal risk level for intermediate exposure based on an analysis of Das (Das 2015, Rogers 2014, Wolf 2010). The HED of the NOAEL of 1 mg/kg/d identified in the Das (2015) developmental toxicity study was selected as the POD for the ATSDR MRL. At this dose, there was no statistical difference from controls for developmental landmarks of eye opening, preputial separation in makes, and vaginal opening in females. A TWA serum PFNA concentration was estimated for dams using the serum concentration in the control group (0.015 µg/mL) as the baseline concentrations and the terminal concentration for the 1 mg/kg/d group (13.67 µg/mL) resulting in an estimated TWA serum concentration of 6.8 µg/mL. Das (2015) provided the serum concentrations directly to the ATSDR. NJDEP (2015) used the same study and the same dose of 1 mg/kg/d, but as a LOAEL for increased liver weight in pregnant mice. DPAG studied the controversy surrounding liver weight and similar effects produced by expression of peroxisome proliferator-activated receptor-alpha (PPARalpha) which is specific to rodents. DPAG agreed with ATSDR's selected POD and further agreed with Michigan's application of the Goeden transgenerational toxicokinetic model to this POD. Interestingly, the resulting MCLG is lower than the MCL determined by NJDEP (2015).

6.a. Summary of Critical Study

This study administered PFNA to pregnant CD-1 mice by oral gavage daily on gestational day 1 - 17 to assess for developmental toxicity outcomes. Treatment groups included 1 mg/kg/d, 3 mg/kg/d, 5 mg/kg/d, and 10 mg/kg/d while controls received deionized water. Mice were allocated to two groups: one group was sacrificed on GD 17 for analysis of gravity uterus, live fetuses, and maternal and fetal liver analysis. The second group was allowed to give birth and pregnancy outcomes and postnatal survival, growth, and development of the pups were monitored.

Mice in the highest 10 mg/kg/d dose group demonstrated overt toxicity beginning on GD 8. Therefore, the highest dose utilized for the remainder of the study was 5 mg/kg/d. The 3 mg/kg/d and 5 mg/kg/d groups demonstrated increased maternal weight gain as compared to controls for GD 11 to GD 17 which of the authors opined was likely due to dose-related enlargement of maternal liver. Increases in absolute and relative liver weight were seen at necropsy on GD 17 at the 1 mg/kg/d, 3 mg/kg/d, and 5 mg/kg/d doses. These changes demonstrated a dose response relationship in pregnant mice but not in non-pregnant mice. The authors noted that liver enlargement is common to PFAA exposure and it's probably mediated by activation of the PPARalpha signaling pathway.

With respect to pregnancy outcomes, there was no effect of treatment group on number of implants, number of life fetuses, or fetal weights. Absolute and relative liver weight was increased in PFNA exposed fetuses as compared to controls; however, there was no dose-response relationship. There was no effect of treatment group on

skeletal or visceral examination of fetuses. Full litter resorption occurred at the 10 mg/kg dose; however, this was associated with overt maternal toxicity, as noted above.

Postnatal survival of pups was decreased at the 5 mg/kg/d dose with deaths starting on PND 2 and only 20% of pups surviving to weaning. Treatment at the two lower dose levels did not affect pup survival. Exposure at the 3 mg/kg/d and 5 mg/kg/d was associated with decreased weight gain in pups with a dose response relationship. Decreased body weight was more persistent in male pups without any evidence of catch up growth in the post weaning period, whereas females typically recovered to control levels by 7 weeks of age. Relative liver weight was increased in pups at all treatment levels as compared to controls. This effect became less strong in the post weaning period and at PND 70 no significant effects remained. There were dose-dependent delays in postnatal development in the 3 mg/kg/d and 5 mg/kg/d groups with respect to eye opening, preputial separation, and vaginal opening.

Analysis of liver mRNA transcripts demonstrated PPARalpha-dependent gene expression in both fetal and neonatal mouse liver with activation of other transcripts regulated by other pathways. PPARalpha activation persisted to young adulthood and then declined, which the authors attributed to body burden of PFNA.

6.b. Development of MCLG

The HED of the NOAEL of 1 mg/kg/d identified in the Das (2015) developmental toxicity study was selected as the POD for the MRL. At this dose, there was no statistical difference from controls for developmental landmarks of eye opening, preputial separation in makes, and vaginal opening in females. (Figure 8)

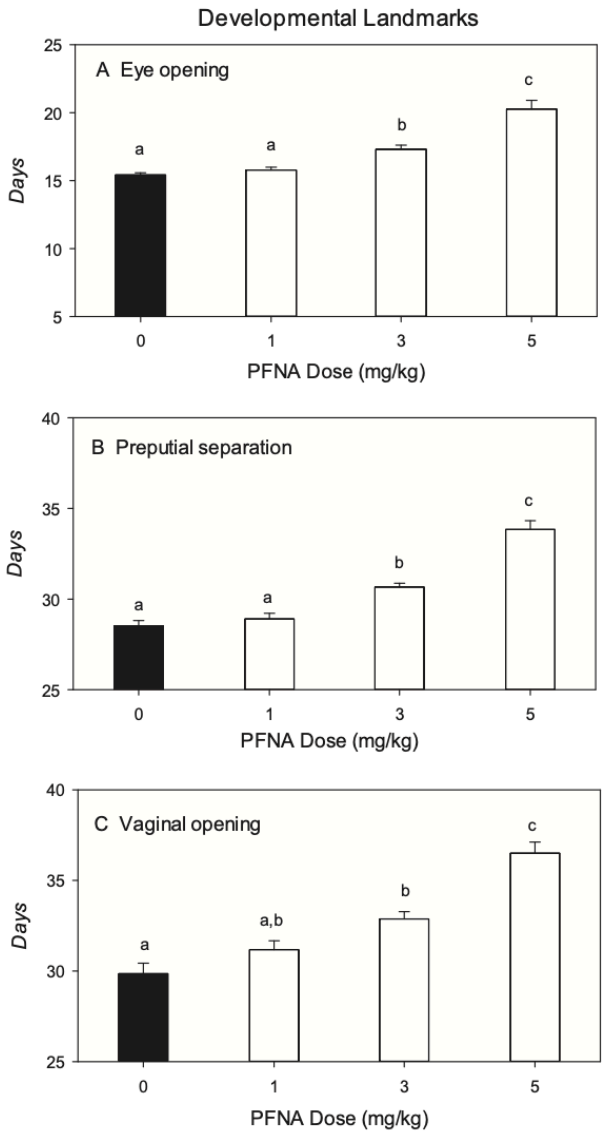

Fig. 9. Developmental landmarks of mouse offspring exposed to PFNA. Panel (A) illustrates eye opening, panel (B) preputial separation in males and panel C vaginal opening in females. ANOVA indicated a significant treatment effect. Points represent means ± S.E. of 6–13 litters. Different letters denote significant differences ($p < 0.05$) among exposure groups determined by Tukey–Kramer test.

Figure 8: PFNA NOAEL of 1 mg/kg identified by Das (2015)

A TWA serum PFNA concentration was estimated for dams using the serum concentration in the control group (0.015 µg/mL) as the baseline concentrations and the terminal concentration for the 1 mg/kg/d group (13.67 µg/mL) resulting in an estimated

TWA serum concentration of 6.8 µg/mL. Das provided the serum concentrations directly to ATSDR. (ATSDR 2018) DPAG agreed with ATSDR's selected POD and UFTs and further agreed with MDH DAF calculations and the use of Goeden transgenerational toxicokinetic model to this POD. Setting the target to protect the breast fed infant as 0.0115 mg/mL (50%RSC), the MCLG for drinking water is recommended to be 6 ng/L (6 PPT) to protect breast fed infants and throughout life. (Figure 8, Table 5)

PFNA	
Dose Response Modeling Method	**NOAEL**
POD	A NOAEL of 1 mg/kg/d was identified for developmental effects. Das (2015) The average serum concentration for NOAEL (1 mg/kg/d) was estimated (6.8 mg/L) in dams using an empirical clearance model (Wambaugh 2013).
HED_{NOAEL} = POD x DAF (mg/kg/d)	DAF = Ke x Vd Ke = 0.000489165 (4.8×10^{-4}) based on a human serum half-life of 1417 days. The human serum half-lives 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. (calculated from Zhang 2013) Vd = 0.2 L/kg (ATSDR 2018; Ohmori 2003) HED_{NOAEL} = POD x DAF (mg/kg/d) HED_{NOAEL} = POD x Ke x Vd HED_{NOAEL} = 6.8 mg/L x 0.000489165 x 0.2 L/kg HED_{NOAEL} = 0.000665 mg/kg/d
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	10
Total Composite (UFT)	300 (as per ATSDR 2018)
RfD = HED/UFT (mg/kg/d)	RfD = HED/UFT (mg/kg/d) RfD = 0.000665 mg/kg/d / 300 RfD = 2.2 ng/kg/day (2.2 x 10-6 mg/kg/d)
THSV = POD/UFT	THSV = POD/UFT THSV = 6.8 mg/L / 300

	THSV = 0.023 mg/L
Receptor	Breast-fed infant, which is also protective of a formula-fed infant Placental transfer of 69%. Breastmilk transfer of 3.2% (MDH 2020) Half-life = 1417 days (3.9 years). (Zhang 2013, MDDHS 2019, ATSDR 2018) Volume of distribution = 0.2 L/kg (ATSDR 2018, Ohmori 2003). Applied to the Goeden Model. 95th percentile drinking water intake, consumers only, from birth to more than 21 years old (Goeden 2019) Upper percentile (mean plus two standard deviations) breast milk intake rate (Goeden 2019) Time-weighted average water ingestion rate from birth to 30-35 years of age (to calculate maternal serum concentration at delivery) (Goeden 2019) 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)
Chronic Non-Cancer MCLG	The model produces a Chronic Non-Cancer MCLG of 6 ppt. This protects health during the growth and development of a breast fed infant. Figure 8

Table 5: Development of Non-Cancer MCLG for PFNA

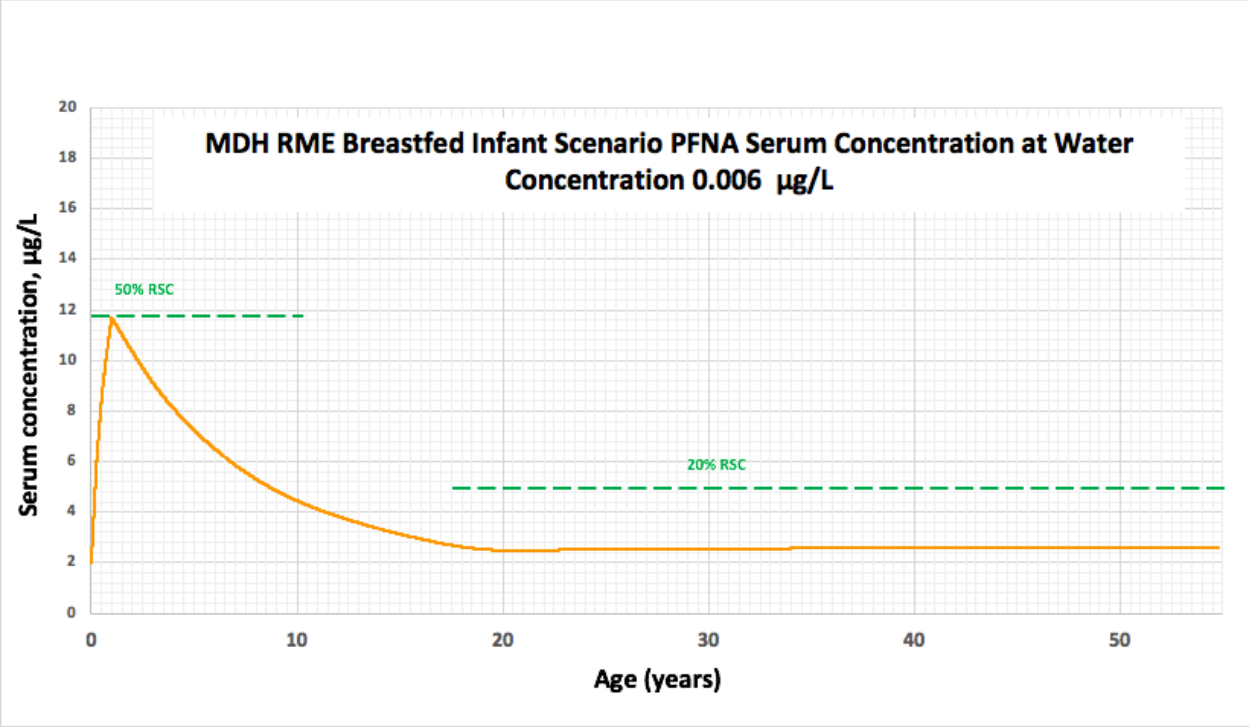

Figure 9. Using the Goeden Model, the reference dose and its parameters for PFNA were converted to an THSV of 0.023 mg/L. An RSC set at 50% means that half of this (0.0115 mg/L) will be from ingested drinking water. The MCLG of PFNA in drinking water should then be set at 0.006 ug/L or 6 PPT to protect from adverse health events.

7. PFHXs

After a literature search and a review of the available evidence and recommendations from various agencies, the DPAG developed an MCLG recommendation for PFHxS based on Non-Cancer endpoints. The critical study selected was Chang S (2018). This study identified reduced litter size following a 14 day prior to pregnancy oral exposure in Adult CD-1 female mice. Serum levels were measured at 14 days. MDHHS (MDHHS (2020 PFHXS) and NTP (2018) identified a POD of 32.4 mg/L serum concentration for male rats based on $BMDL_{20}$ analysis of this study. DPAG had selected a BMR of 10% (hence $BMDL_{10}$) as the preferred method for using BMD to select a POD and therefore rejected the use of $BMDL_{20}$. NHDES and Ali (2019) provided rigorous and more recent analysis and used a BMR of 50% of the Standard Deviation ($BMDL_{0.5SD}$). This was in keeping with EPA guidance on the selection criteria for BMRs and so was acceptable to the DPAG. The $BMDL_{0.5SD}$ derived by Ali (2019) using data from the critical study was 13.9 mg/mL and provided the basis for the MCLG.

7.a. Summary of Critical Study

This study administered potassium perfluorohexanesulfonate (PFHxS) to CD-1 mice to assess for reproductive and developmental toxicity. Both male and female mice were assigned to one of four treatment groups: control, 0.3 mg/kg/d, 1 mg/kg/d, and 3 mg/kg/d with 30 mice of each sex assigned to each treatment group. Following an acclimation period that included observation of female mice for estrous cyclicity, male

and female mice were administered vehicle control or aqueous solution of PFHxS by oral gavage daily beginning 14 days prior to cohabitation. Males were administered vehicle or treatment for a total of at least 42 days with scheduled sacrifice one day post-last dose. F_0 females were administered vehicle or treatment until lactation day 21 with scheduled sacrifice one day later. After weaning on postnatal day 21, F_1 offspring were directly dosed with PFHxS for an additional 14 days at the same respective maternal dose.

F_0 mice were observed daily for clinical signs of toxicity before and 2 hours after oral gavage dosing. No signs of clinical toxicity were noted at any of the treatment levels. Body weights and food consumption were recorded weekly. There was a significant body-weight gain in male mice at the 0.3 mg/kg/d and 1 mg/kg/d dose levels but not at the 3 mg/kg/d dose; therefore, this was not considered to be treatment-related. There were no significant differences in body-weight gain in female mice across all treatment groups. There was no significant difference in food consumption across all treatment groups in either sex.

Functional observational battery and motor activity assessment was performed on 10 mice/sex/treatment group prior to scheduled sacrifice and no significant differences were noted across the treatment groups in any of the measured outcomes or in trend of motor activity over time.

Among F_0 mice, there was no significant difference among treatment groups with respect to any of the reproductive function outcomes investigated. In males, PFHxS did not affect sperm motility, count, density, and morphology. In females, PFHxS did not affect mating index, fertility index, or precoital interval.

With respect to pregnancy outcomes in F_0 mice, there was no significant difference between treatment groups in number of implantations, mean gestation length, number of dams with viable pups, pops born to implant ratio, and sex ratio. The number of pups born per litter and mean live litter size was significantly reduced in the 1 mg/kg/d and 3 mg/kg/d as compared to controls. The authors opined that the toxicological significance of that fighting was unclear due to 1) the lack of a dose response relationship; 2) no significant difference in pup to implant ratio among treatment groups; and 3) the lack of other negative effects on developmental or reproductive outcomes.

At F_0 mice necropsy, there was no significant findings on macroscopic examinations across treatment groups. With the exception of liver weight, there was no difference across treatment groups on absolute or relative organ weights as compared to controls. PFHxS was associated with a significant, dose-dependent increase in both absolute and relative liver weight at the 1 mg/kd/d and 3 mg/kg/d in both male and female mice. This was considered to be an adaptive response.

With the exception of liver tissue, there was no difference across treatment groups in tissue histology. Liver tissue demonstrated primarily centrilobular hepatocellular hypertrophy among treatment groups with a dose-response relationship. In male mice only at the highest 3 mg/kg/d dose, mild microvesicular fatty change and minimal single-cell necrosis was noted in 6 of 10 and 4 of 10 mice, respectively. In female mice only at the highest 3 mg/kg/d dose, a low incidence of cytoplasmic vacuolation was seen in 3 out of 10 mice. Liver tissue findings were considered by the authors to be consistent with an adaptive response.

There was no difference between F_0 treatment groups with any hematology parameters or with serum TSH levels. And male mice only at the highest 3 mg/kg/d dose, there was a significant decrease in serum total cholesterol and bilirubin and a significant increase in alkaline phosphatase. This was considered to be an adaptive change related to increased metabolism of the parasites and unlikely to be of toxicological significance. There were no other significant differences in male mice in clinical chemistry parameters or in female mice in any clinical chemistry parameters.

Among F_1 mice, there was no significant difference between treatment groups on pub survival, body weight at birth or anytime thereafter, balanopreputial separation in males, vaginal patency in females, or areolae/nipple analgen retention in males. In male pups, a significantly increased anogenital distance was seen at all treatment levels as compared to controls; when adjusted to cube root body weight, a significantly increased anogenital distance was seen at the 0.3 mg/kg/d and 3 mg/kg/d treatment levels but not the 1 mg/kg/d treatment level. Among female pups, a decreased anogenital distance relative to cube root body weight was seen at the 1 mg/kg/d treatment level but no other treatment groups. The authors opined that these findings should not be considered toxic logically relevant in that no dose-response relationship was seen and that shortening of the anogenital distance rather than lengthening is indicative of anti-androgenic activity.

At F_1 mice necropsy, with the exception of liver and thyroid weight, there was no difference across treatment groups on absolute or relative organ weight as compared to controls. Absolute liver weight was significantly increased in males at the highest 3 mg/kg/d dose on PND 36 and relative liver weight was increased at the highest 3 mg/kg/d dose in males and females on PND 21 and 36. This was considered an

adaptive response. And female mice only at the highest 3 mg/kg/d dose, there was a significant increase in relative thyroid weight at PND 36 only but not on absolute thyroid weight. However, there were no thyroid histological abnormalities including hypertrophy in that group and no corresponding change in serum TSH levels.

With the exception of liver tissue, there was no difference across treatment groups in tissue histology. Liver tissue demonstrated mild centrilobular hepatocellular hypertrophy in both male and female pups with no evidence of necrosis. This was considered an adaptive response.

Analysis of liver mRNA transcript levels in F_0 and F_1 mice demonstrated increased transcripts that are sensitive to PPAR-alpha activation and CAR activation in the high-dose treatment group as compared to controls across both sexes in F_0 and F_1 mice. Cyp3a11, which is associated with PXR activation, was increased in the high-dose treatment group in F_0 males and F_1 pups of both sexes. Transcripts associated with fatty acid metabolism were increased in the high-dose treatment group across both sexes in F_0 and F_1 mice. However, transcripts associated with cellular stress were not increased.

A second toxicokinetic study was performed by the authors to determine serum and liver PFHxS concentrations at the same daily doses as the main study. The toxicokinetic study was divided into two subsets: 5 mice/sex/dose were administered PFHxS at 0.3 mg/kg/d, 1 mg/kg/d, and 3 mg/kg/d or vehicle control for 14 days prior to scheduled sacrifice. 7 mice/sex/dose were administered PFHxS at 0.3 mg/kg/d, 1 mg/kg/d, and 3 mg/kg/d or vehicle control for 14 days prior to cohabitation. Male mice were dosed for an additional 14 days with scheduled sacrifice one day post-last dose. Female mice were dosed through mating and gestation with scheduled sacrifice on gestation day 18.

Serum and liver sample collections were obtained at necropsy for male and female mice. For fetal serum and liver concentrations, pooled fetal blood and liver sample by litter were obtained at necropsy. The toxicokinetic study found that steady state observations for PFHxS were similar to that seen for PFOS as previously reported in rodent and monkey studies.

The authors concluded that it all doses studied, there was no effect of PFHxS on body weight, food consumption, estrus cyclicity, mating, fertility, gestation length, spermatogenesis, or macro and microscopic evaluation of reproductive organs in F_0 mice. A slight decrease in live litter size what is considered equivocal due to no dose response relationship and no change in the pump to implant ratio. Among F_1 mice, there was no effect of PFHxS on survival, birthweight, or reproductive development. Changes in liver weight, liver tissue microscopy, and clinical chemistry findings were all considered to be adaptive in nature.

7.b. Development of MCLG

The $BMDL_{0.5SD}$ derived by (Ali 2019) using data from the critical study of Chang (2018) was 13.9 mg/mL and provided the basis for the MCLG. (Figure 9)

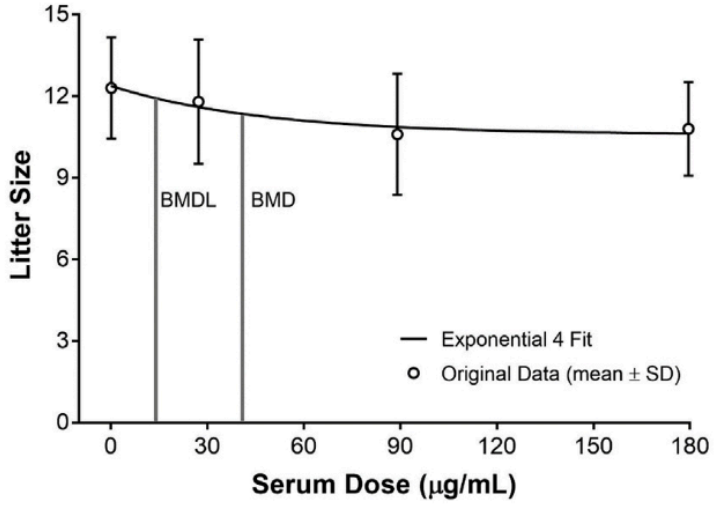

Fig. 2. Reduced litter size in female mice after 14-day oral exposure to K^+PFHxS based on summarized data from Chang et al. (2018). The curve is calculated using the normal, constant variance exponential model. In this model, the BMD is the concentration that elicits a response 0.5 times the standard deviation below the mean of the tested population. The BMDL is the concentration corresponding to the lower 95% confidence interval.

Figure 10: $BMDL_{0.5SD}$ derived by Ali (2019) of 13.9 mg/mL using data from the critical study of Chang (2018).

DPAG agreed with the DAF, UFTs, and application of the Goeden Model by MDH and MDHHS. Setting the target to protect the breast fed infant as 0.023 mg/mL (50%RSC), the MCLG for drinking water is recommended to be 20 ng/L (20 PPT) to protect breast fed infants and throughout life. (Figure 10, Table 6)

PFHxS	
Dose Response Modeling Method	**lower confidence limit on the BMD on 50% of the SD ($BMDL_{0.5SD}$)**
POD	13.9 mg/mL
HED = POD x DAF	DAF based on Chemical-Specific Clearance Rate DAF = Vd (L/kg) x (Ln2/Half- life, days) DAF = 0.25 L/kg x (Ln2/1935 days) DAF = 9.0 x 10^{-2} mL/kg/d HED = POD x DAF HED = 13.9 mg/mL x 8.61x10^{-2} mL/kg/d HED = 1.196 x 10^{-3} mg/kg/d
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3 based on application of DAF
Subchronic to Chronic (UFS)	3 based on extrapolation from Chang S (2018)
LOAEL to NOAEL (UFL)	1
Database (UFD)	3 based on small number of studies
Total Composite (UFT)	300
RfD = HED/UFT (mg/kg/d)	Reference Dose = HED /UFT Reference Dose = 1.196 x 10^{-3} mg/kg/d / 300 Reference Dose = 3.98 ng/kg/d (rounded to 4.0 ng/kg/d)
ITHSL = POD / UFT	ITHSL = 13.9 mg/mL / 300 ITHSL = 0.0463 mg/mL
Receptor	Breast-fed infant, which is also protective of a formula-fed infant. Placental transfer of 70% (MDH 2020 PFHXS). Breastmilk transfer of 1.4% (Li 2019). Half-life = 1935 days. Vd = 0.25 L/kg (USEPA 2016, Han 2012). 95th percentile drinking water intake, consumers only, from birth to more than 21 years old (Goeden [2019]) Upper percentile (mean plus two standard deviations) breast milk intake rate (Goeden 2019) Time-

	weighted average water ingestion rate from birth to 30-35 years of age (to calculate maternal serum concentration at delivery) (Goeden 2019) 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)
Chronic Non-Cancer MCLG	The model produces a Chronic Non-Cancer MCLG of 20 ppt. This protects health during the growth and development of a breast fed infant.

Table 6: Development of Non-Cancer MCLG for PFHxS

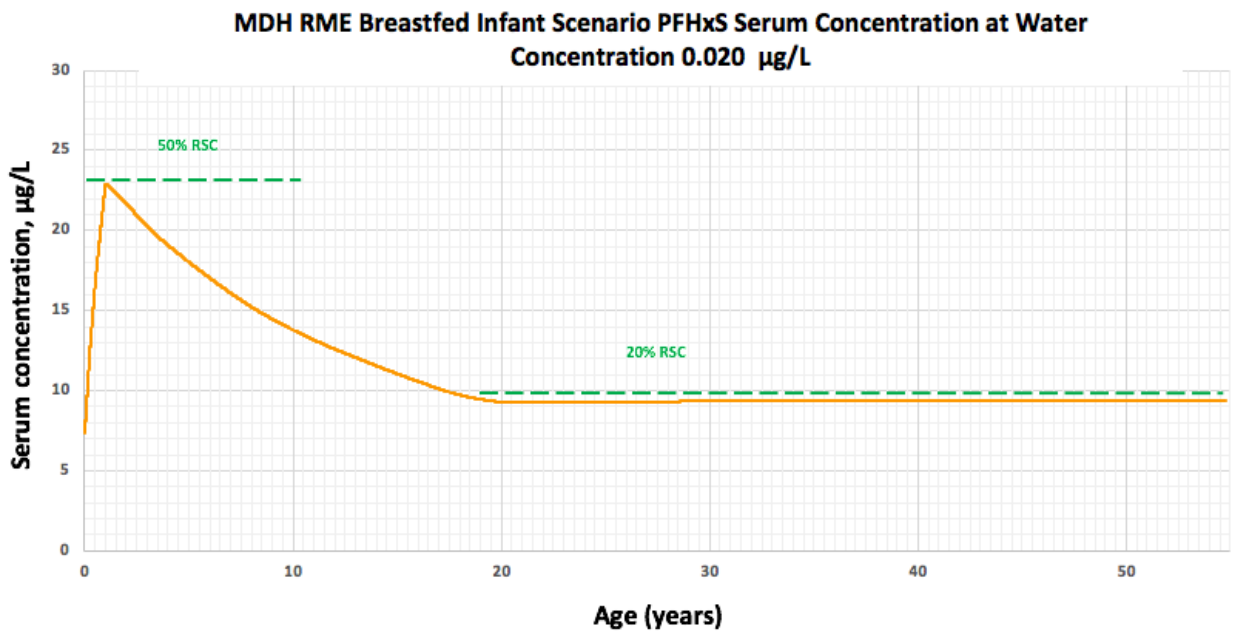

Figure 11. Using the Goeden Model, the reference dose and its parameters for PFHxS were converted to an THSV of 0.046 mg/L. An RSC set at 50% means that half of this (0.023 mg/L) will be from ingested drinking water. The MCLG of PFHXS in drinking water should then be set at 0.020 ug/L or 20 PPT to protect from adverse health events.

8. PFHpA

PFHpA is a difficult compound to develop advisories for because there is a paucity of evidence on its toxicity. The DPAG decided to base recommendations on its chemical structure. MDHHS (2019) has made similar recommendations for other PFAS that lack sufficient scientific evidence to form conclusions about health advisory levels. Like PFOA, PFHpA is a carboxylic acid. PFHpA is a 7-carbon molecule and PFOA is an 8 carbon molecule. The DPAG concludes that the MCLG for PFHpA should be conservatively set at the same threshold for PFOA – 8 PPT.

9. PFBS

After a literature search and a review of the available evidence and recommendations from various agencies, the DPAG developed an MCLG recommendation for PFBS based on Non-Cancer endpoints. The DPAG identified Feng 2017 as the critical study. The ATSDR 2018 considered the available data inadequate for identifying a critical endpoint and evaluating dose-response relationships but did not review Feng 2017. USEPA (2018 PFBS) selected Lieder (2009) and the critical effect of papillary tubular ductal epithelium hyperplasia in P0 females. They applied BMD with a BMR of 10%. The derived $BMDL_{10}$ (HED) of 11.5 mg/kg/d was modified with a UFT of 1000 to achieve a reference dose of 1x10-2 (mg/kg/d). Interestingly, USEPA (2018 PFBS) identified the decreased serum total T4 in newborn (PND 1) mice from Feng 2017 as a critical effect and performed a BMD modeling, but selected a BMR of 20%

over control response rate. The modeled $BMDL_{20}$ and applied a UFT of 300 achieved the same reference dose of 1x10-2 (mg/kg/d) as the kidney critical effect from Lieder 2009. MDHHS identified the kidney effects as a potentially compensatory response and thought the thyroid effects had greater functional significance. However, they removed the allometric scaling used in the draft USEPA (2018 PFBS) and applied the PFBS specific DAF developed by MDH. Thus, MDHHS was able to develop a chemical specific HED. However, MDH did use the $BMDL_{20}$ identified by the US EPA to calculate their HED. DPAG chose to continue with use of the $BMDL_{10}$ as the standard approach where the model fit was valid and used the USEPA (2018 PFBS) BMD modeling which, in addition to the $BMDL_{20}$, included a calculated $BMDL_{10}$ of 1.84 mg/kg/d. This $BMDL_{10}$ POD HED of 1.84 mg/kg/d was divided by 0.149 to remove the DAF employed by USEPA (2018 PFBS) prior to subjecting the data to BMD analysis (USEPA 2018 PFBS). This results in a POD of 12.35 mg/kg/d. DPAG agreed with the application of half-life ratios by MDH of the new chemical specific DAF of 316 (human serum half-life/female mouse serum half-life = 665 hours/2.1 hours = 316). (MDH 2020 PFBS) Dividing by the new chemical specific DAF of 316 (human serum half-life/female mouse serum half-life = 665 hours/2.1 hours = 316) results in a HED of 0.039 mg/kg/d.

9.a. Review of Critical Study

This study investigated the effects of prenatal perfluorobutanesulfonate (PFBS) exposure on perinatal growth and development, people on site, and reproductive and thyroid endocrine system function in female ICR mice. PFBS potassium salt was administered orally to pregnant mice at doses of 50, 200, and 500 mg/kg/d from GD1 to

GD20. Administration of the test substance did not affect weight gain, fetal loss, or behavior of the dams at the doses studied. 30 dams were assigned to one of three experimental groups: 1) sequential examination of perinatal survival and growth, pubertal onset, and ovarian and uterine development; 2) hypothalamic-pituitary-gonadal hormone and hypothalamic pituitary thyroid hormone measurements at postnatal days 1, 30, and 60; 3) measurement of serum levels of PFBS.

Postnatal day 1 body weights of female offspring at the 200 mg/kg/d dose and above were decreased relative to controls. These dose groups remained underweight throughout weaning, pubertal, and adult periods. Delays in eye-opening, vaginal opening, and first estrous period were seen in female offspring at the 200 mg/kg/d dose and above with a dose response relationship.

Absolute and relative ovary weight were decreased at the 200 mg/kg/d dose and above, although no dose response relationship was seen. Number of primordial follicles, primary follicles, secondary follicles, early actual follicles, enter follicles, pre-ovulatory follicles, and corpora lutea were decreased at the 200 mg/kg/d dose and above, although no dose response relationship was seen.

Absolute and relative uterine weight were decreased at the 200 mg/kg/d dose and above, although no dose response relationship was seen. Total uterine diameter, endometrial thickness, and myometrial thickness were decreased at the 200 mg/kg/d dose and above, with a minimal dose response relationship.

Number of days spent in diestrus stage were significantly increased in female offspring at the 200 mg/kg/d dose and above as compared to controls, although no dose response relationship was seen. Levels of serum E2 were decreased at the 200

mg/kg/d dose and above on postnatal day 30 and 60 but not on postnatal day 1 and with no dose response relationship. Levels of luteinizing hormone (LH) were decreased at the 200 mg/kg/d dose and above on postnatal day 30 but not on postnatal day 1 or 60 with no discernible dose response relationship. Levels of P4 were decreased at the 200 mg/kg/d dose and above on postnatal day 60 but not on postnatal day 1 or 30 with no discernible dose response relationship. Levels of gonadotropin-releasing hormone (GnRH) were not affected at any of the doses studied.

Total T3 and total T4 was significantly decreased in female offspring at the 200 mg/kg/d dose and above on postnatal day 1, 30 and 60, although no clear dose response relationship was seen. TSH and hypothalamic *Trh* mRNA were both increased at the 200 mg/kg/d dose and above on postnatal day 30, but not on postnatal day 1 or 60. In dams, total T4, total T3, free T4 were decreased and TSH was increased at the 200 mg/kg/d dose and above without an obvious dose response relationship.

9.b. Development of MCLG

DPAG agreed with USEPA selection of a decreased serum total T4 in newborn (PND 1) mice from Feng 2017 but used the USEPA reported $BMDL_{10}$ of 1.84 mg/kg/d.

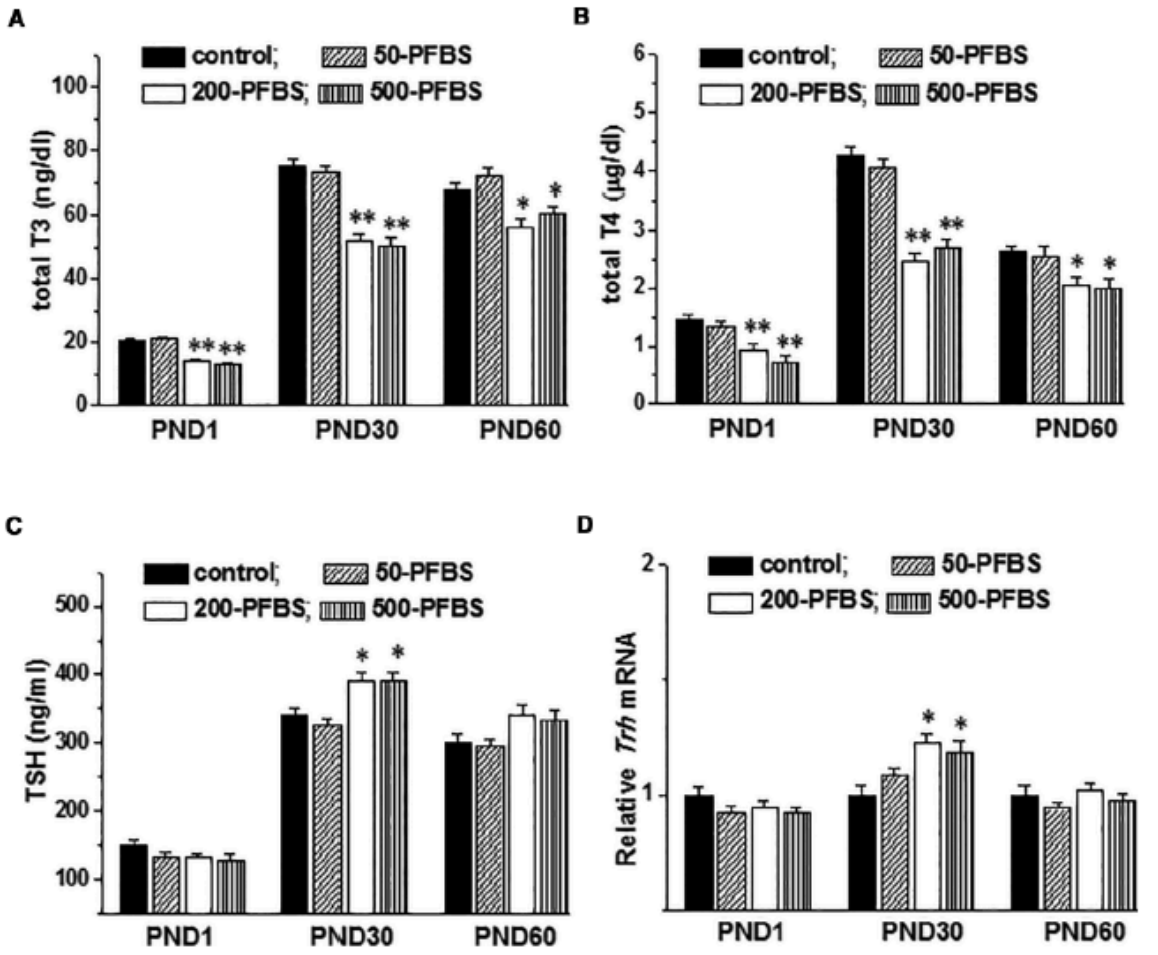

FIG. 4. Influence of prenatal perfluorobutanesulfonate (PFBS) exposure on hypothalamic–pituitary–thyroid hormone levels. Bar graphs show the levels of serum total 3,3′,5-triiodothyronine (T3) (A), total thyroxine (T4) (B), thyroid-stimulating hormone (TSH) (C) and hypothalamic *Trh* mRNA (D) in postnatal day (PND) 1, PND30, and PND60 control offspring and PFBS-offspring. *$P < 0.05$ and **$P < 0.01$ versus control offspring (1-way ANOVA).

Figure 12: Critical effect of PFBS on total thyroxine (T4) levels identified by Feng 2017 used to develop $BMDL_{10}$ POD.

This $BMDL_{10}$ POD HED of 1.84 mg/kg/d was divided by 0.149 (USEPA 2018 PFBS) page F-10 to F-13) to remove the DAF employed prior to subjecting the data to BMD analysis (USEPA 2018 PFBS). This results in a POD of 12.35 mg/kg/d. Dividing by the chemical specific DAF of 316 (human serum half-life/female mouse serum half-life = 665 hours/2.1 hours = 316) (MDH 2020 PFBS) results in a HED of 0.039 mg/kg/d. DPAG agreed with the UFT applied by USEPA. Applying the USEPA ingestion rate for

birth to < 1 year old and a conservative 20% RSC, the MCLG for drinking water is recommended to be 55 ng/L (55 PPT) to protect infants and throughout life. (Table 7)

PFBS	
Dose Response Modeling Method	**$BMDL_{10}$**
POD HED Units	US EPA reported $BMDL_{10}$ of 1.84 mg/kg/d. This was divided by 0.149 (USEPA 2018 PFBS) to derive a POD of 12.35 mg/kg/d.
POD x DAF = HED	DAF = (human serum half-life/female mouse serum half-life) DAF = 665 hours/2.1 hours DAF = 317 (MDH 2020 PFBS). HED = POD ($BMDL_{10}$) / DAF HED = 12.35 mg/kg/d / 317day. HED = 0.0390 mg/kg/d
Uncertainty Extrapolation (USEPA 2018)	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	3 A UFS of 3 is applied because the POD comes from a developmental study of mice. Although this is a susceptible life stage, additional concern over potential hazards following longer-term (chronic) cannot be completely accounted for with this study.
LOAEL to NOAEL (UFL)	1 (BMDL)
Database (UFD)	10 The database lacks studies of chronic duration, neurodevelopment, and immunotoxicity.
Total Composite (UFT)	1000
HED/UFT= Reference Dose (mg/kg-day)	39.0 ng/kg/day (0.000039 mg/kg/d)
Receptor	infant
Ingestion Rate (L/day)	Based on National Health and Nutrition Examination Survey (NHANES) 2005−2010, 95th percentile of water intake for consumers only (direct and indirect consumption) for infants (birth to <1 year old) of 1.106 L/day, per Table 3-17, USEPA Exposure Factors Handbook, 2019.

Body Weight (Kg)	An infant body weight of 7.8 kilograms was used and represents a time-weighted average for birth to 1 year old (Table 8-1, USEPA 2019).
Normalized Drinking Water Intake (L/kg-day)	0.142
Relative Source Contribution	20%
Chronic Non-Cancer MCLG	Chronic Non-Cancer MCLG = RfD x RSC / DWI Chronic Non-Cancer MCLG = 0.055 ug/L or 55 PPT

Table 7: Development of Non-Cancer MCLG for PFBS

10. GenX (HFPO dimer acid and its ammonium salt)

After a literature search and a review of the available evidence and recommendations from various agencies, the DPAG developed an MCLG recommendation for GenX based on Non-Cancer endpoints. US EPA 2018 selected the DuPont oral reproductive/developmental toxicity study in mice as the critical study. (DuPont-18405-1037, 2010). DPAG reviewed this and found it sufficiently robust to provide quality data.

US EPA selected liver effects (single-cell necrosis in male mice) as the critical effect for deriving the subchronic and chronic RfDs for GenX (HFPO dimer acid and its ammonium salt). USEPA (2018) evaluated the relevance of this endpoint in humans and noted that, per Hall, (Hall 2012) liver effects accompanied by effects such as necrosis or inflammation, among others, are indicative of liver tissue damage (USEPA, 2018). This effect is distinct from PPARα-mediated rodent hepatocarcinogenesis. US EPA performed BMD modeling with a BMR of 10%. They reported a $BMDL_{10}$ of 0.15 mg/kg/d based on BMD Multistage 2 model. DAF of 0.15 was developed using allometric scaling, per USEPA (2018 GenX) guidance, since no chemical-specific data on human serum half-life was available that would allow this conversion. Conversely, NCDEQ (NCDDHS 2017) decided against BMD modeling, stating it was statistically unreliable due to poor model fit and large confidence interval. They chose a NOAEL POD and applied a UFT of 1000 to achieve a subsequent RfD at 100 ng/kg/day.

Ultimately, DPAG adopted the approach used by the EPA to develop a HED_{BMDL10}, applied a UFT 300 and produced an RfD of 76.7 ng/kg/day. The ingestion modeling used by NCDEQ to target bottle fed infants was in keeping with the DPAG approach of targeting the most vulnerable populations for protective MCLG. The final MCLG is 108 PPT.

10.a. Review of Critical Study

This study investigated subchronic toxicity of H-28548 (HFPO dimer acid ammonium salt) in Crl:CD1(ICR) mice. Adult male and female mice were administered H-28548 at a dose of 0, 0.1, 0.5, or 5 mg/kg/d by oral gavage with a total of 10 mice per sex per dose for 96 (males) or 97 (females) days. Mice were observed daily for signs of acute toxicity. Body weight, food consumption, and detail the clinical observations were performed weekly. Ophthalmology examination, functional observational battery, and motor activity were evaluated at outset and at the conclusion of the study. Hematology and clinical chemistry studies were performed at study conclusion. Surviving mice were sacrificed and gross and microscopic pathological examinations were performed.

Body weight and body weight gain were increased in the male 5 mg/kg/d dose group relative to control, which was attributed to increased liver weight and not considered an adverse effect. No statistically significant change in body weight or body weight gain were seen any other dose groups. Food consumption and food efficiency were increased in the male 5 mg/kg/d dose group relative to control, which was attributed to increased liver weight and body weight, respectively, and not considered an adverse

effect. No statistically significant change in food consumption or food efficiency were seen any other dose groups.

No acute toxicity or test substance related deaths were seen at any of the doses studied. The test substance had no effect on functional observational battery outcomes at any of the doses studied.

Mean corpuscular hemoglobin (MCHC) was decreased in the male 5 mg/kg/d group relative to controls; because the decrease was minimal (97% of control) and there were no other statistically significant changes in red cell parameters, this outcome was considered to be spurious. Platelet count was increased in males at 0.5 and 5 mg/kg/d, but this did not demonstrate a dose-response relationship, was not associated with clinical signs or pathological changes, and was not seen in a previous 28-day gavage study and was considered to be unrelated to the test substance and not adverse. Absolute monocyte count was decreased in females at 0.1 mg/kg/d. However, similar changes were not demonstrated in the higher dose groups and this effect was considered to be not test substance related or adverse.

AST, ALT, sorbitol dehydrogenase, alkaline phosphatase and total bile acids were increased in the male 5 mg/kg/d group as compared to controls. ALT, sorbitol dehydrogenase, and alkaline phosphatase were increased in the female 5 mg/kg/d group as compared to controls. Changes in these parameters correlated with hepatocellular damage and/or cholestasis and were considered to be adverse effects related to the test substance. Significant differences in liver function parameters were not seen at the lower test doses. Total protein and albumin were increased, and total cholesterol was decreased in male mice at the 5 mg/kg/d dose, however the magnitude

of change was small, was considered to be related to the test substance but non-adverse in nature. Albumin was increased and bilirubin was decreased in the female 5 mg/kg/d group, however the magnitude of change was small and was considered to be non-adverse. Decreased Billy Rubin was also seen in male mice at the 0.5 mg/kg/d dose, but this finding was not replicated at higher doses and was considered to be spurious.

Serum potassium was decreased in male and female mice at the 5 mg/kg/d dose. The changes were not associated with any clinical signs of hypokalemia and this finding was considered to be non-adverse. Chloride was higher in male mice at the 5 mg/kg/d dose, which was considered to be unrelated to the test substance and non-adverse.

Absolute and relative liver weight were increased in male mice at the 0.5 and 5 mg/kg/d those groups relative to control, with a dose response relationship. Absolute and relative liver weight were increased in female mice at the 5 mg/kg/d dose group only. These changes were associated with gross and microscopic pathology findings and were considered to be treatment related.

Relative kidney weight as compared to brain was increased in males at the 5 mg/kg/d dose group; however, absolute and relative kidney weight as compared to body were unchanged and this finding therefore was considered to be of uncertain significance. Relative brain and epididymis weight were lower and relative heart weight as compared to brain was higher in males at the 5 mg/kg/d dose; however, absolute changes in the organ weights were not significant and these findings were not associated with any microscopic pathology findings and were considered to be not related to the test substance. Relative spleen weight was decreased in females at the

0.5 and 5 mg/kg/d dose groups; however, there was no dose response relationship or findings on microscopic pathology examination and these findings were therefore considered spurious and unrelated to the test substance. Absolute and relative ovary weight were increased in females at the 0.5 mg/kg/d dose; however, there was no dose response relationship, the increased ovary weight was attributed to ovarian cysts present in three female mice in that dose group, and this finding was therefore considered spurious and unrelated to the test substance.

There was a significant increase in enlarged and discolored livers in males at the 0.5 and 5 mg/kg/d dose group and in females at the 5 mg/kg/d dose group as compared to controls. These findings were considered to be related to the test substance. There were no other findings on gross pathology examination that were considered to be related to the test substance.

On microscopic examination, hepatocellular hypertrophy without liver cell injury was seen in male mice at the 0.5 mg/kg/d dose, which was considered to be treatment related but not adverse. Hepatocellular hypertrophy, hepatocellular single cell necrosis, and increased pigment concentration in Kupffer cells were seen in both male and and female mice at the 5 mg/kg/d dose. An increased number of mitotic figures were seen in male but not female mice at the same dose. Incidences and severity of liver changes were greater in males as compared to females. These changes correlated with clinical chemistry effects and were considered to be both treatment related and adverse effects. Minimal renal tubular epithelial hypertrophy was seen in male mice at the 5 mg/kg/d dose, but this was not associated with renal tubular cell degeneration or necrosis or any

change in clinical chemistry parameters and was therefore considered to be non-adverse. No other microscopic observations were considered to be treatment related.

An additional pharmacokinetic study was performed in which male and female adult mice were administered the same H-28548 doses at 5 mice per sex dose per timepoint and evaluated for plasma concentration of the test substance approximately two hours after dosing on test days 0, 28, and 95. These mice were also evaluated for bodyweight, food consumption, and clinical signs of overt toxicity but did not have the ophthalmology (postexposure), neurobehavioral, hematology, clinical chemistry, or pathology examinations. Test substance concentration in blood was similar on days 0, 28, and 95 and female mice indicating rapid clearance of the substance from the blood and steady state concentrations achieved on the first day of dosing. In male mice, steady state concentration was achieved by day 28.

10.b. Development of MCLG

DAPG adopted the USEPA performed BMD modeling with a BMR of 10% and a reported $BMDL_{10}$ of 0.15 mg/kg/d based on BMD Multistage 2 model. A DAF of 0.15 was developed using allometric scaling, per USEPA (2018 GenX) guidance, since no chemical-specific data on human serum half-life was available that would allow this conversion. DPAG adopted the approach used by the EPA to develop a HED_{BMDL10}, applied a UFT 300 and produced an RfD of 76.7 ng/kg/day. The ingestion modeling used by NCDHHS (2017) to target bottle fed infants was in keeping the DPAG approach of targeting the most vulnerable populations for protective MCLG (Table 8). The final MCLG is 108 PPT.

GenX	
Method of Administered Dose conversion to Internal Serum Level	BMR 10% $BMDL_{10}$ of 0.15 mg/kg/d based on BMD Multistage 2 model developed by USEPA (2018 GenX)
Method to Derive Human Equivalent Dose	Allometric DAF = $(BWA^{1/4}/BWH^{1/4})$
Dose Response Modeling Method	$BMDL_{10}$ from USEPA (2018 GenX)
HED_{BMDL10} = POD x DAF	DAF = $(BWA^{1/4}/BWH^{1/4})$ DAF = $(0.0372\ kg)^{1/4}/(80\ kg)^{1/4}$ DAF = 0.15 HED_{BMDL10} = POD ($BMDL_{10}$) x DAF HED_{BMDL10} = 0.15mg/kg/d x 0.15 HED_{BMDL10} = 0.0225 mg/kg/d
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	3
LOAEL to NOAEL (UFL)	1 (BMDL)
Database (UFD)	3 (insufficient number of studies)
Total Composite (UFT)	300
RfD = HED/UFT (mg/kg/d)	76.7 ng/kg/day (76.7 x10-6 mg/kg/d)
Receptor	Bottle fed infant
Ingestion Rate (L/day)	Based on National Health and Nutrition Examination Survey (NHANES) 2005−2010, 95th percentile of water intake for consumers only (direct and indirect consumption) for infants (birth to <1 year old) of 1.106 L/day, per Table 3-17, USEPA Exposure Factors Handbook, 2019.
Body Weight BW (Kg)	An infant body weight of 7.8 kilograms was used and represents a time-weighted average for birth to 1 year old (Table 8-1, USEPA 2019).

Normalized Drinking Water Intake (NDWI) (L/kg-day)	0.142
Relative Source Contribution (RSC)	20%
MCLG	MCLG = RfD x RSC / NDWI MCLG = 0.108 ug/L or 108 PPT

Table 8: Development of Non-Cancer MCLG for GenX

11. Summary

The DPAG had the opportunity to build on the diligent work of a great number of US and State agencies who preceded us. We strove to find the best practices wherever possible and apply them in a scientifically valid and data driven manner. As new information becomes available, we would welcome the opportunity to review these MCLG recommendations and modify when appropriate. The summary of recommendations are as follows:

1. These proposed Non-Cancer MCLGs are suggested with the health of the most vulnerable populations in mind
2. Individual MCLGs are advisable and the most scientifically rigorous approach
3. Non-Cancer MCLGs are low enough to protect against Cancer endpoints

PFAS	Reference Dose	MCLG proposed
perfluorooctanoic acid (PFOA)	3.9 ng/kg/day	8 PPT
perfluorooctanesulfonic acid (PFOS)	3.1 ng/kg/day	14 PPT
perfluorononanoic acid (PFNA)	2.2 ng/kg/day	6 PPT
perfluorohexanesulfonic acid (PFHxS)	4.0 ng/kg/day	20 PPT
perfluoroheptanoic acid (PFHpA)	None derived	8 PPT
perfluorobutanesulfonic acid (PFBS)	39 ng/kg/day	55 PPT
ammonium salt of hexafluoropropylene oxide dimer (GenX)	75 ng/kg/day	108 PPT

We would like to thank the Commonwealth of Pennsylvania and the Pennsylvania Department of Environmental Protection for the opportunity to participate in this important work and protect the health and safety of Pennsylvanians.

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Appendix A: Drexel PFAS Advisory Group (DPAG)

Drexel PFAS Advisory Group (DPAG) adhered an evidence-based approach in developing its proposal. (Institute of Medicine (2011), NRC (2009)) The process was transparent and reviewed by PADEP at regular intervals. No member disclosed a conflict of interest. The panel was multidisciplinary and included a wide array of expertise. Literature and scientific evidence were reviewed with a systematic approach that rated the quality of the evidence, grade the strength of recommendations, incorporate values and preferences, and acknowledge differences in opinion. Recommendations were articulated in a structured framework repeatable across each PFA examined. They are now submitted for external review by DEP.

Project Leader and Medical Toxicologist:

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- Rita McKeever MD FAAEM, FACMT, Associate Professor of Emergency Medicine, Drexel University College of Medicine. Board Certified in Medical

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Expert Panel:

- Charles N Haas Ph.D - LD Betz Professor of Environmental Engineering & Head, Dept. of Civil, Architectural & Environmental Engineering, Drexel University
- Christopher Sales Ph.D. Assistant Professor, Architectural & Environmental Engineering, Drexel University
- Marie Kurtz PhD, Senior Scientist; Assistant Research Professor, Academy of Natural Sciences, Drexel University
- Esther D. Chernak, MD, MPH Associate Clinical Professor, Drexel University College of Medicine and Dornsife School of Public Health
- Tom Hipper, MSPH, MA Adjunct Professor, Program Manager of the Center for Public Health Readiness and Communication Dornsife School of Public Health, Drexel University

Appendix B: Acronyms and Abbreviations List

ATSDR: Agency for Toxic Substances and Disease Registry
BMD: benchmark dose
BMDL: lower confidence limit on the benchmark dose
BMR: benchmark response
BW: body weight
Bwa: body weight animal
BWh: body weight human
CDC: Centers for Disease Control and Prevention
CEPA: California Environmental Protection Agency
DPAG: Drexel PFAS Advisory Group
DAF: dosimetric adjustment factor
GD: gestational day
GenX: ammonium salt of hexafluoropropylene oxide dimer
HBV: health-based value
HED: human equivalent dose
$HED_{LOAEL:}$ HED determined by LOAEL
$HED_{BMDL10:}$ HED determined by a BMR of 10%
$HED_{BMDL0.5SD:}$ HED determined by a BMR of 50% of SD
HFPO: hexafluoropropylene oxide
HRA: health risk assessment
THSV = Internal Target Human Serum Value
kg: kilogram
L: liter
LD: lactation day
LHA: lifetime health advisory
LOAEL: lowest observed adverse effect level
MCL: Maximum Contaminant Level
MDH: Minnesota Department of Health
MDHHS: Michigan Department of Health and Human Services
mg: milligram
mg/kg/d: milligrams per kilogram per day
MI: Michigan
ml: milliliter
MPART: Michigan PFAS Action Response Team
NCDHHS: North Carolina Department of Health and Human Services
NHDES: New Hampshire Department of Environmental Services
NHANES: National Health and Nutrition Examination Survey
NJDEP: New Jersey Department of Environmental Protection
ng: nanogram
NOAEL: no observed adverse effect level
OECD: Organization for Economic Co-operation and Development
PA DEP: Pennsylvania Department of Environmental Protection
PFAS: per- and polyfluoroalkyl substances
PFBS: perfluorobutane sulfonic acid
PFHpA : perfluoroheptanoic acid
PFHxA: perfluorohexanoic acid
PFHxS: perfluorohexane sulfonic acid
PFNA: perfluorononanoic acid
PFOA: perfluorooctanoic acid
PFOS: perfluorooctane sulfonic acid
PND: postnatal day
POD: point of departure
PODHED: point of departure human equivalent dose
PPAR: peroxisome proliferator-activated receptor
ppt: parts per trillion
RfD: reference dose
RSC: relative source contribution
TWA: time weighted average
UF: uncertainty factor
µg: microgram
USEPA: United States Environmental Protection Agency

Drexel PFAS Workbook June, 2020

Table of Contents

Definition of Point of Departure (POD) and How to Use It to Calculate Toxicological Reference Dose (RfD)

In toxicology, point of departure (POD) is defined as the point on a toxicological dose-response curve established from experimental data or observational data generally corresponding to an estimated no effect level. It marks the beginning of extrapolation to toxicological reference dose RfD.
US EPA defines RfD as an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral or dermal exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Its unit is usually mg/kg bw/day or mg/kg/day.

BMD = Benchmark Dose (Definition: A dose or concentration that produces a predetermined change in response rate of an adverse effect (called the benchmark response or BMR) compared to background.)
Acronym:

BMR = Benchmark Response (Definition: An adverse effect, used to define a benchmark dose from which an RfD (or RfC) can be developed. The change in response rate over background of the BMR is usually in the range of 5-10%, which is the limit of responses typically observed in well-conducted animal experiments.)

POD = Point of Departure (Definition: The dose-response point that marks the beginning of a low-dose extrapolation. This point can be the lower bound on dose for an estimated incidence or a change in response level from a dose-response model (BMD), or a NOAEL or LOAEL for an observed incidence, or change in level of response.)

The most typical POD used to derive RfD is no-observed-adverse-effect level (NOAEL), lowest-observed-adverse-effect level (LOAEL), or statistical benchmark dose (BMD). Benchmark Dose is derived by entering raw experimental data into a statistical package to determine what dose will cause a certain percentage adverse response. BMD10 for example would be a 10% response compared to an unexposed population. The EPA prefers BMD as the primary means of calculating POD, but data available is not always sufficient to support this approach. In those cases, a LOAEL

RfD values can be calculated by dividing the point of departure with corresponding uncertainty factors (UF). Differences chronic dose response studies are used (often with adjustment factors) to to derive chronic reference dose is necessary. Sometimes, you have to modify the point of departure first before using the equation below.

RfD = [POD * (Adjustment factors)] / Uncertainty Factor * Uncertainty Factor * Uncertainty Factor….

Uncertainty factors are used to address the differences between the experimental data and the human exposure scenarios. They include uncertainties for interspecies differences, intraspecies differences, differences in duration of exposure, issues related to dose-response, quality of data. They are expressed as orders of magnitude of ten. For example, 100 (or 1), 100.5 (or 3), 101 (or 10), 102 (or 100).

From the RfD, a Threshold Level (or Health Advisory Level, MCL, MCLG, etc depending on the authority) is determined by adjusting for the daily water intake (DWI), body weight, and the Relative Source Contribution (percentage of intake from water that is expected to contribute to the body burden of the substance).

Threshold Level = RfD x (Body Weight/Daily Water Intake) x Relative Source Contribution

	Advisory level in PPT	
	PFOA	PFOS
EPA	70*	70*
CA	2	7
MI	10	16
NY	10	10
NH	12	15
NJ	14	13
MA	20*	20*
VT	20	20
MN	35	15
*max sum for all PFAS species		

Table 1: showing PFOA and PFOS Health Advisory Levels (HAL) by State

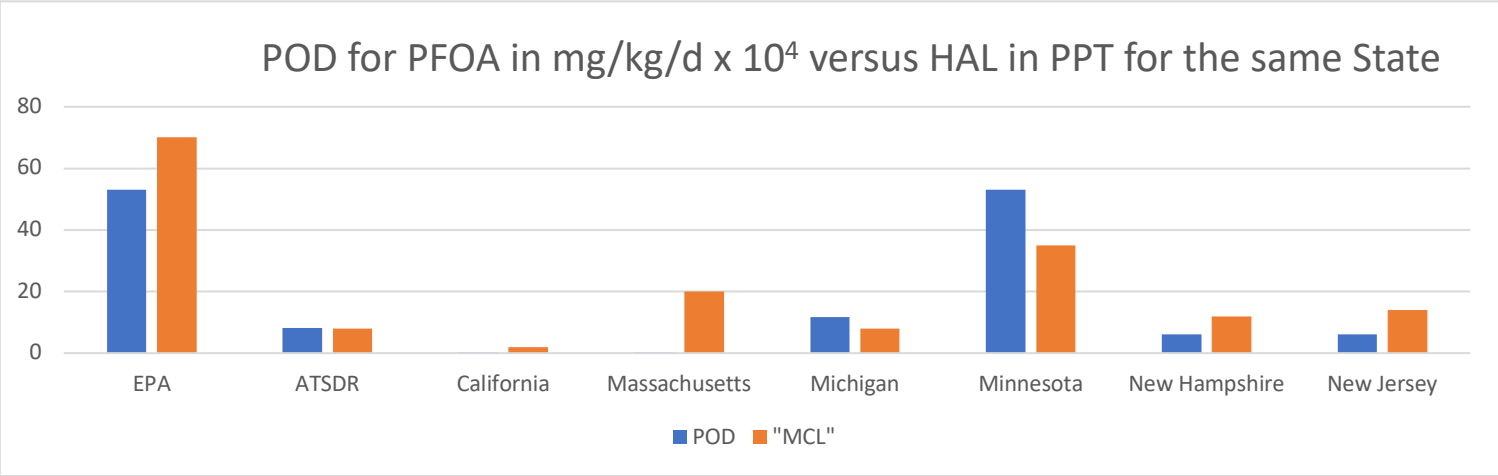

Figure 1: How POD and HAL relate for PFOA by State.

How to use this workbook

Health recommendations are classified by type of PFAS and by State/Authority. The pattern is generally the same State to State but there are notable differences in the adjustment factors, uncertainty factors used, and methods to determine water intake.

1. Critical effect selected

3. POD adjusted by using preferred methods to derive Human Equivalent Dose (HED)

5. Final adjustment made based on intake to derive Threshold Level (e.g. MCL, MCLG, HAL etc)

PFOA	
US EPA	
Office of Water 2016	
Standard / Guidance	Health Advisory
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	0.07 ug/L 70 PPT (PFOA + PFOS cannot exceed this level)
Key Study Information	
Critical Effect Key Study Reference	Developmental (reduced ossification, accelerated puberty) Lau, C., J.R. Thibodeaux, R.G. Hanson, M.G. Narotsky, J.M. Rogers, A.B. Lindstrom, and M.J. Strynar. 2006. Effects of perfluorooctanoic acid exposure during pregnancy in the mouse. Toxicological Science 90:510–518.
Species	Mice
Study Exposure Duration (days)	17 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Modeled AUC
Method to Derive Human Equivalent Dose	Dose adjustment factor of 0.00014 L/kg-day, based on first order kinetic clearance rate (Vd x (ln 2 ÷ t½))
Dose-Response	
Dose Response Modeling Method	LOAEL
POD	38 ug/mL
POD x DAF = Human Equiv Dose	0.0053 mg/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	10
Database (UFD)	1
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	(2 x 10-5 mg/kg-day) or 20 ng/kg/d
Receptor	Lactating women
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg-day)	0.054
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	0.07 ug/L 70 PPT (PFOA + PFOA cannot exceed this level)
Additional Information	90th percentile consumers only estimate of combined direct and indirect community water ingestion for lactating women (see Table 3-81 in USEPA 2011b).
Reference	Health Effects Support Document for Perfluorooctanoic Acid, U.S. Environmental Protection Agency Office of Water (4304T) Health and Ecological Criteria Division, EPA Document Number: 822-R-16-003. May 2016. and Drinking Water Health Advisory for Perfluorooctanoic Acid, U.S. Environmental Protection Agency Office of Water (4304T) Health and Ecological Criteria Division, EPA Document Number: 822-R-16-005. May 2016 https://www.epa.gov/ground-water-and-drinking-water/drinking-water-health-advisories-pfoa-and-pfos

2. POD determined by critical review of study

4. HED divided by Uncertainty Factors to achieve Reference Dose RfD in target population

SUBSTANCE	
STATE	
AUTHORITY AND YEAR	
Standard / Guidance	MCL, HA
Media Type	GW, DW
Threshold Level (ug/L) or (PPT)	Recommendation expressed as ug/L or PPT (repeated below)
Key Study Information	
Critical Effect Key Study Reference	The effect and study are listed here
Species	e.g. mice, rats. Monkeys, etc
Study Exposure Duration (days)	in days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	If there was not a measurable serum level, how was the dose converted to a serum level
Method to Derive Human Equivalent Dose	What method was used to derive the human equivalent dose – e..g. how was the Dose Adjustment Factor (DAF) calculated
Dose-Response	
Dose Response Modeling Method	Benchmark Dose, NOAEL, or LOAEL
POD	POD is listed here
POD x DAF = HED	The HED is calculated here by multiplying the POD by the Dose Adjustment Factor
Uncertainty Extrapolation	
Human Variability (UFH)	Set by the toxicologist interpreting the data
Animal to Human (UFA)	Set by the toxicologist interpreting the data
Subchronic to Chronic (UFS)	Set by the toxicologist interpreting the data
LOAEL to NOAEL (UFL)	Set by the toxicologist interpreting the data
Database (UFD)	Set by the toxicologist interpreting the data
Total Composite (UFT)	The final multiplication of all the UF's
HED/UFT= Reference Dose (mg/kg-day)	The HED is divided by the UFT here
Receptor	Who did they consider (adult, infant, child, breast fed, bottle fed)
Exposure	
Ingestion Rate (L/day)	How many liters a day they assume a person drinks (2L for adult 1 L for child typical)
Body Weight (Kg)	Typically 70 kg adult
Normalized Drinking Water Intake (L/kg/day)	Ingestion rate divided by weight
RSC (Relative Source Contribution)	How much of the PFAS are assumed to come from water as a percentage
Threshold Level (ug/L) or (PPT)	Reference Dose x (Ingestion rate/ Body Weight) x RSC (although not all use this method) Recommendation expressed as ug/L or PPT (repeated above)
Additional Information	
Reference	

PFOA

PFOA	
Canada	
Standard / Guidance	Health Based Value
Media Type	Ground Water and Drinking Water
Threshold Level (ug/L) or (PPT)	0.200 ug/L 200 PPT
Key Study Information	
Critical Effect Key Study Reference	Liver hypertrophy Perkins R, Butenhoff J, Kennedy G, Palazzolo M. 2004. 13- Week dietary toxicity study of ammonium perfluorooctanoate (APFO) in male rats. Drug Chem. Toxicology., 27:361-378.
Species	Rates
Study Exposure Duration (days)	13 weeks (91 days)
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Used administered dose
Method to Derive Human Equivalent Dose	Adjustment of UFA (termed "AK_{UF}") using ratios of PBPK model-(Loccisano 2011, 2012a,b, 2013) predicted dose metrics, using steady-state plasma concentrations. These chemical-specific adjustment factors (CSAFs) and PBPK modelling were used to derive an AK_{UF} reflecting interspecies toxicokinetic differences AK_{UF} = CLanimal/ CLhuman [CL is clearance (e.g., mL/kg bw per day)]
Dose-Response	
Dose Response Modeling Method	Benchmark Dose Modeling
POD	0.05 mg/kg per day is the BMDL10 for hepatocellular hypertrophy
POD x DAF = HED	0.000521 mg/kg-day = (0.05 mg/kg per day) / 96 96 is the dose-specific AKUF for rats in the 0.01 mg/kg bw per day range
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	2.5
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	1
Total Composite (UFT)	25
Toxicity Value RfD (mg/kg-day)	
Receptor	Adult
Exposure	
Ingestion Rate (L/day)	1.5
Body Weight (Kg)	70
Normalized Drinking Water Intake (L/kg-day)	0.02
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	0.200 ug/L 200 PPT
Additional Information	An interspecies uncertainty factor of 2.5 was used to reflect only the toxicodynamic component of the default interspecies uncertainty factor, because the toxicokinetic differences between rats and humans were already incorporated when calculating the PODHEQ. Likewise, a default value of 10 was applied for the intraspecies UF. If further studies of PFOA consistently indicate a 10-fold difference in pharmacokinetics within the population, a higher intraspecies UF might be warranted to ensure that pharmacodynamic differences between humans are also quantitatively addressed. No uncertainty factor was used for subchronic-to-chronic extrapolation, as liver effects were investigated in a chronic study (Butenhoff et al., 2012b), and increasing duration of exposure did not appear to worsen the effects in the key study (Perkins et al., 2004).
Reference	Health Canada. Guidelines for Canadian Drinking Water Quality, Guideline Technical Document, Perfluorooctanoic Acid. December 2018 Loccisano AE, Campbell JL, Jr., Butenhoff JL, et al. 2012a. Comparison and evaluation of pharmacokinetics of PFOA and PFOS in the adult rat using a physiologically based pharmacokinetic model. Reprod Toxicol 33(4):452-467.Loccisano AE, Campbell JL, Jr., Butenhoff JL, et al. 2012b. Evaluation of placental and lactational pharmacokinetics of PFOA and PFOS in the pregnant, lactating, fetal and neonatal rat using a physiologically based pharmacokinetic model. Reprod Toxicol 33(4):468-490. Loccisano AE, Longnecker MP, Campbell JL, Jr., et al. 2013. Development of PBPK models for PFOA and PFOS for human pregnancy and lactation life stages. J Toxicol Environ Health A 76(1):25-57.

PFOA	
US EPA	
Office of Water 2016	
Standard / Guidance	Health Advisory
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	0.07 ug/L 70 PPT (PFOA + PFOS cannot exceed this level)
Key Study Information	
Critical Effect Key Study Reference	Developmental (reduced ossification, accelerated puberty) Lau, C., J.R. Thibodeaux, R.G. Hanson, M.G. Narotsky, J.M. Rogers, A.B. Lindstrom, and M.J. Strynar. 2006. Effects of perfluorooctanoic acid exposure during pregnancy in the mouse. Toxicological Science 90:510–518.
Species	Mice
Study Exposure Duration (days)	17 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Modeled AUC
Method to Derive Human Equivalent Dose	Dose adjustment factor of 0.00014 L/kg-day, based on first order kinetic clearance rate (Vd x (ln 2 ÷ t½))
Dose-Response	
Dose Response Modeling Method	LOAEL
POD	38 mg/L
POD x DAF = Human Equiv Dose	0.0053 mg/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	10
Database (UFD)	1
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	(2 x 10-5 mg/kg-day) or 20 ng/kg/d
Receptor	Lactating women
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg-day)	0.054
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	0.07 ug/L 70 PPT (PFOA + PFOA cann ot exceed this level)
Additional Information	90th percentile consumers only estimate of combined direct and indirect community water ingestion for lactating women (see Table 3-81 in USEPA 2011b).
Reference	Health Effects Support Document for Perfluorooctanoic Acid, U.S. Environmental Protection Agency Office of Water (4304T) Health and Ecological Criteria Division, EPA Document Number: 822-R-16-003. May 2016. and Drinking Water Health Advisory for Perfluorooctanoic Acid, U.S. Environmental Protection Agency Office of Water (4304T) Health and Ecological Criteria Division, EPA Document Number: 822-R-16-005. May 2016 https://www.epa.gov/ground-water-and-drinking-water/drinking-water-health-advisories-pfoa-and-pfos

PFOA	
US DHHS	
ATSDR DRAFT June 2018	
Standard / Guidance	Minimal Risk Level
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	None at present
Key Study Information	
Critical Effect Key Study Reference	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. Pregnant C57BL/6 mice were exposed to 0 or 0.3 mg PFOA/kg/day throughout pregnancy. The critical effects considered were Neurobehavioral effects (decreased number of inactive periods, altered novelty induced activity) at 5-8 weeks of age. Koskela A, Finnilä MA, Korkalainen M, Spulber S, Koponen J, Håkansson 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. Pregnant C57BL/6 mice were exposed to PFOA mixed with food at the dose of 0 or 0.3 mg PFOA/kg/day throughout pregnancy. Group of five offspring (female) were sacrificed at either 13 or 17 months of age. The critical effects considered were skeletal alteration such as bone morphology and bone cell differentiation in the femurs and tibias.
Species	Pregnant C57BL/6 mice
Study Exposure Duration (days)	18 days maternal, 17 days pups
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	The average serum concentration was estimated in the mice (8.29 mg/L) using a three-compartment pharmacokinetic model (Wambaugh et al. 2013) using animal species-, strain-, sex-specific parameters.
Method to Derive Human Equivalent Dose	LOAEL HED = (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 (Thompson et al. 2010)
Dose-Response	
Dose Response Modeling Method	LOAEL
POD	8.29 mg/L
POD x DAF = Human Equiv Dose	0.000821 mg/kg/day or 8.21×10^{-4} mg/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	10
Database (UFD)	1
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	2.7×10^{-6} mg/kg/day (rounded to 3.0×10^{-6} mg/kg/day
Receptor	None selected at present
Exposure	
Ingestion Rate (L/day)	Not determined at present
Body Weight (Kg)	Assuming the ATSDR uses the EPA methodology the Threshold Level would be 8 PPT
Normalized Drinking Water Intake (L/kg-day)	
Relative Source Contribution	
Threshold Level (ug/L) or (PPT)	*8 PPT presumptive*
Additional Information	Draft Commentary awaiting further review
Reference	https://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=1117&tid=237

PFOA	
ALASKA	
Dept. of Environmental Conservation 2019	
Standard / Guidance	Action level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Additional Information	
Reference	https://dec.alaska.gov/spar/csp/pfas/

PFOA	
ALABAMA	
ADEM 2019	
Standard / Guidance	Action level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Additional Information	
Reference	http://adem.alabama.gov/newsEvents/reports/PFASinAlabama.pdf

PFOA	
California	
August 2019	
Standard / Guidance	Noncancer Notification Levels Guidance
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.002 ug/L L or 2 ppt
Key Study Information	
Critical Effect Key Study Reference	Li K, Sun J, Yang J, et al. (2017). Molecular Mechanisms of Perfluorooctanoate- Induced Hepatocyte Apoptosis in Mice Using Proteomic Techniques. Environ Sci Technol 51(19): 11380-11389. Based on hepatic mitochondrial membrane potential changes and increased apoptosis and oxidative DNA damage
Species	Male and female Balb/c mice
Study Exposure Duration (days)	28 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	LOAEL is 0.05 mg/kg-day which corresponds to a serum concentration of 0.97 mg/L
Method to Derive Human Equivalent Dose	Dose adjustment factor of 0.00014 L/kg-day, based on first order kinetic clearance rate (Vd x (ln 2 ÷ t½))
Dose-Response	
Dose Response Modeling Method	LOAEL
POD	0.97 mg/L
PODxDAF=HED (mg/kg/day)	1.35 10-4 mg/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	3
Database (UFD)	3 (potential for developmental toxicity at the point of departure)
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	0.45 ng/kg-day or (0.45 X 10-6 mg/kg/day)
Receptor	
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	0.053 L/kg-day
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	2 ng/L or 2 ppt
Additional Information	Note: California uses an intermediate step called ADD or acceptable daily dose which is expressed as a target serum level and then a dose. This corresponds to the Reference Dose in this table
Reference	Notification Level Recommendations for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS) https://oehha.ca.gov/media/downloads/water/chemicals/nl/final-pfoa-pfosnl082119.pdf

PFOA	
California	
August 2019	
Standard / Guidance	**Cancer Reference Level**
Media Type	one in one million cancer risk from PFOA in tap water
Threshold Level (ug/L) or (PPT)	0.0001 ug/L or 0.1 ppt
Key Study Information	
Critical Effect Key Study Reference	NTP (2018c). TR-598: Technical Report Pathology Tables and Curves - PFOA. National Toxicology Program, Research Triangle Park, North Carolina. https://tools.niehs.nih.gov/cebs3/views/?action=main.dataReview&bin_id=13658 (last accessed March 20, 2019).
Species	
Study Exposure Duration (days)	
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Using the HEDs as the dose metric, multisite benchmark dose modeling was performed to determine the cancer slope factor (CSF) for the hepatic and pancreatic tumors in male rats.
Method to Derive Human Equivalent Dose	
Dose-Response	
Dose Response Modeling Method	$BMDL_{05(human)} = BMDL_{05(animal)} \times (BW_{animal}/BW_{human})^{1/8}$
POD	$BMDL_{05\ animal}$ of 0.000648 mg/kg-day
PODxDAF=HED (mg/kg/day)	$BMDL_{05(human)}$ is 3.5×10^{-4} mg/kg-day. CSF = BMR ÷ $BMDL_{05}$ = 0.05 ÷ 3.5×10^{-4} mg/kg-day = 143 $(mg/kg\text{-}day)^{-1}$
Uncertainty Extrapolation	
Human Variability (UFH)	
Animal to Human (UFA)	
Subchronic to Chronic (UFS)	
LOAEL to NOAEL (UFL)	
Database (UFD)	
Total Composite (UFT)	
HED/UFT= Reference Dose (mg/kg-day)	RL = R ÷ (CSF X DWI) R = default risk level of one in one million, or 10^{-6} RL = 10^{-6} ÷ $(143\ (mg/kg\text{-}day)^{-1}$. 0.053 L/kg-day) = 1.3 . 10^{-7} mg/L
Receptor	All ages: Age sensitivity factors (ASFs) were not applied
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	0.053 L/kg-day
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	0.0001 ug/L or 0.1 ppt (1.3×10^{-7} mg/L)
Additional Information	OEHHA recommends that SWRCB set the final NLs at the lowest levels at which PFOA and PFOS can be reliably detected in drinking water using currently available and appropriate technologies.
Reference	Notification Level Recommendations for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS) https://oehha.ca.gov/media/downloads/water/chemicals/nl/final-pfoa-pfosnl082119.pdf

PFOA	
Colorado	
CPHE 2018	
Standard / Guidance	Action level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Additional Information	
Reference	https://www.colorado.gov/pacific/cdphe/PFCs/health/advisory

PFOA	
Connecticut	
CT DPH 2019	
Standard / Guidance	Action level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA
Additional Information	
Reference	https://portal.ct.gov/DPH/Drinking-Water/DWS/Per--and-Polyfluoroalkyl-Substances

PFOA	
Delaware	
DNREC-DWHS 2018	
Standard / Guidance	Health Advisory Level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Additional Information	
Reference	http://www.dnrec.delaware.gov/dwhs/SIRB/Documents/DWHS%20PFAS%20Sampling%20Policy.pdf

PFOA	
Florida	
DOH 2016	
Standard / Guidance	Health Advisory Level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Additional Information	
Reference	http://www.floridahealth.gov/environmental-health/drinking-water/_documents/pfoa-pfos-fs-20161.pdf

PFOA	
Idaho	
DEQ 2017	
Standard / Guidance	Health Advisory Level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Additional Information	
Reference	https://www.deq.idaho.gov/water-quality/drinking-water/drinking-water-health-advisories/

PFOA	
Iowa	
DNR 2019	
Standard / Guidance	Health Advisory Level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT PFOA
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT PFOA
Additional Information	
Reference	https://programs.iowadnr.gov/riskcalc/Chemical/Index/286

PFOA	
Maine	
DEP 2020	
Standard / Guidance	RAG
Media Type	DW
Threshold Level (ug/L) or (PPT)	PFOA exceeds 0.070 ug/L or 70 or sum of all PFAS exceeds 0.4 ug/L or 400 PPT
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	PFOA exceeds 0.070 ug/L or 70 or sum of all PFAS exceeds 0.4 ug/L or 400 PPT
Additional Information	
Reference	https://www.maine.gov/pfastaskforce/materials/report/PFAS-Task-Force-Report-FINAL-Jan2020.pdf

PFOA	
Maine	
PFAS Task Force 2020	
Standard / Guidance	Health Advisory
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT for PFOS + PFOA, 0.4 ug/L or 400 PPT for all PFAS combined
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	
Relative Source Contribution	
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT for PFOS + PFOA, 0.4 ug/L or 400 PPT for all PFAS combined
Additional Information	
Reference	https://www1.maine.gov/pfastaskforce/materials/report/PFAS-Task-Force-Report-FINAL-Jan2020.pdf

PFOA	
Massachusetts	
DEP 2019	
Standard / Guidance	MCL
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.020 ug/L or 20 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA + PFDA
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	10
Database (UFD)	1
Total Composite (UFT)	300 x 3 = 900
HED/UFT= Reference Dose (mg/kg-day)	5×10^{-6} (mg/kg-day)
Receptor	pregnant women, nursing mothers and infants
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.020 ug/L or 20 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA + PFDA
Additional Information	
Reference	https://www.mass.gov/doc/310-cmr-2200-pfas-amendments/download

PFOA	
Michigan	
Michigan Science Advisory Group 2019	
Standard / Guidance	Health Based Values
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	0.008 ug/L or 8 PPT
Key Study Information	
Critical Effect Key Study Reference	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. Pregnant C57BL/6 mice were exposed to 0 or 0.3 mg PFOA/kg/day throughout pregnancy. The critical effects considered were Neurobehavioral effects (decreased number of inactive periods, altered novelty induced activity) at 5-8 weeks of age. Koskela A, Finnilä MA, Korkalainen M, Spulber S, Koponen J, Håkansson 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. Pregnant C57BL/6 mice were exposed to PFOA mixed with food at the dose of 0 or 0.3 mg PFOA/kg/day throughout pregnancy. Group of five offspring (female) were sacrificed at either 13 or 17 months of age. The critical effects considered were skeletal alteration such as bone morphology and bone cell differentiation in the femurs and tibias.
Species	Pregnant C57BL/6 mice
Study Exposure Duration (days)	18 days maternal, 17 days pups
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	The average serum concentration was estimated in the mice (8.29 mg/L) using a three-compartment pharmacokinetic model (Wambaugh et al. 2013) using animal species-, strain-, sex-specific parameters.
Method to Derive Human Equivalent Dose	LOAEL HED = (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 (Thompson et al. 2010)
Dose-Response	
Dose Response Modeling Method	LOAEL
POD	8.29 mg/L
PODxDAF=HED (mg/kg/day)	0.001163 mg/kg/day or 1.163 x 10-3 mg/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	3
Database (UFD)	3
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	3.9 ng/kg/day (3.9 x 10-6 mg/kg/day) which corresponds to a serum concentration of 0.028 mg/L
Receptor	Breast Fed Infant
Exposure	
Ingestion Rate (L/day)	Breast-fed infant, which is also protective of a formula-fed infant using Minnesota Department of Health Model based on Goeden et al.
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	
Relative Source Contribution	50% Based on NHANES 95th percentiles for 3-11 (2013-2014) and over 12 years old (2015-2016) participants (CDC 2019)
Threshold Level (ug/L) or (PPT)	0.008 ug/L or 8 PPT
Additional Information	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.
Reference	https://www.michigan.gov/documents/pfasresponse/Health-Based_Drinking_Water_Value_Recommendations_for_PFAS_in_Michigan_Report_659258_7.pdf

PFOA	
Minnesota	
DOH 2017	
Standard / Guidance	Health Risk Limit
Media Type	DW & GW
Threshold Level (ug/L) or (PPT)	0.035 ug/L or 35 PPT
Key Study Information	
Critical Effect Key Study Reference	Koskela A, Finnilä MA, Korkalainen M, Spulber S, Koponen J, Håkansson 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.
Species	CD-1 Mice
Study Exposure Duration (days)	18 days maternal, 17 days pups
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	38 mg/L serum concentration (US EPA 2016a predicted average serum concentration for maternal animals from Lau et al 2006) EPA modeled average serum concentration (predicted AUC u/mL/hr divided by (24hr/day x 18 days)
Method to Derive Human Equivalent Dose	DAF Dose adjustment factor of 0.00014 L/kg-day, based on first order kinetic clearance rate (ln 2/t½ of 840 days) x 0.17 L/kg (Vd) (SAME AS EPA)
Dose-Response	
Dose Response Modeling Method	LOAEL
POD	38 mg/L
POD x DAF = HED mg/kg/day	38 mg/L x 0.00014 L/kg/day = 0.0053 mg/kg/day = 5.3 x 10-3 mg/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	3
Database (UFD)	3
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	0.000018 (18 x 10^{-6} mg/kg/d) or 18 ng/kd/d
Receptor	Infant exposure via breastmilk for 1 year, from mother chronically exposed via water, followed by lifetime of exposure via drinking water. Protective for short-term, subchronic and chronic.
Exposure	
Ingestion Rate (L/day)	The 95th percentile water intake rates (Table 3-1 and 3-3, USEPA 2011) or upper percentile breastmilk intake rates (Table 15-1, USEPA 2011) were used.
Body Weight (Kg) Normalized Drinking Water Intake (L/kg/day)	Goeden 2019 Minnesota Model. MDH derived the nHBV based on an internal serum concentration that would not exceed 0.5 (RSC) of the serum concentration corresponding to the RfD (0.13 mg/L) from infancy through lifetime of exposure. RSC was based on ceiling of 80% minus 'background' exposure, based on the most recent NHANES dataset. The 95th percentile water intake rates (Table 3-1 and 3-3, USEPA 2011) or upper percentile breastmilk intake rates (Table 15-1, USEPA 2011) were used. Breastmilk concentrations were calculated by multiplying the maternal serum concentration by a PFOA breastmilk transfer factor of 5.2%. Breastmilk transfer value was based on average breastmilk to maternal serum concentration ratios reported in the literature. The simulated individuals began life with a pre-existing body burden through placental transfer (maternal serum concentration x 87%. Placental transfer value was based on average cord to maternal serum concentration ratios reported in the literature.
Relative Source Contribution	50%
Threshold Level (ug/L) or (PPT)	0.035 ug/L or 35 PPT
Additional Information	MDH Health Based Guidance for Water Health Risk Assessment Unit, Environmental Health Division, 651-201-4899.Toxicological Summary for: Perfluorooctanoic Acid. May 2017 https://www.nature.com/articles/s41370-018-0110-5https://www.health.state.mn.us/communities/environment/risk/guidance/waterguidance.html http://www.legislature.mi.gov/documents/2017-2018/resolutionintroduced/House/htm/2018-HIR-0228.htm https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfoa.pdf

PFOA	
New Hampshire	
NH Department of Environmental Services 2019	
Standard / Guidance	Proposed MCL
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.012 ug/L or 12 PPT
Key Study Information	
Critical Effect Key Study Reference	Increased liver wt. Loveless, S.E., Finlay, C., Everds, N.E., Frame, S.R., Gillies, P.J., O'Connor, J.C., Powley, C.R., Kennedy, G.L. (2006). Comparative responses of rats and mice exposed to linear/branched, linear, or branched ammonium perfluorooctanoate (APFO). Toxicology 220: 203–217. (rejected Macon 2011 Mammary Gland Development because target human serum level was above current serum levels in population)
Species	Mice
Study Exposure Duration (days)	14 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	
Method to Derive Human Equivalent Dose	DAF = 170 mL/kg x (Ln(2)/840 days) = 1.4 x 10-4 L/kg/d
Dose-Response	
Dose Response Modeling Method	lower confidence limit on the BMD (BMDL) for the serum PFOA level resulting in a 10 percent increase in liver weight in mice
POD HED Units	4.35 mg/L
POD x DAF = HED	609 ng/kg/day = 4.35 mg/L * 1.4 x 10-4 L/kg/day (EPA Clearance Factor) = 0.609 mg/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	3
Total Composite (UFT)	100
HED/UFT= Reference Dose (mg/kg-day)	6.1 x 10^{-6} mg/kg/d (RfD)
Receptor	Adult
Exposure	
Ingestion Rate (L/day)	Breast-fed infant, which is also protective of a formula-fed infant using Minnesota Department of Health Model based on Goeden et al.
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	
Relative Source Contribution	50%
Threshold Level (ug/L) or (PPT)	0.012 ug/L or 12 PPT
Additional Information	UFs applied to animal serum level BMDL to obtain Target Human Serum Level of 14.5 ng/mL which is then converted to RfD using 1.4 x 10-4 L/kg/day (EPA Clearance Factor). RSC stated to account for higher exposure of young infants, at least partially.
Reference	https://www4.des.state.nh.us/nh-pfas-investigation/wp-content/uploads/Summary-of-Comments-Responses-with-Attachments.pdf

PFOA	
New Jersey	
Drinking Water Quality Institute 2019	
Standard / Guidance	MCL
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.014 ug/L or 14 PPT proposed
Key Study Information	
Critical Effect Key Study Reference	Increased liver wt. Loveless, S.E., Finlay, C., Everds, N.E., Frame, S.R., Gillies, P.J., O'Connor, J.C., Powley, C.R., Kennedy, G.L. (2006). Comparative responses of rats and mice exposed to linear/branched, linear, or branched ammonium perfluorooctanoate (APFO). Toxicology 220: 203–217. (rejected Macon 2011 Mammary Gland Development because target human serum level was above current serum levels in population)
Species	Mice
Study Exposure Duration (days)	14 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Ke = 0.000489165 (4.8 x 10-4) based on a human serum half-life of 1417 days (calculated from Zhang et al. [2013] as described above)
Method to Derive Human Equivalent Dose	clearance factor (1.4 x 10-4 L/kg/day; USEPA, 2016a)
Dose-Response	
Dose Response Modeling Method	lower confidence limit on the BMD (BMDL) for the serum PFOA level resulting in a 10 percent increase in liver weight in mice
POD HED Units	4.35 mg/L
POD x DAF = HED	4.35 mg/L * 1.4 x 10-4 L/kg/day (EPA Clearance Factor) = 609 ng/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	10
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	2×10^{-6} mg/kg/d (RfD)
Receptor	Adult
Exposure	
Ingestion Rate (L/day)	2
Body Weight (Kg)	70
Normalized Drinking Water Intake (L/kg/day)	.029
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	0.014 ug/L or 14 PPT proposed
Additional Information	UFs applied to animal serum level BMDL to obtain Target Human Serum Level of 14.5 ng/mL which is then converted to RfD using 1.4 x 10-4 L/kg/day (EPA Clearance Factor). RSC stated to account for higher exposure of young infants, at least partially.
Reference	Maximum Contaminant Level Recommendation for Perfluorooctanoic Acid in Drinking Water, Basis and Background. New Jersey Drinking Water Quality Institute. https://www.nj.gov/dep/watersupply/pdf/pfoa-recommend.pdf https://www.nj.gov/dep/watersupply/pdf/pfoa-appendixa.pdf

PFOA	
New York	
Drinking Water Quality Council 2018	
Standard / Guidance	Recommended MCL
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.010 ug/L or 10 PPT proposed
Key Study Information	
Critical Effect Key Study Reference	Mammary gland development Macon MB, Villanueva LR, Tatum-Gibbs K, et al. 2011. Prenatal perfluorooctanoic acid exposure in CD-1 mice: Low-dose developmental effects and internal dosimetry. Toxicol Sci 122(1):134-145.
Species	Mice
Study Exposure Duration (days)	17 day gestational exposure
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Not published
Method to Derive Human Equivalent Dose	Not published
Dose-Response	
Dose Response Modeling Method	Not published
POD HED Units	Not published
POD x DAF = HED	Not published
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	3
Total Composite (UFT)	100
HED/UFT= Reference Dose (mg/kg-day)	1.5×10^{-6} mg/kg/d
Receptor	None given
Exposure	
Ingestion Rate (L/day)	None given
Body Weight (Kg)	None given
Normalized Drinking Water Intake (L/kg/day)	None given
Relative Source Contribution	None given
Threshold Level (ug/L) or (PPT)	0.010 ug/L or 10 PPT proposed
Additional Information	Initial rule making now in the deferral provision phase - Determined by vote at Drinking Water Quality Council (considered 6, 10, and 14 PPT)
Reference	https://www.health.ny.gov/press/releases/2018/2018-12-18_drinking_water_quality_council_recommendations.htm https://totalwebcasting.com/view/?func=VOFF&id=nysdoh&date=2020-02-04&seq=1 https://www.health.ny.gov/environmental/water/drinking/dwqc/

PFOA	
North Carolina	
North Carolina Department of Environment Quality 2019	
Standard / Guidance	Health Advisory
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	0.07 ug/L 70 PPT (PFOA + PFOS cannot exceed this level)
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD	Based on EPA Health Advisories.
POD x DAF = HED	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Lactating women
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Health Advisory
Relative Source Contribution	Drinking Water
Threshold Level (ug/L) or (PPT)	0.07 ug/L 70 PPT (PFOA + PFOS cannot exceed this level)
Additional Information	
Reference	https://files.nc.gov/ncdeq/GenX/SAB/PFOS-and-PFOA-proposed-standard.pdf

PFOA	
Oregon	
Standard / Guidance	Health Advisory
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Additional Information	
Reference	https://www.oregon.gov/oha/PH/HEALTHYENVIRONMENTS/DRINKINGWATER/OPERATIONS/Pages/EmergingContaminants.aspx

PFOA	
Texas	
Office of Water 2016	
Standard / Guidance	Health Advisory
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD	Based on EPA Health Advisories.
POD x DAF = HED	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
Toxicity Value RfD (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Lactating women
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.07 ug/L 70 PPT (PFOA + PFOS cannot exceed this level)
Additional Information	Texas has developed a number of reference dose recommendations for a wide range of PFAS for groundwater but defers to EPA for Drinking Water
Reference	Perfluorcoumpunds (PFCs) January 2016 https://www.tceq.texas.gov/assets/public/implementation/tox/evaluations/pfcs.pdf

PFOA	
Vermont	
Department of Environmental Conservation / Department of Environmental Quality 2018	
Standard / Guidance	Maximum Allowable Concentration
Media Type	Ground Water and Drinking Water
Threshold Level (ug/L) or (PPT)	0.020 ug/mL or 20 PPT applied individually to PFOA, PFOS, PFHxS, PFHpA and PFNA and their sum
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD	Based on EPA Health Advisories.
POD x DAF = HED	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
Toxicity Value RfD (mg/kg-day)	0.000021 (2.1 x 10-5)
Receptor	Infant less than a year
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	0.175
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	0.020 ug/mL or 20 PPT applied individually to PFOA, PFOS, PFHxS, PFHpA and PFNA and their sum
Additional Information	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.
Reference	Drinking Water Health Advisory for Five PFAS (per- and polyfluorinated alkyl substances) July 2018 https://www.healthvermont.gov/sites/default/files/documents/pdf/ENV_DW_PFAS_HealthAdvisory.pdf

PFOA	
West Virginia	
Department of Health and Human Resources 2018	
Standard / Guidance	Health Advisory
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	0.07 ug/L 70 PPT (PFOA + PFOA cannot exceed this level)
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Lactating women
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.07 ug/L 70 PPT (PFOA + PFOA cannot exceed this level)
Additional Information	
Reference	Perfluorinated Compounds Drinking Water Health Advisory https://www.wvdhhr.org/oehs/documents/BPH_pfoa%20pfos_FL.pdf

PFOS

PFOS	
US EPA	
Office of Water 2016	
Standard / Guidance	Health Advisory
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	0.07 ug/L 70 PPT (PFOA + PFOS cannot exceed this level)
Key Study Information	
Critical Effect Key Study Reference	decreased maternal body weight, gestation length and pup survival Luebker DJ, Case MT, York RG, et al. 2005. Two-generation reproduction and cross-foster studies of perfluorooctanesulfonate (PFOS) in rats. Toxicology 215(1-2):126-148.
Species	femaleSprague Dawley rats
Study Exposure Duration (days)	84 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	The average serum concentration was estimated in the mice (6.26 mg/L) using a three-compartment pharmacokinetic model (Wambaugh et al. 2013) using animal species-, strain-, sex-specific parameters.
Method to Derive Human Equivalent Dose	Dose adjustment factor of 0.000081 (8.1 x 10-5) L/kg-day, based on first order kinetic clearance rate (Vd x (ln 2 ÷ t½))
Dose-Response	
Dose Response Modeling Method	NOAEL
POD	6.26 mg/L
POD x DAF = Human Equiv Dose	0.0051 mg/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	1
Total Composite (UFT)	30
HED/UFT= Reference Dose (mg/kg-day)	(2 x 10-5 mg/kg-day) or 20 ng/kg/d
Receptor	Lactating women
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg-day)	0.054
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	0.07 ug/L 70 PPT (PFOA + PFOS cannot exceed this level)
Additional Information	Because the critical effect identified for PFOS is a developmental endpoint and can potentially result from a short-term exposure during a critical period of development, EPA concludes that the lifetime HA for PFOA is applicable to both short-term and chronic risk assessment scenarios. Thus, the lifetime HA of 0.07 µg/L also applies to short-term exposure scenarios (i.e., weeks to months) to PFOA in drinking water, including during pregnancy and lactation.
Reference	https://www.epa.gov/sites/production/files/2016-05/documents/pfos_health_advisory_final_508.pdf

PFOS	
US DHHS	
ATSDR DRAFT June 2018	
Standard / Guidance	Minimal Risk Level
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	None at present in draft phase
Key Study Information	
Critical Effect Key Study Reference	Delayed eye opening and decreased pup body weight Luebker DJ, Case MT, York RG, et al. 2005a. Two-generation reproduction and cross-foster studies of perfluorooctanesulfonate (PFOS) in rats. Toxicol 215: 126-148
Species	Sprague-Dawley rats (P generation)
Study Exposure Duration (days)	18 days maternal, 17 days pups
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	The average serum concentration for NOAEL (0.1 mg/kg/day) was estimated using an empirical clearance model (Wambaugh et al., 2013). The estimated time-weighted average serum concentration corresponding to the NOAEL was 7.43 mg/L.
Method to Derive Human Equivalent Dose	NOAEL HED = 5.5 x 10-5 mg/kg-day = (TWA serum x ke x Vd) TWA serum = 0.674 mg/L (Human Clearance Factor US EPA, 2016b) = 8.1×10^{-5} L/kg-day
Dose-Response	
Dose Response Modeling Method	NOAEL
POD	7.43 mg/L
POD x DAF = Human Equiv Dose	0.000515 mg/kg/day or 5.15×10^{-4} mg/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	10
Modifying Factor (MF)	10
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	$1.7\ 0 \times 10^{-6}$ mg/kg/day rounded to 2.0×10^{-6} mg/kg/day and called a Minimal Risk Level
Receptor	None selected at present
Exposure	
Ingestion Rate (L/day)	Not determined at present
Body Weight (Kg)	Assuming the ATSDR uses the EPA methodology the Threshold Level would be 9 PPT
Normalized Drinking Water Intake (L/kg-day)	
Relative Source Contribution	
Threshold Level (ug/L) or (PPT)	*9 PPT presumptive*
Additional Information	Draft Commentary awaiting further review modifying factor of 10 for concern that immunotoxicity may be a more sensitive endpoint than developmental toxicity Dong et al 2011 was considered and with MF would have resulted in same MRL
Reference	https://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=1117&tid=237

PFOS	
ALASKA	
Dept. of Environmental Conservation 2019	
Standard / Guidance	Action level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Additional Information	
Reference	https://dec.alaska.gov/spar/csp/pfas/

PFOS	
ALABAMA	
ADEM 2019	
Standard / Guidance	Action level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Additional Information	
Reference	http://adem.alabama.gov/newsEvents/reports/PFASinAlabama.pdf

PFOS	
California	
August 2019	
Standard / Guidance	Notification Levels NonCancer
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.007 ug/L or 7 PPT
Key Study Information	
Critical Effect Key Study Reference	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. Decreased plaque forming cell response was the most sensitive endpoint, and a NOAEL of 0.008 mg/kg-day was identified.
Species	adult male mice
Study Exposure Duration (days)	60 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	NOAEL 0.674 mg/L
Method to Derive Human Equivalent Dose	HED = 5.5 x 10-5 mg/kg-day = (TWA serum x ke x Vd) TWA serum = 0.674 mg/L (Human Clearance Factor US EPA, 2016b) = 8.1×10^{-5} L/kg-day
Dose-Response	
Dose Response Modeling Method	NOAEL (no fit found for BMDL)
POD	0.674 mg/L
PODxDAF=HED (mg/kg/day)	HED = 5.5 x 10-5 mg/kg-day mg/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	1
Total Composite (UFT)	30
HED/UFT= Reference Dose (mg/kg-day)	1.8x10-6 mg/kg/day
Receptor	adult
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	0.053 L/kg-day
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	0.007 ug/L or 7 ppt
Additional Information	Note: California uses an intermediate step called ADD or acceptable daily dose which is expressed as a target serum level and then a dose. This corresponds to the Reference Dose in this table
Reference	Notification Level Recommendations for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS) https://oehha.ca.gov/media/downloads/water/chemicals/nl/final-pfoa-pfosnl082119.pdf

PFOS	
California	
August 2019	
Standard / Guidance	Cancer Reference Level
Media Type	one in one million cancer risk from PFOS in tap water
Threshold Level (ug/L) or (PPT)	0.0001 ug/L or 0.1 ppt
Key Study Information	
Critical Effect Key Study Reference	NTP (2018c). TR-598: Technical Report Pathology Tables and Curves - PFOA. National Toxicology Program, Research Triangle Park, North Carolina. https://tools.niehs.nih.gov/cebs3/views/?action=main.dataReview&bin_id=13658 (last accessed March 20, 2019).
Species	Rats
Study Exposure Duration (days)	
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Using the HEDs as the dose metric, multisite benchmark dose modeling was performed to determine the cancer slope factor (CSF) for the hepatic and pancreatic tumors in male rats.
Method to Derive Human Equivalent Dose	
Dose-Response	
Dose Response Modeling Method	BMDL05(human) = BMDL05(animal) × (BWanimal/BWhuman)1/8
POD	BMDL05 of 0.0020 mg/kg-day for male rats
PODxDAF=HED (mg/kg/day)	BMDL05(human) 0.0011 mg/kg- day CSF = BMR ÷ BMDL05 = 0.05 ÷ 3.5 × 10-4 mg/kg-day = 45.5 (mg/kg-day)-1 for males
Uncertainty Extrapolation	
Human Variability (UFH)	
Animal to Human (UFA)	
Subchronic to Chronic (UFS)	
LOAEL to NOAEL (UFL)	
Database (UFD)	
Total Composite (UFT)	
HED/UFT= Reference Dose (mg/kg-day)	RL = R ÷ (CSF X DWI) R = default risk level of one in one million, or 10-6 RL = 10-6 ÷ (45.5 (mg/kg-day)-1 . 0.053 L/kg-day) = 4.2 . 10-7 mg/L RL = 0.4 ng/L or 0.4 ppt (rounded)
Receptor	All ages: Age sensitivity factors (ASFs) were not applied
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	0.053 L/kg-day
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	4.2 . 10-7 mg/L or 0.4 ng/L or 0.4 ppt (rounded)
Additional Information	OEHHA recommends that SWRCB set the final NLs at the lowest levels at which PFOA and PFOS can be reliably detected in drinking water using currently available and appropriate technologies.
Reference	Notification Level Recommendations for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS) https://oehha.ca.gov/media/downloads/water/chemicals/nl/final-pfoa-pfosnl082119.pdf

PFOS	
Colorado	
CPHE 2018	
Standard / Guidance	Action level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Additional Information	
Reference	https://www.colorado.gov/pacific/cdphe/PFCs/health/advisory

PFOS	
Connecticut	
CT DPH 2016	
Standard / Guidance	Action level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA
Additional Information	
Reference	https://portal.ct.gov/DPH/Drinking-Water/DWS/Per--and-Polyfluoroalkyl-Substances

PFOS	
Delaware	
DNREC-DWHS 2018	
Standard / Guidance	Health Advisoru Level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Additional Information	
Reference	http://www.dnrec.delaware.gov/dwhs/SIRB/Documents/DWHS%20PFAS%20Sampling%20Policy.pdf

PFOS	
Florida	
DOH 2016	
Standard / Guidance	Health Advisory Level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Additional Information	
Reference	http://www.floridahealth.gov/environmental-health/drinking-water/_documents/pfoa-pfos-fs-20161.pdf

PFOS	
Idaho	
DEQ 2017	
Standard / Guidance	Health Advisory Level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Additional Information	
Reference	https://www.deq.idaho.gov/water-quality/drinking-water/drinking-water-health-advisories/

PFOS	
Iowa	
DNR 2019	
Standard / Guidance	Health Advisory Level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT PFOS
Additional Information	
Reference	https://programs.iowadnr.gov/riskcalc/Chemical/Index/287

PFOS	
Maine	
PFAS Task Force 2020	
Standard / Guidance	Health Advisory
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT for PFOS + PFOA, 0.4 ug/L or 400 PPT for all PFAS combined
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	
Relative Source Contribution	
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT for PFOS + PFOA, 0.4 ug/L or 400 PPT for all PFAS combined
Additional Information	
Reference	https://www1.maine.gov/pfastaskforce/materials/report/PFAS-Task-Force-Report-FINAL-Jan2020.pdf

PFOS	
Massachusetts	
DEP 2019	
Standard / Guidance	MCL
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.020 ug/L or 20 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA + PFDA
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	10
Database (UFD)	1
Total Composite (UFT)	300 x 3 = 900
HED/UFT= Reference Dose (mg/kg-day)	5×10^{-6} (mg/kg-day)
Receptor	pregnant women, nursing mothers and infants
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.020 ug/L or 20 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA + PFDA
Additional Information	
Reference	https://www.mass.gov/doc/310-cmr-2200-pfas-amendments/download

PFOS	
Michigan	
Michigan Science Advisory Group 2019	
Standard / Guidance	Health Based Values
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	0.016 ug/L or 16 PPT
Key Study Information	
Critical Effect Key Study Reference	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. Decreased plaque forming cell response was the most sensitive endpoint, and a NOAEL of 0.008 mg/kg-day was identified.
Species	adult make mice
Study Exposure Duration (days)	60 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	NOAEL 0.674 mg/L
Method to Derive Human Equivalent Dose	HED = 5.5 x 10-5 mg/kg-day = (TWA serum x ke x Vd) TWA serum = 0.674 mg/L (Human Clearance Factor US EPA, 2016b) = 8.1×10^{-5} L/kg-day
Dose-Response	
Dose Response Modeling Method	NOAEL (no fit found for BMDL)
POD HED Units	0.674 mg/L
POD x DAF = HED	HED = 5.5 x 10-5 mg/kg-day mg/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	1
Total Composite (UFT)	30
HED/UFT= Reference Dose (mg/kg-day)	2.89 x 10-6 mg/kg/day) which corresponds to a serum concentration of 0.022 µg/ml
Receptor	Breast fed infant
Exposure	
Ingestion Rate (L/day)	Breast-fed infant, which is also protective of a formula-fed infant using Minnesota Department of Health Model based on Goeden et al.
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	
Relative Source Contribution	50% Based on NHANES 95th percentiles for 3-11 (2013-2014) and over 12 years old (2015-2016) participants (CDC 2019)
Threshold Level	0.016 ug/L or 16 PPT
Additional Information	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.
Reference	https://www.michigan.gov/documents/pfasresponse/Health-Based_Drinking_Water_Value_Recommendations_for_PFAS_in_Michigan_Report_659258_7.pdf

PFOS	
Minnesota	
DOH 2019	
Standard / Guidance	Health Based Values
Media Type	
Threshold Level (ug/L) or (PPT)	0.015 ug/L or 15 PPT
Key Study Information	
Critical Effect Key Study Reference	increased IL-4 and decreased SRBC specific IgM levels Dong, G., MM Liu, D Wang, L Zheng, ZF Liang, YH Jin, (2011). "Sub-chronic effect of perfluorooctanesulfonate (PFOS) on the balance of type 1 and type 2 cytokine in adult C57BL6 mice." Archives of Toxicology 85: 1235-1244.
Species	adult C57BL/6 male Mice
Study Exposure Duration (days)	18 days maternal, 17 days pups
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	38 mg/L serum concentration (US EPA 2016a predicted average serum concentration for maternal animals from Lau et al 2006) EPA modeled average serum concentration (predicted AUC u/mL/hr divided by (24hr/day x 18 days)
Method to Derive Human Equivalent Dose	DAF = 0.23 L/kg x (Ln(2) ÷ (3.4 y * 365 d/y)) = 1.28x10-1 mL/kg/d
Dose-Response	
Dose Response Modeling Method	NOAEL
POD	2.36 µg/mL(or mg/L)
POD x DAF = HED mg/kg/day	2.36 mg/L x 0.00013 L/kg-d = 0.000307 mg/kg-d
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	3 (impacts on serum thyrozine in developing animals at 1/3 of POD)
Total Composite (UFT)	100
HED/UFT= Reference Dose (mg/kg-day)	0.0000031 mg/kg-d corresponding to a serum concentration of *0.024 mg/L.*
Receptor	Infant exposure via breastmilk for 1 year, from mother chronically exposed via water, followed by lifetime of exposure via drinking water. Protective for short-term, subchronic and chronic.
Exposure	
Ingestion Rate (L/day)	The 95th percentile water intake rates (Table 3-1 and 3-3, USEPA 2011) or upper percentile breastmilk intake rates (Table 15-1, USEPA 2011) were used.
Body Weight (Kg) Normalized Drinking Water Intake (L/kg/day)	Breast-fed infant, which is also protective of a formula-fed infant using Minnesota Department of Health Model based on Goeden et al. Placental transfer of 40% (MDH 2019). Breastmilk transfer of 1.7% (MDH 2019). Human Serum half-life of 1241 days (Li et al. 2018) Volume of distribution of 0.23 L/kg (USA EPA 2016c) 95th percentile drinking water intake, consumers only, from birth to more than 21 years old (Goeden et al. [2019]) Upper percentile (mean plus two standard deviations) breast milk intake rate (Goeden et al. [2019]) Time-weighted average water ingestion rate from birth to 30-35 years of age (to calculate maternal serum concentration at delivery) (Goeden et al. [2019])
Relative Source Contribution	50%
Threshold Level (ug/L) or (PPT)	0.015 ug/L or 15 PPT
Additional Information	https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfos.pdf

PFOS	
New Hampshire	
NH Department of Environmental Services 2019	
Standard / Guidance	Proposed MCL
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.015 ug/L or 15 PPT
Key Study Information	
Critical Effect Key Study Reference	decreased SRBC specific IgM levels Dong, G., MM Liu, D Wang, L Zheng, ZF Liang, YH Jin, (2011). "Sub-chronic effect of perfluorooctanesulfonate (PFOS) on the balance of type 1 and type 2 cytokine in adult C57BL6 mice." Archives of Toxicology 85: 1235-1244.
Species	adult C57BL/6 male Mice
Study Exposure Duration (days)	18 days maternal, 17 days pups
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	
Method to Derive Human Equivalent Dose	DAF = 0.23 L/kg x (Ln(2) ÷ (3.4 y * 365 d/y)) = 1.28 x 10-4 L/kg/d
Dose-Response	
Dose Response Modeling Method	NOAEL
POD HED Units	2.360 ug/mL
POD x DAF = HED	3 x 10-4 mg/kg/d = 2.36 ug/mL x 1.28 x 10-4 L/kg/d = 3 x 10-4 mg/kg/d
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	3
Total Composite (UFT)	100
HED/UFT= Reference Dose (mg/kg-day)	3.0 x 10^{-6} mg/kg/d (RfD)
Receptor	Breast feeding infant
Exposure	
Ingestion Rate (L/day)	Breast-fed infant, which is also protective of a formula-fed infant using Minnesota Department of Health Model based on Goeden et al.
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	
Relative Source Contribution	50%
Threshold Level (ug/L) or (PPT)	0.015 ug/L or 15 PPT
Additional Information	UFs applied to animal serum level BMDL to obtain Target Human Serum Level of 14.5 ng/mL which is then converted to RfD using 1.4 x 10-4 L/kg/day (EPA Clearance Factor). RSC stated to account for higher exposure of young infants, at least partially.
Reference	https://www4.des.state.nh.us/nh-pfas-investigation/wp-content/uploads/Summary-of-Comments-Responses-with-Attachments.pdf

PFOS	
New Jersey	
Drinking Water Quality Institute 2019	
Standard / Guidance	MCL
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.013 ug/L or 13 PPT
Key Study Information	
Critical Effect Key Study Reference	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. Decreased plaque forming cell response was the most sensitive endpoint, and a NOAEL of 0.008 mg/kg-day was identified.
Species	adult make mice
Study Exposure Duration (days)	60 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	NOAEL 0.674 mg/L
Method to Derive Human Equivalent Dose	HED = 5.5 x 10-5 mg/kg-day = (TWA serum x ke x Vd) TWA serum = 0.674 mg/L ||| (Human Clearance Factor US EPA, 2016b) = 8.1×10^{-5} L/kg-day
Dose-Response	
Dose Response Modeling Method	NOAEL (no fit found for BMDL)
POD HED Units	0.674 mg/L
POD x DAF = HED	HED = 5.5 x 10-5 mg/kg-day mg/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	1
Total Composite (UFT)	30
HED/UFT= Reference Dose (mg/kg-day)	1.8x10-6 mg/kg/day
Receptor	Adult
Exposure	
Ingestion Rate (L/day)	2
Body Weight (Kg)	70
Normalized Drinking Water Intake (L/kg/day)	.029
Relative Source Contribution	.2
Additional Information	0.013 ug/L or 13 PPT
Reference	Maximum Contaminant Level Recommendation for Perfluorooctanoic Acid in Drinking Water, Basis and Background. New Jersey Drinking Water Quality Institute. https://www.nj.gov/dep/rules/proposals/20190401a.pdf

PFOS	
New York	
Drinking Water Quality Council 2018	
Standard / Guidance	Recommended MCL
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.010 ug/L or 10 PPT proposed
Key Study Information	
Critical Effect Key Study Reference	
Species	
Study Exposure Duration (days)	
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	
Method to Derive Human Equivalent Dose	
Dose-Response	
Dose Response Modeling Method	
POD HED Units	
POD x DAF = HED	
Uncertainty Extrapolation	
Human Variability (UFH)	
Animal to Human (UFA)	
Subchronic to Chronic (UFS)	
LOAEL to NOAEL (UFL)	
Database (UFD)	
Total Composite (UFT)	
HED/UFT= Reference Dose (mg/kg-day)	
Receptor	
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	
Relative Source Contribution	
Threshold Level (ug/L) or (PPT)	0.010 ug/L or 10 PPT proposed
Additional Information	Determined by vote at Drinking Water Quality Council (considered 6, 10, and 14)
Reference	https://www.health.ny.gov/press/releases/2018/2018-12-18_drinking_water_quality_council_recommendations.htm

PFOS	
North Carolina	
North Carolina Department of Environment Quality 2019	
Standard / Guidance	Health Advisory
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	0.07 ug/L 70 PPT (PFOA + PFOS cannot exceed this level)
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD	Based on EPA Health Advisories.
POD x DAF = HED	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Lactating women
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Health Advisory
Relative Source Contribution	Drinking Water
Threshold Level (ug/L) or (PPT)	0.07 ug/L 70 PPT (PFOA + PFOS cannot exceed this level)
Additional Information	
Reference	https://files.nc.gov/ncdeq/GenX/SAB/PFOS-and-PFOA-proposed-standard.pdf

PFOS	
Texas	
Office of Water 2016	
Standard / Guidance	Health Advisory
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD	Based on EPA Health Advisories.
POD x DAF = HED	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
Toxicity Value RfD (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Lactating women
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.07 ug/L 70 PPT (PFOA + PFOS cannot exceed this level)
Additional Information	Texas has developed a number of reference dose recommendations for a wide range of PFAS for groundwater but defers to EPA for Drinking Water
Reference	Perfluorcoumpunds (PFCs) January 2016 https://www.tceq.texas.gov/assets/public/implementation/tox/evaluations/pfcs.pdf

PFOS	
Vermont	
Department of Environmental Conservation / Department of Environmental Quality 2018	
Standard / Guidance	Maximum Allowable Concentration
Media Type	Ground Water and Drinking Water
Threshold Level (ug/L) or (PPT)	0.020 ug/mL or 20 PPT applied individually to PFOA, PFOS, PFHxS, PFHpA and PFNA and their sum
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD	Based on EPA Health Advisories.
POD x DAF = HED	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
Toxicity Value RfD (mg/kg-day)	0.000021 (2.1 x 10-5)
Receptor	Infant less than a year
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	0.175
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	0.020 ug/mL or 20 PPT applied individually to PFOA, PFOS, PFHxS, PFHpA and PFNA and their sum
Additional Information	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.
Reference	Drinking Water Health Advisory for Five PFAS (per- and polyfluorinated alkyl substances) July 2018 https://www.healthvermont.gov/sites/default/files/documents/pdf/ENV_DW_PFAS_HealthAdvisory.pdf

PFOS	
Connecticut	
CT DPH 2016	
Standard / Guidance	Action level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA
Additional Information	
Reference	https://portal.ct.gov/DPH/Drinking-Water/DWS/Per--and-Polyfluoroalkyl-Substances

PFNA

PFNA	
Connecticut	
CT DPH 2016	
Standard / Guidance	Action level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA
Additional Information	
Reference	https://portal.ct.gov/DPH/Drinking-Water/DWS/Per--and-Polyfluoroalkyl-Substances

PFNA	
Maine	
DEP 2020	
Standard / Guidance	RAG
Media Type	DW
Threshold Level (ug/L) or (PPT)	sum of all PFAS exceeds 0.4 ug/L or 400 PPT
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	sum of all PFAS exceeds 0.4 ug/L or 400 PPT
Additional Information	
Reference	https://www.maine.gov/pfastaskforce/materials/report/PFAS-Task-Force-Report-FINAL-Jan2020.pdf

PFNA	
Massachusetts	
DEP 2019	
Standard / Guidance	MCL
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.020 ug/L or 20 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA + PFDA
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	10
Database (UFD)	1
Total Composite (UFT)	300 x 3 = 900
HED/UFT= Reference Dose (mg/kg-day)	5×10^{-6} (mg/kg-day)
Receptor	pregnant women, nursing mothers and infants
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.020 ug/L or 20 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA + PFDA
Additional Information	
Reference	https://www.mass.gov/doc/310-cmr-2200-pfas-amendments/download

PFNA	
Michigan	
Michigan Science Advisory Group 2019	
Standard / Guidance	Health Based Values
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	0.006 ug/L or 6 PPT
Key Study Information	
Critical Effect Key Study Reference	Developmental endpoints – Delayed eye opening, preputial separation, and vaginal opening in mouse pups Das KP, Grey BE, Rosen MB, et al. 2015. Developmental toxicity of perfluorononanoic acid in mice. Reproductive Toxicology 51:133- 144.
Species	Timed-pregnant CD-1
Study Exposure Duration (days)	17 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	The average serum concentration for NOAEL (1 mg/kg/day) was estimated (6.8 mg/L) in dams using an empirical clearance model (Wambaugh et al., 2013). The estimated time-weighted average serum concentration corresponding to the NOAEL was 6.8 mg/L.
Method to Derive Human Equivalent Dose	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 (calculated from Zhang et al. [2013] as described above) Vd = 0.2 L/kg (ATSDR [2018]; Ohmori et al. [2003]) 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.
Dose-Response	
Dose Response Modeling Method	NOAEL
POD HED Units	6.8 mg/L
POD x DAF = HED	The time-weighted average serum concentration of 6.8 mg/L was converted to the HED using the below equation. HED= (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 (calculated from Zhang et al. [2013] as described above) Vd = 0.2 L/kg (ATSDR [2018]; Ohmori et al. [2003])
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	10
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	2.2 ng/kg/day (2.2 x 10-6 mg/kg/day) which corresponds to a serum concentration of 0.023 mg/L
Receptor	Breast fed infant
Exposure	
Ingestion Rate (L/day) Body Weight (Kg) Normalized Drinking Water Intake (L/kg/day)	Breast-fed infant, which is also protective of a formula-fed infant using Minnesota Department of Health Model based on Goeden et al. Placental transfer of 87% (MDH 2017). Breastmilk transfer of 5.2% (MDH 2017). Human Serum half-life of 840 days (Bartell et al. 2010) Volume of distribution of 0.17 L/kg (Thompson et al. [2010]) 95th percentile drinking water intake, consumers only, from birth to more than 21 years old (Goeden et al. [2019]) Upper percentile (mean plus two standard deviations) breast milk intake rate (Goeden et al. [2019]) Time-weighted average water ingestion rate from birth to 30-35 years of age (to calculate maternal serum concentration at delivery) (Goeden et al. [2019])

Relative Source Contribution	50% Based on NHANES 95th percentiles for 3-11 (2013-2014) and over 12 years old (2015-2016) participants (CDC 2019)
Threshold Level	0.006 ug/L or 6 PPT
Additional Information	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.
Reference	https://www.michigan.gov/documents/pfasresponse/Health-Based_Drinking_Water_Value_Recommendations_for_PFAS_in_Michigan_Report_659258_7.pdf

PFNA	
New Hampshire	
NH Department of Environmental Services 2019	
Standard / Guidance	Proposed MCL
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.011 ug/L or 11 PPT
Key Study Information	
Critical Effect Key Study Reference	Increased liver weight in pregnant mice Das KP, Grey BE, Rosen MB, et al. 2015. Developmental toxicity of perfluorononanoic acid in mice. Reproductive Toxicology 51:133- 144.
Species	Timed-pregnant CD-1
Study Exposure Duration (days)	17 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	
Method to Derive Human Equivalent Dose	Toxicokinetic Adjustment based on Chemical-Specific Clearance Rate = Volume of Distribution (L/kg) x (Ln2/Half- life, days) = 200 mL/kg x (Ln2/1570 days) = 8.83 x 10 -2 mL/kg/d
Dose-Response	
Dose Response Modeling Method	lower confidence limit on the BMD (BMDL) for the serum PFNA level resulting in a 10 percent increase in liver weight in mice
POD HED Units	4.9 mg/L
POD x DAF = HED	4.3×10^{-6} mg/kg/d = 4.9 mg/L x 8.83 x 10 -2 mL/kg/d
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	3
Total Composite (UFT)	100
HED/UFT= Reference Dose (mg/kg-day)	4.3×10^{-6} mg/kg/d (RfD)
Receptor	Breast Fed Infant
Exposure	
Ingestion Rate (L/day)	Breast-fed infant, which is also protective of a formula-fed infant using Minnesota Department of Health Model based on Goeden et al.
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	
Relative Source Contribution	50%
Threshold Level (ug/L) or (PPT)	0.011 ug/L or 11 PPT
Additional Information	
Reference	https://www4.des.state.nh.us/nh-pfas-investigation/wp-content/uploads/Summary-of-Comments-Responses-with-Attachments.pdf

PFNA	
New Jersey	
Drinking Water Quality Institute	
Standard / Guidance	MCL
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	0.013 ug/L or 13 PPT
Key Study Information	
Critical Effect Key Study Reference	10% increase from the mean liver weight in the pregnant control mice pups Das KP, Grey BE, Rosen MB, et al. 2015. Developmental toxicity of perfluorononanoic acid in mice. Reproductive Toxicology 51:133- 144.
Species	Timed-pregnant CD-1
Study Exposure Duration (days)	17 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	"Because the half-life of long-chain PFCs such as PFNA is much longer in humans (several years) than in rats and mice, a given administered dose (mg/kg/day) results in a much greater internal dose (as indicated by serum level) in humans than in these animal species. Therefore, comparisons between effect levels in animal studies and human exposures were made on the basis of serum levels rather than administered dose"
Method to Derive Human Equivalent Dose	
Dose-Response	
Dose Response Modeling Method	BMDL
POD HED Units	4.9 mg/L
POD x DAF = HED	None derived
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	10
LOAEL to NOAEL (UFL)	1
Database (UFD)	3
Total Composite (UFT)	1000
Target Human Serum Level	4.9 x 10-3 mg/L or 4.9 x10-3 ug/mL target human serum level
Receptor	Lifetime
Exposure	
Ingestion Rate (L/day) Body Weight (Kg) Normalized Drinking Water Intake (L/kg/day)	Based on an assumed daily drinking water intake of 16 ml/kg/day (USEPA, 2011), the corresponding increase in daily dose of PFNA (ng/kg/day) that results in a 1 ng/ml increase in PFNA in blood serum is 0.08 ng/kg/day/(ng/ml). Based on an assumed daily drinking water intake of 16 ml/kg/day (USEPA, 2011), the corresponding increase in daily dose of PFNA (ng/kg/day) that results in a 1 ng/ml increase in PFNA in blood serum is 0.08 ng/kg/day/(ng/ml). Therefore, ongoing exposure to drinking water with 150 ng/L PFNA (the highest concentration reported in public drinking water in New Jersey or elsewhere) is estimated to increase PFNA serum levels, on average, by 30 ng/ml (µg/L; ppb) in serum. Based on the 200:1 ratio between PFNA serum levels and drinking water concentration, an increase in PFNA serum level of 2500 ng/L is expected to result from ongoing exposure to 12.5 ng/L
Relative Source Contribution	50 % RSC = 100 X (Target human serum level – 95th % NHANES serum level)/ Target Human Serum Level PFNA RSC = 100 x (4.9 ng/ml – 2.5 ng/ml) /4.9 ng/ml = 49.0% (rounded to 50%)
Threshold Level	0.013 ug/L or 13 PPT = 200 / 2.5 ng/mL rounded up
Additional Information	
Reference	https://www.nj.gov/dep/watersupply/pdf/pfna-health-effects.pdf

PFNA	
Vermont	
Department of Environmental Conservation / Department of Environmental Quality 2018	
Standard / Guidance	Maximum Allowable Concentration
Media Type	Ground Water and Drinking Water
Threshold Level (ug/L) or (PPT)	0.020 ug/mL or 20 PPT applied individually to PFOA, PFOS, PFHxS, PFHpA and PFNA and their sum
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD	Based on EPA Health Advisories.
POD x DAF = HED	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
Toxicity Value RfD (mg/kg-day)	0.000021 (2.1 x 10-5)
Receptor	Infant less than a year
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	0.175
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	0.020 ug/mL or 20 PPT applied individually to PFOA, PFOS, PFHxS, PFHpA and PFNA and their sum
Additional Information	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.
Reference	Drinking Water Health Advisory for Five PFAS (per- and polyfluorinated alkyl substances) July 2018 https://www.healthvermont.gov/sites/default/files/documents/pdf/ENV_DW_PFAS_HealthAdvisory.pdf

PFHxS

PFHxS	
Connecticut	
CT DPH 2016	
Standard / Guidance	Action level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA
Additional Information	
Reference	https://portal.ct.gov/DPH/Drinking-Water/DWS/Per--and-Polyfluoroalkyl-Substances

PFHxS	
Maine	
DEP 2020	
Standard / Guidance	RAG
Media Type	DW
Threshold Level (ug/L) or (PPT)	sum of all PFAS exceeds 0.4 ug/L or 400 PPT
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	sum of all PFAS exceeds 0.4 ug/L or 400 PPT
Additional Information	
Reference	https://www.maine.gov/pfastaskforce/materials/report/PFAS-Task-Force-Report-FINAL-Jan2020.pdf

PFHxS	
Massachusetts	
DEP 2019	
Standard / Guidance	MCL
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.020 ug/L or 20 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA + PFDA
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	10
Database (UFD)	1
Total Composite (UFT)	300 x 3 = 900
HED/UFT= Reference Dose (mg/kg-day)	5×10^{-6} (mg/kg-day)
Receptor	pregnant women, nursing mothers and infants
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.020 ug/L or 20 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA + PFDA
Additional Information	
Reference	https://www.mass.gov/doc/310-cmr-2200-pfas-amendments/download

PFHxS	
Michigan	
Michigan Science Advisory Group 2019	
Standard / Guidance	Health Based Values
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	0.051 ug/L or 51 PPT
Key Study Information	
Critical Effect Key Study Reference	decreased serum free thyroxin (T4) level 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
Species	Sprague Dawley Rats
Study Exposure Duration (days)	28 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	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 Benchmark Dose modeling and provided modeling run data in the HRA Toxicology Review Worksheet for PFHxS, last revised 3/8/2019.
Method to Derive Human Equivalent Dose	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 [Sundstrom et al. [2012], half-life = 1935 days [Li et al. 2018]) for a human equivalent dose of 0.00292 mg/kg/day.
Dose-Response	
Dose Response Modeling Method	POD of 32.4 mg/L serum concentration for male rats based on BMDL20.
POD HED Units	32.4 mg/L
POD x DAF = HED	0.00292 mg/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	10
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	9.7 ng/kg/day (9.7 x 10-6 mg/kg/day) which corresponds to a serum concentration of 0.11 µg/ml
Receptor	Breast fed infant
Exposure	
Ingestion Rate (L/day) Body Weight (Kg) Normalized Drinking Water Intake (L/kg/day)	Breast-fed infant, which is also protective of a formula-fed infant. Placental transfer = 0.8 Breastmilk transfer = 0.012 Half-life = 3100 days (ATSDR 2018: Olsen et al. 2007) Volume of distribution = 0.287 L/kg (ATSDR 2018) 95th percentile drinking water intake, consumers only, from birth to more than 21 years old (MDH 2017b: US EPA 2011) 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) Background Document: Toxicokinetic Model for PFOS and PFOA and Its Use in the Derivation of Human Health-based Water Guidance Values. Minnesota Department of Health.
Relative Source Contribution	50% Based on NHANES 95th percentiles for 3-11 (2013-2014) and over 12 years old (2015-2016) participants (CDC 2019)
Threshold value	0.051 ug/L or 51 PPT
Additional information	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.
Reference	https://www.michigan.gov/documents/pfasresponse/Health-Based_Drinking_Water_Value_Recommendations_for_PFAS_in_Michigan_Report_659258_7.pdf

PFHxS	
Minnesota	
DOH 2019	
Standard / Guidance	Health Based Guidance
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.047 ug/L or 47 PPT
Key Study Information	
Critical Effect Key Study Reference	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) Critical effect: decreased serum free thyroxin (T4) levels was observed in adult male rats at the lowest PFHxS dose administered (0.625 mg/kg/day) Co-critical effects: decreased free and total T4, triiodothyronine (T3), and changes in cholesterol levels and increased hepatic focal necrosis https://tools.niehs.nih.gov/cebs3/views/?action=main.dataReview&bin_id=3874
Species	Adult Sprague Dawley rates
Study Exposure Duration (days)	
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	
Method to Derive Human Equivalent Dose	Toxicokinetic Adjustment based on Chemical-Specific Clearance Rate = Volume of Distribution (L/kg) x (Ln2/Half- life, days) = 0.25 L/kg x (0.693/1935 days) = 0.000090 L/kg- day. (Half-life from Li et al 2018)
Dose-Response	
Dose Response Modeling Method	MDH modeled BMDL20%
POD	32.4 µg/mL (or mg/L) serum concentration (male rats - NTP 2018, MDH modeled BMDL20%)
POD x DAF = HED mg/kg/day	POD x DAF = 32.4 mg/L x 0.000090 L/kg/d = 0.00292 mg/kg/d
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	10
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	HED/Total UF = 0.00292/300 = 0.0000097 mg/kg-d (or 9.7 ng/kg-d)
Receptor	Infant exposure via breastmilk for 1 year, from mother chronically exposed via water, followed by lifetime of exposure via drinking water. Protective for short-term, subchronic and chronic.
Exposure	
Ingestion Rate (L/day)	The 95th percentile water intake rates (Table 3-1 and 3-3, USEPA 2011) or upper percentile breastmilk intake rates (Table 15-1, USEPA 2011) were used.
Body Weight (Kg) Normalized Drinking Water Intake (L/kg/day)	Breast-fed infant, which is also protective of a formula-fed infant using Minnesota Department of Health Model based on Goeden et al. Placental transfer of 87% (MDH 2017). Breastmilk transfer of 5.2% (MDH 2017). Human Serum half-life of 840 days (Bartell et al. 2010) Volume of distribution of 0.17 L/kg (Thompson et al. [2010]) 95th percentile drinking water intake, consumers only, from birth to more than 21 years old (Goeden et al. [2019]) Upper percentile (mean plus two standard deviations) breast milk intake rate (Goeden et al. [2019]) Time-weighted average water ingestion rate from birth to 30-35 years of age (to calculate maternal serum concentration at delivery) (Goeden et al. [2019])
Relative Source Contribution	50%
Threshold Level (ug/L) or (PPT)	0.047 ug/L or 47 PPT
Additional Information	https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfhxs.pdf

PFHxS	
New Hampshire	
NH Department of Environmental Services 2019	
Standard / Guidance	Proposed MCL
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.018 ug/L or 18 PPT
Key Study Information	
Critical Effect Key Study Reference	Reduced litter size in mice following a 14 day prior to pregnancy oral exposure Chang S, et al. 2018. Reproductive and developmental toxicity of potassium perfluorohexanesulfonate in CD-1 mice. Reproductive Toxicology 78: 150-168.
Species	Adult CD-1 female mice
Study Exposure Duration (days)	14 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Serum concentrations on day 14
Method to Derive Human Equivalent Dose	Toxicokinetic Adjustment based on Chemical-Specific Clearance Rate = Volume of Distribution (L/kg) x (Ln2/Half- life, days) = 213 mL/kg x (Ln2/1716 days) = 8.61 x 10 -2 mL/kg/d
Dose-Response	
Dose Response Modeling Method	lower confidence limit on the BMD (BMDL)
POD HED Units	13.9 mg/L
POD x DAF = HED	4.3 x 10^{-6} mg/kg/d =134.9 mg/L x 8.61 x 10 -2 mL/kg/d
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	3 (14 day exposure study)
LOAEL to NOAEL (UFL)	1
Database (UFD)	3
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	4.0 x 10^{-6} mg/kg/d (RfD)
Receptor	Breast Fed Infant
Exposure	
Ingestion Rate (L/day)	Breast-fed infant, which is also protective of a formula-fed infant using Minnesota Department of Health Model based on Goeden et al.
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	
Relative Source Contribution	50%
Threshold Level (ug/L) or (PPT)	0.018 ug/L or 18 PPT
Additional Information	
Reference	https://www4.des.state.nh.us/nh-pfas-investigation/wp-content/uploads/Summary-of-Comments-Responses-with-Attachments.pdf

PFHxS	
Vermont	
Department of Environmental Conservation / Department of Environmental Quality 2018	
Standard / Guidance	Maximum Allowable Concentration
Media Type	Ground Water and Drinking Water
Threshold Level (ug/L) or (PPT)	0.020 ug/mL or 20 PPT applied individually to PFOA, PFOS, PFHxS, PFHpA and PFNA and their sum
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD	Based on EPA Health Advisories.
POD x DAF = HED	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
Toxicity Value RfD (mg/kg-day)	0.000021 (2.1 x 10-5)
Receptor	Infant less than a year
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	0.175
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	0.020 ug/mL or 20 PPT applied individually to PFOA, PFOS, PFHxS, PFHpA and PFNA and their sum
Additional Information	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.
Reference	Drinking Water Health Advisory for Five PFAS (per- and polyfluorinated alkyl substances) July 2018 https://www.healthvermont.gov/sites/default/files/documents/pdf/ENV_DW_PFAS_HealthAdvisory.pdf

PFHpA

PFHpA	
Connecticut	
CT DPH 2016	
Standard / Guidance	Action level
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.070 ug/L or 70 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA
Additional Information	
Reference	https://portal.ct.gov/DPH/Drinking-Water/DWS/Per--and-Polyfluoroalkyl-Substances

PFHpA	
Maine	
DEP 2020	
Standard / Guidance	RAG
Media Type	DW
Threshold Level (ug/L) or (PPT)	sum of all PFAS exceeds 0.4 ug/L or 400 PPT
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
HED/UFT= Reference Dose (mg/kg-day)	Based on EPA Health Advisories.
Receptor	Child (0-6 years) residential, non-cancer
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	sum of all PFAS exceeds 0.4 ug/L or 400 PPT
Additional Information	
Reference	https://www.maine.gov/pfastaskforce/materials/report/PFAS-Task-Force-Report-FINAL-Jan2020.pdf

PFHpA	
Massachusetts	
DEP 2019	
Standard / Guidance	MCL
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.020 ug/L or 20 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA + PFDA
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	10
Database (UFD)	1
Total Composite (UFT)	300 x 3 = 900
HED/UFT= Reference Dose (mg/kg-day)	5×10^{-6} (mg/kg-day)
Receptor	pregnant women, nursing mothers and infants
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.020 ug/L or 20 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA + PFDA
Additional Information	
Reference	https://www.mass.gov/doc/310-cmr-2200-pfas-amendments/download

PFHpA	
Vermont	
Department of Environmental Conservation / Department of Environmental Quality 2018	
Standard / Guidance	Maximum Allowable Concentration
Media Type	Ground Water and Drinking Water
Threshold Level (ug/L) or (PPT)	0.020 ug/mL or 20 PPT applied individually to PFOA, PFOS, PFHxS, PFHpA and PFNA and their sum
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD	Based on EPA Health Advisories.
POD x DAF = HED	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	Based on EPA Health Advisories.
Animal to Human (UFA)	Based on EPA Health Advisories.
Subchronic to Chronic (UFS)	Based on EPA Health Advisories.
LOAEL to NOAEL (UFL)	Based on EPA Health Advisories.
Database (UFD)	Based on EPA Health Advisories.
Total Composite (UFT)	Based on EPA Health Advisories.
Toxicity Value RfD (mg/kg-day)	0.000021 (2.1 x 10-5)
Receptor	Infant less than a year
Exposure	
Ingestion Rate (L/day)	
Body Weight (Kg)	
Normalized Drinking Water Intake (L/kg/day)	0.175
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	0.020 ug/mL or 20 PPT applied individually to PFOA, PFOS, PFHxS, PFHpA and PFNA and their sum
Additional Information	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.
Reference	Drinking Water Health Advisory for Five PFAS (per- and polyfluorinated alkyl substances) July 2018 https://www.healthvermont.gov/sites/default/files/documents/pdf/ENV_DW_PFAS_HealthAdvisory.pdf

PFDA

PFDA	
Massachusetts	
DEP 2019	
Standard / Guidance	MCL
Media Type	DW
Threshold Level (ug/L) or (PPT)	0.020 ug/L or 20 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA + PFDA
Key Study Information	
Critical Effect Key Study Reference	Based on EPA Health Advisories.
Species	Based on EPA Health Advisories.
Study Exposure Duration (days)	Based on EPA Health Advisories.
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Based on EPA Health Advisories.
Method to Derive Human Equivalent Dose	Based on EPA Health Advisories.
Dose-Response	
Dose Response Modeling Method	Based on EPA Health Advisories.
POD HED Units	Based on EPA Health Advisories.
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	10
Database (UFD)	1
Total Composite (UFT)	300 x 3 = 900
HED/UFT= Reference Dose (mg/kg-day)	5×10^{-6} (mg/kg-day)
Receptor	pregnant women, nursing mothers and infants
Exposure	
Ingestion Rate (L/day)	Based on EPA Health Advisories.
Body Weight (Kg)	Based on EPA Health Advisories.
Normalized Drinking Water Intake (L/kg/day)	Based on EPA Health Advisories.
Relative Source Contribution	Based on EPA Health Advisories.
Threshold Level (ug/L) or (PPT)	0.020 ug/L or 20 PPT total PFOA + PFOS + PFNA + PFHxS + PFHpA + PFDA
Additional Information	
Reference	https://www.mass.gov/doc/310-cmr-2200-pfas-amendments/download

PFBS

PFBS	
Michigan	
Michigan Science Advisory Group 2019	
Standard / Guidance	Health Based Values
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	0.420 ug/L or 420 PPT
Key Study Information	
Critical Effect Key Study Reference	decreased serum total thyroxine (T4) in newborn (PND 1) mice as this was protective of kidney effects as well Feng, X; Cao, X; Zhao, S; Wang, X; Hua, X; Chen, L; Chen, L. (2017). Exposure of pregnant mice to perfluorobutanesulfonate causes hypothyroxinemia and developmental abnormalities in female offspring. Toxicol Sci 155: 409-419. decreased serum total thyroxine (T_4) in newborn (PND 1) mice
Species	PND1 Newborn mice
Study Exposure Duration (days)	20 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	The USEPA PODHED of 4.2 was divided by 0.149 (USEPA example DAF) to obtain a BMDL20 of 28.19 mg/kg/day.
Method to Derive Human Equivalent Dose	The BMDL20 of 28.19 mg/kg/day was divided by the Dose Adjustment Factor of 316 (human serum half-life/female mouse serum half-life = 665 hours/2.1 hours = 316)
Dose-Response	
Dose Response Modeling Method	BMDL20
POD HED Units	28.19 mg/kg/day (BMDL20) for decreased serum total T4 in newborn (PND 1) mice
POD x DAF = HED	HED = 0.0892 mg/kg/day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	10
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	300 ng/kg/day (0.0003 mg/kg/day)
Receptor	infant
Exposure	
Ingestion Rate (L/day)	95^{th} percentile of water intake for consumers only (direct and indirect consumption) for infants (birth to <1 year old) of 1.106 L/day, per Table 3-1, USEPA Exposure Factors Handbook, 2019.
Body Weight (Kg)	An infant body weight of 7.8 kilograms was used and represents a time-weighted average for birth to 1 year old (Table 8-1, USEPA 2011).
Normalized Drinking Water Intake (L/kg-day)	0.142
Relative Source Contribution	20%
Threshold value	0.420 ug/L or 420 PPT
Additional information	As insufficient human serum data was available to assess the population's exposure to PFBS from sources other than drinking water, a default Relative Source Contribution of 20% was selected consistent with USEPA (2000) guidance
Reference	https://www.michigan.gov/documents/pfasresponse/Health-Based_Drinking_Water_Value_Recommendations_for_PFAS_in_Michigan_Report_659258_7.pdf

PFHxA

PFHxA	
Michigan	
Michigan Science Advisory Group 2019	
Standard / Guidance	Health Based Values
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	400 ug/L or 400,000 PPT
Key Study Information	
Critical Effect Key Study Reference	Critical effect renal tubular degeneration and renal papillary necrosis in female rats 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. Luz, AL, Anderson, JK, Goodrum, P, Durda, J. (2019) Perfluorohexanoic acid toxicity, part I: Development of chronic human health toxicity value for use in risk assessment. Reg. Toxicol. Pharmacol. 103: 41-55.
Species	male and female Crl:CD rats
Study Exposure Duration (days)	104 weeks
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	BMDL10 = 90.4 mg/kg/day (Luz et al., 2019)
Method to Derive Human Equivalent Dose	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. (Luz et al., 2019)
Dose-Response	
Dose Response Modeling Method	BMDL10
POD HED Units	90.4 mg/kg/day (Luz et al., 2019).
POD x DAF = HED	HED 24.8 mg/kg/d
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	1
LOAEL to NOAEL (UFL)	1
Database (UFD)	10
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	83,000 ng/kg/day (8.3 mg/kg/day)
Receptor	adult
Exposure	
Ingestion Rate (L/day)	95th percentile of water intake for consumers only (direct and indirect consumption) for adults > 21 years old 3.353 L/day
Body Weight (Kg)	80 kg
Normalized Drinking Water Intake (L/kg-day)	
Relative Source Contribution	20%
Additional Information	0.420 ug/L or 420 PPT
Reference	https://www.michigan.gov/documents/pfasresponse/Health-Based_Drinking_Water_Value_Recommendations_for_PFAS_in_Michigan_Report_659258_7.pdf

GenX

GenX	
Michigan	
Michigan Science Advisory Group 2019	
Standard / Guidance	Health Based Values
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	0.370 ug/L or 370 PPT
Key Study Information	
Critical Effect Key Study Reference	Oral (Gavage) Reproduction/ Developmental Toxicity Study in Mice (OECD TG 421; modified according to the Consent Order) DuPont18405-1037 (2010) (also contains 90-day toxicity study information and outcomes - that information is not described here) (Adopted draft USEPA 2018 over North Caroline 2017)
Species	Crl:CD1(ICR) mice
Study Exposure Duration (days)	40 days
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	A candidate POD HED was derived from the BMDL10 for liver effects using a BW3/4 allometric scaling approach.
Method to Derive Human Equivalent Dose	DAF for the allometric scaling of doses from mice to humans is 0.15. Using the BMDL10 of 0.15 mg/kg/day to complete the calculation results in a PODHED for single-cell necrosis of the liver from DuPont18405-1037 (2010) of 0.023 mg/kg/day (USEPA 2018).
Dose-Response	
Dose Response Modeling Method	BMDL10
POD HED Units	
POD x DAF = HED	HED 24.8 mg/kg/d
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	3
Subchronic to Chronic (UFS)	3
LOAEL to NOAEL (UFL)	1
Database (UFD)	3
Total Composite (UFT)	300
HED/UFT= Reference Dose (mg/kg-day)	77 ng/kg/day (7.7 x10-5 mg/kg/day)
Receptor	adult
Exposure	
Ingestion Rate (L/day)	95th percentile of water intake for consumers only (direct and indirect consumption) for adults > 21 years old 3.353 L/day
Body Weight (Kg)	80 kg
Normalized Drinking Water Intake (L/kg-day)	
Relative Source Contribution	20%
Threshold value	0.370 ug/L or 370 PPT
Additional Information	Workgroup decided that the drinking water HBV below based on liver effects would be sufficiently conservative to be protective of infant exposure.
Reference	https://www.michigan.gov/documents/pfasresponse/Health-Based_Drinking_Water_Value_Recommendations_for_PFAS_in_Michigan_Report_659258_7.pdf

GenX	
North Carolina	
Standard / Guidance	Health Based Values
Media Type	Drinking Water
Threshold Level (ug/L) or (PPT)	0.140 ug/L or 140 PPT
Key Study Information	
Critical Effect Key Study Reference	liver toxicity endpoints from two sub-chronic studies provided by Chemours/DuPont during the U.S. EPA Toxic Substances Control Act
Species	mice
Study Exposure Duration (days)	mice (28-day study and a reproductive screen)
Kinetics	
Method of Administered Dose conversion to Internal Serum Level	Used UF adjustment
Method to Derive Human Equivalent Dose	Used UF adjustment
Dose-Response	
Dose Response Modeling Method	NOAEL
POD HED Units	0.1 mg/kg-day
POD x DAF = HED	0.1 mg/kg-day
Uncertainty Extrapolation	
Human Variability (UFH)	10
Animal to Human (UFA)	10
Subchronic to Chronic (UFS)	10
LOAEL to NOAEL (UFL)	1
Database (UFD)	1
Total Composite (UFT)	1000
HED/UFT= Reference Dose (mg/kg-day)	0.0001 mg/kg-day
Receptor	Bottle fed infant
Exposure	
Ingestion Rate (L/day)	1.1 liters per day = Intake rate of drinking water for a bottle-fed infant, 1.1 liters per day
Body Weight (Kg)	7.8 kg BW infant
Normalized Drinking Water Intake (L/kg-day)	
Relative Source Contribution	20%
Threshold Level (ug/L) or (PPT)	0.140 ug/L or 140 PPT
Additional Information	BMD modeling performed and determined to be statistically unreliable due to poor model fit and large confidence interval
Reference	https://files.nc.gov/ncdeq/Energy%20Mineral%20and%20Land%20Resources/DEMLR/SAB-GenX-Report-FINAL-Appendices-10-30-2018.pdfdf

Appendix A: Abbreviations and Acronyms

Regulatory Agency
CDC = Center for Disease Control & Prevention
CEQ = Commission on Environmental Quality
DEC = Dept. of Environmental Conservation
DENR = Dept. of Environment and Natural Resources
DEP = Dept. of Environmental Protection
DEQ = Dept. of Environmental Quality
DES = Dept. of Environmental Services
DOH = Dept. of Health
DNR = Dept. of Natural Resources
DPH = Division of Public Health
EPA = Environmental Protection Agency

Standard or Guidance
AGQS = ambient groundwater quality standard
BCL = basic comparison level
CL = groundwater cleanup level
ES = environmental standard
GCC = Generic Cleanup Criteria (Part 201)
HA = lifetime health advisory
HNV = human non-cancer value for surface drinking water
HRL = health risk limit
ILR = initiation level
IMAC = interim maximum allowable standard
ISGWQC = interim specific groundwater quality criterion
MAC = maximum allowable concentration
MCL = maximum contaminant level
MEG = maximum exposure guideline
PCL = protective concentration level
PGWES = primary groundwater enforcement standard
PHG = public health goal
RAG = remedial action guideline
RL = reporting level

RSL = regional screening level (calculated)
Type of Medium
DW = drinking water
GW = groundwater
SW = surface water and/or effluent

EXHIBIT B

May 8, 2017

Patrick McDonnell
Acting Secretary, Department of Environmental Protection
P.O. Box 2063
Harrisburg, PA 17105-2063

Re: Rulemaking Petition

Dear Secretary McDonnell,

Enclosed please find the Delaware Riverkeeper Network's rulemaking petition submitted pursuant to 25 Pa. Code § 23 to set a drinking water maximum contaminant level for Perfluorooctanoic Acid (PFOA) not to exceed 6 parts per trillion.

We appreciate your prompt review and consideration. Should you have any questions or comments, please do hesitate to contact me.

Sincerely,

_____

Tracy Carluccio
Deputy Director
tracy@delawareriverkeeper.org

cc:

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PETITION TO SET A DRINKING WATER STANDARD MAXIMUM CONTAMINENT LEVEL FOR PFOA NOT TO EXCEED 6 PARTS PER TRILLION

I. PETITIONER INFORMATION

Name: Maya K. van Rossum, the Delaware Riverkeeper
Tracy Carluccio, Deputy Director
Nicholas Patton, Staff Attorney
Delaware Riverkeeper Network

Mailing Address: 925 Canal St., Suite 3701,
Bristol, PA 19007

Telephone number: 215-369-1188

Date: May 8, 2017

II. PETITION INFORMATION

A. The petitioner requests the Environmental Quality Board to amend a regulation (citation 25 Pa. Code § 93.9e).

The Delaware Riverkeeper Network (DRN) requests that the Environmental Quality Board (EQB) and the Department of Environmental Protection (Department) set a maximum contaminant level for Perfluorooctanoic Acid (PFOA) not to exceed 6 parts per trillion[1] (ppt).

B. Why is the petitioner requesting this action from the Board? (Describe problems encountered under current regulations and the changes being recommended to address the problems. State factual and legal contentions and include supporting documentation that establishes a clear justification for the requested action.)

Perfluorinated compounds (PFC), like PFOA, are currently unregulated at the federal level, one of thousands of chemicals in use without safe drinking water standards and, in many cases, without any requirement for monitoring for their presence. These unregulated chemicals can enter the environment and show up in water supplies without being detected. Those with

[1] Note: ppt is equivalent to ng/L (nanogram per liter).

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toxic properties can expose people to dangerous levels that can result in disease and environmental degradation. The lack of federal regulation of PFCs means that people have been exposed to PFCs in some cases for years without knowing the risks.

There is no federal safe drinking water standard for any PFC and Pennsylvania has not established a standard. Some states have issued guidance levels or adopted maximum contaminant levels; New Jersey is in the process of adopting a safe drinking water standard.

DRN has been working on the problems posed by the presence of perfluorinated compounds in our local environment since 2005 when our staff collected tap water samples in the neighborhoods close to DuPont's Chambers Works facility in Deepwater, New Jersey on the Delaware River. We suspected that there may be a problem because of news reports about a lawsuit that had been brought in West Virginia against DuPont for releasing PFOA into the environment there. Our sampling revealed the presence of PFOA in the drinking water being used by people in the local community near DuPont's Chambers Works plant. We notified the residents and filed the information with New Jersey Department of Environmental Protection (NJDEP), setting off alarm bells and a chain of events that eventually led to NJDEP investigating the occurrence of perfluorinated compounds throughout the state and the issuance of a guidance level of 40 ppt for PFOA in 2007. Since then, after years of scientific study and research, New Jersey Drinking Water Quality Institute (NJDQWI) has recommended that New Jersey adopt a maximum contaminant level (MCL) for PFOA of 14 ppt.

EPA issued short term provisional health advisories for PFOA at 400 ppt and Perfluorooctanesulfonic acid (PFOS), another PFC, at 200 ppt in 2009. These advisory levels were based on short term exposure (5 days to 20 days, approximately) and did not mandate that water suppliers remove PFOA or PFOS from drinking water; it was just an advisory, not a federal enforceable standard. Such a short term advisory is not a valid level to use as a measurement of what is safe for drinking water on an ongoing basis. As a result of the publicity surrounding the disclosure of data from USEPA's Unregulated Contaminant Monitoring Rule 3 (UCMR3) water supply sampling, many locations across the nation took action as a result of the discovery of the presence of PFOA or other PFCs in their water supply. Most turned first to EPA for guidance. After much controversy and confusion at the federal and state levels, in 2016 EPA issued a lifetime PFOA and PFOS health advisory level (HAL) of 70 ppt when found singly or a combined total of 70 ppt when both are found. The HAL, while designed to address long term exposure, is not mandatory and does not require removal of PFOA and PFOS from drinking water. But it is being used by water suppliers and, in some cases, by those responsible for releasing the compounds into the environment, such as military bases, as an enforceable limit for drinking water.

As demonstrated below, this HAL limit has been shown to be ineffective at protecting the public health and a more protective standard not to exceed 6 ppt must be set for PFOA.

1. Legal Standard

The Department has broad authority to protect the drinking water of its citizens from emerging contaminants of concern like PFCs or PFOA. As outlined within this petition, because

PFOA in drinking water creates a substantial health risk to citizens of the Commonwealth, the Department must set a protective maximum contaminant level (MCL) for PFOA not to exceed 6 ppt.

Importantly, the Pennsylvania Constitution recognizes that the people of the Commonwealth have a constitutional right to pure drinking water. Namely, Article I Section 27 of the Pennsylvania Constitution states:

> The people have a right to clean air, pure water, and to the preservation of the natural, scenic, historic and esthetic values of the environment. Pennsylvania's public natural resources are the common property of all the people, including generations yet to come. As trustee of these resources, the Commonwealth shall conserve and maintain them for the benefit of all the people.[2]

Article I Section 27 requires the state government to ensure the preservation of the state's natural resources, including the provision to safe drinking water. This means the state government is responsible for protecting Pennsylvania's environment on behalf of its citizens.

The General Assembly has responded by passing the Pennsylvania Safe Drinking Water Act (SDWA) and declaring that "an adequate supply of safe, pure drinking water is essential to the public health, safety and welfare and that such a supply is an important natural resource in the economic development of the Commonwealth."[3] The SDWA also recognizes that the people of the Commonwealth have a constitutional right to pure drinking water.[4]

The SDWA created a state program to establish drinking water standards and to implement and enforce those standards to ensure the supply of safe drinking water to the public.[5] The Commonwealth also was required to develop a process for implementing plans to provide safe drinking water in times of emergencies and provide public notice of potentially hazardous conditions that may exist in the water supply.[6]

The Pennsylvania Department of Environmental Protection (Department) is tasked with administrating a program to accomplish the objectives of the SDWA.[7] One power and duty of the Department involves the establishment of a maximum contaminant level (MCL) related to drinking water quality standards.[8] MCLs must be no less stringent than those promulgated under the Federal act and regulations.[9] However, the MCLs and treatment technique requirements may be more stringent than those promulgated under the federal act, and the Department has the

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[2] Pa. Const. Art. I, § 27.
[3] 35 Pa. Stat. § 721.2(a)(1).
[4] 35 Pa. Stat. § 721.2(b).
[5] 35 Pa. Stat. § 721.2(b)(1).
[6] 35 Pa. Stat. § 721.2(b)(2) and (b)(3).
[7] 35 Pa. Stat. § 721.5.
[8] 35 Pa. Stat. § 721.4(a).
[9] 35 Pa. Stat. § 721.4(a).

power to adopt an MCL for an unregulated contaminant, like PFOA, "***on a case-by-case basis for a public water system in which an unregulated contaminant creates a health risk to the users of the public water system.***"[10]

Notably, the EQB has clear and expansive authority to adopt "a maximum contaminant levels or treatment technique requirements for any contaminant that a maximum contaminant level or treatment technique requirement has not been promulgated under the national primary and secondary drinking water regulations."[11] The MCL development process is intended to create "standards limiting the concentration of contaminants in public drinking water to protect the consumer from possible short-term and long-term adverse health effects. Contaminants are usually selected for regulation based on potential health risks and their occurrence or potential occurrence in drinking water."[12]

Furthermore, if the Department has reason to believe a contaminant is present in the public water system and creates a health risk to the users of the public water system, the Department "may require a public water supplier to conduct special monitoring for an unregulated contaminant."[13] The Department, and its agents and employees, "may also conduct inspections of public water systems and related activities, whenever a person presents information to the Department which gives the Department reason to believe that a condition exists which may threaten the public health, safety or welfare or the environment."[14]

PFOA has been found in many of the water supply systems in Pennsylvania at alarming levels. Because the consumption of PFOA results in significant adverse health consequences, Pennsylvania must protect its citizens and set an MCL for PFOA not to exceed 6 ppt. A failure to do so would be an abrogation of its duties under the SDWA and the Pennsylvania Constitution.

2. PFOA Background and Health Effects

PFOA is part of a larger group of chemicals referred to as perfluorinated compounds. PFOA is not found naturally in the environment, yet it is ubiquitously present. This is because PFCs were widely used in the production of goods from the 1950's until recently by companies like DuPont and 3M to make products more stain-resistant, waterproof and/or nonstick (e.g. Teflon). In particular, PFOA been used in the following products: cookware, carpets, clothing, fabrics for furniture, paper packaging for food, and other materials. It is also been used in firefighting foams and in a number of industrial processes.[15]

Problematically, PFOA is quite resistant to biodegradation, which contributes to its widespread presence. Blood studies show the presence of PFCs in the blood of 96% of people in

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[10] 25 Pa. Code § 109.203 (emphasis added).
[11] 35 Pa. Stat. § 721.4; see also 25 Pa. Code § 109.203.
[12] Commonwealth of Pennsylvania: Bureau of Water Standards and Facility Regulation, *Citizen's Guide to Volatile Synthetic Organic Chemicals in Drinking Water*, http://pa-montgomerycounty.civicplus.com/DocumentCenter/View/916, (last updated August 2008).
[13] 25 Pa. Code § 109.302(c).
[14] 25 Pa. Code § 109.6(d).
[15] PADEP Fact Sheet available here: http://files.dep.state.pa.us/Water/DrinkingWater/Perfluorinated%20Chemicals/PFC%20Info%20Sheet.pdf

the United States, at approximately 4 ng/mL (nanogram/milliliter). It has even been found in polar bears in the Arctic. Its durable nature also causes the chemical to build up in the human body and is difficult to excrete, the levels in an individual's blood is about 105 times the amount in their drinking water. According to the U.S. Environmental Protection Agency (EPA): "Because PFOA can remain in the body for a long time, drinking water that contains PFOA can, over time, produce concentrations of PFOA in blood serum that are higher than the concentrations of PFOA in the water itself."[16] This magnification of PFOA in the blood can have serious health effects.

The scientific literature and the data gleaned from health studies show that PFCs, and PFOA, are linked to serious disease, including cancers, and detrimental human health conditions.[17] Fetuses, infants, and children are the most vulnerable populations due to negative developmental impacts, which also affect pregnant women, women of child bearing age and women who are breastfeeding. Chief among the new bodies of data and findings available for PFOA are those from the court-ordered C8 Health Panel and the C8 Health Project in West Virginia, related to the Dupont facility there. Among the conclusions of this multi-year study of human subjects, their blood and scientific reports, it was found that PFOA is correlated with Kidney Cancer, Testicular Cancer, Thyroid Disease, High Cholesterol, Pregnancy-Induced Hypertension/Preeclampsia, and Ulcerative Colitis.[18] In addition to the six diseases with probable links, the study also verifies probable links to decreased birth weight and decreased response to vaccines. A report reviewing all of the studies on low birth weight concluded that PFOA does reduce human birth weight. [19]

According to the New Jersey Drinking Water Quality Institute's recent report:

> Human exposure to PFOA has also been associated with increased risk of cancer, including increased risk of kidney and testicular cancer in communities with contaminated drinking water after adjustment for smoking and other relevant factors. These studies accounted for smoking history and other relevant factors. In 2006, the U.S. EPA Science Advisory Board described PFOA as "likely to be carcinogenic to humans." based on the criteria provided in U.S. EPA cancer risk assessment guidance. More recently, the International Agency for Cancer Research concluded that PFOA is possibly carcinogenic to humans. In 2016, the U.S. EPA Office of Water described it as having suggestive evidence of carcinogenic potential.[20]

Sources of exposure to PFOA and/or its precursors include drinking water, food and food packaging, treated fabrics, protective sprays and waxes, cosmetics and personal care products,

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[16] EPA-SAB-06-006 SAB Review of EPA's Draft Risk Assessment of Potential Human Health Effects Associated with PFOA and Its Salts

[17] https://www.epa.gov/sites/production/files/2015-09/ucmr-3-occurrence-data.zip

[18] http://www.c8sciencepanel.org/newsletter10.html

[19] http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4181929/pdf/ehp.1307893.pdf

[20] NJDWQI (2016). Health-based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA). New Jersey Drinking Water Quality Institute Health Effects Subcommittee, Public Review Draft, June 27, 2016. Retrieved from http://www.nj.gov/dep/watersupply/pdf/pfoa-hb--mcl-public-review-draftwithappendices.pdf (Enclosed as **Attachment 1**), p. 8.

house dust, and inhalation of indoor and outdoor air.[21] The contribution of ingested drinking water to total exposure from all sources (e.g. diet, consumer products, etc.) is dependent on the concentration of PFOA in the drinking water, and relatively low concentrations in water substantially increases the human body burden.[22]

Furthermore, exposures to PFOA may be higher in young children than in older individuals because of age specific behaviors such as greater drinking water and food consumption on a body weight basis, hand-to-mouth behavior resulting in greater ingestion of house dust, and more time spent on floors where treated carpets are found.[23]

In 2006, the eight major U.S. producers of PFOA voluntarily agreed to reduce emissions and product content of PFOA and related substances, including precursors of PFOA, on a global basis by 95% by 2010 and to work towards elimination of these substances by 2015.[24] According to the U.S. EPA, reports submitted by the participating companies in 2013 and 2014 indicated that they were on track to achieve the goal of phasing out these chemicals by the end of 2015. Nonetheless, PFOA remains ubiquitously present in our environment and presents a serious public health threat as it is linked to serious disease, including cancers, and detrimental human health conditions.[25]

3. PFC Presence in Pennsylvania

PFOA is significantly elevated in many Bucks and Montgomery County water supplies at levels that far exceed EPA's HAL and the more protective 6 ppt standard that we advocate for in this petition. Other locations in Pennsylvania have PFOA water supply contamination as well. Furthermore, if the Commonwealth undertook appropriate study of likely sources of PFOA, it is highly likely that it would identify scores of other locations with similar contamination issues.

a. PFCs in PA: Initial Findings

The PFOS and PFOA levels found in public wells in Bucks County and Montgomery Counties were among the ten highest sampling results in the nation.[26] Sampling done in Warminster, Warrington and Horsham Townships reported that the groundwater that feeds public and private wells for tens of thousands of people in the area was found to be among the worst in the nation, most all in the vicinity of the former Naval Air Station Joint Reserve Base at Willow Grove, the current Horsham Air Guard Station in Horsham and the site of the former Naval Air Warfare Center in Warminster. In Doylestown Township where testing was done for UCMR3 as a smaller representative public water supply, the Municipal Authority had the sixth highest PFOA sample report in the nation but to date the source is not publicly known.[27] Sampling of drinking water in other parts of the state has also revealed contamination problems

[21] *Id*. at 4.
[22] *Id*. at 5.
[23] *Id*.
[24] *Id*.
[25] *Id*. at 22.
[26] https://www.epa.gov/sites/production/files/2015-09/ucmr-3-occurrence-data.zip
[27] Ibid.

and there could well be more places, as yet undetected, where people are currently drinking contaminated water containing PFCs.

Sampling for the UCMR3 2015 report that revealed the presence of PFCs in Pennsylvania at levels above specific reporting levels (for PFOA UCMR3 reporting level was 20 ppt; for PFOS it was 40 ppt), all in parts per trillion or ppt[28] include:

- Aqua PA, Bristol: PFOA 20 and 26
- Warminster Municipal Authority Well 2: PFOA 34, PFOS 57
- Warminster Municipal Authority Well 5: PFOA 23
- Warminster Municipal Authority Well 9: PFOA 20
- Warminster Municipal Authority Well 10: PFOA 89, PFOS 190
- Warminster Municipal Authority Well 13: PFOA 122, PFOS 160
- Warminster Municipal Authority Well 14: PFOA 25, PFOS 65
- Warminster Municipal Authority Well 26: PFOA 350, PFOS 1090
- Warrington Township Water and Sewer Wells 1, 2, and 6 treatment plant: PFOA 120, PFOS 670
- Warrington Township Water and Sewer Well 3: PFOA 20, PFOS 62
- Warrington Township Water and Sewer Well 9: PFOA 29
- Quakertown Borough Well 13: PFNA 35 and 32
- Doylestown Municipal Utilities Authority Cross Keys: PFOA 210 and 130, PFNA 26
- Ambler Borough Water Department: PFNA 29
- Horsham Water and Sewer Authority Well 10: PFOA 26, PFOS 45
- Horsham Water and Sewer Authority Well 17: PFOA 26, PFOS 97
- Horsham Water and Sewer Authority Well 21: PFOS 140
- Horsham Water and Sewer Authority Well 26: PFOA 290, PFOS 700

[28] DRN is only reporting here results for PFOA, PFOS and PFNA; some wells show presence of other PFCs; all data available at UCMR3 occurrence text file for method 537 at https://www.epa.gov/sites/production/files/2015-09/ucmr-3-occurrence-data-by-method-classification.zip

- Horsham Water and Sewer Authority Well 40: PFOA 63, PFOS 1000

- EMMAUS Borough Public Water: PFNA 22

- United Water PA Airport (PWS ID# PA7220015, Harrisburg): PFOA 38, PFOS 363, PFNA 47

Many of these samples exceed EPA's HAL for PFOA of 70 ppt, and all samples exceed New Jersey's proposed PFOA MCL of 14 ppt and the more protective 6 ppt MCL we are requesting.

b. Current Conditions in Bucks and Montgomery Counties

The former Naval Air Station Joint Reserve Base at Willow Grove, the current Horsham Air Guard Station in Horsham and the site of the former Naval Air Warfare Center in Warminster are the primary sources of PFC contamination in Bucks and Montgomery Counties. The military has been using firefighting foams for decades at these locations.

The Naval Air Warfare Center in Warminster, Bucks County was designated for closure in 1995 by the Defense Base Closure and Realignment Commission (BRAC) program and now operates under BRAC as the Naval Air Development Center. It is classified as a CERCLA National Priority List (NPL) "Superfund" site due to contamination of area groundwater, primarily trichloroethylene (TCE), tetrachloroethylene (PCE) and carbon tetrachloride documented in 1989.[29] A treatment system is operating on the site that removes Volatile Organic Compounds from the groundwater under BRAC's supervision. The site is 824 acres and is located in Warminster Township, Ivyland Borough and Northampton Township. The area has used groundwater for both public and private water supplies. A groundwater analysis is being conducted by the Navy to assess where the PFC pollution plume is and where it is going, according to the Willie Lin of BRAC, but no results are available publicly at this time.[30]

The Horsham Air Guard Station opened in 2011 after the 2006 base closure of the adjacent former Willow Grove Naval Air Station Joint Reserve Base. It is located on approximately 207 acres in Horsham Township, Montgomery County. In the 1990s, the Willow Grove Naval Air Station Joint Reserve Base investigated groundwater contamination from volatile organic compounds (VOC's) in the aquifer; cleanup was carried out of underground storage tank leaks. The site was classified as a Superfund site in 1995. Site remediation needs and the impact of proposed site changes regarding water resources, air quality hazardous materials and solid and hazardous waste, utilities and other affected environmental aspects are still being assessed.[31]

[29] https://cumulis.epa.gov/supercpad/cursites/csitinfo.cfm?id=0302466
[30] Statement of Willie Lee, US NAVY BRAC as per Tracy Carluccio, DRN, at Northampton Township Board of Supervisors meeting, 1.25.2017.
[31] http://horshamlibrary.org/docview.aspx?docid=28289

PFCs were discovered at these bases during the 2012 Five Year Superfund Review of the Naval Air Warfare Center in Warminster, according to the EPA.[32] The public subsequently found out about PFOA and PFOS in their drinking water through the UCMR3 reporting from 2013 to 2015. The Navy shut down two Warminster water wells as early as 2014 because of PFCs.

At the commencement of 2017, 22 public drinking water wells and 230 private drinking water wells have been shut down by a variety of agencies because they exceed the 70 ppt EPA HAL in Warminster, Warrington and Horsham Townships. As testing is completed in more places, more wells report contamination by PFOA and/or PFOS, some at concentrations that exceed the EPA HAL and some that are under that level.

Since the UCMR3 sampling, additional water testing in the region around the military bases has revealed PFOA and PFOS contamination in more locations. The most recent count is seventeen communities in Bucks and Montgomery Counties where some level of PFOA/PFOS has been discovered. The seventeen communities in Bucks and Montgomery Counties with water wells that have been found to contain some concentration of PFOA and/or PFOS at levels leading to the shutdown of the well or at levels lower than the EPA HAL include:

- Horsham
- Warminster
- Warrington
- Abington
- Bristol Township
- Bristol Borough
- Buckingham Township
- Doylestown Borough
- Northampton Township
- East Rockhill Township
- Upper Southampton Township
- Doylestown Twp
- Ivyland
- Plumstead Township
- Upper Dublin
- Warwick
- Hatboro

Some communities in the region are provided drinking water by municipal authorities, some are served by regional water companies, and some people have private individually owned wells. Water systems proximate to the military bases are testing the water they provide for PFOA and PFOS even though there is no maximum contaminant level in force, presumably under PADEP guidance. For instance, Aqua America serves multiple municipalities in Bucks and Montgomery Counties and has set up a testing protocol and a web page, making data available to the public.[33] The company has found PFCs in some of their water sources and their data shows that concentrations change in their wells over time. PFOA and PFOS has also been found in surface water the company uses, most notably high concentrations of PFOA and PFOS at their Neshaminy Creek intake.[34] Aqua America is using the EPA HAL to measure if the water

[32] Ibid.
[33] http://www.waterfacts.com/
[34] http://www.theintell.com/news/horsham-pfos/pfcs-increasing-in-neshaminy-creek-widespread-in-northampton-wells/article_1813c26a-7f4f-11e6-8aaa-1fe2604163a6.html

is safe.[35] In some instances, water companies and municipal authorities are blending water that contains some level of PFCs with cleaner water to dilute the concentration to below the EPA HAL.

The Navy is employing the EPA HAL as if it were a safe drinking water level and only taking action if PFOA and PFOS concentrations exceed 70 ppt. The Navy is replacing or treating water supplies in some municipalities that have contaminated wells and are connecting some private wells with systems that meet the EPA HAL. The Navy has committed publicly to pay for the replacement or treatment of all water supplies that exceed the EPA HAL (70 ppt) if the Navy concludes that the water was contaminated by their facility. So far, $19 million has been committed. However, this does not cover the cost of remediating all contamination and sampling has not been done of all water supplies that could have been contaminated.

Action by the Navy has not addressed all of the contamination issues; indeed actions have varied greatly from municipality to municipality. For instance, the Navy has sampled some but not all private water wells in the region. Where the Navy's sampling of private wells show an exceedance of the EPA HAL, the Navy is supplying bottled water while permanent connections are being arranged. However, where sampling has not been done by the Navy, the private well owner is on their own to test their water and, if there are PFCs, the Navy will not commit to taking any action until they are ready and will not commit to reimbursing homeowners who install treatment systems.[36] This leaves private well owners left to buy point-of-entry treatment systems on their own at a cost of thousands of dollars.

The delivery of contamination-free water is not uniform and some municipalities have set different policies that are resulting in different outcomes in terms of the presence of PFCs. For instance, Warminster, Warrington and Horsham Townships are employing plans to remove PFOA and PFOS to a concentration of "non-detect" due to zero tolerance for any PFCs in their water supplies. The Navy's use of the EPA HAL does not align with the municipalities' plans and excludes many water wells and systems that contain PFC contamination from being acted on by the Navy. Municipalities do not have the funds to address all of the contamination. The municipalities' "non-detect" policy is being adopted by municipal officials and supported by some state and federal representatives from the region seeking protection of residents from exposure to PFOA and PFOS due to concern for an increased risk of developing disease and adverse health impacts, especially for vulnerable populations such as infants, children, and women of childbearing age. It is being carried out despite an expected substantial rate hike for customers in some municipalities such as Horsham.[37] Warminster Township has estimated their costs could go as high as $26 million.[38]

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[35] Ibid.

[36] Statement of Willie Lee, US NAVY BRAC as per Tracy Carluccio, DRN, at Northampton Township Board of Supervisors meeting, 1.25.2017.

[37] http://www.theintell.com/news/horsham-pfos/horsham-residents-to-pay-surcharge-for-additional-north-wales-water/article_e129c946-712c-11e6-8c8c-bf8764ec9b59.html

[38] http://www.theintell.com/news/horsham-pfos/state-rep-kathy-watson-hints-at-upcoming-pfc-bill/article_7c904d36-cdf5-11e6-bcd1-37acef07f06e.html

In addition, the Horsham Air Guard Station is acting independently from the Navy BRAC, with uneven results. For instance, it was stated by State representative Kathy Watson that military officials said that Horsham Air Guard Station is still contributing PFC contaminated flow to the Little Neshaminy Creek and Park Creek.[39] Both creeks flow to the Neshaminy Creek, the source of drinking water for downstream residents, including Aqua America that wheels water to several locations not obviously local to the military bases.[40] The result of the lack of a comprehensive plan for addressing the problem has led to some residents receiving more protection than others and the lack of a uniform and thorough military response shouldering the cleanup costs has meant that much of the burden of increased costs is being carried by the public and some communities have a greater burden than others.

In Horsham Township, for instance, granulated activated carbon (GAC) treatment to remove PFCs is being installed and water that has any level of PFOA or PFOS is being replaced with water that has no detection of the compounds for both public and private water users.[41] A $10 million grant will come from the Pennsylvania Infrastructure Investment Authority to help defray some of the costs of attaining PFC-free water for township residents.[42] The total costs, however, are not yet tallied. Yet, other communities have not adopted a "non-detect" policy and are only applying the EPA HAL as a trigger for action. This is resulting in uneven protection for residents in the region.

Many water systems may have PFOA and PFOS in the water they provide to consumers but they do not know it because the water is not being sampled or is not being tested to or reported at a low enough level to find where it is occurring. This is especially a problem for areas where there may have been releases of PFCs to the environment from unknown sources. This is the case in Doylestown Township, Bucks County. Doylestown Township was included in the UCMR3 sampling from 2013-2015, which is when the contamination was first discovered but no action was taken until the EPA HAL was issued.

After the EPA issued its HAL, the Doylestown Township Cross Keys well was shut down by the Township's Municipal Authority in May 2016.[43] The pollution source is unknown but the well is located near the Doylestown Airport and a site that handled waste for a laboratory. See more information on this occurrence later in this report.

c. PFCs in Other Pennsylvania Locations

Elsewhere in Pennsylvania, the Susquehanna Area Regional Airport Authority (SARAA) sells water to Suez[44], formerly known as United Water Pennsylvania. SARAA shut down three wells in June 2014 after being notified of "elevated" levels of PFOS. In 2014, EPA's provisional

[39] Ibid.
[40] http://www.theintell.com/news/horsham-pfos/pfcs-increasing-in-neshaminy-creek-widespread-in-northampton-wells/article_1813c26a-7f4f-11e6-8aaa-1fe2604163a6.html
[41] https://www.horshamwater-sewer.com/news/short-term-plan-progress
[42] http://www.theintell.com/news/horsham-pfos/rep-todd-stephens-confirms-million-for-pfc-clean-up/article_ddec8bfc-e34f-11e6-835c-f7387628e06d.html
[43] http://bit.ly/2kCtcje
[44] http://www.mysuezwater.com/about-us

health advisory was 200 ppt and the SARAA sampling showed the concentration to be from 400 to 1100 ppt, which triggered the shutdown. The areas that get their drinking water from those wells were reportedly Lower Swatara Township and Highspire. That connection served about 2,700 customers, including Penn State's Capitol Campus, and may also have provided water to Harrisburg International Airport (HIA) and commercial customers north of the Airport but the precise number of customers that have been exposed to the PFC-contaminated water is unclear. As per EPA's UCMR3 Report, other PFCs were found in at least one well at the Airport: PFOA at 38 ppt and PFNA at 47 ppt. Those results indicate the EPA HAL combined concentration for PFOA and PFOS of 70 ppt issued in 2016 was exceeded based on the 2014-2015 data available. The 2015 UCMR3 sampling documented a PFOS concentration of 363 ppt.

In response to the UCMR3 sampling results, PADEP and SARAA changed pumping regimes to use water that was below the provisional HAL for PFOS in 2014 (200 ppt) and United Water closed the interconnection with SARAA.[45] A plan for long-term treatment to remove PFCs is in process, as reported by EPA.[46]

The source of the PFC contamination is not settled but the EPA Superfund Report for Middletown Air Field sheds light on the history of groundwater contamination at the Air Field, operated by the United States Air Force (USAF).[47] The Harrisburg International Airport (HIA) occupies part of the Middletown Air Field. In 1983 when groundwater contamination by chlorinated solvents was discovered in wells at HIA, the Pennsylvania Department of Transportation owned the airport. The state of Pennsylvania, represented by PADEP, and the USAF were involved in the cleanup of the Superfund site in the 1980's and 1990's. The site was removed from the Superfund National Priorities List in 1997. Groundwater continued to be supplied through water wells at HIA through United Water, now Suez, until the three wells were shut down and pumping changes went into effect in 2014.

Since 2014, sampling of some private wells in the Harrisburg area has been conducted under the federal Hazardous Sites Cleanup Act of 1988.[48] Currently, in instances when the wells' PFC level exceeded the EPA HAL, alternative drinking water supplies have been provided, according to PADEP's website.[49] Sampling was done in Middletown Borough as well but the data for these and other wells is not available publicly except that EPA states that the concentrations did not exceed the EPA HAL.[50]

Other locations where PFCs found in exceedance of the EPA HAL are being investigated in Pennsylvania, according to PADEP are:

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[45] https://cumulis.epa.gov/supercpad/cursites/dsp_ssppSiteData1.cfm?id=0301295
[46] Ibid.
[47] Ibid.
[48] http://www.dep.pa.gov/Citizens/My-Water/drinking_water/Perfluorinated%20Chemicals%20%e2%80%93PFOA%20and%20PFOS%20%e2%80%93%20in%20Pennsylvania/Pages/DEP-Program-Involvement.aspx
[49] ibid.
[50] ibid.

- Ridge Run Road, Bucks County - parts of East Rockhill and West Rockhill Townships, and Perkasie Borough. One public water supply well exceeded the HAL; one was just under the HAL. Private water well sampling is underway for a one mile radius.[51] A survey and letter to residents within the radius was sent by PADEP in November 2016.[52] The contamination source is not publicly identified.[53]
- Easton Road, Bucks County – parts of Doylestown, Plumstead and Buckingham Townships. The Doylestown Municipal Utilities Authority "Cross Keys" public water supply well was documented to contain combined concentrations of PFOA and PFOS that exceeded the EPA HAL as part of the UCMR3 sampling. Concentrations of PFOA at 210 and 130 ppt and PFNA at 26 ppt were reported in the 2015 UCMR3 sampling results.[54] PADEP took no action to shut down the well until EPA issued the lifetime HAL in 2016; the well was then shut down by the Water Authority and since then water from approximately 280 private wells within 1 mile of the contaminated well has been sampled.[55] PADEP reports that bottled water is being provided to any resident whose water exceeds the EPA HAL.[56] The contamination source has not yet been identified.[57] Round One sampling results show that most private wells sampled had the presence of PFCs with three wells above the combined EPA HAL for PFOA and PFOS of 70 ppt and many wells above 10 ppt.[58,59] PADEP announced they will conduct a second round of sampling in an expanded area and a third round to re-sample properties with results that exceeded 40 ppt.[60]

In addition to these sites under investigation in Pennsylvania, there are other sites that are likely sources of PFCs, particularly PFOA and PFOS. These sites include: military facilities, firefighting and aviation testing sites; fire departments where foam was stored, used and/or tested; aqueous firefighting foam manufacturers, testers, and suppliers; airports; wastewater treatment facilities and their discharge points; sewage sludge and dredge spoils application sites; and manufacturing sites that manufactured or used PFCs in their process. Some of these sites

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[51] http://files.dep.state.pa.us/Water/DrinkingWater/Perfluorinated%20Chemicals/Ridge-Run-Map.jpg
[52] http://files.dep.state.pa.us/Water/DrinkingWater/Perfluorinated%20Chemicals/RidgeRunWellSurvey.pdf
[53] http://www.ahs.dep.pa.gov/NewsRoomPublic/articleviewer.aspx?id=21105&typeid=1
[54] https://www.epa.gov/sites/production/files/2015-09/ucmr-3-occurrence-data.zip
[55] http://files.dep.state.pa.us/RegionalResources/SERO/SEROPortalFiles/Community%20Info/EastonRoadPFC/Site%20area%20radius%20with%20TWPs.pdf
[56] http://files.dep.state.pa.us/RegionalResources/SERO/SEROPortalFiles/Community%20Info/EastonRoadPFC/Easton%20Road%20Information%20Sheet_final.pdf
[57] http://files.dep.state.pa.us/Water/DrinkingWater/Perfluorinated%20Chemicals/Easton%20Rd%20PFC%20FAQ_final%20(1)%20%20.docx
[58] http://files.dep.state.pa.us/Water/DrinkingWater/Perfluorinated%20Chemicals/EastonRoadPFCSite/Sampling%20Summary%20-%20Round%201.pdf
[59] http://files.dep.state.pa.us/Water/DrinkingWater/Perfluorinated%20Chemicals/EastonRoadPFCSite/Sampling%20Map%20-%20Round%201.pdf
[60] http://files.dep.state.pa.us/Water/DrinkingWater/Perfluorinated%20Chemicals/EastonRoadPFCSite/rls-DEP-SERO-Easton%20Road%20update%2012062016.pdf

can be researched through Department of Defense; USEPA regulatory and reporting programs; EPA Superfund (C.E.R.C.L.A.) sites; the Hazardous Sites Cleanup Program; and Resource Conservation and Recovery Act sites. Sampling at these sites is essential to provide an informed and accurate assessment of the scope of the PFC contamination problem in Pennsylvania. The cost of sampling should be borne by the Department of Defense in locations that could have been affected by a military facility or activity and can be paid for through existing programs that are reimbursed by responsible parties. Sampling cannot wait. It is crucial that people know if their drinking water is contaminated by the presence of PFCs.

By way of illustration, military facilities where firefighting foam was used or training for firefighting was carried out include several sites in Pennsylvania that have the potential to cause groundwater contamination by PFCs, particularly PFOA and PFOS. These sites could enormously increase the locations where PFC-contaminated drinking water is discovered within Pennyslvania. These include:

- **Letterkenny Army Depot (originally Letterkenny Ordnance Depot)**, the Center of Industrial and Technical Excellence (CITE) for Air Defense and Tactical Missile Systems under the U.S. Army Aviation and Missile Command encompassing 18,000 acres in Letterkenny Township and extending into Greene Township and Hamilton Township, all in Franklin County.
- **Fort Indiana Gap** under the U.S. Army in Lebanon and Dauphin Counties is still an active National Guard Training Center and is the headquarters for the Pennsylvania National Guard and Pennsylvania Department of Military and Veterans Affairs.
- **NORTH PENN USARC** owned by the U.S. Army, near Worcester, Montgomery County is a private airport and heliport where there was a fire training area burn area.
- **Tobyhanna Army Depot,** located in Coolbaugh Township, Monroe County, under the command of the Department of Defense was a military equipment and firefighting training center, today specializing in electronic systems and intelligence for all branches of the Armed Forces.
- **Philadelphia Naval Shipyard** in Philadelphia was a centrally important shipyard under the U.S. Navy for more than 200 years on the Delaware River. Closed in the 1990's, it was used as a fire training site in addition to an active ship yard.
- **Defense Logistics Agency Susquehanna** is located in New Cumberland and Mechanicsburg, and is the Department of Defense's largest distribution processing facility. Firefighting testing and products are handled there.

The inventory of aqueous firefighting foam that contains PFOA and PFOS in the United States is estimated at about 9.9 million gallons, rounded off to the nearest tenth of a million gallons. These are located at military bases, aviation facilities, merchant ships, fire departments, oil refineries, petro-chemical facilities, and other locations. These stockpiles are geographically scattered and all are a potential source of PFC release to the environment.

Examples of a widely dispersed potential source of PFCs are fire departments and firefighting schools. There are over 27,000 fire departments in the U.S. and hundreds in Pennsylvania.[61] Firefighting foam is an essential tool used routinely by these stations. Aqueous firefighting foam containing PFCs has not been phased out yet, especially because the shelf life of this foam is about 20 years. The proper disposal of foams and the containment of foam when used to put out a fire are not regulated in a manner that prevents inadvertent release into the environment. When used, foams end up in the air and/or on land and in water.

Another example of geographically dispersed locations where PFCs may have been released is airports. There are more than 140 active or decommissioned airports in the Commonwealth. Airports are a known source of PFC contamination in New Jersey and other locations. Foam manufacturers are also locations where groundwater should be sampled. For instance, National Foam in West Chester, PA is a large aqueous firefighting foam manufacturer and supplier. [62]

Pennsylvania should sample all locations where PFCs may have been released to the environment to discover the occurrence in the state. Pennsylvania is a large state with many locations where this is likely to have occurred. PFCs, resistant to being broken down in the environment and extremely durable, are carried into waterways and percolate into groundwater from the soil, carrying toxic properties that persist indefinitely. To protect human health and the environment, it is crucial that surface water, groundwater and groundwater wells, as well as soils and other media where releases could have occurred, be sampled to discover if PFCs are present as the first step towards the adoption of a maximum contaminant level for PFOA in the Pennsylvania. The Pennsylvania Safe Drinking Water Act compels the EQB to act to protect the public from these highly toxic compounds that are, at present, unregulated. Once it is known how widespread these contaminants are in the state, it will become clear that a MCL is immediately necessary for PFCs statewide, starting with PFOA.

4. A MCL for PFOA not to exceed 6 ppt must be set in Pennsylvania

The New Jersey Drinking Water Quality Institute (NJDWQI) has recently recommended that the NJDEP adopt a MCL for PFOA at 14 ppt. However, a recent study by the Cambridge Environmental Consulting (CEC) demonstrates that the NJDWQI has overlooked recent relevant studies, failed to account for children in their analysis, and recommends that a MCL of 1 ppt is feasible and most protective of human health, but at minimum a MCL for PFOA should not exceed 6 ppt.[63]

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[61] https://en.wikipedia.org/wiki/List_of_Pennsylvania_fire_departments

[62] http://nationalfoam.com/

[63] Oliaei, F.Z., & Kriens, D.L. (2016). Proposed Health-Based Maximum Contaminant Level (MCL) for Perfluorooctanoic Acid (PFOA) in Drinking Water. Technical Analyses of New Jersey Drinking Water Quality Institute, Cambridge Environmental Consulting, November 18, 2016. Retrieved from http://www.delawareriverkeeper.org/sites/default/files/cvr%20ltr%20PFOA%20mcl%20cmnt11.19.combinedpdf.pdf (Enclosed as **Attachment 2**).

a. Summary of the NJDWQI Report (14ppt PFOA MCL)

On March 21, 2014, NJDEP Commissioner Bob Martin requested that a MCL be developed for PFOA in drinking water in New Jersey.[64] The NJDWQI issued a public review draft of a MCL for PFOA of 14 ppt dated June 27, 2016 on September 12th.[65] A 60 day comment period followed from September 22 – November 21, 2016. On February 16, 2017, the NJDWQI members unanimously approved its recommendation of 14 ppt.[66] On March 15, 2017 the NJDWQI transmitted to NJDEP with its Basis and Background document its recommendation of a health-based Maximum Contaminant Level for PFOA of 14 ppt.[67] The MCL was developed with guidance from the 2005 USEPA draft risk assessment for PFOA and also considered were the conclusions of the USEPA Science Advisory Board in 2006.[68]

The NJDWQI deviated from some of USEPA's conclusions because the 2005 USEPA draft risk assessment problematically did not develop a cancer slope factor or Reference Dose (Rfd) for PFOA, and it did not address the relationship between human body burden and drinking water concentration, as measured by blood serum level.[69] Comparisons between effect levels in human exposures and animal studies were made by the NJDWQI on the basis of serum levels rather than external dose because the half-life of PFOA is much longer in humans (several years) than in the animal species used in the toxicological studies (several hours to 30 days).[70]

Seven health endpoints were evaluated comprehensively by the NJDWQI in the development of the MCL. These included: liver enzymes, liver disease, serum cholesterol/lipids, thyroid function, thyroid disease, uric acid, and antibody concentrations following vaccination. Some of the factors considered in selection of these endpoints were the consistency and extent of the data, evidence for reverse causality, and whether the effect has been observed at exposures relevant to potential drinking water exposures.[71] In total, 54 epidemiological studies from the United States, Canada, and several Asian and European countries were utilized.[72]

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[64] NJDWQI (2017). Maximum Contaminant Level Recommendation for Perfluorooctanoic Acid in Drinking Water, Basis and Background. New Jersey Drinking Water Quality Institute, March 15, 2017. http://www.nj.gov/dep/watersupply/pdf/pfoa-recommend.pdf (Enclosed as **Attachment 3**) at 2.

[65] See **Attachment 1** (NJDWQI (2016). Health-based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA). New Jersey Drinking Water Quality Institute Health Effects Subcommittee, Public Review Draft, June 27, 2016. Retrieved from http://www.nj.gov/dep/watersupply/pdf/pfoa-hb--mcl-public-review-draftwithappendices.pdf at 23).

[66] http://www.nj.gov/dep/watersupply/pdf/minutes170216.pdf

[67] See **Attachment 3** (NJDWQI (2017). Maximum Contaminant Level Recommendation for Perfluorooctanoic Acid in Drinking Water, Basis and Background. New Jersey Drinking Water Quality Institute, March 15, 2017. http://www.nj.gov/dep/watersupply/pdf/pfoa-recommend.pdf)

[68] See **Attachment 1** (NJDWQI (2016). Health-based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA). New Jersey Drinking Water Quality Institute Health Effects Subcommittee, Public Review Draft, June 27, 2016. Retrieved from http://www.nj.gov/dep/watersupply/pdf/pfoa-hb--mcl-public-review-draftwithappendices.pdf at 23).

[69] *Id*. at 23.

[70] *Id*. at 23.

[71] *Id*. at 60.

[72] *Id*. at 60.

The NJDWQI also used data from animal studies in developing its MCL.[73] In humans, it has been estimated that as much as 55% of PFOA exposure comes from drinking water.[74] The range of health-based drinking water concentrations for the seven endpoints assessed was 40-260 ppt, and multiple concentrations fell within a similar range (40, 50, 60, 70, and 80 ppt).[75] The most sensitive endpoints were hematological (blood) effects and decreased body weight in adult female rats in a chronic dietary study, which resulted in a drinking water concentration of 40 ppt.[76]

The health-based MCL developed by the NJDWQI was intended to be protective for lifetime (chronic) exposure through drinking water.[77] It was based on well-established and sensitive animal toxicology endpoints that are considered relevant to humans based on mode of action data.[78] Delayed mammary gland development from exposure around birth is the most sensitive systemic endpoint for PFOA. However, the Health Effects Subcommittee decided not to recommend a Health-based MCL with the RfD for delayed mammary gland development as its primary basis because it believed the use of this endpoint as the basis for human health criteria is a currently developing topic.[79] Therefore, the NJDWQI did not calculate an MCL based on delayed mammary gland development.

Instead, increased liver weight was the primary endpoint for the NJDWQI's Heath-based MCL.[80] Increased relative liver weight is a well-established effect of PFOA that is more sensitive than most developmental/reproductive effects and other toxicological effects such as immune system toxicity. A Relative Source Contribution (RSC) factor that accounts for non-drinking water sources including water, air, soil, food, and consumer products was used in the development of health-based drinking water concentrations based on non-carcinogenic effects. In addition to its use by the NJDWQI, an RSC is used by the USEPA for Maximum Contaminant Level Goals and by other states in development of similar health-based drinking water values. The RSC is intended to prevent total exposure from all sources from exceeding the RfD.[81]

The Health Effects Subcommittee concluded that there are insufficient data to develop a chemical-specific RSC for PFOA.[82] There are no New Jersey-specific biomonitoring data for PFOA, and its frequent occurrence in public water supplies suggests that New Jersey residents may also have higher exposure from non-drinking sources than the general population in the U.S.[83] The exposure factors used to develop the Health-based MCL are based on body weight and an adult drinking water consumption rate. Exposures to infants, both those who consume

[73] *Id.* at 203.
[74] *Id.* at 41.
[75] *Id.* at 24.
[76] *Id.* at 24.
[77] *Id.* at 203.
[78] *Id.* at 204.
[79] *Id.* at 205.
[80] *Id.* at 210.
[81] *Id.* at 215.
[82] *Id.* at 215.
[83] *Id.* at 215.

formula prepared with contaminated drinking water and those who are breastfed, are much higher than in older individuals and therefore a default RSC of 20% was used.[84]

For carcinogenic effects, dose-response modeling was based on administered PFOA dose to rats (mg/kg/day) instead of internal dose (serum PFOA level) since serum PFOA levels were not measured in the study.[85] As per the 2005 USEPA guidelines for carcinogen risk assessment, converting the doses from rats to humans was made based on drug interaction differences between species instead of through the default adjustment based on body weight.[86] In calculating the MCL, the half-lives used for this adjustment were 7 days for male rats and 840 days for humans.[87] The human lifetime cancer risk was one in one million (1×10^{-6}) and default drinking water assumptions were 2 L/day with a body weight of 70 kg.[88] Using these values, the NJDWQI recommended a MCL of 14 ppt for both increased liver weight and carcinogenic risk.[89]

Ongoing exposure to the recommended health-based MCL of 14 ppt is expected to increase blood serum PFOA levels by about 1.6 ng/ml with average daily water consumption and 2.8 ng/ml with upper percentile daily water consumption in adults on average.[90] The proposed Health-based MCL includes an uncertainty factor to protect for more sensitive developmental effects. It is unknown whether it is sufficiently protective for more subtle effects that may occur later in life that may result from low exposures during the developmental period.[91] The chronic studies did not assess effects such as carcinogenicity which might result from exposures during the critical developmental stages that are identified to be sensitive periods for PFOA toxicity.[92]

There are also uncertainties about whether the human relevance of effects seen in animals is applicable to all risk assessments based on animal data.[93] Finally, the toxicity of PFOA and other PFCs may also be additive because the modes of action and target organs are typically similar for PFOA and other PFCs such as PFNA. Although PFOA and other PFCs, including PFNA, are known to co-occur in some NJ public water supplies, the potential for additive toxicity between these compounds was not considered in development of the Health-based MCL.[14] For these reasons, the NJDWQI recommended MCL may not be protective enough.

b. <u>The MCL for PFOA must be set not to exceed 6 ppt</u>

The Cambridge Environmental Consulting (CEC) prepared a technical analysis of the NJDWQI Health-based Maximum Contaminant Level Support Document for PFOA that was summarized above. CEC has concluded that the proposed drinking water MCL of 14 ppt for PFOA that is based on increased relative liver weight is not adequately protective of all

[84] *Id.* at 216.
[85] *Id.* at 219.
[86] Id. at 219.
[87] *Id.* at 220.
[88] Id. at 220.
[89] Id. at 220.
[90] *Id.* at 221.
[91] *Id.* at 221.
[92] *Id.* at 221.
[93] *Id.* at 221.

population segments.[94] Instead, CEC has recommended that the proposed MCL for PFOA should be lowered to 1 ppt, or alternatively, should be no higher than 6 ppt.[95]

CEC's recommendation of a MCL of 1 ppt is consistent with the values found pursuant to the immunotoxic epidemiologic study and/or animal studies showing adverse developmental effects. However, if these values are excluded, the CEC has identified that the PFOA MCL should be no greater than 6 ppt to assure protection of children.[96]

In particular, CEC disagrees with the NJDWQI's conclusion that the "review of epidemiologic studies provides evidence of consistent findings among studies of decreased antibody concentrations following vaccination and PFOA. However, while there is epidemiologic evidence of temporality, evidence of an exposure-response is limited."[97] Rather, CEC identifies that there is strong, significant epidemiologic evidence that includes quantitative data to enable derivation of a benchmark dose level (BMDL) and such data should be taken into account in derivation of the MCL.[98] CEC cites to a study by Grandjean and Budtz-Jørgensen that represents the greatest sensitivity to PFOA so far studied, un-confounded by exposure to other chemical contaminants.[99] The NJDWQI report does not refer to this study (although it does refer to an unrelated 2012 study by the same authors). Based on the acceptable dose level identified by Grandjean and Budtz-Jørgensen, CEC calculated that the MCL for PFOA should be 0.5 ppt.

CEC also disagreed with NJDWQI's decision to use increased liver weight as its primary endpoint when delayed mammary gland development is the more sensitive endpoint. The NJDWQI's reasoning to exclude the mammary gland endpoint (lack of precedent for delayed mammary gland development), does not explain why NJDWQI arbitrarily applied an additional 10 uncertainty factor to an unrelated endpoint (increased liver weight that forms the basis for their MCL derivation) as compensation.[100] Because adequate toxicity data already exists for the more sensitive delayed mammary gland development endpoint, this endpoint must be used when calculating a MCL.[101] Taking delayed mammary gland development into account, CEC proposed that the MCL for PFOA be 1 ppt.[102]

CEC also disagrees with NJDWQI's use of adult default exposure values because it omits protection for the population's most vulnerable exposure group, children. Children have a greater

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[94] See **Attachment 2** (Oliaei, F.Z., & Kriens, D.L. (2016). Proposed Health-Based Maximum Contaminant Level (MCL) for Perfluorooctanoic Acid (PFOA) in Drinking Water. Technical Analyses of New Jersey Drinking Water Quality Institute, Cambridge Environmental Consulting, November 18, 2016. Retrieved from http://www.delawareriverkeeper.org/sites/default/files/cvr%20ltr%20PFOA%20mcl%20cmnt11.19.combinedpdf.pdf)

[95] Id. at 3.

[96] *Id.* at. 3.

[97] *Id*. at 5.

[98] *Id*. at 5

[99] *Id*. at 5-6.

[100] *Id.* at 7.

[101] *Id*. at 7.

[102] *Id*. at 7.

rate of food and drinking water consumption based on body weight than adults do.[103] Calculation of an MCL using adult default values results in a reference dose (RfD) to children (age group 1-6) that significantly surpasses that deemed allowable by NJDWQI based on the increased liver weight toxicity endpoint.[104] Although the MCL should be based on human immunotoxicity and/or the delayed mammary gland development shown in test animals, CEC assert that, at a minimum, MCL calculations using increased liver weight as an endpoint should be based on children exposure values for drinking water intakes and body weight.[105] Using children group ages 1-6, the consultants conclude that an MCL of 5.65 ppt (rounded to 6 ppt) be promulgated.[106]

In conclusion, absent lowering the proposed PFOA MCL to 1 ppt, the MCL should be no higher than 6 ppt because (i) animal studies show significant delayed mammary gland development are appropriate and sufficient to use in the MCL determination and the NJDQWI failed to use this endpoint, (ii) substantial epidemiological evidence (e.g. study by Grandjean and Budtz-Jørgensen) show a significant association between PFOA and suppression of antibody responses in children, (iii) children exposure values mandate heightened protection, and (iv) toxic effects from PFOA exposures in early childhood may persist into adulthood and could result in more profound disease in later life.[107]

5. The challenges the PA Department of Environmental Protection identifies with setting a MCL for PFOA do not outweigh the significant public health risk of continued exposure to contaminated drinking water supplies

The Department identifies a number of perceived challenges with setting a state MCL for PFOA on their website.[108] Those include (a) lack of state funding and resources; (b) lack of data evaluating whether PFOA contamination is a statewide problem; and (c) lack of funding and resources to develop the science in support of a PFOA MCL.[109] Provided these challenges are resolved, the Department believes these additional steps must be complete prior to setting a MCL: (d) evaluate whether a PFOA MCL is technically feasible; (e) conduct a cost/benefit analysis of the proposed MCL; and (f) develop the necessary justification for proposing a standard that is more stringent than what the EPA has set.[110]

Importantly, all of these challenges can be resolved if the Commonwealth recognizes the serious health consequences of PFOA drinking water contamination and prioritizes committing the resources to set a MCL. This is particularly true for the first three challenges identified by the Department. Regarding the lack of data evaluating whether PFOA contamination is a statewide problem, as discussed at II.B.3. above, there is already ample data showing that PFOA

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[103] *Id*. at 8.
[104] *Id*. at 8.
[105] *Id*. at 8.
[106] *Id*. at 8.
[107] *Id*. 9-10.
[108] See http://www.dep.pa.gov/Citizens/My-Water/drinking_water/Perfluorinated%20Chemicals%20%e2%80%93PFOA%20and%20PFOS%20%e2%80%93%20in%20Pennsylvania/Pages/Establishing-a-State-MCL.aspx
[109] *Id*.
[110] *Id*.

presents a significant health risk in Bucks and Montgomery Counties' drinking water sources, effecting, at a minimum, over 70,000 residents. The Department need only engage in a targeted review of other similarly situated facilities statewide that are likely sources of PFOA and PFOS. These sites include: military facilities, firefighting and aviation testing sites; fire departments where foam was stored, used and/or tested; aqueous firefighting foam manufacturers, testers, and suppliers; airports; wastewater treatment facilities and their discharge points; sewage sludge and dredge spoils application sites; and manufacturing sites that manufactured or used PFCs in their process. Finally, the Department's claim that it lacks funding and resources to develop the science in support of a PFOA MCL fails to account for the NJDWQI's Draft Reports from its Health Effects, Treatment, and Testing Subcommittees which have provided the Department a substantial head start with its research.

Like the three challenges identified by the Department, the three additional next steps are also achievable. Much of the work involved in evaluating whether a PFOA MCL is technically feasible has been performed by NJDWQI subcommittees and DRN's consultants. The NJDWQI Treatment Subcommittee evaluated the treatment technologies for PFOA removal and found that granulated activated carbon was an effective removal technology.[111] Further research by DRN's experts Cambridge Environmental Consulting conclude that while granulated activated carbon has been highly effective in removing PFCs, the best available and economically achievable technology to remove PFOA from dilute aqueous streams at public water supplies is reverse osmosis.[112] Additionally, the NJDWQI Testing Subcommittee has recommended a practical quantification limit (PQL) of 6 ng/L for PFOA.[113] DRN's experts Cambridge Environmental Consulting have reviewed the PQL recommendation from the NJDWQI Testing Subcommittee and conclude that by using the method detection limit (MDL) approach a PQL of 3.0 ppt is achievable and by using the minimum reporting level (MRL) approach to determine a PQL for PFOA, a MRL of 2.0 ppt is achievable.[114]

The additional step requiring the Department to conduct a cost/benefit study of the proposed MCL for PFOA can be accomplished by the Department recognizing the imminent health consequences of PFOA in the public drinking water supply and prioritizing this work. The Department need only remember that the General Assembly has entrusted it to protect the drinking water supply of the citizens of Pennsylvania and that the Pennsylvania Constitution provides that each citizen has a right to clean and safe drinking water.

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[111] See NJDWQI Treatment Subcommittee Draft Report, Addendum to Appendix C: Recommendation on Perfluorinated Compound Treatment Options for Drinking Water (August 2016), p. 3. The draft report can be found here: http://www.nj.gov/dep/watersupply/pdf/addendum-public-review.pdf and is enclosed as **Attachment 4**.

[112] See **Attachment 2**, p. 7 (Cambridge Environmental Consulting, Technical Analysis of New Jersey Drinking Water Quality Institute – Recommendation of Perfluorinated Compound Treatment Options for Drinking Water (Nov. 18, 2016)).

[113] NJDWQI Testing Subcommittee Draft Report on the Development of a Practical Quantification Level for Perfluorooctanoic Acid (PFOA) in Drinking Water, 8/29/2016, p. 19. The NJDWQI Testing Subcommittee Draft Report is enclosed as **Attachment 5** and can be found here: http://www.nj.gov/dep/watersupply/pdf/testing-subcompql-pfoa-8.29.16KA.pdf.

[114] See **Attachment 2**. (Cambridge Environmental Consulting, Technical Analysis of New Jersey Drinking Water Quality Institute – Development of a Practical Quantitation Level for Perfluorooctanoic Acid (PFOA) in Drinking Water (Nov. 18, 2016).

C. Describe the types of persons, businesses and organizations likely to be impacted by this proposal.

All users of treated water, including residents, workers, businesses, agricultural animals and pets, and manufacturing such as consumer and food products, will benefit from the treatment of drinking water to a safe standard that protects human health. Infants, fetuses, women of childbearing age and children, known to be highly vulnerable populations to harm from PFOA exposure, will especially benefit from the use of safe drinking water. The dependency of these populations on adult decisionmaking put them at additional risk of exposure. Benefits include greater protection from disease that is correlated with exposure to PFOA and the multiple benefits of the removal of other potentially dangerous contaminants that are filtered out by the employed treatment technology, specifically through the use of recommended activated carbon filtration. As stated in the New Jersey Drinking Water Quality Institute's Report on Perfluorinated Compound Treatment Options for Drinking Water:

> Activated carbon is commonly used to adsorb contaminants found in water. It is used to remove synthetic organic chemicals, natural organic compounds, and other compounds affecting taste and odor in drinking water treatment.[115]

Persons who have been exposed to concentrations of PFOA in their drinking water for a period of time will benefit by having the compound removed so their body can excrete the compound over time. PFOA does not break down in the human body, accumulating and staying in the blood for years. The only way to reduce or eliminate its presence in the body is to stop exposure and allow for the slow process of natural elimination to take its course.

Treatment of water to remove PFOA will benefit groundwater and the environment because it will filter out the compound, allowing the residue to be disposed of in a safe manner. Since PFOA does not biodegrade, it persists in the environment indefinitely as a toxin. Groundwater, soil, vegetation, and other environmental media contain PFOA, allowing it to migrate to fish and fishlife. Delaware River Estuary surface water and fish flesh in the Delaware River Estuary contain concentrations of PFOA and other PFCs.[116] The treatment and removal of PFOA from drinking water will reduce the concentrations and distribution of PFOA, reducing the exposure to wildlife and to humans who consume fish, reducing the population's intake of PFOA-contaminated food.

Increased property values are also expected by improving the quality of available drinking water to communities that are now suffering depressed home and land values due to known drinking water contamination. Improved water quality can also increase the property values of nearby communities. According to Kauffman's report on the Socioeconomic Value of the Delaware River Basin:

> Several studies along rivers, estuaries, and coasts throughout the United States indicate that improved water quality can increase shoreline property values by 6% to 25% (Table

[115] http://www.nj.gov/dep/watersupply/pdf/pfna-pfc-treatment.pdf, p. 3.
[116] http://www.nj.gov/drbc/library/documents/contaminants-of-emerging-concernAug2013rev.pdf and http://www.nj.gov/drbc/library/documents/contaminants-of-emerging-concernAug2013rev.pdf

17). The EPA (1973) estimated that improved water quality can raise property values by up to 18% next to the water, 8% at 1000 feet from the water, 4% at 2000 feet from the water, and 1.5% at 3000 feet from the water. Leggett, et al. (2000) estimated that improved bacteria levels to meet state water quality standards along the western shore of the Chesapeake Bay in Maryland raised shoreline property values by 6%. The Brookings Institution (2007) projected that investments of $26 billion to restore the Great Lakes would increase shoreline property values by up to 10%. For this analysis, shoreline property values within 2000 feet of the waterways are estimated to increase by an average of 8% due to improved water quality in the Delaware Estuary.[117]

Finally, the federal and state Safe Drinking Water Act requires the provision of safe water to customers. Municipal, County and State government units and their water supply facilities will benefit from the treatment and removal of PFOA from drinking water by delivering on government's duty to supply safe drinking water under federal and state Safe Drinking Water Acts. The trust, reliability and service that community members and commerce require from elected officials and government agencies is supported when those entities fulfill their responsibility under these statutes and is eroded when contaminated water is consumed by the public and businesses, regardless of intentionality.

D. Does the action requested in the petition concern a matter currently in litigation? If yes, please explain.

No, to our knowledge, the action requested in the petition does not concern a matter currently in litigation.

E. For stream redesignation petitions, the following information must be included for the petition to be considered complete. Attach supporting material as necessary.

DRN's petition is not a petition for stream redesignation.

[117] Kauffman, G. (October 11, 2011). Socioeconomic Value of the Delaware River Basin in Delaware, New Jersey, New York, and Pennsylvania, p. 50. Enclosed as **Attachment 6**.