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ENVIRONMENTAL PROTECTION

WATER RESOURCE MANAGEMENT

DIVISION OF WATER SUPPLY AND GEOSCIENCE

Discharges of Petroleum and Other Hazardous Substances Rules; **Ground Water Quality Standards Rules; Private Well Testing Act Rules; Safe Drinking Water Act Rules; and New Jersey Pollutant Discharge Elimination System Rules**

Ground Water Quality Standards and Maximum Contaminant Levels (MCLs) for Perfluorooctanoic Acid (PFOA) and Perfluorooctanesulfonic Acid (PFOS)

Adopted Amendments: N.J.A.C. 7:1E Appendix A, 7:9C Appendix Table 1, 7:9E-2.1, 7:10-5.2, and 12.30; and 7:14A-4 Appendix A and 7.9

Proposed: April 1, 2019, at 51 N.J.R. 437(a).

Adopted: March 31, 2020, by Catherine R. McCabe, Commissioner, Department of Environmental Protection.

Filed: March 31, 2020, as R.2020 d.059, **with non-substantial changes** not requiring additional public comment or response, pursuant to N.J.A.C. 1:30-6.3.

Authority: N.J.S.A. 13:1B-3 et seq., 13:1D-1 et seq., 13:1D-125 through 133, 13:1E-1 et seq., 26:2C-1 et seq., 13:1K-1 et seq., 58:10-23.11, 58:10-46 through 50, 58:10A-1 et seq., 58:11-9.1 et seq., 58:11-23 et seq., 58:11-49 et seq., 58:11-64 et seq., 58:11A-1 et seq., 58:12A-1 et seq., and 58:12A-26 et seq.

DEP Docket Number: 02-19-03.

Effective Date: June 1, 2020.

Expiration Dates: April 4, 2021, N.J.A.C. 7:9C;

January 23, 2022, N.J.A.C. 7:9E;

March 29, 2024, N.J.A.C. 7:10; and

This rule adoption may be viewed or downloaded from the Department's website at http://www.nj.gov/dep/rules/adoptions.html.

The Department is adopting amendments to the New Jersey Safe Drinking Water Act (SDWA) rules at N.J.A.C. 7:10 to establish, as recommended by the New Jersey Drinking Water Quality Institute (Institute), a maximum contaminant level (MCL) for perfluorooctanoic acid (PFOA) of 0.014 micrograms per liter (µg/l) and an MCL for perfluorooctanesulfonic acid (PFOS) of 0.013 µg/l. PFOA and PFOS are part of a larger class of substances referred to as per- and polyfluoroalkyl substances (PFAS, previously referred to by the Institute as perfluorinated compounds, or PFCs), which have been detected in drinking water supplies in New Jersey and which, as explained further below, pose serious health threats to consumers. The Department previously established an MCL for another PFAS, perfluorononanoic acid (PFNA), on September 4, 2018 (see 50 N.J.R. 1939(a)). Currently, there are no Federal drinking water standards for these contaminants.

The MCLs apply to public community and public noncommunity water systems. Public community and public noncommunity water systems are required to routinely monitor for contaminants for which MCLs have been established and to treat water when there is an exceedance of an MCL. Public community water systems are water systems that have at least 15 service connections used by year-round residents, or regularly serve at least 25 year-round residents. Public noncommunity water systems include public nontransient noncommunity and public transient noncommunity water systems. Public nontransient noncommunity water systems do not serve year-round residents but do serve at least 25 of the same individuals for more than six months of any calendar year. Examples include schools or office parks that have their own water source.

Monitoring requirements for PFOA and PFOS for public community and public nontransient noncommunity water systems are being adopted and the existing rules at N.J.A.C. 7:10-5.2 will be recodified accordingly. In addition, the adopted amendments delineate the information regarding PFOA and PFOS that public community water systems must include in the annual consumer confidence report (CCR) describing the quality of the water delivered to customers.

Further, the Department is also adopting amendments to the Private Well Testing Act (PWTA) rules at N.J.A.C. 7:9E to require testing of private wells subject to sale or lease and to amend the SDWA rules to require testing of newly constructed wells for public noncommunity water systems and nonpublic water systems for PFNA, PFOA, and PFOS.

The Department is adopting amendments to the Ground Water Quality Standards (GWQS) at N.J.A.C. 7:9C to establish a specific ground water quality standard for PFOA of 0.014 µg/l and a specific ground water quality standard for PFOS of 0.013 µg/l. The Department previously established a specific ground water quality standard for PFNA on January 16, 2018 (see 50 N.J.R. 334(a)). Once adopted, the new ground water quality standards for PFOA and PFOS will also serve as the remediation standards for cleanup of contaminated ground water in accordance with N.J.A.C. 7:26D-2.2(a).

Further, and in accordance with the New Jersey Pollutant Discharge Elimination System (NJPDES) rules at N.J.A.C. 7:14A, the Department is adopting the addition of PFNA, PFOA, and PFOS to the Permit Application Testing Requirements/Pollutant Listings and the Requirements for Discharges to Ground Water.

PFOA and PFOS exist as acids and anions. However, because established testing and reporting requirements use the acid form of both contaminants, the adopted amendments to the GWQS, and the PWTA, SDWA, and NJPDES rules reference the acid form.

Lastly, the Department is adopting the addition of PFOA and PFOS to the List of Hazardous

Substances at N.J.A.C. 7:1E, Discharges of Petroleum and Other Hazardous Substances (DPHS) rules. The owners or operators of major facilities that store PFOA and PFOS may store these substances in multiple forms. Therefore, the listing will reference these forms, which include acids, anions, salts, and esters. In addition, the Department is adopting the addition of PFNA's anionic form, salts, and esters to the List of Hazardous Substances.

Summary of Hearing Officer's Recommendation and Agency's Response:

The Department held a public hearing on the notice of proposal on Wednesday, May 15, 2019, at 2:00 P.M., in the Department's Public Hearing Room, 401 East State Street, Trenton, New Jersey. Filina Poonolly, an Environmental Engineer for the Division of Water Supply and Geoscience, was the hearing officer. Ten persons commented at the public hearing. After considering the testimony at the public hearing and the written comments received, the hearing officer recommended that the Department adopt the amendments. The Department accepts the recommendation. A record of the public hearing is available for inspection in accordance with applicable law by contacting:

Department of Environmental Protection

Office of Legal Affairs

Attn: DEP Docket Number: 02-19-03

401 East State Street, 7th Floor

Mail Code 401-04L

PO Box 402

Trenton, New Jersey 08625-0402

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Summary of Public Comments and Agency Responses:

The following persons timely submitted comments on the notice of proposal:

1. Jill Arbuckle

2. Eric Benson, New Jersey Campaign Director, Clean Water Action

3. Richard Bizub, Director for Water Programs, Pinelands Preservation Alliance

4. Mary Brosius

5. Richard Burgstresser, Turpin Realtors

6. Kerry Butch, Rutgers University Center for Environmental Exposures and Disease

7. Richard Calbi Jr.

8. Raymond Cantor, Vice President Government Affairs, New Jersey Business Industry Association

9. Tracy Carluccio, Deputy Director, Delaware Riverkeeper Network

10. Christopher J. Connors, Senator, 9th District Legislative Offices

11. James Cosgrove, Vice President, Kleinfelder on Behalf of Sussex County Municipal Utilities Authority

12. Delaware River Keepers Network

13. John Demaio

14. Michael Egenton, Executive Vice President, New Jersey State Chamber of Commerce

15. Margaret Elis, Laboratory Manager, J.R. Henderson Labs, Inc.

16. Norm Farmer, SGS Orlando

17. Mark Feitelson, President, Precision Analytical Services, Inc.

18. Kathleen Fultz, Water Quality Association

19. Michael Furrey, Agra Environmental and Lab Services

20. Margaret Gallos, Association of Environmental Authorities

21. Joshua Greene, Vice President Government and Industry Affairs, A. O. Smith Corporation

22. Izhar Groner

23. Dennis Hart, Executive Director, Chemistry Council of New Jersey

24. John Hoertz, Acting Regional Environmental Coordinator, Department of Defense

25. Phyllis Howe

26. Karen Isky

27. Samantha Jones, Director of Regulatory Affairs, Site Remediation Industry Network

28. Dan Kennedy, Utility and Transportation Contractors Association of New Jersey

29. Harvey Klein, Laboratory Director, Garden State Laboratories

30. Christopher Len, Executive Director, Clean Water Advocacy Center, Inc.

31. Howard Levison, Township of South Orange Village

32. David Loveday, Government Affairs Director, Water Quality Association

33. Grant Lucking, Vice President of Environmental Affairs, New Jersey Builders Association

34. Rocco Mercuri, Gilmore & Associates, Inc.

35. New Jersey Realtors

36. Joseph Noonan, EWMA, Inc.

37. Kimberly Ong, Senior Attorney, Natural Resources Defense Council

38. Dennis Palmer, Executive Director/Chief Engineer, The Landis Sewerage Authority

39. Linnet Pereira

40. Kimber Ray, New Jersey Council of Watershed Associations

41. Steven Risotto, Senior Director, American Chemistry Council

42. Jeffery Sepesi, 3M

43. Bruce Shapiro, Deputy Director of Regulatory Affairs, New Jersey Realtors

44. Judy Shaw

45. Vikram Sikand

46. Fay M. Smith, BHHS Fox & Roach Realtors

47. Michelle Smith, Senior Project Scientist, Newfields

48. Eileen Snyder, Alpha Analytical, Inc.

49. Joseph Stanley, Chair, American Water Works Association New Jersey Section

50. Jeff Tittel, Director, New Jersey Sierra Club

51. James Votaw, Responsible Science Policy Coalition

52. Sam Weinstein, Princeton Public Affairs Group on Behalf of American Water Works Association New Jersey Section

53. Suzanne Wilder

The comments received and the Department's responses are summarized below. The number(s) in parentheses after each comment identify the respective commenter(s) listed above.

Comments in support of the amendments

1. COMMENT: The proposed amendments are supported. (1, 2, 3, 6, 9, 12, 13, 16, 18, 19, 20, 21, 22, 28, 29, 30, 32, 33, 36, 37, 39, 40, 45, 48, 49, 50, 52, and 53)

2. COMMENT: Given the high prevalence and wide distribution of these chemicals found in New Jersey and their known adverse health effects, there is concern about the presence of these highly toxic compounds in drinking water, the environment, and the risks they pose to the families and communities of New Jersey. Swift action by the Department is needed to adopt the proposed changes to regulations. (12)

RESPONSE TO COMMENTS 1 AND 2: The Department acknowledges the comments in support of the amended rules. The Department is charged with the protection of the environment and public health and continues to ensure that there is clean and safe drinking water for all of New Jersey's citizens. The Department agrees that promulgation of MCLs is an important part of protecting public health and, therefore, has prioritized this adoption accordingly.

3. COMMENT: There is some concern over the lack of "local" laboratories, turnaround time, and capacity for these parameters. There is no need for the laboratory to be located within the State of New Jersey. Certain laboratories are located in New Jersey and can be used as a drop off point for samples, they also provide a courier service that may be utilized for bottle kit delivery or sample pick up. They will then ship the samples to the appropriate laboratory for analysis. (16)

4. COMMENT: The laboratories in attendance at the stakeholder meeting were confident that they could provide the adequate capacity necessary for both the Private Well Testing Act Rule proposal, as well as the other rules. (48)

RESPONSE TO COMMENTS 3 AND 4: The Department agrees with the commenters. There are currently 19 laboratories certified to conduct the required testing, including two in New Jersey. Upon adoption, all

public community water systems and public nontransient noncommunity water systems will begin monitoring for PFOA and PFOS within the first quarter of 2021. Owners of private wells subject to sale or lease will be required to test for PFNA, PFOA, and PFOS starting 18 months after the amended rules are effective. The Department believes this will allow additional laboratories time to purchase equipment, train staff, and obtain certification in New Jersey, as necessary, and to coordinate with public water systems to ensure samples are collected and reported in accordance with proposed requirements, thus, avoiding monitoring violations. In addition, this will allow enough time to address the technical complexity of sampling and analysis for these parameters in accordance with the PWTA. The Department anticipates additional laboratories will become certified as this rulemaking is implemented.

5. COMMENT: We support the adoption of the proposed regulations regarding PFAS chemicals, specifically PFOA and PFOS and the additional proposals for PFNA. Given the high prevalence and wide distribution of these chemicals found in New Jersey and their known adverse health effects, we are deeply concerned about the presence of these highly toxic compounds in drinking water, the environment around us, and the risks they pose to families and communities. We support swift action by the Department to adopt the proposed changes to regulations and offer some suggestions for stricter standards.

We are concerned for our health, our family's and our community's health. PFOA and PFOS build up in the human body, are difficult to excrete, and even tiny concentrations in drinking water can have adverse health effects. Many of us have been drinking contaminated water for decades.

PFAS are linked to devastating disease and adverse health conditions. For PFOA these include Kidney Cancer, Testicular Cancer, Thyroid Disease, High Cholesterol, Pregnancy-Induced Hypertension/Preeclampsia, and Ulcerative Colitis. For PFOS these include decreased vaccine response and increased cholesterol, with toxicological effects in animals to the liver, immune system, endocrine, metabolic, and neurological systems. The developing fetus and young show several damaging developmental effects. (37)

6. COMMENT: We believe these new rules would be an important and overdue step to establishing stricter standards needed for PFOA and PFOS. This is critical for protecting our drinking water and groundwater. PFOA and PFOS are water soluble, and once they bioaccumulate in a body they never leave. (50)

7. COMMENT: We have been waiting for this change for nine years. Over that time the bioaccumulation of the chemicals in the environment has only magnified the health hazards. (50)

RESPONSE TO COMMENTS 5, 6, AND 7: The Department agrees that a public health-protective approach in addressing PFOA and PFOS in drinking water and ground water designated for potable supply use is warranted because these chemicals are extraordinarily persistent in the environment, bioaccumulate in humans, are slowly excreted with human half-lives of several years, and there is substantial evidence that even relatively low exposures increase the risk of multiple human health effects.

8. COMMENT: There is no time left to protect our health as the dangers are already present in our water supplies, communities, and have contaminated our bodies. In the face of widespread public concern about PFOS chemicals, contaminated sites around the State, occurrence in drinking water sources and mounting evidence of a wide array of health effects, we encourage the Department to continue using all available authority to address this issue. We need to know more about where PFOS chemicals are manufactured and are used, and how they get into our environment and how to clean them up. We need to stop PFOS discharges into the environment including into surface and groundwater that provide our drinking water. (2)

RESPONSE: The requirements set forth in the amendments to the Department's SDWA rules will reduce human exposure to this contaminant in drinking water by ensuring public community water systems and public nontransient noncommunity water systems consistently monitor the water to ensure compliance with the MCLs and treat to remove the contaminant(s) as necessary. The adopted specific ground water quality standards for PFOA and PFOS will ensure that current and scientifically based standards to protect, maintain, and restore ground water quality are in place. Permitted discharges to ground water and remediation of contaminated ground water will be required to achieve these health-based standards, which will reduce potential adverse impacts to public health and the environment from these contaminants in the ground water.

Testing requirements being adopted under the PWTA will help ensure that all buyers and sellers of real property are provided with information regarding the quality of onsite

potable well water in order to protect themselves from exposure to these contaminants, if detected. Similarly, landlords of properties where the source of potable water is a well subject to the PWTA will also be required to test for these contaminants and to advise tenants accordingly.

These requirements will also result in the collection of more data on the quality of water Statewide through required reporting of PWTA and remediation and remedial action permit sample results to the Department. The Department will utilize the data to ascertain ground water quality throughout the State and to provide information to counties, municipalities, other government entities, and the public. This will assist the Department and local health authorities in identifying areas of health concerns and directing resources to reduce or eliminate human exposure to drinking water contaminants in those areas.

Upon adoption, PFOA and PFOS will be listed as hazardous substances on the DPHS rules at N.J.A.C. 7:1E Appendix A. N.J.A.C. 7:1E Appendix A lists all substances that, in addition to petroleum and petroleum products, are considered hazardous substances under the Spill Compensation and Control Act (Spill Act), N.J.S.A. 58:10-23.11 et seq. The Spill Act provides strict liability for cleanup and removal costs resulting from any discharge of a hazardous substance.

In addition, the listing of PFOA and PFOS under the DPHS Appendix A List of Hazardous Substances will also require owners and operators of industrial establishments who are subject to the Industrial Site Recovery Act (ISRA), N.J.S.A. 13:1K-6 et seq., to remediate applicable sites prior to their sale or transfer or upon cessation of business operations. The inclusion of PFOA

and PFOS to the DPHS Appendix A List of Hazardous Substances will impose upon all responsible parties, regardless of the environmental statute they are liable under, the obligation to identify and remediate PFOA and PFOS discharges.

Compliance with the Safe Drinking Water Act and the Administrative Procedure Act

9. COMMENT: The Department appears to have inadequately addressed, or ignored, the significant costs that will be faced by public agencies, businesses, homeowners, and taxpayers if these proposals are enacted.

Specifically, the Department estimates that 207 public water systems in the State have levels of PFOA above the proposed MCL, and that PFOS levels in 97 systems exceed the proposed MCL. Assuming that there is some overlap between the two lists, the total number of potentially affected systems could exceed 200. By some estimates, total capital costs to comply with the proposed MCLs would exceed $260 million.

What's more, based on the Department's estimate of $80,000 per year, total operating costs would exceed $16 million annually. Not surprisingly, the Department's draft suggests MCL compliance costs "will be ultimately passed on to consumers" but makes no reference to the "limits of practicability and feasibility" standard prescribed in the SDWA. (14)

10. COMMENT: Since these capital and maintenance costs will ultimately be passed onto the customers of the water systems, it is imperative that the Department evaluates how the cost of compliance with the proposed MCLs will impact the households served by the systems. In addressing the costs for individual households, the United States Environmental Protection

Agency's (USEPA) National Drinking Water Advisory Council (NDWAC) recommends that a given drinking water standard be considered affordable if the annual cost per customer to meet the standard does not exceed 1.0 percent of the median household income for the median system in each drinking water system size category. Without estimating the increased cost to households served by the affected water systems, the Department cannot determine whether its proposed MCLs are affordable, and, thus, whether they can be considered practicable and feasible. (27)

11. COMMENT: As part of the proposed rule, the Department discusses the potential economic impact of the MCLs and other proposed testing and remediation requirements. However, the proposal's economic impact analysis is inadequate for several reasons. First, the agency does not provide a quantified estimate of the proposed rule's costs. Second, since the Department does not provide a cost estimate, it cannot demonstrate how these costs will be distributed to New Jersey households, businesses, public utilities, and local governments. Third, the proposed rule's discussion omits the adverse economic impact from delayed redevelopment and its attendant economic activity. (51)

12. COMMENT: If water rates rise proportionately to pay for these costs, lower income households will face a disproportionate burden. Households bear two types of costs from the regulation: (1) their water bills increase; and (2) since water is an essential component in many goods, prices of basic consumer goods are also likely to increase. Since lower income households spent a greater percentage of their income on water services and on basic

consumer goods, the Department's proposed regulation will have a disproportionately greater effect on lower income households. (51)

RESPONSE TO COMMENTS 9, 10, 11, AND 12: As stated in the notice of proposal's Economic Impact statement, the impacts of the amendments depend on various factors, including the number of water systems that install treatment, the type of treatment being implemented, site conditions, existing treatment, background quality of the source water, the size of the installation, and the concentration of the target contaminant in source water. According to Department records, the estimated cost of installing a GAC treatment system has ranged from $500,000 to $1 million for a one million-gallon-per-day (one MGD) treatment plant (serving about 10,000 people). Costs will be project specific, ranging from simply replacing filter media in existing GAC vessels to full treatment plant construction and upgrades. For example, systems that require a new treatment plant will incur higher costs for design, building and infrastructure construction, labor, and treatment components such as pumps, chemical storage and feed systems, monitoring instruments, and holding tanks. Costs associated with the operation and maintenance of a GAC system, which include periodic regeneration or replacement of the carbon, vary depending on such factors as the background quality of the source water, the size of the installation, and the concentration of the target contaminant in the source water. Operating costs are estimated to be approximately $80,000 per year for a one MGD plant but can increase depending on the number of wells requiring treatment and the level of contamination, as carbon filters will need to be replaced more frequently in case of higher levels.

To offset costs, the Department also offers low interest loans to eligible water systems through the New Jersey Water Bank, as treatment of emerging contaminants such as PFNA, PFOA, and PFOS is now a high priority for State funding. For example, the estimated average annual debt repayment for a typical publicly owned Drinking Water State Revolving Fund project (50 percent interest free and 50 percent at AAA market rate) with $1 million financed over 30 years would be $43,039.63. For a 1 MGD treatment plant serving 10,000 people, that would be $4.30 per person annually, if all debt repayment costs passed down to the customer. For a family of four, this would amount to $17.20 per year, or $1.43 per month. The true costs to customers will vary depending on factors such as system size and population served, existing treatment, water system rates and profits, availability and use of funding sources, and how the system ultimately determines costs that will be passed on to their customers. However, the Department does not believe that pass through costs to the customer would be significant on an individual basis.

As a result of this rulemaking, up to 506 public community water systems and 715 public nontransient noncommunity water systems will be required to monitor for PFOA and PFOS. The Department estimates that of these systems, 207 may have detections of PFOA and 97 systems may have detections of PFOS over their respective MCLs. If a public community or public nontransient noncommunity water system has PFOA and/or PFOS above the proposed MCLs, the system will be required to take action to reduce levels below the MCLs, which may include the utilization of an alternate water source or the installation of treatment.

The Department acknowledges that some costs may be passed on to consumers. However, these costs are necessitated by the statutory mandate at N.J.S.A. 58:12A-2 to ensure the provision of safe drinking water and to protect public health. The adopted amendments will reduce human exposure to these contaminants in drinking water and have a positive social and economic impact by protecting consumers from the health effects associated with PFOA and PFOS. Further, these amendments, which establish the information regarding these contaminants to be included in the CCR, will ensure that customers of public community water systems are informed on the quality of their water.

13. COMMENT: Job impacts should consider water utilities' need to hire more staff to monitor and maintain the new treatment units. Additionally, the tier requirement for the system water operator will increase as new treatment goes online at each utility. Achieving a higher license tier has experience and testing constraints that will have to be met by each water system operator. (7)

RESPONSE: The Department anticipates that the amendments will have a positive impact on jobs related to the design and installation of treatment systems once adopted. These amendments may create additional work for water systems based on additional testing requirements and operation and maintenance of new systems designed to treat for PFOA and PFOS.

The Department notes that the addition of a new treatment may not always require a utility to hire additional higher-level staff. Under the Licensing of Water Supply and

Wastewater Treatment System Operators rules at N.J.A.C. 7:10A, a public water treatment system is classified according to a range of points assigned based on the size, water supply source, and treatment in use at the system. Each new treatment adds additional points to the system classification that may or may not elevate the water system into a higher classification or "tier requirement" depending on the existing treatment processes already in place. In addition, N.J.A.C. 7:10A-1.10(b)1 states that if a system is reclassified by the Department, the existing licensed operator may continue to serve as the licensed operator of that system regardless of the new classification.

14. COMMENT: The information provided in the Economic Impact statement regarding remediation technologies for PFOA and PFOS has since been updated and we recommend the Department revise the information on point-of-use (POU) treatment and point-of-entry treatment (POET) technologies to reflect these updates.

In 2017, a National Sanitation Foundation (NSF) Standards task group was formed and charged with developing PFOA/PFOS treatment protocols for three technologies: activated carbon, reverse osmosis, and anion exchange. The scope of this charge was to formalize the NSF P473 protocol and officially ballot into NSF/American National Standards Institute (ANSI) Standard 53 and NSF/ANSI Standard 58.

In 2019, the protocols were adopted into NSF Standard 53 and NSF Standard 58 for carbon-based systems, as well as reverse osmosis systems; continued work is underway for anion exchange based systems. Now, products are being certified by ANSI-accredited

Certification Bodies to NSF/ANSI 53 and NSF/ANSI 58 for reduction of PFOA and PFOS, replacing the NSF P473 protocol.

Presently, there are 25 companies with numerous products currently certified at NSF, Water Quality Association, and International Association of Plumbing and Mechanical Officials for the reduction of PFOA/PFOS. We anticipate this to continue to grow in number, as well as scope of PFAS chemicals able to be claimed as additional research and information is developed within this family of products.

Finally, there is reference to small granular activated carbon (GAC) POET systems that remove PFAS in the water distributed throughout the house or building costing between $1,500 and $2,000 to install. It should be noted that POU treatment systems certified to remove PFAS at a single tap, such as for drinking or cooking cost approximately $30.00 to $300.00 depending on the device technology. (18, 21, and 32)

RESPONSE: The Department acknowledges the adoption of testing protocols for the reduction of PFOA and PFOS into the NSF/ANSI Standard 53 and NSF/ANSI Standard 58 in 2019. However, the Department notes that to comply with these standards and earn certification, devices are only required to reduce PFOA and PFOS to the USEPA Health Advisory of 0.07 µg/l. These devices are not certified to remove PFOA and PFOS to below the Department's MCLs for PFOA and PFOS of 0.014 µg/l and 0.013 µg/l, respectively. Under the PWTA, treatment is not required for private well owners and installation of these systems is at the discretion of the home or business owner. Prior to purchasing a POU treatment device, the Department recommends home or business owners confirm that the device is capable of reducing PFOA and PFOS to

below the Department's MCLs. Home and business owners should consult the NSF website and the manufacturer to confirm that the device is capable of meeting these recommendations (see https://www.nsf.org/consumer-resources/water-quality/drinking-water/perfluorooctanoic-acid-and-perfluorooctanesulfonic-acid-in-drinking-water).

15. COMMENT: Under the Department's proposals, even monitoring of community water systems for PFOA and PFOS will be costly. (14)

16. COMMENT: The $1,200 cost estimate underestimates the total cost to water systems to implement the sampling program. Considering the low part per trillion PQLs and the relative ubiquitous nature of perfluorinated like materials, for example, Teflon coated clothing, gloves, gaskets, packing material, etc., we ask that the Department reassess the estimated analytical costs and factor in costs of preparing sample collection procedures, training sampling staff, and other factors associated with sample collection. (31)

RESPONSE TO COMMENTS 15 AND 16: To determine an approximate cost of analysis for PFOA and PFOS, the Department estimated sampling costs per sampling point. The Department estimated that the cost of analysis (EPA Method 537) for the group of PFAS that includes both PFOA and PFOS was approximately $300.00 per sample, and that a public water system will spend approximately $1,200 in the first year for quarterly sampling at each point of entry. The Department further estimates that a public water system that monitors at a reduced monitoring frequency will spend as little as $300.00 per point of entry every three years.

The Department expects the cost for sample analysis to diminish with time after an MCL is adopted, as more laboratories are certified by the Department for analysis of these contaminants and as market competition increases. The analytical method commonly used to test for PFOA and PFOS, EPA Method 537, also detects PFNA. Thus, as systems are required to monitor for PFNA, the Department anticipates little to no additional cost to implement a sampling program to monitor for PFOA and PFOS.

The Department acknowledged these costs in its Economic Impact statement in the notice of proposal but determined that, because of the health effects associated with exposure to PFOA and PFOS, the costs were outweighed by the benefits provided by protection of public health.

17. COMMENT: The values listed for testing costs should be doubled. Each point of entry requires a field blank, which is tested for the same parameters and at the same cost. (7)

RESPONSE: Costs associated with analysis of a field blank will be site-specific and will depend on whether detections are determined above or below the minimum reporting level. If detections are above the minimum reporting level, the field blank is necessary to verify that contaminants have not been inadvertently introduced into the compliance sample. A field blank is a water sample prepared in the field that is exposed to the same environmental conditions as water sample used by the laboratory for compliance. A field blank ensures that contaminants were not inadvertently introduced into the compliance sample. Without the field blank analysis to

confirm the detection, the water system may take unnecessary actions, such as installing treatment.

18. COMMENT: The cost for treatment installation is woefully low. Ridgewood Water is installing a one MGD treatment plant and the total cost for planning, engineering and the awarded construction contract (not including financing costs) is $3.17 million. (7)

19. COMMENT: Estimated capital cost of installing a GAC system has ranged from $500,000 to $1 million for a one MGD plant. The estimated capital costs appear to represent the GAC process equipment only. We ask that the Department review the economic impacts to account for the range of system costs for situations where treatment facilities/stations must be expanded, or new buildings erected. In these situations, the total cost will include mechanical, structural, electrical, and architectural improvements in addition to GAC process improvements. (31)

RESPONSE TO COMMENTS 18 AND 19: As stated in the notice of proposal's Economic Impact statement, the cost of treatment, including costs for construction, operation, and maintenance, varies based on the type of treatment selected, site conditions, initial concentration of the contaminant, the presence of other contaminants and organic materials in the raw water, the need for pre-treatment, and the size of the water system. Although costs may be higher or lower depending on site-specific conditions and treatment chosen, these costs are necessitated by the statutory mandate at N.J.S.A. 58:12A-2 to ensure the provision of safe drinking water and to protect public health. In addition, the Institute has advised that, "GAC and/or an equally

efficient technology be considered for treatment of PFNA, PFOA and PFOS if they are detected above the [Institute's] recommended MCLs subject to the on-site pilot testing performance results." Pursuant to N.J.A.C. 7:10-5.7(a), a water system is required to take any action necessary to remove a contaminant when an MCL is exceeded. Thus, the Department does not specify a particular treatment process for the removal of PFOA and/or PFOS below the MCL.

The cost range provided in the Department's Economic Impact statement was estimated from data, including anticipated project costs and plant capacity, submitted to the Department by water systems in conjunction with applications for permits to install GAC. As of February 2020, 12 community water systems have submitted and received approval for the installation of GAC to treat for PFAS, including PFNA, PFOA, and PFOS. Estimated costs cited range from $31,350 to $16,067,300 for treatment capacities between 0.115 MGD and 21 MGD. Costs were project specific, ranging from simply replacing filter media in existing GAC vessels to full treatment plant construction and upgrades. Systems requiring a new treatment plant incurred higher costs for design, building and infrastructure construction, labor, and treatment components such as pumps, chemical storage and feed systems, monitoring instruments, and holding tanks. For example, in August 2012, New Jersey American Water-Penn's Grove submitted estimated costs of $7,780,000 for the construction of a new 2.2 MGD treatment building with GAC treatment, on-site chlorine generation, and three holding tanks. The number of GAC units needed was also site-specific, ranging from two to 24 vessels. In January 2018, Brick Township Municipal Utilities Authority was issued a permit to treat 16 MGD for PFAS with 12 pairs of GAC vessels in parallel at an estimated cost of $16,067,300. This cost included

additions and alterations to the existing treatment plant, such as concrete pads, enclosures, and piping modifications. Greenwich Township Water Department was issued a permit in January 2017, for additions and alterations to an existing 1.008 MGD capacity treatment plant comprised of two GAC vessels for the treatment of PFAS at an estimated cost of $614,257.

The Ridgewood system cited by the commenter submitted a permit in 2018 for treating water pulling from five wells, each with detections of both PFOA and PFOS above the limits. The permit involved the installation of GAC at a 1.44 MGD treatment facility. Estimated costs cited in the permit were $3,144,000, or $2,183,333 per MGD. While this value is higher than the range estimated, the Department acknowledges that costs may be higher or lower than the range estimated in the Economic Impact statement depending on site-specific conditions and treatment chosen.

20. COMMENT: The Department's rule proposal references several Treatment Subcommittee reports that provide recommendations on perfluorinated compound treatment options for drinking water (dated June 2015; August 2016; November 2017). These reports summarize existing water treatment technology collected from various research organizations, as well as information from various operating GAC systems.

It appears that the Department has not fully assessed the economic impacts of the proposed standards as they relate to treatment costs. Several of the references state that "samples taken after GAC treatment" were either "non-detectable" or "have remained below the recommended [MCL]." The Treatment Subcommittee fails to recognize that several

examples provided have raw water PFOS levels that are at or below the proposed MCL (NJAW – Logan System and NJAW – Penn's Grove). As such, while we agree that GAC is effective for removal of PFOS and PFOA, a comparison of treatment effectiveness for these systems seems inappropriate and skews the economic impact analysis for the proposed PFOS MCL. (42)

21. COMMENT: The Subcommittee fails to recognize that several of the treatment system operational costs are provided for treatment below higher drinking water guidance values (MDH – 0.3 µg/l; Department 0.04 µg/l). There are no costs provided or calculated to meet the proposed MCL. Given the traditional GAC isotherm it would be expected that operating costs would be higher to operate a system at the proposed MCL. (42)

RESPONSE TO COMMENTS 20 AND 21: As stated in the notice of proposal Summary, the role of the Institute's Treatment Subcommittee is to evaluate the best available and feasible treatment technologies for attaining removal of the contaminants from drinking water to achieve the health-based level, while considering the limits of available testing methodologies. The Treatment Subcommittee reviewed both relevant literature, as well as data from drinking water treatment plants, with full-scale treatment for long-chain PFAS, such as PFNA, PFOA, and PFOS, and concluded that the ability to remove these contaminants is not a limiting factor in setting an MCL. The Treatment Subcommittee also states that selection of the most cost-effective treatment option is site-specific and should be determined by careful design and bench and/or pilot testing studies and cost analysis.

The Department reviewed the Treatment Subcommittee reports and agrees with its conclusion. There are multiple full-scale facilities with varying influent and effluent

concentrations referenced in the Treatment Subcommittee reports that establish that it is both practical and feasible to treat for long-chain PFAS below the MCL of 0.013 µg/l that is being adopted for PFOS. For example, the raw water concentrations of PFOS at two sites in Oakdale, Minnesota were 0.540 µg/l and 0.620 µg/l, respectively. In addition, raw water concentrations of PFOS at Horsham, Pennsylvania were on average 0.629 µg/l. These sites were all able to achieve PFOS levels below 0.013 µg/l after GAC treatment. The Department determined that the annual operating cost of these two locations was estimated between $83,000 and $104,000 per site, respectively. These operational costs included cost of media replacement, insurance, sampling, personnel, and/or energy. As stated in the notice of proposal's Economic Impact statement, operating costs can increase based on the number of wells that require treatment and the level of contamination. Carbon filters will need to be replaced more frequently with higher levels of PFOA and PFOS.

22. COMMENT: While GAC is an effective removal technology for a wide range of drinking water constituents, a GAC treatment unit will be taxed to remove all the other stuff in the water at the part per thousand, part per million, part per billion, and finally part per trillion concentrations that are typically found in water matrices. GAC will remove all of the easily adsorbable constituents before removing the part per trillion PFOS and PFOA compounds. The constituent burden could result in premature GAC exhaustion and, thus, frequent replacement. The Department should review the MCLs in light of potential cost and operational challenges of using GAC as a treatment option. (31)

23. COMMENT: The $80,000 per year operating cost estimate for a one MGD plant could vary widely depending upon the source water matrix and constituent burden that GAC will be taxed to remove. We ask that the Department review the operating cost estimates and expand accordingly to account for the range of typical New Jersey water quality to be treated and adjust the potential GAC replacement frequency to meet the proposed MCLs. (31)

24. COMMENT: The Department's analysis of the costs associated with compliance obligations of its notice of proposal accurately points out that the costs associated with the changes to New Jersey's SDWA will be passed on to consumers in the case of public and nonpublic water systems. However, we recommend that the Department may want to review its supply and demand analysis of the reoccurring operations and maintenance (O&M) costs of acquiring GAC for the public water systems affected by the proposed SDWA amendments. It is our experience as a global manufacturer that procures activated carbon for its products, elevated demand levels will affect the market price of the underlying commodity, which may in turn elevate O&M costs for the entire marketplace. In addition, we would recommend that the Department further evaluate the impact of increased use of GAC treatment by public water systems and the environmental externalities associated with incineration and landfilling of PFAS. (21)

RESPONSE TO COMMENTS 22, 23, AND 24: Many manufacturers of GAC offer off-site carbon regeneration services, which include thermal reactivation of spent GAC. This process can result in desorption from the media and destruction of PFAS when done at appropriate temperatures. Reactivated GAC can be reused, with PFAS removal efficiencies similar to new, or virgin, GAC.

Regeneration of spent GAC through thermal reactivation can offer cost benefits when compared to media replacement with virgin GAC, reduce the costs associated with disposal of spent GAC through incineration or landfilling, and help prevent reintroduction of PFOA and PFOS into the environment. The cost of the regeneration process is assessed by the manufacturer as part of the cost associated with supply of carbon.

As stated in the notice of proposal Economic Impact statement, costs associated with the operation and maintenance of a GAC system, which include periodic regeneration or replacement of the carbon, will vary depending on site specific factors, such as the background quality of the source water, the size of the installation, market variations in the price of GAC, and the concentration of the target contaminant in the source water.

25. COMMENT: The current universe of systems implementing PFOA/PFOS treatment is limited to a handful of states and, thus, the small number of impacted systems may be insufficient to move the treatment technology equipment manufacturing community to invest in developing and commercializing new technologies that will treat and remove PFOA/PFOS at lower costs than today. We ask that the Department revise this section for the current status of regulatory review and technology adoption and rationalize the new technology development projections accordingly. If the Department is aware of new technologies in the technical readiness/commercial trials stage, we ask the Department to communicate these new technology developments. If would be helpful if the Department incorporated other effective

technologies, such as reverse osmosis into the capital and operating cost assessments section to provide a fuller range of potential costs impacts. (31)

26. COMMENT: The Department assumes, without adequate supporting evidence, little or no additional costs for treating PFOA and/or PFOS treated by granular activated carbon and provides no documentation to support its separate claim that treatment costs will decrease over time. (14)

27. COMMENT: The Department provides no estimate for how many water systems have already installed, or are installing, GAC systems for the treatment of PFNA and will, therefore, incur "little to no additional cost for the treatment of PFOA and/or PFOS." In fact, the available evidence suggests that the number of systems incurring little to no cost would be small since PFOA and PFOS were not found at the four public water systems where PFNA was reported in the USEPA's Unregulated Contaminant Monitoring Rule (UCMR) Occurrence Database. The Department also provides no evidence to support its contention that "the costs of treatment are likely to decrease over time." (27)

RESPONSE TO COMMENT 25, 26, AND 27: As stated in the notice of proposal Economic Impact statement, the Department anticipates costs of treatment to decrease over time as treatment technologies develop and become more readily available. The Institute's Treatment Subcommittee reviewed relevant literature and data from drinking water treatment plants, including facilities in New Jersey, with full-scale treatment for long-chain PFAS, including PFNA, PFOA, and PFOS. The Subcommittee also reviewed technologies including powder-activated carbon, membrane filtration, advanced oxidation, and anion exchange. However, the

Subcommittee's review did not identify any drinking water facilities treating for long-chain PFAS using non-GAC treatment technologies, such as reverse osmosis; thus, limited information, including cost, was available for these technologies. That does not mean that these technologies are not available to water systems as treatment options. The Department recognizes that new information relevant to PFOA and PFOS treatment technologies, such as anion exchange, continues to become available and that systems should choose treatment based on site-specific factors.

Although there are currently a limited number of states with standards, several states are in the process of proposing, recommending, or drafting standards. For example, in December 2018, the New York Drinking Water Quality Council recommended to the New York State Health Commissioner MCLs of 0.010 µg/l for PFOA and PFOS, which are currently going through an approval process. In March 2020, Michigan adopted health-based values for seven PFAS, including 0.008 µg/l for PFOA and 0.016 µg/l for PFOS. As of February 2020, 10 states have set PFOA and PFOS guidelines or standards below the USEPA Health Advisory of 70 ppt (0.070 µg/l).

In the first quarter of 2019, public community water systems in New Jersey that use ground water as a source and serve a population of 10,000 or less, and all public nontransient noncommunity water systems in New Jersey began the required monitoring for PFNA and completed their first year of quarterly monitoring in December 2019. As of February 10, 2020, 1,107 water systems submitted PFNA monitoring data to the Department. Eleven of those systems received MCL violations. These public water systems have one year to take action,

which may include installation of treatment, to bring the water into compliance with the applicable MCL. If these systems also have exceedances of the PFOA and PFOS MCLs, they may incur little to no additional cost for the treatment of PFOA and/or PFOS, if a technology that can treat for these compounds is used.

28. COMMENT: As with the consideration of the affordability of the proposed MCLs, the Department must consider the potential cost impacts on residents for compliance with the groundwater standards. Many of the active remediation sites, and sites that will be subsequently identified, are owned by municipalities who will be required to bear the cost of compliance with the standards. They will, in turn, be required to pass those higher costs onto residents through higher local taxes or fees. Given the diverse and diffuse nature of the historic use of PFOA and PFOS, it often may not be possible to identify a responsible party. (27)

RESPONSE: In proposing these amendments, the Department did consider the potential cost impacts on residents for compliance with the ground water quality standards.

Costs for remediation will vary based on site specific circumstances. The Department estimates that the costs of installing a GAC pump and treatment system for ground water remediation will be similar to treatment costs for water systems. As stated above, costs associated with the operation and maintenance of a GAC system, which include periodic regeneration or replacement of the carbon, vary depending on such factors as contaminant loading, the background quality of the source water, the size of the installation, and the concentration of the target contaminant in the source water.

While it is true that it can be difficult to identify responsible parties, several tools can be used to offset costs. First, the addition of PFOA and PFOS to the DPHS Appendix A List of Hazardous Substances will, in accordance with the Spill Act, enable an eligible public water system or person who has incurred costs because of a PFOA or PFOS discharge to seek reimbursement for, among other things, the cost of remediating the PFOA or PFOS contamination, provided the person is not the responsible party. Listing PFOA and PFOS will also enable the Department to require the discharger, or a person in any way responsible, to remediate discharges of these substances. Second, hazardous substance-based funding sources are available, and when determined necessary, the Department may use those sources to conduct remediation should the responsible party refuse to do so, is not financially viable, or is unknown. Additionally, the Department may undertake cost recovery and damages actions against the responsible party or parties, including manufacturers of PFOA and/or PFOS and/or products containing one or both substances.

29. COMMENT: The Economic Impact Statement regarding costs for GAC treatment on wastewater treatment plants being similar to potable water treatment systems is questionable. (20 and 38)

RESPONSE: The Department notes that USEPA determined that wastewater constituents, such as biochemical oxygen demand (BOD), organics, and total suspended solids (TSS), can adversely affect GAC treatment, but TSS is the constituent of most concern for a GAC system (see https://www3.epa.gov/npdes/pubs/carbon_absorption.pdf). In order to comply with the

requirements of the NJPDES discharge to ground water permit, tertiary treatment is typically provided. This level of treatment generally removes the contaminants that would adversely affect the GAC system. While there are no promulgated ground water quality standards for TSS and BOD, the tertiary treatment typically provided by NJPDES-regulated facilities should result in TSS and BOD values similar to potable water.

30. COMMENT: The Department did not fully evaluate the economic impact on wastewater treatment plants to treat for PFOA/PFAS. Most water treatment plants can easily change out sand filtration for activated carbon and most wastewater treatment plants do not have an existing filtration process to change out. Also, most wastewater treatment plants would not have the hydraulic profile that would allow for gravity flow into the GAC system. Finally, space is at a premium at many wastewater treatment plants, so having room for an additional building at the end of the treatment process is certainly not a given. (11)

RESPONSE: The Department based its assessment of costs of treatment at wastewater treatment facilities on the cost of treatment per gallon of water processed. The Department did not calculate the cost to treat for PFNA, PFOA, and PFOS for each individual potable water or wastewater treatment facility, because individual costs are site-specific. Potable water and wastewater treatment plants have similar considerations and engineering challenges.

31. COMMENT: The Department failed to evaluate the economic impact to solid waste stormwater basins. It is not clear as to the Department's intentions in implementing the proposed regulations and applicability thereof to stormwater basins. (11)

RESPONSE: Municipal solid waste facilities are one of the targeted discharge to ground water facilities that will require sampling for PFNA, PFOA and PFOS, and the economic impact with respect to stormwater basins should be minimal. At a solid waste facility, such as a landfill, there are two potential discharge to groundwater regulated units: basins sited within the footprint of a liner that receive stormwater that may contact source material over the lined portion of the site, but do not discharge into ground water due to the liner; and basins sited outside the liner that receive stormwater from the remainder of the site that has not contacted source material. In either case, the cost of future compliance inclusive of these amendments will not substantially change since these solid waste facilities' designs minimize or eliminate pathways by which PFNA, PFOA, and/or PFOS are discharged to ground water.

32. COMMENT: Requiring treatment for otherwise uncontaminated stormwater would require significant cost that was not considered in the supporting evaluation for the proposed rules. The need for treatment is best determined under the Department's Site Remediation and Waste Management Program rather that the New Jersey Pollution Discharge Elimination System – Discharge to Ground Water (NJPDES-DGW) permitting program. (23)

RESPONSE: In general, a NJPDES-DGW permit will not contain requirements for treatment for stormwater that does not contact source material that may contain PFNA, PFOA, and/or PFOS.

Facilities with discharges of stormwater to ground water will be required to monitor for PFAS where these compounds are reasonably expected to be present in the facility's discharge. If PFNA, PFOA, and/or PFOS are detected in the discharge above the ground water quality standards, the facility will be required to attempt to track down the source of the PFAS and remove it from the waste stream before treatment would be required. This can be achieved by physically locating and removing contaminated material from the site, implementation of stormwater best management practices (BMPs), and/or implementing drainage control measures to direct stormwater away from affected areas until those sources can be removed. If it is determined the source is from off-site, the facility may need to update its drainage control plan to eliminate run-on from other properties or take other measures. For further discussion, see Response to Comment 225.

33. COMMENT: The Environmental Impact statement did not address the tons of spent GAC that will be saturated with PFOA/PFOS and where it will go. (38)

RESPONSE: As stated above in Response to Comments 22, 23, and 24, many manufacturers of GAC offer off-site carbon regeneration services, which include thermal reactivation of spent GAC. This process results in desorption from the media and destruction of PFAS when done at appropriate temperatures. Reactivated GAC can be reused, with PFAS removal efficiencies similar to new, or virgin, GAC. Regeneration of spent GAC through thermal reactivation greatly reduces the need for disposal of spent GAC through incineration or landfilling, helps prevent

reintroduction of PFOA and PFOS into the environment, and can offer cost benefits when compared to media replacement with virgin GAC.

Compliance with Executive Order Nos. 2 (2010) and 63 (2019)

34. COMMENT: Investment in a proactive public outreach campaign is a necessary component of the implementation process for the new MCLs. Providing means for the public to understand the acute and long-term impacts of drinking water from sources with PFOS and PFOA levels above the proposed MCLs is a necessary component of this process. Providing public water systems with this information as they work with the public to define appropriate measures for interim protection of public health while treatment system upgrades are pending is of utmost importance. (49)

RESPONSE: The Department increases awareness through education and outreach efforts. However, in the Department's experience, outreach efforts alone are not enough to adequately inform residents of the condition of their drinking water and are, therefore, not sufficiently protective of public health. As part of this rulemaking, the Department is amending the health effects information for systems to include in their annual CCRs to include specific language for PFOA and PFOS. All water systems are required to provide information regarding regulated contaminants and those unregulated contaminants that are sampled for pursuant to the Federal Unregulated Contaminant Monitoring Rule in their CCRs. CCRs must be provided to consumers annually and made publicly available. In addition, the Department provides

information regarding regulated contaminants at all public water systems through Drinking Water Watch (see https://www9.state.nj.us/DEP_WaterWatch_public/NJMap.jsp).

35. COMMENT: Executive Order No. 63 (2019) states "where [F]ederal regulation is inadequate to protect the environment, health, safety, and welfare of New Jersey's residents and communities, New Jersey should develop its own regulatory framework where it has the legal authority to do so," but "where [F]ederal regulation adequately protects the environment, health, safety, and welfare of New Jersey's residents and communities, New Jersey should operate under that framework in order to minimize confusion and complexity." On this count, New Jersey's efforts also fail. While there may be disagreement over the pace of USEPA's regulation of PFOA and PFOS, USEPA action is not non-existent. The current drinking water health advisories (DWHAs) provide a national reference point, as USEPA's recent recommendations for groundwater remediation based on the DWHAs illustrate. Further, the USEPA's semi-annual regulatory agenda shows that USEPA is on pace with meeting the requirements of the Federal SDWA and will make an initial regulatory determination on setting MCLs for PFOA and PFOS by the end of the year. (42)

36. COMMENT: We are concerned, that New Jersey is not unique in its need for more stringent PFOA and PFOS drinking water standards and we question the difference in scientifically derived drinking water standards from what is being proposed and what remains in place at the Federal level. (8)

RESPONSE TO COMMENTS 35 AND 36:

As explained in the Department's Federal Standards Statement in the notice of proposal, the Department's Safe Drinking Water Act (SDWA) rules at N.J.A.C. 7:10 incorporate by reference the National Primary Drinking Water Regulations (National Regulations) at 40 CFR 141. The Department's SDWA rules are, therefore, the Federal standards, except where the Department has determined, as authorized by the Federal SDWA and allowed by the National Regulations, to establish New Jersey-specific requirements. Pursuant to the SDWA, the Department is authorized to promulgate MCLs after considering the recommendation of the Institute, if there are adverse health effects associated with the contaminant and the contaminant may be found in public water supplies in New Jersey. There is substantial evidence that even relatively low exposures to PFOA and PFOS in drinking water increase the risk of multiple serious human health effects. Both PFOA and PFOS were detected in public water systems in New Jersey through sampling conducted during the third iteration of the Federal Unregulated Contaminant Monitoring Rule (UCMR3) and Department-initiated sampling. Currently, there are no Federal drinking water standards for these contaminants.

37. COMMENT: The Department's failure to conduct a complete quantitative cost analysis or economic impact assessment is striking due to a recent New Jersey Executive Order on regulation. Under Executive Order No. 63 (EO 63), signed April 2, 2019, the Department is required to compare the proposed benefit to the public with the anticipated burden to the public. In addition, Executive Order No. 63 states that agencies should consider the

distributional impact of a regulation on "various subsets of the population and economy." Particularly relevant for an analysis of the distributional impact of a drinking water regulation is the effect on low-income households or small businesses.

Since safe water is an essential good for life and for public health, lower income households spend proportionally more of their household income on basic goods like water, sewage, and energy than more affluent households. The proposed regulation will necessarily have a disproportionately higher impact on lower income households that, in turn, are disproportionately comprised of certain racial and social groups. Since Executive Order No. 63 requires the Department to give "due consideration" to "Environmental Justice" and "address … disproportionately high and adverse human health or environmental effects of the program, policy, or activity on minority and low-income populations," the agency should carefully consider the distributional impacts of its proposed regulation. But the Department's proffered comparison of benefits and costs is cursory and incomplete in the proposed rule. As a result, the public cannot identify or evaluate the distributional impacts and the magnitude of the disproportionately high impact of the Department's regulation on low-income populations. (51)

38. COMMENT: Governor Murphy recently signed Executive Order No. 63, "Establishing new regulatory principles to foster economic growth and government efficiency." Compared to these goals, the Department's proposed rules are deficient. The exceedingly low proposed MCLs are ill-considered and not well-framed. They are not "informed" because the proposal ignores sound science in its MCL study selection and derivation. These values are no more protective in any real sense than the USEPA's current Drinking Water Health Advisory of 70

parts per trillion (ppt) because both sets of values carry large margins of safety built into them. Neither are a bright line between safety and harm. They are, however, burdensome on business, local governments, and water utilities. Nothing in the Department's rulemaking offers any reasoned determination that the benefits of the proposed MCLs justify its costs. In fact, other than limited cost considerations, such as monitoring costs by water utilities, the Department by and large ignores the larger costs to the whole of New Jersey's economy and citizens. (42)

RESPONSE TO COMMENTS 37 AND 38: As stated in the notice of proposal's Economic Impact statement, costs incurred to comply with the SDWA rules are standard business expenses for public water systems. The costs incurred as a result of the proposed amendments will be ultimately passed on to consumers and are necessitated by the statutory mandate at N.J.S.A. 58:12A-2 to ensure the provision of safe drinking water and to protect public health. The prevention of the known negative effects on human health will create eventual savings in avoided medical costs and avoided losses to productivity associated with illness.

39. COMMENT: The application of limits and sampling of sewage treatment plants are not consistent with EO 63. Specifically, "[g]overnmental decisions should be based upon the best available data, including scientific data, if applicable. Where science evidence is an important element in developing or evaluating a rule, State entities should seek out and make productive use of scientific expertise available to them." There are no data, none, addressing wastewater sampling, analysis, and treatment. (38)

RESPONSE: In developing this rulemaking, the Department sought out all data available at the time to guide the Department's rulemaking. This survey of best available information at the time included analysis of data derived by the Site Remediation and Waste Management Program that demonstrates both the presence of these contaminants at concerning levels in groundwater, as well as the viability of treatment for these parameters. In addition, the Department's Division of Science and Research has performed studies on the presence of these parameters in wastewater. National data surveyed by the Department also supported the Department's conclusions that these contaminants exist in wastewater and in some wastewater discharges. As discussed above in the Response to Comment 30, the Department based its assessment of costs of treatment at wastewater treatment facilities on the cost of treatment per gallon of water processed. The Department did not calculate the cost to treat for PFNA, PFOA, and PFOS for each individual potable water or wastewater treatment facility because individual costs are site-specific.

As discussed in the Response to Comments 219, 220, 221, and 222, the Department's Office of Quality Assurance certifies laboratories for user-defined, or laboratory developed, analytical methods that are applicable to non-potable water. The OQA ensures that these user-defined methods include rigorous quality control measures, including those that provide a measure of the method's suitability to a given matrix.

40. COMMENT: We are disappointed the Department stakeholder process did not provide an opportunity for meaningful public participation by the wastewater industry and did not

better align with EO 63. The principles of an open and effective rulemaking process are well known, and the Department has previously utilized the stakeholder process in an open and effective manner. Even absent an Executive Order, we would have expected the Department to engage with affected communities, provide opportunities to work in partnership with affected communities, and gather information through community meetings.

With respect to this rulemaking, the Department held stakeholder meetings, but the focus was on the potable water and environmental communities. The wastewater community did not fully appreciate the impact on their NJPDES permits until very recently. In essence, the rulemaking sets new effluent limits for wastewater treatment plants that discharge to groundwater. We do not believe that is understood by the wastewater community, even today.

A meaningful stakeholder process with the wastewater industry was not undertaken, and the wastewater industry was unable to provide meaningful input prior to publication of this proposal. (11)

RESPONSE: The Department included a wide range of stakeholders who participated in a series of discussions as part of this rulemaking initiative. In addition to several informal stakeholder meetings convened to discuss potential amendments to the GWQS held between 2016 and 2019, a formal stakeholder meeting was convened by the Department on January 18, 2019, specifically to discuss ground water quality standards for PFOA and PFOS and the addition of PFNA, PFOA, and PFOS to the Permit Application Testing Requirements/Pollutant Listings and the Requirements for Discharges to Ground Water in the NJPDES rules. Information regarding

these meetings is available on the Department's website at https://www.nj.gov/dep/workgroups/gwqs.html.

Stakeholders included facilities likely to be affected by this rulemaking, as well as other interested parties including existing NJPDES-Discharge to Ground Water (DGW) permittees. A public hearing on the rulemaking was also held on May 15, 2019. Notification of the rulemaking was also provided through email to NJPDES stakeholders and through the Department's Listserv to all entities who subscribe to receive updates on the GWQS (see https://www.nj.gov/dep/wms/listservs.html).

Maximum Contaminant Levels for PFOA and PFOS

Development of the MCLs for PFOA and PFOS

41. COMMENT: The Department failed to consider information for the proposed MCLs that was submitted in response to the Institute summary documents that are the basis of this rulemaking (27)

42. COMMENT: Upon reviewing the health effect documents prepared by Institute's Health Effects Subcommittee, we respectfully disagree with the proposed MCLs, as well as its conclusions regarding the human health effects associated with exposure to PFOS and PFOA. Key scientific evidence was not fully considered by the Institute's Health Effects Subcommittee or the Department, which led to incorrect scientific assumptions resulting in underestimations of the proposed MCLs. (42)

43. COMMENT: The proposed MCLs are overly conservative, technically flawed, do not reflect recently published studies and provide no additional protection compared to the USEPA's current drinking water health advisories. They are merely lower. (42)

44. COMMENT: The MCL proposal ignores the best available scientific evidence and arbitrarily selects studies and toxicity endpoints to drive to lower MCLs. This was done without applying scientific rigor or assessing the reliability of testing for such low values. (42)

45. COMMENT: The Department should reanalyze its health assessment for PFOA and PFOS to consider new, high-quality scientific studies and to follow best practices for health assessments. Due to the substantial economic impact of the proposed regulation, the Department should ensure its health assessment is based on the best available science. (51)

46. COMMENT: In the proposed rule, the Department relies on the 2017 findings of the Institute as the basis of the estimated human health risk from PFOA and PFOS. There have been a substantial number of high-quality scientific studies published since the Institute's findings. These studies call into question the Institute's findings of the likelihood and magnitude of potential adverse effects. (51)

RESPONSE TO COMMENTS 41 THROUGH 46: The MCLs were developed based on recommendations from the Institute, which reviewed the most current science prior to finalizing a recommendation. The Department considered the Institute's recommendations and performed additional research, as necessary, to determine whether new information is available. Specifically, the MCLs are based on a thorough evaluation and documentation of relevant epidemiological, toxicological, and mode of action studies.

The USEPA's Health Advisories are not sufficiently protective because the USEPA did not consider more sensitive toxicological endpoints that are well-established, adverse, and relevant to humans. Further, exposure to drinking water at the level of the USEPA Health Advisories will result in levels of PFOA or PFOS in blood serum that are well above the range associated with multiple human health effects. The Michigan PFAS Science Advisory Panel also concluded that "[i]f one accepts the probable links between PFOA exposure and adverse health effects detected in the epidemiological literature as critical effects for health risk assessment, then 70 ppt [0.070 µg/l] in drinking water might not be sufficiently protective for PFOA" (Michigan PFAS Science Advisory Panel, 2018).

As discussed further in other responses, the Department also considered the more recent studies mentioned in the public comments submitted on the proposed MCLs.

47. COMMENT: The Department's basis for the proposed MCLs regarding the toxicity and potential for exposure to these substances is inappropriate and overly conservative. (27)

48. COMMENT: The Department should avoid the layering of very conservative parameter choices to derive an MCLs because this is NOT the same as being protective in a true and sound public health protection sense. A lower MCL value will not be any more protective than a higher MCL if the latter value already provides an adequate margin of safety. Unnecessarily low values do not provide more public health protection, but will impose unwarranted costs on the public and instill unnecessary fear and anxiety in communities. (42)

RESPONSE TO COMMENTS 47 AND 48: The Department believes that the proposed MCLs have a sound scientific basis and does not agree that their basis is inappropriate and overly conservative regarding toxicity and exposure potential. Specific issues related to toxicity and exposure potential are addressed in other responses to comments. The Institute and the Department considered all information that was submitted related to development of MCLs for PFOA and PFOS, both in the request for information prior to development of the Institute's recommendations in the public comment period on the draft PFOA and PFOS MCL recommendation documents.

The Department disagrees that monitoring and treatment of drinking water will cause the public to fear consumption of the water supply. As explained in the Social and Economic Impact statements in the notice of proposal, public water systems routinely make decision regarding the operation of their systems in order to deliver safe drinking water to consumers. Rather, the Department believes that failure to regulate these contaminants will have the negative social impact of eroding consumer confidence in drinking water.

49. COMMENT: The weight of scientific evidence does not support either MCL proposed by the Department. In addition to equating presence in the environment with harm, the Department treats PFOS and PFOA's long serum half-lives and tendency to accumulate in the blood at low exposures as synonymous with increased health risk and higher toxicity. The body of credible science does not support such a conclusion. (42)

RESPONSE: The adopted MCLs for PFOA and PFOS are not based on equating presence in the environment with harm, and they are not based on an assumption that health effects and toxicity necessarily result from a compound's accumulation in blood. The MCLs are based on toxicological effects in laboratory animals, with supporting evidence from associations of PFOA and PFOS with health effects in humans. Blood serum PFOA and PFOS levels, which reflect internal dose, are used as the dose metric in development of the MCLs.

50. COMMENT: We recommend that the Department conduct an updated review of the scientific literature according to the best practices recommended by the National Academy of Sciences (NAS) and use its findings to develop new proposed MCLs.

These best practices apply to the USEPA and to any agency seeking to provide hazard information to the public. They are the basis of sound public policy decisions. The Department did not follow these best practices. There was no systematic review, no independent peer review, and no transparent evidence integration. As it reconsiders the Institute assessment based on the newer, high-quality scientific studies, the Department also should follow the NAS recommendations for conducting hazard assessments. (51)

51. RESPONSE: The Institute's literature search was comprehensive and is described in the DWQI (2017) and DWQI (2018).

The Department's MCLs for PFOA and PFOS were developed in accordance with the SDWA. Specifically, the Institute is established by the SDWA, at N.J.S.A. 58:12A-20, and has a statutorily specified role as an advisory body to evaluate scientific information and make

recommendations to the Commissioner of the Department for the implementation of the Department's drinking water quality program, including MCLs. While the MCLs recommended by the Institute did not undergo additional peer review, the majority of the Institute membership consists of scientific and technical experts from outside of New Jersey government agencies, and there are multiple opportunities for public input during the MCL development process. The PFOA and PFOS Health-based MCL Support Documents thoroughly discuss the decision process for selection of the critical toxicology studies and endpoints used as the basis for the MCLs, including supporting information from other toxicology studies, as well as epidemiology and mode of action studies.

51. (**OAL Note:** Inadvertently, this number was not used and is intentionally left blank upon publication.)

52. COMMENT: The MCLs for PFOA and PFOS should be set at ≤ 1.0 ng/l based on immunotoxicity endpoints. Strong, significant epidemiologic evidence that include quantitative data for immune suppression is available to derive PFOA and PFOS lower confidence limits on benchmark dose (BMDLs). The Grandjean and Budtz-Jorgensen study represents the greatest sensitivity to PFOA thus studied, un-confounded by exposure to other chemical contaminants (Grandjean and Budtz-Jorgensen 2013). That study found a strong association between serum PFOA and PFOS concentrations and serum antibody concentrations against tetanus and diphtheria toxoids. Regression modeling of PFOA and PFOS as independent variables along with

potential confounders of sex, age, and booster type at age five and seven, with antibody concentrations as outcome, determined a benchmark dose (BMD) and response. (9)

RESPONSE: The Department generally supports the use of epidemiologic studies in quantitative risk assessment. However, due to the observational nature of human epidemiology, there is a high bar for its use as the quantitative basis for risk assessment. There is evidence for association of PFOA and PFOS with decreased vaccine response in humans, and this evidence is particularly strong for PFOS. However, the Department maintains that the epidemiologic database is insufficient at this time to support the use of this endpoint as the basis for quantitative risk assessment of PFOA and PFOS. In particular, the strong correlation between PFOS and PFOA limited the researchers' ability to mutually adjust for both, thereby preventing inference in regard to causal attribution to a specific compound. It remains unclear that the effects of PFOS and PFOA can be separated from each other or from the effects of other PFAS. Although the database for antibody response following vaccination is currently not conclusive enough to use as the primary basis for quantitative risk assessment, it clearly supports the need for a protective approach in the risk assessment based on animal data. If future studies provide additional support for a relationship between PFOS and decreased response to vaccinations, including appropriate dose-response data, then this endpoint could be reconsidered for use as the basis for quantitative risk assessment.

53. COMMENT: We believe that, based on the most up-to-date scientific and health-based research, the standards for PFOA and PFOS should each be five ppt. (50)

RESPONSE: The Institute makes MCL recommendations to the Department based on the health effects of the targeted contaminant, as well as the certified laboratories' ability to test for the contaminant, and the availability of treatment removal technologies. For PFOA and PFOS, the recommended MCLS are the health-based levels, which were developed using accepted methods of risk assessment and current scientific data. As stated by the Institute (DWQI), the health-based levels are based on lifetime exposure and are expected to be protective of all age groups.

54. COMMENT: Rather than develop separate MCLs for PFOA and PFOS, New Jersey should develop a combined MCL for PFOA and PFOS. These structurally similar contaminants likely have additive and synergistic effects on human health. It is the combined level of PFOA and PFOS in our bodies that is relevant for human health, rather than the level of each contaminant individually. A combined MCL of 13 ppt for these two contaminants would be an improvement over the proposed regulations, which evaluate the concentrations of PFOA and PFOS separately. (37)

55. COMMENT: We need a combined standard for both PFOA and PFOS of 13 ppt because this is a persistent and pervasive chemical that directly affects human health. (50)

RESPONSE TO COMMENTS 54 and 55: The potential for additive toxicity of PFOA and PFOS is acknowledged in DWQI (2017a) and DWQI (2018a). However, the toxicological effects and mode of action of PFOS differ in some respects from PFOA. Additionally, because the dose-response for some health effects is steepest at low exposures and approaches a plateau at

higher exposures, dose-response for mixtures may be complex and dose-dependent. Although cumulative effects were not considered in developing the Health-based MCL, an important benefit of addressing exceedances of the Health-based MCL is that treatment removal processes intended to remove PFOA and/or PFOS may also partially or totally remove other types of PFAS, and other unrelated contaminants that may be present at levels of public health concern (see Post et al., 2017).

56. COMMENT: The Department should not minimize primate toxicity data. When it comes to human relevance and risk assessment, given the many issues in extrapolating toxicology data from rodents (a lower order species) to humans (the highest order), primate data have always valued as the most scientifically appropriate species for human risk assessment because it is the second-highest order species next to humans. (42)

RESPONSE: The available primate studies of PFOA and PFOS were reviewed in detail by DWQI (2017a) and DWQI (2018a), with the exception of Chang et al. (2017), which was published after the Institute's PFOS literature review was conducted. The Institute noted that one of the primate studies related to PFOA (Butenhoff et al., 2002) had problematic issues, including toxicity that prevented a substantial percentage of the dosed monkeys from completing the study, and the occurrence of mortality possibly attributed to PFOA. Additionally, serum PFOA were highly variable between animals and over time in the same animal, and they did not increase proportionally with dose. In general, the primate studies of PFOA and PFOS, including Chang et al. (2017), did not evaluate the most sensitive effects of PFOA and PFOS, such as

developmental effects or immune system function. Consequently, the studies were not used as part of the basis for the quantitative risk assessments for PFOA or PFOS. The quantitative risk assessments are based on studies of effects that are sensitive, well-established, and considered relevant to humans.

57. COMMENT: Many epidemiological studies regarding PFOS or PFOA are cross-sectional by design. This type of study design cannot address temporality (that is, time-dependent associations). This issue is important to acknowledge because confounding and reverse causation has now been shown to be the explanation for several different health outcomes initially reported in cross-sectional studies as indicating an association between PFOS or PFOA exposure and the outcome (for example, chronic kidney disease, lower birth weight, early onset menopause). (42)

RESPONSE: As mentioned in the Response to Comment 56, DWQI (2017a) and DWQI (2018a) discuss the commenter's point that many epidemiological studies of PFOA and PFOS are cross-sectional, and that studies of this type cannot address temporality. The Department agrees that associations of PFOA and/or PFOS with chronic kidney disease and early onset menopause are explained by reverse causality. These conditions decrease the excretion of PFOA and, thus, lead to higher serum PFOA levels. However, the Department does not agree that reverse causality or confounding fully explains the associations of PFOA with lower birth weight, as explained in DWQI (2017a) . Reverse causality and/or confounding is not known to be an explanation for several other human health effects associated with PFOA and/or PFOS, such as

increased cholesterol (PFOA and PFOS), increases in the liver enzyme ALT (PFOA), decreased immune response (PFOA and PFOS), and testicular and kidney cancer (PFOA).

58. COMMENT: The Institute's use of adult default exposure values of 70 kg for body weight and 2.0 l/day for water intake results in excessive, unsafe PFOS and PFOA risk to nearly all ages of children. This is especially disconcerting since many epidemiologic studies have shown associations between PFOS and PFOA exposure and health effects in children including adverse effects in serum lipids (high total cholesterol), delayed onset of puberty (associated with altered risk of adult disease: diabetes mellitus, heart disease, bone disease, substance abuse, and asthma), associations between renal function and serum PFC levels, and suppression of vaccine mediated antibody response. Animal studies have also shown a number of adverse reproductive and developmental effects, including delayed mammary gland development with increased vulnerability to later disease development.

The proposed MCLs of 13 ng/l for PFOS and 14 ng/l for PFOA do not protect a large segment of the population. Children two months through age 13 may receive PFOA and PFOS daily doses that exceed the allowable reference doses if 14 ng/l and 13 ng/l are established as MCLs for PFOA and PFOS. Although an uncertainty factor applied for human variability in the risk assessment accounts for variable sensitivity within the broader population, including variability in sensitivity within children sub-populations, appropriate age-specific exposure values for body weights and water intakes should be used to derive MCLs for children subpopulations to enable protection. (9)

59. COMMENT: Fetuses, infants, and children are more vulnerable to exposure-related health effects than adults. The young may be more sensitive to the effects of PFOA and PFOS due to their immature, developing biological systems (such as the immune system), and rapid body growth during development. For example, exposure to PFAS before birth or in early childhood may result in decreased birthweight, decreased immune responses, and hormonal effects later in life. (37)

60. COMMENT: When determining a Maximum Contaminant Level Goal (MCLG), New Jersey should consider adverse health risks to sensitive subpopulations, such as infants, children, the elderly, and those with compromised immune systems and chronic diseases. (37)

61. COMMENT: New Jersey uses a default drinking water exposure parameter for adults that is not an accurate accounting of which populations are most vulnerable to PFOA and PFOS contamination. Sensitive members of the population, such as fetuses, infants, children, nursing mothers, and those with certain preexisting conditions, face particular risk from chemicals of such persistence, and which demonstrate clear adverse effects at very low levels of exposure. The Department should develop a health threshold protective of the of the most vulnerable populations, particularly developing fetuses, infants, and children, by accounting for these sensitive subgroups in the evaluation of data gaps, the selection of uncertainty factors, and the choice of exposure parameters to use.

Infants are more likely to have higher exposure than adults to these contaminants because they ingest more water per kilogram of body weight than adults, and breastfeeding infants are likely to have even greater exposure due to higher levels of PFOA and PFOS in

breastmilk compared to drinking water. It is, therefore, important to account for these differences in exposure when setting New Jersey's MCL, so that exposures to PFOA and PFOS during this crucial developmental window are limited.

Three states, Vermont, Minnesota and Michigan, have already incorporated exposure estimates for infants. Vermont has used 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, 0.175 L/kg-day. An alternative modeling approach was taken by Minnesota, which incorporated infant exposures, using chemical specific toxicokinetic parameters including placental and breast milk transfer. This new model prepared by Minnesota takes into account that babies are already born with a transgenerational body burden from placental transfer based on maternal accumulation, and that infants may also experience subsequently higher exposures, due to higher body weight adjusted water intake rates and/or the partitioning of PFAS in breast milk. The model has been peer-reviewed and was published in the Journal of Exposure Science & Environmental epidemiology on January 10, 2019. New Hampshire stated on February 21, 2019, that it may incorporate use of this model for derivation of MCLs, stating that "health-based drinking water or groundwater standards for PFOA and PFOS would potentially be lowered significantly below the initial proposal figures." Michigan has incorporated the use of the Minnesota model for its screening levels for PFOA, PFOS and PFHxS, and PFNA. As a consequence, Michigan's screening levels are lower than New Jersey's proposed MCLs for PFOA and PFOS (nine ppt for PFOA and eight ppt for PFOS).

Importantly, either approach to accounting for the unique exposure situation of infants would significantly reduce New Jersey's proposed MCLG for both PFOA and PFOS. If New Jersey used Vermont's infant exposure assumptions, its proposed MCLGs would then become two ppt for PFOA and PFOS. The MCLGs for each contaminant would be lowered below one ppt if an additional uncertainty factor was applied to ensure adequate protection of fetuses, infants and children, as recommended by the National Academy of Sciences and as required in the Food Quality Protection Act. Notably, the MCLG for PFOA would be below one ppt for all exposure populations if it was based on altered mammary gland development.

Another approach to assessing New Jersey's proposed MCLs is to input them into Minnesota's exposure model for breastfed infants. At 14 ppt PFOA in drinking water, blood serum levels of PFOA would exceed New Jersey's target human serum level of 14.5 ug/L within the first year of life and stay above this level until approximately four years of age (peaking at approximately 25 ng/ml), solely from drinking water and no other routes of PFOA exposure. Drinking water would then contribute more than 50 percent of the total target human serum level up to the age of eight, increasing the likelihood that other routes of PFOA exposure could push a child's blood serum level over New Jersey's target human serum level. At 13 ppt for PFOS in drinking water, drinking water would contribute more than 50 percent of New Jersey's total target human serum level of 22 µg/l for the first eight years of life (peaking at approximately 18 µg/l), increasing the likelihood that other routes of PFOS exposure could push a child's blood serum level over New Jersey's target human serum level. Interestingly, incorporating fetal, infant, and childhood exposures increases an adults steady-state blood

serum levels for both PFOA and PFOS above 20 percent (the relative source contribution limit from drinking water selected by New Jersey) of New Jersey's target human serum level at least until age 55, where the model ends. (37)

62. COMMENT: If MCLs are based on adult body mass, they are probably insufficient to protect smaller individuals and especially children. (30)

RESPONSE TO COMMENTS 58, 59, 60, 61, AND 62: As discussed in detail in DWQI (2017a, b) and DWQI (2018a, b), it is acknowledged that infants and children have higher exposures to PFOA and PFOS from contaminated drinking water than adults and that they are susceptible subpopulations for the effects of PFOA and PFOS. Higher risks to sensitive subpopulations, such as infants and children (as well as the elderly and those with compromised immune systems and chronic diseases, mentioned by one of the commenters) are considered through the intraspecies uncertainty factor of 10, which is applied to protect more sensitive individuals within the population. However, the adopted MCLs for PFOA and PFOS are not based on exposure factors (consumption rate, which is based on volume ingested per day and body weight) for infants or children because of uncertainties related to toxicokinetic considerations. Specifically, it is not scientifically supportable to use the higher drinking water consumption rates for infants and children with a reference dose based on a steady-state serum level of PFOA or PFOS, since steady-state is reached from exposure to a constant dose of PFOA or PFOS over a period of many years. In contrast, the higher exposure rates in infants and children vary at different age periods and occur over a time period shorter than needed to reach steady-state. These issues are addressed in the Minnesota transgenerational toxicokinetic model

(Goeden et al., 2019), which is recommended by one of the commenters. Although this model was not available when PFOA and PFOS MCLs were developed, the Department has reviewed Goeden et al. (2019). This review did not alter the Department's determination to proceed with this rulemaking.

While the Department recognizes that new information and models relevant to PFOA and PFOS risk assessment continue to become available, the Department's MCLs are based on the information and models that were available at the time when they were developed. As discussed in DWQI (2017a) and DWQI (2018a), the use of a relative source contribution (RSC) factor of 20 percent (the most stringent value within the recommended range of 20 percent to 80 percent), while not explicitly intended for this purpose, partially accounts for the higher PFOA and PFOS exposures in young infants, because this age group is not expected to have substantial exposures from non-drinking water sources. It is noted that Michigan, Minnesota, and New Hampshire, which used the transgenerational toxicokinetic model in development of PFOA and PFOS drinking water guidelines, selected an RSC of 50 percent. If all other parameters are equal, this would result in a health-based drinking water level that is two and a half times higher than the Department's PFOA and PFOS MCLs.

Relative Source Contribution (RSC)

63. COMMENT: We are concerned with the extreme conservancy associated with the Department's 20 percent RSC assumption. In developing the proposed MCLs, the Department assumes an RSC of 20 percent, despite acknowledging that PFOA and PFOS use has "decreased

substantially." Although 20 percent is often used as a default assumption for the exposure resulting from drinking water, the available evidence suggest that other sources of potential exposure to PFOA and PFOS have declined drastically. According to data collected by the CDC, mean serum levels of PFOS declined by 85 percent in the US population between 1999 and 2016. According to CDC, mean serum levels of PFOA declined by 60 percent over the same timeframe. Given those dramatic declines, it is inappropriate to assume that 80 percent of exposure to these substances comes from sources other than drinking water. While a few other states have assumed an RSC of 50 or 60 percent, it is likely that the contribution of drinking water to overall exposure is even higher, particularly in areas where drinking water contamination has been detected. (23, 27, and 41)

64. COMMENT: The Department should Increase the RSC for PFOS. The Department chose a RSC of 20 percent for its PFOS MCL derivation citing that: "there are insufficient data to develop a chemical-specific RSC for PFOS" The available chemical-specific data from PFOS drinking water affected communities, as reported by Landsteiner et al. (2015) and Li et al. (2018), provided substantial evidence that elevated PFOS levels in the drinking water can be the primary route of PFOS exposure. Therefore, the Department could consider raising the RSC for PFOS. Other states, such as Minnesota and New Hampshire, have used 50 percent.

Also, it is incorrect for the Department to state that "There are no New Jersey-specific biomonitoring data for PFOS, and its more frequent occurrence in New Jersey public water systems as compared to the U.S. as a whole suggests that New Jersey residents may also have higher exposure from non-drinking sources than the U.S. general population (e.g. NHANES)."

Limited data reported by Graber et al. (2019) did not show that New Jersey residents have higher exposure (of PFOS). Study by Graber et al. was a cross-sectional biomonitoring study in Paulsboro area (New Jersey) where higher PFNA levels were detected in the community water supply system in 2009. Although PFOS concentration in the water was not reported, 13 PFAS serum concentrations were measured, including PFOS from 165 residents (greater than 12 years old). Compared to the representative data from NHANES, there was no difference in the PFOS serum levels from these community residents. (42)

65. COMMENT: The Department chose a relative source of contribution (RSC) of 20 percent for its PFOA MCL derivation citing that: “there are insufficient data to develop a chemical-specific RSC for PFOA.” This is incorrect. The available chemical-specific data from PFOA drinking water affected communities, as reported by Emmett et al. (2006) and Landsteiner et al. (2015), provided substantial and compelling evidence that elevated PFOA levels in the drinking water will become the primary route of PFOA exposure. Therefore, the Department should raise the RSC for PFOA. States, such as Minnesota and New Hampshire, have used 50 percent RSC. (42)

RESPONSE TO COMMENTS 63, 64, AND 65: As discussed in DWQI (2017a) and DWQI (2018a), the Institute concluded that there are insufficient data to develop chemical-specific RSC factors for PFOA or PFOS that can be applied to PFAS exposures in New Jersey residents, and, therefore, the default RSC of 20 percent was used. The frequent occurrence of PFOA and PFOS in New Jersey drinking water may also result in more frequent occurrence in other

environmental media. Therefore, New Jersey residents may also have higher exposure from non-drinking water sources than the U.S. general population.

For example, contamination of drinking water with PFOA and/or PFOS, as well as PFOS at levels of potential human health risk in fish (Goodrow et al., 2018), have been found in specific locations in many areas of the State. Potential sources have been identified in some instances, while sources are unknown in other locations. The PFAS biomonitoring data reported by Graber et al. (2019) comes from a location where PFOS contamination is not known to be present. Therefore, elevated levels of PFOS in the serum of the population studied by Graber et al. (2019) is not expected.

Additionally, as mentioned above, the default RSC of 20 percent, while not explicitly intended to account for the higher exposures in infants, is necessary to at least partially account for the higher PFOA and PFOS exposures in infants, which the Department does not otherwise account for in the exposure assumptions used. As discussed in detail in DWQI (2017a) and DWQI (2018a), exposures to infants, both breastfed and consuming formula prepared with contaminated drinking water, are several-fold higher than in than older individuals. These higher infant exposures must be considered because toxicological effects of concern occur from short-term exposures relevant to elevated exposures in infancy, including when exposure occurs only through lactation. The information about the PFOA and PFOS drinking water risk assessments developed by New Hampshire and Minnesota mentioned in the comment are not relevant to the Department's MCLs for PFOA and PFOS. In the New Hampshire and Minnesota risk assessments, a transgenerational toxicokinetic model is used to

account for the much higher exposures of breastfed infants, and the RSC of 50 percent applies specifically to exposures to breastfed infants, while the Department's MCLs are based on default adult exposure assumptions and a more stringent RSC.

USEPA Health Advisories

66. COMMENT: New Jersey is not unique in its need for more stringent PFOA and PFOS drinking water standards and we question the difference in scientifically derived drinking water standards from what is being proposed and what remains in place at the Federal level.

The USEPA has not established an MCL for PFOA or PFOS. Rather, it has set a health advisory for PFOA and PFOS combined at 70 ppt. The USEPA webpage states that they have set these levels "based on the agency's assessment of the latest peer-reviewed science." It is our understanding that the USEPA and the Department calculate health effects and risks from contaminants in drinking water using essentially the same risk analysis methodologies. Thus, we presume, the difference in the conclusions between the two agencies cannot be explained by the implementation of differing calculations.

As the USEPA has acted on the exact same information as the Department and the same risk methodologies have been used, the Department should resolve the discrepancy between the State and Federal levels and explain why its levels are significantly lower than the USEPA health advisories. (8)

67. COMMENT: The NJDEP should more fully address the difference between this rulemaking and the 70 ppt USEPA Drinking Water Health Advisory for PFOS and PFOA. (49)

RESPONSE TO COMMENTS 66 AND 67: The Department agrees that the same risk assessment approaches were used to develop both the USEPA Health Advisories and the Department MCLs for PFOA and PFOS. The USEPA and Department risk assessments are generally based on review of the same scientific information, although the Institute evaluations considered more recent studies. The reasons for the differences in the USEPA and Department risk assessments are thoroughly explained in Appendix 2 of DWQI (2017a) and DWQI (2018a). Additional supporting information is in the Institute's responses to comments from the USEPA to the draft Institute Heath-based MCL for PFOA (DWQI, 2017b). In summary, the Institute concluded that the USEPA Health Advisories for PFOA and PFOS are not sufficiently protective of public health. The USEPA Health Advisories do not consider sensitive toxicological endpoints that meet the criteria for consideration in risk assessment. Further, they do not consider that exposure to drinking water at 70 ppt (0.070 µg/l) will result in increases in blood serum PFOA and PFOS levels well above the serum levels associated with several health effects in human studies. The Department also notes that seven other states have concluded that the USEPA Health Advisories for PFOA and PFOS are not sufficiently protective and have proposed or established drinking water standards or guidance values lower than the USEPA Health Advisories.

Agency for Toxic Substances and Disease Registry (ATSDR)

68. COMMENT: The two studies selected by ATSDR, Onishchenko et al. (2011) and Koskela et al. (2016), lacked fundamental scientific rigor (for example, using a single dose study without any dose-response, small sample size with only six pregnant dams; no details on the reproductive nor the developmental hallmarks, litter bias, non-standard testing methods, no

internal serum PFOA dosimetry data, etc.). Given these flaws, the proposed ATSDR Minimal Risk Levels (MRLs) were not derived using best available science and do not provide support for the Department proposal. (42)

RESPONSE: The Health-based MCL for PFOA (DWQI, 2017a) is not based on the same studies as the draft ATSDR (2018) MRL for PFOA. Although the Institute did not select these studies as the primary basis for the PFOA MCL, these studies and the MRL derived in ATSDR (2018) support the Institute's conclusion that developmental effects of PFOA occur at lower doses than other effects.

69. COMMENT: The Department should consider that in deriving their proposed guidance values, both ATSDR and the USEPA apply uncertainty factors on the assumption that humans are more sensitive than rodents to these effects. This has not been shown to be the case, however. Published data strongly support that rodents are likely to be much more sensitive to PFAS-induced effects than humans. ATSDR has acknowledged the impact on these various differences on the reliability of its risk assessment, noting that "for the most part, adverse health effects in studies in animals have been associated with exposure concentrations or doses that resulted in blood levels of perfluoroalkyl compounds that were significantly higher than those reported in perfluoroalkyl workers or in the general population." This, along with "profound differences in toxicokinetics between humans and experimental animals," (such as the differences in half-lives between species) and issues related to peroxisome proliferator

activated receptor-alpha (PPAR-alpha), "make it somewhat difficult at this time to determine the true relevance of some effects reported in animal studies to human health." (42)

RESPONSE: The same administered dose of PFOA or PFOS results in a much higher internal dose in humans than in rodents due to a slower excretion rate of PFOA and PFOS in humans than in rodents (see Post et al., 2017). This toxicokinetic difference is accounted for in the PFOA and PFOS risk assessments by using internal dose (serum PFAS level) as the dose metric. Additionally, the blood serum PFOA and PFOS levels that are associated with human health effects are generally much lower than those at which toxic effects are seen in rodent studies, suggesting that the conclusion that humans are less sensitive to the effects of PFAS than rodents is not valid.

70. COMMENT: The CDC and the Agency for Toxic Substances and Disease Registry (ATSDR) have just announced that they will be conducting exposure assessments in communities near current or former military bases and communities that are known to have had PFAS in their drinking water. This information will provide the basis for development of future studies on the effects of PFAS on human health, and the Department should wait until a proper study of the effects of PFAS health is completed and the data have been properly evaluated. (23, 27, and 41)

RESPONSE: The Department is aware of the CDC/ATSDR exposure studies mentioned by the commenter, as well as the ATSDR-funded multi-site PFAS studies which have not yet begun. The health studies at each individual site will take five years to complete, and additional time will be

needed to consolidate the data from all of the sites so that overall conclusions for the multi-site can be made. The Department recognizes that new information on PFAS continues to become available. However, the Department believes that the large body of health effects information available on PFOA and PFOS provides sufficient data for MCL development. Delay of these MCLs will pose a threat to public health.

European Food Safety Authority (EFSA)

71. COMMENT: With respect to EFSA, the Department said "the EFSA tolerable weekly intakes and associated daily intake values provide additional support for the Institute's reference doses for PFOA and PFOS" because the EFSA daily intake values are near, or lower than, the Institute's PFOA and PFOS reference doses. The EFSA based its tolerable levels on a novel approach of using human epidemiological studies concerning cholesterol to perform quantitative risk assessment to calculate the tolerable intakes. The Department's embrace of this approach contradicts a recent published position taken by Institute members in December 2017. The article stated "there is a high bar for use of human epidemiology in quantitative risk assessment due to its observational nature … limitations in the current human database such as inability to determine the dose-response relationships for individual PFAAs due to cooccurrence of other PFAAs, preclude the use of human data as the primary basis for PFAA drinking water guidelines." (42)

RESPONSE: The primary basis of the quantitative risk assessment for PFOA and PFOS presented in DWQI (2017a) and DWQI (2018a) is toxicological effects from animal studies, for the reasons

cited to Post et al. (2017) by the commenter. However, DWQI (2017a) and DWQI (2018a), as well as Post et al. (2017), concluded that there is substantial evidence for several human health effects from relatively low exposures to PFOA and PFOS. The consistency between the tolerable daily intakes described in EFSA (2018), which are based on human data, and the reference doses utilized by the Institute for PFOA and PFOS, which are based on animal data, supports the Institute's conclusions.

On February 24, 2020, the EFSA posted a "Public consultation on the draft scientific opinion on the risks to human health related to the presence of perfluoroalkyl substances in food" for public comment (see https://www.efsa.europa.eu/en/consultations/call/public-consultation-draft-scientific-opinion-risks-human-health). In this document, EFSA develops a tolerable daily intake of 1.16 ng/kg/day for the total of four long-chain PFAS (PFOA, PFOS, PFNA, and perfluorohexanoic acid [PFHxS]). The tolerable daily intake is based on a recent study (Abraham et al., 2020) of the association of these PFAS with decreased vaccine response in one-year-old children. To account for the higher exposures to PFAS in breastfed infants and young children, the EFSA's tolerable daily intake is based on the daily maternal PFAS dose resulting in serum PFAS levels associated with decreased vaccine response in their breastfed children at age one year.

Health Canada and the Australia PFAS Science Panel

72. COMMENT: The vast body of scientific evidence does not establish that PFOS or PFOA cause any adverse health effects in humans at current exposure levels, or even at the

historically higher levels found in blood. The Department dismisses careful and detailed evaluations of PFOS and PFOA made by the USEPA, Health Canada, Australia's PFAS Science Panel, and other organizations. The Department focused on studies and findings reviewed and rejected by these entities. (42)

73. COMMENT: A recently released review of studies involving perfluoroalkyls exposed populations commissioned by the Australian government also supports the lack of evidence of harm. In the May 2018 report by the Australian Expert Health Panel, "[t]he Panel concluded there is mostly limited or no evidence for any link with human disease from these observed differences. Importantly, there is no current evidence that supports a large impact on a person's health as a result of high levels of perfluoroalkyl exposure." The report further stated, "[a]fter considering all the evidence, the Panel's advice to the Minister on this public health issue is that the evidence does not support any specific health or disease screening or other health interventions for highly exposed groups in Australia, except for research purposes." This point is illustrated by the following table summarizing recent drinking water standards and guidance levels for PFOA and PFOS set by USEPA, German, Dutch, Canadian, Swedish, and Australian environmental authorities. As indicated by the chart, different national environmental protection authorities have arrived at different toxicity values and drinking water guidance levels for the same chemicals. Nonetheless, all of these drinking water guidance values are five times or more higher than the MCLs proposed by New Jersey. (42)

74. COMMENT: In the proposed rule, the Department discusses draft toxicological profiles of six PFAS from ATSDR and an assessment by EFSA. We presume that the Department cited

only these two organizations since they have produced draft or final human hazard assessments that produce threshold levels comparable to the Department's proposed MCLs. There are three problems with the Department's selective citation of these sources as justification for its hazard assessment. First, the draft ATSDR toxicological profile is only a draft and, like the Institute's Health Effects report, omitted key studies relevant to Department's PFOA and PFOS draft drinking water standards. The draft ATSDR toxicology profiles also did not have a public peer review or follow many of the other NAS recommendations for high quality health assessments. Second, ATSDR MRLs are intended to be used only as a screening tool to identify populations potentially at risk, to help public health professionals decide where to look more closely. They are not maximum safe exposure levels and may be set orders of magnitude lower than levels shown to be non-toxic in laboratory animals. Third, other public health agencies have reviewed the same literature and, after notice and comment, have reached very different scientific conclusions than Institute and ATSDR.

In December 2018, Health Canada published its final Guidelines for: Canadian Drinking Water Quality's Guideline Technical Document for PFOA. Developed after public comment, the final Health Canada assessment evaluated the same literature and potential mechanisms/modes of action as the USEPA, ATSDR, and the Institute. More importantly, Health Canada applied a structured evidence integration analysis as the NAS recommended. Its evidence integration methodology, based on the World Health Organization (WHO) and Bradford-Hill criteria, properly screened out, due to study limitations and inconsistencies with other research findings, evidence that states like Minnesota and New Jersey relied on.

In March 2018, an expert panel formed by the Australian Ministry of Health gave its comprehensive evaluation of the literature concerning potential PFAS health effects. The expert panel consisted of academic researchers and medical practitioners independent of the ministry. The expert panel explicitly applied a structured evidence integration framework consistent with the NAS recommendations. The panel concluded that differences between those with the highest and lowest exposures are generally small, with the highest groups generally still being within the normal ranges for the whole population. The panel also found there is mostly limited or no evidence for an association with human disease accompanying these observed differences. Lastly, the panel found there is no current evidence that supports a large impact on an individual's health. In particular, there is no current evidence that suggests an increase in overall cancer risk.

As part of its consideration of the new scientific data published since the Institute evaluation, the Department should also evaluate the analysis of Health Canada, the Australian Ministry of Health, and other public health organizations before adopting MCLs. (51)

RESPONSE TO COMMENTS 72, 73, AND 74: The ATSDR Draft Toxicological Profile (2018) was reviewed by external peer reviewers, including a non-governmental panel, and was made available for public review. ATSDR bases its MRLs on non-carcinogenic effects, and they are developed using the same risk assessment approaches used to develop the USEPA and Department reference doses for drinking water contaminants. It is generally true that human health values, including reference doses developed by the USEPA, as well as MRLs developed by ATSDR, for some environmental contaminants are set at orders of magnitude below the No

Observed Adverse Effects Levels (NOAELs) from animal studies; this is not unique to PFOA, PFOS or PFAS, or to other MRLs developed by ATSDR. These human health-based levels are set lower than the NOAELs from animal studies to account for uncertainties about interspecies differences in sensitivity, gaps in the toxicological database, and other factors.

The Australian Expert Health Panel conclusions that are cited are not the basis for development of quantitative risk assessments (such as reference dose, health-based drinking water, or groundwater levels) for PFOA and PFOS, but rather address the weight of evidence for the association of PFOA and PFOS with human health effects. It should be noted that the report also states: "[a]lthough the evidence on health effects associated with PFAS exposure is limited, the current reviews of health and scientific research provide fairly consistent reports of associations with several health outcomes, in particular: increased cholesterol, increased uric acid, reduced kidney function, altered markers of immunological response, levels of thyroid and sex hormone levels, later menarche and earlier menopause, and lower birth weight."

Further, an observable increase in overall cancer risk is not the basis for a public health-protective guideline for an environmental contaminant. The MCLs are intended to protect from cancer at the one-in-one million lifetime risk level, much lower than the increase in overall risk from an environmental contaminant that is detectable in the human epidemiology studies of PFOA and PFOS.

Hall et al. (2012) presented criteria for the use of hepatocellular hypertrophy and increased liver weight in risk assessment. Health Canada based its PFOA risk assessment on hepatocellular hypertrophy, an effect that is closely related to increased liver weight, which was

used as the basis for the Department's PFOA risk assessment. Therefore, Health Canada's findings are consistent with the Department's on use of this criteria.

The Department has reviewed the basis for the standards and guidance levels established by other state and national governments that are higher than the Department's adopted MCLs. In general, differences among drinking water standards and guidance levels for PFOA and PFOS arise from differences in toxicity factors and/or exposure assumptions. DWQI (2017a) and DWQI (2018a) provide detailed support for the conclusion that the USEPA Health Advisories of 70 ppt (0.070 µg/l) for PFOA and PFOS are not sufficiently health-protective and the same conclusion applies even more so to the values developed by others that are higher than the USEPA Health Advisories. The standards and guidance values submitted by the commenter are not based on the most sensitive toxicological endpoints that are well-established and relevant to humans.

Additionally, as discussed in DWQI (2017a) and DWQI (2018a), multiple human health effects are associated with serum PFOA and PFOS levels well below those that will result from consumption of drinking water at the USEPA Health Advisory of 70 ppt (0.070 µg/l). Accordingly, DWQI (2017a) and DWQI (2018a) concluded that the blood serum PFAS increases at 70 ppt (0.070 µg/l) "are not desirable and may not be protective of public health." The Department notes that several other states have developed drinking water guidelines for PFOA and PFOS that are lower than the USEPA Health Advisories, including four states with PFOA and/or PFOS drinking water guidelines lower than the New Jersey MCLs.

Development of the MCL for PFOA

Critical Effects

75. COMMENT: As noted in the Institute's summary document for PFOA that is the basis for the proposed standards, systemic effects have been observed in experimental animals exposed to PFOA, including effects on liver, immune system, and developmental effects. However, not all of the observed animal effects are adverse, and not all animal adverse effects are relevant to humans. (23, 27, and 41)

RESPONSE: As detailed in DWQI (2017a) and DWQI (2018a), the hepatic, immune system, and developmental effects of PFOA and PFOS in animal studies are considered to be adverse effects or precursors to adverse effects. According to USEPA risk assessment guidelines (USEPA, 2002), adverse effects and effects that can progress to adverse effects are appropriate for use as the basis for human health risk assessment. Regarding the relevance of animal effects to humans, based on the mode of action analyses presented in DWQI (2017a) and (2018a), the Department considers the adverse hepatic, developmental, and immune system effects of PFOA and PFOS to be relevant to humans.

76. COMMENT: Based on increased relative liver weight effects observed in mice by Loveless et al. (2006), the Department (through its Health Effects Subcommittee within the Institute) developed an MCL for PFOA in drinking water at 0.014 µg/l. In addition to increased absolute and relative liver weights, direct evidence of hepatic peroxisome proliferator-activated receptor alpha (PPAR-alpha) activation with PFOA exposure was also demonstrated by Loveless

et al. (2006) shown as increased cyanide insensitive hepatic peroxisomal β-oxidation activity from PFOA-treated animals.

We respectfully disagree with Departments' use of rodent liver weight as a critical effect to establish a point-of-departure for derivation of a reference dose for PFOA. It is inconsistent with USEPA guidelines and published expert opinions on the distinction between liver hypertrophy as a non-adverse adaptive change and other endpoints representing liver toxicity. Moreover, the observational human data, as well as a significant body of mechanistic experimental data that relates to the liver response to exposure to PFOA strongly suggests that rodent liver weight as an endpoint for the human-health risk assessment of PFOA is inappropriate and needlessly conservative. (42)

77. COMMENT: Increased relative liver weight is a common effect of PFOA in animal studies that has been reported to occur at lower levels of exposure than those causing effects on other organ systems. Extrapolation of liver effects seen in animals to humans must be approached with caution, however, in light of the conclusions of the C8 Health Project and recent human data reported by Convertino et al. (2018) and strong evidence for rodent-specific adaptive responses. (27)

78. COMMENT: Increased liver weight due to hepatocellular hypertrophy can be an adaptive (protective) effect in animals due to up-regulation of detoxification enzymes, leading toxicologists to revisit key liver endpoint studies. Research has shown that many metabolic effects of exposure to PFOA and PFOS in rodents can be explained by the activation of xenosensor nuclear receptors, such as the peroxisome proliferator activated receptor (PPAR-

alpha) in the liver. These effects are of questionable relevance for risk assessment since the associated proliferative response in mice has not been observed in humans. (27)

79. COMMENT: The endpoint selected for MCL development is increased liver weight in male mice. An expert panel convened by the European Society of Toxicologic Pathology (ESTP) defined what constitutes an adverse hepatic effect. Hall et al. (2012) summarized the findings of the ESTP workgroup, and per the ESTP criteria, increases in liver weight without histological evidence, such as necrotic changes or degeneration of liver cells may be a normal adaptive response and, therefore, not considered adverse or relevant for human risk assessment. Hall et al. (2012) does not appear to have been considered in selecting increased liver weight as the MCL endpoint as it is not included in the references. (24)

RESPONSE TO COMMENTS 76, 77, 78, AND 79: As stated in DWQI (2017a), "…. numerous studies of PFOA have demonstrated that increased liver weight co-occurs with and/or progresses to more severe hepatic effects including increased serum liver enzymes, hepatocellular necrosis, fatty liver, and/or hyperplastic nodules. Additionally, recent studies [such as Quist et al., 2015] show that cellular damage indicative of liver toxicity persists until adulthood following developmental exposure to PFOA." Similarly, Butenhoff et al. (2012a) concludes that the observations at one year and two years in a chronic study in rats suggest a progression of lesions "from hepatocellular hypertrophy to fatty degeneration to necrosis followed by regenerative hyperplasia." DWQI (2018a) provides a detailed review of the data from the numerous studies showing co-occurrence or progression of hepatocellular hypertrophy and/or increased liver weight, to more severe histopathological changes in the

liver and increased liver enzymes and/or bile acids to the use of increased liver weight as the basis for the reference dose for PFOA.

DWQI (2017b) also references Hall et al. (2012), which states that increased liver weight or hepatocellular hypertrophy are adverse when they co-occur with, or progress to, other types of liver toxicity, such as those associated with PFOA. It should be noted that the primary focus of Hall et al. (2012) is pre-clinical toxicity studies for drug development, for which less-than-chronic durations of exposure are most relevant, while the reference dose and MCL for PFOA are intended to protect for chronic (lifetime) exposure. Hall et al. (2012) also emphasizes that the expected duration of exposure must be considered in determining the adversity of effects, such as increased liver weight. Such effects may be reversible if the anticipated duration of exposure is short, while progression to more severe hepatic effects may occur from longer exposures to the same dose. These duration of exposure considerations are relevant to safety evaluation of drugs, since they are normally only taken for a limited period of time. However, because the Department's adopted MCL for PFOA is intended to protect for lifetime exposure, reversibility of effects when exposure ends is not a relevant consideration.

Similarly, USEPA (2002) guidance also emphasizes that the potential for progression to more severe types of toxicity with longer exposure must be considered in determining whether liver weight/hepatocellular hypertrophy in studies of less-than-chronic duration is adverse.

Several other risk assessments of PFOA, including Health Canada (2016), which is recommended in other comments from the same commenters, are based on increased liver weight and/or hepatocellular hypertrophy. Health Canada (2016) cites the conclusions of Hall

et al. (2012), and additionally concludes that increased liver weight and hepatocellular hypertrophy in rats are an appropriate basis for its risk assessment of PFOA. The evaluation notes that these effects can progress to more serious hepatic toxicity with continued exposure.

DWQI (2017a) includes detailed evaluations of the primary data from numerous relevant studies that support the human relevance of hepatic effects of PFOA in rodents. While some PPAR-alpha activating chemicals have been shown to cause hepatic toxicity, particularly tumors, through a PPAR-alpha activation mode of action that may not be relevant to humans, several lines of evidence support the conclusion that hepatic effects of PFOA should be considered to be relevant to humans. DWQI (2017a) notes that the dose-response curves for liver toxicity and PPAR-alpha activation from PFOA are similar in non-human primates and rats. Additionally, PFOA causes hepatic toxicity in PPAR-alpha null strains of mice lacking PPAR-alpha, and liver toxicity in these PPAR-alpha null mice is more severe in some cases than in wild type mice of the same strain. Also, increased liver weight does not correlate with magnitude of PPAR-alpha activation even in standard outbred strains of rodents.

Regarding hepatic PPAR-alpha activation (as indicated by increased cyanide insensitive palmitoyl CoA hepatic peroxisomal β-oxidation [PCO] activity) in rodents dosed with PFOA in Loveless et al. (2006), DWQI (2017a) shows that increased relative liver weight did not correlate with hepatic peroxisome proliferation, as indicated by PCO activity. As stated by DWQI (2017a), "[t]hese results illustrate the involvement of PPAR-alpha independent processes in the increased relative liver weight caused by PFOA even in standard strains of rodents with normal PPAR-alpha function." Furthermore, two additional studies (Quist et al., 2015; Li et al., 2017)

reported PPAR-alpha independent liver toxicity in mice at very low doses of PFOA (0.01 mg/kg/day and 0.05 mg/kg/day, respectively); a No Observed Adverse Effect Level (NOAEL) was not identified in either study. Quist et al. (2015) was not considered as the basis for the DWQI (2017a) reference dose for PFOA because it did not provide the serum data needed for dose-response modeling. Li et al. (2017) was not available to DWQI (2017a), but was the basis of the PFOA reference dose of 1.8 ng/kg/day developed by California USEPA (2019).

80. COMMENT: We disagree with the Institute's selection from the Loveless et al. (2006) study of only the data for mice that received linear and branched ammonium PFOA treatment for its MCL derivation. Only linear PFOA was detected in the general population in the latest NHANES 2015-2016 cycle analyses. Branched PFOA was not detected. If the Institute continues to use the Loveless et al. (2006) study as the basis of its PFOA MCL, it should use a subgroup of the mice data that were treated with linear ammonium PFOA. This data results in a BMDL10 of increased relative liver weight of 7,973 ng/mL (which is 1.8 times higher than the current BMDL10 used by the Institute). Using a BMDL10 would result in a higher PFOA MCL to 0.026 µg/L by considering this parameter alone. (42)

RESPONSE: The data from the mice dosed with a mixture of linear PFOA (in which carbons atoms are in a straight chain) and branched PFOA (in which the carbon chain is branched) are appropriate for use in risk assessment because there was a dose-related increase in relative liver weight over the whole range of doses used in the study. Additionally, the relevance to effects in humans is supported by the observation that increased liver weight is not correlated

with hepatic PPAR-alpha activity. The data for linear and branched PFOA is appropriate for development of an MCL because drinking water may be contaminated with both linear and branched PFOA, depending on the source of the contamination. Though branched PFOA was not detected in NHANES 2015-2016 (see https://www.cdc.gov/exposurereport/pdf/FourthReport_UpdatedTables_Volume1_Jan2019-508.pdf), the NHANES data are from the general population whose exposure is primarily from food and consumer products, not drinking water, which can be contaminated with branched PFOA. It is noted that branched PFOA is excreted more quickly than linear PFOA in both rodents (DWQI, 2017a) and humans (Zhang et al., 2013). Rapid excretion of branched PFOA diminishes the likelihood of detection in blood serum if low levels of exposure are occurring. The Department notes that other toxicological studies of PFOA, including for example Perkins et al. (2004), also utilized linear and branched PFOA.

81. COMMENT: In addition to assessing liver effects, the Institute considered Health-based MCLs based on evidence of delayed mammary gland development and testicular cancer in laboratory studies. Many metabolic effects of exposure to PFOA in rodents, including developmental effects, are associated with a proliferative response in mice that has not been observed in humans. (23, 27, and 41)

RESPONSE: The “proliferative response” mentioned in the comment is assumed to refer to responses related to PPAR-alpha activation. The statement that this response does not occur in humans is not accurate. As discussed in DWQI (2017a), PPAR-alpha, other PPARs, and other

nuclear receptors are found in many tissues in humans, as well as rodents and other species. In general, PPARs, including PPAR-alpha, affect many biological processes beyond stimulation of peroxisome proliferation in rodents, the effect for which they were originally named. Regarding developmental effects of PFOA, DWQI (2017a) states that:

> PFOA is associated with decreased fetal growth in humans. PPAR-alpha and other PPARs are present in human fetal tissues and are expected to have important roles in reproduction and development. Therefore, PPAR-alpha mediated effects of PFOA on development are considered relevant to humans for the purposes of risk assessment. The developmental effects from exposure to PFOA in rodents appear to occur primarily through PPAR-alpha dependent mechanisms, while some reproductive effects such as full litter resorptions appear to be PPAR-alpha independent. However, high affinity "pure" PPAR-alpha activators [compounds whose primary or effect is to activate PPAR-alpha at low concentrations] (WY [Wyeth 14,643; 4-Chloro-6-[2,3-xylidino]-2-pyrimidinylthioacetic acid] and clofibrate) do not cause the developmental effects in mice that were caused by PFOA.

Regarding delayed mammary gland development, DWQI (2017a) reviews the evidence in support of the conclusions that rodents are an appropriate model for studying the effects of environmental contaminants on human mammary gland development and that the role of PPAR-alpha in the effects of PFOA on mammary gland development in rodents is not known.

Regarding testicular tumors, DWQI (2017a) states that the mode of action for testicular Leydig cell tumor induction by PFOA is unknown. While several modes of action suggesting that these tumors may not be relevant to humans have been proposed, data from the only chronic mechanistic rat study (Biegel et al., 2001) do not support one of the key events in each of these proposed modes of action (Klaunig et al., 2012). As such, the testicular tumors caused by PFOA in rats are considered to be relevant to humans.

82. COMMENT: If the Institute insists on using liver weight as a sensitive endpoint, the Institute should include additional available studies in mice and rats which capture sensitive life stages (that is, gestation exposure) or with longer-term exposure duration (that is, 13-week treatment). (42)

83. COMMENT: Given that the Department emphasized that “the developmental period is a sensitive lifestage for PFOA’s hepatic effects, and that increased relative liver weight is a relative and appropriate endpoint for PFOA’s toxicity,” the increases in relative liver weights observed in lactating dams in mice should be considered by the Department, such as the study by Abbott et al. (2007). A key strength of using pregnant and lactating animal data is to reflect PFOA exposure in lactating women; another advantage of considering the above-mentioned studies is that Abbott et al. (2007) not only administered PFOA during gestation to wild type mice, they also utilized PPARα null (knockout) mice as well. The inclusion of PPAR-alpha null mice (in addition to wild type) is of particular importance because the Department has established its position that non-PPAR-alpha mechanism can cause hepatic hypertrophy with PFOA. Therefore, the data obtained from a non-PPAR-alpha responsive mouse model (for

example, PPARα null mice) should provide even more relevance than those obtained from wild type.

In their review of the studies, the Health Effects Subcommittee within the Institute had excluded the study by Abbott et al. (2007) for MCL consideration because serum PFOA concentration data (from lactating dams) were obtained at three weeks after last PFOA dosing (that is, end of weaning / lactation). The difference in the timing of the tissue collection should not be the basis for data exclusion because the benchmark dose variables evaluated by the Institute were exposure (serum PFOA concentration) and effect (increased relative liver weight). Even though Abbott et al. (2007) did not measure serum PFOA concentration in dams until the end of lactation, there were still appreciable large amount of PFOA in the blood of these animals (mainly due to slow serum elimination half-life). Therefore, the Department should also consider evaluating the mouse dam data from Abbott et al. (2007), which encompassed more sensitive life stages (gestation and lactation) than the adult male mice from Loveless et al. (2006). (42)

84. COMMENT: On the sole premises of increased liver weight effects in non-pregnant rodents, another study that the Institute should consider is a 90-day dietary study in male Sprague Dawley rats by Perkins et al. (2004). Sprague Dawley rats were also included in the 14-day study by Loveless et al. (2006). The study by Perkins et al. was also excluded by the Health Effects Subcommittee within the Institute, citing a lack of time-dependent responses in increased liver weight over the study period. This is incorrect. In this study, serum PFOA appeared to have reached steady state by four weeks into the study and the attainment of

steady state is a common observation in laboratory animals when perfluoroalkyls were administered at high doses or for extended exposure durations. This corresponded to a "saturation" status where (latter) additional PFOA administered was not absorbed efficiently. This natural occurrence does not invalidate the study data given that at every single time point of the study (four, seven, or 13 weeks post-dose), there were dose-dependent increases in serum PFOA concentrations, as well as increases in relative liver weight. In addition, based on the data reported by Loveless et al. (2006), male Sprague Dawley rats appeared to be more sensitive than male CD-1 mice in terms of higher body burden when similar PFOA doses were administered. A key strength of including Perkins et al. (2004) study data reflects on the dietary PFOA exposure route used in the study design, which is a major pathway considered by the Department as potential PFOA source of exposure. Therefore, the Department should also consider evaluating the longer-term rat data from Perkins et al. (2004) for its PFOA assessment. (42)

RESPONSE TO COMMENTS 82, 83, AND 84: The increased relative liver weight data from Abbott et al. (2007) did not meet the DWQI (2017a) criteria for use as the basis for risk assessment because serum PFOA levels were not measured until three weeks after dosing ended. As stated in DWQI (2017a), "studies providing serum PFOA data at the end of the dosing period are most appropriate for dose-response evaluation in risk assessment, because serum levels are highest at this time point and thus represent the maximum internal doses that could have caused the observed effect." The use of serum PFOA data from a timepoint several weeks

after dosing ended potentially underestimates the exposure level that caused the effect, resulting in an overly stringent risk assessment.

The commenter's interpretation of the reason that Perkins et al. (2004) was not selected by DWQI (2017a) is not accurate. Perkins et al. (2004) was not selected as the basis for risk assessment because the observed dose-response curves for increased relative liver weight were almost identical at four, seven, and 13 weeks. This was cited by DWQI (2017a) as evidence supporting the use of shorter-duration studies of relative liver weight as the basis for risk assessment, particularly because the use of serum PFOA levels as the dose metric eliminates uncertainty about whether serum PFOA levels are increasing with longer exposure duration.

The mouse data from Loveless et al. (2006) was selected as the basis for risk assessment instead of the rat data from Perkins et al. (2004) because there was a more definitive dose-response for increased liver weight over the dose range used by Loveless et al. (2006) and because increased relative liver weight was strongly correlated with PPAR-alpha activity in rats dose with PFOA in Perkins et al. (2004) at weeks four and seven, while this was not the case in mice in Loveless et al. (2006), as discussed above.

85. COMMENT: We applaud New Jersey for recognizing altered mammary gland development as an adverse health effect associated with PFOA and agree that it can lead to a number of health effects, including difficulty in breastfeeding and an increase in susceptibility to breast cancer later in life—for these reasons, we urge New Jersey to base its MCLG for PFOA on altered mammary gland development directly. There is sufficient evidence to support this

conclusion: A workshop of experts in mammary gland biology and risk assessment also concluded that changes in mammary gland growth and differentiation, including changes in developmental timing, are a health concern. Furthermore, three human studies have reported that maternal PFOA exposure is associated with decreased duration of breastfeeding. (37)

86. COMMENT: The Department should use the toxicity endpoint of delayed mammary gland development as the most sensitive endpoint in the PFOA MCL derivation. This effect was shown in nine different studies (DWQI 2016). Delayed mammary gland development is concerning since adverse effects related to delayed mammary gland development persist into adulthood and become permanent. (9)

RESPONSE TO COMMENTS 85 AND 86: The Department agrees that the effects of low doses of PFOA on mammary gland development in mice are relevant to humans. As discussed below, delayed mammary gland development from perinatal exposure is the most sensitive systemic endpoint for PFOA that provides serum PFOA data that can be used for the dose-response analysis needed for reference dose development. It is a well-established toxicological effect of PFOA that is considered to be adverse and relevant to humans for the purposes of risk assessment. However, to the knowledge of the Institute and Department, a reference dose for delayed mammary gland development has not previously been used as the primary basis for health-based drinking water concentrations or other human health criteria for environmental contaminants.

87. COMMENT: We disagree with the Institute that mammary gland development is a robust endpoint for PFOA related toxicity in laboratory animals and there were a number of specific concerns that warrant careful consideration before using data from Macon et al. (2011) for risk characterization. (42)

88. COMMENT: The study by Macon et al. (2011) was flawed in several important aspects of study design and had numerous instances of inappropriate data interpretation. The authors failed to consider all aspects of biology and rather than scope out the best objective endpoints for the assessment, the study gave very few quantitative measures. The authors attributed various phenotypic consequences (that is, reduction in mammary gland development) to the direct effects of PFOA. Alternative interpretations suggest that PFOA may be affecting mammary gland function in the lactating dams. Without any supporting evidence for maternal well-being, the data presented by Macon et al. are built on a great deal of speculation with a lack of definitive reproductive data combined with a lack of quantitative mammary gland analysis. The fact that the effects of PFOA on mammary gland development cannot be consistently described and quantified in all mouse models brings into question the biological significance of this phenotype as described, and its relevance to human health is unclear. (42)

RESPONSE TO COMMENTS 87 AND 88: Delayed mammary gland development in mice from developmental exposures is a well-established endpoint for PFOA toxicity. This effect has been reported in nine separate studies presented in five publications; all of these studies are thoroughly reviewed in DWQI (2017a), including those that reported effects at low doses (White et al., 2011; Macon et al., 2011; Tucker et al., 2011). Only one study (Albrecht et al.,

2013) did not find an effect on mammary gland development, as discussed further below. Histological changes in the mammary glands of exposed offspring persisted until adulthood and were considered permanent. Delayed mammary gland development in mice occurs in a dose-related fashion, and there is no information indicating it is not relevant to humans. For these reasons, the Department considers delayed mammary gland development from exposures to PFOA to be a sensitive and relevant endpoint for toxicity.

89. COMMENT: While the study by Macon et al. (2011), used by the Institute as the basis for an alternative reference dose, observed a delay in mammary gland development in CD-1 mice, the results in other mouse studies are equivocal and support a PPAR-alpha-activated mechanism of questionable relevance to humans. Albrecht et al. (2013) did not find alterations in mammary gland development in offspring of wild type, PPAR-alpha-null, or PPAR-alpha humanized mice following in utero exposure to PFOA. In a multi-generational study in CD-1 mice, moreover, no clear dose-response was reported and the investigators noted that the delay in mammary gland development did not appear to affect lactational support based on normal survival and growth of the second generation (F2) offspring. (23, 27, and 41)

90. COMMENT: An MCL based on delays in mammary gland development was calculated and is used to support application of additional uncertainty factors in the calculation of the MCL based on increased liver weight. In its development of a Minimal Risk Level for PFOS, the ATSDR noted “[r]eproductive and developmental toxicity studies have identified very low LOAELs of ≥0.0024 mg/kg/day for delays in mammary gland development in dams and offspring …

however, the mammary gland effect did not result in an adverse effect on lactational support at maternal doses as high as 1 mg/kg/day (i.e., the effect was transient and did not adversely impact organism reproduction, development or growth), (White et al. 2011a). Given that milk production was adequate to support normal growth and survival in F2 pups, the biological significance and adverse nature of the delayed development of the mammary gland is uncertain and was not considered a suitable basis for the MRL." (24)

RESPONSE TO COMMENTS 89 AND 90: The basis of the commenters' statement that the results in mouse studies of mammary gland development other than Macon et al. (2011) "are equivocal and support a PPAR-alpha-activated mechanism of questionable relevance to humans" is unclear. As discussed in DWQI (2017a), delayed mammary gland development from prenatal/early life exposure in mice was observed in nine separate studies reported in five publications. Only one study, Albrecht et al. (2013) did not find an effect on mammary gland development. However, the Institute's consideration of this study was limited due to uncertainty whether postnatal lethality is actually significantly increased by PFOA in wild type pups; the statistical comparison used appears to be inappropriate. Therefore, the basis for the conclusion that wild type, but not humanized PPAR-alpha, mice are sensitive to developmental effects of PFOA is uncertain.

Additionally, elevated PFOA levels (up to greater than 1,000 ng/ml) were found in liver and serum from some control fetuses, pups, and dams in Albrecht et al. (2013), but no information is provided about the groups of animals that these samples came from, the number of samples with elevated PFOA concentrations, or the summary statistics (for example, means

and standard deviations) for serum levels in the control samples. Data from control animals with elevated PFOA exposures appear to have been excluded from the comparisons of endpoints of toxicity in control and treated groups. Exclusion of these data from the control animals could have affected the results of these comparisons, especially since serum levels in some of the treated groups were only a few fold higher than those in some of the controls.

Furthermore, developmental effects (other than delayed mammary gland development) observed in the same strain of mice (SV/129) in another study (Abbott et al., 2007) at lower doses (0.6 and one mg/kg/day) were not observed at the higher dose (three mg/kg/day) used by Albrecht et al. (2013). Both studies used SV/129 mice, but they were obtained from different sources, and Albrecht et al. (2013) suggest that pharmacokinetic differences in the wild type mice from the two different sources may explain the differences in effects of PFOA in these mice in the two studies. Although not mentioned by Albrecht et al. (2013), the serum levels in wild type pups at which no developmental effects occurred were higher than the serum levels in wild type pups at which delayed eye opening and postnatal mortality were reported by Abbott et al. (2007). Finally, the serum PFOA data for wild type dams on postnatal day 20 appear to be inconsistent within the publication. These general issues with this study create uncertainty about its conclusions related to mammary gland development. Regarding the functional significance of delayed mammary gland development, the only study which evaluated nursing capability was White et al. (2011b).

For the reasons provided by DWQI (2017a), including that the ability to provide nutritional support to offspring has been evaluated in only one study, the DWQI (2017a)

concluded that "the available toxicological information is not sufficient to make conclusions about the effects of developmental exposure to PFOA on lactational function." Also, as noted in DWQI (2017a) as possibly relevant to this issue, three human studies suggest that maternal exposure to PFOA may be related to shorter duration of breastfeeding. Additionally, developmental exposure to PFOA consistently caused structural changes observed through microscopic evaluation of whole mounts of mammary glands, and histological changes in the mammary glands persisted and were considered permanent in the only study in which developmentally exposed mice were followed through adulthood. Such structural changes, particularly when permanent, are considered adverse for the purposes of risk assessment, regardless of whether functional effects are detected.

Uncertainty Factors

91. COMMENT: While New Jersey recognized mammary gland development as the most sensitive endpoint and even calculated a reference dose based on this endpoint, it did not use this endpoint to calculate its MCLs. Instead it used a reference dose for increased liver weight. This was a mistake. Delayed mammary gland development can lead to a number of health effects including difficulty in breastfeeding and an increase in susceptibility to breast cancer later in life. If the reference dose for mammary gland development had been used, New Jersey's MCL for PFOA, for example, would be notably lower, coming in at less than one part per trillion. (37)

92. COMMENT: The Institute chose not to use delayed mammary gland development reference dose as the basis for a recommended Health-based MCL, not because of uncertainty about the scientific validity of doing so, but rather because of a lack of precedent to use this endpoint as the primary basis for health-based criteria for environmental contaminants. Instead, an additional 10 UF was applied to an unrelated endpoint (increased liver weight that forms the basis of the MCL derivation) to compensate. (9)

93. COMMENT: While the Institute applied an extra uncertainty factor of 10 to protect against more sensitive effects like altered mammary gland development, an uncertainty factor of 10 is insufficient to protect against this health effect. Indeed, if New Jersey's reference dose for mammary gland development had been used, New Jersey's MCLG for PFOA would be less than one part per trillion. (37)

94. COMMENT: In its analysis, the Institute includes a composite or total uncertainty factor (UFtotal) of 300 in the derivation of the MCL for PFOA. The proposed UFtotal includes a 10-fold uncertainty to account for variability in susceptibility across the human population (UFH), a factor of three to account for the toxicodynamic differences between humans and animals (UFA), and an additional factor of 10 for database uncertainties (UFD). The UFtotal is applied to adjust the human-equivalent dose (HED) to add conservatism to the calculation of a reference dose from which the MCL is calculated. According to its summary report, the Institute applied a UFD of 10 to account for "sensitive effects that are not otherwise considered," specifically citing mammary gland development and hepatic toxicity not associated with liver weight. According to the USEPA, the UFD is intended to account for the potential for deriving an under-protective

reference dose as a result of an incomplete characterization of the chemical's toxicity. In addition to identifying toxicity information that is lacking, review of existing data may also suggest that a lower reference value might result if additional data were available. Consequently, in deciding to apply this factor to account for deficiencies in the available data set and in identifying its magnitude, the assessor should consider both the data lacking and the data available for particular organ systems, as well as life stages. A UFD is generally applied when reproductive and developmental toxicity studies are missing since they have been found to provide useful information for establishing the lowest NOAEL. If the reference dose is based on animal data, a factor of three is often applied if either a prenatal toxicity study or a two-generation reproduction study is missing, or a factor of 10 may be applied if both are missing.

The reproductive and development databases for PFOA are robust, however, and do not suggest the need to account for an incomplete characterization of toxicity. As discussed above, evidence of mammary gland developmental effects in mice are equivocal and support a PPAR-alpha-activated mechanism of questionable relevance to humans. Similarly, the Institute's concern about liver toxicity is misplaced in light of the available epidemiological evidence and the likely contribution of PPAR-alpha activation. (23, 27, and 41)

RESPONSE TO COMMENTS 91, 92, 93, AND 94: DWQI (2017a) has considered issues related to the database uncertainty factor to protect for more sensitive developmental effects, including delayed mammary gland development and low-dose liver toxicity. As discussed in DWQI (2017a) and DWQI (2017b), the Institute's Health Effects Subcommittee decided not to use delayed mammary gland development as the primary basis for the quantitative risk assessment

because there is no precedent for doing so. Delayed mammary gland development from perinatal exposure is the most sensitive systemic endpoint for PFOA that provides serum PFOA data that can be used for the dose-response analysis needed for reference dose development. It is a well-established toxicological effect of PFOA that is considered to be adverse and relevant to humans for the purposes of risk assessment. However, to the knowledge of the Institute and the Department, a reference dose for delayed mammary gland development has not previously been used as the primary basis for health-based drinking water concentrations or other human health criteria for environmental contaminants.

Additionally, limited data regarding the effects of developmental exposure to PFOA on lactational function are available. For example, a single study assessed this endpoint in mice and did not observe a decrease in body weight of offspring nursed by dams with earlier developmental exposure. However, at least three human studies from different locations found associations of maternal PFOA exposure with decreased duration of breast-feeding, suggesting a potential effect of PFOA on human mammary gland function (Fei et al., 2010; Romano et al., 2015; Timmermann et al., 2016). Because the use of this endpoint as the basis for human health criteria is a currently developing topic, the Institute in DWQI (2017a) decided not to recommend a Health-based MCL with the reference dose for delayed mammary gland development as its primary basis. However, the occurrence of delayed mammary gland development and other effects (such as persistent liver toxicity following developmental exposure; Quist et al., 2015) at doses far below those that cause increased relative liver weight

(the endpoint used as the primary basis for the recommended Health-based MCL) clearly requires application of an uncertainty factor to protect for these more sensitive effects.

According to the current USEPA risk assessment guidance, an uncertainty factor should be incorporated into a reference dose to account for effects that may be more sensitive than the effect used as its primary basis "if there is concern that future studies may identify a more sensitive effect, target organ, population, or lifestage" (USEPA, 2016). The primary basis for the PFOS reference dose is increased liver weight, and DWQI (2017a) concluded that it is most appropriate to account for effects on mammary gland development and other low-dose developmental effects, such as persistent liver toxicity from developmental exposures (Quist et al., 2015), through incorporation of the default uncertainty factor of 10 into the reference dose. The Department notes that these low-dose developmental effects (delayed mammary gland development and persistent liver toxicity) occur at much lower doses than the developmental effects used as the basis for the USEPA Health Advisory (delayed ossification and accelerated male puberty) for PFOA.

As discussed above, the Department agrees with the Institute's conclusions that delayed mammary gland development caused by PFOA in mice is considered to be relevant to humans, and the persistent liver toxicity from developmental exposure to PFOA in mice (Quist et al., 2015) is not dependent on PPAR-alpha activation.

95. COMMENT: The Institute allocated a database uncertainty factor of 10 to account for "sensitive effects that are not otherwise considered," specifically citing mammary gland

development and hepatic toxicity not associated with liver weight. This decision lacks a logical scientific basis and contrary to USEPA guidance.

USEPA guidance provides that the uncertainty factor for database uncertainty is intended to account for the potential for deriving an under-protective toxicity value when there is an incomplete characterization of the chemical's toxicity. In contrast, the toxicology database for PFOA is quite comprehensive. The convoluted action taken by the Institute for the allocation of an uncertainty factor of 10 is contrary to USEPA guidance

The Institute attempts to create an aura of database uncertainty by focusing on mammary gland development concerns. In fact, the Institute derived a BMDL for PFOA and mammary gland development findings based on the study reported by Macon et al. (2011). It elected, however, not to proceed further for MCL derivation because this endpoint "has not previously been used as the primary basis for health-based drinking water concentrations or other human health criteria." Therefore, it is improper for the Institute to include an uncertainty factor because there are "more sensitive effects that are not otherwise considered." when it had considered mammary gland effects. Furthermore, the effect of PFOA exposure on mammary gland development in laboratory mice have not been consistently described in published literature. Contrary to the Institute's assertion, it is not a robust endpoint. The study by Macon et al. (2011) used by the Institute had numerous technical deficits that preclude a meaningful interpretation in addition to its biological significance and relevance to human health.

Accordingly, the UF should be reduced to three. Changing this parameter would increase the proposed PFOA MCL to 0.042 µg/L. (42)

RESPONSE: The rationale for the inclusion of an uncertainty factor of 10 to account for more sensitive developmental effect is provided in the response to the other comments on this uncertainty factor addressed elsewhere in this notice of adoption. Regarding the additional specific points mentioned in this comment, the conclusion that an additional uncertainty factor to protect for more sensitive developmental effects should not be applied because DWQI (2017a) conducted a formal evaluation and developed a reference dose for this effect is not logical. The Department has determined that an uncertainty factor of 10, rather than the lower value of three, is not excessively conservative because the BMDL and reference dose referenced in DWQI (2017a) are about 200-fold lower for delayed mammary gland development than for increased liver weight.

As discussed in the response to prior comments, the Institute in DWQI (2017a) concluded that delayed mammary gland development caused by PFOA in mice is well-established and relevant to humans.

Adverse Effects

96. COMMENT: In March 2017, the Institute recommended to the Department an MCL of 14 ng/L for PFOA and 13 ng/L for PFOS, which was based on deliberations of its Health Effects Subcommittee. According to this Subcommittee, as stated by the Department, exposure to PFOA has been associated with health effects including increased cholesterol, increased liver

enzymes (an indication of liver damage), decreased vaccine response, decreased birth weight, and testicular and kidney cancer. Unfortunately, the Subcommittee on Health Effects chose to not consider, discount, or not have available for review (due to not considering publications after March 2015), important information that suggests these reported associations are highly likely to be noncausal. We believe the following research that was not reviewed by the Institute's Health Effects Subcommittee due to either timing of publication or incorrect data interpretation, is germane to the conclusion of a misunderstanding of the biological associations reported with PFOA (or PFOS) and, therefore, impacts the proposed MCLs through the misguided attempt by the Institute's Health Effects Subcommittee to apply unjustified uncertainty factors for PFOS, as well as incorrectly calculates the MCL for PFOA. A brief discussion follows for each of the epidemiologic associations listed above. (42)

RESPONSE: The Department notes that an association refers to a statistical association between exposure and a health effect, but does not address whether the exposure caused the health effect. DWQI (2017a) considered all information available at the time of its review of PFOA in making its conclusions about the associations and evidence for causality for each of the health endpoints mentioned. Based on a review of the primary scientific literature, the Institute in DWQI (2017a) concluded that the evidence for an association with PFOA exposure was strongest with increased serum cholesterol, the liver enzyme alanine aminotransferase (ALT), and uric acid. DWQI (2017a) also concluded that there was some level of evidence of an association between PFOA exposure and decreased antibody concentrations following vaccination, low-density lipoproteins (LDL), the liver enzymes gamma-glutamyl transferase

(GGT) and aspartate aminotransferase (AST), bilirubin, liver disease, and thyroid disease. Of these health effects, DWQI (2017a) concluded that the epidemiological evidence supports multiple criteria for a causal relationship between PFOA and both serum cholesterol and ALT. For some other epidemiological endpoints (decreased birth weight, testicular and kidney cancer), the Institute's conclusion that there is evidence for an association was based on evaluation of reviews conducted by other authoritative bodies. For these endpoints, the Institute did not evaluate whether or not the epidemiological evidence supports criteria for a causal relationship.

The intent of the comment stating that the interpretation of the epidemiology data by the Institute's Health Effects Subcommittee impacts the PFOA and PFOS MCLs and resulted in applying "unjustified uncertainty factors for PFOS as well as incorrectly calculat[ing] the MCL for PFOA" is not clear since the quantitative basis for the Health-based MCL for PFOA recommended by DWQI (2017a) is data from animal studies. While the Institute in DWQI (2017a) concluded that "the human epidemiological data support the use of a public Health-protective approach in developing a Health-based MCL recommendation based on animal toxicology data," the Health-based MCL for PFOA is not dependent on a conclusion that there is an association, either causal or non-causal, for PFOA and any specific human health effect.

97. COMMENT: Several studies of ALT and PFOA should be carefully evaluated because of the large study population with drinking water exposure to PFOA (Gallo et al., 2012, Darrow et al., 2016); they were occupational (Olsen et al., 2007; Sakr et al., 2007a; Sakr et al., 2007b;

Olsen et al., 2012) or they are experimental (Convertino et al.). Taken together, they do not indicate "liver damage" as defined by a two- to four-fold increase in ALT. Even if statistically significant, the increase in ALT is small and PFOA explains only a small percent of the variation.

Post and Gleason overinterpret the epidemiological data as it relates to ALT and PFOA and the use of the phrase "liver damage" is misunderstood. ALT is a "leakage" enzyme and may be increased due to necrosis, injury, or repair. Increases of two- to four-fold in rodents, canines, non-human primates, and humans indicate hepatic injury. As defined by Hall et al. (2012), "[b]ased on the recommendations of regulatory authorities ... increases in ALT activity of two-to threefold should be considered as indicated of 'hepatocellular damage.'"

Those studies that have suggestion of an elevation of ALT remain well within the expected physiologic range of measuring ALT. Using the term 'damage' in this context is, therefore, misleading. It is also possible to have quite modest but statistically significant increases in ALT that are not toxicologically relevant (Cattley and Cullen, 2013). Finally, it should be noted that the human half-life of ALT is approximately 47 hours (Hall et al. 2012). This is often not mentioned when cohort studies are conducted examining estimated (modeled) serum PFOA concentrations over time when there is only a single ALT value reported.

Finally, it should also be noted that nonalcoholic fatty liver disease is the most common cause of mild elevations of liver enzymes (Gianni et al. 2005). Several studies are worthy of careful evaluation as they relate to ALT and PFOA either because of the size of the population studied that was exposed to PFOA through the drinking water, they were occupational populations, or the study was experimental and based on a phase 1 clinical trial in humans

designed to ascertain the maximum tolerated dose of PFOA (ammonium salt). Two studies were from the C8 Science Panel (one cross-sectional (Gallo et al., 2012), the other longitudinal based on an estimated cumulative serum (ng/mL-year) model (Darrow et al., 2016), four are occupational studies (two cross-sectional (Olsen et al. 2007; Sakr et al., 2007a) and two longitudinal (Sakr et al., 2007b; Olsen et al., 2012), and one experimental phase 1 clinical trial (Convertino et al., 2018). Collectively, these studies do not suggest "liver damage" – see above - (two- to four-fold increase) as measured by ALT associated with increasing serum concentrations of PFOA.

Although some studies' regression coefficients for PFOA may be statistically significant, the percent variation explained of ALT by PFOA is minimal, at best, and the elevation of ALT very modest (generally an increase of one to three IU ALT). Nor is there any evidence of increased mortality from increased liver disease in epidemiologic analyses of community-based exposure to PFOA (Darrow et a. 2016) or in occupational cohort mortality studies (Steenland et al., 2012; Raleigh et al., 2014).

A study of genetically engineered mice enabled to mimic human lipid metabolism observed an increase in ALT (U/l) only at the highest concentration, which approximated a serum concentration 144,000 ng/mL PFOA (Pouwer et al., 2019). (42)

98. COMMENT: Increased relative liver weight is a common effect of PFOA in animal studies that has been reported to occur at lower levels of exposure than those causing effects on other organ systems. Extrapolation of liver effects seen in animals to humans must be approached with caution, however, in light of the conclusions of the C8 Health Project and

recent human data reported by Convertino et al. (2018) and strong evidence for rodent-specific adaptive responses.

The C8 Health Project is a large epidemiological study conducted in communities surrounding a manufacturing facility in Parkersburg, West Virginia that used PFOA from the 1950s until 2002. The study included over 32,000 adult residents and facility workers. The Science Panel formed as part of this project concluded that "there is not a probable link between exposure to C8 (also known as PFOA) and liver disease."

The conclusions of the C8 Science Panel are supported by the recent work of Convertino et al. who reported no differences in clinical measures (including triglycerides, urea, glucose, AST, GGT, alkaline phosphatase, total bilirubin, fibrinogen, PTT, and aPTT) at weekly PFOA doses as high as 1,200 milligrams (about 16 milligrams/kilogram (mg/kg)), among a sensitive sub-population of cancer patients. The authors concluded that the disparity between animal and human liver endpoint studies, emphasizing a lack of risk of human enlarged liver, fatty liver, or cirrhosis, can be attributable to mode of action differences. (23, 27, and 41)

RESPONSE TO COMMENTS 97 AND 98: All of the epidemiological studies of PFOA and its association with alanine transaminase (ALT) listed and summarized above, except Convertino et al. (2018), were reviewed in DWQI (2017a), including individual tables for each of the studies. Regarding the magnitude of the increase in ALT associated with PFOA, the Department stated in its comments on the draft ATSDR (2018) Toxicological Profile:

> We disagree … that, even if the evidence for association is strong, small changes such as have been found for … serum liver enzymes are not likely to be

> "biologically relevant" because they are "within normal limits." Exposure to environmental contaminants at levels that, based on the weight of evidence, are likely to cause these effects should not be dismissed as being of no public health concern … Within a large population, relatively small population–level changes in parameters such as serum liver enzymes (which are markers of liver damage) result in a shift in the overall distribution of values such that the numbers of individuals with clinically abnormal values is increased. Also, small changes in a clinical biomarker that has been measured may be an indicator of other effects which were not assessed.

The Department notes that clinical websites that recommend use of ALT in evaluating potential liver disease, including American Family Physician - Nonalcoholic Fatty Liver Disease: Diagnosis and Management; NASPGHAN Clinical Practice Guideline for the Diagnosis and Treatment of Nonalcoholic Fatty Liver Disease in Children: Recommendations from the Expert Committee on NAFLD (ECON), and the North American Society of Pediatric Gastroenterology, Hepatology and Nutrition (NASPGHAN), do not mention that a two- to three-fold increase in the ALT level is required to indicate increased risk.

The Department also notes that the National Toxicology Program (NTP, 2019) report on 28-day rat studies of PFOA and other perfluoroalkyl carboxylates state that PFOA and the other compounds tested caused "mild (≤onefold) increases in serum ALT, AST, and/or [succinate dehydrogenase] SDH activities …" NTP (2019) does not mention that a several-fold increase is needed to indicate hepatic damage, but rather states that "… these enzymes are biomarkers of

hepatocellular injury and their increases correlate with the observed hepatocellular necrosis, as well as the finding of cholestasis as hepatocellular injury can occur secondary to cholestasis." It is unclear how the relatively short (47 hours) human half-life of ALT could affect the observed associations of ALT with PFOA. Similarly, it is unclear how the fact that nonalcoholic fatty liver disease is the most common cause of mild elevations of liver enzymes, as mentioned by the first commenter, could affect the association of PFOA and ALT. Regarding Convertino et al. (2018), as discussed in the response to the a prior comment regarding effects on plasma cholesterol, limitations of this study noted by the study authors include small sample size, very short length, limited power of study, and potential altered metabolic state of the study group consisting of late-stage cancer patients who had failed standard therapy. Observations in these patients cannot be considered relevant to healthy individuals because their nutritional and physiological status was likely affected by their severe illness.

Regarding association of PFOA and liver disease, a recent study (Girardi and Merler, 2019) of occupationally exposed workers with very high serum PFOA levels (geometric mean: 4,048 ng/ml; range 19-91,900 ng/ml) found an association of PFOA with hepatic cancer and hepatic cirrhosis. While noting that this was "a small observational study with limited control over confounding factors," the authors conclude "toxicological studies on PFOA and PFOS provide support for causality for the observed association with the risk for liver cirrhosis and liver cancer."

Regarding the study of mice that mimic human lipid metabolism (Pouwer et al., 2019), the relevance of the increase in ALT in this study in this specific strain is unclear. DWQI (2017a)

provides a thorough review of increases in ALT caused by PFOA in numerous studies of monkeys, wild type mice, PPAR-alpha null mice, and rats.

99. COMMENT: Studies by regulatory bodies and expert health panels have shown inconsistent findings that do not support an association between PFOA and a reduced vaccine response in humans. (42)

RESPONSE: While aspects of the studies mentioned by the commenter focus on potential associations of PFOA with incidence of infectious disease, the Department did not make any conclusions about associations of PFOA with this endpoint. Additionally, the Institute in DWQI (2017b) stated "[a]lthough the database for antibody response following vaccination is currently not conclusive enough to use as the primary basis for risk assessment [for PFOA], it supports the need for a protective approach in the risk assessment based on animal data."

100. COMMENT: While the Institute's Health Effects Subcommittee was aware of a phase 1 dose-escalation clinical trial study that administered PFOA to cancer patients due to the anti-tumorigenic properties of PFOA, it did not cite the existence of this study because it was only presented as an abstract (Macpherson et al., 2010) published in the journal Clinical Oncology. According to the Institute's Health Effects Subcommittee, the abstract was based on a clinical conference and was not peer-reviewed in the literature nor was there sufficient information in the abstract provided to even understand study design. Therefore, the Subcommittee chose to not consider the abstract. On the other hand, it should be noted that the draft 2018 ATSDR

(https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf) report discussed this abstract. Regardless, such an explanation to cite or not cite an abstract is now moot as the findings from this phase 1 clinical trial study have been publicly available in the peer reviewed scientific literature since May 2018 (Convertino et al., 2018). To the best of our knowledge, this is the only experimental study of PFOA conducted in humans. All other epidemiologic research reported in the scientific literature is observational - whether it is from the general population, communities exposed to PFOA, or occupational. Aas noted by the Institute's Subcommittee on Health Effects, most of these studies were cross-sectionally designed studies, which means temporality between exposure and outcome cannot be determined. Thus, the Department needs to understand not only the phase 1 clinical trial study results because of its unique study design with direct exposure to humans. The Department also needs to consider an important toxicological study recently published in the same premier toxicological journal (Toxicological Sciences) where a genetically engineered mouse model designed to mimic human lipoprotein metabolism was used to assess administered PFOA (ammonium salt) dosages that resulted in environmental, occupational, and toxicological (similar to phase 1 clinical trial) concentrations of PFOA reported in humans (Pouwer et al., 2019). Reduction of serum cholesterol in mice fed a Western diet was only observed at toxicological concentrations that were similar in magnitude to the phase 1 clinical trial in humans. Findings from these two studies indicate the epidemiologic observations are associative rather than causal. (42)

101. COMMENT: The Department should evaluate a 2018 published phase 1 clinical trial data with (ammonium) PFOA in 49 human subjects. As a human clinical trial with controlled doses,

the study does not have the substantial uncertainties inherent in extrapolating from effects in animals to humans. Contrary to the Department's assertion and the European Food Safety Agency's (EFSA) preliminary conclusions regarding purported associations between PFOA and cholesterol, these phase 1 clinical data show that at concentrations that are several orders of magnitude higher than those reported in the general population, PFOA unequivocally reduced the cholesterol. But these observations are inconstant with the observational epidemiological associations showing higher cholesterol with markedly lower PFOA concentrations. (51)

RESPONSE TO COMMENTS 100 AND 101: The Department has reviewed Convertino et al. (2018) and concluded that it is not useful in the evaluation of potential health effects of chronic drinking water exposure to PFOA in the general population. The paper by Convertino et al. (2018) describes a Phase 1 trial that was conducted to assess the chemotherapeutic potential of PFOA and focuses on the establishment of a maximum tolerated dose in the setting of clinical therapy. This study involved experimental administration of extremely high doses of PFOA to advanced cancer patients with solid tumors (types not specified) in whom other treatments had failed. Limitations of this study include small sample size, very short length, limited power of study, and potential altered metabolic state of study group consisting of late-stage cancer patients. Observations in these patients cannot be considered relevant to healthy individuals because their nutritional and physiological status was likely affected by their severe illness. For these reasons, the results of this study do not negate or call into question the consistent associations of PFOA with increased cholesterol in the general population at environmentally relevant exposures.

An earlier abstract (Macpherson et al., 2010) regarding this study was reviewed by the Institute in DWQI (2017b). Convertino et al. (2018) states that "no more than one subject showed DLT [drug related toxicity] at any dose." However, as mentioned by DWQI (2017b), Macpherson et al. (2010) reported that one of the patients dosed with 600 mg weekly (about 1.2 mg/kg/day, assuming 70 kg body weight) experienced drug related toxicity (DLT) consisting of "grade 5 renal failure and transaminitis" (indicative of liver damage), and these effects were noted as "possibly drug related" in the abstract. DWQI (2017b) noted that the information provided by Macpherson et al. (2010) indicates the potential for PFOA to cause renal and hepatic toxicity in humans. It is unclear why the observation of "possibly drug related" kidney and liver toxicity reported by Macpherson et al. (2010) is not mentioned by Convertino et al. (2018).

More importantly, observations at the extremely high exposures in this study are not relevant to exposures at environmentally relevant concentrations. For example, the median serum PFOA level in the C8 Study (see http://www.c8sciencepanel.org/index.html) of communities exposed to PFOA in contaminated drinking water was 28 parts per billion (ppb); the median serum PFOA level in U.S. general population is currently approximately two ng/ml [2 ppb]). Convertino et al. (2018) report decreased serum cholesterol only in the three categories of patients with the highest exposures (above approximately 200,000 ng/ml [200,000 ppb] plasma PFOA), but not in the seven lower exposure categories that also had extremely high plasma PFOA levels of up to approximately 200,000 ng/ml (200,000 ppb). The plasma PFOA concentrations at which cholesterol was reported to be decreased by Convertino

et al. (2018) are similar to the serum PFOA levels at which cholesterol is decreased in animal studies, presumably through activation of PPAR-alpha. As discussed in DWQI (2017a), drugs used to reduce serum cholesterol in humans act through activation of PPAR-alpha. The decreased cholesterol at these extremely high plasma PFOA concentrations in humans is consistent with the action of these drugs, while the increased cholesterol at serum PFOA levels several orders of magnitude lower likely occurs through a different mechanism. Further, serum cholesterol was decreased in mice fed a high fat diet in Tan et al. (2013) and Rebholz et al. (2016). Additionally, a recent 28-day study of PFOA in rats that was conducted by the National Toxicology Program (NTP, 2019) reported significantly increased serum cholesterol in female rats while serum levels were unchanged or decreased in the dosed groups of male rats. It is noted that the levels of PFOA in the serum of female rats in which cholesterol was decreased were lower than in any of the dosed groups of male rats.

Finally, as stated by the Minnesota Department of Health in its development of a chronic drinking water guideline for PFOA (Minnesota Department of Health, 2018):

> Convertino et al. (2018) describes PFOA as an agent that causes endoplasmic reticulum stress, acts as a fatty acid mimetic, and an inhibitor of PIM kinases in tumor cells. While tumor cells may be more susceptible to these effects, these processes are also involved in normal functions within the body. For example, heightened endoplasmic reticulum stress response has been implicated in insulin resistance and inflammatory processes. Chemotherapeutic agents are typically selected based on their biological activity and cytotoxicity (e.g., killing) towards

tumor cells. The purported use of PFOA to kill living cells, even tumorigenic cells, conflicts with the idea that PFOA does not alter biological systems.

102. COMMENT: The Institute's Health Effects Subcommittee mentioned two toxicological studies, Tan et al., 2013 and Rebholz et al., 2016, which reported wild-type mice fed a Westernized (high fat) diet containing PFOA resulted in increased cholesterol. However, this Subcommittee appeared not to appreciate the fact that rodent lipoprotein metabolism is characterized by fast clearance of apolipoprotein B(apoB)-containing lipoproteins and the absence in the rodents of cholesteryl ester transfer protein (CETP) that results in a higher proportion of high density lipoprotein (HDL)-cholesterol relative to LDL cholesterol in the rodent. In contrary, humans have a much higher proportion of LDL-cholesterol relative to HDL-cholesterol due to the presence of CETP, which results in transfer of cholesterol sterol from HDL-cholesterol to the much slower clearing apoB-containing lipoproteins in exchange for triglycerides. Therefore, wild-type mice are not the most suitable species to study human lipid metabolism in addition to the relevance and translatability of their findings to the human situation. It is premature for the Institute, inferring from the study conclusion by Tan et al. and Rebholz et al., to conclude that PFOA can cause hypercholesterolemia.

Instead, the APOE*3-Leiden.CETP mouse model has been commonly used to study the effect of pharmaceuticals on lipid metabolism and atherosclerosis for human evaluation. The APOE*3-Leiden.CETP mouse model was designed to mirror human lipoprotein metabolism with

incorporation of cholesterol ester transfer protein expression and a delayed apoB clearance that occurs in humans.

Pouwer et al. (2019) used the APOE*3-Leiden.CETP mouse model to study the effect of PFOA on plasma cholesterol and triglyceride metabolism at concentrations relevant to humans relative to environmental, occupational, and toxicological (above phase 1 clinical trial) plasma PFOA concentrations. The other objective was to elucidate the mechanisms for the effects reported. The data reported in this study are consistent with the findings from the phase 1 clinical trial in humans that demonstrated high serum or plasma PFOA concentrations result in lower cholesterol levels. (42)

RESPONSE: As is the case for the human data from Convertino et al. (2018), the observation by Pouwer et al. (2019) that serum cholesterol levels were decreased in the highest dose group of mice that had serum PFOA levels of greater than 90,000 ng/ml, comparable to those in the clinical trial in advanced cancer patients (Convertino et al., 2018), is not relevant to the much lower serum/plasma PFOA levels (far below 100 ng/L) associated with increased cholesterol in the general population and communities with PFOA-contaminated drinking water. Decreased cholesterol at similar serum PFOA levels has also been observed in wild type mice that do not have humanized lipoprotein metabolism, as found in Loveless et al. (2006), while no effect on serum cholesterol was observed in the mice in the two lower dose groups that had much lower serum PFOA levels (less than 100 ng/L and 1,300 through 1,500 ng/L) in Pouwer et al. (2019). The decreased cholesterol at these higher plasma PFOA concentrations in both mice and humans is consistent with a mechanism involving activation of PPAR-alpha in both species,

consistent with the action of drugs that reduce cholesterol in humans through PPAR-alpha activation. As stated in the responses to prior comments regarding Convertino et al. (2018), the increased cholesterol in humans at serum PFOA levels several orders of magnitude lower than those at which cholesterol is decreased in both humans and mice likely occurs through a different mechanism.

103. COMMENT: Another cholesterol-related paper worthy of consideration by the Department is Vanden Heuval (2013). Vanden Heuval (2013) was a commentary to the premise offered by Fletcher et al. (2013), a member of the C8 Science Panel, who suggested that exposure to PFOA created a “hypercholesterolemic environment.” As reviewed by Vanden Heuval (2013), a paper that was not cited by the Institute’s Subcommittee on Health Effects, reverse cholesterol transport and cholesterol efflux involves HDL to stimulate the efflux of cholesterol from peripheral tissues, transport in plasma, uptake in the liver, and then involve biliary excretion. Specific to the premise of anti-atherogenic properties of HDL, macrophage reverse cholesterol transport involves efflux of cholesterol from macrophage foam cells in the artery wall, which involves many genes including the ABC transporters in the transport of free cholesterol from the cell. In their paper, Fletcher et al. (2013) reported inverse associations between serum PFOA levels and whole blood expression level of a small subset of genes involved in cholesterol transport among 290 mid-Ohio river valley subjects. Based on these data, Fletcher et al. suggested PFOA could be increasing circulating cholesterol and decreasing cholesterol efflux from macrophages in humans. However, according to Vanden Heuval,

Fletcher et al. only examined 11 of 67 genes engaged in macrophage cholesterol efflux and reverse cholesterol transport and they did not take into account the redundancy and overlapping functions in maintaining cholesterol homeostasis. Furthermore, most laboratory studies have examined the hepatic, not extrahepatic expression sites (for example, peripheral lymphocytes and macrophages as was done by Fletcher et al.) that involve cholesterol metabolism. A mode of action was not formally studied by Fletcher et al. whereas the major target of the effects for PFOA (and PFOS) in laboratory studies involve PPARα, and likely other nuclear receptors, such as PXR and CAR. Also, when Vanden Heuval compared cholesterol metabolism genes in mouse liver to those of peripheral lymphocytes, there was considerable inconsistency. Laboratory studies have often shown (for example, see above study by Pouwer et al., 2019) decreases in cholesterol levels with exposure to PFOA, including the study by Loveless et al. (2006), which is being used by the Institute's Subcommittee on Health Effects for the point of departure for PFOA to set an MCL based on increased liver weight. (42)

RESPONSE: The Department has reviewed Vanden Heuvel (2013), which is a commentary on Fletcher et al. (2013). As stated in DWQI (2017a), Fletcher et al. (2013) investigated:

> [T]he biological plausibility of the association of PFOA and serum cholesterol … in a study of associations of serum PFOA and changes in the expression of genes involved in cholesterol metabolism. In this cross-sectional study, the expression of 13 genes involved in cholesterol metabolism (cholesterol biogenesis, peroxisome proliferation, cholesterol transport, downstream transcriptional activation of PPAR-alpha, and mobilization of cholesterol) was evaluated in

whole blood from 290 subjects from a highly exposed community (geometric mean serum PFOA, 32.2 ng/ml). Statistically significant associations between genes involved in cholesterol transport and mobilization and PFOA were found, and the affected genes differed in men and women. The authors state that these change in gene expression "appear consistent with PFOA promoting a hypercholesterolemic environment."

As noted by Vanden Heuvel (2013), Fletcher et al. (2013) evaluated only a subset of the potentially relevant genes. Nevertheless, the associations of PFOA with gene expression that were observed by Fletcher et al. (2013) are consistent with effects of PFOA on cholesterol transport that result in increased serum cholesterol levels. While not definitive without replication and additional research, these results add support to the conclusion, discussed in the responses to other comments, that there is evidence for a causal association between PFOA and cholesterol. The decrease in cholesterol at the higher plasma PFOA concentrations in the rodent studies that are mentioned by the commenter is consistent with a mechanism involving activation of PPAR-alpha. As stated in the responses to other comments regarding both Pouwer et al. (2019) and Convertino et al. (2018) above, the increased cholesterol in humans at serum PFOA levels several orders of magnitude lower than the levels at which cholesterol is decreased in both humans and rodents likely occurs through a different mechanism.

104. COMMENT: It is highly premature to suggest a low dose causal association between PFOA and cholesterol can be based on the current observational epidemiologic data. Neither the Institute's Health Effects Subcommittee, EFSA (2018) or ATSDR (2018) reviewed any study, except Butenhoff et al. (2012b), that examined a hypothesized, peer-reviewed published research mode of action study for the low dose response association observed between PFOA and cholesterol, as evidenced in several (but not all) observational epidemiologic studies. On the other hand, there is considerable evidence to indicate a mode of action for the decreased cholesterol associated with PFOA seen in both human and animal high-dosed experimental studies. Several areas of investigation have been proposed to examine potential modes of action in low dose response studies including: 1) the possibility of decreased glomerular filtration rate (GFR) with dyslipidemia that would confound an association between cholesterol and PFOA (or PFOS); 2) saturation of an underlying physiologic mechanism given the nonlinear association between PFOA (or PFOS) and cholesterol; 3) examination of shared organic anion transporters between lipids and PFOA (or PFOS) in the human in the small and large bowel, liver, and bile (as seen with uric acid salts (urate) in the kidney proximal tubules; and 4) understanding the toxicokinetics of lipoprotein maturation with the possibility of incorporation of PFOA (or PFOS) into this maturation process of these lipoproteins. Until these and other hypotheses are thoroughly investigated, the low dose response association based on observational epidemiologic data, that has been suggested by some epidemiologists to be causal, continues to remain only a hypothesis elusive of a foundational mode of action and not supported by experimental data. (42)

RESPONSE: Regarding a causal association, the Institute in DWQI (2017a) did not definitively conclude that PFOA causes an increase in serum cholesterol but rather states "[i]n summary, the epidemiologic database for serum cholesterol and PFOA, which included [20] studies, provides evidence of consistency, strength and dose-response, including some evidence of temporality. Associations with clinically defined hypercholesterolemia were reported in some studies. These findings provide evidence supporting a causal relationship between PFOA and serum cholesterol."

Butenhoff et al. (2012b) evaluated whether PFOA distributes into serum lipoproteins such that increased serum lipoproteins would cause increased serum PFOA through reverse causality, but found that this is not an explanation for the association of PFOA and cholesterol. An additional study, Fletcher et al. (2013), discussed above, also "examined a hypothesized, peer-reviewed published research mode of action study for the low dose response association observed between PFOA and cholesterol" and concluded that the observed changes in gene expression "appear consistent with PFOA promoting a hypercholesterolemic environment." While the commenter criticizes the Institute and other authoritative bodies for not reviewing other such mode of action studies, the Department is unaware of any additional studies and the commenter does not provide citations for additional studies.

Several studies, including Butenhoff et al. (2012), have explored whether PFOA and cholesterol could be jointly affected or whether the associations were due to reverse causality, that is, whether increased cholesterol resulted in increased serum PFOA levels. Butenhoff et al. (2012) examined the issues of whether PFOA distributes into serum lipoprotein fractions, and

whether increases in serum lipoproteins would result in increases in serum PFOA. They concluded that there was limited distribution to plasma lipoproteins, and that increased serum PFOA due to increased serum lipoproteins did not provide support for reverse causality.

While the Steenland et al. (2009) study found slightly lower serum PFOA levels (four percent) among individuals taking cholesterol lowering medication, as compared to those not taking medication, they noted that the statistical significance of the small difference in serum PFOA levels between the two groups was primarily a function of the large sample size and that this small difference does not support a conclusion of reverse causality.

To the Department's knowledge, the four potential modes of action listed by the commenter are speculative, with no evidence to support them. Based on the review of some of the potential factors mentioned by the commenter and other potential factors that could result in reverse causality, EFSA (2018) concluded that "it is likely that associations between serum PFOS and PFOA levels and serum cholesterol are causal, i.e. that increased levels of PFOS and PFOA cause increased levels of serum cholesterol."

105. COMMENT: Convertino et al., 2018 did not report clinically relevant changes in thyroid stimulating hormone (TSH), the primary thyroid hormones measure for clinical thyroid assessment. Again, contrary to the Institute's assertion that PFOA may depress thyroid hormone levels at very low doses based on animal studies, a high-quality study in human

volunteers show no effect on TSH thyroid hormone levels when they were exposed to much higher doses than are typically found in the general population. (51)

RESPONSE: DWQI (2017a) states, "Overall, studies evaluating thyroid hormones, TSH, and thyroid disease provide inconsistent evidence of any associations with PFOA." DWQI (2017a) also discusses evidence from animal studies, along with other factors used to evaluate potential effects of PFOA on human thyroid function, stating that "Although thyroid endpoints were not evaluated in most toxicology studies of PFOA, data from a limited number of studies, … support the biological plausibility of effects of PFOA on human thyroid function."

Additionally, NTP (2019), which was not available to DWQI (2017a), involved a comprehensive 28-day rat toxicology study of several perfluoroalkyl carboxylates, including PFOA. The doses of PFOA were 0, 0.625, 1.25, 2.5, 5, or 10 mg/kg/day in males, and 0, 6.25, 12.5, 25, 50, or 100 mg/kg/day in females. Total thyroxine (T4), free T4, and triiodothyronine (T3) were significantly decreased in all male dose groups (except T3 at 10 mg/kg/day), thyroid stimulating hormone (TSH) was significantly decreased in five and 10 mg/kg/day males, and TSH was significantly increased in all female dose groups. These recent results provide further support for the conclusion in DWQI (2017a) that effects of PFOA on human thyroid function are biologically plausible.

106. COMMENT: Gleason and Post (2019) concluded, "Based on review of the relevant information, it was concluded that confounding by GFR does not account for the major portion of the decrease in fetal growth that is associated with PFOA." Post and Gleason (2019) limited

their review to papers published by 2015. Subsequent research has shown the opinion of Post and Gleason (2019) is clearly not supported by the scientific evidence.

After a collection of studies conducted by Woodruff et al. (2014), Koustas et al. (2014), Johnson et al. (2014), and Lam et al. (2014) concluded, using a systematic review process, that "developmental exposures to PFOA adversely affects human health based on sufficient evidence of decreased fetal growth in both human and nonhuman mammalian species." A critical question arose as to whether the epidemiologic data presented on fetal growth was confounded by the maternal glomerular filtration rate (GFR). Vesterinen et al. (2015) did a systematic review of the literature based on the hypothesis that reduction in fetal growth would lead to less plasma expansion resulting in less GFR and subsequently reduced filtration of exogenous chemicals. This would result in higher concentrations in cord or maternal blood suggesting an association between lower fetal growth and higher cord or maternal blood concentrations of a chemical, including PFOA. Vesterinen et al. (2015) then asked the question whether there was an association established in the published literature between fetal growth and GFR (that is, confounding or reverse causation). Based on their review, Vesterinen et al. (2015) concluded that there was insufficient evidence to support the plausibility of a reverse causation hypothesis between exposure to environmental chemicals during pregnancy and fetal growth; however, further research would be needed to confirm or disprove this hypothesis. Post and Gleason (2019) mention the subsequent research by Morken et al. (2014) and the PBPK model/Monte Carlo simulation models by Verner et al. (2015) that definitely indicated there was an association between GFR and fetal growth, as well as the confounding between

GFR in the association between fetal growth and measured PFOA concentrations. Verner et al. concluded such confounding could be upwards of 50 percent. More importantly, and what Post and Gleason failed to recognize from the Verner et al. study, was this association between fetal growth and maternal measurement of PFOA was seen only in the second and third trimesters, not the first trimester, likely because the effect of GFR would be subsequent of plasma volume expansion that occurs in the first trimester.

A meta-analysis (not cited by Post and Gleason 2019) was published in 2017 by Negri et al. (2017). They included 16 studies in their meta-analysis. The meta-analyses by Negri et al. used both the untransformed and natural log transformations of PFOA and PFOS. For PFOA, they reported a -12.8 g untransformed birthweight (95 percent CI -23.21, -2.38) and -27.12 (95 percent CI -50.64, -3.6) g (natural log transformed) change per ng/mL PFOA. Based on their sensitivity analyses, there were stronger associations from studies conducted in Asia and significant heterogeneity was observed when the measurement of PFOA/PFOS was done later in the pregnancy or using cord blood. The latter is consistent with the simulation PBPK modelling done by Verner et al. (2015) as it relates to the potential confounding influence of maternal GFR with the timing of when PFOA is measured during pregnancy. Negri et al. also examined the laboratory animal data (results not reported here) and concluded the animal data showed similar dose-response trends but the effective serum concentrations in rodents were 100 to 1,000 times higher than in humans based on the epidemiological evidence. This led Negri et al. to increase their degree of uncertainty as to the biological plausibility of a causal relationship between PFOS exposure and lower birthweight in humans. This doubt led these

authors to suggest there might be some, not yet identified, confounding factors that lead to this spurious association of lower birth weight and perfluoroalkyl measurements in humans. For reasons not explained, Negri et al. did not reference the Verner et al. (2015) PBPK simulation study which aptly demonstrated the potential confounding of maternal GFR, the timing of measurement of PFOS during and through pregnancy and reported birth weight.

Steenland, Barry, and Savitz (Steenland et al., 2018) did recognize this distinction from the Verner et al. study. They conducted a meta-analysis of 24 epidemiologic studies – 15 more than done by Johnson et al. (2014) and eight more than Negri et al. (2017). They stratified their results as to whether the maternal PFOA concentration was measured in the first or the combined second and third trimesters. Steenland et al. reported with first trimester measurements of maternal PFOA, there was a -3.3 gram (95 percent CI -9.6, 3.0) reduction in birthweight per ng/mL55 PFOA. When PFOA was measured second/third trimester, there was a -17.8 gram reduction (95 CI -25.0, -10.6) in birthweight per ng/mL PFOA. Steenland et al. (2018) concluded “restriction to studies with blood sampling conducted early in pregnancy or shortly before conception showed little or no association such that these results are consistent with confounding and/or reverse causation being responsible for the inverse association seen in studies with low background exposure levels and blood sampling conducted later in pregnancy, when confounding and/or reverse causality are likely to be more important.” This statement clearly contradicts Post and Gleason’s (2019) opinion that confounding by GFR does not account for the major portion of the decrease in fetal growth that is associated with PFOA.

Subsequent to the Steenland et al. (2018) meta-analysis, other studies have been, and will continue to be, published regarding associations about fetal growth and the timing of measurements of PFAS, including those studies by Buck et al. (2018), Buck Louis et al. (2018), Manzano-Salgado et al. (2017), Marks et al. (2019), Meng et al. (2018), Shoaff et al. (20180, and Starling et al. (2017). The essential message from the meta-analyses conducted to date indicate physiological aspects of pregnancy, including plasma volume expansion, GFR, and when the maternal PFAS measurement was made during gestation, are critical important points to evaluate. (42)

The association reported between fetal growth (few gram reduction) per ng/mL PFOA is likely not causal but rather consistent with confounding and/or reverse causation via GFR.

RESPONSE: The Department presumes that the commenter, when referencing "Gleason and Post (2019)" and "Post and Gleason (2019)" is referring to the Department's Technical Support Document: Interim Specific Ground Water Criterion for Perfluorooctanoic Acid (PFOA, C8) (2019).

The Institute in DWQI (2017a) reviewed Morken et al. (2014) and Vesterinen et al. (2015) regarding the potential relationship between glomerular filtration rate (GFR) and birthweight, and Verner et al. (2015) regarding the potential impact of GFR on the association of PFOA and birthweight. The Institute agreed with the conclusion of Verner et al. (2015), stating "[r]ather than suggesting that GFR is the sole driver of the association between prenatal PFAS and birth weight, our results indicate that a portion of the association may be attributable to confounding by GFR …"

The Department has reviewed Negri et al. (2017). The meta-analysis in this study, which included more recent studies not considered by Johnson et al. (2014), also reported a quantitative relationship between decreased birthweight and serum PFOA and PFOS levels. Based on a qualitative consideration of both human and animal data for PFOA and birthweight, Negri et al. (2017) concluded that the causal relationship "falls in the 'likely' category." Negri et al. (2017) also concluded that the much higher serum PFOA concentrations at which decreased birthweight was reported in animal studies as compared to human studies increases the uncertainty about the biological plausibility of a causal relationship. However, it is important to note that Negri et al. (2017) did not consider that the changes in birthweight reported in the human studies are much smaller (on a percent basis) than in the animal studies. Because the study group sizes in the animal studies are much smaller than in the human studies, the small percentage changes in birthweight observed in the human studies at low serum PFOA levels would not be detectable in the animal studies. Thus, it cannot be concluded that the exposures at which decreased birthweight occur are inconsistent between humans and experimental animals.

Steenland et al. (2018) considered additional studies not included in the two earlier meta-analyses (Johnson et al., 2014; Negri et al., 2017), including the large studies from the C8 Health Study in which serum PFOA levels during pregnancy were modeled from pre-pregnancy serum PFOA data. Although Steenland et al. (2018) concluded that use of modeled or pre-pregnancy serum data may be preferable to serum levels measured during pregnancy because they would not be affected by potential reverse causality/confounding related to expansion of

maternal plasma volume during pregnancy or renal GFR. Johnson et al. (2014) concluded that results from studies without measured serum data during pregnancy are too uncertain to include in a meta-analysis.

The Department has also reviewed the additional studies mentioned by the commenter (Buck et al., 2018; Buck Louis et al., 2018; Manzano-Salgado et al., 2017; Marks et al., 2019; Meng et al., 2018; Shoaff et al., 201; Starling et al., 2017). The results of these studies are generally consistent with the existing literature on associations of PFOA with birthweight and other markers of fetal growth, such as birth length. Notably, serum PFOA levels in the first trimester of pregnancy were associated with decreased birth weight in two of these studies (Manzano-Salgado et al., 2017; Meng et al., 2018), and the authors stated that these associations were not related to GFR. Additionally, another study (Sagiv et al., 2017) concluded that the association of serum levels of PFOA and other PFAS in pregnant women with decreased birthweight were not affected by adjustment for GFR or plasma albumin. In summary, the review of the additional studies discussed above, which were not available to the Institute for inclusion in DWQI (2017a), does not alter the Institute's previous finding that "confounding by GFR does not account for the major portion of the decrease in fetal growth that is associated with PFOA."

Cancer Risk

107. COMMENT: The Department states that human exposure to PFOA has been associated with kidney cancer. Although factually correct, it is highly misleading and improper to cite the

USEPA's 2006 Science Advisory Board panel's (not unanimous) conclusion that PFOA is "likely" carcinogenic to humans. This long-outdated decision preceded the important studies subsequently published from the C8 Science Panel, 3M, and others that resulted in the "downgrading" of the classification to "suspected" by the USEPA Office of Water which is comparable in hazard rating to the IARC "possibly carcinogenic to humans." The Institute should also acknowledge that the Raleigh et al. (2014) study did not show increased incidence of kidney cancer among the PFOA manufacturing workers who had been reported to have the highest serum concentrations of PFOA in occupational settings. (42)

RESPONSE: The Institute in DWQI (2017a) provided a complete and balanced overview of the conclusions of authoritative scientific bodies and peer-reviewed publications regarding the carcinogenic potential of PFOA. Specifically, DWQI (2017a) includes the conclusions of the USEPA Science Advisory Board (2006), USEPA Office of Water (2016), and IARC (2016), as well as the conclusions of several peer-reviewed publications on this topic including Chang et al. (2014). In addition, the Institute supplemented its review of the sources listed above with a review of relevant epidemiological studies not considered in one or more of the USEPA or IARC reviews. These included Steenland and Woskie (2012), Raleigh et al. (2014), Vieira et al. (2013) and Barry et al. (2013). In relation to the 2006 USEPA Science Advisory Board study, DWQI (2017a) acknowledges that the USEPA conclusion was made prior to the publication of the [more recent] epidemiology studies … [Steenland and Woskie (2012), Raleigh et al. (2014), Vieira et al. (2013), Barry et al. (2013)] and was based on the toxicology and mode of action data available at that time." DWQI (2017a) then states, "[m]ore recently, the USEPA Office of

Water (2016a) concluded that PFOA has suggestive [not "suspected" as stated by the commenter] evidence of carcinogenic potential for PFOA."

The Institute in DWQI (2017a) independently reviewed Raleigh et al. (2014) and stated "[t]here was no evidence of elevated risk for kidney cancer [in Raleigh et al., 2014]." DWQI (2017a) also summarized IARC's conclusions, which stated that Raleigh et al. (2014) "did not find evidence for increased incidence of kidney cancer." Additionally, the DWQI (2017a) summary of conclusions of Steenland and Woskie (2012) does not provide any additional interpretations. Steenland and Woskie (2012) acknowledged that tetrafluoroethylene (TFE) is classified as a rodent kidney carcinogen. While TFE may be a confounder because it was used at the facility whose workers were studied, Steenland and Woskie (2012) noted that appreciable exposures would have been unlikely, since TFE exposure would have been well controlled due to TFE's explosive and volatile nature. In conclusion, DWQI (2017a) summarizes the conclusions of available authoritative reviews, both historical and current, of the carcinogenic potential of PFOA, as well as providing a more detailed summary of current epidemiological studies including Raleigh et al. (2014). This information provides support for the development of a Health-based MCL based on carcinogenicity using the approaches recommended in the USEPA Guidelines for Carcinogen Risk Assessment (2005) for chemicals with suggestive evidence of carcinogenicity.

108. COMMENT: In assessing the association between PFOA exposure and testicular cancer, the Institute focuses on the same study as that considered by the USEPA in its 2016 evaluation,

but generates a potency factor that is 36x lower than that calculated by the USEPA. The disparity results from the Institute's decision to use a dose adjustment factor based on biological half-life rather than the default body-weight adjustment chosen by the USEPA. While the Institute cites the USEPA's cancer risk guidelines for the need to adjust for pharmacokinetic differences between species, it provides no rationale for abandoning the default approach, nor does the Institute attempt to compare its conclusion with that reached by the USEPA.

By adjusting the dose for biological half-life instead of body weight, the Institute's analysis places PFOA among the more potent chemicals for which cancer potency factors have been calculated. Such a conclusion is not consistent with the animal data, which suggest a modest cancer response in rats exposed up to 14.2 mg/kg/day, or with the information available from the C8 Health Project. In its analysis, the C8 Science Panel noted that the association with testicular cancer was stronger in community residents than among workers, whose exposures were higher, and that there was little evidence of increasing risk among the residents when compared to the U.S. population. (23, 27, and 41)

RESPONSE: The USEPA (2005) cancer risk assessment guidelines state that toxicokinetic data, when available, should be used for interspecies extrapolation, in preference to the default approach. As stated by the Institute in DWQI (2017a), "[a]lthough USEPA considered interspecies pharmacokinetic differences in animal-to-human conversion for non-cancer effects, the default animal-to-human extrapolation (ratio of body weights to the [three-fourths] power) was used by USEPA for cancer risk assessment. This default approach does not account for interspecies pharmacokinetic differences. USEPA's use of an approach that does not

account for pharmacokinetic differences for cancer risk assessment, although pharmacokinetic differences were considered for non-cancer risk assessment, does not appear to be logical or consistent."

As further stated in DWQI (2017b), "[t]he default approach used by USEPA (body weight to the [three-fourths] power) and the explanation provided by USEPA regarding 'half-life to lifespan ratios' … do not account for these interspecies internal dose differences. Use of internal dose, rather than administered dose, reduces the uncertainty in the risk assessment. This is especially important for a chemical such as PFOA for which the half-life is much longer in humans than experimental animals, resulting in much higher serum levels in humans than in animals from the same administered dose."

The basis of the assertion that the slope factor developed by the Institute is inconsistent with the PFOA dose that caused tumors in the rat study and that it is inconsistent with the testicular cancer data from the C8 Health Project is not clear. The slope factor applies to the risk of cancer in humans, not the risk in experimental animals, and accounts for the higher internal dose in humans than in rats from the same administered dose. The C8 Health Study did not evaluate the concentration of PFOA in drinking water that would result in the one-in-one million increased lifetime cancer risk used as the basis for New Jersey MCLs. This risk level is too low to be detectable in the study population of the size (approximately 15,000 males) evaluated in the C8 Health Study.

In the C8 Health Study (Barry et al., 2013), testicular cancer was validated in about one in 1,000 male study participants (19 validated cases in 14,894 male participants) who were

exposed to drinking water contaminated with concentrations of PFOA greater than 50 nanograms per liter (ng/L). The assertion that "association with testicular cancer was stronger in community residents than among workers, whose exposures were higher" is misleading. The association is stronger in community residents than in workers because, as stated by Barry et al. (2013), the "[r]esults for the worker cohort are limited by low sample size for cancers of interest." Finally, it is noted that data from a more recent chronic toxicology and carcinogenic rat study of PFOA conducted by the National Toxicology Program (NTP, 2019) support a much higher cancer slope factor than the rat testicular tumor data from Butenhoff et al. (2012a) used by the Institute and the USEPA. The California Environmental Protection Agency (CalEPA) developed a cancer slope factor of 143 (mg/kg/day)-1 (approximately 7000-fold more stringent than the Institute slope factor of 0.021(mg/kg/day)-1) based on the incidence of benign and malignant pancreatic acinar cell tumors in male rats in the NTP study (CalEPA, 2019). This slope factor and a one-in-one-million lifetime cancer risk level were used by CalEPA to develop a recommended Notification Level for PFOA in drinking water of 0.1 ng/L.

Development of the MCL for PFOS

Critical Effects

109. COMMENT: We strongly support New Jersey's selection of immune suppression as the most sensitive endpoint for PFOS. As documented in the Institute's health-based maximum contaminant level support document for PFOS and ATSDR's profile, both animal and epidemiology studies provide strong evidence linking PFOS exposure to immunotoxic effects,

including decreased antibody response to vaccines in humans, decreased host resistance to viruses, and suppressed immune response to antigens in animals. The National Toxicology Program also conducted a systematic review to evaluate immunotoxicity data on PFOA and PFOS in 2016. It concluded that both chemicals are presumed to constitute immune hazards to humans based on a high level of evidence that they suppress antibody response in animal studies and a moderate level of evidence from studies in humans. They also identified evidence linking PFOS exposure to suppressed disease resistance and lowered immune cell activity. Importantly, as noted by the Michigan PFAS Science Advisory Panel, "the developing immune system is especially sensitive to environmental stressors ... [d]isruption of immune development is likely to have broader impacts than the antibody changes that are directly measured in these studies and may have long lasting consequences." (37)

RESPONSE: The Department acknowledges the comments in support of the Department's assessment of the immunotoxic effects of PFOS.

110. COMMENT: It is not clear thatthe endpoint selected for development of the MCL represents an adverse effect. In the ATSDR Toxicological Profile for Perfluoroalkyls, it is noted the National Toxicology Program (NTP, 2016) concluded that there is moderate confidence that exposure to PFOS is associated with suppression of the antibody response and that there is low confidence that exposure to PFOS is associated with increased incidence of infectious disease (or lower ability to resist or respond to infectious disease). Changes in the immune system

measures (for example, antibody titers) should not be considered adverse unless they result in a change in disease incidence and severity in the organism. (24)

RESPONSE: The confidence ratings used by the NTP (NTP, 2016) are not indicators of the adversity of a given immune outcome, such as suppression of the antibody response. The endpoint selected for development of the MCL, decreased plaque forming cell response in mice as reported in Dong et al. (2009), does represent an adverse effect. As reviewed in detail in DWQI (2018a) and by the Department (see Pachkowski et al., 2019), decreases in antibody response following vaccination are associated with exposure to PFOS.

This is supported by experimental evidence showing that PFOS exposure in rodents results in decreased antibody response to foreign antigens and also decreased host resistance, as indicated by increased mortality following influenza virus challenge (Guruge et al., 2009). Additionally, the International Programme on Chemical Safety (IPCS) in "Guidance for Immunotoxicity Risk Assessment for Chemicals" states, in reference to interpreting laboratory animal studies, that "any statistically significant effect [on immune function] should be considered meaningful, provided the quality of the animal data is sufficient" (IPCS, 2012). Therefore, increased incidence of infectious disease is not necessary. IPCS (2012) bases this conclusion on "the assumption that a linear relationship exists between loss of immune responsiveness and increased risk of developing disease [which is] consistent with our understanding of immunological processes and is supported by both laboratory animal … and human studies … in which changes in immune tests correlated progressively with increased incidence of disease over a broad range." These statements from IPCS (2012) support the relevance of decreased plaque forming cell response as an appropriate endpoint for risk assessment and MCL development. Additionally, consistent with the IPCS guidance, the Health Effects Subcommittee concludes that Dong et al. (2009) is of sufficient quality to serve as the principal study for the derivation of a Health-based MCL

(DWQI, 2018a). Finally, as stated in DWQI (2018a), the USEPA's Integrated Risk Information System (IRIS) program has used decreased plaque forming cell response as the basis for the derivation of reference doses for several other contaminants.

As discussed below, the Department (Pachkowski et al., 2019) concluded that PFOS exposure is associated with an increased incidence of childhood infections. The Department based this conclusion on its review of relevant studies, including several that were not available to NTP in 2016 (such as Dalsager et al., 2016, Goudarzi et al., 2017, Impinen et al., 2018). Together, these observations demonstrate that PFOS exposure in humans results in a change in an immune system measure as well as an increase in disease incidence.

111. COMMENT: The Department's proposal offers little support for the relevance of the available animal and human data, which NTP is clear to caution may not be related to actual health effects in humans. (23, 27, and 41)

RESPONSE: The Department agrees with the National Toxicology Program's conclusions about the relevance of the animal and human data to health effects in humans. NTP (2016) concluded that "… exposure to PFOS is presumed to be an immune hazard to humans based on a high level of evidence that PFOS suppressed the antibody response from animal studies and a moderate level of evidence from studies in humans." In support of this conclusion, NTP (2016) states "[t]he production, release, and increase in circulating levels of antigen-specific antibodies are important for protection against the infectious agent and preventing or reducing severity of influenza, respiratory infection, colds, and other diseases as part of the humoral immune response. Reduced antibody production is an indication of decreased immune function or immunosuppression that may indicate a greater risk of disease." NTP (2016) further states that "[a]ntigen-specific IgM [immunoglobulin M] to a T-cell-dependent antigen

(e.g., SRBC [sheep red blood cells]) is considered one of the most predictive measures of overall immune function because proper response requires cooperation between T-cells, B-cells, and antigen-presenting cells to develop an antibody response (Luster et al., 1992). This antibody response can be examined by measuring antigen-specific antibody levels after vaccination in humans and after challenge with SRBC or other antigens in laboratory animals."

112. COMMENT: While asserting that the SRBC response in mice are "analogous" to decreased vaccine response in humans, the Department offers no supporting information and neither the USEPA nor Health Canada have reached a similar conclusion. (23, 27, and 41)

RESPONSE: The Institute concluded that the plaque forming cell response (PFCR) to sheep red blood cells (SRBC) in mice and antigen-specific antibody production in response to vaccination in humans are analogous, as both are indications of T-cell dependent antigen-specific antibody responses to analogous antigen challenges. The Institute made this same conclusion in DWQI (2018b). The Department agrees with this conclusion.

Additionally, a review of the epidemiological and toxicological evidence for the immunotoxicity of PFAS stated that "the TDAR is widely regarded as robust and sensitive, and translatable to humans. The analogous human response is antibodies generated toward a specific vaccine, which can be measured in human populations exposed to particular exogenous agents" (DeWitt et al., 2019).

113. COMMENT: For PFOS, the Institute report could not consider more recent and more relevant toxicity studies. The Institute evaluation did not use a 2017 published clinical study with monkeys exposed to PFOS for its risk assessment. Since these non-human primates have

much more physiological resemblance to humans than rodents, the results are more relevant to determine potential human effects than the rodent studies that are the basis of the Institute's recommendation. (51)

RESPONSE: The Department does not consider Chang et al. (2017) to be an appropriate basis for its MCL for PFOS because this study exposed monkeys to the contaminant only three times over the course of one year. Such an exposure regimen is not relevant to the daily, chronic exposure that occurs through the consumption of PFOS-contaminated drinking water. Additionally, this study did not evaluate the immunotoxic effects of PFOS, which the Department identified as among the most sensitive effects reported from laboratory animal studies.

114. COMMENT: Among the immunotoxicity data that had been reported for PFOS, inconsistent and inconclusive findings are being reported. When Peden-Adams et al. (2008) reported the immune suppression with PFOS at such low serum level (approximately 91 ng/mL) in mice, Qazi et al. (2009a; 2010; 2009b) carefully designed and performed a series of studies trying to see if the results reported by Peden-Adams et al. could be replicated. They were not able to replicate the results. The weight of evidence would suggest that immunotoxicity responses are not rigorous nor robust to support the Department's risk characterization. (42)

RESPONSE: The Department does not agree that findings for PFOS immunotoxicity, in particular for decreased plaque forming cell response, are "inconsistent and inconclusive." As stated in DWQI (2018b), "[t]he Health Effects Subcommittee does not agree with the characterization of immunosuppressive effects of PFOS as 'inconsistent across studies.'" Only one of five PFOS studies that evaluated plaque forming cell response in mice was negative for this effect. Relevant to this point, the

USEPA (2016a) Health Effects Support Document for Perfluorooctane Sulfonate (PFOS) notes a "… consistent suppression of SRBC response [such as plaque forming cell response] in animals …" Additionally, the Institute in DWQI (2018b) and the Department (see Pachkowski et al., 2019) discuss the sole study (Qazi et al., 2010) that did not report a decrease for plaque forming cell response with PFOS exposure. NTP (2016) concluded that "exposure to PFOS is presumed to be an immune hazard to humans based on a high level of evidence that PFOS suppressed the antibody response from animal studies and a moderate level of evidence from studies in humans." Additionally, NTP (2016) states "[t]he evidence indicating that PFOS suppresses multiple aspects of the immune system supports the overall conclusion that PFOS alters immune function in humans." The Department maintains that such conclusions support the use of immunotoxicity as the basis for its MCL for PFOS. Finally, the Department does not agree with the commenter that the Qazi et al. studies were a replication of the Peden-Adams et al. (2008) study. The Institute in DWQI (2018a) and the Department (Pachkowski et al., 2019) have noted major study design differences between the studies.

115. COMMENT: The animal immunological data relied on by the Department are inconsistent. Five studies have investigated potential effects on the immune system (NK cell activity and SRBC response) in mice exposed to PFOS. Although the studies reported immune effects, the USEPA concluded that the differences in the levels at which effects were reported (and conflicts in the direction of the effects) "highlight the need for additional research to confirm the NOAEL and LOAEL for the immunological endpoints." Health Canada reached a similar conclusion noting that "[f]urther exploration should be performed to address the nearly two orders of magnitude difference in LOAELs in the studies before these endpoints can be

reliably considered as a basis for risk assessment." The inconsistency of these study results is detailed below.

The 2008 study by Peden-Adams et al. (2008) identified decreased SRBC response in male B6C3F1 mice exposed to 0.0017 mg/kg/day after 28 days of treatment, although no overt signs of toxicity were observed at doses up to 0.166 mg/kg/day. Additionally, the study observed enhanced NK cell activity at the lowest PFOS doses, but suppressed activity at higher doses. In the study by Keil et al., also published in 2008, B6C3F1 mice exposed during gestation had decreased NK cell activity in males (at one mg/kg/day) and females (at five mg/kg/day) at postnatal week eight – the opposite of the effect reported by Peden Adams et al. SRBC response was suppressed in males, but at doses several orders of magnitude higher (five mg/kg/day) than in the study by Peden-Adams et al. No SRBC response was reported in females. A 2009 study by Zheng et al. reported decreased NK cell activity in male C56BL/six mice exposed to one mg/kg/day over seven days. Additionally, SRBC response was observed in males at five mg/kg/day, consistent with the report from Keil et al.

In the mouse study by Dong et al. (2009), NK cell activity was reported to increase at 0.083 mg/kg/day and to decrease at doses 10-fold higher (0.833 mg/kg/day) after 60 days. Decreased SRBC response also was reported in C57BL/six males at 0.083 mg/kg/day, well below the LOAEL reported in the Keil study. (23, 27, and 41)

RESPONSE: The Department agrees with the Institute's conclusions in DWQI (2018b) regarding the consistency of results between Dong et al. (2009) and other mouse studies assessing plaque forming cell response. The differences in NOAELs and LOAELs between the animal studies assessing immunotoxicity, particularly plaque forming cell response, may reflect methodological differences between studies, (such as dose selection, strain, source of SRBCs). The study selected as the basis for the Health-based MCL was not the most or the least sensitive of the four studies showing that PFOS causes this effect, and

it was selected for reasons discussed by Pachkowski et al. (2019) and the Institute in (DWQI, 2018a). The database for immunotoxicity of PFOS clearly demonstrates a consistent observation of decreased plaque forming cell response. This conclusion is supported by other recent assessments of PFOS. In its 2016 "Health Effects Support Document for Perfluorooctane Sulfonate (PFOS)," the USEPA acknowledges "… the consistent suppression of SRBC response in animals." Additionally, the NTP (2016) systematic review concluded that there is "a high level of evidence that PFOS suppressed the antibody response from animal studies."

NK cell activity is not related to the SRBC response and was not used as the basis for the Health-based MCL.

116. COMMENT: The Department fails to provide its rationale for selecting the SRBC response data from Dong et al. (2009) to generate the MCL when they conflict with findings reported by the same group in a subsequent study and by other researchers. (23, 27, and 41)

RESPONSE: The Department notes that the Institute addressed the differences in results reported in the two Dong et al. (2009, 2011) studies in DWQI (2018b), which states that, "[t]he two studies [Dong et al. (2009) and (2011)] measured different endpoints following SRBC inoculation. In Dong et al. (2009), 'SRBC response' was evaluated with the plaque forming cell assay, which is an assessment of immune function. In Dong et al. (2011), the 'SRBC response' was assessed by measuring serum levels of IgM, which is an observational immune endpoint and does not address specific antibody function." In regard to the comment that the results of Dong et al. (2009) conflict with the findings from other researchers, both DWQI (2018a) and the Department (see Pachkowski et al., 2019) have provided in-depth reviews showing that the decrease in plaque forming cell response from Dong et al. (2009) is supported by four other studies that also demonstrated a decrease in plaque forming cell response following a challenge

with sheep red blood cells. Only one study (Qazi et al., 2010) did not find such a response, which was likely due to differences in study design and/or non-study design factors (DWQI, 2018a; Pachkowski et al., 2019).

117. COMMENT: The 2016 NTP systematic review of the animal data concluded that it cannot be confident in the outcome assessment of the Dong et al., 2009 study that is the basis for the proposed MCL. (23, 27, and 41)

RESPONSE: The Department agrees with the Institute that NTP (2016) did not provide a definitive statement regarding its confidence in the outcome assessment in Dong et al. (2009). NTP (2016) noted that Dong et al. (2009) did not report whether outcome assessors were blinded to the exposure groups. This is the case for most toxicology studies published in peer-reviewed journals and does not necessarily indicate a flaw in the design or conduct of the study. Additionally, NTP noted that "well-established methods" were used to measure PFCR in this study. Based on consideration of a number of relevant factors, including evidence of dose-response across multiple studies, magnitude of the effect, consistency, potential for publication bias, and several others, NTP (2016) concluded that there is high confidence that exposure to PFOS is associated with suppression of the antibody response based on the available animal studies.

118. COMMENT: The Department appears to have selected Dong et al., 2009 as its critical study merely because it yielded the lowest possible MCL from its list of candidate MCLs. (42)

RESPONSE: The Department notes that Dong et al. (2009) was neither the most nor least sensitive of the available studies. For example, Peden-Adams et al. (2008) reported that PFOS caused decreased

plaque forming cell response (the immune system effect used as the basis for the PFOS MCL) in male mice at PFOS doses approximately 50-fold lower than in Dong et al. (2009). The Department relied on the best available science, and it does not agree that studies and endpoints were arbitrarily selected to drive lower MCLs for PFOS. In regard to scientific rigor, the proposed MCLs are based on a thorough evaluation and documentation of the relevant epidemiological, toxicological, and mode of action studies, including screening of over 2,800 peer-reviewed studies identified in a literature search. The Department does not agree that Dong et al. (2009) was selected as the critical study "merely because it yielded the lowest possible MCL ..." As reviewed in DWQI (2018a) and by the Department (Pachkowski et al., 2019), the Dong et al. (2009) study was selected as the critical study after a thorough review of nearly 200 human and animal studies.

119. COMMENT: The Dong et al. (2009) study used by the Institute as the point of departure for the PFOS MCL was based on immunotoxicity. From a fundamental immunology perspective, there were several important technical aspects that Dong et al. (2009) failed to address. The study lacked overall scientific validity to support the conclusion that PFOS causes immune suppression. It is well-known that body weight plays a critical role in studying immune response and any factors that can influence body weight will likely indirectly affect immune responses. Although Dong et al. claimed that body weight was not affected in the first two lower dose groups (0.5 and five mg/kg TAD), based on simple ANOVA and Dunnett's t tests, there appeared to be a difference in mean body weight change between the control group (mean body weight = 3.10 ± 0.13 g) and the NOAEL dose group at 0.5 mg/kg/day (mean body weight = 2.58 ± 0.15 g). With one-sided test, the final body weights in the 0.5 mg/kg/day dose group were

significantly lower than the control group at α (alpha) = 0.10 (0.05 < p < 0.10). With two-sided test, it was statistically significantly different at α (alpha) = 0.20 (0.15 < p < 0.20). Therefore, Dong et al. (2009) data may have been confounded by decreased body weight effect which hindered the overall interpretation. (42)

RESPONSE: The commenter's statement that there was "significance" in the difference in body weight change between controls and the lowest dose is based on the calculation using less stringent criteria, alpha of 0.1 (one-sided) and 0.2 (two-sided), for statistical significance than used by Dong et al. (2009). Dong et al. (2009) stated that there was no statistical difference (alpha ≤ 0.05) and did not conclude that body weight change was a consideration in their findings of plaque forming cell response. Therefore, the Department does not agree that this is a "flaw" or relevant to the use of decreased plaque forming cell response as the basis for the PFOS MCL.

120. COMMENT: Dong et al. (2009), which was used by the Institute for its recommendation for an MCL for PFOS, had several important technical aspects that Dong et al. (2009) failed to address. The study lacked overall scientific validity to support the conclusion that PFOS causes immune suppression. The standard clinical marker for antibody titers to vaccinations are secondary immunoglobulin G (IgG) antibody isotype, not primary IgM. Dong et al. reported the PFOS dose-dependent reductions in SRBC-induced IgM plaque forming cell assay in vitro; they did not evaluate IgG or other potential antibody responses that can develop, including IgG or IgE. In addition, the use of the SRBC-induced antibody response to measure antigen-induced

antibody response is very crude and non-specific to T cell activation. There are better T-cell dependent antigens available for use in the immunology research (that is, ovalbumin) and Dong et al. did not acknowledge such fact. (42)

121. COMMENT: There is insufficient evidence to support immunotoxicity with PFOS. Although NTP conducted a systematic review in 2016 and concluded that PFOS is presumed to be immune hazards to humans in connection with vaccine antibody response, there were several areas of the NTP systematic review where insufficient animal data were used as supporting evidence for human findings and its final hazard conclusion. In particular, suppression of the T cell dependent antibody response (TDAR) in mice, which evaluates suppression of the "primary" IgM response, is used to support suppression of antibody titers to vaccinations in humans. However, because vaccine antibody titers reflect the secondary IgG response, the observation in human epidemiological data was in great discrepancy with animal data in that no suppression of the secondary IgG response was observed in mice. (42)

RESPONSE TO COMMENTS 120 AND 121: While the plaque forming cell response to the sheep red blood cells was likely to have been mounted by the mice primarily on the basis of IgM antibodies, IgM is the first-line defense against foreign antigens. As stated by the NTP in its systematic review (NTP, 2016) of the immunotoxicity of PFOS and PFOA, "[a]ntigen-specific IgM to a T-cell-dependent antigen (for example, SRBC) is considered one of the most predictive measures of overall immune function." Furthermore, IgM enhances the downstream IgG response. A possible explanation for IgM-mediated enhancement is that B lymphocytes capture IgM-antigen-complement complexes and transport the complexes into areas in the

spleen where efficient immune responses are generated.

Consistent with this, and as reviewed by the Department (Pachkowski et al., 2019), strong epidemiologic evidence that PFOS reduces the vaccine response (an IgG response) strongly suggests that PFOS additionally affects the IgG response. The Department asserts that the fact that Dong et al. (2009) did not evaluate the IgG response does not detract from the health significance of a decrease in the IgM response. Additionally, the fact that Dong et al. (2009) did not evaluate the immunoglobin E (IgE) response, a hypersensitivity-related outcome, is not relevant to the PFOS-mediated immunosuppression, a decrease in plaque forming cell response, demonstrated in this study.

The plaque forming cell response to sheep red blood cells following PFOS exposure is a quantitative demonstration of a functional deficit in immune response to a foreign antigen. While the specific mechanisms of this deficit (such as the effect of PFOS on T cell activation) are of interest from the standpoint of mechanistic toxicology, they do not alter the interpretation of the functional deficit in immune response resulting from PFOS exposure, and they do not alter the relevance of this deficit as a point-of-departure for human health risk assessment.

The studies by Peden-Adams et al. (2008), Zheng et al. (2009), and Keil et al. (2008), which were reviewed by the Institute and the Department, all used the plaque forming cell response assay with sheep red blood cell immunization as their measure of the functional effect of PFOS on the immune response.

122. COMMENT: The Dong et al. (2009) study used by the Institute as the point of departure for the PFOS MCL had numerous technical deficiencies that Dong et al. (2009) did not consider. While Dong et al. claimed that the antibody response was reduced based on IgM plaque forming cell response data; the IgM plaque forming cell response activity was only evaluated in spleen cells. The authors should have also looked at thymus and serum for IgM levels to illustrate that the responses are consistent in other primary immune organs. By way of similar scientific rationale, Dong et al. should have looked at IgG in addition to IgM, as well as evaluated IgG levels in thymus and serum. (42)

RESPONSE: Regardless of effects on the thymus and serum, the observations from Dong et al. (2009), Zheng et al. (2009), and Peden-Adams et al. (2008) based on response of the splenocytes in the plaque forming cell response assay are of notable health significance. The Department also notes that PFOS caused a decrease in serum IgM levels in Dong et al. (2011), which supports the decrease in plaque forming cell response reported in Dong et al. (2009). As explained in the response to prior comments, the Department does not agree that the lack of IgG measurement in Dong et al. (2009) represents a technical flaw or diminishes the significance of the decreased IgM response reported in this study.

123. COMMENT: While the immune cell populations were reported by Dong et al. in spleen and thymus, they did not look at these cell populations in another key immune organ: bone marrow. Similarly, while NK cell activity was reported for the spleen, it was not done for the thymus. These were major technical omissions that the Department should have considered. (42)

RESPONSE: The Department does not agree with this comment. Measurements of immune cell

populations, such as those in bone marrow, are considered to be observational immune endpoints and are less predictive of immunotoxicity than measures of immune function, such as plaque forming cell response, which was assessed by Dong et al., 2009 (NTP, 2016). NK cell activity is a separate endpoint from plaque forming cell response, and the fact that Dong et al. (2009) did not evaluate NK cell activity in the thymus is not relevant to the use of plaque forming cell response data from this study as the basis for the MCL.

124. COMMENT: Dong et al. reported a negative effect of PFOS and the splenic lymphocyte proliferation as a way of demonstrating that the immune cells were not "proliferating" upon challenge. However, two major technical flaws associated with the study design limit a scientific support for this conclusion:

Dong et al. reported Concanavalin A (ConA)-mediated responses as antigen specific T cell receptor-based proliferation in vitro. However, ConA stimulates T cells through a different set of pathways than through the T cell receptor. The more appropriate method would have been using anti-CCD3/CD28 antibodies to mimic antigen specific cell stimulation in vitro.

The second concern is the use of the 3-(4,5-Dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) assay to determine T cell proliferation in vitro. The MTT assay determines metabolic activity, not cell numbers. It is simply an indicator of the mitochondrial respiration state of cells and is not a reflection any proliferative response(s). The standard assay for cell proliferation would be bromodeoxyuridine (BrDU) assay or proliferating cell nuclear antigen (PCNA) staining, neither of which was used by Dong et al. and the

Department was misinformed. (42)

RESPONSE: The Department does not agree that the manner in which lymphocyte proliferation was measured is a technical flaw in Dong et al. (2009) or diminishes the significance of the plaque forming cell response results from Dong et al. (2009). The Department notes that the NTP, as part of its risk of bias analysis of Dong et al. (2009) determined that "… lymphocyte proliferation assay, antibody plaque forming cell assay (response to SRBC administration) and natural killer cell activity were measured with well-established methods" (NTP, 2016).

125. COMMENT: The human immunological data relied on by the Department is inconsistent. The USEPA cautioned that "lack of human dosing information ... precludes the use of these immunotoxicity data in setting the [reference dose]." (23, 27, and 41)

RESPONSE: The Department does not agree with the USEPA's contention that immunotoxicity data cannot be used in deriving a reference dose for PFOS due to a "lack of human dosing information," particularly since animal data are used as the basis for the Reference Dose for the MCL. The Department asserts that the rationale for the USEPA's dismissal of immunotoxicity data for the derivation of a PFOS reference dose is poorly described and does not appear to be scientifically valid. Additionally, this statement is taken out of context by the commenter, since the USEPA (2016a) states in the sentence preceding the quoted text, "[t]aken together, the lower antibody titers associated with PFOS levels in humans and the consistent suppression of SRBC response in animals indicates a concern for adverse effects on the immune system." Furthermore, it is unclear what "lack of human dosing information" means since the dose metric for PFOS is serum PFOS levels, and serum PFOS data are provided in the relevant studies.

126. COMMENT: The epidemiologic evidence relied on by the Department is inconsistent and does not support an association between PFOS exposure and decreased vaccine response in humans. The USEPA (2016b) has made the following conclusions regarding immunotoxicity and exposure to PFOS and other perfluoroalkyls: "[a]nother limitation of epidemiology studies that evaluate the immune response following PFOS exposure is that these studies have not demonstrated whether immune parameters measured in clinically normal individuals accurately reflect the risk of future immunological diseases. Given the immune system's capacity for repair and regeneration, apparent abnormalities that are detected at one point in time might resolve before producing any adverse clinical health effect. Thus, biomarkers that do not accurately diagnose or predict the presence or absence of a clinical health condition are not clinically useful." (42)

RESPONSE: DWQI (2018a) includes a detailed review of the USEPA Health Advisory for PFOS (USEPA, 2016b, cited in the comment). In general, it concludes that the USEPA rationale for dismissing immunotoxicity as the basis for the Health Advisory is not scientifically supportable, and that the USEPA Health Advisory is not sufficiently protective of human health. Specifically, the Department disagrees with the USEPA (2016b) conclusion that is cited by the commenter. Decreased antibody response to vaccination is clearly an adverse effect that is appropriate as the basis for risk assessment, particularly when it is observed in humans from environmentally relevant exposures, and this effect has lasting consequences if it is present at the time of vaccination. Furthermore, the Department has determined that PFOS has been associated with the clinical effect of increased infection, and there are now additional data to support this conclusion. Finally, it noted that Grandjean et al. (2012) found a

statistically significant odds ratio for diphtheria and tetanus vaccine antibodies falling below the clinically protective standard as a function of PFOS.

127. COMMENT: The existing epidemiologic studies do not provide consistent evidence of a significant association between PFOS exposure and decreased vaccine responses. Contrary to the Institute's assertion that "study findings are consistent and support a potential for PFOS to reduce vaccine response" (DWQI 2018), mostly null findings have been reported across all studies and the results are inconsistent by vaccine type. For example, among the five existing studies that have examined antibody responses to the tetanus vaccine (the most commonly studied vaccine type) relative to serum PFOS levels, only one study reported a significant decrease in antibody levels (Grandjean et al., 2012). The other four studies, including a follow-up study of Grandjean et al., 2012, did not observe a significant decrease in tetanus antibody levels (Grandjean et al., 2017). More specifically, of the 11 statistical measures of association reported across these five studies, only one was significant (Grandjean et al., 2012). Clearly, the evidence from epidemiology studies examining tetanus vaccine response does not support a potential for PFOS to reduce this toxoid response as the Institute infers. (42)

128. COMMENT: The Department asserts that human exposure to PFOS has been associated with decreased vaccine response. Contrary to this assertion considerable inconsistencies have been observed among the nine epidemiological studies that have examined PFOS exposure to antibody responses to 10 distinct vaccine antigens. Because of these inconsistencies, these

studies do not support an association between PFOS exposure and decreased vaccine response in humans. (42)

RESPONSE TO COMMENTS 127 AND 128: The Department acknowledges there are inconsistencies in the epidemiological data for vaccine response. Multiple vaccinations were assessed in the studies of human vaccine response that were reviewed and summarized in DWQI (2018a) and a peer-reviewed publication by the Department (Pachkowski et al., 2019). However, there were few repeated observations for any given vaccine antibody across the studies. Nonetheless, decreased responses to four different vaccine antibodies in six studies were found to be associated with PFOS exposure (Pachkowski et al, 2019). These observations strongly suggest a general effect across vaccines. Additionally, different vaccines (and different preparations of the same vaccine) can differ in their inherent antigenicity. Thus, it may be that a given vaccine (for example, the influenza vaccine) with strong antigenic properties is less subject to the titer-reducing effects of PFOS than those vaccines that yielded significant associations. The commenter's discussion of only one type of vaccine response (tetanus) ignores other evidence that DWQI (2018a) considered in determined the consistency of the human vaccine response database. Specifically, as reviewed in DWQI (2018a) and by the Department in Pachkowski et al. (2019), multiple studies have found an association between PFOS exposure and decreased antibody response following vaccination for diphtheria and rubella. Additionally, although assessed in one study each, PFOS exposure was also associated with decreased vaccine response to mumps, as well as enterovirus 71 (EV71) and coxsackievirus A 16 (CA16) (as addressed in a response to a comment below). Collectively, the human database for vaccine response strongly points to a general effect across vaccines. This is consistent with NTP (2016) which states: "[t]aken together, these studies provide evidence that higher developmental, childhood, or adult serum concentrations of PFOA and

PFOS are associated with lower specific antibody responses to commonly used vaccines. The data are considered a consistent pattern of findings for PFOA- and PFOS-associated antibody suppression."

129. COMMENT: The MCL recommendation for PFOS was based on a decreased plaque forming cell response in adult mice (Dong et al., 2009). The Institute argues that this effect is supported by epidemiological evidence for an "analogous effect" of decreased vaccine response in humans. The Institute's review of the epidemiology literature is outdated and fails to accurately reflect the inconsistencies and mostly null findings across studies. (42)

130. COMMENT: The Institute acknowledges only five epidemiology studies; however, there are nine published studies that have examined PFOS exposure and antibody responses to vaccines in children, adolescents, and adults (Grandjean et al., 2012; Grandjean et al., 2017; Granum et al., 2013; Kielsen et al., 2016; Looker et al., 2014; Mogensen et al., 2015, Stein et al., 2016a; Stein 2016b; Zeng et al., 2019). These studies have measured antibody responses to 10 distinct vaccines: tetanus, diphtheria, rubella, measles, mumps, influenza A (H1N1), influenza A (H3N2), influenza B, enterovirus, and coxsackievirus. While tetanus and diphtheria are the most commonly studied vaccine types, other vaccines have only been reported in one or two studies. (42)

RESPONSE TO COMMENT 129 AND 130: The Department has reviewed the four studies not originally reviewed in DWQI (2018a). In brief, Mogensen et al. (2015) is a further analysis and confirmation of Grandjean et al. (2012) in that the both studies observed an inverse relationship between serum PFOS concentration and vaccine response for diphtheria and tetanus in the same study population. In Mogensen et al. (2015) these observations were based on PFOS and antibody measurements at age

seven, whereas the Grandjean et al. (2012) data were based on PFOS measurement at age five. Stein et al. (2016) is a study of 78 adults assessing antibody response following influenza vaccination, specifically FluMist. Although the authors did not find an association between PFOS exposure and vaccine response, they noted that, "[g]iven the study's many limitations, however, it does not rule out impaired vaccine response to other vaccines or vaccine components in either children or adults." Grandjean et al. (2017) is an analysis of a smaller-sized birth cohort from the same location (the Faroe Islands) as Grandjean et al. (2012). Although no statistically significant associations between serum PFOS and vaccine antibodies (tetanus, diphtheria) were observed, pooled analysis of this smaller cohort with that of Grandjean et al. (2012) did reveal a significant decrease in diphtheria vaccine antibody. Zeng et al. (2019) is a study of three-month old infants that observed statistically significant associations between elevated PFOS concentrations in cord blood samples and decreased antibody measurements for enterovirus 71 (EV71) and coxsackievirus A 16 (CA16), two viral agents responsible for hand, foot, and mouth disease. The doubling of cord blood PFOS concentrations was also associated with increased risk for EV71 and CA16 antibody concentrations falling below clinically protective levels. In addition to these four studies, the Department also notes a study by Timmermann et al. (2019) that reported a statistically significant association between increased serum PFOS levels and decreased measles antibody concentrations in children. These studies add to the database of studies reviewed in DWQI (2018a) and by the Department (Pachkowski et al., 2019). Although this database consists of positive and null results for some antibodies, as well as limited results for others, the database strongly points to a general effect across vaccines.

131. COMMENT: The Department should not interpret antibody responses to these distinct vaccine types should not be interpreted as a single health outcome, such as decreased vaccine

response. Rather, antibody responses to each vaccine type should be considered separately as vaccines differ depending on the nature of the vaccine antigen. Tetanus and diphtheria, for example, are toxoid vaccines whereas measles, mumps, and rubella are live attenuated vaccines. Influenza vaccines are inactivated, conjugate, or live attenuated depending on the strain and method of administration. Consequently, each vaccine type elicits an immune response through various molecular and cellular mechanisms of the immune system. Additionally, all vaccines contain various excipients including adjuvants to improve the antibody response, preservatives, stabilizers, and vehicles for delivering the vaccine, which may differ substantially depending on the vaccine (Baxter, 2007).

The National Toxicology Program acknowledged the differences in immune response across vaccines, and stated that "[t]he strength of an antibody response in terms of antibody level and length of time that an elevated/effective antibody response is maintained is known to differ across vaccines" (NTP, 2016). Granum et al. (2013), also concluded that "different vaccines may stimulate different components of the immune system, which can explain the vaccine-dependent differences in the effect of PFAS exposure." Therefore, observed changes in antibody response to a particular vaccine type should not be interpreted as consistent with changes in the antibody response to another vaccine. (42)

RESPONSE: As discussed in the responses to prior comments, the Department acknowledges there are inconsistencies in the epidemiological data for antibody response following vaccination in humans. In discussing sources of heterogeneity in the human antibody response dataset, NTP (2016) concluded that "[t]aken together, these studies provide evidence that higher developmental, childhood, or adult serum

concentrations of PFOA and PFOS are associated with lower specific antibody responses to commonly used vaccines. The data are considered a consistent pattern of findings for PFOA- and PFOS-associated antibody suppression." Additionally, as reviewed in the responses to prior comments, newer studies (for example, Zeng et al., 2019 and Timmermann et al., 2019) not available to DWQI (2018a) and NTP (2016) lend further support to the conclusion that increased serum PFOS levels are associated with decreased antibody levels.

132. COMMENT: In support of the proposed MCL for PFOS, the Department and the Institute assert the relevance of reduced SRBC response observed in mice to reduced resistance to infection in humans. Yet, the human studies generally report no increase in infection in children or adults, and both the USEPA and Health Canada have questioned whether the small variations in the antibodies observed in the available studies are sufficient to result in adverse health effects in humans. (23, 27, and 41)

133. COMMENT: Small changes in antibody response, as observed in some studies, do not necessarily translate to an increased risk of infectious disease, as the Department and Institute asserts. Several epidemiologic studies (Dalsager et al., 2016; Fei et al., 2010; Impinen et al., 2018; Looker et al., 2014; Okada et al., 2012; Goudarzi et al., 2017; Granum et al., 2013) have examined PFOS levels and infectious disease outcomes (that is, occurrence of common colds and otitis media, symptoms of infections, mortality from infectious and parasitic diseases, and hospitalizations from infectious diseases). Across all reported measures, mostly inconsistent

associations between PFOS levels and increased risk of infectious disease outcomes have been observed. (42)

134. COMMENT: Hypothesized vaccine response effects appear to have no clinical significance as the data does not support a causal association between PFOS exposures and an increased risk of infectious disease. As a result, they do not provide collaborative support to the immunotoxicity findings in laboratory studies in mice claimed by the Department. (42)

RESPONSE TO COMMENTS 132, 133, AND 134: The Department does not agree that there is a lack of association between PFOS exposure and increased infectious disease. The Institute in DWQI (2018) and the Department in Pachkowski et al., (2019) reviewed seven studies (six in children and one in adults) assessing the association between PFOS exposure and infectious disease. Four of the six studies in children observed statistically significant associations between the incidence of infectious disease and serum PFOS concentrations, which were in the range of the serum levels found in the U.S. general population. These four studies included different age ranges (even among children), evaluated different endpoints, and provided different statistical measures of association. Taken together, these studies provide evidence that PFOS exposure is associated with a clinically meaningful measure of health risk, specifically the incidence of infectious disease. Contrary to the questions of the USEPA and Health Canada, regarding whether small variations in antibodies levels are sufficient to affect human health, the National Toxicology Program acknowledges the health significance of antibody levels. In reference to antibody reductions as reported in Stein et al. (2016) and Grandjean et al. (2012) from PFOS exposure, NTP (2016) states, “[i]t is unknown if this level of reduction would affect the immune response to a viral or bacterial challenge for these individuals. Nevertheless, immune suppression resulting in a lower antibody response is not a desirable outcome and any lowering of the antibody

response may be considered adverse on a population level such that individuals with lower antibody levels may be less able to mount a defense against viruses or bacteria (WHO 2012)." As discussed in the Response to Comment 136, three of the four studies reporting associations of PFOS and infectious disease in children were not available when NTP (2016) made its conclusion about uncertainty regarding the response to a bacterial or viral challenge. Finally, the Department notes that at least five other states, California, New York, Michigan, Minnesota, New Hampshire, and Washington have concluded that immune effects are valid endpoints for developing drinking water guidelines and have based their PFOS reference doses on immunotoxicity. Similarly, the ATSDR MRL for PFOS also considers immunotoxicity as a sensitive endpoint for PFOS toxicity.

135. COMMENT: Relative to continuously declining PFOS concentrations in the general population, World Health Organization data on the time series distribution of tetanus and diphtheria in the United States do not suggest a population whose immunity to tetanus or diphtheria might have been compromised by a decreased antibody response due to PFOS exposure. These data do not suggest there is an increased risk of infection to tetanus or diphtheria in a high-vaccinated population, such as in the United States. Therefore, it is highly speculative to suggest there are immune system deficits beyond these "2 specific bacteria" when such risks do not exist for tetanus or diphtheria as expressed in Grandjean et al. (2012), which the Institute relied upon. (42)

RESPONSE: The Department notes that the decreased response to vaccination reported in epidemiological studies is an indicator of immunosuppression, and it is not necessary to observe an increase in the cases of the corresponding disease, for example tetanus and diphtheria, to consider a

decrease in vaccine response as an adverse effect. Additionally, the Department notes that the commenter's assertion that "relative to continuously declining PFOS concentrations in the general population, [the data submitted by the commenter] do not suggest a population whose immunity to tetanus or diphtheria might have been compromised by a decreased antibody response due to PFOS exposure" ignores incidence data for other diseases. The Department notes that the incidence of mumps has increased over the years despite "… continuously declining PFOS concentrations in the general population …" (see https://apps.who.int/immunization_monitoring/globalsummary/incidences?c=USA).

136. COMMENT: The National Toxicology Program concluded that there is low confidence that exposure to PFOS is associated with increased incidence of infectious disease (or lower ability to resist or respond to infectious disease) (NTP, 2016). (42)

RESPONSE: The Department notes that since the release of the National Toxicology Program systematic review in 2016, additional epidemiological studies have reported an association between PFOS exposure and the incidence of infectious disease. Several studies (Dalsager et al., 2016; Goudarzi et al., 2017; Impinen et al., 2018) were not available to the National Toxicology Program (2016) when it conducted its systematic review and arrived at the conclusion of low confidence for PFOS exposure and incidence of infectious disease. Each of the three later studies reported a statistically significant association between elevated serum PFOS concentration and incidence of infectious disease. These studies strengthen the evidence that PFOS exposure is associated with increased incidence of infectious disease. This conclusion is further supported by experimental evidence showing that PFOS exposure in rodents decreases host resistance, as indicated by increased mortality following influenza virus challenge

(Guruge et al., 2009). The relevance of NK cell activity to the PFOS MCL is unclear, since NK cell activity is unrelated to the endpoint used as the basis for the MCL (decreased plaque forming cell response).

137. COMMENT: Other regulatory bodies and expert health panels have made conclusions that are contrary to those of the Department regarding immunotoxicity and exposure to PFOS and other perfluoroalkyls. (42)

RESPONSE: The Department does not agree with the Australia Expert Health Panel, Food Standards Australia New Zealand, and Health Canada assessments. The Health Canada assessment did not have access to more recent studies that strengthen the evidence for an association between PFOS exposure and vaccine response and infectious disease. Similarly, it is not clear that the conclusions of the Australia Expert Health Panel and Food Standards Australia New Zealand are based on a full consideration of all the available studies. The Department also notes that immune system toxicity (decreased antibody response to a foreign antigen in mice, analogous to decreased vaccine response in humans) is the basis for the PFOS Reference Doses developed by six other states (California, Michigan, Minnesota, New Hampshire, New York, and Washington) and is also considered in the draft ATSDR (2018) PFOS Minimal Risk Level. Additionally, the EFSA (2108) tolerable weekly intake for PFOS is based on human health effects including decreased vaccine response. Furthermore, it is emphasized that human epidemiology data are not used as the primary basis for the PFOS MCL. The primary focus of the Health Effects Subcommittee's risk assessment is on the controlled animal experiments that show a clear cause-and-effect relationship between serum PFOS concentrations in mice and decreased response to a foreign antigen (SRBC). Based on these studies, it is clear that immunosuppression is a valid and adverse endpoint for PFOS exposure.

The epidemiology data provide support for the relevance of the animal data to human environmental exposure. In seven of nine peer-reviewed human studies, PFOS exposure levels prevalent in the general population were significantly associated with decreased response to at least one vaccine. These studies of vaccine response differ in the ages at which PFOS exposure and vaccine antibodies were measured, the time between inoculation and the measurement of antibody levels, the specific vaccines that were evaluated, the study populations, and the study design. Nevertheless, the observation of an association of decreased vaccine antibodies with some measure of PFOS exposure for at least one vaccine antibody in most of the studies is suggestive of an association between increased PFOS serum levels and decreased antibody response across different populations and different study designs.

Further, given the different inherent antigenicity of different vaccines, there is no *a priori* reason to expect that the effect of PFOS exposure on all vaccine antibodies will be consistent. As noted above, several of the relevant studies showing associations of PFOS with infectious disease were not available when assessments cited by the commenter were developed. These more recent studies of clinical disease and PFOS exposure are consistent with the results of the studies of vaccine antibodies and PFOS exposure. Combined, they provide strong evidence that PFOS exposure in the general population can cause immunosuppression at the clinical level. In three studies in different populations, exposures to PFOS at levels prevalent in the general population were significantly associated with increased risk/incidence of infectious disease in children, including hospitalization for infections in girls. As such, the Department concludes that the epidemiology data strongly support the conclusion that exposure to PFOS at current population levels can lead to immunosuppression. This conclusion is supported by NTP (2016) which concludes, “The evidence indicating that PFOS suppresses multiple aspects of the immune system supports the overall conclusion that PFOS alters immune function in humans.” Although the

specific mechanisms that can explain differences in responses between sexes, across different vaccines, different types of infections, or different ages at which PFOS exposure is measured are not yet clear, it is more noteworthy PFOS is associated with immunosuppression in humans at general population exposure levels in a variety of populations, across a variety of vaccines, and for a variety of infections. While these human data do not easily lend themselves to quantitative dose-response analysis, such quantitative analysis is not necessary to establish the qualitative relevance to humans of the MCL derived from the animal database.

Uncertainty Factor for Duration of Exposure

138. COMMENT: The Dong et al., 2009 study of decreased plaque forming cell response, predictive of immunotoxicity, resulted in the lowest (most sensitive) point of departure (POD) and was used as the Institute toxicity endpoint for PFOS. We concurred with this assessment but disagree with the UF (uncertainty factor) used to determine the PFOS target human serum level. The Institute applied a UF of unity (1.0) for sub-chronic versus chronic testing used in Dong et al., 2009 study even though this study of 60 days is of sub-chronic duration. Sub-chronic duration is greater than 30 day to less than or equal to 90 days. A UF of 10 is normally applied when sub-chronic is used instead of chronic testing to estimate a NOAEL.

The Institute asserts that an uncertainty factor to extrapolate sub-chronic to chronic is not needed because the immunotoxicity studies of sub-chronic duration did not show a greater effect (response) at longer duration (but within the sub-chronic duration period) among the three studies reviewed. The Institute notes that for the same PFOS serum concentration of 1 x 105 ng/ml, plaque forming cell response decreased by the same 60 percent in two studies

despite the difference in duration between these two studies, (Zheng et al., 2009) at seven-days duration and (Dong et al., 2009) at 60-days duration. The Institute asserts, therefore, that the decrease in plaque forming cell response does not progress at longer exposure duration.

Although suggestive of a lack of progression over time, these tests are of very short duration (seven to 60 days) and we believe they do not fully explain whether this premise holds true at longer chronic durations of six months or more. Further, the mechanistic basis for the immunotoxic effect of PFOS is unknown, and whether further long-term exposures may indeed accelerate this effect.

Omission of a UF for sub-chronic-to-chronic in risk assessments should not be done on the basis of results taken solely from short term studies, especially without an understanding of the mechanism of toxicity. A sub-chronic to chronic UF should be applied. In lieu of some (limited) evidence of no increase in effect in dose-response between the seven-day and 60-day short-term sub-chronic studies we would apply a UF of 3 (versus 10) as a reasonable compromise. Applying a UF for sub-chronic to chronic is important since serum PFOS levels in the general U.S. population are already near the range of central tendency serum PFOS levels in the epidemiology studies of the general population that found associations with decreased immune response. (9)

RESPONSE: The Department agrees with the Institute's conclusion stated in DWQI (2018b), "… an uncertainty factor (UF) for duration of exposure of 1 was used because studies with varying durations suggest that PFOS causes decreased PFCR [plaque forming cell response] within a short timeframe, and that this effect does not occur from longer exposure to lower doses. The use of serum PFOS levels for dose-response evaluation further support an UF of 1 for duration of exposure. Because the dose-response is based on serum PFOS levels rather than administered dose, application of an uncertainty factor to account for potential increases in serum PFOS levels with longer exposure durations is not necessary."

Benchmark dose modeling

139. COMMENT: The decision to use the Dong et al., 2009 data is further invalidated by the results of the Institute's BMD modeling, which reveals that the SRBC response data failed to provide an acceptable fit to any of the dose-response models included in the USEPA's BMD software. The inability of BMD modeling to yield a valid Point of Departure (POD) suggests that the SRBC response data reported in the Dong et al., 2009 study are not sufficiently robust. (23, 27, and 41)

140. COMMENT: The Department's analysis is silent on its inability to fit the SRBC data from Dong et al., (2009) to any of the dose-response models included in the USEPA's BMD software. (23, 27, and 41)

141. COMMENT: The Institute made a serious technical error in its evaluation of BMD modeling. This error led the Institute to use a serum PFOS NOAEL of 674 ng/mL as the POD for calculating the PFOS MCL instead of a properly calculated serum PFOS BMDL. If the Institute's BMD modeling error is corrected, and the Department uses the preferred BMD modeling approach, a serum PFOS BMDL1SD at 3,400 ng/mL can be successfully determined as the point of departure (POD)for the PFOS MCL. This would raise the PFOS MCL to 0.064 µg/L, five times higher than the proposed MCL. (42)

142. COMMENT: The Institute selected a study by Dong et al. (2009) as the POD study for deriving a PFOS MCL based on an immunotoxic endpoint. The Institute made a serious technical error in its BMD modeling by using the standard error of the mean (SEM) from the Dong et al. (2009) study, rather than the required standard deviation. This error led the Institute to reject

the otherwise preferred BMD modeling approach in this instance and to instead use a serum NOAEL of 674 ng/mL as the POD for calculating the PFOS MCL. If the Institute's BMD modeling error is corrected by using the standard deviation (rather than SEM), a serum BMD can be properly calculated and used as the POD for the PFOS MCL. Correcting the Institute's error results in a PFOS BMDL1SD at 3,400 ng/mL. Using this value as POD results in a PFOS MCL of 0.064 µg/L, five times higher than the proposed MCL. (42)

143. COMMENT: Our review of the Institute's BMD modeling discovered a major technical error in the Institute's BMD modeling. If corrected, an acceptable serum PFOS BMDL can be derived; specifically, a BMDL1SD of 3,400 ng/mL.

As the Department has recognized, a BMD and/or BMDL is the recommended and "preferred" approach for deriving a POD value. Accordingly, the Department should adopt the serum BMDL1SD and revise its POD value for PFOS. Because the serum BMDL1SD (3,400 ng/mL) is five times higher than the serum NOAEL (674 ng/mL), the PFOS MCL should be raised by a factor of five to 0.065 µg/L (0.013 µg/L x 5 = 0.065 µg/L).

The Institute erroneously used standard error and not the required standard deviation in its BMD modeling. Doses, number of animals, mean responses, and standard deviation are required to model summarized continuous response data using the USEPA's Benchmark Dose Software (BMDS).

In addition, BMDL1SD 3,400 ng/mL should be the POD for Dong et al. (2009) plaque forming cell response data. With this corrected value, the dataset from Dong et. al. (2009) was

modeled using USEPA BMDS version 3.1., a lowest BMDL1SD (3,400 ng/mL serum PFOS) and lowest AIC and was deemed to be the "best" fit for the dataset

Further, the Institute's rationale for concluding that the Dong et al. (2009) plaque forming cell response data is not amenable to benchmark dose modeling was incorrect. The Institute performed benchmark dose modeling after excluding the high dose group which yielded four models with acceptable fits to the dataset: "Restricted Hill Model, constant variance"; "Restricted Hill Model, non-constant variance"; "Unrestricted Hill Model, constant variance"; and "Restricted Hill Model, non-constant variance."

The models that assumed constant variance were rejected because the constant variance test failed (Test 2 P-value was less than 0.05), and we agree that the BMDLs calculated for these models should be used with caution. However, the version of BMDS that the Institute used, "version 2.6.0.1," was unable to calculate BMDLs for non-constant variance Hill models. This software-based limitation has since been resolved in the more recent release of BMDS version 3.1. In fact, when we repeated the Institute's analysis (dropping the top dose and incorrectly entering standard error into the standard deviation column) using the most up-to-date version of the software, there were three viable models with calculated BMDLs obtained under the assumption of non-constant variance: "Restricted Exponential"; "Restricted Exponential"; and "Restricted Hill."

Lastly, it should be noted that even if the highest dose group is included in the BMD modeling with the more recent release of BMDS version 3.1, there are no viable models that can be attained with the full dataset. The complete dataset would yield three potential models

for BMDL consideration: “Unrestricted Hill Model, non-constant variance”; “Unrestricted Polynomial, Degree 4 Model, non-constant variance”; and “Unrestricted Polynomial Degree 3 Model, non-constant variance.”

However, in the unrestricted Hill Model, the ratio between BMD:BMDL greater than five reflects large uncertainty associated with the “true” shape of the dose-response curve in the low-dose region and caution should be used when selecting BMDLs from such models (Haber et al., 2018).

The other two viable models (“Poly 4” and “Poly 3”) have multiphasic curves with multiple inflection points, which indicated non-monotonicity. Taken together, these results suggest that all three unrestricted models should be excluded from consideration with BMDL selection which would mean no viable models were attained with the full dataset. (42)

RESPONSE TO COMMENTS 139 THROUGH 143: The Department acknowledges that the standard error instead of the standard deviation was inadvertently used in the BMD modeling of the data from Dong et al. (2009) as presented in DWQI (2018a). However, the results of this modeling were not used as the basis for the PFOS reference dose or MCL because none of the models gave an acceptable fit to the data, thereby preventing the identification of a BMDL as a point of departure. The USEPA (2012) benchmark modeling guidance recommends that a NOAEL (or LOAEL if there is no NOAEL) be used as the point of departure when a BMDL cannot be developed. In accordance with that guidance, the NOAEL of 0.674 μg/ml (674 ng/ml) from Dong et al. (2009) was used as the point of departure for the reference dose.

The BMD modeling in DWQI (2018a) was performed using version 2.6.0.1 of the BMDS, the version that was available at the time. The commenter calculated Benchmark Doses for the data from Dong et al. (2009) using the more recent BMDS Version 3.1 using the full dataset and with the highest dose excluded. The USEPA (2012) states that the highest dose group should not be dropped if the full dataset provides an adequate fit. The full dataset provided BMDLs based on serum PFOS levels from three viable models: 0.83 µg/ml - Hill, non-constant variance (recommended); 7.87 µg/ml - Polynomial Degree 3, non-constant variance (alternate); and 3.79 µg/ml - Polynomial Degree 4, non-constant variance (alternate). The commenter states that none of these three viable models from the full dataset should be considered and that the recommended BMDL of 3.40 µg/ml from the modeling with the highest dose group excluded should be used instead. The reasons provided by the commenter for not considering the models from the full dataset are: 1) The two Polynomial (alternate) models have multiphasic curves with multiple inflection points which indicate non-monotonicity; and 2) the Hill (recommended) model has a BMD:BMDL ratio greater than five, and the commenter states that Haber et al. (2018) recommended that caution should be used when selecting BMDLs from such models. However, this is not an accurate representation of the conclusions on this issue from Haber et al. (2018).

Haber et al. (2018) states that the developers of the BMDS software do not provide any rationale for including the BMD:BMDL ratio as part of the decision logic for model choice or for recommending a ratio of greater than five as a cutoff point for providing a “caution” notation. In contrast, Haber et al. (2018) further states, “[w]hile one might intuitively expect that the

BMD:BMDL ratio reflects model uncertainty, a mathematical evaluation reveals that the BMD:BMDL ratio can reflect data quality." Therefore, based on the results from BMDS Version 3.0, the BMDL from the Hill model with non-constant variance of 0.83 µg/ml (830 ng/ml), which is close to the NOAEL of 674 ng/ml, would be recommended. However, BMDS Version 2.6.0.1 (the version available at the time that the Institute performed the BMD modeling) was unable to calculate benchmark doses for the non-constant variance Hill models due to a software-based limitation that has been corrected in Version 3.0.

The Department's MCLs are based on the information and models that were available at the time when they were developed, and new information and models relevant to risk assessment of PFOA and PFOS have continuously become available since that time. Similarly, the peer reviewed transgenerational toxicokinetic model (Goeden et al., 2019) developed by the Minnesota Department of Health provides valuable quantitative information on the much higher exposures in breastfed infants (a susceptible subgroup for the effects of PFOS and PFOA) from maternal consumption of contaminated drinking water. Use of this model by the Department in MCL development would result in PFOA and PFOS MCLs lower than those proposed by the Department. However, the model is not used because it was not available when the Department's PFOA and PFOS MCLs were developed. In summary, the NOAEL of 0.674 µg/ml (674 ng/ml) is used as the point of departure for the PFOS reference dose. It is noted that the USEPA (2016b, 2016c) Health Advisories for both PFOS and PFOA are also based on the NOAEL/LOAEL approach.

Adverse Effects

144. COMMENT: The Department also asserts that human exposure to PFOS has been associated with increased cholesterol. In experimental studies, PFOS has not been shown to cause an increase in cholesterol. The low dose response association based on certain observational epidemiologic data continues to remain only a hypothesis elusive of a foundational mode of action and not supported by experimental data. Association seen in observational studies is a hypothesis – no mode of action and not supported by experimental studies.

The European Food Safety Authority (EFSA) CONTAM Panel in March 2018 issued a tolerable weekly intake of 13 ng/kg body weight per week for PFOS. The EFSA CONTAM Panel considered an increase in serum total cholesterol with PFOS to be the critical effect that was based on three BMR models of cross-sectional epidemiologic studies (Steenland et al., 2009; Eriksen et al., 2013, and Nelson et al., 2010) where similar BMDL5 values were calculated (2530 ng/mL, 22 ng/mL, and 21 ng/mL, respectively). The BMDs were 27 ng/mL, 31 ng/mL, and 31 ng/mL, respectively. The much larger of the three cross-sectional studies, Steenland et al. (2009) appeared to have a plateauing of response at approximately 50 ng/mL. While the Institute's Subcommittee on Health Effects acknowledged that most of these studies were cross-sectional by design, which means temporality between exposure and outcome cannot be determined, neither CONTAM Panel nor the Institute considered a clinical chemistry study in monkeys with PFOS (Chang et al., 2017) in their assessments.

The study by Chang et al. (2017) evaluated the potential associations between serum PFOS and changes in serum clinical chemistry parameters in purpose-bred young adult cynomolgus monkeys (Macaca fascicularis). While the highest serum PFOS achieved was approximately 165,000 ng/mL, administration of PFOS to monkeys did not result in any toxicologically meaningful or clinically relevant changes in serum clinical measurements for coagulation, lipids, hepatic, renal, electrolytes, and thyroid-related hormones. A slight decrease (not increase) in serum cholesterol (primarily in HDL fraction), was observed. The corresponding lower-bound fifth percentile benchmark concentrations (BMCL1sd) were 74,000 and 76,000 ng/ml for male and female monkeys, respectively.

The findings from a mechanistic study published by Bijland et al. (2011) are corroborated by the data from the Chang et al. (2017) study of monkeys dosed with PFOS. Using the APOE*3-Leiden.CETP mouse model that expresses a human-like lipoprotein profile, Bijland et al. (2011) reported that high levels of PFOS lowered serum total cholesterol with enhanced lipoprotein lipase activity, as well as decreased the rate of HDL particle maturation. At end of the three experimental studies where these mice were fed a Western higher fat composition diet, the PFOS concentrations ranged between 85,600 ng/mL and 124,700 ng/mL. Therefore, given the toxicological data (Chang et al., 2017; Bijland et al., 2011), it is highly premature to suggest a low dose causal association between PFOS and cholesterol can be based on the current observational epidemiologic data without a well-defined mode of action.

As with the association between cholesterol and PFOA, the toxicological evidence suggests a decrease in cholesterol with high concentrations of PFOS. The low dose associations

between cholesterol and PFOS noted in certain observational epidemiologic studies, like that of PFOA, are likely due to yet-to-be discovered mode of actions or confounding factors and are not causal. (42)

RESPONSE: The Department notes that, an association refers to a statistical association between exposure and a health effect. It does not address whether the exposure caused the health effect, and neither the Institute nor the Department presented conclusions about whether or not the association of PFOS and cholesterol is causal. The EFSA (2018) tolerable weekly intake based on increased cholesterol in human studies is provisional at this time, and both DWQI (2018a) and EFSA (2018) acknowledged that most studies were cross sectional by design.

Regarding the monkey study by Chang et al. (2017) and the mouse study by Bijland et al. (2011), serum cholesterol was decreased at serum PFOS levels several orders of magnitude higher than those associated with increased cholesterol in human epidemiology studies. The decreased cholesterol at these higher plasma PFOS concentrations in both mice and monkeys is consistent with a mechanism involving activation of PPAR-alpha in both species, consistent with the action of drugs that reduce cholesterol in humans through PPAR-alpha activation.

Similar to the association of PFOA and cholesterol discussed above, the increased cholesterol in humans at serum PFOS levels several orders of magnitude lower than those at which cholesterol is decreased in both these animal studies may occur through a different mechanism. Based on review of potential factors that could result in reverse causality, EFSA (2018) concluded that “it is likely that associations between serum PFOS and PFOA levels and

serum cholesterol are causal, i.e. that increased levels of PFOS and PFOA cause increased levels of serum cholesterol."

145. COMMENT: The Department asserts that human exposure to PFOS has been associated with lower birth weight. The association with birth weight has been demonstrated to be the result of confounding or reverse causation. (42)

RESPONSE: The Department did not assert that human exposure to PFOS has been associated with lower birth weight. As stated in the Department's notice of proposal Summary, human exposure to PFOS has been associated with health effects including decreased vaccine response and increased cholesterol. The Institute in DWQI (2018a) also did not conclude that PFOA is associated with decreased birth weight, but stated "[a]lthough there is a suggestion of a relationship between maternal PFOS exposure and decreased birthweight from epidemiological studies, the evidence is not consistent."

PFOS Half-life

146. COMMENT: The Department used a human serum elimination half-life estimate of 5.4 years from a retiree occupational population in the MCL derivation for PFOS, which does not reflect overall general population demographics, as well as age-dependent renal function. The Department should use 3.4 years as the serum elimination half-life estimate for PFOS for its MCL calculation. This estimate is based on a more recent study (Li et al., 2018) of a Swedish

population whose demographic characteristics are more similar to the community population used by the Institute for its selection of a PFOA serum half-life. (42)

147. COMMENT: Chemical-specific clearance factors (CL) used in risk assessment calculations are highly dependent on human half-life estimates. The human half-life estimates for PFOS have been reported in longitudinal studies across various age-groups and populations since 2007 ranging from 3.1 to 5.4 years for PFOS.

For the PFOS MCL derivation, the Institute selected an arithmetic mean serum elimination half-life estimate of 5.4 years (Olsen et al., 2007). This half-life estimate was based on a small study of 26 retired fluorochemical workers (24 males), whose mean age at the end of the study was 66 years. While the half-life estimate of 5.4 years is the most conservative estimate reported in the literature, it is not appropriate to use in the derivation of the PFOS MCL for the reasons discussed below.

Serum elimination half-lives are dependent on several factors including age, sex, and renal clearance of the study subjects. It is well-recognized that the glomerular filtration rate (GFR), an essential component of renal clearance of PFOS and other perfluoroalkyls, substantially declines with advancing age. The overall rate of decline in GFR in healthy persons is approximately 6.3 to 8.7 ml/min/1.73m2 per decade (Berg, 2006; Linderman et al., 1985; Rule et al., 2010). Thus, the higher PFOS half-life estimate of 5.4 years, based on retired workers, is likely explained by lower GFR and slower renal clearance of perfluoroalkyls in these older study subjects. This was not considered by the Institute.

The Institute also did not recognize that approximately 85 percent of the New Jersey general population is less than 65 years of age. Therefore, it is not justifiable to use a mean half-life estimate based solely on occupationally exposed retirees (who were primarily male) with markedly lower GFRs than most of the general population. The Institute should consider alternative serum elimination half-life estimates that reflect overall general population demographics and GFRs. At a minimum, the Institute should present sensitivity analyses using these collective data.

Recently, a study by Li et al. (2018) reported PFOS half-life estimates from a community exposed to perfluoroalkyls through a contaminated water supply from a nearby military airfield. Upon the installation of GAC filters into the municipal water source, there was an abrupt mitigation of exposure to PFOS and other perfluoroalkyls in the drinking water. There were 1,006 study subjects ages four to 83 years, who were biomonitored a total of seven times during a 26-month period following the installation of GAC filters. A serum elimination half-life of 3.4 years for PFOS was reported for these 106 subjects. Males (n=20) and females (n=30), ages 15 through 50 years, had half-lives of 4.6 and 3.1 years, respectively. It is well-known that various time-dependent physiological events (for example, pregnancy, lactation, menstruation) affect clearance pathways that can result in lower concentrations in females.

Although the Institute briefly discussed the Li et al. (2018) study, they failed to provide sufficient justification for not using the overall PFOS half-life estimate of 3.4 years reported in this study (DWQI 2019). Rather, the Institute provided the following statement to justify their decision to use the half-life of 5.4 years from the Olsen et al. (2007) study:

> "Although the men in Olsen et al. (2007) were all older than 50 years of age, the mean half-life of 4.6 years for men age 15-50 years from Li et al. (2018) is in reasonable agreement with the mean half-life of 5.4 years from Olsen et al. (2007). Additionally, the 95 percent CI of 3.9-6.9 years from Olsen et al. (2007) overlaps with the 95 percent CI of 3.7-6.1 years for men age 15-50 from Li et al. (2018)."

The Institute did not address the fact that the PFOS half-life estimate of 5.4 years does not reflect overall general population demographics and age-related declines in GFR, as discussed above. Furthermore, there is no scientific basis for stating that the mean half-life of 4.6 years from Li et al. (2018) is in "reasonable agreement" with the mean half-life of 5.4 years from Olsen et al. (2007). In fact, there is a difference of 292 days between these two half-life estimates, which has a substantial impact on the derived MCL. If the Institute had used 4.6 years (1,679 days) for the PFOS half-life, based on men only, the MCL would be 15 ng/L. This MCL is 15 percent higher than the proposed MCL of 13 ng/L.

Regardless, the Institute should have used the PFOS half-life of 3.4 years (for both males and females, ages 4 to 83 years) from the Li et al. (2018) study, as this estimate is most representative of the general population. Furthermore, using this half-life would be consistent with the Institute's decision to use a half-life for PFOA (2.3 years) based on another population that had contaminated drinking water mitigated following the installation of GAC filters (Bartell et al., 2010) rather than using the PFOA half-life based on the study of retired workers by Olsen at al. (2007). Thus, the same Institute decision-making process should be considered for PFOS as it was for PFOA. If the Institute used 3.4 years (1,241 days) for the PFOS half-life while all the

other parameters remain unchanged, the CL factor would become 1.28 x 10-4 L/kg/day and the MCL would be 20 ng/L. This MCL is 54 percent higher than the proposed MCL of 13 ng/L. Relevant to this point, it is noted that the Minnesota Department of Health used the mean half-life of 3.4 years from the Li et al. (2018) study in the derivation of their 2019 Health-Based Value for PFOS. In conclusion, the Institute used a half-life estimate of 5.4 years for PFOS that was not representative of general population demographics and GFRs, and not supported by the published literature. The Institute should revise the MCL for PFOS to include a more appropriate and scientifically justifiable half-life estimate for PFOS consistent with their decision-making process for PFOA. (42)

RESPONSE TO COMMENTS 146 AND 147: In deriving the PFOS MCL, the Institute in the DWQI (2018a) used data from Olsen et al. (2007) and Li et al. (2018), which show that the half-life for PFOS is clearly longer in males than in the overall population, indicating that a half-life from males (or mostly males, as in Olsen et al., 2007) is appropriate for use in risk assessment. This is especially true because a central tendency half-life, rather than an upper percentile half-life, is used. In contrast to PFOS, it is appropriate to use the half-life for males and females combined from Bartell et al. (2019) for PFOA risk assessment because the PFOA half-life did not differ in males versus females in this study. Also relevant to this point, there were outliers with much longer PFOS half-lives than the other subjects in both Li et al. (2018) and Olsen et al. (2007). This suggests that there may be a subpopulation with much slower PFOS elimination than the mean, possibly due to physiological/biochemical differences, such as differences in the organic anion transporters responsible for reabsorption of perfluoroalkyl acids in the kidney. The

potential existence of such a subgroup further supports the choice of a longer half-life value for use in the risk assessment.

Although the PFOS half-life estimate from Olsen et al. (2007) was based on a small study of 26 individuals, the Department notes that the PFOS half-life estimate for males age 15 to 50 from Li et al. (2018) is based on an even smaller group size of 20. The commenter also states that serum was sampled a total of seven times during a 26-month period in Li et al. (2018) to measure the decline in serum PFOS levels after granular activated carbon (GAC) filters were installed. However, Li et al. (2018) states that the median number of sampling rounds per subject was six, not seven, and, in Olsen et al. (2007) the serum was sampled more frequently and longer than in Li et al. (2018) - eight times for most subjects and at least seven times for almost all subjects, for five years for almost all (24) subjects and two to three years for the other two subjects.

Further, the mean half-life in men ages 15 to 50 from Li et al. (2018) is only 15 percent lower than the mean half-life from Olsen et al. (2007), a difference that does not substantially impact the reference dose. The commenter also noted that the Minnesota Department of Health (MDH) used the mean half-life of 3.4 years for males and females combined from Li et al. (2018) to develop its recent Health Based Value (HBV) for PFOS. Relevant to this point, MDH also used a transgenerational toxicokinetic model that accounts for the much higher exposure of breastfed infants to PFOS from contaminated drinking water (Goeden et al., 2019) in developing its HBV for PFOS. An MCL based on this model would be much lower than the MCL based on the exposure assumptions used by the Department. It is noted that the MDH HBV for

PFOS (15 ng/L) is very close to the Department's MCL for PFOS (0.013 µg/l, or 13 ng/L), despite the fact that it used a less stringent half-life, relative source contribution factor, and PFOS reference dose than those used by the Department. In summary, the half-life of 5.4 years from Olsen et al. (2007) will be used in development of the Department's MCL for PFOS.

PQL for PFOA and PFOS

148. COMMENT: The PFOA PQL of six ng/L is approaching the scientific limit of what is analytically sustainable. Establishing a PQL of six ng/L requires sophisticated laboratories to perform the analyses that will limit the universe of qualified laboratories and will require specialized procedures and training for field sample preparation and collection procedures. We ask that the Department consider PQLs that will allow more local laboratories to analyze for PFOA and PFOS. (31)

RESPONSE: The Institute's Testing Subcommittee (DWQI, 2016, 2017) identified acceptable methods for New Jersey-certified laboratories to analyze PFOA and PFOS in drinking water samples and developed PQLs for PFOA and PFOS. The PQL is the minimum concentration to which the contaminant can be reliably quantified within acceptable limits of uncertainty. When developing the PQLs, the Testing Subcommittee considered performance data available at the time from robust analytical methods and accredited laboratories. Of 17 laboratories used to determine the PQL for PFOA, 12 had lowest calibration standards at or below the PQL of 0.006 µg/L (DWQI 2016). As stated in the notice of proposal Summary, the start of monitoring in 2021 will allow laboratories time to purchase equipment, train staff, and obtain certification in New

Jersey, as necessary, and to coordinate with public water systems to ensure samples are collected and reported in accordance with the adopted requirements, thus, avoiding monitoring violations. The Department will also prepare training materials, guidance documents, forms, standard operating procedures, and data systems.

In 2015, at the time that the Institute's Testing Subcommittee developed and recommended the PQL for PFNA, only six laboratories were certified to analyze PFAS in drinking water. There are currently 19 laboratories certified to conduct the required testing for PFOA and PFOS, including two in New Jersey. Therefore, the Department anticipates additional laboratories will continue to become certified as this rulemaking is implemented due to the increased testing needs.

Consumer Confidence Reports

149. COMMENT: The body of scientific data for PFAS does not support the adverse human health effects that the Department associates with PFOS and PFOA and lists in its proposed Health Effects Language for Consumer Confidence Reports. (42)

150. COMMENT: The proposed amendments to N.J.A.C. 7:10-5.2(b)4, regarding the language to be included in Consumer Confidence Reports provided by community water systems that have detections of PFOA or PFOS, contains several inaccurate statements about the potential health effects of the substances. The proposed language implies a level of certainty as to the causative nature of PFOA and PFOS exposure that does not exist. While notification of the public is an important aspect of ensuring the public's confidence in the drinking water supply, it

is essential that the information provided be accurate and avoid inflammatory and misleading statements. (27)

RESPONSE TO COMMENTS 149 AND 150: As stated in the notice of proposal Summary, the National Primary Drinking Water Regulations (National Regulations) require public community water systems to deliver a Consumer Confidence Report each year to their customers and provide information regarding the quality of the water delivered by the system. The Consumer Confidence Report summarizes information regarding sources used for drinking water, any detected contaminants, and any violations of the SDWA rules, including MCLs, as well as health effects information. Because the National Regulations do not establish an MCL for PFOA or PFOS, the Federal Consumer Confidence Report rule does not specify the health effects language that must be included in the Consumer Confidence Report if there is a detection of either compound. Accordingly, the Department's amendments update the number of State-regulated contaminants for which there is no Federal MCL and provide health effects information for systems to include in the Consumer Confidence Report.

The Consumer Confidence Report language is based on the Department's conclusions about potential health effects based on review of the human and animal studies cited in the Institute's Health Effects Subcommittee reports on PFOA and PFOS (DWQI 2017 and DWQI 2018).

Monitoring and testing for PFOA and PFOS

151. COMMENT: The Department is proposing a monitoring threshold of 0.002 µg/L for PFOA and PFOS as part of the monitoring requirements for community water systems under N.J.A.C. 7:10-5.2(a)5. While detection techniques and limits of detection will no doubt continue to improve over time, it is not clear that levels of these substances can be reliably detected at such a low level. For its latest national sampling results under the Unregulated Contaminant Monitoring Rule (UCMR), for example, the USEPA listed minimum reporting levels of 0.01 µg/L or higher for these two substances. The most recent version of the USEPA's methodology for measuring PFAS in drinking water (EPA Method 537.1) indicates that, while detection limits for the four substances range from 0.00053 to 0.0014 µg/L, "accurate quantification is not expected at [these] levels."

More importantly, however, the Department itself has determined PQLs for PFOA and PFOS of 0.004 and 0.006 µg/L, respectively. The PQL is defined as the minimum concentration to which the contaminant can be reliably quantified within acceptable limits of uncertainty. It is not clear how community water systems would be able to comply with the proposed monitoring threshold that is below the practical detection limits determined by both the Department and the USEPA. This is particularly problematic given the limitations of certified laboratories. Setting the threshold below the practical qualification limits will increase monitoring costs, generate inaccurate information, and likely encourage the use of unvalidated testing methods without providing any clear benefit. Our recommendation is to set the

threshold at 10 nanograms per liter (ng/L) or higher, allowing certified laboratories to follow validated testing methods. (14 and 27)

RESPONSE: As stated in the notice of proposal Summary, the monitoring requirements for PFOA and PFOS are based on the framework in the National Regulations at 40 CFR 141 for volatile organic compounds (VOCs), which establishes a threshold of 0.0005 mg/l (0.5 µg/l). The VOC threshold was established as a regulatory minimum detection limit above which there is a presence of the contaminant in a public water system's drinking water (see 56 FR 30,266). A public water system with a confirmed presence of the contaminant in its drinking water has an increased probability of exceeding an MCL level when compared to a water system without detections of the contaminant. The threshold is established as a detection level above which a public water system is required to increase monitoring frequency due to this higher risk. The threshold is intentionally set below the PQL, or the minimum concentration to which a contaminant can be reliably quantified within acceptable limits of uncertainty.

The National Regulations do not establish an MCL for PFOA or PFOS, and the quarterly monitoring threshold for VOCs is much higher than the Department's MCLs. Thus, the Department's threshold is established at 0.002 µg/l, which corresponds with the threshold value established for PFNA (see 50 N.J.R. 1939(a)). In 2019, approximately 1,097 public water systems voluntarily submitted PFOA and PFOS data to the Department as part of their PFNA monitoring requirements. These samples were analyzed by 11 different laboratories, with detection levels ranging from 0.0017 µg/l to 0.002 µg/l. This supports the Department's

proposed threshold of 0.002 µg/l. In addition, the threshold value recently set for monitoring PFNA is also 0.002 µg/l.

152. COMMENT: The specific impacts of implementing the monitoring program for the various sizes/types of systems (that is, private and public water systems) should consider the subsequent loading impacts to laboratories to manage the volume and reporting of samples. (19)

153. COMMENT: Requiring private well homeowners, all public community water systems and non-community water systems to begin compliance with the regulations at the same time is likely to create bottlenecks in the above areas. The Department should determine certified laboratory capacity by consulting with the Department's Office of Quality Assurance (OQA) prior to additional compliance testing. (19 and 49)

RESPONSE TO COMMENTS 152 AND 153: As stated in the Department's notice of proposal Summary, all public community water systems and public nontransient noncommunity water systems will begin monitoring for PFOA and PFOS within the first quarter of 2021. Owners of private wells subject to sale or lease will be required to test for PFNA, PFOA, and PFOS starting 18 months after the amended rules are effective. This will allow laboratories time to purchase equipment, train staff, and obtain certification in New Jersey, as necessary, and to coordinate with public water systems to ensure samples are collected and reported in accordance with proposed requirements, thus, avoiding monitoring violations. In addition, the Department

believes that this will also allow enough time to ensure there is sufficient laboratory capacity available to meet testing needs.

154. COMMENT: We request clarification on the details of compliance calculations for PFOA and PFOS and the time allowed to attain compliance with the MCL once a violation is determined. The time to attain compliance should factor in the public procurement process, request for proposals, contract award and execution, detailed design, bid advertisement, bid award and contract execution, and construction. (49)

RESPONSE: Pursuant to N.J.A.C. 7:10-5.7(a), if a water sample demonstrates an exceedance of an MCL that constitutes a violation, the supplier of water must take any action necessary to bring the water into compliance with the applicable MCL within one year after receipt of the results of the test that demonstrate an exceedance. Selecting an appropriate treatment requires a case-by-case evaluation of site-specific factors, such as the background quality of the source water, the size of the installation, and the concentration of the target contaminant in the source water. The time needed to design and install treatment will vary depending on these above factors. Pursuant to N.J.A.C. 7:10-5.7(c), the Department may extend the deadline by which the water system must come into compliance with the applicable MCL after a public hearing and its determination that the extension will not pose an imminent threat to public health.

155. COMMENT: Regarding the sampling schedule for PFOA and PFOS, we advocate that sampling of public water systems start earlier than 2021. Since PFNA sampling began in January 2019, laboratories have already had time to purchase equipment and training, so we do not support an entire year of delay and advocate that PFOA and PFOS sampling begin in the first quarter after the effective date of the rule. People have been exposed far too long to these toxic compounds that build up in human blood, even from tiny amounts in drinking water, increasing risk of disease; there is no good reason to allow exposure to continue after the effective date of this rulemaking. (9)

156. COMMENT: The timing to implement treatment should be considered as a limiting factor for the new rules, as all impacted utilities will be vying for testing, feasibility planning, engineering design, and recommended treatment procurement at the same time. (7)

RESPONSE TO COMMENTS 155 AND 156: Upon the effective date of this rulemaking, all public community water systems and public nontransient noncommunity water systems will begin monitoring for PFOA and PFOS within the first quarter of 2021. As stated in the notice of proposal Summary, the start of monitoring in 2021 will allow laboratories time to purchase equipment, train staff, and obtain certification in New Jersey, as necessary, and to coordinate with public water systems to ensure samples are collected and reported in accordance with the adopted requirements, thus, avoiding monitoring violations. The Department will also prepare training materials, guidance documents, forms, standard operating procedures, and data systems.

In addition, the amendments include a "grandfathering" provision that allows a public water system that begins compliance monitoring for PFNA prior to 2021 to submit monitoring data for PFOA and PFOS. The Department will use this data to determine whether monitoring frequency can be reduced to an annual basis. Factors the Department will consider in making this determination include whether there are at least four consecutive quarters of operational data for PFOA and PFOS with levels consistently below the MCLs. Public water systems with insufficient data will not be able to show, with confidence, that they can remain consistently below the MCL. As of February 10, 2020, 1,107 water systems have submitted monitoring data for PFNA to the Department. Of those systems, 1,092 have also submitted monitoring data for PFOA and PFOS. For these reasons, the Department expects water systems will continue to start to monitor for PFOA and PFOS prior to 2021, in order to be eligible for reduced monitoring. Further, pursuant to N.J.A.C. 7:10-5.7(a), if a water sample demonstrates an exceedance of an MCL that constitutes a violation, the supplier of water shall take any action necessary to bring the water into compliance with the applicable MCL. Water systems that start monitoring for PFOA and PFOS prior to 2021, and have detections above the MCLs, may elect to install treatment in order to lower the levels of these contaminants prior to 2021.

Treatment for PFOA and PFOS

157. COMMENT: The role of the Institute is to provide recommendations for New Jersey specific drinking water standards and MCLs within the limits of medical, scientific, and

technological feasibility for carcinogens with the goal of eliminating all adverse health effects from ingestion within the limits of practicability and feasibility for non-carcinogens.

The Department is contemplating standards which will be among the most stringent of any state or country. Proposed MCLs of low double-digit part per trillion concentrations is at the technological limit of what is sustainable for drinking water treatment processes. (31)

158. COMMENT: The granular activated carbon treatment technology chosen by the Institute Treatment Subcommittee will not consistently and reliably remove PFOA and will not efficiently remove other perfluorinated compounds (such as the short- chain perfluorinated compounds) likely to be present (and pose toxicity) in drinking waters contaminated with these chemicals. Although long-chain PFAS have similar properties, as stated by the subcommittee, there are significant differences in treatability due to differences in structural chemistry, as demonstrated at many treatment installations and research projects.

The Treatment Subcommittee recommends GAC to remove PFOS and PFOA. The subcommittee states that treatment options are expected to be the same for the long-chain PFAS (perfluoroalkyl substances) because of the compounds' similar properties (for example persistence in the environment, water solubility, similar structure, strong-carbon fluorine bonds, and high polarity). However, there are differences in chemical structure and polarity among long-chain perfluoroalkyl substances that leads to, at times, significant differences in removal capability by GAC. Specifically, the charged functional group, carboxylic or sulfonic acid, affects the adsorption capability of activated carbon. PFSAs (perfluoroalkyl sulfonic acids, for example, PFOS) are stronger acids and more hydrophobic and thus more strongly adsorbed

onto carbon compared to PFCAs (perfluoroalkyl carboxylic acids, for example, PFOA, PFBA). Therefore, perfluoroalkyl sulfonic acids (for example, PFOS) tendency to adsorb onto activated carbon is greater. Persistence in the environment usually has little relationship to treatability by GAC.

Differences in GAC capability to remove long-chain PFAS, including PFOS and PFOA, and short-chain PFAS, including PFBS and PFBA, has been demonstrated at a number of peer-reviewed research projects and full-scale installations. Reverse osmosis has been found to be superior to GAC in removal of PFOS, PFOA, and other PFAS, including short-chain PFAS, such as PFBS and PFBA. Although GAC is generally efficient at removing PFOS, it is less efficient to reliably remove PFOA and is relatively inefficient to remove shorter-chain PFAS, such as PFBA, PFBS, PFHxA, and PFPeA.

The Water Research Foundation (WRF) study of 15 full-scale water treatment systems in the U.S., including two potable reuse treatment systems, found that full-scale anion exchange and GAC column treatments were more effective at removing long-chain PFSAs (for example, PFOS) than PFCAs (for example, PFOA, PFBA) (Water Research Foundation 2016). This study also found that full-scale reverse osmosis systems demonstrated significant removal for all perfluorinated compounds, including the smallest, perfluorobutanoic acid (PFBA). The WRF study further evaluated nanofiltration (NF) for removal of PFCAs and PFSAs and noted that NF "has been deemed potentially effective (less than 95 percent) in bench-scale experiments using NF270 membranes" (WRF 2016; Steinle-Darling and Reinhard 2008). WRF (2016) indicated that

NF may be as capable of rejecting (treating) perfluorinated compounds as reverse osmosis at lower cost.

Although GAC adequately removes PFOS, PFOA is at times marginally removed, and other PFAS may be poorly removed. Reverse osmosis offers the most robust technology to consistently remove all PFAS potentially present, including PFOA and short chain PFAS (PFBA and PFBS), as well as unidentified contaminants in the drinking water. Reverse osmosis or equivalent high-pressure membrane technology (such as nanofiltration) should be required as Best Available Technology to remove PFAS in New Jersey drinking water supplies and in discharges to ground water. In some cases, pre-treatment with GAC may be appropriate to reduce the mass load of PFAs in the reverse osmosis reject, depending upon the reject discharge location. (9)

159. COMMENT: The Treatment Subcommittee advises that “GAC and/or an equally efficient technology be considered for treatment …” The term “equally efficient” is vague and fails to consider the economic impacts of technology that may have equal treatment efficiency to GAC for PFOS and PFOA, but much higher capital and operating costs than GAC. (42)

160. COMMENT: The Department appears to assume that granular activated carbon will be an effective treatment technology but does not appear to allow for other effective technologies that may already be in use, or coming into use, around the country. (14)

161. COMMENT: The recommendation of one particular technology does not allow consideration of other equally suitable technologies that are readily available. In addition, GAC is known to be less effective for removal of short chain PFAS. If additional criteria are

formulated for short chain PFAS components after installation of GAC, a different treatment system may be selected. We recommend that the Department remove the single technology reference and recognize that many new products are entering the market. (27)

RESPONSE TO COMMENTS 157 THROUGH 161: As stated in the notice of proposal Summary, the role of the Institute's Treatment Subcommittee is to evaluate the best available and feasible treatment technologies for attaining removal of the contaminants from drinking water to achieve the health-based level, while considering the limits of available testing methodologies. The Subcommittee reviewed the relevant literature, as well as data from drinking water treatment plants, including facilities in New Jersey, with full-scale treatment for long-chain PFAS, such as PFNA, PFOA, and PFOS, and concluded that the ability to remove these contaminants is not a limiting factor in setting an MCL. The Subcommittee's review did not identify any drinking water facilities treating for long-chain PFAS using treatment technologies other than granular activated carbon (GAC), such as reverse osmosis. Thus, limited information regarding alternate treatment technologies was available for review. Given the data available, the Institute recommended the use of "granular activated carbon or an equally efficient technology" but also noted that GAC is the most commonly used treatment for PFAS contamination (see http://www.nj.gov/dep/watersupply/pdf/pfna-pfc-treatment.pdf).

The Institute noted that based on site-specific factors an "equally efficient technology" may be used for a particular water system. Pursuant to N.J.A.C. 7:10-5.7(a), a water system is required to take any action necessary to remove a contaminant when the MCL is exceeded. Actions could include the use of an alternative water supply or the installation of treatment.

Therefore, the Department does not specify a particular treatment process for the removal of PFOA and/or PFOS below the MCL.

The Department reviewed the Subcommittee report and agrees with its conclusion. The Subcommittee report references multiple full-scale facilities using GAC treatment with varying influent and effluent concentrations. The combined experience establishes that GAC treatment is both practical and feasible to treat for PFOA and PFOS below the applicable MCLs. The Department recognizes that new information relevant to PFOA and PFOS treatment technologies, such as anion exchange, continues to become available and that systems should choose treatment based on site-specific factors.

New Jersey Water Bank

162. COMMENT: We understand that recovery of funds from polluters is not guaranteed, nor is it an expedient process, which emphasizes the importance for the State to refine the level of impact and estimated costs for public water systems to comply with the proposed regulations. The State will need to dedicate funds to the New Jersey Infrastructure Bank, pursue Drinking Water State Revolving Fund funding through the USEPA, or take other measures to aid public water systems in funding these improvements. Public water systems will also need to estimate projected rate adjustments necessary to plan for anticipated capital investments and adjust their long-term financing needs accordingly. (19 and 49)

163. COMMENT: The State of New Jersey and the Federal government must increase funding to assist systems make these investments through the Drinking Water State Revolving Fund. In

addition, any money recovered from responsible parties should be repurposed to assist public water systems offset these costs. The Department must work with the Board of Public Utilities and the New Jersey Department of Community Affairs to ensure they understand that costs of installation of treatment technologies must be easily recoverable within their rate structures. (28)

164. COMMENT: The New Jersey Water Bank is reporting limited availability to fund their current projects. We ask that the Department coordinate and adjust rule adoption and implementation/compliance dates based on the available Water Bank funding. (31)

RESPONSE TO COMMENTS 162, 163, AND 164: The New Jersey Water Bank, which is a partnership between the Department and the New Jersey Infrastructure Bank, offers low interest loans to eligible water systems. The Department has been working closely with the New Jersey Infrastructure Bank in recognition of the anticipated needs of some public water systems as a result of this rulemaking. The treatment of emerging contaminants, such as PFNA, PFOA, and PFOS, is a high priority for State funding. Water systems may be eligible to receive loans for installation of treatment to address levels of PFNA, PFOA, and PFOS. The Department is also currently exploring alternative sources of funding that may be available for public water systems.

Amendments to the Private Well Testing Act Rules

Private Well Testing

165. COMMENT: The Department is proposing to require all public community water systems and public nontransient noncommunity water systems begin monitoring within the first quarter of 2021. However, what is not clear is when compliance with the proposed MCLs would become effective for private well owners. (21)

RESPONSE: While the water delivered to the public by public water systems must meet all applicable State and Federal drinking water standards, private well owners are not required to meet an MCL under the Private Well Testing Act (PWTA). The PWTA requires testing at the time of a real estate transaction, or every five years for rental properties, and notification to potential buyers and tenants. Testing is intended to provide buyers and tenants with information regarding their drinking water quality. A potential home buyer or tenant can consider treatment, for example, the installation of a point of entry treatment (POET) or point of use (POU) treatment system, or alternate sources of water. Installation of treatment is at the discretion of the home or business owner.

166. COMMENT: Instead of testing at points of sale, why not require each well owner to test every five years? That way prospective sellers can ensure their well water is drinkable at the time of sale. (4)

167. COMMENT: As this test is available to homeowners, consideration could be given to offering the analysis on a voluntary basis similar to Radon testing. (15)

168. COMMENT: Testing requirements should apply to all private wells, not just those that are the subject of real estate transactions. (33)

169. COMMENT: Consideration for sampling of private wells should be given to define a regulatory compliance period, for example a three-year compliance period, within which one investigatory sample would be required to be collected and analyzed regardless of how many times the property was sold. (49)

RESPONSE TO COMMENTS 166, 167, 168, AND 169: The testing of private wells in New Jersey is required pursuant to the Private Well Testing Act (PWTA), N.J.S.A. 58:12A-26 et seq., and the Department's PWTA rules at N.J.A.C. 7:9E. The PWTA requires testing at the time of a real estate transaction or every five years for rental properties. Testing is intended to provide buyers and tenants with information regarding their drinking water quality. Testing every five years ensures renters have current test results. Although testing is only required by the PWTA during times of real estate transactions, private well owners may conduct voluntary testing of their private wells at any time and are encouraged to do so to be better informed on their drinking water quality.

170. COMMENT: Instead of addressing water quality when a home is sold, the Department should instead scrap this rule and provide funding immediately through the Spill Compensation Fund to cover testing and treatment costs in all homes with well water, not just those being sold. (35)

171. COMMENT: The State must provide funding for all homeowners with private wells through the Spill Compensation Fund, not just placing a costly requirement on those selling their homes, to test and, if necessary, treat their water for PFOS, PFOA, and PFNA. (35 and 43)

RESPONSE TO COMMENTS 170 AND 171: The Department is adding PFOA and PFOS to the DPHS rules List of Hazardous Substances at N.J.A.C. 7:1E Appendix A. The listing of these contaminants in the DPHS Appendix A List of Hazardous Substances enables an eligible person who has incurred damages due to a discharge of PFOA and PFOS to seek reimbursement from the Spill Compensation Fund under the Processing of Damage Claims Pursuant to Spill Compensation and Claims rules, N.J.A.C. 7:1J. The testing of private wells in New Jersey is required by the PWTA and N.J.A.C 7:9E. Businesses or persons who determine after the required testing that they have been damaged by the discharge of PFOA, PFOS, and/or PFNA may be eligible for compensation under the Spill Act for those damages.

172. COMMENT: Requiring that PFOS and PFOA compounds be added to the suite of compounds analyzed in private wells under the PWTA seems premature and not necessary for a large group of citizens who own private wells. As an example, northern New Jersey has many private wells in areas that are predominantly farmed or forested. They are not located in areas of known manufacturing using fluorine chemistry and/or near commercial airports or military airports that would have used fire-fighting foam. Other parts of New Jersey are similar as being a low risk for contamination. Requiring homeowners in low risk areas to be burdened with the added cost of PFAS analysis seems unnecessary. (8)

173. COMMENT: The Department should wait to review results from the 2019 sampling of public nontransient noncommunity water systems and public water systems to get an idea of

how widespread and where this contaminant is found. After reviewing the data from 2019-2020, an informed decision can then be made about the implementation into the PWTA. (17)

174. COMMENT: The Department should evaluate the test results of all public water systems prior to finalizing a PWTA testing requirement. A buyer or seller can decide if PFAS testing is necessary based on their individual needs and concerns for the health and safety of the residents. The testing should be voluntary and not mandated by any legislation. (19)

RESPONSE TO COMMENTS 172, 173, AND 174: As stated in the notice of proposal Summary, the high occurrence of PFOA and PFOS in drinking water is throughout New Jersey and has been documented through sampling conducted by the Department, public water systems, and third parties, including during Federal required sampling under the third iteration of the Federal Unregulated Contaminant Monitoring Rule (UCMR3). For example, an occurrence study was conducted by the Department between July 2009 and February 2010, to determine whether PFOA, PFOS, and eight other PFAS occur in drinking water sources throughout New Jersey, or only in the targeted study areas of the State near potential sources of contamination. Sample sites in the 2009-2010 study were located throughout New Jersey and included 33 source water samples from 31 different public water systems supplied by both surface water and ground water sources. PFOA was detected at or above 0.005 µg/l in 18 of the 33 samples. PFOS was found at or above 0.005 µg/l in nine of 33 samples. In 2019, approximately 1,097 public water systems voluntarily submitted PFOA and PFOS data to the Department as part of their PFNA monitoring requirements. As of January 2020, 169 public water systems in 19 out of 21 counties

have had detections of PFOA and PFOS over the MCLs, further supporting the Department's prior evaluations that the level of occurrence warrants Statewide testing.

175. COMMENT: The Department should wait a minimum of one year before implementing the requirement for PFNA analysis under the PWTA to allow New Jersey laboratories to purchase equipment, attain certification, and handle the additional volume of work generated by the PWTA. It will also allow for more competition, allow for more rapid turn-around-time for results, and lower the price of this test to the public.

The New Jersey laboratory community and the New Jersey real estate industry strongly opposes the immediate implementation of required testing for PFNA under the Private Well Testing Act and hopes the Department will consider the delay of implementation of PFNA testing under the PWTA for at least a year. (17)

176. COMMENT: There is not enough capacity with the current laboratories that are certified to handle the additional volume of samples generated by the PWTA. The out-of-State laboratories currently contracted to perform PFOA analysis are already near capacity with samples generated from the public community water systems and public nontransient noncommunity water systems.

When gross alpha was added to the PWTA, there was not enough capacity for certified laboratories to handle the volume and the regulation was phased in. This allowed more laboratories to get certified to handle the volume of samples with a quick turn-around time. (17)

RESPONSE TO COMMENTS 175 AND 176: To allow time to address the technical complexity of sampling and analysis for these parameters, the testing of private wells subject to sale or lease for PFNA, PFOA, and PFOS will be required starting 18 months after the amended rules are effective. There are currently 19 laboratories certified to conduct the required testing, including two in New Jersey. This number has increased from the six laboratories who were certified to analyze for PFAS in drinking water at the time of the Institute's Testing Subcommittee's recommendation in 2015. Therefore, the Department anticipates additional laboratories will continue to become certified as this rulemaking is implemented due to the increased testing needs.

As stated in the notice of proposal, it could take a minimum of 12 months for laboratories to obtain certification. Laboratories must follow the standardized procedure for obtaining certification. Prior to a laboratory submitting a complete package, it must have obtained instrumentation, as well as instrument operators with at least six months experience on the instrument technique who have participated in the appropriate training course. The exact time needed to obtain certification will depend on the circumstances at the laboratory, the quality of documentation submitted, and the results of an on-site assessment. Therefore, the requirements for testing private wells subject to sale or lease for PFNA, PFOA, and PFOS will start 18 months after the effective date of the rulemaking.

177. COMMENT: There are currently no laboratories located in New Jersey certified to run these analyses. All analyses have to be shipped to an out-of-state laboratory. (5)

178. COMMENT: There are no commercial laboratories in New Jersey certified for this parameter yet. Although several New Jersey laboratories have applied for this certification, most of these "applied" laboratories still do not have the equipment to run this analysis and have not begun the certification process. These laboratories will not likely invest in the equipment and personnel necessary to perform this analysis until the regulation is promulgated. (17)

179. COMMENT*:* The major issue is there are no accredited companies to do the testing or remediation, nor is there even a certification schooling process in place to create remediation specialists. The relatively few available to do the testing and remediation are not even located in our own State. This will cause a doubling of the length of time a seller will need to provide the test results and costs to acquire a satisfactory certificate in order to close on a sale. (46)

RESPONSE TO COMMENTS 177, 178, AND 179: There are currently 19 laboratories certified by the Department's Office of Quality Assurance to conduct the required analysis for PFOA and PFOS. Two of these laboratories are located in New Jersey. In 2015, at the time that the Institute's Testing Subcommittee developed and recommended the PQL for PFNA, only six laboratories were certified to analyze PFAS in drinking water. The Department anticipates additional laboratories will continue to become certified as this rulemaking is implemented due to the increased testing needs.

The Department disagrees that there are laboratories who have applied for certification for the analysis of PFOA and PFOS without having the necessary instrumentation. Under the Regulations Governing the Certification of Laboratories and Environmental Measurements at

N.J.A.C. 7:18, laboratories seeking certification to test for PFNA, PFOA, and PFOS will need to purchase and train in the operation of new testing instrumentation and operate the new instrumentation for a minimum of six months prior to applying to the Department for certification. Any laboratory listed as applied would be required to have the instrument before an application is submitted.

180. COMMENT: There will need to be a ramp up of testing capacity within the State, including certified laboratory capacity to handle what will be multiple thousands of PFOA and PFOS samples. The Department should consider allowing for expeditious qualifying process for out-of-State third-party laboratories to administer and provide testing analysis and results. In addition, we recommend a similar process to allow for certified field-testing companies to be qualified to perform field testing in the State. (21)

RESPONSE: The laboratory certification program includes allowances for reciprocity of out-of-State laboratories that participate in the New Jersey-National Environmental Laboratory Accreditation Program (NJ-NELAP), a program that offers certification based on nationwide criteria. This process already allows for an expedited certification process by granting certification through reciprocity with another NELAP State Accreditation Body (AB). An out-of-State laboratory must still first meet all the requirements of the regulations and methods prior to obtaining certification through another NELAP AB before reciprocity will be granted. Laboratories not participating in the NJ-NELAP must meet all the requirements of N.J.A.C. 7:18 and the method requirements before certification will be granted. The Department will not

lessen the requirements for laboratory certification in order to expedite certifications, regardless of what type of certification is sought. The Department's laboratory certification rules are in place to ensure consistency amongst laboratories and to ensure data quality meets the requirements of the programs for which the testing is being conducted. All laboratories conducting testing for the Department are held to the highest standards, and allowance for reduced requirements will not be considered. All laboratories must meet the requirements detailed in the applicable rules, regulations, and methods without exception.

181. COMMENT: The PWTA analysis has a four-week turnaround time for reporting test results from the date the laboratory receives the samples. This may be as much as three days after the water is sampled at the house with the current workload. PWTA testing may double the workload, extending the turnaround time. The earliest closing that can be scheduled is five to six weeks from the day the water is sampled. Sending samples to out-of-State laboratories is not supportive of the property sellers or buyers. (5)

182. COMMENT: We are concerned with potential time delays in obtaining analytical results given the lack of laboratory capacity at this time. As much as a two- to three-week delay is anticipated. This delay can have an enormous negative impact on the residential real estate market. While the rules do provide an 18-month delay in this mandate, we would suggest that if the changes to the PWTA are adopted that it be delayed until such time as the Department determines that sufficient laboratory capacity exists, so as not to delay real estate transactions. (8)

183. COMMENT: We are concerned about the time required to conduct PWTA testing, as there are no laboratories in New Jersey capable of testing for PFOS, PFOA, and PFNA. At the May 15, 2019, public hearing on this rulemaking, testing companies testified that obtaining results for tests could take up to one month. The timely submission of test results could be critical to real estate closings and project completions. Accordingly, the implementation of this rule should be delayed until New Jersey laboratories can be certified for testing PFOS, PFOA, and PFNA. (33)

184. COMMENT: We are concerned about the time it will take to conduct PWTA testing. As indicated at the May 15, 2019 public hearing, there are no laboratories in New Jersey capable of testing for PFOS, PFOA, and PFNA. The notice of proposal states that about 8,000 samples from private wells will have to be tested at time of sale or lease per year, which raises a concern that even though the PWTA requirements would not take effect for 18 months, there could be lengthy delays when homes are sold or that laboratories capable of conducting these tests could increase their prices due to lack of competition. At the May 15th public hearing, one laboratory testing company indicated it could take up to 15 days to get test results for a PFNA, PFOA, and PFOS test while another indicated it could take as much as one month. Given these facts and statements, at the very least, the portion of this rulemaking concerning the PWTA should not be considered until such time as there are a sufficient number of in-State laboratories capable of testing for PFNA, PFOA, and PFOS to ensure tests are run in a timely and cost-effective manner. (43)

185. COMMENT: These amendments will cause a doubling of the length of time a seller will need to provide the test results and costs to acquire a satisfactory certificate in order to close on a sale. If a seller tries to get an early jump on getting the certificate needed, and obtains it too early, the certificates/results will be too old to use for a closing, which could be four months from when they had tests done. (46)

RESPONSE TO COMMENTS 181 THROUGH 185: Individual laboratories will have different lengths of time necessary for the analysis and reporting of test results for PFNA, PFOA, and PFOS. These times will vary depending on travel/shipping time and pre-made agreements with the customer requesting the analysis, regardless of whether the laboratory is located within New Jersey. However, sellers will have the flexibility to both collect the PWTA sample early in the process of selling their home and to select a laboratory that is able to meet their time requirements. Under the PWTA rules at N.J.A.C. 7:9E-3.3, analytical results, except for total and fecal coliform remain valid for one year from the date of sample collection, except for cases when a new source of water has been installed.

The Department agrees that time is needed to address the technical complexity of sampling and analysis for these parameters and to ensure there is sufficient laboratory capacity available to meet testing needs. This is why the requirements for testing private wells subject to sale or lease PFNA, PFOA, and PFOS will start 18 months after the effective date of the rulemaking. There are currently 19 laboratories certified to conduct the required testing. The Department anticipates that this number will increase after adoption based on comments during the public hearing for this rulemaking, helping to provide sufficient laboratory capacity.

In addition, given the health effects associated with exposure to PFOA and PFOS, the Department believes that it is necessary to promulgate these amendments to the PWTA rules to require testing and notification to potential buyers and tenants regarding their water quality.

Private Well Treatment

186. COMMENT: The Institute recommends an MCL for PFOA of 0.014 micrograms per liter (µg/l) and an MCL PFOS of 0.013 µg/l. These levels are very stringent and may pose challenges for detection and treatment at the private well or very small nonpublic water system level. (21)

RESPONSE: The Institute considered the limits of available testing methodologies in recommending MCLs for PFOA and PFOS. The Institute's Testing Subcommittee determined a PQL, the minimum concentration to which the contaminant can be reliably quantified within acceptable limits of uncertainty, for PFOA of 0.006 µg/l and PFOS of 0.0042 µg/l based on data collected from laboratories. The Department agrees with these conclusions.

Treatment is not required for private well owners under the PWTA. Installation of treatment is at the discretion of the home or business owner. Unlike private wells, treatment is required for nonpublic water systems that exceed an MCL, under the Department's Safe Drinking Water Act (SDWA) rules at N.J.A.C. 7:10-12.30(h). The Department's SDWA rules do not require a specific type of treatment, so long the drinking water provided is in compliance with the applicable MCL.

187. COMMENT: The Department should clarify that NSF certified treatment systems were tested under the National Sanitation Foundation (NSF) P473 protocol, which has a performance goal of 70 ppt or less, as opposed to the lower levels proposed in the new rule. (7)

RESPONSE: As stated in the notice of proposal Economic Impact statement, the NSF P473 protocol certification evaluates whether a point-of-use (POU) treatment system can reduce levels of PFOA and PFOS. The Department acknowledges that the NSF P473 test protocol uses the combined PFOA and PFOS USEPA Health Advisory of 0.07 µg/l as the level to which a device must reduce levels of PFOA and PFOS. These devices are not certified to remove PFOA and PFOS to below the Department's MCLs for PFOA and PFOS of 0.014 µg/l and 0.013 µg/l, respectively. As stated in the Response to Comment 14, under the PWTA, treatment is not required for private well owners and installation of these systems is at the discretion of the home or business owner. Prior to purchasing a POU treatment device, the Department recommends home or business owners confirm that the device is capable of reducing PFOA and PFOS to below the Department's MCLs. Home and business owners should consult the NSF website and the manufacturer to confirm that the device is capable of meeting these recommendations (see https://www.nsf.org/consumer-resources/water-quality/drinking-water/perfluorooctanoic-acid-and-perfluorooctanesulfonic-acid-in-drinking-water).

Cost of Additional Testing Requirements

188. COMMENT: The cost of this analysis alone is $300.00. This is in addition to the major cost increase that has recently occurred due to the new testing requirements for gross alpha,

uranium and trichloropropane instituted by the Department in September 2018, and again in March of 2019. (5)

189. COMMENT: When the PWTA was adopted by the Legislature, tests were required for a limited number of contaminants and the total cost of those tests were under $200.00. Over time, more contaminants were added and costs have risen. The average test today is approximately $700.00. Adding PFOA and PFOS to the PWTA mandated contaminants will add $300.00 to this total, with some estimates being as high at $500.00. This will drive up the mandated testing cost up to $1,000 - $1,200, a significant sum. We believe this cost is burdensome and, thus, PFOA and PFOS should not be added to the PWTA list at this time. We do not believe the benefits justify the cost. We note, as well, that nothing would prevent a prospective property owner from doing these tests at the time of purchase or at some later point in time. We only object to this being a mandate under the PWTA. (8)

190. COMMENT: With regards to the costs associated with the proposed amendment to N.J.A.C. 7:9E-2.1, we are extremely concerned that if adopted, this rule would increase testing costs under the Private Well Testing Act by 43 percent. As stated during the Department's May 15, 2019, public hearing on this proposal by several laboratories present, the current testing cost under the PWTA is approximately $700.00. The notice of proposal indicates testing costs for PFOA, PFOS, and PFNA would be another $300.00, meaning the cost to conduct testing under the PWTA would increase by 43 percent. (43)

RESPONSE TO COMMENTS 188, 189, AND 190: The testing of private wells in New Jersey is required under the PWTA at N.J.S.A. 58:12A-26 et seq., and the PWTA rules at N.J.A.C. 7:9E. The testing of private

wells ensures that all buyers and sellers of real property are provided with information regarding the quality of onsite potable well water in order to protect themselves from exposure to these contaminants, if detected. Similarly, landlords of property with a well subject to the PWTA will also be required to test for these contaminants and to advise tenants accordingly. The Department acknowledges that the amendments to the PWTA will increase costs for either the seller or buyer, whomever assumes the cost of testing, as well as a landlord, or tenants, if a landlord chooses to pass the cost on. Testing costs are expected to increase by an average of $300.00 due to additional testing for PFNA, PFOA, and PFOS. However, the Department expects this cost to decrease as more laboratories are certified to perform analysis of these contaminants. In addition, given the health effects associated with exposure to PFOA and PFOS, these costs are minimal compared to the value of the water quality information provided.

191. COMMENT: The Department just implemented comprehensive and expensive changes to the PWTA in September of 2018, and in March of this year. This already raised the average cost of a PWTA test to about $700.00. This new additional test will add another $300.00 to the PWTA raising the average cost of a PWTA water test to about $1,000. In addition, field blanks may be necessary to analyze, in addition to the sample, raising the cost an additional $300.00. This places an unfair burden to consumers with the cost of this test driven high due to the lack of competition and capacity. (17)

192. COMMENT: Currently, whenever water samples for PFOS are taken, a field blank is also taken just to confirm that there is no cross-contamination. Out of the 235 samples taken so far at our laboratory, we have not had a single field blank contamination issue. For public water

systems, that's not that critical. There is a charge for the field blank analysis. For the PWTA, we recommend that the Department allow samples to be done without a field blank requirement. The reason being that if we do a sample and we get contamination, even above two PPT, not even above the MCL, the normal procedure is to run the field blank. That would increase the price for the PWTA by an additional $275.00 to $300.00, approximately. We make the recommendation that the Department waive the requirements for field blanks in PWTA. (29)

RESPONSE TO COMMENTS 191 AND 192: As stated above in the Response to Comment 17, a field blank is a water sample prepared in the field that is exposed to the same environmental conditions as the water sample used by the laboratory for compliance. The analytical methods used for testing PFNA, PFOA, and PFOS require the collection of field blanks. Analysis of field blanks is required when detections of a compound of concern are determined above the minimum reporting level. If detections are determined above the minimum reporting level, the field blank is necessary to verify that PFNA, PFOA, and PFOS have not been inadvertently introduced into the compliance sample.

193. COMMENT: There is concern that the proposal to add PFNA, PFOA, and PFOS to testing required under the PWTA, would unfairly place the burden of treating contamination on innocent property owners engaging in real estate transactions. The rulemaking indicates testing costs for PFNA, PFOA, and PFOS would increase by approximately $300.00 per well. Additionally, the Department notes that treatment systems can cost between $1,500 and

$2,000 to install. These additional costs are significant and would only be incurred by individuals who did not cause the contamination. (33)

194. COMMENT: At the May 15, 2019 public hearing, concerns were raised about the costs associated with this proposed rule and that they can be too expensive to install, as well as that the rulemaking only addresses homes being sold or rented. Clean Water Action's New Jersey Campaign Director in his testimony said, "[t]hese residents often install expensive in-home filtration systems, but low income and working-class families cannot afford these systems," and "New Jersey families should not have to spend extra money trying to ensure that the water they drink with, cook with and bathe in is safe."

We agree with both of these sentiments that, given the costs of these systems, property owners should not be required to pay for what polluters placed in their water. (43)

195. COMMENT: These are costs the State of New Jersey should cover through the Spill Fund for all homes with well water, not only homeowners when selling their homes - which would make it more expensive to and increase the time it takes to buy and sell homes, while also making it more difficult to obtain mortgages on homes with well water. (35)

196. COMMENT: There is the question as to why, if private well testing is deemed necessary, does the proposed rule only apply to cases involving the sale of properties? Why is State funding not being appropriated for this State mandate, especially as it concerns water quality? (10)

197. COMMENT: Given the serious health concerns raised by PFOA, PFOS, and PFNA, and the presumed innocence of affected property owners, public funding should be made available to test and treat for these new requirements under the PWTA. (33)

RESPONSE TO COMMENTS 193 THROUGH 197: The PWTA requires the testing of individual private wells as a condition of sale or lease of properties served by private potable wells to ensure that prospective purchasers and lessees are made aware of the quality of the drinking water source. The Department expects the testing costs to increase by an average of $300.00 due to additional testing for PFNA, PFOA, and PFOS. However, this cost is expected to decrease as more laboratories become certified to perform analysis of these contaminants. Given the health effects associated with exposure to PFOA and PFOS, the Department has determined that these costs are minimal compared to the value of the water quality information provided.

Treatment is required for nonpublic water systems but is not required for private well owners under the PWTA. As of 2019, a small GAC POET system that removes PFAS costs between $1,500 and $2,000 to install.

In addition, PFOA and PFOS will be listed in the DPHS rules List of Hazardous Substances at N.J.A.C. 7:1E Appendix A alongside PFNA, which was listed in 2018. The DPHS Appendix A List of Hazardous Substances lists all substances that, in addition to petroleum and petroleum products, are considered hazardous substances under the Spill Act. The Spill Act provides strict liability for cleanup and removal costs as a result of any discharge of a hazardous substance on this list. The listing of PFOA and PFOS to the DPHS Appendix A List of Hazardous Substances alongside PFNA, allows the Department to access hazardous substance based public funding

sources to conduct remediation when the Department determines the use of such funds is necessary to conduct remediation where the responsible party refuses to do so, is not financially viable, or is unknown. In addition, the Spill Compensation Fund may be able to make funding available to eligible persons who have incurred damages due to a discharge of PFOA or PFOS.

198. COMMENT: This proposed rule would disparately and negatively impact property owners living in more rural areas of the State, including a large portion of the 9th Legislative District, mainly consisting of Pineland areas.

There are substantial costs associated with the proposed rule for homeowners, which would only make living in our State less affordable while also detrimentally impacting real estate markets in affected areas. In the 9th Legislative District, affected areas would include those still working to recover from Superstorm Sandy and/or casino closures.

We are requesting the Department reconsider moving forward with this proposed rule and, instead, address not only the underlying economic issues but the inherent disparity in how the proposed rule would be implemented. (10)

199. COMMENT: There is concern the new costs and time constraints in this rulemaking will make it more expensive to sell homes in areas covered by well water, lead to decreased property values, will increase the amount of time it takes to sell a home, or lead to home sales falling through. (35)

200. COMMENT: The Department should keep in mind that in areas of the State where many homes received their water from private wells, home sales prices are dramatically different than that across New Jersey. For example, in March of this year, the median home sales price in New Jersey was $305,000. In that same month the median sales price in Warren County was $160,000. In Cumberland County, it was $121,500. If the cost for testing and treatment (which would likely be negotiated as part of a real estate transaction even though not required by this rule), the Department could increase the cost to purchase a home in Cumberland County by 4.4 percent and a home in Warren County by 3.3 percent, again excluding installation and maintenance costs.

While the Housing Affordability Impact Analysis states "[t]he Department anticipates the proposed amendments will have minimal impact on the affordability of housing because it is unlikely that the amendments will evoke a major change in the average costs associated with housing," we believe that the additional costs that this rulemaking would require when a home is sold are substantial, especially when considering what homes are currently selling for in certain parts of New Jersey where there are private wells. Even when using the Department's cost estimate of $2,000 for a treatment system, this would increase a home sale price by two percent in Cumberland County, again making it more expensive to purchase a home in New Jersey. (43)

RESPONSE TO COMMENTS 198 200: The testing of private wells in New Jersey is required pursuant to N.J.S.A. 58:12A-26 et seq., and the PWTA rules at N.J.A.C. 7:9E. The testing of private wells ensures that all buyers and sellers of real property are provided with information

regarding the quality of onsite potable well water in order to protect themselves from exposure to these contaminants, if detected. Similarly, landlords of property with a well subject to the PWTA will also be required to test for these contaminants and to advise tenants accordingly. The Department acknowledges that the amendments to the PWTA will increase costs for either the seller or buyer, whomever assumes the cost of testing. Testing costs are expected to increase by an average of $300.00 due to additional testing for PFNA, PFOA, and PFOS. This represents a 0.19 percent increase in the median sales price in Cumberland County, based on the U.S. Census median value of owner-occupied housing units between 2014 and 2018 of $165,500 (see https://www.census.gov/quickfacts/fact/table/cumberlandcountynewjersey/PST045218). In Warren County, this would be a 0.12 percent increase, based on the 2014-2018 U.S. Census median value of owner-occupied housing units of $260,000 (see https://www.census.gov/quickfacts/fact/table/warrencountynewjersey/PST045218). However, the Department expects the cost of testing to decrease as more laboratories become certified to perform analysis of these contaminants. In addition, given the health effects associated with exposure to PFOA and PFOS, these costs are minimal compared to the value of the water quality information provided. This is especially true in rural areas, which may not have additional water quality information from previously collected data.

Installation of treatment is at discretion of the home or business owner or tenant who can consider the installation of a POET treatment system, or alternate sources of water. As of 2019, a small GAC POET system that removes PFAS, costs between $1,500 and $2,000 to install.

In addition, as stated in the Response to Comments 193 through 197, the Spill Compensation Fund may be able to make funding available to eligible persons who have incurred damages due to a discharge of PFOA or PFOS.

201. COMMENT: The testing process for PWTA is quite onerous and is recognized as having the real potential for false positives, driving costs up even more. Under typical protocol for sampling identified in New Jersey's Field Sampling Procedures Manual (FSPM), samples are typically collected from a faucet and not directly from the well itself. The FSPM indicates "… sample as close to the well head as possible and upstream of the storage tank or any treatment system. Homeowners' plumbing systems should not be tampered with in any way, except for removal of the faucet screen (aerator) with permission of the homeowner. Under no circumstances shall a pump be pulled from a homeowner's well unless the removal is authorized by the homeowner and is carried out by a licensed pump installer."

When combining the low threshold of parts per trillion in the interim specific ground water quality criteria with the widespread use of Teflon products in pumps and plumbing equipment, materials and fixtures, the likelihood of false positives is significantly increased. Many water systems utilize LDPE, MDPE, and HDPE piping, which may influence sample results. When a positive result is obtained, the first response should be to re-sample under a more rigid sampling protocol, thus, adding to the cost for the homeowner. This added cost could include hiring a licensed plumber and/or well driller to assist the laboratory in sample collection. To do adequate testing may require that a well pump be pulled. To pull the pump using a licensed

well driller, as required by State regulation, and collect a truly representative sample from a well would be extremely costly. (8)

RESPONSE: Resampling due to a detection of a contaminant is at the discretion of the private well owner. However, the Department does not anticipate that these amendments will lead to substantial increases in private well pumps being removed in order to obtain representative samples.

The requirements for sampling a private well, including location, are established in the Department's PWTA rules at N.J.A.C. 7:9E-2.3. These requirements are not being altered as part of these amendments and remain the same for all parameters tested for under the PWTA. Samples must be collected from a primary cold water, non-aerated spigot or tap if no treatment system is in place. If treatment is in use by the private well owner, the sample may be collected by disconnecting or otherwise disabling treatment prior to collection, or it may be collected at a location prior to the water treatment system. In the case of new well construction and installation where there is no spigot or tap on the property, the sample may be collected directly at the well head.

202. COMMENT: The cost of purchasing the equipment to become certified is approximately $150,000. (5)

203. COMMENT: The equipment necessary to analyze PFNA costs over $300,000. Laboratories will be very hesitant to make that huge investment before regulations are enacted to regulate this compound under the PWTA. When the Department considered regulating

perchlorate, several years ago, laboratories invested in the analytical equipment to perform this analysis, and then the Department reversed its position and never regulated perchlorate. And investment of $50,000 for this equipment was a mistake for these laboratories. (17)

RESPONSE TO COMMENTS 202 AND 203: The Department estimates that the cost of purchasing the required equipment will most likely be above $150,000. Laboratories will need to consider this cost when deciding if they wish to obtain certification. The requirements for testing private wells subject to sale or lease PFNA, PFOA, and PFOS will start 18 months after the effective date of the rulemaking. The Department anticipates that the delayed implementation period will be sufficient to allow laboratories time to consider their investment with more confidence and obtain the required instrumentation and certification.

Amendments to the Ground Water Quality Standards

204. COMMENT: We support that Ground Water Quality Standards be adopted for PFOA and PFOS to protect groundwater quality and to provide uniform standards for the cleanup of contaminated sites; the proposed MCLs would become the standards. Independent toxicological reports commissioned by Delaware Riverkeeper Network (DRN) advocates even stricter standards to protect the developing fetus and young children and to utilize endpoints that are more sensitive. We support the Ground Water Quality Criteria and Standard be lowered to no more than five ppt for PFOS and for PFOA, one ppt, or alternatively, no higher than six ppt. (9 and 37)

RESPONSE: The numerical values promulgated as MCLs are the same as the promulgated ground water quality standards for PFOA and PFOS. However, in accordance with N.J.A.C. 7:9C-1.7(c)3i, the health-based levels used to establish the MCLs are the specific ground water quality criteria for the constituents. Further, the ground water quality standard (referred to as a "constituent standard") is the higher of the criterion and the applicable PQL. For both PFOA and PFOS, the ground water quality criteria are higher than the corresponding PQL. Therefore, the ground water quality standards for each constituent are the same as the MCLs. The basis for the MCLs are generally discussed in Response to Comment 53 above.

205. COMMENT: For PFOA and PFOS combined, we recommend a Ground Water Quality Criteria and Standard no higher than 11 ppt. (9)

RESPONSE: As discussed in the Response to Comments 30 and 31, the ground water quality standards for PFOA and PFOS are based on the health-based levels recommended by the Institute used to establish the respective MCLs, in accordance with N.J.A.C. 7:9C-1.7(c)3i. The Department developed the MCLs for PFOA and PFOS individually, not for the total concentration of both compounds. The potential for additive toxicity of PFOA and PFOS is acknowledged in DWQI (2017a) and DWQI (2018a). However, the toxicological effects and mode of action of PFOS differ in some respects from PFOA. Additionally, because the dose-response for some health effects is steepest at low exposures and approaches a plateau at higher exposures, dose-response for mixtures may be complex and dose-dependent (Post et al., 2017). Although cumulative effects were not considered in developing the Health-based MCL,

an important benefit of addressing exceedances of the Health-based MCL is that treatment removal processes intended to remove PFOA and/or PFOS may also partially or totally remove other types of PFAS, and other unrelated contaminants that may be present at levels of public health concern (Post et al., 2017).

206. COMMENT: This rulemaking estimates that nearly 5,500 active groundwater remediation sites in the State could be potentially impacted by the proposed GWQS. Based on the Department's estimate of the percent of public water systems that exceed the proposed MCL for PFOA (17 percent), one can estimate that more than 900 of the active sites will be required to conduct sampling, laboratory analysis, and treatment for PFOA and PFOS contamination. Although some of these sites may already be impacted by the groundwater standard for PFNA, the drinking water data suggest that the number of sites already conducting per- and polyfluoroalkyl substances (PFAS) remediation will be small.

The New Hampshire Department of Environmental Services (NHDES) estimates capital costs of up to $2.2 million for waste sites to treat PFOA and PFOS contamination, plus as much as $1.0 million in annual maintenance costs. Using these estimates, the total cost impact of the proposed groundwater standards is staggering – as much as $2 billion in capital costs and $900 million in annual operating costs. (27)

RESPONSE: The commenter's estimate of sites that will require ground water remediation is not valid because the commenter assumes that the percentage of public water systems that exceed the MCL for PFOA is the same as that for active ground water remediation sites. In the

Department's experience, the rate of PFAS detections at a ground water remediation site is dependent on the sources of contamination at that site, and public water supply well contamination is not necessarily correlated with the number of ground water remediation sites. A ground water remediation site may contaminate multiple public water supply wells, or none at all. Thus, the number of impacted wells and remediation sites do not have a linear correlation.

207. COMMENT: Currently, the only practical groundwater remediation technology is pump-and-treat. It is indicated that, at active remediation sites, the new MCLs will be used as criteria. At all 14 sites investigated, PFOS was identified above the criteria and additional remedial effort is required. The data shows the widespread diffuse nature of these compounds and elevated background concentrations. Given the ubiquitous nature of PFOA and PFOS, no practical remedial endpoint can be achieved other than infinite pump-and-treat, and it will be infeasible in many cases to attribute PFAS components in groundwater to individual site owners and sources; we request that the Department considers this consequence when adopting the MCLs as GWQS for active and new remediation projects.

In relation to this, it is noted that there is currently no groundwater in-situ treatment technology available that is proven at field scale, and groundwater remediation will rely on pump-and-treat systems. Given the ubiquitous nature of the compounds, large scale extraction of groundwater will be required, which may cause significant depletion of groundwater resources and may negatively impact groundwater resources. We request that the Department

considers both quality, as well as quantity (volumes) aspects of groundwater resources when setting targets. (27)

RESPONSE: Remedial action selection for contaminated sites must be conducted in accordance with the Technical Requirements for Site Remediation at N.J.A.C. 7:26E-5.1, which requires site specific evaluation to determine the necessary actions required to reduce or eliminate exposure to contaminants to the applicable standards. The rules require an evaluation of many factors including, but not limited to, the use of engineering and institutional controls and whether the proposed groundwater remediation will be active or passive in achieving the objectives. The potential impacts to ground water resources will be considered as part of this evaluation.

208. COMMENT: Given the extremely low proposed standards, operation of a pump-and-treat system will result in true and significant economic hardship for small businesses. These materials were used by small businesses under the assumption that they were safe, and, in fact, were often used for the purpose of employee safety. System operation and treatment media disposal costs will be significant over time. We recommend that the Department include hardship provisions in these rules to protect small businesses from financial ruin. (27)

RESPONSE: The Department acknowledges the commenter's recommendation. As stated in the Economic Impact statement in the notice of proposal, cost for remediation will vary based on site specific circumstances. The Department has estimated that the costs of installing a GAC pump and treatment system for ground water remediation will be similar to treatment costs for

water systems. Costs associated with the operation and maintenance of a GAC system, which include periodic regeneration or replacement of the carbon, will vary depending on such factors as contaminant loading, the background quality of the source water, the size of the installation, and the concentration of the target contaminant in the source water.

209. COMMENT: Does the Department have authority to require the investigation of PFAS (that is, regulation by website, statutory authority to add new compounds on ISRA case after PA)? How does the establishment of GWQS translate into an affirmative obligation on the part of a person responsible for conducting the remediation of a site contaminated with a substance or substances other than PFOA/PFOS to evaluate whether there is the potential for PFOA/PFOS contamination? (34)

RESPONSE: As stated in the Response to Comment 207, remedial action selection for contaminated sites must be conducted in accordance with the Technical Requirements for Site Remediation at N.J.A.C. 7:26E-5.1, which requires site specific evaluation to determine the necessary actions required to reduce or eliminate exposure to contaminants to the applicable standards. The rules require an evaluation of many factors including, but not limited to, the use of engineering and institutional controls and whether the proposed groundwater remediation will be active or passive in achieving the objectives.

Amendments to the Discharges of Petroleum and Other Hazardous Substances (DPHS) Rules (N.J.A.C. 7:1E), Appendix A: List of Hazardous Substances

210. COMMENT: Ongoing releases must be identified, publicized, and remediated. Acute sources of PFAS to the environment, largely from military bases and airports where firefighting foam is commonly used, must be identified and monitored. The public must be made aware of these threats, so that they can make informed decisions and advocate for their own health interests. The Department must ensure PFAS waste is properly disposed; at a minimum, PFOA and PFOS should be added to the List of Hazardous Substances under the Spill Compensation and Control Act. The Department should kickstart the cleanup process, so that contaminated sites do not serve as ongoing sources of PFAS contamination into drinking and surface waters. (30)

RESPONSE: Upon adoption, PFOA and PFOS will be listed as hazardous substances on the Discharges of Petroleum and Other Hazardous Substances rules at N.J.A.C. 7:1E Appendix A. N.J.A.C. 7:1E Appendix A lists all substances that, in addition to petroleum and petroleum products, are considered hazardous substances under the Spill Compensation and Control Act (Spill Act), N.J.S.A. 58:10-23.11 et seq. The Spill Act provides strict liability for cleanup and removal costs resulting from any discharge of a hazardous substance.

In addition, the listing of PFOA and PFOS under the DPHS Appendix A List of Hazardous Substances will also require owners and operators of industrial establishments who are subject to the Industrial Site Recovery Act (ISRA), N.J.S.A. 13:1K-6 et seq., to remediate applicable sites prior to their sale or transfer or upon cessation of business operations. This will ensure that hazardous substances existing at industrial sites are remediated prior to their transfer to a new owner or operator. The inclusion of PFOA and PFOS to the DPHS Appendix A List of Hazardous

Substances will impose upon all responsible parties, regardless of the environmental statute they are liable under, the obligation to identify and remediate PFOA and PFOS discharges.

211. COMMENT: The Department proposes to add PFOA and PFOS and hazardous substance under the Spill Act. The Department failed, however, to provide specific and adequate explanation and justification for this addition. The Department merely makes a conclusory statement that it "has determined that because PFOA and PFOS in the environment pose an unacceptable risk to public health, it is appropriate to include PFOA and PFOS on the DPHS Appendix A List of Hazardous Substances." The Department merely offers a passing comment as to the persistence of PFOS and PFOA and a short recap of alleged health effects, for which no causation has ever been established in the scientific literature. The Department makes no effort to explain why the presence of PFOA and PFOS in the environment pose an unacceptable risk to public health. The Department's rulemaking instead spends the bulk of its text discussing the outcome of adding PFOS and PFOA onto the list hazardous substances, such as cleanup liability under the Spill Act. (42)

RESPONSE: As discussed in the responses to other comments and in the Institute's recommendations to the Department, PFOA and PFOS are developmental toxicants, liver toxicants, and immune system toxicants that are possibly carcinogenic and bioaccumulate in humans. In addition, PFOA and PFOS are extremely persistent in the environment and soluble and mobile in water. Therefore, the Department has determined that PFOA and PFOS pose an unacceptable risk to public health. Accordingly, the Department is adding PFOA and PFOS to the

DPHS Appendix A List of Hazardous Substances to help address the extensive areas of PFOA and PFOS contamination in New Jersey for which no party has acknowledged or assumed responsibility to remediate. To further prevent contamination of the waters in this State with PFOA and PFOS, the Department has determined that additional protection is necessary. The discharge prevention and control provisions of the DPHS rules, as applicable to major facilities, will help ensure ongoing protection of the waters in this State from potential PFOA and PFOS contamination.

212. COMMENT: The inclusion of PFNA, PFOS, and PFOA compounds as regulated "hazardous substances" under N.J.A.C. 7:1E, Discharges of Petroleum and Other Hazardous Substances Rules, would make firefighting foams and their associated aboveground storage tanks, at major facilities, subject to all the requirements of N.J.A.C. 7:1E, which offers little benefit to the protection of the environment at a significant cost. (27 and 47)

RESPONSE: Major facilities that store aqueous film forming foam that contains PFOA and/or PFOS will be subject to all the requirements of the Spill Act and DPHS rules, including those for the transfer and storage of hazardous substances. PFOA and PFOS are developmental toxicants, liver toxicants, and immune system toxicants that are possibly carcinogenic and bioaccumulate in humans. In addition, PFOA and PFOS are extremely persistent in the environment and soluble and mobile in water. Therefore, the Department has determined that PFOA and PFOS pose an unacceptable risk to public health and it is appropriate to add these contaminants to the DPHS Appendix A List of Hazardous Substances.

The Department additionally notes that many commonly used aqueous film forming foams contain glycol ethers, zinc compounds, or other substances which may be currently regulated as hazardous substances. Thus, the addition of PFOA and PFOS to the DPHS Appendix A List of Hazardous Substances may have minimal impact on affected facilities.

213. COMMENT: The inclusion of PFNA, PFOS, and PFOA compounds as regulated "hazardous substances" pursuant to N.J.A.C. 7:1E would require notification to the Department Hotline if any firefighting foam were "discharged," except as provided under N.J.A.C. 7:1E-5.3(e). Regulating lawful use of firefighting foam in the same manner as the discharge of other hazardous substances does not seem reasonable and could result in numerous additional notifications adding burden to the reporting system. The definition of "discharge" excludes actions pursuant to conditions of a valid Federal or State permit. It is recommended that the lawful use of firefighting foams be considered in the same manner as actions pursuant to valid Federal or State permits pursuant to N.J.A.C. 7:1E-5.3(e) and, thus, be exempted from the "discharge" notification requirement to the Department Hotline.

N.J.A.C. 7:1E-1.11(a) specifies, "No person shall cause, suffer, allow or permit a discharge of a hazardous substance." Use of firefighting foams containing these compounds in emergency circumstances is a lawful use and may be necessary to protect lives, assets, and minimize air emissions from open burning. In certain circumstances, such use might result in a "discharge," which would, under the proposed rules, represent a violation and unreasonably and unfairly subject the user to penalties under N.J.A.C. 7:1E-6. In addition, a "major facility"

subject to the rules may have limited control over emergency providers (for example, municipal fire departments) who might use foams containing the referenced compounds at the facility in emergency circumstances or require the facility to test the foam system that might result in a "discharge," yet the facility might be held accountable and subject to penalties. Sufficient time frames are required for alternative foams to be developed and replace the existing foams. It is recommended that the Department grant a three-year delay for the applicability of the rules to firefighting foams containing the referenced compounds. (27 and 47)

RESPONSE: A discharge is defined at N.J.A.C. 7:1E as any intentional or unintentional action, unless pursuant to, and in compliance with, the conditions of a valid and effective Federal and State permit, resulting in the releasing, spilling, pumping, pouring, emitting, emptying, or dumping of a hazardous substance into the waters or onto the lands of the State. Because PFOA and PFOS are hazardous substances that are extremely persistent in the environment, it is important that the Department is notified when aqueous film-forming foam (AFFF) containing these substances are used. Therefore, if an AFFF containing PFOA and/or PFOS is discharged for any purpose, the responsible party must comply with the reporting requirements at; N.J.A.C. 7:1E, and conduct remediation as necessary.

Although notifications have been issued to reduce or eliminate the use of AFFFs that contain PFOA and PFOS, the Department recognizes that this material may still be in use. The Department intends to use its enforcement discretion, when considering first responders in an emergency situation.

To reduce the risk of future use of AFFF containing PFOA and PFOS, the Department and the Department of Community Affairs are spearheading an effort to "take-back" AFFF made before 2003, which contains high levels of PFOA and PFOS. Both State agencies are committed to working closely with communities and local governments to reduce the use of this material. Additionally, in July 2019, the Department of Community Affairs provided to all New Jersey fire departments guidance from the State Fire Marshall, Division of Fire Safety, which urges New Jersey fire departments to restrict the use of AFFFs to critical operations in which alternative options are not available.

In addition, to help defray the expense associated with remediating sites contaminated by AFFF containing PFOA and PFOS, the Department filed a lawsuit in 2019 against the manufacturers of AFFF products and the companies that supplied the PFAS that was used to make those products. The lawsuit seeks to hold these companies responsible for the harm caused by the products they manufactured. The lawsuit alleges that the manufacturers of these products understood the environmental and health risks associated with their products, but concealed those risks from users, including New Jersey firefighters. The Department's lawsuit seeks to have these manufacturers pay for cleanup and other costs.

As discussed in the Response to Comment 214, the Department will grant the owner or operator of a major facility a reasonable period of time, in light of all circumstances, including economic feasibility, to upgrade existing equipment and procedures to meet the requirements at N.J.A.C. 7:1E-2, provided that the facility proves to the Department that such a time period is needed. The facility must provide a reasonable schedule in which it will become compliant with

the amendments. The Department may approve or deny any proposed schedule based on the submitted information.

214. COMMENT: It is recommended that the Department grant a three-year compliance period for engineering, design, construction, and installation of impermeable secondary containment or diversion system on firefighting foam tanks. Promulgation of the proposed rule without modification would immediately put many facilities in a non-compliant situation until impermeable secondary containment or diversion systems to accommodate the largest tank plus six-inches of precipitation could be constructed. (27 and 47)

RESPONSE: In accordance with N.J.A.C. 7:1E-1.11(b), the Department will grant the owner or operator of a major facility a reasonable period of time, in light of all circumstances, including economic feasibility, to upgrade existing equipment and procedures to meet the requirements at N.J.A.C. 7:1E-2, provided that the facility proves to the Department that such a time period is needed. The rate of upgrades should be proposed as part of the Discharge Prevention, Containment, and Countermeasure (DPCC) and Discharge Cleanup and Removal (DCR) plans submitted to the Department. The facility must provide a reasonable schedule in which it will become compliant with the amendments. The Department may approve or deny any proposed schedule based on the submitted information.

Amendments to the New Jersey Pollutant Discharge Elimination System (NJPDES) Rules

215. COMMENT: N.J.A.C. 7:14A-4 Appendix A is applicable to all NJPDES permits. However, only discharges to ground water permitting are within the described intended scope of the proposed rules. The Department did not consider the effect their rulemaking would have on other NJPDES permits. (23 and 27)

216. COMMENT: Table VI is being added to N.J.A.C. 7:14A-4, Appendix A. N.J.A.C. 7:14A-4, Appendix A is applicable to all NJPDES permit applications. Only NJPDES-DGW permitting, however, is within the described intended scope of the proposed rules. Effect on other permits (for example, surface water, stormwater) was not considered and addressed in the analysis by the Department supporting the proposed rules. (47)

RESPONSE TO COMMENTS 215 AND 216: This rulemaking creates a new table labelled "Table VI: Toxic Pollutants and Hazardous Substances Required to be Identified by Dischargers if Expected to be Present." This Table is referenced at N.J.A.C. 7:14A-7.9, General requirements for applications for discharge to Ground Water Permits, which only applies to NJPDES-DGW permits. Therefore, Table VI is not applicable to any other NJPDES discharge categories.

217. COMMENT: The Department should provide clarification on whether the minimum annual PFNA, PFOS, and PFNA sampling requirement will be imposed on surface impoundments, monitoring wells as listed in the NJPDES-DGW permit, or both. (47)

RESPONSE: The Department is not requiring sampling for these parameters in lined surface impoundments since these units are not designed to discharge to ground water. These regulated units are required to ensure a permeability rate of 1×10^{-7} cm/sec for the liner. Since

these regulated units are designed not to discharge to ground water, the requirements of these rule amendments are not applicable. However, as with any unintentional discharge, the discharger is still liable and required to comply with the GWQS if the liner were to fail.

Sampling for PFNA, PFOA, and PFOS in ground water monitoring wells will be required for targeted facilities that do not have a regulated discharge outfall, such as facilities that utilize natural attenuation to meet the ground water quality limits at the property boundary or nearest sensitive receptor. Facilities with discharges to ground water that the Department has reason to believe may contain PFAS will be required to monitor for PFNA, PFOA, or PFOS at a point prior to discharging to the preferred discharge mechanism, such as infiltration/percolation lagoons or underground injection. If discharge sampling at a facility indicates the presence of PFNA, PFOA, and/or PFOS above the GWQS, the Department's preferred alternative response to the contravention of the GWQS would be to identify the contributing source and remove the source from the waste stream. If that option is not possible, the permittee has the option to provide treatment prior to the discharge or provide documentation that natural attenuation in existing ground water will allow the permittee to achieve the GWQS at downgradient ground water monitoring wells, in accordance with N.J.A.C. 7:14A-7.6, prior to the nearest sensitive receptor or the property boundary. The latter option would require monitoring of ground water monitoring wells. Based on best available science, it is unlikely natural attenuation will be a compliant method.

218. COMMENT: The Department should provide clarification on whether NJPDES-DGW permits will be modified immediately to include annual sampling requirements or not until the next five-year permit renewal cycle. (47)

RESPONSE: The Department will incorporate sampling requirements for PFNA, PFOA, and PFOS in NJPDES-DGW permits immediately subsequent to the effective date of this rulemaking through Department-initiated modifications to existing permits and through renewals of permits that are currently expired.

NJPDES Testing

219. COMMENT: The ability for EPA Method 537 to accurately test for the PFOA/PFOS compounds in treated wastewater that may have been chlorinated or treated for odor and corrosion control is disputed. EPA Method 537 has limitations on total dissolved solids (TDS) and total organic carbon (TOC). Wastewater treatment facilities that discharge to ground water also add alkalinity and hardness can approach 300 mg/l. (20 and 38)

220. COMMENT: The rulemaking does not provide, with any rigor or review or background basis to, support any analytical sampling and analyses of a wastewater matrix. The rulemaking has not provided legal or scientific support to validate sampling results that are not potable. How can a Discharge to Ground Water facility sample and track down a source or discharger if the Department has not offered any, not one, supporting report on testing treated and raw wastewater matrix and associated interferences? (20 and 38)

221. COMMENT: There are concerns regarding the ability to adequately analyze wastewater for PFOA and PFOS, as there is no USEPA approved method. (11)

222. COMMENT: There are currently no widely accepted methods for ground water and effluent matrices. USEPA Method 537.1 is a drinking water method that has not been developed and validated for analysis of ground water and effluents. These matrices are prone to interferences from other natural or manmade constituents. Given the proposed extremely low standards, the regulated community will not be able to provide reliable data of known quality in the absence of appropriate analytical techniques. The analytical determination error resulting from the application of inappropriate analytical techniques will render compliance with these standards impossible, and the Department should wait until the USEPA has published appropriate analytical methods. (23 and 27)

RESPONSE TO COMMENTS 219, 220, 221, AND 222: It is correct that there are currently no official USEPA- or Department-approved methods to quantify PFAS substances in non-drinking water matrices. Consequently, the Department's Office of Quality Assurance certifies laboratories for user-defined, or laboratory developed, methods in non-potable water. The OQA ensures that these user-defined methods include rigorous quality control measures, including those that provide a measure of the method's suitability to a given matrix. This includes either the use of isotope dilution or the use of surrogate compounds. Additionally, each extraction batch must contain a matrix spiked sample, providing further information regarding matrix effects. Also, these user-defined wastewater methods would account for TDS, TOC, alkalinity, and hardness variations associated with wastewater.

Currently, there is a sufficient number of laboratories certified to sample for PFNA, PFOA, and PFOS using these methods. Accordingly, NJPDES permits requiring PFNA, PFOA, and PFOS sampling will

include language specifying the requirement to utilize a laboratory certified by the Department for the user-defined methods until such time as the USEPA publishes an applicable analytical method. As for the reference to "raw" wastewater, all sampling in NJPDES-DGW permits would be on the effluent after treatment and not the raw wastewater influent.

223. COMMENT: Many of the referenced compounds are ubiquitous in the environment and arise from various historical sources. Where the permitted discharge to groundwater is stormwater and not a process wastewater, the presence of the compounds could be unrelated to the permitted facility and due to off-site sources, such as precipitation, that are outside the control of the permitted facility. Their presence in groundwater might also be due to historical activities unrelated to the permitted discharge to groundwater. The permitted facility might, therefore, be held accountable for costly removal or treatment of the compounds unrelated to the permitted discharge to groundwater. These compounds should not be incorporated into the NJPDES DGW permitting program at this time because of the ubiquitous nature of these compounds and likelihood their presence would be unrelated to the permitted discharges to groundwater. The permitted facility might be held accountable for costly removal or treatment of the referenced compounds unrelated to permitted discharge to ground water.

In view of the possibility that these compounds may be present from sources other than the permitted discharge to groundwater, establishment of background concentrations of these compounds in background monitoring wells and statistical evaluations should also be considered and incorporated as appropriate into DGW permits. (23 and 27)

RESPONSE: When background concentrations, in the context of the NJPDES program, prevent a permittee from complying with the ground water quality standards, permittees will be required to demonstrate compliance with the PFNA, PFOA, and PFOS ground water quality standards prior to discharge, by the property line or sensitive receptor.

224. COMMENT: The Department should not require monitoring if PFAS may only be expected due to their presence in source waters and not through the manufacturing process. (23)

RESPONSE: At this time, the Department is prioritizing the requirement to monitor for PFNA, PFOA, and/or PFOS for NJPDES-DWG permits on facilities where the presence of these compounds is reasonably expected, such as publicly owned treatment works, aquifer storage and recovery facilities, airports, military bases, and manufacturing facilities with potential to have these contaminants in their discharge. Facilities that do not have manufacturing processes that currently use, or have previously used, PFAS, and do not fit into the other listed categories where the presence of PFAS is reasonably expected, will not be required to monitor for PFNA, PFOA, and/or PFOS. For facilities that are required to sample and subsequent sampling events demonstrate that these contaminants are not present in the discharge, the permittee may, under the existing rules, request a major modification to reduce or eliminate sampling to these contaminants in accordance with N.J.A.C. 7:14A-7.6(f).

225. COMMENT: Where the permitted discharge to ground water is stormwater and not process water, the presence of the referenced compounds could be unrelated to the permitted facility and due to off-site sources, such as precipitation, that are outside the control of the permitted facility. (23 and 27)

RESPONSE: If offsite sources, like precipitation, contain PFNA, PFOA, and/or PFOS and the stormwater is directed to a regulated discharge to groundwater unit, such as an infiltration lagoon, the facility must comply with the Department's groundwater quality standards at N.J.A.C. 7:9C. The Department is requiring monitoring of PFNA, PFOA, and/or PFOS in cases where these compounds are reasonably expected to be present in the facility's discharge to groundwater. This may include facilities that are reasonably expected to store or process source materials containing PFNA, PFOA, and PFOS that direct stormwater runoff to regulated discharge to groundwater units. Facilities that have regulated discharges of stormwater to groundwater are required to have drainage control at the facility, which requires the stormwater to be directed away from source material on-site. Best management practices are also implemented to reduce or eliminate potential contact with stormwater that cannot be directed away from source material on-site. If PFNA, PFOA, and/or PFOS are detected above the ground water quality standards, the NJPDES-DGW permit will require an investigation of the source and removal (if possible) from the waste stream. If the investigation determines that the presence of these compounds is due to off-site sources, the permittee will need to either update its drainage control plan to eliminate run-on from other properties or sample the precipitation during the storm event to determine compliance with the ground water quality

standards. A permittee may also evaluate background quality, such as precipitation, to determine its contribution to a facility's compliance with the standards. In the event PFNA, PFOA, and/or PFOS are detected in the background, like precipitation, below the applicable standard and the facility's ground water discharge data also demonstrates concentrations below applicable ground water quality standards, then the facility is in compliance with the standards. However, if a combination of background contribution and source material exposure yield monitoring results above the applicable ground water quality standards, the permittee is obligated to take measures to ensure compliance. As mentioned above, if non-compliance is the result of stormwater contacting source material containing PFNA, PFOA, and/or PFOS, then the permittee should immediately take measures to eliminate or minimize exposure of the source material to stormwater.

NJPDES Treatment

226. COMMENT: The rulemaking does not address the ability for GAC to treat wastewater that contains millions to billions of bacteria per gallon. This microscopic life would prematurely foul the GAC or resin unit. (38)

RESPONSE: There are seven municipal wastewater treatment plants with NJPDES permits for discharges to ground water, which all provide disinfection prior to discharge. Disinfection significantly reduces, if not eliminates, all bacteria present in the wastewater. The required effluent limitation for fecal coliform bacteria in NJPDES-DGW permits is 200 colonies per 100 milliliters. If a facility was unable to achieve this requirement, modification or upgrade to the

disinfection system would be required. The GAC used to treat for PFNA, PFOA, and/or PFOS should be installed after the disinfection to prevent any premature fouling of the GAC (or resin unit) resulting from bacteria.

227. COMMENT: The notice of proposal Summary states that dischargers to ground water can use the same treatment technology as the drinking water purveyors, which is incorrect. Because of contaminants in wastewater and the volumes and velocity of a discharge, carbon filters are not a treatment option. (8)

RESPONSE: In general, there are some contaminants in wastewater that could adversely affect the performance of GAC, however, in order to comply with the requirements of the NJPDES discharge to ground water permit, tertiary treatment is typically provided. This level of treatment generally removes the contaminants that would adversely affect GAC treatment. The potential issues with volume and velocity associated with wastewater treatment plants can be overcome through standard engineering modifications to these plants. Based upon literature research and data from the USEPA, GAC is a viable method of treatment for PFOA, PFOS, and PFNA, as well as many other organic compounds. As discussed in the Response to Comment 29, although the USEPA has determined that wastewater constituents, such as biological oxygen demand (BOD), organics, and total suspended solids (TSS) can adversely affect GAC treatment, it is TSS the constituent of most concern for a GAC system (see https://www3.epa.gov/npdes/pubs/carbon_absorption.pdf). In order for GAC to perform efficiently, TSS concentrations should be less than 20 mg/l. As for the quality of the wastewater

associated with the seven municipal wastewater treatment plants that discharge to ground water and will be required to sample for PFNA, PFOA, and PFOS, these seven plants are presently meeting ground water quality standards for parameters of concern, as well as providing disinfection prior to discharge. The Department has examined the sampling results submitted over the past three years from the municipal wastewater plants in question and determined that TSS and BOD concentrations in the discharge of these plants should not adversely affect GAC treatment for PFNA, PFOA, and PFOS. The highest TSS average over that time period was 14.5 mg/l, and several facilities had TSS averages at five mg/l or less. Therefore, based on the Department's assessment of the effluent quality, GAC is a viable treatment option.

228. COMMENT: The costs associated with a treatment system to remove pollutants from stormwater could be extremely high and requiring the treatment of stormwater to GWQS for stormwater discharges to surface water was not considered in the evaluation of the proposed rules. (27)

RESPONSE: This rulemaking is amending NJPDES requirements related to direct discharges to ground water, and not discharges of stormwater to surface water. Therefore, the Department did not consider treatment costs associated with stormwater to surface water.

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Federal Standards Statement

N.J.S.A. 52:14B-1 et seq. (P.L. 1995, c. 65) requires State agencies that adopt, readopt, or amend State rules that exceed any Federal standards or requirements to include in the rulemaking document a Federal Standards Analysis.

The Department's SDWA rules at N.J.A.C. 7:10 incorporate by reference the National Regulations at 40 CFR 141, promulgated by the USEPA pursuant to the Federal Safe Drinking Water Act, 42 U.S.C. §§ 300f et seq., including all siting requirements, filtration and disinfection requirements, maximum contaminant levels, monitoring and analytical requirements, reporting requirements, public notification requirements, and recordkeeping requirements as the New Jersey primary drinking water rules, applicable to all public water systems. The Department's SDWA rules are, therefore, the Federal standards, except with respect to those areas for which the Department has determined, as authorized by the SDWA and allowed by the National Regulations, to establish New Jersey-specific requirements.

As described above, the Institute has recommended an MCL for PFOA of 0.014 µg/l and an MCL for PFOS of 0.013 µg/l. Pursuant to the SDWA, N.J.S.A. 58:12A-13, the Department is authorized to promulgate an MCL based on this recommendation. Under the existing rules, the Department has MCLs for 14 contaminants that are more stringent than the Federal standards and for seven contaminants for which no Federal standard has been established. With the addition of PFOA and PFOS, New Jersey will have nine State-established MCLs where no Federal standard exists.

The Institute's process for recommending MCLs is similar to the Federal process, with the differences noted below. The Institute considers three factors when recommending MCLs: health effects, technological ability to measure the contaminant level, and ability of existing treatment technologies to meet the MCL. For MCLs based on effects other than cancer (noncarcinogens), New Jersey's goal is the elimination of all adverse health effects resulting from ingestion, within the limits of

practicability and feasibility. With respect to carcinogens, the recommended MCL is to be established, within limits of medical, scientific, and technological feasibility, at a level which permits cancer in no more than one in one million persons ingesting that chemical for a lifetime. The health-based goal, known as the maximum contaminant level goal, for Federal MCLs for carcinogens is zero, and cost-benefit may be considered. The Institute evaluated the most current information available regarding PFOA and PFOS in drinking water before recommending MCLs to the Department.

The development of New Jersey-specific MCLs for PFOA and PFOS is necessary to protect public health. As stated in the Institute's Health Effects Subcommittee reports, PFOA and PFOS are persistent in humans with a half-life for elimination of several years, exposure to relatively low drinking water concentrations is expected to substantially increase human body burden and the toxicological effects in laboratory animal studies are relevant to humans.

PFOA was detected over the proposed MCL in 18 percent of public water systems sampled during UCMR3 and Department-initiated sampling as part of the 2006 and 2009-2010 Statewide occurrence study. PFOS was detected over the proposed MCL in nine percent of public water systems sampled. PFOA and PFOS were found more frequently in New Jersey than in other parts of the country based on results of sampling conducted pursuant to the UCMR3. While the Department has encouraged public water systems with elevated levels of PFOA and PFOS to continue to monitor and, where necessary, install treatment to remove these contaminants, those systems are under no obligation to comply with this request because an MCL has not yet been established. Therefore, without an adopted State-MCL, the Department cannot reduce exposure and protect public health. Through the Department's stakeholder process some water systems expressed support for the adoption of MCLs for unregulated contaminants because adopted rules provide predictability. Design of treatment systems in the absence

of a removal target, such as an MCL, can be both challenging and risky as the target is susceptible to change. Thus, systems are hesitant to invest in treatment without an MCL.

The PWTA rules, N.J.A.C. 7:9E, are not promulgated under the authority of, or in order to implement, comply with, or participate in any program established under Federal law or under a State statute that incorporates or refers to Federal law, Federal standards, or Federal requirements. Therefore, the Department has determined that a Federal standards analysis is not required.

The GWQS, N.J.A.C. 7:9C, are not promulgated under the authority of, or in order to implement, comply with, or participate in any program established under Federal law or under a State statute that incorporates or refers to Federal law, standards, or requirements. The proposed ground water quality standards for PFOA and PFOS do not exceed any Federal standards or requirements. The authority for the ground water quality standards comes solely from New Jersey law and has no Federal counterpart. Because the NJPDES rules require all discharges to ground water to comply with the GWQS, the NJPDES rules are proposed for amendment to be consistent with the GWQS rule changes. The proposed amendments to the NJPDES rules are governed by State statutes, including the New Jersey Water Pollution Control Act, which has no Federal counterpart, except regarding underground injection wells. The USEPA regulates injection wells under its rules for the Federal Underground Injection Control Program created pursuant to the Federal Safe Drinking Water Act. The proposed amendments to the NJPDES rules do not exceed Federal underground injection control mandates. Therefore, the Department has determined that a Federal standards analysis is not required.

The DPHS rules, N.J.A.C. 7:1E, are not promulgated under the authority of, or in order to implement, comply with, or participate in, any program established under Federal law, or under a State statute that incorporates or refers to Federal law, Federal standards, or Federal requirements. While there are Federal regulations promulgated pursuant to the Federal Water Pollution Control Act and the

Comprehensive Environmental Response, Compensation and Liability Act that govern discharge prevention and reporting that are generally analogous to the DPHS rules, PFOA and PFOS are not among the substances to which those Federal programs apply. The Department has determined that because PFOA and PFOS in the environment pose an unacceptable risk to public health, it is appropriate to include PFOA and PFOS on the DPHS Appendix A List of Hazardous Substances. Doing so will require responsible parties to notify the Department of a discharge and initiate remediation with a Licensed Site Remediation Professional and require owners and operators of industrial establishments who are liable under ISRA to, among other things, undertake an investigation of their industrial establishment and remediate any discharges of PFOA or PFOS that are discovered prior to their sale or transfer or upon cessation of business operations. In addition, including PFOA and PFOS on the DPHS Appendix A List of Hazardous Substances will enable the Department to, in accordance with the Spill Act, direct persons with Spill Act liability to remediate discharges of PFOA and PFOS, use available hazardous substance-based funding sources, as necessary, to conduct remediation of PFOA and PFOS, and undertake cost recovery actions against the party responsible for the discharge.

Full text of the adoption follows (additions to proposal indicated in boldface with asterisks ***thus***; deletions from proposal indicated in brackets with asterisks *[thus]*):

CHAPTER 9E

PRIVATE WELL TESTING ACT RULES

SUBCHAPTER 2. SAMPLING AND TESTING REQUIREMENTS

7:9E-2.1 Parameters for which testing is required

(a) Each water sample shall be analyzed for the following parameters:

NOTE: THIS IS A COURTESY COPY OF THIS RULE ADOPTION. THE OFFICIAL VERSION WILL BE PUBLISHED IN THE JUNE 1, 2020 NEW JERSEY REGISTER. SHOULD THERE BE ANY DISCREPANCIES BETWEEN THIS TEXT AND THE OFFICIAL VERSION OF THE ADOPTION, THE OFFICIAL VERSION WILL GOVERN.

1.-9. (No change.)

10. Gross alpha particle activity, determined using the 48 Hour Rapid Gross Alpha Test, in accordance with N.J.A.C. 7:18;

11. As of March 3, 2019, the synthetic organic compounds, 1,2,3-trichloropropane, ethylene dibromide, and 1,2-dibromo-3-chloropropane; and

12. As of *[(18 months after the effective date of this amendment)]* ***December 1, 2021***, the per- and polyfluoroalkyl substances perfluorononanoic acid (PFNA), perfluorooctanoic acid (PFOA), and perfluorooctanesulfonic acid (PFOS).

(b)-(c) (No change.)

CHAPTER 10

SAFE DRINKING WATER ACT

SUBCHAPTER 5. STATE PRIMARY DRINKING WATER REGULATIONS

7:10-5.2 Discretionary changes to National Regulations

(a) In accordance with the discretionary authority permitted by the National Regulations, for compliance with the State primary drinking water regulations, the following shall apply:

1.- 4. (No change.)

5. MCLs for the State-regulated per- and polyfluoroalkyl substances perfluorononanoic acid (PFNA), perfluorooctanoic acid (PFOA), and perfluorooctanesulfonic acid (PFOS) shall be those established at (a)5i, ii, and iii below. Monitoring requirements for PFNA, PFOA, and PFOS shall be those established under the National Regulations at 40 CFR 141.24(f) and at (a)7 below. For PFNA, the conditions at (a)5i apply. For PFOA, the conditions at (a)5ii apply. For PFOS, the conditions at (a)5iii apply.

i. (No change from proposal.)

ii. For PFOA, the MCL shall be 0.014 µg/l. Monitoring requirements shall begin as set forth at (a)5ii(1) below and are subject to the conditions at (a)5ii(2) and (3) below.

(1)-(2) (No change from proposal.)

(3) A public community water system or a public nontransient noncommunity water system may submit monitoring data for PFOA to the Department for a determination of whether the system may reduce monitoring frequency to an annual basis provided:

(A) (No change from proposal.)

(B) The monitoring data are reported to the Department in accordance with N.J.A.C. 7:10-5.4 on or before *[(the effective date of this amendment)]* ***June 1, 2020***.

iii. For PFOS, the MCL shall be 0.013 µg/l. Monitoring requirements shall begin as set forth at (a)5iii(1) below and are subject to the conditions at (a)5iii(2) and (3) below.

(1)-(2) (No change from proposal.)

(3) A public community water system or a public nontransient noncommunity water system may submit monitoring data for PFOS to the Department for a determination whether the system may reduce monitoring frequency to an annual basis provided:

(A) (No change from proposal.)

(B) The monitoring data are reported to the Department in accordance with N.J.A.C. 7:10-5.4 on or before *[(the effective date of this amendment)]* ***June 1, 2020***.

6. (No change.)

7.-13. (No change from proposal.)

(b) (No change from proposal.)

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SUBCHAPTER 12. STANDARDS FOR THE CONSTRUCTION OF PUBLIC NONCOMMUNITY WATER SYSTEMS AND NONPUBLIC WATER SYSTEMS

7:10-12.30 Water quality analysis and treatment

(a)-(b) (No change from proposal.)

(c) Upon completion of construction of a water system, the owner of a nonpublic water system shall sample and analyze the raw water from the system for the parameters listed at (c)1 through 12 below. The administrative authority may require sampling and analysis for inorganic chemicals, volatile organic compounds, and/or radionuclides**,** as appropriate**,** based on the region and the aquifer in which the water source is located.

1.-9. (No change.)

10. As of *[(18 months after the effective date of this amendment)]* ***December 1, 2021***, the per- and polyfluoroalkyl substances PFNA, PFOA, and PFOS;

11.-12. (No change from proposal.)

(d) – (i) (No change.)